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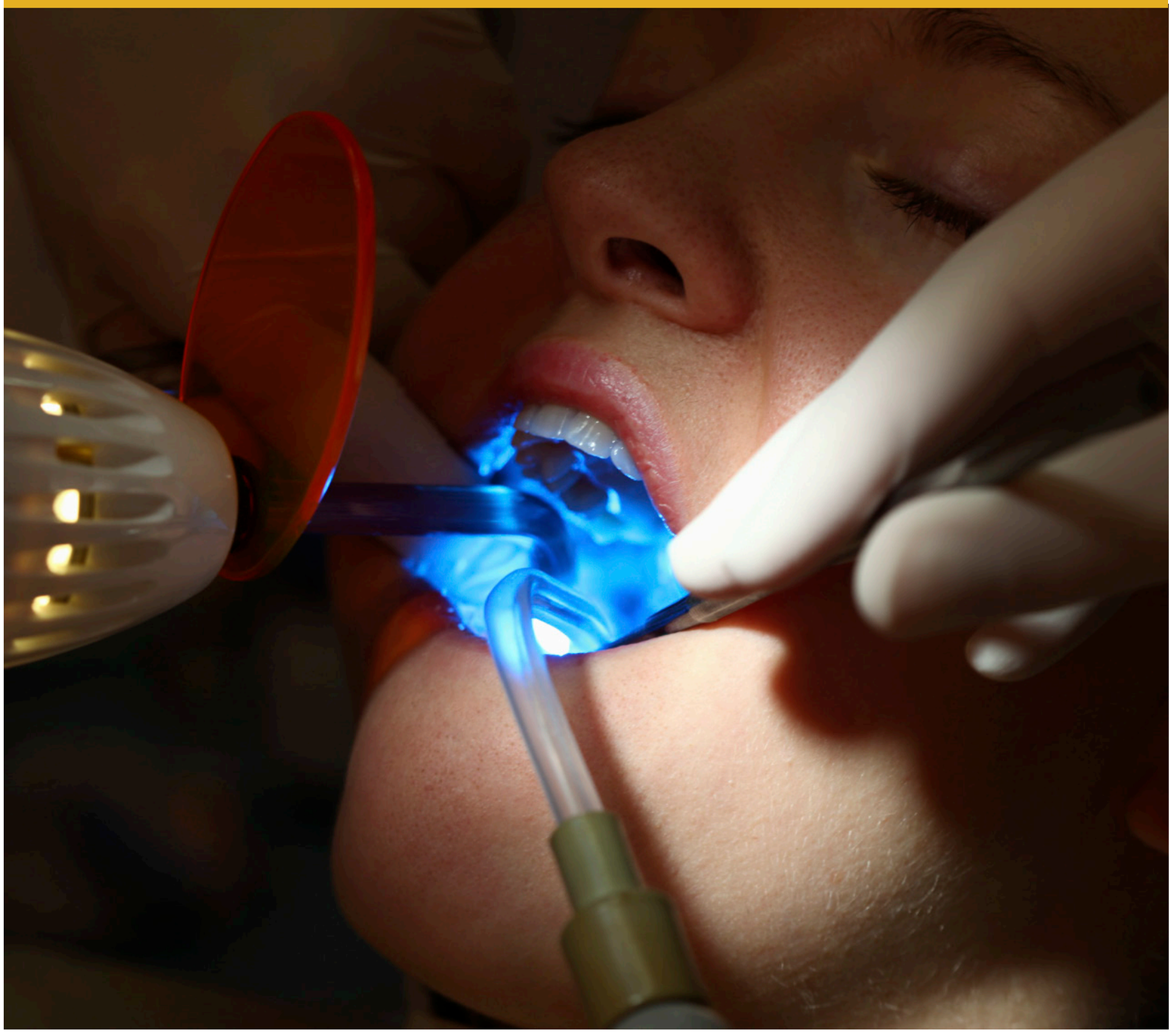
MARCH 2012

Volume 6, Issue 3: Orthopaedics, Oral & Maxillofacial



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Volume 6, Issue 3

Foundation Years Journal

Foundation Years Journal is an international peer-viewed journal which seeks to be the pre-eminent journal in the field of patient safety and clinical practice for Foundation Years' doctors and educators. The Journal welcomes papers on any aspect of health care and medical education which will be of benefit to doctors in the Foundation training grade in the UK or international equivalents.

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FOUNDATION YEARS JOURNAL 2012

Volume 6, Issue 3

Volume 6, Issue 3: Orthopaedics, Oral & Maxillofacial

Foundation Years Journal is the ONLY journal for Foundation Years, doctors and educators, specifically written according to the MMC curriculum. It focuses on one or two medical specialties per month and each issue delivers practical and informative articles tailored to the needs of junior doctors. The journal closely follows the Foundation Years syllabus to provide the best educational value for junior doctors. In addition to good clinical and acute care articles, assessment questions give junior doctors the chance to gauge their learning. Each issue provides comprehensive clinical cases for trainees as well as practical teaching assessments for educators. Readers will benefit from:

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Editorial For Orthopaedics, Oral & Maxillofacial Issue Of Foundation Years Journal 2012

Many persons like to hold a book or journal in the hand. The ability to browse by turning pages, for those to annotate, who are prepared to deface paper copies, to read without needing to find electronic apparatus to enable viewing (whether by computer, by Kindle device or otherwise), all are powerful stimuli to keep to conventional hard copy, paper publications. The feel of a book, the smell of the paper (maybe the binding), the colourful printing, and the variations in font and style all contribute to this sensual experience. However, paper copies become dated and cannot easily be amended except in loose-leaf form where they lose much of their aesthetic appeal. They are more expensive to produce at the point of the user. They decay with use, whether aided by fingers, thumbs or by mice, and they are bulky for publishers and readers to transport. Hence, this trends towards electronic publishing. Electronic journals have many advantages and can be accessed from computers worldwide. This journal offers all of these advantages and on this occasion brings to readers aspects of important neurological topics relevant to Foundation Years practitioners.

The neurosciences, of which everyday clinical neurology forms a part, have made amazing progress over the last couple of decades. The interactions between laboratory and clinical research, and with clinical medicine that deals with illness in patients at its most elementary level, have contributed to these advances. However, sometimes research and cutting edge thinking from the laboratory is difficult to apply to some of the immediate clinical problems exhibited by patients. Common sense (whatever that is) and thinking is needed with acute problems and so is rapid decision making. Some of the topics covered in this issue deal with acute medicine, and neurology is now very much part of this since nearly one fifth of those admitted acutely have neurological problems, and others with less acute matters still get admitted to hospital. Papers published here express some of the most important points that Foundation Years doctors experience during their everyday duties, lessons they wish to share with others in order to help prevent mishaps.

Indeed, such practitioners are encouraged to submit to this journal. There is so much to be learned from our everyday activities and our patients are in many ways our best teachers, using their symptoms and signs to make us think. It is in many ways a moral imperative to share this information with others and to publish for the widest circulation. Specific lessons that may be drawn from the papers in this issue of the Foundation Years Journal, include epilepsy and the causes of blackouts together with some useful tips on the use of the EEG in diagnosis, an important supportive test in some patients.

Stroke is now an emergency in more ways than previously (since more can be done), a brain attack that needs handling acutely and which can result from venous sinus thrombosis, two more areas covered in this journal. The techniques of lumbar puncture are still important although much that was investigated previously by this technique now is revealed by the increasingly complicated imaging processes that have become available.

Acute neuromuscular weakness is a further presenting feature that has many causes and this condition may be quite puzzling in many patients. Increasingly complicated drugs and drug regimes may lead to toxicity, an important cause of disability that can easily be overlooked; baclofen is a useful drug for spasticity and intoxication is disabling. Trigeminal neuralgia can be treated in many ways; not all being effective and a paper on this topic should help guide those who deal with its early manifestations.

And what of that imperative to publish? Here we are guided in the values of clinic letters and of the role of the doctor as educator. All very important stuff, hopefully interesting, certainly enlightening, and without doubt we hope a stimulus for readers to provide further papers dealing with the many topics in neurology that may perplex all of us including those working in the Foundation Years.

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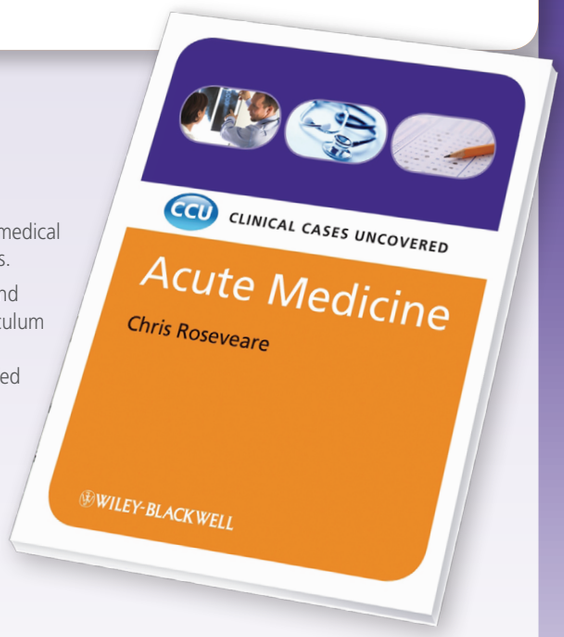
Acute Medicine

CLINICAL CASES UNCOVERED

Chris Roseveare,
Southampton General Hospital



Acute Medicine is the central part of foundation and specialist general medical training and is one of the most rapidly expanding UK hospital specialties.

Acute Medicine: Clinical Cases Uncovered combines patient cases and outcomes, drawn from real-life experiences, with reference to the curriculum for Training in General (Acute) Medicine. It provides self-assessment MCQs, EMQs and SAQs to give Foundation Programme doctors and allied healthcare professionals the perfect preparation for life on the wards.

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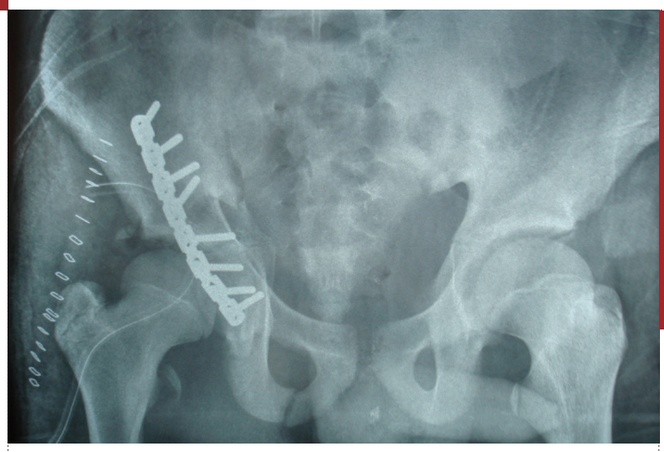
WILEY-BLACKWELL

MANAGEMENT OF ACETABULAR FRACTURES & PELVIC INJURIES

R Kakkar, J Cochrane and P Banaszkiewicz



Management of acetabular fractures and pelvic injuries.
Patient Management.

Abstract

The majority of pelvic ring fractures are due to high-energy blunt trauma. Disruption of the retroperitoneal arteries and venous plexus with compromise of pelvic ring integrity can lead to massive blood loss. We provide a case presentation of such an injury to better guide Foundation doctors in their management of such injuries.

Fractures of the acetabulum involve the joint surface of the pelvis. In terms of presentation and management these are different injuries compared to pelvic ring fractures and this is explored in the case presentation.

History

A 42-year-old previously healthy woman was admitted to the emergency department following a road traffic accident. She was an unrestrained backseat passenger in a stationary car which was hit from behind by a van travelling at 40-50mph resulting in her being thrown forward between the front two seats. Pre-hospital examination documented chest and abdominal pain and right sided pelvic and thigh pain and an obvious rotational deformity of the leg.

1. What would be your initial management?

The initial management would consist of a primary survey according to ATLS protocol by the trauma team which should consist of at least an A+E consultant/ Middle grade, Orthopaedic surgeon, General surgeon and Anaesthetic doctor with assistant.

Primary survey

The purpose of the ATLS primary survey is to quickly identify immediate life threatening conditions in order of priority and deal with them before addressing the next system.

Airway and cervical spine control: the patient had a patent airway and c-spine was immobilised at the scene.

Breathing: The patient was complaining of shortness of breath with difficulty taking deep inspirations. On examination respiratory rate was 18/min and chest sounded clear bilaterally without any obvious pathology.

Circulation: HR 140bpm, BP 100/40 indicating grade 3 haemorrhagic shock. No obvious external sites for blood loss therefore sites for occult blood loss (chest, pelvis and abdomen) were checked. Pelvis appeared intact but tender on the right side. The right thigh was swollen, with shortening and external rotational deformity. Abdomen was intact, with abrasions to left upper quadrant which was mildly tender but soft on palpation.

Management of shock

This would require: two large calibre intravenous access cannulae with aggressive fluid resuscitation including blood. Temporary pelvic stabilisation with pelvic binder/sheet and splinting of the right leg to decrease the volume of the space where the patient can bleed. As the pelvis can be compared to a sphere in terms of volumetric dimensions, the volume of the pelvis is directly proportional to r^3, where r is the radius of the pelvis. Decreasing the radius has a significant effect on reducing pelvic volume and therefore reduces the risk exsanguinating life-threatening haemorrhage.

Disability: GCS was 15/15 throughout assessment with pupils equal and reactive. A left sided hemiparesis, with reduced sensation and power was noticed. No obvious bony tenderness to the whole spine but tender over posterior sacrum and SI joints. Per rectum examination was normal but blood was found in the urethral meatus.

2. What investigations would you request?

Initial investigations:

Bloods

Full blood count, Urea and electrolytes, Liver function tests, Coagulation screen, Group and save with cross match

Imaging

Chest x-ray - No abnormalities demonstrated

Pelvic x-ray - Open book pelvic fracture
Right posterior column acetabular fracture
Dislocated right hip and femoral head fracture
Posterior acetabular wall fracture

MANAGEMENT OF ACETABULAR FRACTURES & PELVIC INJURIES

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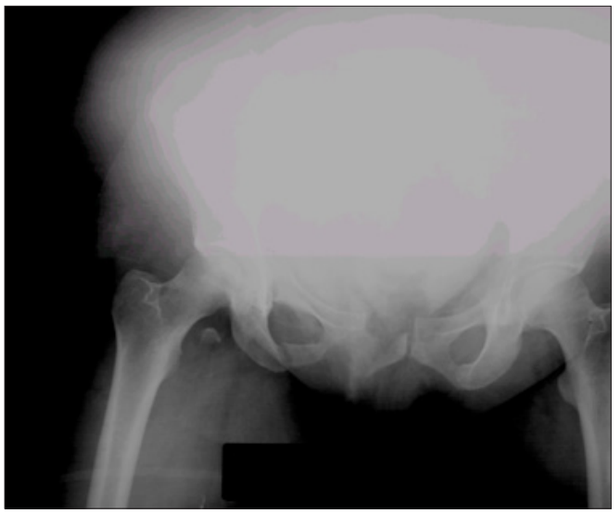

Figure 1: Anteroposterior (AP) radiograph demonstrating open book pelvis.

FAST scan - Used to assess occult blood loss in the abdomen but in this patient the views were insufficient to exclude visceral injury. Therefore a CT abdomen should be performed if the patient is haemodynamically stable. CT abdomen showed a small laceration to the liver and a small splenic laceration but was unable to rule out free fluid in the abdomen.

CT head - Left hemiparesis noted on initial examination which may suggest intracerebral haemorrhage.

CT spine - no spinal fractures noted and thus spine cleared.

3. What other injuries can be associated with a pelvic/acetabular fracture?

This patient had a urethral injury and also suffered acute renal failure and a right middle cerebral artery infarct and right optic neuropathy due to right carotid dissection. Furthermore, she had fat embolus syndrome and TRALI (transfusion-related lung injury) during her ITU stay.

Urethral injuries occur in up to 10% of the pelvic fractures but this is more common in males and requires urgent urology referral. Urinary bladder injuries are also seen in 20% of the cases although this patient did not have any. If significant damage to the bladder or urethra is suspected a urethrogram should be performed prior to catheterisation

Other injuries to look out for are bowel and perineal/rectal injuries. Small bowel mesenteric or shearing tears (leading to perforation or infarction) may present with abdominal rigidity, loss of bowel sound and abdominal distension. A careful neurological examination should be performed in conscious patients to rule out lumbosacral plexus and nerve root injuries.

4. What would be the priorities of management now?

In this patient's case, airway was satisfactory, cervical spine was clear, breathing was not a problem but circulation was still a issue despite fluid resuscitation as the unstable pelvic injury which was the source of bleeding had not been addressed. Therefore the pelvis was stabilised with an external fixator and exploratory laparotomy carried out by general surgeons to exclude an intra-abdominal source of bleeding. No obvious abdominal source was found and 4 packs were left in situ as a precaution.

The usual source of the retroperitoneal bleed in pelvic injuries is due to bleeding from the posterior pelvic venous plexus but in some cases the source can be arterial, where angiographic embolisation may play a role. This is specially the case if circulation continues to be a problem despite closing of the pelvic volume by an external fixator as this indicates continued haemorrhage due to arterial bleeding.

Further evaluation of pelvic and acetabular injuries revealed a very unstable right hip due to a posterior acetabular column and posterior wall fracture with a fractured femoral head. This was stabilised with leg traction to keep the femoral head in the acetabulum. The patient was transferred to ITU for further care. After physiological stabilisation the patient was transferred to the regional centre for definitive pelvic stabilisation surgery.

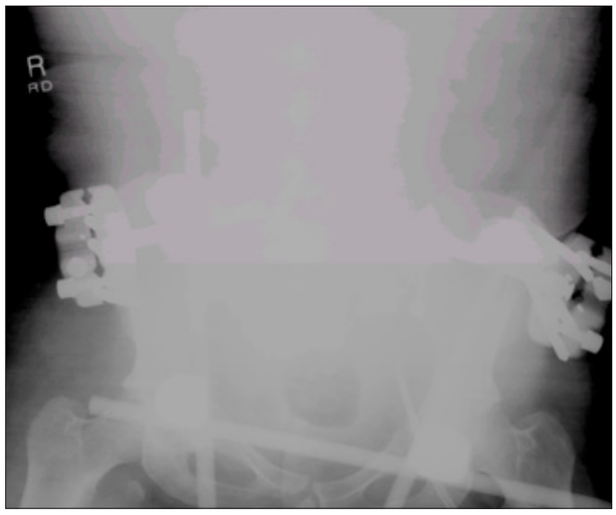

Figure 2. AP pelvis after application of external fixator.

MANAGEMENT OF ACETABULAR FRACTURES & PELVIC INJURIES

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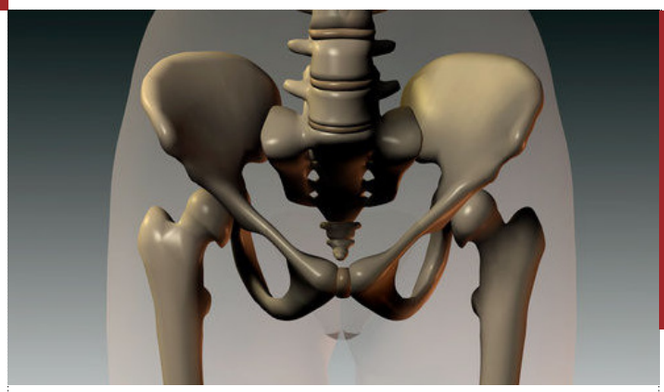

Management of acetabular fractures and pelvic injuries.
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5. How would you further evaluate the pelvic/acetabular injuries?

Pelvic Fractures

Radiographic evaluation of pelvic fractures
The AP pelvis is the standard initial radiograph. Markers of pelvic instability include avulsion fracture of the transverse process of the 5th lumbar vertebra, ischial spine (sacrospinous ligament) and lateral border of the sacrum (sacrotuberous ligament). Further information can be obtained by performing Inlet and Outlet views of the pelvis.

Inlet view - this is taken with the patient supine and the x-ray tube directed 60° caudally perpendicular to the pelvic brim. This is useful to determine AP and rotational displacement of the SI joint, sacrum or the iliac wing.
Outlet view - taken with patient supine and tube directed 45° cephalad. This view allows assessment of vertical displacement of hemipelvis and also shows any widening of the SI joint, sacral fractures and foramina disruption.
CT scan - allows assessment of posterior pelvis accurately and is the modality of choice.

Classification of pelvic fractures

Pelvic fractures are most commonly described using one of two classification systems.

1. The Tile classification system is based on the integrity of the posterior sacroiliac complex.

- In type A injuries, the sacroiliac complex is intact. The pelvic ring has a stable fracture that can be managed nonoperatively.

- Type B injuries are caused by either external or internal rotational forces resulting in partial disruption of the posterior sacroiliac complex. These are often unstable. Therefore these injuries are rotationally unstable but vertically stable.

- Type C injuries are characterized by complete disruption of the posterior sacroiliac complex and are both rotationally and vertically unstable. These injuries are the result of great force, usually from a motor vehicle crash, fall from a height, or severe compression. They are typically associated with massive haemorrhage.

2. The Young and Burgess Classification system is based on mechanism of injury:

- Lateral compression
- Anteroposterior compression
- Vertical shear
- Combination of forces

Lateral compression (LC) fractures involve transverse fractures of the pubic rami, either ipsilateral or contralateral to a posterior injury.

- Grade I - Associated sacral compression on side of impact
- Grade II - Associated posterior iliac ("crescent") fracture on side of impact
- Grade III - Associated contralateral sacroiliac joint injury

Anterior-posterior compression (APC) fractures, involve symphyseal diastasis or longitudinal rami fracture

- Grade I - Associated widening (slight) of pubic symphysis or of the anterior sacroiliac (SI) joint, while sacrotuberous, sacrospinous, and posterior SI ligaments remain intact

- Grade II - Associated widening of the anterior SI joint caused by disruption of the anterior SI, sacrotuberous, and sacrospinous ligaments; posterior SI ligaments remain intact

- Grade III (open book) - Complete SI joint disruption with lateral displacement and disrupted anterior SI, sacrotuberous, sacrospinous, and posterior SI ligaments

Vertical shear (VS) involves symphyseal diastasis or vertical displacement anteriorly and posteriorly, which is usually through the SI joint, though occasionally through the iliac wing or sacrum.

Combined mechanical (CM) fractures involve a combination of these injury patterns, with LC/VS being the most common.

In this patient's case posterior sacro iliac tenderness indicated disruption of the posterior complex and thus an unstable injury. This injury along with the open book pattern indicated an APC type III.

MANAGEMENT OF ACETABULAR FRACTURES & PELVIC INJURIES

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Acetabular Fractures

Most acetabular fractures occur after significant trauma secondary to either a motor vehicle accident or a high-velocity fall. The direction and magnitude of the force, as well as the position of the femoral head, determine the pattern of acetabular injury.

The articular surface of the acetabulum is in the shape of an inverted horseshoe with the anterior and posterior halves forming parts of the anterior and posterior columns respectively.

The anterior column of the acetabulum includes most of the iliac wing, the anterior acetabulum, and the superior pubic ramus while the posterior column begins at the sciatic notch and includes the posterior portion of the acetabulum and the ischium.

Radiographic evaluation of acetabular fractures

Evaluation of acetabular fractures on an AP pelvis film is made easier by looking for 6 structures.

1. The iliopectineal, or iliopubic, line is the landmark for the anterior column. It begins at the sciatic notch and travels along the superior pubic ramus to the symphysis pubis.
2. The ilioischial line demarcates the posterior column. It also begins at the sciatic notch, coursing inferiorly to the medial border of the ischium/lateral border of obturator foramen. The ilioischial line should pass through the acetabular teardrop on the AP view. If it does not overlap the teardrop, the ilioischial line and, thus, the posterior column are disrupted.
3. Teardrop is a composite shadow of the inferomedial structures that compose the acetabulum and is important to determine central impaction in acetabular injuries.
4. Superior weight bearing area/Dome
5. Anterior acetabular line, disruption of this line indicates anterior wall fracture and
6. Posterior acetabular line is easier to see as it lies more lateral in comparison to the anterior acetabular line. Disruption indicates posterior wall fracture.

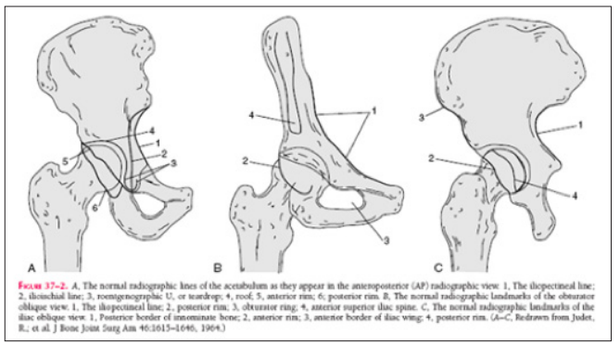

FIGURE 37–2. *A*, The normal radiographic lines of the acetabulum as they appear in the anteroposterior (AP) radiographic view. 1, The iliopectineal line; 2, ilioischial line; 3, roentgenographic U, or teardrop; 4, roof; 5, anterior rim; 6, posterior rim. *B*, The normal radiographic landmarks of the obturator oblique view. 1, The iliopectineal line; 2, posterior rim; 3, obturator ring; 4, anterior superior iliac spine. *C*, The normal radiographic landmarks of the iliac oblique view. 1, Posterior border of innominate bone; 2, anterior rim; 3, anterior border of iliac wing; 4, posterior rim. (*A–C*, Redrawn from Judet, R., et al. J Bone Joint Surg Am 46:1615–1646, 1964.)

Figure 3: Radiographic evaluation acetabular fractures.

Further evaluation of acetabular injuries can be gained by obtaining the Judet Iliac and Obturator oblique views.

An Iliac oblique view best demonstrates injuries to the posterior column (ilioischial line is seen fully), anterior wall of the acetabulum and the iliac wing. The iliac wing is a part of the anterior column and thus fractures here indicate anterior column injury.

The Obturator oblique view better depicts the anterior column, posterior acetabular wall, and the obturator ring. The integrity of the obturator ring is an important feature to recognize. Certain fracture patterns (such as those of the column and T-shaped fractures) characteristically include fractures through the obturator ring.

A CT scan provides detailed information about the fracture line orientation, size and position of the column fragments and the degree of comminution. This is the modality of choice along with Judet views in helping plan operative management of acetabular fractures. A CT scan slice obtained at the level of the dome shows that transverse-type acetabular fractures have a vertical (sagittal) orientation. Column-type fractures have a horizontal (coronal) orientation and wall fractures have an oblique orientation.

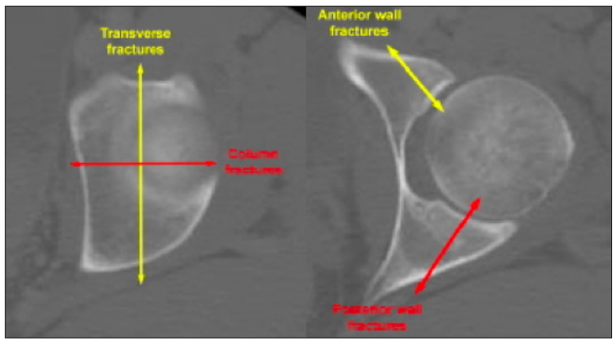

Figure 4: CT scan acetabulum demonstrating fracture lines.

Classification of acetabular fractures

Fractures of the acetabulum are most commonly classified according to the system described by Judet and Letournel. The system is based on the orientation of the fractures and the structures involved on the radiographs.
This classification divides acetabular fractures into 5 elemental and 5 associated fracture types. Elementary fractures account for 20% of acetabular fractures and involve 1 primary fracture plane while associated types account for 80% of acetabular fractures and involve more than 1 fracture plane usually combinations of the elementary fracture patterns.

Elementary fractures include

1. Anterior wall,
2. Posterior wall,
3. Anterior column,
4. Posterior column, and
5. Transverse fractures.

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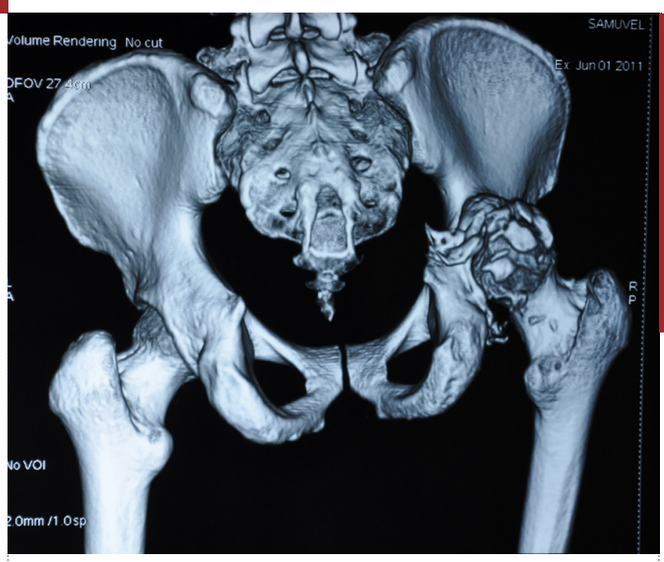

Associated patterns include

1. Both-column fractures,
2. Posterior column fractures with posterior wall fractures,
3. Transverse fractures with posterior wall fractures,
4. T-shaped fractures, and
5. Anterior column fractures with posterior hemitransverse fractures.

Our patient had an associated type fracture as both the posterior column and posterior wall were involved.

6. How would you definitively manage these fractures?

Pelvic injuries can be managed either non-operatively or operatively depending on the type of fracture and instability present. Most Lateral Compression-I and APC-I fractures can be managed non-operatively. Operative interventions include external fixation or internal fixation. Internal fixation can take the form of screw fixation for SI joint instability and anterior plate stabilisation for pubic symphysis diastasis.

Similarly, acetabular injuries can be managed non-operatively and operatively. In general if femoral head congruency is maintained with fracture displacement less than 2-3mm and a stable hip joint, non-operative treatment is chosen.

Indications for acetabular fixation include

- Significant articular fracture displacement
- Inability to maintain joint congruency without traction
- Large posterior wall fragment
- Intraarticular loose body
- Lack of parallelism between the femoral head and acetabular roof

Management of acetabular fractures and pelvic injuries.
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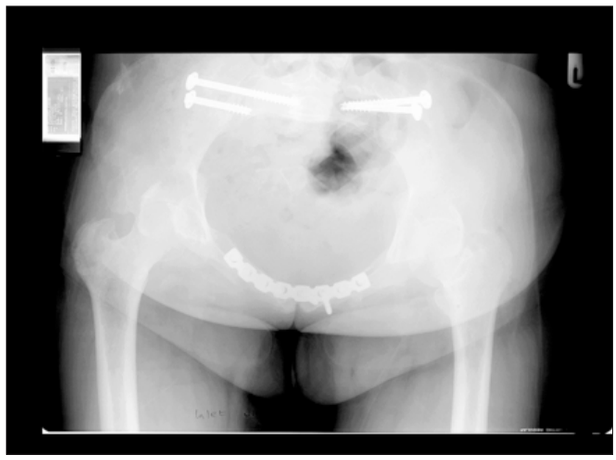
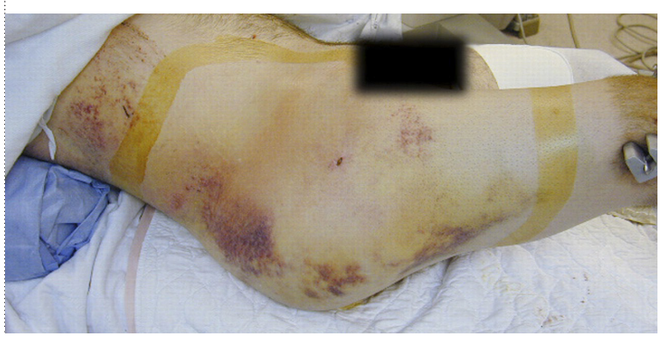
Surgery consists of internal fixation with plates and screws as deemed necessary.



In this woman's case, definitive management consisted of anterior pubic symphysis fixation with an 8-hole plate and percutaneous sacroiliac screws posteriorly and stabilisation of posterior acetabular wall using screws to maintain a relatively stable congruent joint.

7. What are the complications associated with these injuries and their management?

Pelvic and acetabular injuries are most commonly seen in victims of high velocity trauma and are often associated with other life threatening injuries. In general pelvic injuries tend to be life threatening while acetabular injuries pose more of a threat to the function of the limb.



MANAGEMENT OF ACETABULAR FRACTURES & PELVIC INJURIES

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The most common complications with these injuries are:

1. Infection - up to 25% incidence has been quoted. The presence of a Morel-Lavalle lesion (internal skin shear degloving injury) is a bad prognostic factor as up to a third of cases get bacterial colonisation.
2. Mortality - if haemodynamically stable there a 3% incidence but in unstable patients a 38% incidence has been quoted.
3. Thromboembolism - due to disruption of the venous vessels and immobilisation. Therefore the patients should be kept on anticoagulants.
4. Malunion - especially in acetabular fractures which may result in degenerative posttraumatic hip osteoarthritis. Malunion of pelvic fractures may result in leg length inequalities, low back/SI joint pain and pelvic outlet obstruction, which can be a significant problem in young females.
5. Nerve injury - sciatic, femoral and superior gluteal nerves along with sacral roots can be injured during operative stabilisation of these fractures via various surgical approaches.
6. Avascular necrosis of femoral head- approximately 7% incidence.
7. Chondrolysis of femoral head cartilage-leading to osteoarthritis.

MCQs - True or False?

1. Urethral injury complicate about 10% of the pelvic fractures.

2. Mortality of pelvic ring fractures is 1%.

3. Elemental fractures account for 20% of acetabular fractures.

4. The internal (obturator) Oblique Judet view shows the iliopectineal line, anterior column of the pelvis and posterior wall.

5. The presence of a Morel-Lavalle lesion is a good prognostic sign for wound healing.

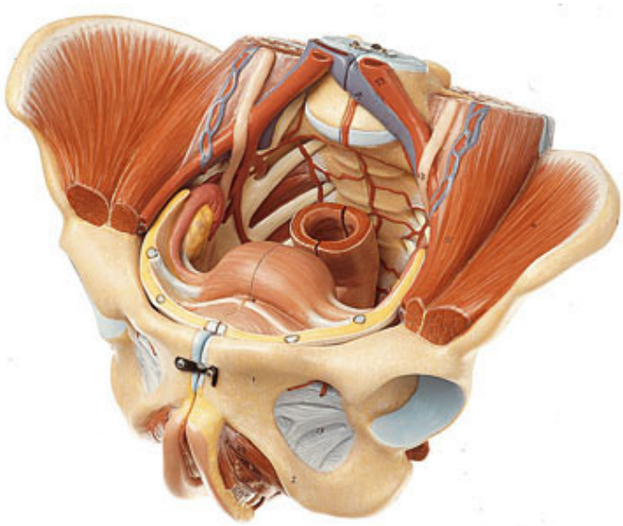
Answers

1. True.
In males the incidence is higher nearer 15%. Urethral injury should be suspected if there is;

- Blood at the penile meatus
- Blood in the scrotum
- A high riding or non palpable prostrate
- Perineal ecchymosis

2. False.
The mortality of pelvic ring fractures is between 3-38% dependent on associated injuries and blood loss

3. True.



4. True.
The patient is supine with the uninvolved side of the pelvis rotated anteriorl 45° and the beam directed vertically towards the affected hip

5. False.
The Morel-Lavalle lesion is a closed degloving injury, which commonly occurs over the greater trochanter. The subcutaneous tissue is torn from the underlying fascia creating a cavity that places the tissue at risk for infection and/or poor wound healing

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ORTHOPAEDIC RADIOLOGY QUIZ

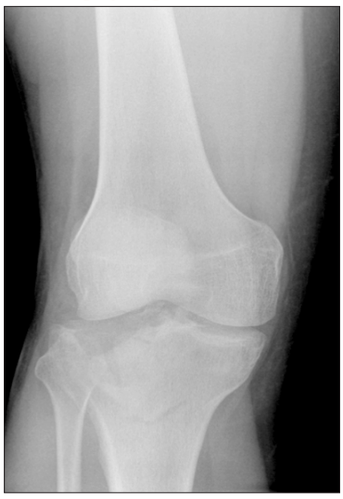
D Ferguson, S Kothapalli & K Haendlmayer

Orthopaedic Radiology Quiz.
Test Yourself.

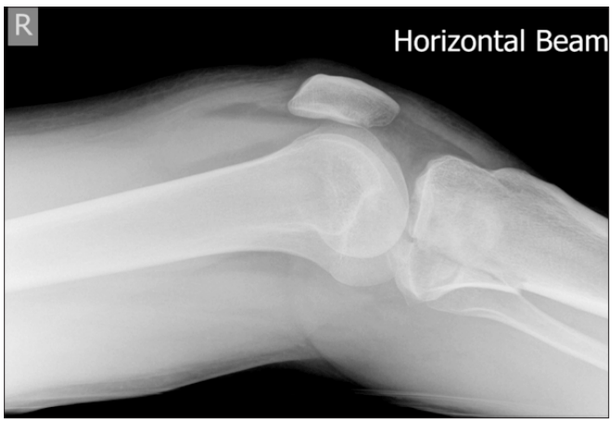
A 27 year old man presents to A&E following a motor vehicle accident. He complains of severe pain and swelling in the right knee, and is unable to weight bear.

On examination, his knee is tender, swollen and has severely restricted range of movement. There are no visible wounds around the knee. Antero-posterior and lateral view radiographs of the right knee are performed.

AP Radiograph right knee



Lateral radiograph right knee



1st Questions

Question 1.1
What are the findings from the AP radiograph?

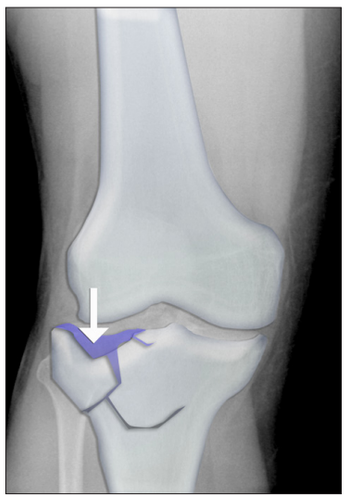
Question 1.2
Describe a typical feature seen on the lateral radiograph.

Question 1.3
What is the immediate management plan?
Does this patient need further investigations?

Question 1.4
What treatment options are available for this fracture?

1st Answers

Answer 1.1



This is an AP view of right knee joint showing an intra-articular displaced lateral tibial plateau fracture. It is classified as a Schatzker type II tibial plateau fracture because there is a split depression fracture of the lateral tibial plateau (white arrow). The shaded blue area represents depression of the joint surface.

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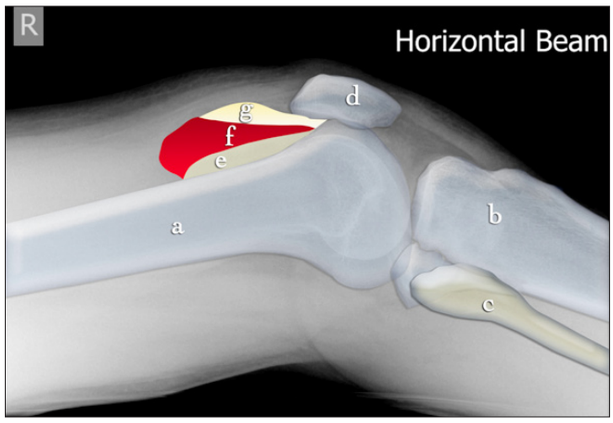
Answer 1.2



This lateral radiograph shows widening of the supra-patella pouch indicating a post-traumatic knee joint effusion, proximal to the patella (d). There is a lipohaemarthrosis, which suggests an intra-articular fracture will be present somewhere within the knee. When the patient lies flat for the cross-table lateral radiograph, the less dense fat globules (g) float above the more dense blood (f). There is another darker area below the blood which is normal fatty tissue (e) adherent to the anterior surface of the femur (a). The fracture of the tibia (b) can be seen in the posterior third, behind the fibula (c).

Answer 1.3

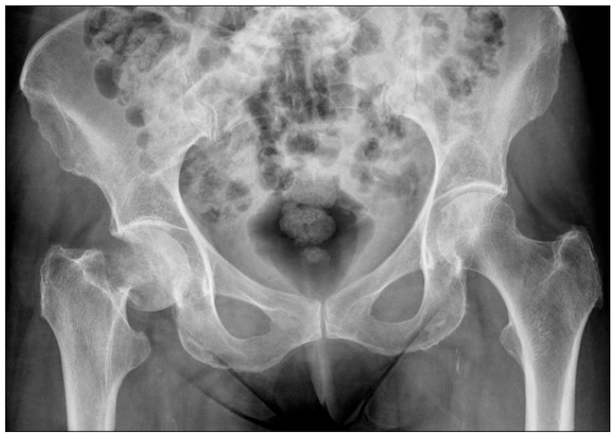
Initial management of this type of injury should be in accordance with Advanced Trauma Life Support guidelines[1]. These trauma patients may have other more life threatening problems that should be dealt with first. The limb should be monitored for compartment syndrome, elevated to help reduce the swelling, and splinted to protect against further damage. Computed tomography scans are useful for looking at the bony fracture configuration, whereas magnetic resonance imaging is more accurate at assessing associated soft tissue injuries. Investigations should always be guided by findings from a thorough history and examination.

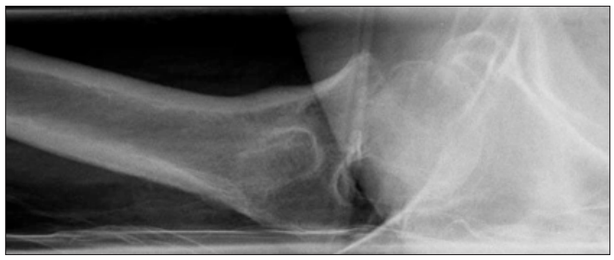
Answer 1.4

This kind of fracture is best treated with an operation. Intra-articular fractures should be anatomically reduced and stabilized. A step in the articular surface greater than 5mm has a high risk of causing arthritis to develop[2]. Most of these fractures can be fixed with a buttress plate, with the tibial plateau elevated to the normal level and bone graft packed into any void underneath. In this particular case, the buttress plate should be placed relatively posterior through a posterolateral approach to the knee. Care must be taken not to damage the common peroneal nerve. Severely comminuted and complex fractures may be more suitable for a circular frame (a form of external fixation).

Case 2

An 85 year old lady falls in her own home, landing on her right hip. It is too painful for her to walk. On examination, the right leg looks shorter than the left, and is externally rotated. There is an antero-posterior (AP) pelvis and lateral radiograph available.





2nd Questions

Question 2.1

What are the findings from the AP pelvis radiograph?

Question 2.2

What are the findings from the lateral right hip radiograph?

Question 2.3

What is the choice of treatment and why?

Question 2.4

What are the complications of this fracture?

ORTHOPAEDIC RADIOLOGY QUIZ

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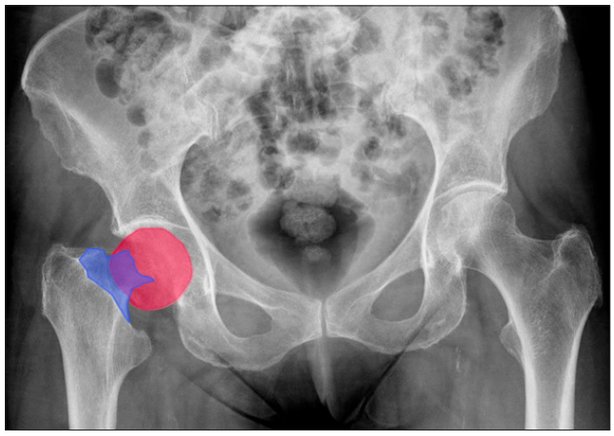
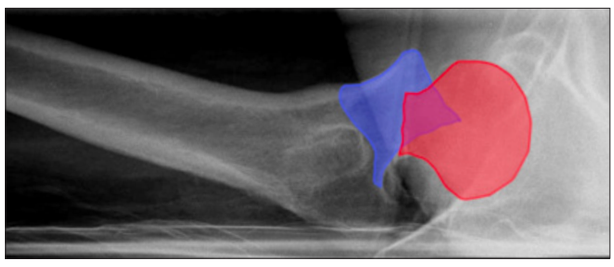
Orthopaedic Radiology Quiz.
Test Yourself.

2nd Answers



Answer 2.1

This AP pelvis radiograph shows an intracapsular fracture of right femoral neck with superolateral displacement and external rotation of the femoral shaft. The lesser trochanter on the right side is more pronounced than on the left indicating external rotation. This occurs due to the unopposed pull of iliopsoas. Superolateral displacement, or shortening, occurs because of the strong pull of both iliopsoas and the hip abductors. The capsule surrounds the neck (shaded blue) and contains the main blood supply to the femoral head in this age group. The head (shaded red) has completely separated from the femoral shaft.



Answer 2.2

This is a lateral view radiograph of right hip showing anterior displacement of the remaining femoral neck with the femoral head completely free and lying in the acetabulum.

Answer 2.3

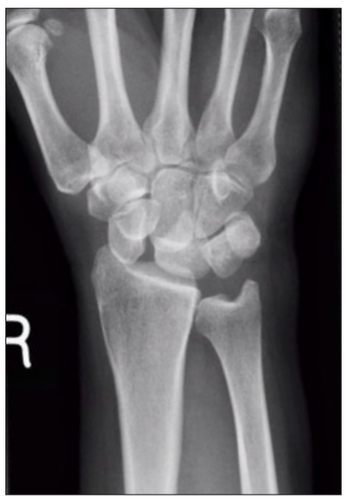
The most common method of treating this kind of fragility fracture in the United Kingdom is by inserting a form of hip arthroplasty. This can be either hemiarthroplasty, or total hip replacement. There are many designs and techniques for these kinds of implants, such as whether cement should be used or not. The National Institute for Health and Clinical Excellence has issued guidelines to help surgeons treat hip fractures effectively[3].

Answer 2.4

This group of patients unfortunately has a relatively poor prognosis. The mortality rate at one year[4] for osteoporotic fractured neck of femur patients is over 30%. If left untreated, the fracture is at risk of non-union and the femoral head may dissolve away due to avascular necrosis. New recommendations have been made for managing these patients with joint efforts from orthopaedic surgeons and ortho-geriatricians[5].

Case 3

A 45-year-old housewife slips in her garden, landing on an outstretched hyper-extended right hand. She immediately feels pain at her wrist and is reluctant to move it. There is some generalized swelling around the wrist, and maximal tenderness over the wrist joint. Dorso-palmar and lateral radiographs of the right wrist are taken.



ORTHOPAEDIC RADIOLOGY QUIZ

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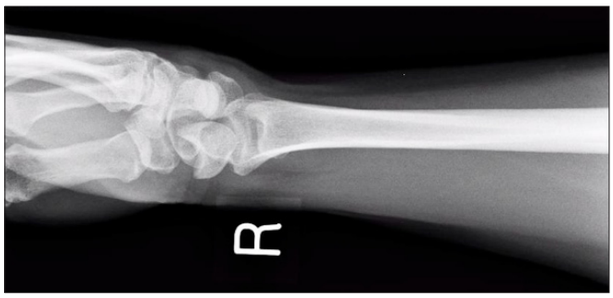


3rd Questions

Question 3.1
Name the carpal bones from the DP radiograph.

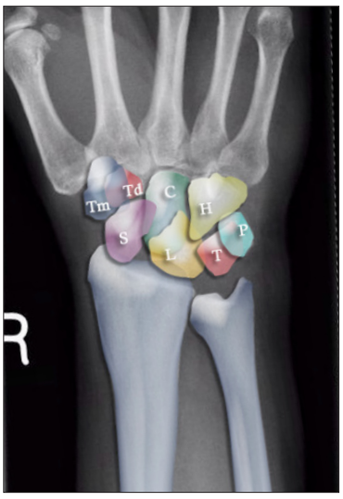
Question 3.2
What is the diagnosis?

Question 3.3
Are any further investigations required?

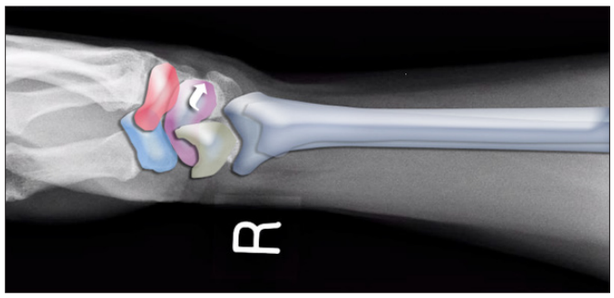
Question 3.4
What is the best treatment for this injury?

Question 3.5
What are the complications of this injury?

3rd Answers



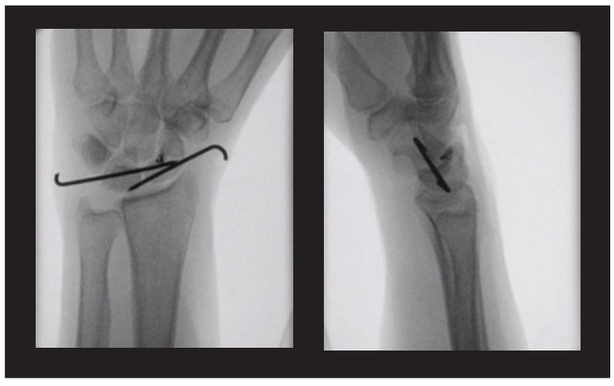
Answer 3.1
The carpal bones from left to right, and proximal row to distal row are as follows: (S) scaphoid, (L) lunate, (T) triquetrum, (P) pisiform, (Tm) trapezium, (Td) trapezoid, (C) capitate and (H) hamate.



Answer 3.2
The diagnosis is of an acute right wrist perilunate dislocation. The DP radiograph shows disruption of Gilula's lines[6] and widening of the scapho-lunate gap more than 2mm. There are four stages that have been described by Mayfield for this injury[7]. This is a stage III injury as the lunate has not dislocated volarly into the space of Poirier. The lunate appears more triangular on the DP radiograph, but appears in the correct place, albeit slightly rotated volarly on the lateral radiograph. The other carpal bones however are dislocated dorsally as shown by the white arrow on the lateral radiograph.

Answer 3.3
Usually plain radiographs alone are sufficient to make the diagnosis, but it is common for non-orthopaedic doctors to miss. These injuries can be associated with scaphoid fractures and high-energy polytrauma. A thorough examination is essential to pick up other injuries.

Answer 3.4



This kind of dislocation requires urgent reduction. For stage III and IV dislocations, this can be difficult to achieve closed so it is recommended that the patient be worked up for theatre. Open reduction with acute repair of the torn interosseous ligaments has been shown to give superior outcomes compared to closed reduction and immobilization alone[8,9]. This can be achieved by using Kirschner wires as joysticks to reduce the scaphoid and lunate. Suture anchors in the bone can be used to repair the torn ligaments. The Kirschner wires can further be used to protect the ligament repair temporarily (removed after 6 weeks).

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Answer 3.5

There are many complications from perilunate dislocations that patients should be warned about. These include avascular necrosis, carpal instability, osteoarthritis, median nerve damage and complex regional pain syndrome. Early reduction and adequate fixation has been shown to reduce the incidence of these problems. If there are median nerve symptoms during initial presentation, decompression of the carpal tunnel should also be performed.

References

1. American College of Surgeons. Advanced Trauma Life Support (ATLS) instructor manual, 8th ed. Chicago, IL: American College of Surgeons, 2008

2. Lasinger O, Bergman B, Korner L, Andersson GB. Tibial condylar fractures. A twenty-year follow-up. J Bone Joint Surg Am 1986; 68:13-9

3. National Clinical Guideline Centre, (2011) [The Management of Hip Fracture in Adults]. London: National Clinical Guideline Centre. Available from: **www.ncgc.ac.uk**

4. Keene GS, Parker MJ, Pryor GA Mortality and morbidity after hip fractures British Medical Journal, 13 November 1993, 307, pp1248-50

5. British Orthopaedic Association, The Care of Patients With Fragility Fracture, September 2007.

6. Gilula LA, Destouet JM, Weeks PM, et al. Roentgenographic diagnosis of the painful wrist. Clin Orthop Relat Res. 1984;187:52-64.

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Test Yourself.

7. Mayfield JK, Johnson RP, Kilcoyne RK. Carpal dislocations: pathomechanics and progressive perilunar instability. J Hand Surg [Am]. 1980;5:226-41.

8. Apergis E, Maris J, Theodoratos G, et al. Perilunate dislocations and fracture-dislocations. Closed and early open reduction compared in 28 cases. Acta Orthop Scand. 1997;275 (Suppl):55-9.

9. Adkison JW, Chapman MW. Treatment of acute lunate and perilunate dislocations. Clin Orthop Relat Res.1982;164:199-207.

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DEVELOPMENTAL DYSPLASIA OF THE HIP: A JUNIOR DOCTORS GUIDE

U Abah

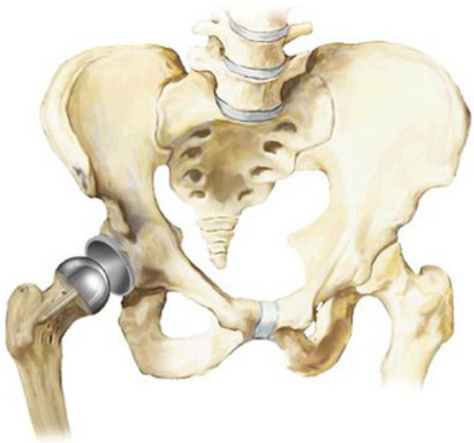
Developmental Dysplasia of the Hip: A Junior Doctors Guide Good Clinical Care.

Abstract

Developmental dysplasia of the hip (DDH) is a condition which covers a spectrum of abnormal hip development, ranging from acetabular dysplasia, through hip instability to fixed dislocation of the hip. It is important for medical professionals involved in the detection of DDH to have a good examination technique, as evidence has shown late presentation of DDH to be higher when screening is carried out by poorly trained staff and this can have serious implications for the future of the individual patient. This article discusses the natural history of DDH, and describes the recommended methods for the screening and management.

Introduction

Developmental dysplasia of the hip (DDH) is a condition which covers a spectrum of abnormal hip development ranging from acetabular dysplasia, through hip instability to fixed dislocation of the hip. The term DDH has replaced the earlier nomenclature of congenital dislocation of the hip (CDH), in order to more accurately describe the wide spectrum of anatomical and clinical abnormalities with which DDH presents. It is key for junior doctors involved in the detection of DDH to have an understanding of the natural history of the condition and a good examination technique. Evidence has shown late presentation of DDH to be higher when screening is carried out by poorly trained junior doctors and this can have serious implications for the future of the individual patient. Without treatment DDH can lead to poor mobility and function, it is a leading cause of premature arthritis requiring total hip replacement. The aim of this article is to give an overview of the aetiology, diagnosis and management of DDH for junior doctors.



Aetiology

The aetiology of DDH is unknown. DDH is found in 2 in 1000 live births and unstable hips in 5-20 in 1000 live births. It is important to recognise that all unstable hips do not constitute DDH, but rather joint laxity which may naturally resolve with development. The majority of cases of DDH are idiopathic; however neuromuscular disease and some syndromes can lead to teratological hips which often represent severe forms of the condition, commonly presenting with bilateral fixed dislocations, which are assessed and treated differently. This article will consider only the idiopathic form of DDH. DDH has been shown to be associated with family history in an immediate relative; it is more common in girls (80%) and in the left hip and has been found to be bilateral in 20% of cases.

Conditions which affect intrauterine space and positioning increase the risk of DDH. Breech presentation in the third trimester (even if the delivery is cephalic!), first born child, oligohydramnios and multiparous pregnancy are all associated with an increase in the risk of DDH. Post natal factors such as swaddling have also been associated with a higher risk. A number of congenital conditions have been linked to DDH. These most likely represent the reduction in intrauterine space. Conditions include; metatarsus adductus, torticollis, calcaneovalgus, positional talipes (equinovarus) and plagiocephaly.

Screening

The concept of early recognition resulting in improved outcomes is undisputed; however the method by which to ensure early detection of infants with DDH remains controversial. Some centres advocate universal ultrasound examination for all (as in Germany and Austria), some screen high risk patients, whilst others really upon detection by routine clinical examination. Evidence shows that clinical examination in those trained and familiar with DDH to be an adequate screening tool, however currently in the UK examinations are carried out by junior doctors lacking in experience and training. A UK national screening programme was introduced in 1986 by the Standing Medical Advisory Committee & Standing Nursing and Midwifery Committee. This encompassed the newborn health check, 6 week and 5-9 month review. However published literature suggests there has been no reduction in the rate of late diagnosis of DDH with the blame placed upon junior doctors being a key factor in the screening process.

DEVELOPMENTAL DYSPLASIA OF THE HIP: A JUNIOR DOCTORS GUIDE

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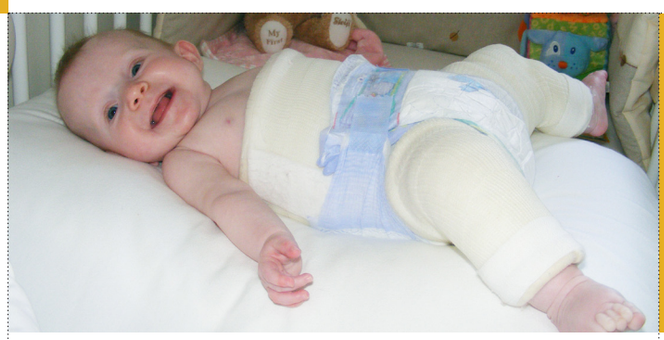


Developmental Dysplasia of the Hip: A Junior Doctors Guide Good Clinical Care.

Diagnosis

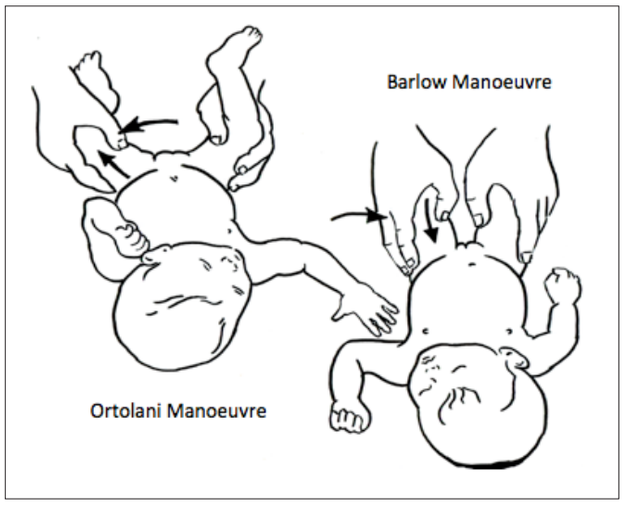
The mainstay of diagnosis is based upon clinical examination and confirmation is made with appropriate imaging. The Ortolani manoeuvre and Barlow test are used to examine every newborn baby and at 6 weeks as a legal requirement in the UK.

Ortolani Manoeuvre - for a dislocated hip

Place the infant supine with hips and knees flexed at 90º, starting with the knees together slowly abduct the hips, with ones fingers placed over the greater trochanter and thumb on the inner surface of the thigh in order to feel if the femoral head 'clicks' back into the acetabulum. There should be a visible and palpable movement, as the hip relocates. If the examination is not clear, exam one hip at a time using the other hand to stabilise the pelvis.

Barlows test - for a dislocatable hip

Place the infant supine with hips and knees at 90º flexion, then place a downward and adduction pressure on the hip joint to establish if it is unstable/dislocatable.



Both of these commonly used examination methods have the advantage of a very high specificity, but unfortunately they do have a low sensitivity. They are useful in the diagnosis of neonates but become difficult after the age of approximately 8 weeks due to development of soft tissue contractures, increased muscle tone and the size of the child.

Once soft tissue contractures have developed, the most sensitive sign of DDH is reduced hip abduction. During examination there are signs which give clues to the presence of a dislocated hip. Lower limb length discrepancy can be demonstrated by Galeazzi sign. This describes when the child is placed supine with both hips and knees flexed and feet placed on examination couch and the examiner views the height of the knees and a difference is noted. Asymmetrical gluteal/thigh/posterior knee skin creases are seen in DDH. However asymmetrical skin folds are also demonstrated in 25% of normal babies.

Investigations

The mainstay of investigation for DDH consists of ultrasonography and radiographs based upon the age of the child. Ultrasound scanning is used for infants prior to the ossification of the femoral head which occurs at approximately between 4 -6 months. There are two common methods used: static and dynamic scanning. The most common static method was described by Graf, it uses a static ultrasound image to create angles, which gives a morphological group (Graf a-d), which can be used to guide prognosis and management. Dynamic ultrasonography described by Harke involve stressing the hip as in Barlow's test under ultrasound visualisation to detect instability of the hip. Following the ossification of the femoral head (4-6 months) radiographs can be used to estimate dysplasia. A line drawn through the tri-radiate cartilages (Hilgenreiners line), intersecting with a line drawn at the bony acetabular cup, creates an angle: the acetabular index which is used to determine prognosis and management.

DEVELOPMENTAL DYSPLASIA OF THE HIP: A JUNIOR DOCTORS GUIDE

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Management

DDH is managed by a consultant orthopaedic surgeon with a specialist interest in paediatric orthopaedics. The mainstay of management involves conservative management in a flexion-abduction harness. The harness holds the hip in flexion and abduction which causes the femoral head to sit firmly in the acetabulum and encourage normal acetabular development. Success rates of 80-95% have been shown with flexion-abduction splints. There is a significant reduction in success rates of conservative treatment following the age of 7 weeks. In the case of late presentation or failure of management with a flexion-abduction harness, closed or open reduction may be required. Closed reduction involves manipulation of the hip under anaesthesia, normally accompanied by an adductor or psoas tenotomy to release soft tissue contractures; this is followed immobilisation in a plaster cast or abduction brace. Open reduction procedures can include pelvic or femoral ostetomies to improve the anatomical and mechanical morphology of the joint.

Conclusion

DDH of the hip is commonly screened for by junior doctors, during newborn health checks. Early detection of the condition leads to improved outcomes. A good understanding and examination technique of those involved in detection (paediatric/general practitioner trainees) of the condition is crucial to reduce late diagnosis rates.

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References

1. Eastwood DM, Neonatal Hip Screening. Lancet 2003;361:595-7
2. Furnes O, Lie SA, et al. Hip disease and the prognosis of total hip replacements. A review of 53,698 primary total hip replacements reported to the Norwegian Arthroplasty Register 1987-99. J Bone Joint Surg (Br) 2001;83:579-86.
3. Barlow TG. Early diagnosis and treatment of congenital dislocation of the hip. J Bone Joint Surg(B) 1962;44:292-301.
4. Sewell MD, Rosendahl, et al. Developemental Dysplasia of the Hip. BMJ 2009;339:b4454
5. Stein-Zamir C, Volovik I, et al. Developmental dysplasia of the hip: risk markers, clinical screening and outcome. Pediatr Int. 2008 Jun;50(3):341-5.
6. Higginbottom MC, Jones KL, et al. Intrauterine constraint and craniosynostosis. Neurosurgery. 1980 Jan;6(1):39-44.
7. Bialik V, Fishman J, et al. Clinical assessment of hip instability in the newborn by an orthopaedic surgeon and a paediatrician. J Piatr Orthop 1986;6:703-06.
8. Standing Medical Advisory Committee and Standing Nursing and midwifery Advisory Committee. Screening for the detection of congenital dislocation of the hip. London: Department of Health and Social Security, 1986.
9. Godward s, Dezateux C. Surgery for congenital dislocation of the hip in the UK as a measure of outcome of screening. Lancet 1998;351:1149-52.
10. Graf R. The diagnosis of congenital hip-joint dislocation by the ultrasonic Combound treatment. Arch Orthop trauma Surg 1980;97:117-133.
11. Van der Sluijs JA, De Gier L, et al. Prolonged treatment with the Pavlik harness in infants with developemental dysplasia of the hip. J Bone Joint Surg (Br) 2009;91:1090-3
12. Atalar H, Sayli U, et al. Indicators of successful use of the Pavalik Harness in infants with developmental dysplasia of the hip. Int Orthop 2007; 31:145-150.

SEPTIC ARTHRITIS OF THE PAEDIATRIC HIP - A CASE STUDY

D Ferguson & K Haendlmayer

Septic Arthritis of the Paediatric Hip – A Case Study. Patient Management.

Abstract

Paediatric hip pain is very common and has many different causes. One of the most concerning and significant causes is septic arthritis. Diagnosis of this disorder can be difficult. Our aim in this case based review article is to describe the management by example. We present a case of a 12 year-old-girl with a 2-day history of pain in her right hip. Key elements in her history and examination reveal the cardinal features of septic arthritis and are then confirmed on further investigation. We recommend using a scoring system to decide which children are most at risk of septic arthritis, in order to guide initial management. We also describe the surgical treatment for septic arthritis of the hip, once the diagnosis has been made.

A 12 year-old-girl presents with a 2-day history of a painful right hip. Her parents carry her into the emergency department because it is too painful for her to walk.

How would you approach this patient if you were asked to review her?

Start by taking a detailed history from the patient and her family. It is important to ask about specific points in the history (table 1). Severe pain is a major feature and should be explored thoroughly. Pay specific attention to pain at night or at rest. True hip pain tends to radiate to the groin and is worse on weight-bearing. Knee pain may also be a feature as it shares a nerve supply with the hip. Abdominal pain can radiate to the hip, so the history should also enquire about abdominal symptoms (bowel and bladder function). This can be the case in appendicitis, where a right hip pain is experienced due to irritation of the psoas muscle[1].

The most striking feature of this case is that her hip pain is so severe that her parents had to carry her into hospital. This is a worrying feature that should be taken extremely seriously.

A history of recent illness may be significant, as the most common cause for paediatric hip pain is transient synovitis. This can accompany a recent viral illness and cause joints to spontaneously fill with fluid. This is a self limiting condition that requires analgesia alone.

A septic hip often has acute severe pain that does not completely settle, wakes children from their sleep and has associated nausea, vomiting and fever. There is not usually a history of trauma. If the child has not completed their immunization schedule, then they may be at increased risk of certain infections. A septic hip can occur at any age so a high index of suspicion should always be present. Age is still an important point as the differential includes Legg-Calvé-Perthes' disease (usually boys aged 4-9)[2] and slipped upper femoral epiphysis (14 to 16 years in boys, 11 to 13 years in girls)[3].

Pain details including site, onset, character, radiating, associated symptoms, timing, exacerbating / alleviating factors, severity
Trauma
Bowel and bladder function
Recent illness
Immunization history
Age of patient
Family history
Current medication (including any antibiotics)
Drug allergies

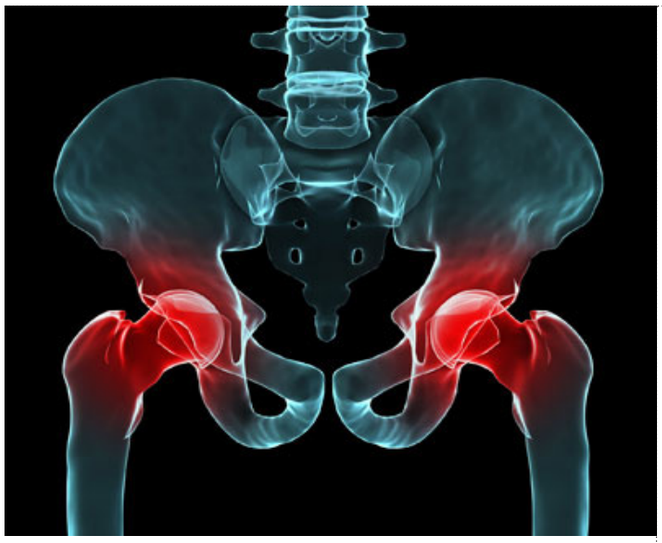
Table 1: History Key Points

When examining the child, attention should be made to their gait as they try to walk. Septic hips are usually held flexed and the child will guard against any movement. Internal and external rotation may also be limited. The hip joint may feel hot and may have overlying erythema.

Make sure that in males, there are no undescended testicles. Also check for abdominal discomfort on palpation.

SEPTIC ARTHRITIS OF THE PAEDIATRIC HIP - A CASE STUDY

D Ferguson & K Haendlmayer



The history reveals that her pain started suddenly and has gradually got worse over the past 2 days. She now has pain at rest that radiates to the groin, and is holding the hip to prevent you moving it. Her parents tell you that she has had a fever overnight and has vomited twice. On examination, the girl's hip is severely painful to move in all directions. She has a temperature of 39 degrees Celsius and has a pulse rate of 90. Her blood pressure is 120/80 mmHg. There is no abdominal tenderness and she is up to date with her immunisations.

What investigations would you like to perform?

It is useful to obtain a full blood count and an inflammatory marker such as erythrocyte sedimentation rate (ESR). Kocher et al, 1999 used these tests with the history to calculate the chance of a child's hip being septic. Blood cultures should also be taken to identify organisms and their sensitivities to antibiotics. This can take 48 hours, so the sooner the cultures are sent, the sooner the results will be back.

If the child has a history of fever, is non weight bearing, has an ESR greater than 40mm per hour and has a serum white cell count greater than 12.0 x 10^9 cells per litre then the predicted probability of septic arthritis is 99.6%. With just three of these factors, it drops to 93.1%, two factors 40.0%, one factor 3% and no factors <0.2% chance of septic arthritis[4]. Children with all four factors should have their hip aspirated in theatre, as there is a high chance that pus will be aspirated and therefore the hip will need to be washed out. Ultrasound scans are safe and useful investigations in children with hip pain but lacking other risk factors as they can demonstrate effusions (abnormal if >5mm[5]). If an effusion is present, the hip can also be aspirated at the same time. If there is no hip effusion seen on ultrasound examination, then septic arthritis is unlikely to be the cause of hip pain.

The blood tests reveal that she has an ESR of 40mm per hour, and a white cell count of 14x10^9 cells. She also has an ultrasound scan whilst awaiting the blood results, which is shown below (figure 1). What should you do next?

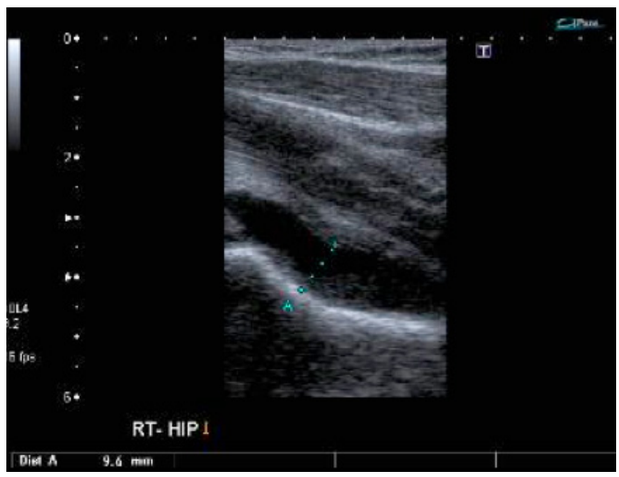


Figure 1: An ultrasound scan revealed the presence of a 9.6mm effusion.

Given all the clinical findings, there is a strong possibility that this girl has septic arthritis of her hip. She is not in extremis so antibiotics should ideally be started once an aspirate has been performed. The hip could not be aspirated in the radiology department because there were no radiologists available to perform this intervention. You decide to take her to theatre and attempt aspiration of the hip under fluoroscopic control.

How do you aspirate a childs hip under fluoroscopic control?

One of the easiest and safest ways to aspirate the hip is through a lateral approach, sliding a long spinal needle over the anterior aspect of the greater trochanter and through the hip capsule, as shown in figure 2. The child must be lying supine on a radioluscent table, and consented for aspiration plus proceed to open washout if required.

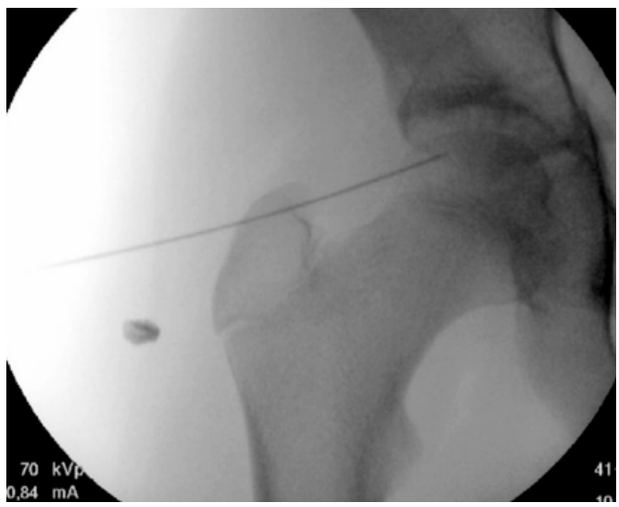


Figure 2: Aspiration technique for hip effusions in theatre.

SEPTIC ARTHRITIS OF THE PAEDIATRIC HIP - A CASE STUDY

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In this case, after you insert the needle, frank pus is withdrawn. This is sent off for immediate microbiological studies (Gram staining) and you proceed to open the hip joint through an anterior Smith-Peterson approach to wash the joint out. The child can then be started on intravenous Benzylpenicillin and Flucloxacillin as the most common organism to cause joint infections is Staphylococcus aureus.

Professional Dilemma MCQs

1. What is the most common cause for paediatric hip pain?

2. What 4 factors best predict a child has septic arthritis?

3. When should antibiotics be started when septic arthritis is suspected?

4. Which is the most common organism responsible for septic arthritis?

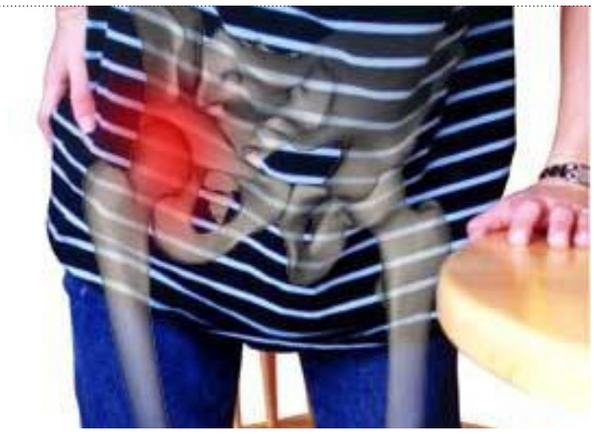
MCQs Answers

1. Transient synovitis

2. Non weight bearing, fever, WCC >12x10^9 cells/L, ESR>40 mm/hour

3. Ideally after an aspirate and blood cultures have been taken to allow targeted antimicrobial therapy, although empirical antibiotics are indicated if there are generalised signs of sepsis.

4. Staphylococcus aureus



Key Points

- Septic arthritis is an orthopaedic emergency
- Keep patients fasted until this diagnosis has been ruled out
- Use a scoring system to predict the likelihood of septic arthritis

References

1. Silen M. L., Tracy T. F. The right lower quadrant "revisited". Paediatr Clin North Am 40:1201-1211, 1993
2. Hall A. J. Perthes Disease: Progress in aetiological research. In: Catterall A (ed) Recent Advances in Orthopaedics 5. Churchill Livingstone, Edinburgh, pp 187-199
3. Hagglund G, Hanson L. I., Ordeberg G. Epidemiology of slipped capital femoral epiphysis in Southern Sweden. Clinical Orthopaedics and Related Research 1984: 191; 82-94
4. Kocher M. S., Zurakowski D., Kasser J. R. Differentiating between septic arthritis and transient synovitis of the hip in Children: An evidence-based clinical prediction algorithm. JBJS Am Dec 1999; 81-A, 12: 1662-1670
5. Schmidt WA, Schmidt H, Schicke B, Gromnica-Ihle E. Standard reference values for musculoskeletal ultrasonography. Ann Rheum Dis2004;63:988–94.

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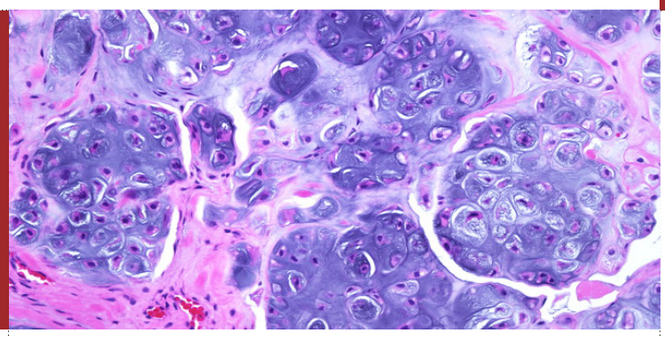
SYNOVIAL CHONDROMATOSIS OF THE DISTAL RADIOULNAR JOINT: A CASE REPORT



Synovial Chondromatosis of the Distal Radioulnar Joint: A Case Report.
Patient Management.

Abstract

Synovial chondromatosis is a rare condition, which results in the formation of loose bodies within joints, with associated pain and limitation in the range of movement and function. The disorder has been most commonly observed in the knee, the hip and the elbow. A case of the condition affecting the wrist is presented here, together with a brief discussion of the management of the condition.

A 39-year-old female patient, who is an accountant by profession, presented with a history of instability and discomfort in the right wrist, localised to the ulnar aspect of the wrist. This was also causing night-time waking from sleep. These symptoms had been ongoing for four years. The patient denied any history of trauma.

On examination, her pain was localised to the ulnar aspect, and was exacerbated by pronation as well as supination movements. The piano key test was found to be positive.

There was initially a suspicion of triangular fibrocartilage complex tear. X-rays showed some increased calcification in relation to distal radio-ulnar joint and possibly in soft tissues.

An MR arthrogram showed multiple small round low intensity loose bodies in the distal radioulnar joint of the right wrist, and a small effusion in the distal radioulnar joint. No erosive changes noted in the distal radioulnar joint or within the ulnar styloid process. There was no evidence of a tear in the triangular fibrocartilage complex.

Synovial chondromatosis is known to be a recurrent condition. Spontaneous regression is rare. Also, a previous literature review examined 24 published cases of synovial chondromatosis affecting the wrist, and found only three cases of recurrence.[6] The vast majority of these cases had been managed with excision of the loose bodies, and in many cases with synovectomy in addition.[6] As the patient reported troublesome, persistent symptoms, it was therefore decided that surgical intervention offered the best chance of a satisfactory outcome.

The patient underwent wrist arthroscopy, open synovectomy and removal of loose bodies. Plain film radiographs confirmed complete removal of all loose bodies.

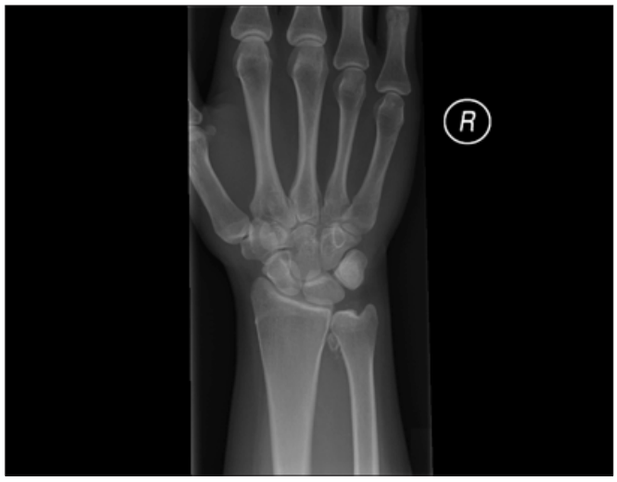


Figure 1. Plain film radiograph of wrist before surgery, demonstrating osteochondral body in distal radioulnar joint.

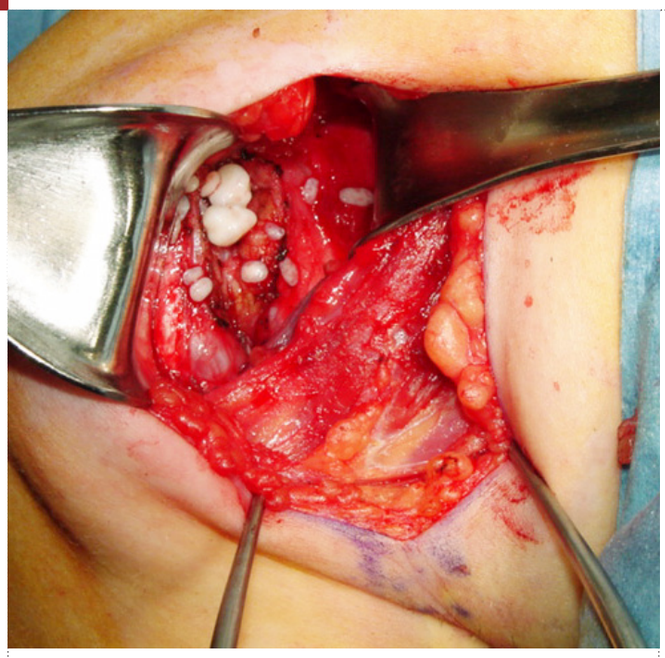
Histopathological analysis found sections showing numerous cartilaginous and osteo-cartilaginous nodules of varying size growing below a synovial lining and lacking any cytological atypia or any suspicious features. This was consistent with synovial chondromatosis.

At review 3 months after surgical excision of the loose bodies, she reported that she was not experiencing any pain, and that she was back to full function.

She was advised that there was a risk of recurrence, as synovial chondromatosis is a recurrent condition.

At review 12 months after surgery, she remained free of symptoms. It was decided that she would be monitored for recurrence of symptoms, and provisionally decided that she would be reviewed after 5 years.

SYNOVIAL CHONDROMATOSIS OF THE DISTAL RADIOULNAR JOINT: A CASE REPORT



However, six months later (18 months after surgery), the patient presented with a recurrence of symptoms, with a limitation of range of wrist movement, specifically supination. Plain film radiographs demonstrated a recurrence of loose bodies in the distal radioulnar joint. At her next review, another six months later, she reported that her symptoms were progressing further. The patient also reported locking of the wrist on occasion.

As the patient's symptoms were again causing significant limitation of function, it was decided that further surgery was required.

It was decided to proceed with minimal surgical intervention, with removal of the loose bodies and synovectomy, so as to facilitate rehabilitation and restoration of function. More aggressive options, including hemiresection of the distal ulna and further synovectomy, are reserved for a later stage, as these can themselves carry the risk of causing problems with instability and recurrent aches.

The procedure was successful and there were no complications.

At review two months following surgery, she was found to be making good progress, with a reasonable recovery in range of motion and function.

At her next review 5 months post-surgery, she remained symptom-free.

Considering that the patient previously had recurrence of symptoms after an interval of approximately eighteen months following surgery, the overall outcome is perhaps best judged following long-term follow-up and review of the patient over a period of several years.

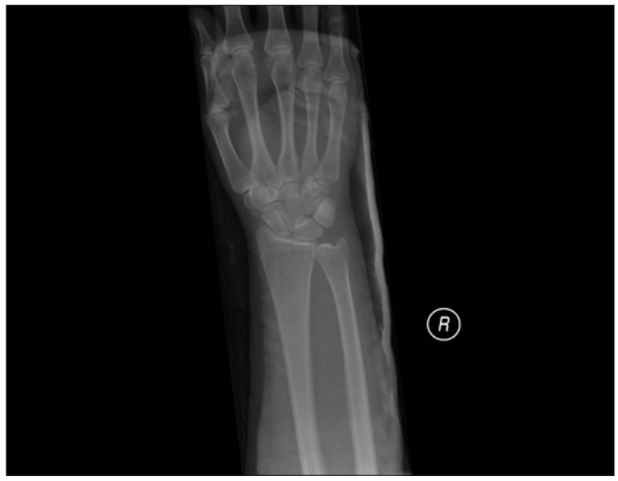


Figure 2. Radiograph demonstrating successful removal of loose body in DRUJ.

Discussion

Primary synovial chondromatosis is a rare condition. It is characterized by the proliferation of islands of irregularly hypercellular cartilage in the synovium of a major joint, or, occasionally, in a tendon sheath.[1]

Microscopic examination reveals discrete nodules of lobulated cartilaginous tissue in the synovium, characterized by cellular crowding with cytologic atypia; many binucleate cells as well as myxoid areas may be present.[1]

As described by Milgram, the three phases of the disease process are: (i) active intrasynovial proliferation, (ii) active intrasynovial disease and formation of free loose bodies, and (iii) no active disease, only free loose bodies within the joint.[2,3] Identification of cartilaginous metaplasia within the synovium confirms the diagnosis of synovial chondromatosis.[2]

The lesion usually presents during the third to fifth decades of life.[4] Men appear to be affected about twice as frequently as women.[1,4]

The usual symptoms are pain and stiffness, gradual in onset, or an enlarging mass around the affected joint. Limitation of motion is a characteristic finding on clinical examination.[1] The most commonly affected joints are the knee, the hip and the elbow.[1,4] The majority of the remaining cases occur in the tendon sheaths of the hands or feet.[1]

SYNOVIAL CHONDROMATOSIS OF THE DISTAL RADIOULNAR JOINT: A CASE REPORT

Radiographic and MRI findings can be vague. In the early stages, radiographs may show little more than soft tissue swelling. Later, with increased ossification, small radio-opaque masses will be demonstrated in the joint.[2] The situation is similar for magnetic resonance imaging. In the earlier stages the findings may be similar to joint effusions, as the signal intensity of the process resembles that of fluid, potentially resulting in misdiagnosis. In later stages, however, MRI is very accurate; calcification of peripheral portions of the lesions can be identified.[2] Other radiologic signs of this disorder include effusions, degenerative arthrosis, subchondral sclerosis and osteophytes.[4] When radiographs are negative, MRI usually demonstrates multiple filling defects.[1,4]

At surgery, there are usually multiple cartilaginous loose bodies, both free in the joint and attached to the synovium. The larger cartilage loose bodies often have a multinodular surface.[1]

The treatment is based on the stage of the disease: synovectomy for earlier phases, and loose body excision with or without synovectomy for the later phases.[2]

As described by Slesarenko et al., treatment options include arthroscopic synovectomy, open synovectomy and loose body removal. Spontaneous resolution is rare and surgical synovectomy with excision of the loose bodies is the most effective treatment, however, owing to difficulty of achieving complete excision, the condition frequently recurs.[1,4]

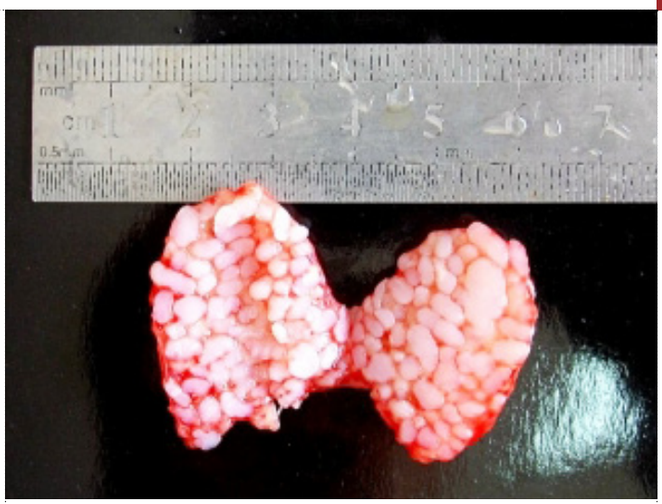
Test yourself questions

1. Which of the following is one of the possible differential diagnoses of synovial chondromatosis?

a. Pigmented villonodular synovitis
b. Tumoral calcinosis
c. Osteoarthritis
d. Rheumatoid arthritis
e. All of the above

2. Ulnar deviation of the hand is a function of:

a. flexor carpi ulnaris and extensor carpi ulnaris
b. extensor carpi radialis longus
c. extensor carpi radialis brevis
d. flexor carpi radialis
e. none of the above



Answers

1. Answer – e. All of the choices.

- **Pigmented villonodular synovitis (PVNS):**

Is a locally aggressive synovial tumor. It affects both large joints and tendon sheaths. It is frequently found as a solitary nodule and rarely as a diffuse multinodular lesion. The joints most commonly involved are the knees and fingers, but it sometimes occurs in the hip, ankle, foot, or wrist. It is usually painless or only mildly painful. The pain tends to be more severe when the lesion is diffuse throughout a major joint. In general, the condition is confined to a single joint or tendon sheath.(1)

The radiologic signs of PVNS depend on the site of occurrence. In the finger or toe, only soft tissue swelling may be evident, although cortical bone erosion may occur. In the knee, the only consistent radiographic change is soft tissue swelling in and around the joint. In the hip, joint narrowing and lytic defects in the bone may be present on both sides of the joint. Local juxta-articular bone erosion may also be quiteprominent in joints such as the wrist, knee, and ankle.(1)

MRI and computed tomography are useful to assess the extent of the lesion. On T1-weighted MRI, iron deposits may show up as punctate signal voids within the lesion. (1)

- **Tumoral calcinosis:**

Tumoral calcinosis is a rare inherited condition that is seen most frequently – but not exclusively – in patients of African descent who are otherwise in good health. It usually presents in the second decade of life and is characterized by deposition of painless calcific masses around the hips, elbows, shoulders, and gluteal areas (i.e., areas subject to movement and/or pressure). Familial incidence has been reported. In rare instances, intra-articular or intraosseous deposits are also observed. (1)

SYNOVIAL CHONDROMATOSIS OF THE DISTAL RADIOULNAR JOINT: A CASE REPORT

- **Osteoarthritis and rheumatoid arthritis:**

Separated fragments of bone and cartilage from a damaged joint surface may become incorporated into the synovial membrane and digested, or may remain free as loose bodies in the joint cavity. Under certain circumstances, cartilage cells proliferate on the surface of these loose bodies and as a result, they enlarge, their centres becoming necrotic and calcified. In histologic sections, periodic extension of this central calcification may be observed, in the form of concentric rings increasing in number as the loose body grows larger. (1)

Sometimes the bodies may reattach to the synovial membrane, in which case they are invaded by blood vessels. The loose bodies become bony as a result of endochondral ossification. (1)

Occasionally, in cases of OA, the loose bodies are so numerous that they must be distinguished from those that occur in primary synovial chondromatosis. There is also some degree of loose body formation in many types of arthritis, including inflammatory RA, in which fibrinous loose bodies may be numerous. (1)

2. Answer: a. flexor carpi ulnaris and extensor carpi ulnaris

Muscle	Proximal attachment	Distal attachment	Innervation	Main action
Flexor carpi ulnaris	Humeral head: medial epicondyle of humerus; Ulnar head: olecranon and posterior border of ulna	Pisiform bone, hook of hamate bone, and 5th metacarpal bone	Ulnar nerve (C7 and C8)	Flexes and adducts hand (at wrist)
Flexor carpi radialis	Medial epicondyl of humerus	Base of second metacarpal bone	Median nerve (C6 and C7)	Flexes and abducts hand (at wrist)
Extensor carpi radialis brevis	Lateral epicondyle of humerus	Base of 3rd metacarpal	Deep branch of radial nerve (C7 and C8)	Extend and abduct hand at wrist joint
Extensor carpi radialis longus	Lateral supracondylar ridge of humerus	Base of 2nd metacarpal	Radial nerve (C6 and C7)	Extend and abduct hand at wrist joint
Extensor carpi ulnaris	Lateral epicondyle of humerus and posterior border of ulna	Base of 5th metacarpal	Posterior interosseus nerve (C7 and C8), the continuation of deep branch of radial nerve.	Extends and adducts hand at wrist joint
Flexor digitorum superficialis	Humeroulnar head: medial epicondyle of humerus, ulnar collateral ligament, and coronoid process of ulna Radial head: superior half of anterior border of radius	Bodies of middle phalanges of medial four digits	Median nerve (C7, C8, and T1)	Flexes middle phanalges at proximal interphalangeal joints of medial four digits; acting more strongly, it also flexes proximal phalanges at metacarpophalangeal joints and hand.
Flexor digitorum profundus	Proximal three-fourths of medial and anterior surfaces of ulna and interosseous membrane	Bases of distal phalanges of medial four digits	Medial part: ulnar nerve (C8 and T1) Lateral part: median nerve (C8 and T1)	Flexes distal phalanges at distal interphalangeal joints of medial four digits; assists with flexion of hand

Table: Muscles acting on the wrist (5).

Synovial Chondromatosis of the Distal Radioulnar Joint: A Case Report.
Patient Management.

References

1. Bullough PG. Orthopaedic Pathology. 5th ed. Mosby; 2009

2. Rogachefsky RA, Zlatkin MB and Greene TL. Synovial Chondromatosis of the Distal Radioulnar Joint: A Case Report. J Hand Surg Am. 1997 Nov;22(6):1093-7

3. Milgram JW. Synovial Osteochondromatosis: a histopathological study of thirty cases. J Bone Joint Surg Am. 1977 Sep;59(6):792-801.

4. Slesarenko YA, Hurst LC and Dagum AB. Synovial Chondromatosis of the Distal Radioulnar Joint. Hand Surg. 2004 Dec;9(2):241-3.

5. Moore KL, Dalley AF. Clinically Oriented Anatomy. 4th Ed. Lippincott Williams & Wilkins; 1999.

6. Loonen MPJ, Schuurman AH. Recurrent synovial chondromatosis of the wrist: Case report and literature review. Acta Orthop. Belg. 2005 Apr;71(2):230-5.

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Oral and Maxillofacial Surgery.
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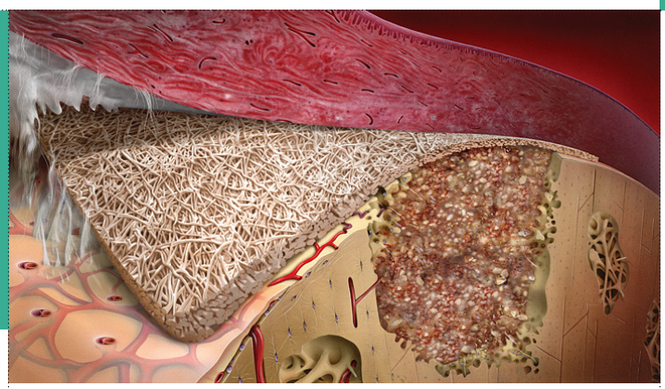

Abstract

This two-part article provides an overview of the surgical skills necessary and training requirements for a career in Oral and Maxillofacial Surgery. Structured pathways for trainees originating from both medical and dental backgrounds are outlined (Part I). In the second part, the clinical features and management of common Oral and Maxillofacial injuries are described, with particular relevance to what is expected of a foundation trainee (Part II).

Part I: A career in Oral and Maxillofacial Surgery: Do you have what it takes?

Oral and Maxillofacial Surgery (OMFS) is the surgical specialty concerned with the diagnosis and management of diseases of the mouth (oral), jaws (maxillo) and face[1]. In the United Kingdom the foundations of OMFS are in dentistry and the subsequent need during both World Wars for specialist treatment of devastating mouth, face and jaw injuries. In the post war period, hard and soft tissue reconstructive techniques practiced by Oral and Maxillofacial (OMF) surgeons were put to use in patients with traumatic injuries following motor vehicle accidents, as it became clear there was a need for specialist surgical management.

What does an OMF surgeon do?

In keeping with the anatomical and surgical knowledge of the head and neck region, the scope of OMFS has grown rapidly and now 'includes the diagnosis and management of facial injuries, head and neck cancers, salivary gland diseases, facial disproportion, facial pain, temporomandibular joint (TMJ) disorders, impacted teeth, cysts and tumours of the jaws as well as numerous problems affecting the oral mucosa such as mouth ulcers and infections' [2].

- **Management of facial trauma:** This ranges from soft tissue injuries of the face to complex fractures of the facial bones. The management of these patients challenges the surgeon in both soft and hard tissue reconstruction in the most unforgiving part of the human body - an individual's face.

- **Head and neck cancer:** The OMF surgeon is a key-figure in the head and neck cancer multidisciplinary team, and is intimately involved in the diagnosis, treatment planning, surgical resection and reconstruction of head and neck cancer patients. Oral cancer is an extremely debilitating disease and the difficulties of reconstruction are coupled with providing a good post-operative quality of life. Many patients will now have a primary resection and neck dissection, combined with immediate micro-vascular reconstruction of the defect with free tissue transfer, using a variety of soft and hard tissue flaps.

- **Dento-alveolar surgery:** Involves the extraction of wisdom teeth, removal of cysts from the jaws, placement of dental implants and biopsies of intra-oral lesions.

- **Salivary gland surgery:** Incorporates both traditional and minimally invasive techniques in the treatment of obstructive and neoplastic salivary gland disease.

- **Facial deformity:** Essentially this is the management of congenital and acquired facial skeletal disproportion using modern surgical orthognathic techniques.

- **Cleft lip and palate surgery:** An OMF surgeon with subspecialist training can manage the patient from birth (primary cleft repair) to adulthood (rhinoplasty), as part of a cleft multidisciplinary team.

- **TMJ management:** This includes both conservative and surgical treatment, ranging from minimal access procedures to full joint replacement.

- **Facial aesthetics:** This is becoming an increasing area of expertise for the OMF surgeon, who plays an important role in surgically excising and reconstructing patients with facial cutaneous malignancies. Our knowledge of the head and neck allows resection and local flap reconstruction under local anaesthetic with excellent cosmetic results. Further to this, cosmetic based procedures such as face-lifts and blepharoplasties are also becoming increasingly common, with OMFS trainees undertaking Interface Fellowships in Aesthetic Facial Surgery.

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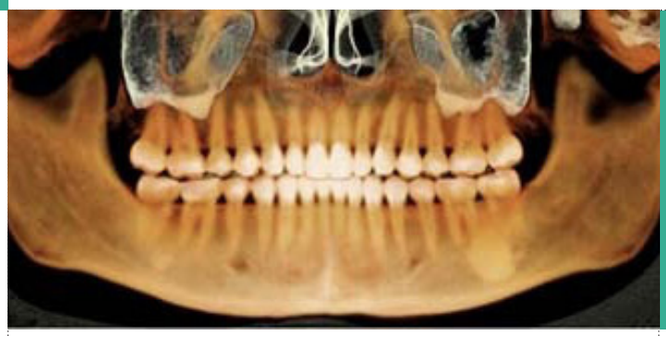

Oral and Maxillofacial Surgery. Teaching & Training.

Is it true that you need a medical and dental degree to be an OMF surgeon?

Yes, Oral and Maxillofacial surgery is the youngest of the nine surgical specialities of the Royal College of Surgeons and unique in the fact that it requires both dental and medical degrees to pursue higher training in the speciality.

In its infancy, many OMF surgeons were dentally qualified, however, as a hospital based surgical specialty it became mandatory for higher trainees to possess both medical and dental qualifications and this has been the case since the 1980's [3]. Despite the need for two primary degrees, it has been shown that the training pathway to becoming a Consultant Oral and Maxillofacial Surgeon is not significantly longer than other surgical specialties [4]. This is made possible by the availability of shortened medical and dental degrees for budding OMF surgeons, and also the drive and determination of OMF trainees.

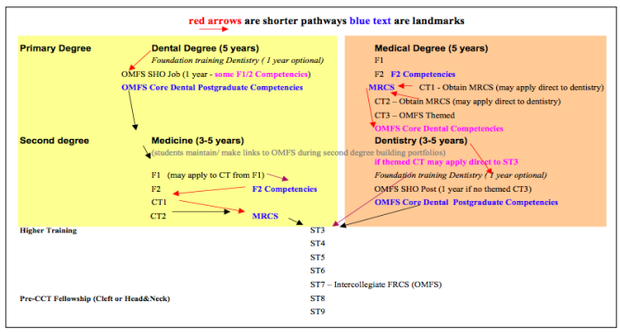

Fig 1: Outline of OMFS training pathways. (Reproduced with kind permission of Mr. Patrick Magennis)

Dentistry first	Medicine first
5 years BDS	5 years MBBS
1 year OMFS SHO	2 years FY
3 years MBBS	1 year CT Surgery/MRCS
1 year FY	3 years BDS
1 year CT Surgery/MRCS	1 year OMFS SHO
5 years ST OMFS	5 years ST OMFS
Total = 16 years	**Total = 17 years**
11 years after primary degree	**12 years after primary degree**

Fig 2: The shortest pathway to becoming an OMF surgeon. (Reproduced with kind permission of Mr. Patrick Magennis)

I would like to get more exposure to OMFS. What can I do during my Foundation training?

There are a number of avenues to explore:

- *Introduce yourself to your local OMFS unit:* We are a friendly specialty and if you show enthusiasm this will not go unnoticed.

- *Undertake an OMFS audit:* This could then be presented at a regional meeting or even better at an international conference. This will stand out on your curriculum vitae and surgical portfolio.

- *Contact the Junior Trainees' Group of the British Association of Oral and Maxillofacial Surgeons (JTG of BAOMS):* The JTG of BAOMS represents all OMFS trainees' up to the level of ST3 in the UK. The group has hundreds of members and communicates via an online forum, the trainees' section of the BAOMS website, the JTG Gazette which is a biannual newsletter and most importantly holds the annual JTG of BAOMS conference. The conference hosts international speakers and is a relaxed environment to meet other OMFS trainees.

- *The National Training Day (NTD) in OMFS:* This is an annual event that has a number of streams running throughout the day to offer up to date information on applying for your second degree, how to undertake research in OMFS and applying for core surgical training.

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· *Register your interest in OMFS:* In doing this you are automatically added to a mailing list from which you receive all the current and relevant information to your stage of training. This is an invaluable source of information to all trainees. The link is: www.baoms.org.uk

· *Consider a Core Surgery OMFS themed post:* This will enable you to experience OMFS first hand.

· *Develop your surgical portfolio and logbook:* This is essential to show your commitment to the specialty. The eLogbook is the only logbook approved for OMFS trainees' and it is highly recommended that you use it. www.elogbook.org.

I have done the above and OMFS is for me, so what next?

You should undertake core surgical training, complete your MRCS and apply to dental school. The JTG of BAOMS has up-to date information regarding all shortened dental courses in the UK and the authors would strongly advise that you contact the JTG of BAOMS committee via the BAOMS website.

On completion of your dental training, assuming that you fulfil the person specification as set out on the MMC website 5, including dental and medical degrees and MRCS, you will now be eligible to apply for an ST3 post in OMFS. National selection days are co-ordinated by the Severn Deanery 6, in the spring and autumn of each year.

A career in Oral and Maxillofacial Surgery: Do you have what it takes?

Oral and Maxillofacial Surgery offers enormous surgical diversity and challenges that will make no day in your career the same. If this inspires you then you may have what it takes to be an OMF surgeon.

Further information:

BAOMS website **(www.baoms.org.uk)** or alternatively contact the author at scott.omfs@me.com

Part II: The management of Oral and Maxillofacial trauma as a Foundation doctor

The acute management of OMF trauma forms part of the Advanced Trauma Life Support (ATLS) course and its detailed description is beyond the scope of this article. However, the following three areas are those most likely to be encountered by Foundation trainees, especially if they rotate through the Emergency Department.

1. Facial lacerations

2. Orbito-zygomatic complex fractures

3. Mandibular fractures

1. Facial Lacerations

When first seen these can be extremely daunting and you should not attempt to close these wounds unless you have previous experience or supervision. Careful consideration to the anatomy is essential when suturing facial lacerations as what appears to be a simple injury may involve numerous structures.

· *Neck lacerations deep to the platysma muscle*

Lacerations that should be referred to the OMFS team are:

· Those involving nerves, blood vessels and specialised structures such as the salivary glands and their ducts
· Full-thickness lacerations of the lip
· Lacerations communicating to the oral cavity
· Injuries involving tissue loss
· Lacerations associated with hard tissue trauma. Closing the laceration may hide important clinical information and the laceration itself may be used to access the fracture site

If the laceration is appropriate for you to suture then do not delay. Here are some important principles to guide you:

· *Equipment:* Good light, fine suturing set, local anaesthetic, dental syringe, dental needle, swabs, irrigating solution such as tispets (chlorhexidine and saline), 10 or 20 ml syringe, sutures and 'steri-strips'.

· *Position:* Begin by getting the patient and yourself into a comfortable position, preferably the patient should lay on an adjustable trolley with good head support, rather than sitting upright in a chair.

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· *Local anaesthetic:* Infiltration is best performed with a dental needle and syringe. These needles are fine bore and are less traumatic to the surrounding tissues and less painful than other needles stocked in Emergency Departments! OMF surgeons tend to prefer lignocaine with adrenaline as it can offer better haemorrhage control in the area you are operating. Ideally a nerve block is used, as local infiltration can distort the anatomy.

· *Wound exploration and cleaning:* You should assess the depth of the wound, the structures involved, any possible underlying bone injury, the degree of tissue loss, and level of contamination. Once you have thoroughly made this assessment and are happy you can begin cleaning and irrigating the wound. Foreign bodies can often become trapped in tissue planes so it is essential to irrigate copiously or you risk the formation of a wound tattoo or infection. This stage should not be rushed as a poorly irrigated wound will breakdown, become infected and leave a disastrous cosmetic result. All tissue should be handled in the most delicate manner and the skin should not be grasped with toothed forceps as puncture holes will be visible.

· *Suturing:* The key to obtaining a good cosmetic result is the correct apposition of layers when suturing. Muscle to muscle closure should be with a non-dyed resorbable suture such as a 4/0 Vicryl Rapide. Ideally vertical and horizontal mattress sutures are used and the knots are buried which ensure that that do not interfere with closure of the subsequent layer.

As you reach the skin, the edges should rest together neatly and tension free if the deep layers have been closed correctly. Now interrupted, non-resorbable, monofilament sutures can be placed. The authors recommend 5/0 Novafil or Prolene for skin closure, but you should be mindful that monofilament sutures have a habit of unwinding. Therefore you must ensure that you lay your knots flats and lock the suture correctly. Placing 'steri-strips' over the wound is optional but it can help to avoid obvious contamination and aid in relieving wound tension.

· *Post-operatively:* Ensure that the patient's tetanus status is up to date. If the wound was contaminated or communicated with the oral cavity then oral antibiotics should be provided. Advise the patient not to get the wound wet, and arrange suture removal in 5-7 days.

2. Orbito-zygomatic complex fractures

The orbit and zygomatic complex are intimately related and therefore fractures of either bone go hand in hand. Fractures of the orbito-zygomatic complex are the second most common facial fracture and are usually seen in young adult males. The main cause is interpersonal violence with the majority of fractures being unilateral and on the left side resulting from a right-handed punch [7].

Clinical features:

· Flattening of the cheek.
· Periorbital ecchymosis and swelling.
· Bony step deformities around the orbit.
· Restricted mouth opening, due to the zygomatic arch impinging on the coronoid process of the mandible.
· Paraeasthesia in distribution of the infra-orbital nerve on the affected side.
· Decreased range of movements of the globe of the eye, especially if the ocular muscles are trapped in a fracture of the orbital wall or floor.
· Diplopia.
· Subconjunctival haematoma.
· Enophthalmus as a result of an increase in orbital volume due to a fracture in the orbital floor or walls.
· Surgical emphysema if the patient has blown their nose. This occurs when air has been forced into the tissue from the nasal system.

Investigations:

Occipito-mental views (OM views) at 10 and 30 degrees are the initial views of choice, however, CT scanning of the orbito-zygomatic complex is becoming more common and demonstrates fractures clearly.

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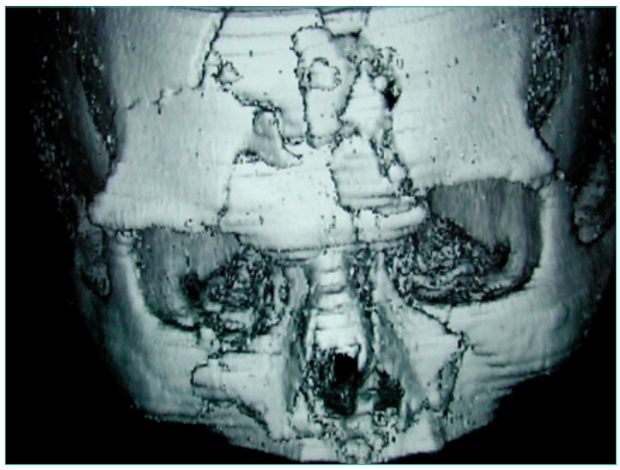

Fig 3: CT scan with 3-dimensional reconstruction showing extensive facial trauma.

Documentation: It is vital to record the visual acuity, range of eye movements, pupillary reflexes and the presence of paraesthesia clearly in the notes for comparison at the follow up appointments and post-operatively.

Initial management: If these are isolated injuries in adult patients then they may be discharged home and followed up in 4-5 days in the OMF outpatient clinic. However, children with a suspected orbito-zygomatic fracture should not be discharged until examined by a member of the OMFS team, as early surgery is sometimes required.

On discharging all patients it is crucial to give them the following information[8]:

- Advice on analgesia.

- Patients must be told to return if they develop:
- *Increasing pain in or around the eye*
- *Deterioration in vision*
- *Increasing swelling in or around the eye*

- They must avoid:
- *Nose blowing*
- *Contact sports*

3. Mandibular fractures

The first description of a mandibular fracture was around 1650 BC [9]. The aetiology of the fractures has not changed greatly since this time and currently interpersonal violence; sports injuries and road traffic collisions are the main causes of fractured mandibles in the United Kingdom [10].

The most common sites of mandibular fractures are [11]:

• *Condyle*	*36%*
• *Body*	*21%*
• *Angle*	*20%*
• *Parasymphysis*	*14%*
• *Ramus*	*3%*
• *Alveolar process*	*3%*
• *Coronoid process*	*2%*
• *Symphysis*	*1%*

Clinical features

Extra oral examination

Inspection: There is a high probability that there will be swelling and bruising at the site of the fracture and this will lead to facial asymmetry. Ask the patient if they have any sensory disturbance of their lower lip and chin. It is essential to clearly document this in the notes. The presence of any lacerations over the mandible should raise your suspicions that there is an underlying fracture.

Palpation: Gently palpate in a systematic approach, starting at the temporomandibular joint (TMJ) on the non-painful side and work your way around the lower border of the mandible noting any deformities.

Intra oral examination

Assess the following:

- Degree of mouth opening.
- Deviation of the mandible upon opening.
- Ask the patient to bite together and assess any disruption to their occlusion.
- Look for obvious step deformities. A compound fracture may be visible.
- Look for a sublingual haematoma. This should alert you to the fact that there is a fracture.

Investigations: As with all fractures two radiographic views at 90 degrees to each other are required to ensure that a fracture is not missed. Fractured mandibles require an Orthopantomogram (OPG) and posterior-anterior mandible view (PA mandible). Any missing teeth must be accounted for and a chest radiograph may be required.

Documentation: This is crucial as often in mandibular fractures there is a sensory disruption to the lower lip and chin as a result of trauma to the inferior alveolar nerve.

Initial management: Ensure that the patient has a good level of analgesia and intra-venous fluids prescribed as they will be nil by mouth. Ultimately it is likely the fracture will require open reduction internal fixation in theatre using titanium mini plates to provide semi-rigid fixation.

A minority of fractured mandible will be treated conservatively but this decision rests with the OMFS team on call.

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References

1. Careers in oral and Maxillofacial Surgery. NHS careers. h**ttp://www.nhscareers.nhs.uk/specialtytraining/oral-and-maxillofacial-surgery.shtml.**
2. British Association or Oral and Maxillofacial Surgeons. 'About the specialty'. **http://www.baoms.org.uk/landing.asp?id=3**
3. The history of the British Association of Oral and Maxillofacial Surgeons. **http://www.baoms.org.uk/page.asp?id=47**
4. Cameron M, Westcott L. Maxillofacial training is no longer than other surgical specialties. Ann R Coll Surg Eng 2008; 90: 146-149
5. Person Specification for OMFS ST3 in 2011. **http://www.mmc.nhs.uk/specialty_training/specialty_training_2011_final/recruitment_process/stage_2_choosing_your_specia/person_specifications_2011.aspx**
6. Severn Deanery website: **http://www.severndeanery.nhs.uk/**
7. Perry M. Head, Neck and Dental Emergencies. 1st edn. Oxford University Press 2005; 249
8. Bater, M. Ramchandani, P. and Brennan, P. (2005) Post-traumatic eye observations. British Journal of Oral and Maxillofacial Surgery 43 p.410-416
9. Thomas KH. Historical review of methods advocated for treatment of jaw fractures with ten commandments for modern fracture treatment. Am J Orthodont Oral Surg. 1944; 30: 39910. Patient UK: Fractured mandibles. **http://www.patient.co.uk/doctor/Mandibular-Fractures.htm**
11. Perry M. Head, Neck and Dental Emergencies. 1st edn. Oxford University Press 2005; 236

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MAXILLOFACIAL ONCOLOGY AND RECONSTRUCTIVE SURGERY

B Musgrove

Maxillofacial Oncology and Reconstructive Surgery. Good Clinical Care.

Abstract

This article outlines the role that Maxillofacial surgeons play in treating patients with tumours of the Head and Neck (H&N), in particular surgical reconstruction following ablative surgery to remove a tumour of the H&N. The vast majority of H&N tumours are squamous cell carcinomas (SCC) of the upper aero digestive tract (UAT) but there is a wide range of less common tumours which affect the H&N. This article cannot cover them comprehensively. However the principles of managing them are very similar. The challenge that confronts the clinician treating these tumours is the complexity of the anatomical region, and the constraints that this imposes on the clinical team in their quest to eradicate the disease whilst at the same time maintaining an acceptable quality of life (QOL) for their patients.

The majority of Head and Neck Cancers in the UK are now managed by the Multidisciplinary Team (MDT), a strategy which is part of the National Cancer Plan and Improving Outcomes Guidance[1, 2].

The MDT for H&N cancer comprises a wide range of professionals, including Head and Neck Surgeons, Oncologists, Radiotherapists, Neuroradiologists, Restorative Dentists, Speech Therapists, Dieticians and Specialist Head and Neck Nurses.

This composition of special skills reflects the complex problem of managing patients with H&N cancer which affects basic physiological functions including breathing, swallowing and speech.

Cancers of the H&N comprise a large variety of malignancies, the majority of which are cancers of the mucosal lining of the UAT, and include salivary gland cancers. Thyroid cancers and skin cancers, although often treated by the same clinicians, usually come under the auspices of a separate MDT and will not be discussed in this article.

Other tumours of the Maxillofacial Region are less common and usually less troublesome with a few exceptions such as some rare sarcomas.

Sarcomas represent 1% of malignancies but 10%-15% of H&N malignancies. In paediatric oncology 1 in 3 sarcomas occur in the H&N.

These tumours may be associated with rare genetic disorders or radiotherapy but frequently no obvious aetiology is recognised.

The commonest sarcomas of the head and neck include osteosarcomas, malignant fibrous histiocytoma, angiosarcoma and rhabdomyosarcoma. Surgery is usually included in the treatment with the exception of some paediatric tumours for which chemotherapy is the mainstay of treatment.

Hamartoma is a term that often confuses but essentially describes a type of tumour. It is a dysmorphic proliferation of tissue which is native to the anatomical area, does not have the capacity for independent growth but parallels the development of the surrounding tissue and first appears in childhood [3]. Examples include haemangioma and squamous odontogenic tumours. They are not infiltrative and spontaneously cease growing. They may give problems because of their anatomical position. Haemangioma of the jaws for example can cause very severe bleeding following tooth extraction and severe disfigurement.

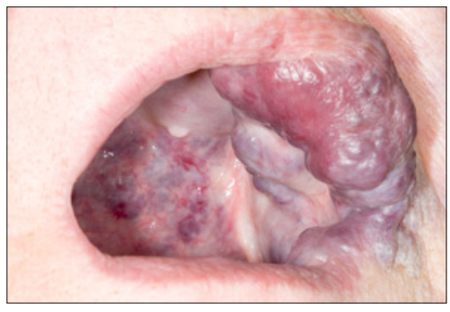

Fig 1: Haemangioma of the face and upper jaw.

Of special interest to Maxillofacial Surgeons are odontogenic tumours. which are derived from the embryonic layers of the developing tooth germ The majority of odontogenic tumours are benign and they are usually adequately managed with curettage, but a proportion need resection of the jaw and reconstruction because of their locally destructive nature and predisposition to recur after simple curettage. Ameloblastomas are included in this category and are the commonest odontogenic tumour but still only have an incidence of one per million of the population per year. There is a large variety of histological ameloblastomas[4], derived from the ameloblasts of the developing tooth which under normal circumstance go on to form mature enamel. They present as a cystic or multicystic expansion of the jaws.

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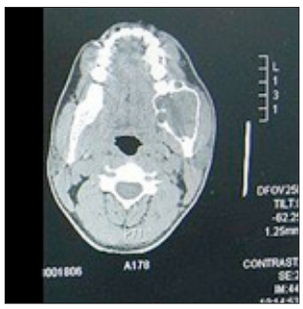

Fig 2: CT scan of an ameloblastoma of the jaw. Note that the lesion appears as a muticystic tumour with solid contents.

Odontogenic myxomas are benign tumours of odontogenic mesenchyme which are similar to ameloblasomas in clinical presentation. Cementoblastomas are hamartomatous growths of cementoblasts. They cause damage to surrounding anatomical structures, particularly the teeth and pathological fractures of the jaws. A small number of odontogenic tumours, the odontogenic carcinomas and odontogenic sarcomas are malignant. Clear cell odontogenic carcinoma and the ameloblastic fibrosarcoma are two such examples.

Salivary glands are divided into major and minor glands. The major glands comprise the paired parotid, submandibular and lingual salivary glands. The minor glands are those found scattered throughout the mucosa of the oral cavity. Tumours of the salivary glands have a great variety of histopathological properties and a varied malignant potential. The parotid gland is the commonest site for tumours, 80% of which are benign, whereas tumours of the minor salivary glands have a benign to malignant tumour ratio of 1:1. Most salivary gland tumours are derived from ectodermal cells but there are other tumours found in the salivary glands, such as lymphomas, melanomas and skin cancer metastasis.

Salivary gland cancers can invade the surrounding anatomical structures such as the base of skull and the facial nerve in the case of the parotid gland and the minor salivary gland tumours of the palate can destroy the palate and extend into the nose and sinuses.

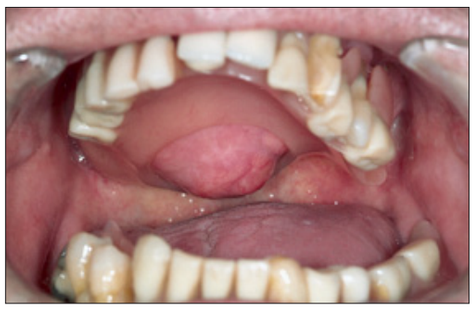

Fig 3: Adenocystic carcinoma of a minor salivary gland displacing the patient's upper denture.

Cancers of the upper aerodigestive tract:

a. Site specific tumours of the UAT include squamous cell carcinoma of the oral cavity, oropharynx, nasopharynx, hypopharynx and larynx, representing 90% of cancers of the H&N.

b. The less common malignancies include cancers of the sinunasal tract, salivary glands, and lachrymal glands.

c. The overall five year survival rate of carcinomas of the UAT is 50%.

Aetiology

UAT cancers are associated with older men, over 50, who consume alcohol and tobacco excessively[5,6,7].However there has always been a group of patients who do not readily fall into this category.

A recent trend has been an increasing incidence in younger patients with oral cancer and there is an overall increase in oral cancer in the UK[8].

The male to female ratio varies from 2:1 to 15:1 depending on the site of the cancer in the H&N.

Tobacco is carcinogenic for all the sites of the UAT and alcohol increases the risk of squamous carcinoma in the oral cavity, oropharynx, hypopharynx and larynx. The combination of tobacco usage and alcohol is synergistic, for all sites in the UAT.[9]

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In some Asian communities PAAN usage[10] can cause oral cancer. PAAN (a mixture if spices, slaked lime, areca nut wrapped in betel leaf) Fig 4, is held in the oral cavity between the buccal mucosa and the gums where is gives rise to squamous cell carcinoma. It is a habit practised particularly in field workers in Asia which has elevated the incidence of oral cancer to one of the most common cancers in Asia. PAAN causes submucous fibrosis of the oral mucosa which presents as restricted mouth opening because of fibrosis in the submucosa and connective tissue of the cheeks. Sub mucous fibrosis is a premalignant lesion which can progress onto squamous cell carcinoma. PAAN can be purchased in Asian shops and restaurants in the UK.

Fig 4: PAAN is a mixture of spices, areca nut, slaked lime and sometimes toacco rolled in a betel leaf and is held in the mouth between the cheek and the gums where is can cause squamous cell carcinoma or submucous fibrosis, a precursor of SCC.

Human Papilloma virus (HPV) sero-positivity, in particular types 16 and 18 is associated with oral and oropharyngeal cancer[11, 12]. In the USA this is becoming the commonest presentation of oropharyngeal cancer and seems to be on the increase. Vaccination for HPV may reduce or even eliminate HPV related carcinoma of the UAT.

Dietary factors have been demonstrated to influence the risk of developing cancers of the UAT. Mediterranean diets have been shown to reduce the risk of oral/oropharyngeal and laryngeal cancers, particularly those rich in polyunsaturated fats, thiamine and vitamin B12 which play a role in maintaining the health of the mucosa and reducing the affect of carcinogens on the mucosa. Iron deficiency anaemia can cause post-cricoids carcinoma.

Genetic factors can play a role and the rare genetic disorder of Fanconis Anaemia is associated with Proliferative Verrucous Leukoplakia, a premalignant condition which progresses to carcinoma.[13]

Nasopharyngeal cancer is associated with Epstein Barr virus, malignancies of the sinunasal tract with smoking and woodworkers and lip cancer with smoking but more commonly exposure to ultraviolet light. Lower lip cancer is ten times more common than upper lip cancer.

Premalignant conditions

White or red patches, leukoplakia and erythroplakia and speckled leukoplakia (a mixture of red and white) are recognised as having the potential to transform into cancer (4%-25%) depending on the type and is a common cause of cancer of the mouth.

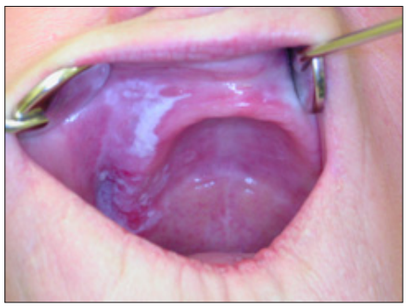

Fig 5: Speckled leukoplakia of the oral mucosa, a premalignant condition with a high potential for malignant transformation.

The Scottish Referral Guidelines for Cancer recommend urgent referral of any patients exhibiting the following:

Red or red and white patches of the oral mucosa which persist for more than three weeks at any particular site, ulceration of oral mucosa or oropharynx which persists for more than three weeks, oral swellings which persist for more than three weeks, unexplained tooth mobility not associated with periodontal disease persistent, particularly unilateral, discomfort in the throat for more than four weeks, pain on swallowing persisting for three weeks that does not resolve with antibiotics, dysphagia which persists for more than three weeks, hoarseness which persists for more than three weeks, stridor (requires same day referral), unresolved head or neck mass which persists for more than three weeks, unilateral serosanguinous nasal discharge which persists for more than three weeks, particularly with associated symptoms facial palsy, weakness or severe facial pain or numbness orbital masses ear pain without evidence of local ear abnormalities.

The IOG for Head and Neck cancer stipulate that any patient should have their treatment commenced by the relevant specialist within 62 days of referral from the primary clinician to a specialist team.

MAXILLOFACIAL ONCOLOGY AND RECONSTRUCTIVE SURGERY

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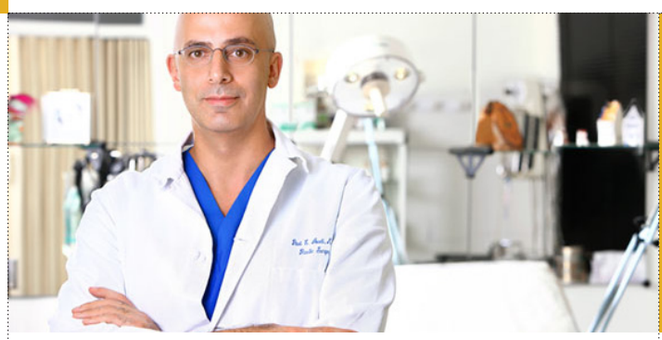

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Diagnosis relies on clinical examination, endoscopy, fine needle aspiration cytology (FNA) of neck lumps and open biopsy of the primary lesion possibly with an examination under anaesthetic (EUA) if necessary.

On presentation the disease need to be staged with the help of special investigations including CT and MRI scans of the H&N, together with either a chest radiographs or a CT of the chest depending on local protocol.

Sometimes a patient may present with metastases but no detectable primary cancer which is then described as an occult primary. Positron Emmision Tomography (PET) may then be employed to help detect the primary which may not necessarily be in the H&N. Fig 6 Positron Emission Tomography showing a hot spot in the neck, likely to be a metastasis.

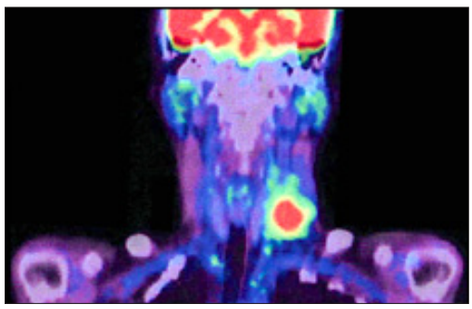

Fig 6: Positron Emission Tomography showing a hot spot in the neck, likely to be a metastasis.

IOG for H&N cancer advises submitting details of any patient with H&N cancer to the H&N MDT and this is usually done following histopathological confirmation and the necessary investigations. Staging which describes the extent of the lesion then informs the MDT to enable best practice treatment to be recommended.

The Maxillofacial Surgeon is most likely to be presented with cancer of the oral cavity and oropharynx. The oral cavity includes the buccal mucosa, mucosal surface of the lip, the floor of the mouth, the anterior two thirds of the tongue and the retromolar fossa, hard palate and alveolus. The oropharynx includes the posterior third of the tongue, the soft palate and the tonsil. Cancers at these sites can extend into the neck, the sinuses, the infratemporal fossa, the orbit and the base of skull. Spread to the regional nodes in the neck occurs by means of lymphatic spread. Distant metastatic spread tends to occur late in the course of the disease and the most common sites are the lungs and bone.

Staging:

In the UK staging follows the Union International Contre le Cancer (UICC)/TNM Classification of Malignant Tumours. T stands for the size of the tumour, N for the degree of nodal involvement in the neck and M for the extent of distant metastasis. The cancer is divided into early disease, (stage 1 and 2), and locally advanced disease, (stage 3 and 4)[14]. Early detection and treatment of H&N cancer, including oral cancer, improves the prognosis.

Cancers of the maxillofacial complex can seriously impair the following:

- Swallowing
- Speech
- Chewing
- Airway
- Smell
- Sight
- Sensation
- Taste
- And disfigurement including loss of motor activity of the tongue and face.

Contemporary treatment modalities attempt to preserve these functions and to restore them when possible. This usually involves ablative and reconstructive surgery and for malignant tumours treatment will often be a combination of surgery, radiotherapy and chemotherapy.

The rest of this article will discuss the reconstruction of the maxillofacial complex following ablative surgery for tumours of the face mouth and jaws, The QOL of patients treated with surgery has improved considerably over the last 25 years because of the advances in reconstructive surgical techniques. The most significant advance has been the development of microvascular surgery.

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The history of reconstructive surgery of the H&N:

Modern reconstructive surgery of the H&N grew out of the two world wars when pioneers such as Gillies and McIndoe developed means of transferring skin flaps to the face to reconstruct severe facial injuries caused by shrapnel and burns.

The Reconstruction Ladder:

After the benign or malignant tumour has been excised then the surgical defect has to be repaired. The choice of surgical technique depends on the size of the surgical defect and the anatomical structures that need to be preserved or restored. For example, early disease of the tongue might be excised and allowed to heal by itself (called healing by secondary intention), Fig 7, or repaired by suturing whereas removing part of the mandible because of a large benign tumour such as an ameloblastoma or a cancer will demand reconstruction of the surgical defect with a microvascular free flap incorporating bone.

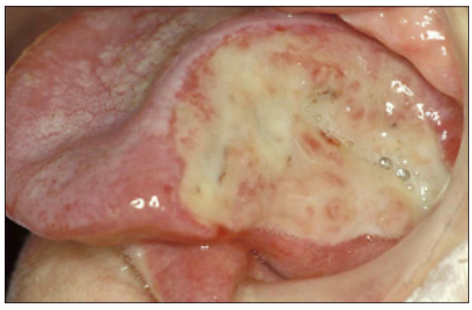

Fig 7: tongue healing by secondary intention after laser excision of a small SCC.

The surgical ladder may be summarised as follows :

1. Primary closure
2. Healing by secondary intention
3. Skin grafting (split or full thickness)
4. Local flaps
5. Regional pedicle flaps
6. Free tissue transfer

Primary closure is best for small and linear lesions when suturing will not create tension on the wound edge with consequent wound dehiscence. Healing by secondary intention is suitable for small cancers of the tongue.

Local flaps are skin or mucosal flaps which are left attached and depend on the dermal and sub-dermal vascular supply to the flap to maintain its viability. These skin flaps are commonly used for treating skin cancers. when the surgical defect is relatively simple.

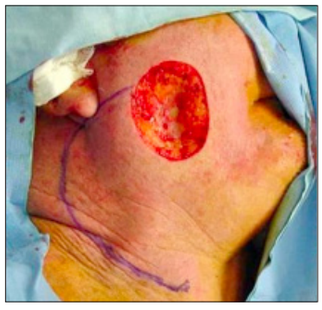

Fig 8: Excision of malignancy of cheek with the proposed local skin flap marked out.

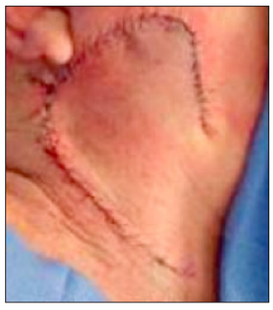

Fig 9: Flap has been dissected in the subcutaneous plane and rotated into the surgical defect.

A versatile flap for use in reconstruction of the oral lining is the nasolabial flap.

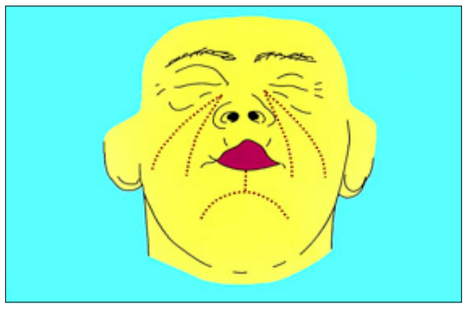

Fig 10: Nasolabial flap outlined in the nasolabial fold of the cheek.

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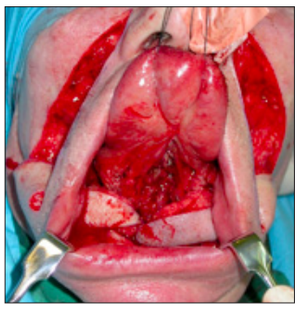

Fig 11: The nasolabial flaps have been raised in a subcutaneous plane, passed through a tunnel made in the cheek and will be sutured into the floor of the mouth under the tongue to repair the surgical defect.

Axial pattern flaps have a more dominant vascular supply and two such flaps which were in common usage until the advent of free tissue transfer were the deltopectoral flap which derives its blood supply from perforating branches of the internal mammary artery and the forehead flap which is nourished by the superficial temporal artery and can either be used to cover facial defects or in the case of the forehead flap passed inside the zygomatic arch to replace the lining of the mouth. The combination of the forehead flap for oral lining and a deltopectoral flap for external skin cover was a common way to replace full thickness defects of the face for some time before the advent of microvascular surgery.

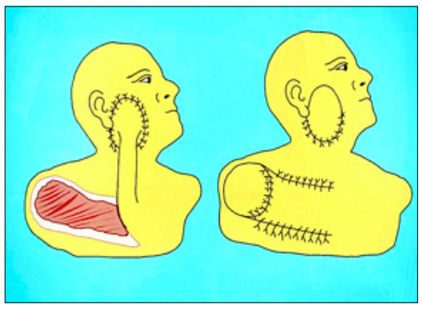

Fig 12: The delto-pectoral flap which has a blood supply from the perforating branches of the internal mammary artery.

Fig 13: The forehead flap supplied by the superficial temporal artery, after it has been lifted in the subaponeurotic plane and tunnelled through the cheek or under the zygomatic arch into the mouth.

Both of these techniques suffer from being a two stage operation, the second stage being to divide the base of the flap after three weeks when a collateral blood supply growing in from the surrounding tissues has had time to establish itself.

They were superseded in H&N reconstruction with the advent of myocutaneous flaps, which depend for their blood supply on the perforating branches of the vascular pedicle supplying the muscle. One workhorse is the pectoralis major flap. Fig 14 which can be combined with a neck dissection and tunnelled through the neck into the mouth or pharynx. The other workhorse has been the latissimus dorsi myocutaneous flap Fig 15, which again is tunnelled into the neck but also needs to be combined with a neck dissection in which the sternomastoid muscle has been removed. There are other examples but they all suffer from being limited in their reach by still being attached by their vascular pedicle. It was almost a decade later that surgeons realized that the pedicle could be divided and transferred using microvasular surgery

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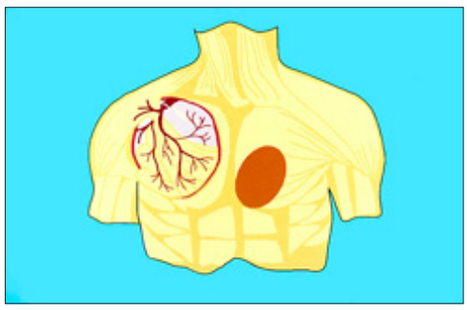

Fig 14: The Pectoralis major myocutaneous flap. The skin paddle is elevated on a portion of the pectoralis major muscle which has as its blood supply the pectoral branch of the thoracoacromial artery, a branch of the second part of the axillary artery.

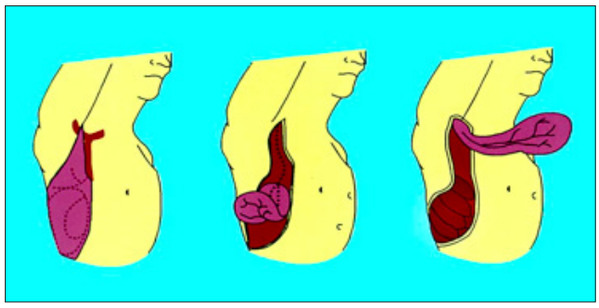

Fig 15: The latissimus dorsi myocutaneous flap which is a skin paddle lying on the latissimus dorsi muscle which is supplied by the thoracodorsal artery a branch of the subscapular artery, the largest branch of the axillary artery.

2. Microvascular surgery and free tissue transfer

By dividing the vascular pedicle and anatomising the blood vessels to vessels at the recipient site then the way was opened up to move any tissue with a vascular pedicle from any part of the body to the H&N. This technique has facilitated the development of chimeric flaps composed of different tissue types such as bone, skin, muscle and nerve to reconstruct more complex anatomical defects which may occur following surgical ablation of H&N tumours. The flap can fail if the anastomosis becomes obstructed by a blood clot but survival rate is above 95% in experienced hands.

.A large number of microvascular free flaps have now been described which bring diversity to the reconstructive surgeon's armamentarium and enable the surgeon to perform more ambitious surgery safe in the knowledge that he will have the reconstructive techniques at his disposal to keep complications at acceptable limits and afford an acceptable QOL to his patient.

There follows a description of how modern techniques attempt to restore key anatomical structures which comprise the craniofacial anatomy. These structures include the lips, the tongue, the mandible, the maxilla, the nose, the cheeks, the orbit and the cranial base.

Swallowing and Speech

Lip reconstruction

The lips are important structures not only from an aesthetic point of view but also from a functional one. The lips are important for speech but equally as important for swallowing. If an adequate seal cannot be maintained and the lips are rendered incompetent then it is impossible to retain liquid and solids at the commencement of the swallowing reflex. Microvascular free transfer to date is not sophisticated enough to re-establish the oral sphincter and it is more effective to transfer full thickness lip from one lip to another to achieve this. Local flaps include the Abbe flap Figs16-18, the Estlander flap and the Karapandzic flap. These techniques can restore a competent sphincter for up to two thirds loss of the lip but when the surgical defect is too large for reconstruction with a lip flap, then a free flap will be necessary, but this gives a poor functional result because because the tissue merely provides a static platform with which the opposing forms a seal.

Fig 16: Abbe Flap marked out on the upper lip. A small cancer is visible on the lower lip. The blood supply is from the labial artery.

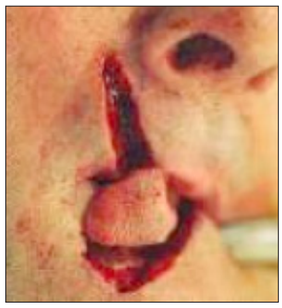

Fig 17: The full thickness flap rotated on the lip pedicle containing the labial artery.

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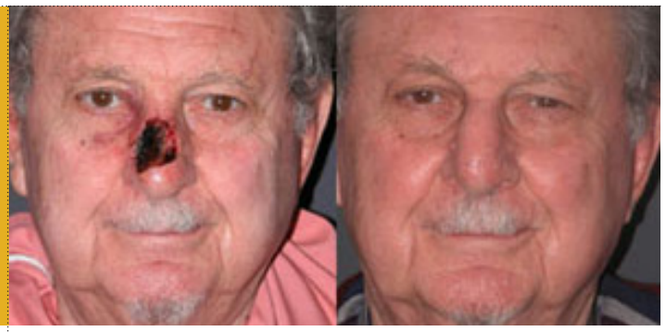

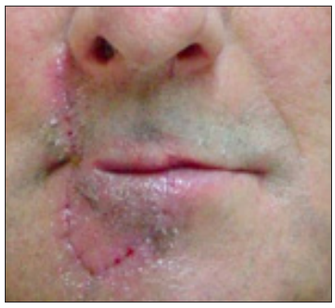

Fig 18: The lip repair three weeks later. The commisure of the mouth will be restored with a small surgical revision.

Tongue reconstruction

Resections of the tongue and the floor of the mouth affect swallowing, speech and taste. Speech may become unintelligible and swallowing so dysfunctional that any attempt to swallow solid or liquids can result in aspiration pneumonia. Post-operative swelling can obstruct the airway which may need to be protected with a temporary tracheotomy until swallowing can be assessed. If any sizeable resection of the tongue is intended then a temporary tracheotomy should be considered at the same time but prior to starting the primary surgery in order to help the anaesthetist to keep the anaesthetic tube away from the operative site.

Small cancers of the tongue can be excised with laser and the surgical wound sutured or allowed to heal by secondary intention. This applies to small T1 squamous cell carcinomas of the tongue. Resection of larger cancers with a clear margin of 5mm cause significant loss of function and most maxillofacial surgeons would transfer tissue into the surgical defect to reduce the amount of scarring and distortion of the anatomy that could otherwise occur with healing by secondary intention.

Patients having any form of glossectomy for cancer usually have a neck dissection at the same time to remove any lymph nodes in the drainage field for the tumour and to expose suitable recipient blood vessels in the neck for the microvascular transfer of a free flap.

The most commonly used microvascular flap for reconstructing the tongue is the Radial Free Forearm Flap (RFFF), which has the advantage that it is a thin pliable skin paddle that serves to replace the floor of the mouth (FOM) and tongue whilst preserving the function of the remaining anatomy. When the (FOM) has to be removed together with most of the tongue then flaps incorporating muscle and skin may provide a better reconstruction by filling the volume defect and minimising the "sump" in the reconstructed FOM where saliva can pool. Appropriate flaps include the rectus abdominis flap and the antero-lateral thigh flap.

Jaw Reconstruction

Prior to the introduction of free tissue transfer with microvascular - anastomosis restoring the integrity of the jaws was much more problematic than it is today. Small non-vascularised bone grafts may survive particularly if the periosteum is preserved. Often the periosteum cannot be preserved, particularly with cancer patients. The non-vascularised bone graft is vulnerable to infection from the mouth and to absorption.

Transferring tissue with its vascular pedicle significantly reduces these complications to the extent that survival of transferred tissue is better than 95%. Moreover employing this technique facilitates the design of complex reconstructions so that bone, skin, muscle, vascular and neural tissue can all be transferred to reconstruct a surgical defect.

By virtue of the new blood supply wound healing is promoted. And postoperative complications reduced including salivary fistula formation, infection and wound breakdown.

There are four donor sites for bone transfer using microvascular surgery which are commonly utilized to reconstruct the jaws. These are the fibula, transferred on the peroneal artery, the superior iliac crest transferred on the deep circumflex iliac artery (DCIA flap), the lateral border of the scapula on the circumflex scapular artery and the radius on the radial artery. Each has its own merits depending on the anatomical defect to be restored and the medical circumstance of the patient.

The RFFF can be transferred with part of the radius if bone is required to reconstruct the jaw. Fig 19-21, although there are more appropriate bone flaps because the bone is quite small, not suitable for dental implants and there is a risk of post-operative fracture of the radius. The Forearm has to be immobilized in an above elbow cast for six weeks.

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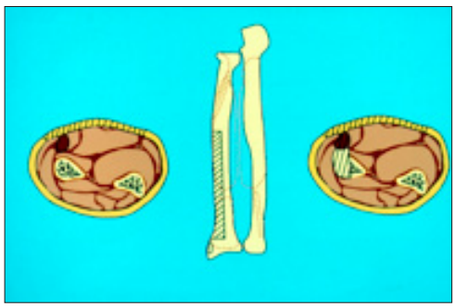

Fig 19: The thin skin of the volar surface of the forearm is raised with the radial artery or with the radial artery and upto half the radius.

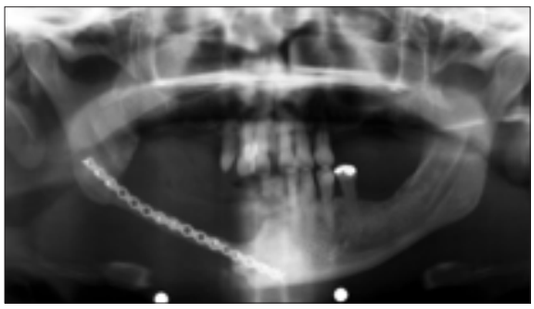

Fig 20: Reconstructed mandible and floor of mouth with a composite forearm flap.

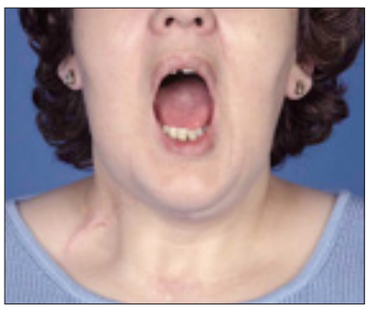

Fig 21: One year postoperative, same patient demonstrates good function and cosmesis and able to wear a denture over the reconstructed jaw.

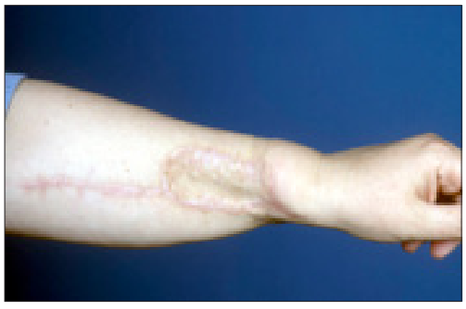

Fig 22: Donor site on the none dominant arm.

In 1975 Taylor and colleagues described the free fibula flap, while Hidalgo applied the technique for mandibular reconstruction in 1989[15]. This has several advantages over the RFFF including a much thicker and longer piece of bone and one that is big enough to accept dental implants. Figs 23-24.

Urken described the use of the DCIA flap also in 1989. This is arguably provides the best bone for dental implants and is particularly suitable for reconstructing the anterior mandible in the dentate patient Figs: 25-26.

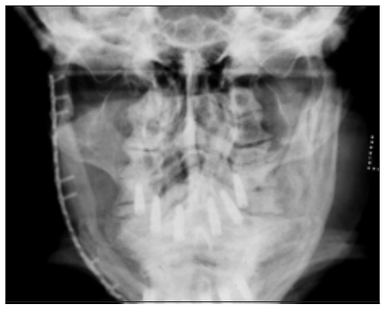

Fig 23: Mandible reconstructed with fibula flap and dental implants ready for dental restoration.

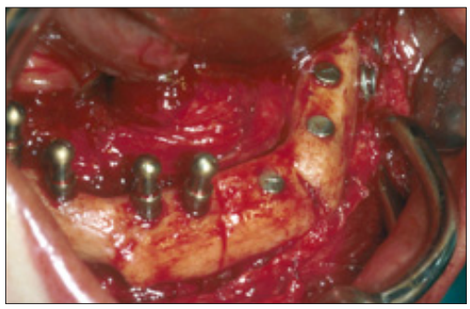

Fig 24: Fibula graft after placement of dental implants.

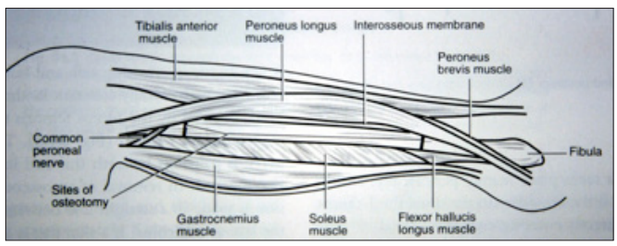

Fig 25: Illustration showing length of fibula to be harvested and the peroneal artery which supplies it.

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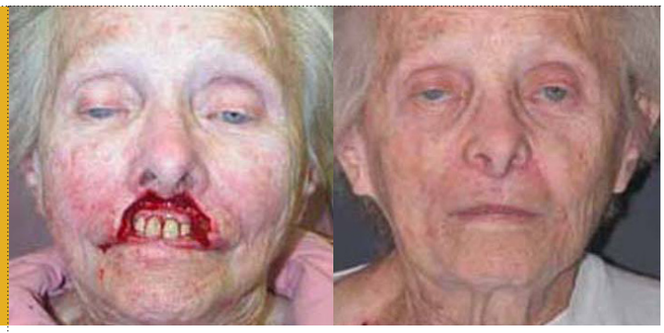

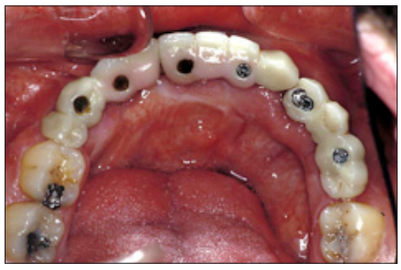

Fig 26: Anterior mandible which has been reconstructed with an Iliac crest graft based on the deep circumflex Iliac artery, after dental implants have been inserted and a dental bridge fitted.

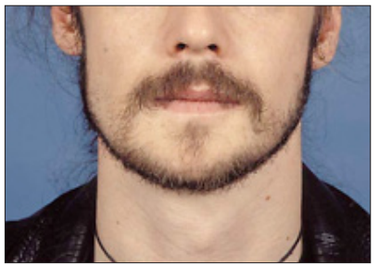

Fig 27: Post reconstruction with dental prosthesis fitted illustrating good bone height and lip support with good symmetry of the jaw.

Chimeric flaps

When it is necessary to transfer a variety of tissue types to enable reconstruction of multiple tissue layers then those free flaps supplied by the subscapular artery Fig: 28, are suitable. An example is illustrated of a patient who underwent resection of his cheek mandible and lip. Fig: 29 The reconstruction comprised a scapula flap to restore the lining of the cheek, the mandible and the overlying skin, reconstruction of the commisure of the lip with an Estlander flap and restoration of facial movement by anastomosing the stump of the buccinator branch of the facial nerve to the thorocodorsal nerve innervating the latissimus dorsi muscle.

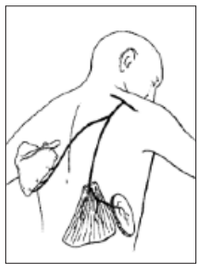

Fig 28: chimeric flap comprising bone from the scapula, latissimus dorsi muscle and a skin paddle all supplied by the subscapular artery, a branch of the axillary artery.

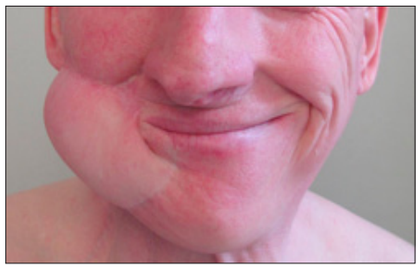

Fig 29: Result one year after resection of a large carcinoma of cheek which had recurred following radiotherapy, using a chimeric flap based on the subscapular artery, which replaced the lining, the mandible, muscle which was innervated by anastomosing the thoracodorsal nerve innervating latissimus dorsi muscle to the proximal branch of the buccinator nerve, and a paddle of skin for the cheek skin.

Establishing the blood supply to a flap

Microvascular surgery depends on accurate anastomosis of small blood vessels, the artery supplying blood and the veins draining blood. These vessels are usually 1 to 3 mm in diameter and the anastomosis is performed with the aid of a microscope. Fig 30. In specialist centres the flap success rate is in the order of 95% or better.

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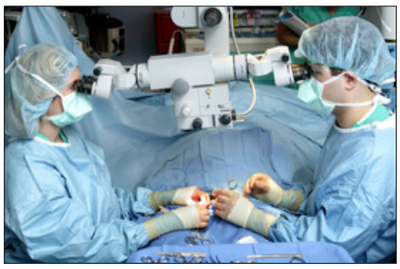

Fig 30: Operating microscope with a surgeon and his assistant performing a microvascular anastomosis.

Reconstruction plates

The introduction of reconstruction plates made from biocompatible material such as titanium which can be prefabricated on accurate three dimensional models of the jaws has helped to reproduce the morphology of the jaw at surgery. These models are constructed using data from CT scans which is fed into a milling machine to produce an accurate three dimensional model of the jaws in plastic. If the anatomy is so badly damaged by disease then a mirror image of the normal side can be used to reproduce the jaw or any other part of the facial skeleton. These models can then be used as a template for prefabricating a reconstruction plate. Fig. 31.

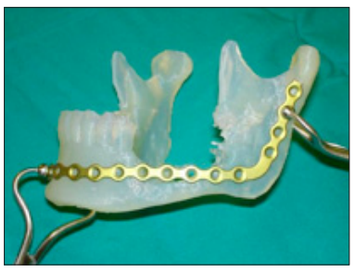

Fig 31: A reconstruction plate being prefabricated on a model of the mandible which has been invaded by cancer of the retramolar fossa.

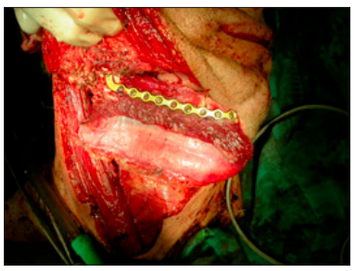

Fig 32: bone is osteotomised and fitted to plate which has been prefabricated and fitted to mandible. The arterial anastomosis is then performed followed by anastomosing the veins.

Dental rehabilitation

Following the reconstruction it can be difficult to replace teeth or provide dentures. This is because the new shape of the reconstructed jaw might not be retentive and if the patient has had radiotherapy then the quality and quantity of saliva will be reduced which compounds the difficulty in retaining a dental prosthesis. Dental Implants can be fixed into the reconstructed mandible or maxilla to retain a prosthesis Figs 32-33. All MDTs now have as part of the team a Dental Surgeon skilled in dental rehabilitation using dental implants. These implants have also been modified to retain artificial eyes, noses and ears.

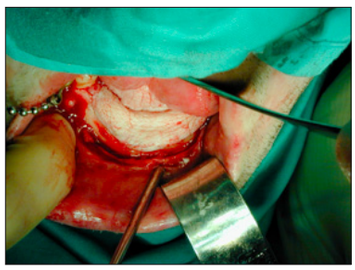

Fig 32: The alveolus of the mandible has been removed together with a cancer of the floor of the mouth and reconstructed with a RFFF.

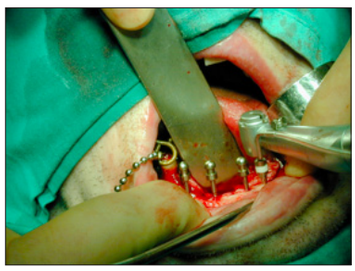

Fig 33: At One year following the initial surgery, dental implants are fixed into the mandible to support a dental bridge.

Following a maxillectomy the surgical defect can be obturated with a prosthesis, retained on implants if necessary. The surgical defect can also be reconstructed with a free flap. Both methods aim to prevent nasal escape on speaking, oronasal-reflux on eating and drinking and to restore the dentition and facial aesthetics of the patient.

Cranial base

Tumours that invade the cranial base have always been difficult to deal with because of the necessity to seal the cranial cavity from the nose, sinuses and mouth. Open communication between the cranial cavity and the sinunasal tract will lead to intracranial infection. Microvascular surgery can be used to seal off the cranial cavity and prevent ascending infection. It is particularly important to close the meninges to avoid a cerebro-spinal fluid (CSF) leak. There is a risk of non-vascularised tissue repairs breaking down and vascularised tissue is much safer, particularly if the tumour has been irradiated. Consequently more ambitious craniofacial surgery for malignancies is now possible.

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Osteoradionecrosis (ORN)

A common problem that the H&N Reconstructive Surgeon is faced with is ORN. Radiotherapy damages the cellular matrix of the jaws, reduces the blood supply and the ability for cellular repair. Although it can occur spontaneously it more usually follows a surgical insult, especially to the mandible. The surgical insult is usually a tooth extraction, and sometimes a biopsy or traumatic ulcer caused by dentures. It can vary from mild to severe but is progressive and causes extreme pain from dying and dead bone, soft tissue infections, sinuses and fistulae. Figs 34-35. It is imperative that the dentition of H&N patients is screened prior to treatment starting so that any potentially compromised teeth can be dealt with to avoid the need for extractions. Following treatment the patients need to be followed up by a dental specialist conversant with the risks of ORN in H&N patients. It is estimated that the risk of ORN of the mandible following radiotherapy can be as large as 8%.when the dose of radiotherapy is over 60Gy and even greater when combined with chemotherapy.

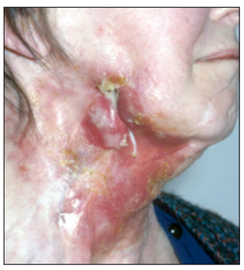

Fig 34: A patient with severe ORN of the lower jaw with necrotic bone and a pathological fracture, fistulae and damaged skin.

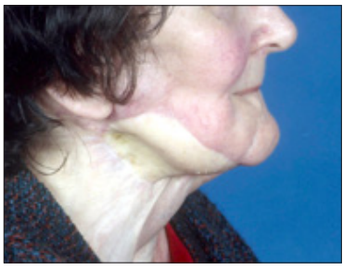

Fig 35: Following resection of the affected mandible, oral mucosa and skin and repair with a microvascular composite flap (fibula and RFFF).

Flap monitoring

Microvascular anatomises are susceptible to failure because of blood clotting at the suture line. For this reason flaps need to be monitored carefully for colour, capillary refill and temperature, for the first 72 hours following surgery table 1. The commonest cause of failure is a blood clot at the venous or arterial anastomosis caused by faulty suturing technique, pressure from haematomas, surgical drains etc.

Observation	Normal Flap	Arterial Insufficiency	Venous congestion
Colour	normal skin tone	pale	purple/mottled
Turgidity	soft	flacid	turgid
Temperature	warm	cold	cold
Capillary refill	2-3 seconds	Absent/sluggish more than 6 seconds	Brisk less than 3 seconds

Table 1 : Observation of the skin of a transplanted tissue flap, which should be performed at regular intervals for the first 72 hours.

Clinical observation of the flap has several weaknesses since it depends on the interpretation of the appearance of the flap. The skin paddle may be difficult to see, if it is in the oropharynx for example. It may be impossible to see because it is buried when used in a pharyngeal reconstruction and if bone is used without a skin paddle. In these circumstances Doppler monitors which give an audible signal can be used to warn the medical attendants of any interruption in the blood flow across the anastomosis. Microdoppler probes can be fixed around the vessel and led from the surgical site to a monitor at the bedside.

A more recent technique employs microdialysis of tissue fluid which is regularly sampled from the flap and its glucose and lactate levels measured. An increase in the lactate to glucose ratio indicates ischaemia in the flap. The monitor provides a trace for easy interpretation of the chemical trend in the extracellular fluid of the flap and can give a five to six hour window for re-exploring the anastomosis and salvaging the flap. If the flap dies then it has to be removed and another reconstruction performed.

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Airway management;

Nursing and medical staff must be familiar with managing tracheotomies on the ward and being able to recognise and deal with their failures. The following instructions are taken from the guidelines of the H&N Unit at the Manchester Royal Infirmary; The management of temporary tracheotomies:

1) Observe patient: including colour, respiratory rate and breathing pattern.

2) Suction via tracheostomy as indicated.

3) Ensure adequate humidified oxygen is utilised to prevent drying of secretions thus maintain patency of tube, saline nebulisers should be considered.

4) Ensure ties are secure to hold tube in place.

5) Ensure site around tracheostomy clean and dry, Lyofoam T insitu.

6) If site shows signs of infection swab and send for C&S.

7) Ensure cuff tolerated down prior the removal of the tube and the introduction of speaking tubes or valves. For eating and drinking cuff must be tolerated down or at least partially down: Patient must be able to cough effectively in to mouth and have adequate swallow prior to cuff deflation.

8) If the patient is ready to eat please ensure that a swallow assessment is carried out by a speech therapist (or trained personnel).

9) Ensure the inner tube is cleaned at least once a shift,more if required: Use small brush provided and sterile water, store in sealed pot.

10) If inner tube kinked or broken replacement inner must be used.

11) If the patient is having difficulty breathing, ensure tube is patent- try suctioning the tracheostomy tube. If the tube remains blocked remove the inner tube and replace with new white inner tube. If the tube is still blocked – deflate cuff – assess whether the tube can be removed and administer 100% O_2 via face mask seek medical advice.

Nutritional Support

Patients with H&N cancer of the UAT require nutritional support if they have not been eating prior to surgery, and when surgery is extensive and when combined with radiotherapy, chemotherapy or both. Naso-gastric tubes are not well tolerated by H&N cancer patients. Percutaneous endoscopic gastrostomy feeding has become the most acceptable and safest method of long term nutritional support in these patients.

Fine bore gastrostomy tubes can be inserted with the aid of an endoscope as an outpatient procedure. The endoscope transilluminates the gastric wall and facilitates the passing of a transcutaneous tube which is then held in place with an internal balloon.

Complications are low, major complications between 3% and 8% and minor around 14%. Mortality from the procedure is less than 1%.

Major complications include internal leakage and gastric perforation leading to peritonitis, gastric bumper syndrome (migration of the fixation device into the skin and muscle of the abdominal wall and aspiration. The incidence of aspiration is low compared to naso-gastric tubes, especially when the patient has swallowing difficulties.

Minor complications include skin infections, dislodgement, and tube blockages.

Occasionally they are contraindicated if for example there is stenosis of the oesophagus and the tube cannot be passed endoscopically in which case an alternative technique is the radiologically inserted gastrostomy tube. Obesity, and previous gastric surgery and other anatomical abnormalities may also make it difficult . Rarely it may be necessary to insert the tube surgically.

An important advantage of gastrostomy tube feeding is that it can be managed at home by the patient and their helpers and they are cost effective compared to alternative strategies for nutritional support.

MCQs

Which statements are true?

Question 1 - The National Cancer Plan:

1. The National Cancer Plan has been designed to centralise cancer services.
2. Improving Outcomes Guidance is a component of the national Cancer Plan.
3. The IOG recommends that major cancer specialties have designated Multidisciplinary Teams (MDT).
4. The National Cancer Plan organises Cancer Research.
5. Robust data from cancer activity in the UK has guided policymakers.

Question 2 - Which of the following statements are true:

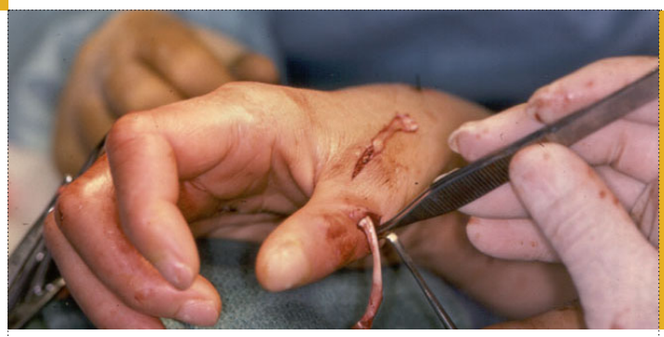
1. Head and Neck Cancer includes benign and malignant tumours of the H&N.
2. The commonest cancers of the H&N are odontological cancers.
3. Odontological tumours are derived from the endothelial lining of the mouth.
4. Cancer of the oesophagus is treated by H&N Surgeons
5. The commonest cancer of the H&N is squamous cell carcinoma of the mucosa.

Question 3 - Tumours:

1. Ameloblastomas are not dangerous and can be treated with curettage only
2. Odontogenic tumours can cause displacement and absorption of teeth.
3. The commonest salivary gland tumour is a Pleomorphic Adenoma
4. Pleomorphic adenomas are always benign.
5. A swelling of the palate is most likely to be caused by an infected tooth.

MAXILLOFACIAL ONCOLOGY AND RECONSTRUCTIVE SURGERY

B Musgrove



Maxillofacial Oncology and Reconstructive Surgery. Good Clinical Care.

Question 4 - MCQ 4 Microvascular Surgery:

1. Monitoring microvascular reconstructions on the ward is necessary for 24 hours.
2. If the flap fails the most likely change in its appearance is that it will turn pale.
3. If using microdalysis to monitor the flap a rise in the extracellular lactate and a fall in the glucose reading will mean that the flap is healthy
4. Vascularized bone is at high risk of becoming infected
5. It is important to keep firm pressure on the flap to stop it bleeding.

Question 5 - Airway management:

1. A temporary tracheostomy is always necessary in H&N Surgery.
2. A temporary tracheostomy can never block.
3. A blocked tracheostomy can be removed to clear the airway.
4. Stridor and confusion are signs of a blocked airway.
5. The way to manage a blocked airway is to phone for help.

Answers

Question 1. Correct answers 1 and 3.

The following extract summarises the aims of the National Cancer Plan. It has not intended to prescribe centralised units but has intended to encourage interdisciplinary organisation into MDTs. It has not taken over nor funded any research but has pressed MDTs to show evidence of research activity at its peer review.. There has been a dearth of good data and one of the stipulations is that each cancer specialty should produce national data on its activity and outcomes

The NHS Plan (July 2000) presents the government's strategy for investment and reform across the NHS, and gives cancer services high priority. This document provides a detailed account of the government's comprehensive national programme for investment in and reform of cancer services in England, which aims to reduce death rates and improve prospects of survival and quality of life for cancer sufferers by improving prevention, promoting early detection and effective screening practice, and guaranteeing high quality treatment and care throughout the country.

The Cancer Plan is particularly committed to addressing health inequalities through setting new national and local targets for the reduction of smoking rates, the setting of new targets for the reduction of waiting times, the establishment of national standards for cancer services, and investment in specialist palliative care, the expansion and development of the cancer workforce, cancer facilities, and cancer research.

Question 2. Answer: 5.

Head and Neck Cancer means cancer of the UAT, 90% of which are squamous cell cancers. Malignant odontological tumours are very rare.
Odontological tumours are derived from the embryonal layers of the developing tooth germ.

Cancer of the oesophagus does not fall into the remit of the H&N Surgeon except for the cervical oesophagus which would betreated by the ENT Surgeons.

Question 3. Answers 2, 3, 5 are correct.

Ameloblastomas can only be treated with curettage if small and not near to any vital structures. There is a high risk of recurrence after curettage and if the tumour involves the orbit and base of skull it can be very difficult to eradicate. The commonest site for salivary gland tumours is the parotid gland and 80% of them are benign Pleomorphic Adenomas. can undergo malignant change iinto carcinoma ex-pleomorphic adenoma, a risk that increases with time. Dental infection is still the commonest cause of swellings around the mouth but they will respond to dental extraction or drainage of the abscess.However there are plenty of other causes such as a carcinoma of the antrum which may have other signs and symptoms to suggest the diagnosis including, nose bleeds, displacement of the eye, double vision, hearing loss, numbness of the face.

MAXILLOFACIAL ONCOLOGY AND RECONSTRUCTIVE SURGERY

B Musgrove

Question 4. Answers: None.

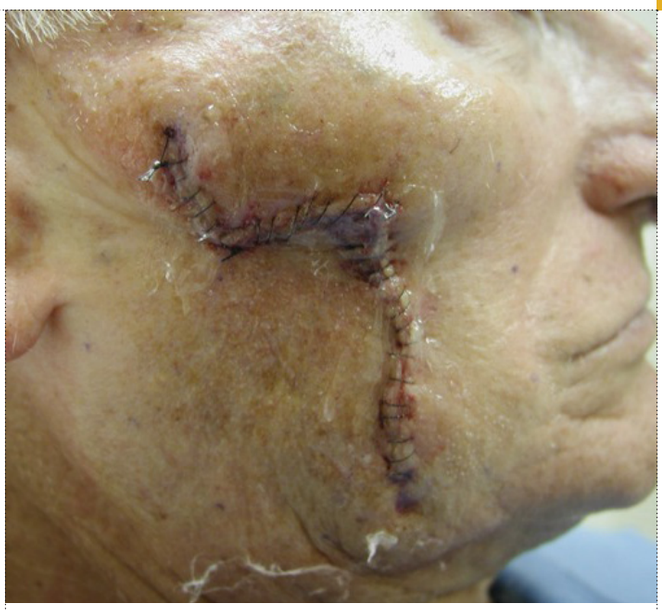
The flap needs to be monitored for a minimum of 72 hours. The commonest cause of flap failures is clotting at the venous anastomosis in which case the flap will become mottled or purple. Increasing lactate to glucose trend indicates anaerobic metabolism and ischaemia. Vascularised bone is very good in resisting infection and is one reason why it is successful in irradiated patients. Active bleeding is an indication for returning the patient to theatre to have the bleeding stopped to prevent pressure on the anastomosis.

Question 5. Answers: 3 and 4 are correct.

A tracheostomy is not always necessary and some clinicians choose not to use a tracheostomy in all patients but will leave the patient intubated on the ICU until the day after surgery and then assess the amount of postoperative swelling before extubating the patient. The Tracheostomy tube is there as a precaution against postoperative swelling blocking the airway. A temporary tracheostomy can block and will do so if not properly managed with regular suction. Ideally the tracheostomy tube should be cleared but if this is difficult a flexible endoscope should be introduced down the tube to discover the cause of the obstruction. In extremis then the tube should be removed but a new tube with a narrower bore should be available to replace it. Phoning for help can waste valuable time. There should always be somebody on the ward who is properly trained to manage an emergency.

References

1. The NHS Cancer plan: a plan for investment, a plan for reform **http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4009609.**
2. Scottish Intercollegiate Guidelines Network (SIGN) **www.sign.ac.uk.**
3. Marx RE, Stern D. Oral and maxillofacial pathology. A rationale for diagnosis and treatment. Illinois: Quintessence Publishing Co, Inc; 2003.
4. WHO histological classification of tumours of odontogenic tumours **http://screening.iarc.fr/atlasoralclassifwho2.php.**
5. B alaram P, Sridhar H, Rajkumar T, Vaccarella S, Herrero R, Nandakumar A, et al. Oral cancer in Southern India: The influence of smoking, drinking, paan-chewing and oral hygiene. Int J Cancer 2002;98(3):440-5.
6. De Stefani E, Boffetta P, Oreggia F, Mendilaharsu M, Deneo-Pellegrini H. Smoking patterns and cancer of the oral cavity and pharynx: a case-control study in Uruguay. Oral Oncol 1998;34(5):340-6.
7. Franceschi S, Levi F, La Vecchia C, Conti E, Dal Maso L, Barzan L, et al. Comparison of the effect of smoking and alcohol drinking between oral and pharyngeal cancer. Int J Cancer 1999;83(1):1-4.
8. Macfarlane GJ, Boyle P, Scully C. Oral cancer in Scotland: changing incidence and mortality. BMJ. 1992;305:1121-3.
9. Head and neck cancer—Part 1: Epidemiology, presentation, and prevention H Mehanna, V Paleri, C M L West, C Nutting BMJ 25 September 2010 Vol 341 10Balarmi P Sridhara H, Rajkumar T, Vaccarella S,Herrer R, Nandakumar A, Ravichandran K et al Oral cancer in Southern India: The influence of Smoking, Drinking, Paan-Chewing and Oral Hygiene `Int. J. Cancer: 98, 440–445 (2002).



11. Dahlstrom KR, Adler-Storthz K, Etzel CJ, Liu Z, Dillon L, El-Naggar AK, et al. Human papillomavirus type 16 infection and squamous cell carcinoma of the head and neck in never-smokers: A matched pair analysis. Clin Cancer Res 2003;9(7):2620-6.
12. Herrero R, Castellsague X, Pawlita M, Lissowska J, Kee F, Balaram P, et al. Human papillomavirus and oral cancer: the International Agency for Research on Cancer multicenter study. J Nat Cancer Inst 2003;95(23):1772-83.
13R. Lowy, Maura L. Gillison A New Link Between Fanconi Anemia and Human Papillomavirus–Associated Malignancies Douglas Journal of the National Cancer Institute, Vol. 95, No. 22, November, 2003.
14. Sobin LH, Wittekind C, editors. TNM Classification of Malignant Tumours. 6th Edition ed. Hoboken, NJ: Wiley; 2002. Altier.
15. Hidalgo DA. Fibula free flap mandibular reconstruction. Clin Plast Surg. Jan 1994;21(1):25-35.
16. Department of Dental Sciences. Screening for oral cancer. London: Royal College of Surgeons of England; 1994.
17. Dahlstrom KR, Adler-Storthz K, Etzel CJ, Liu Z, Dillon L, El-Naggar AK, et al. Human papillomavirus type 16 infectionand squamous cell carcinoma of the head and neck innever-smokers: A matched pair analysis. Clin Cancer Res 2003;9(7):2620-6.
18. Herrero R, Castellsague X, Pawlita M, Lissowska J, Kee F, Balaram P, et al. Human papillomavirus and oral cancer: the International Agency for Research on Cancer multicenter study. J Nat Cancer Inst 2003;95(23):1772-83.
19. Schurink CA, Tuynman H, Scholten P, et al; Percutaneous endoscopic gastrostomy: complications and suggestions to avoid them. Eur J Gastroenterol Hepatol. 2001 Jul;13(7):819-23. [abstract]

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FIBRO-OSSEOUS LESIONS OF THE JAWS AND CRANIAL BONES

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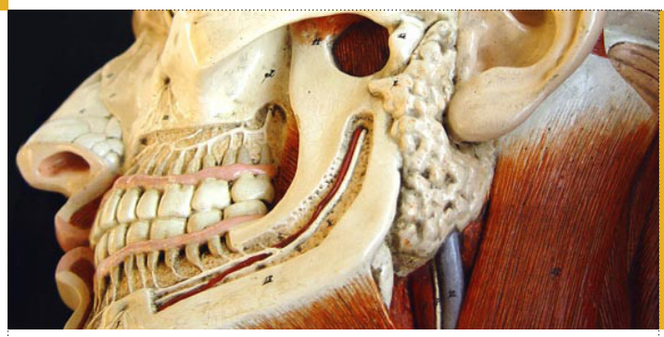

Fibro-osseous lesions of the jaws and cranial bones. Good Clinical Care.

Abstract

Fibro-osseous lesion is a broad term used to describe a group of conditions that are characterised by the replacement of normal bone by a tissue composed of collagen fibres, fibroblasts and varying amounts of mineralised substances. In spite of their similar histological features, they exhibit wide clinical behaviours and therefore each lesion requires a different approach to their management. Accurate diagnosis is therefore imperative. This is achieved through good history taking, thorough clinical examination, and appropriate imaging and in some cases histological examination. We describe here the clinical and radiological features of fibro-osseous lesions of the jaws and cranial bones and their management.

Introduction

Fibro-osseous lesion (FOL) of the jaws and cranial bones is a generic term used to describe a diverse group of conditions. These are characterized by the replacement of normal bone by a tissue composed of collagen fibres and fibroblasts and they contain varying amounts of mineralised substances, which may be bony or cementum-like in appearance. Most of the lesions contain a mixture of these calcifications [4, 11]. Their clinical presentations can vary from asymptomatic localised lesions, found incidentally on routine radiographic examination to large lesions that cause facial disfigurement and other functional disturbances depending on the location in the cranio-facial region. Although FOLs have similar histological features, they exhibit wide clinical behaviours which determine the management approach. These may range from clinical follow-up to surgery. Accurate diagnosis is therefore imperative. This is achieved through good history taking, thorough clinical examination, and appropriate imaging and in some cases histology examination [6, 11].

Classification of fibro-osseous lesions

FOL of the jaws and cranial bones are classified as:

1. Fibrous dysplasia
- *Monostotic*
- *Polyostotic*
- *Craniofacial*

2. Ossifying fibroma and Juvenile active ossifying fibroma

3. Cemento-osseous dysplasias
- *Periapical*
- *Focal*
- *Florid*

Fibrous dysplasia

Fibrous dysplasia (FD) is one of the most common fibro-osseous lesions of the jaws and cranial bones. Its aetiology is unknown but it is thought to be a developmental disorder of growing bones. It is characterized by a gradual replacement of normal bone by cellular fibrous connective tissue containing irregularly shaped trabeculae of immature woven bone. The irregularly arranged woven bone is often referred to as Chinese character trabeculae because of it's resemblance to Chinese script writing. FD may involve one or several bones. When it involves a single bone, it is referred to as monostotic fibrous dysplasia and when the disease involves more than one bone, it is referred to as polyostotic fibrous dysplasia. Monostotic fibrous dysplasia does not progress to the polystotic form of the disease. In the cranial bones, FD may involve several contiguous bones and such cases are referred to as cranio-facial FD. A variety of endocrine abnormalities have been associated with polyostotic FD. The most commonly associated endocrine abnormality is precocious puberty in girls. When polyostotic FD is accompanied by precocious puberty and café au lait skin pigmentation, it is referred to as McCune - Albright syndrome[8].

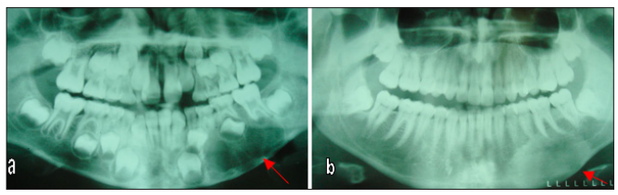

Fig 1: Orthopantomogram showing (a) early fibrous dysplasia of the body of the mandible as a multi-locular radiolucency and (b) a ground glass appearance at a latter stage in the same patient.

FIBRO-OSSEOUS LESIONS OF THE JAWS AND CRANIAL BONES

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Clinical features of FD

FD presents in early life, typically between late childhood and early adolescence. The majority of cases stabilise in early adulthood when skeletal maturity has been reached. Patients with polyostotic FD generally tend to present earlier than those with monostotic FD. The most common presenting complaint in the jaws and cranial region is a painless swell or facial asymmetry. The swelling grows very slowly over a long period of time, and patients and their family usually are unable to recall when the swelling first began. Depending on the bones involved in the cranio-facial region, patients may present with other symptoms. They may present with visual impairment when the sphenoid and ethmoid bones are involved and hearing loss when the temporal bones are affected. Patients may also present with displacement of the teeth when the jaw bones are involved[7].

Radiological features FD

The radiographic appearance of FD depends on the stage of the disease. Early lesions may appear radiolucent whereas late lesion may appear sclerotic, classically described as 'cotton-wool' sclerosis. However, the most common radiographic characteristic of FD is ground-glass or peau d'orange appearance of the bone on plain radiographs (fig 2). The involved region is ill-defined and tends to blend indiscernibly into the adjacent uninvolved bone[11]. These different radiographic features may be present in different parts of the same lesion. Computed tomography (CT) is useful in demonstrating the extent of the disease in the jaws and cranial bones especially when surgery is contemplated. CT images of FD on bone windows can display the range of opacities observed on plain radiographs (fig 3 and 4).

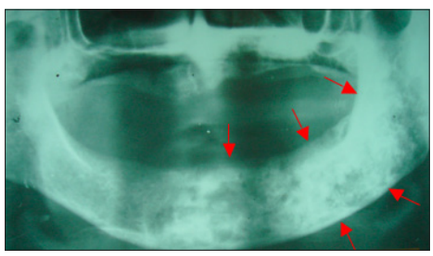

Fig 2: Orthopantomogram showing sclerotic fibrous dysplasia of the left body and ramus of mandible.

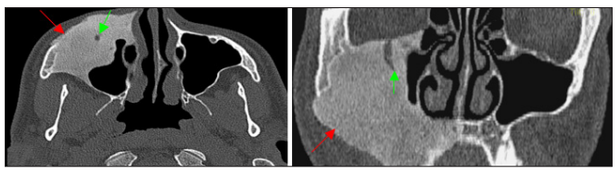

Fig 3: Axial and coronal CT scans showing monostotic fibrous dysplasia of right maxilla (red arrow). NB the right infra-orbital nerve (green arrow).

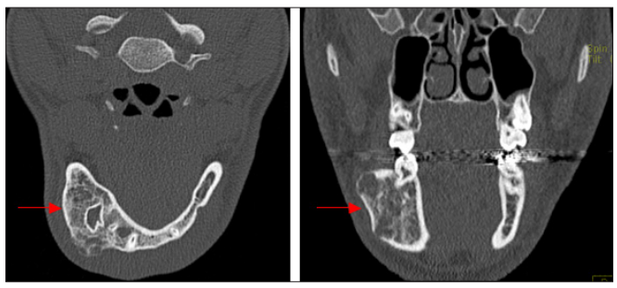

Fig 4: Axial and coronal CT scans, showing monostotic fibrous dysplasia of the right body of mandible.

Treatment of FD

Treatment of FD is tailored to each patient's symptoms. Asymptomatic patients with minimal bone involvement require no treatment and all that may be needed is routine follow-up. However, patients with severe disease causing facial deformity may require surgery to reduce the extent of the deformity. In such situations, surgery should be deferred until early adult life after the active growth phase. Surgery may also be required in patients with orbital cavity involvement to prevent blindness[6]. Malignant change, usually into osteosarcoma, rarely occurs in fibrous dysplasia. The osteosarcomatous change that has been reported has occurred in patients who have been previously treated with radiotherapy for their FD[9]. Radiotherapy is of no value in the treatment of fibrous dysplasia. There is some evidence to suggest that bisphosphonates may have some effect in relieving pain, stabilise or even decrease the size of the swelling in FD[3]. However, as the level of the evidence is not strong and also because of the possible causal relation between bisphosponates and osteonecrosis of the jaws[2] and the fact that FD is a self limiting condition, the use of bisphosphonates should perhaps be limited to cases where there is significant pain and where there is risk to vital structures such as the optic nerve.

Ossifying Fibroma

Ossifying fibroma (cemento-ossifying fibroma) is a benign slowly growing, predominantly fibrous lesion with varying amount of woven and lamellar bone trabeculae and cementum-like tissue[5]. Plump osteoblasts may rim the trabeculae[7]. Lamellar bone formation and osteoblastic rimming found in OF and the presence of a capsule differentiates it from FD.

Clinical features OF

OF presents as a slowly growing, localized swelling predominantly in the jaw bones. Rarely they may reach a very large size and result in considerable deformity. They present usually in the second to fourth decade of life with women more frequently affected than men. The most commonly involved bone appears to be the mandible with a predilection to the premolar and molar region, in close proximity to the roots of the teeth[8].

FIBRO-OSSEOUS LESIONS OF THE JAWS AND CRANIAL BONES

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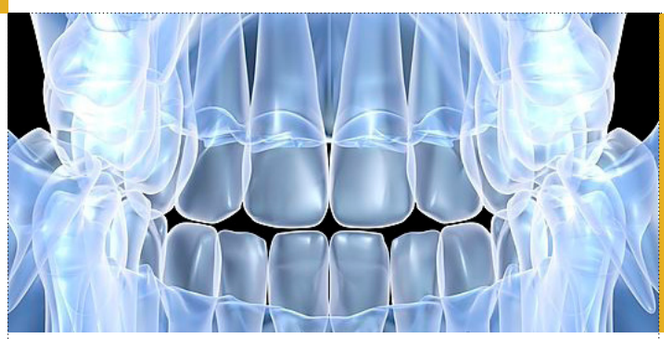

Fibro-osseous lesions of the jaws and cranial bones. Good Clinical Care.

Radiographic features of OF

In its early stage, OF appears on a plain radiograph as a well-defined round or oval radiolucent lesion. At a later stage of maturation, it may appear radio-opaque with a uniform radiolucent rim. As the lesion grows, it may cause displacement and destruction of adjacent structures. In the jaws, adjacent teeth are displaced away from the lesion but root resorption is not common. Large mandibular lesions may cause a characteristic thinning and downward bowing of the inferior border. On a CT scan OF exhibits a thin but intact cortex.

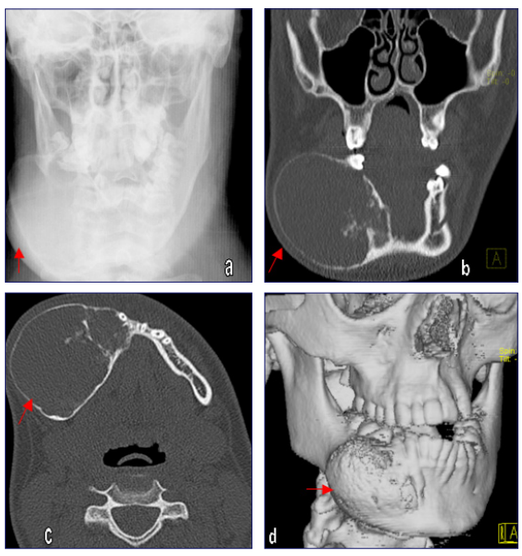

Fig 5: Images of a patient with juvenile active ossifying fibroma of the right body of mandible. (a) posterio-anterior plain radiograph, (b) coronal CT, (c) axial CT and (d) 3-D reconstructed CT views.

Treatment of OF

OF is treated by simple enucleation of the lesion. The presence of a capsule allows surgical removal with relative ease. Recurrence after complete enucleation is unusual.

Juvenile Active Ossifying Fibroma

Juvenile active ossifying fibroma (JAOF) is a rapidly growing destructive fibrous-osseous lesion. It consists of a cell-rich fibrous stroma, containing bands of cellular osteoid without osteoblastic lining, together with trabeculae of more typical woven bone.

Clinical features of JAOF

JAOF typically present as a rapidly growing swelling or mass. It occurs in children and adolescence, usually under the age of 15 years with the maxilla being the most common site affected. Symptoms are related to the site of involvement and include epistaxis, proptosis, exophthalmos, nasal obstruction, headaches and diplopia. Pain is only rarely described.

Radiological features of JAOF

Radiographically JAOF usually appear as a well-defined unilocular or multilocular radiolucent lesion that may contain fine spots of radiopacity (fig 5). Some lesions may show a ground-glass appearance[1].

Treatment OF JAOF

These tumors have a tendency for local recurrence. However, radical surgery does not appear to be appropriate because recurrences may be managed by local excision. Sarcomatous transformation has not been reported[1]. Lesions showing aggressive behaviour however, may require a more extensive surgical resection.

Cemento-osseous Dysplasia

These are a group of FOLs with similar histological features separated only by their clinical presentation and radiographic features. As a group, they are all more common in middle-aged black women[10]. They are almost exclusively related to the alveolar process of the jaws. They appear to develop in close relationship with the roots of teeth or in the alveolar process of edentulous parts of the jaws[11]. When they occur in the mandible, they are found above the inferior dental canal. The histology consists of cellular fibrous tissue with varying amounts of bone and cementum-like calcifications. The proportions of these structures depend on the age of the lesion. The lesions included in cemento-ossifying dysplasias are periapical cemento-ossous dysplasia, focal cemento-osseous dysplasia and florid cemento-osseous dysplasia.

Periapical Cemento-osseous Dysplasia

PCODs are asymptomatic lesions and are usually detected incidentally during routine radiographic examinations. PCOD typically involve the bone associated with the teeth in the anterior part of the mandible. The associated teeth are usually found to be vital.

The radiographic features depend on the stage of the lesion. Early lesions appear as ill-defined or well-defined radiolucencies associated with the apices of the teeth involved.

FIBRO-OSSEOUS LESIONS OF THE JAWS AND CRANIAL BONES

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Early lesions may mimic a periapical inflammatory process. This early lesion may remain unchanged or progress to an intermediate stage of mixed radio-density. In the final stage, the lesion appears sclerotic, often completely calcified with a radiolucent rim[1, 11]. The lesions are usually small and rarely exceed 1.5cm in diameter. PCOD can be diagnosed on the clinical and radiological features alone and no surgical treatment is needed.

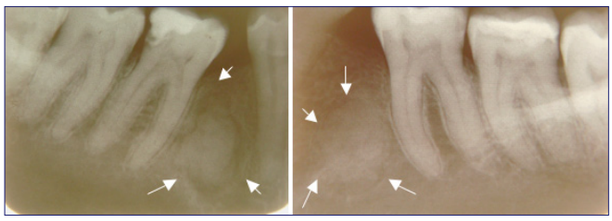

Fig 6: Peri-apical radiographs depicting focal cemento-osseous dysplasia in the missing second premolar regions bilaterally.

Focal cemento-osseous dysplasia

Focal cemento-osseous dysplasias occur in the alveolar process of the jaws. Many seem to occur in the endentulous areas of previously extracted teeth. However, they may occur in dentate areas of the jaws, associated with the periapical areas of the teeth. They are usually asymptomatic and are found on routine radiographic examination. They appear radiographically as either a well defined radiolucency or of mixed radio-density depending on the age of the lesion. They may or may not have a sclerotic border and they rarely exceed 2cm in diameter (fig 6). Once a diagnosis is made, they require no treatment. The lesions normally persist without any significant change.

Florid cemento-osseous dysplasia

The clinical presentation of florid cemento-osseous dysplasia depends on the size and extent of the lesion. Small lesions may be asymptomatic whiles larger ones may present as a sclerotic masses which can cause expansion of the affected jaw. The lesions usually involve more than one quadrant of the jaws. However the most common presentation is bilateral densely sclerotic lesion in the molar regions of the edentulous mandible. The lesions may become symptomatic if the sclerotic masses become exposed to the oral environment. This may occur as a result of extraction, biopsy of the lesion or progressive atrophy of the alveolar bone. This may result in infection and pain. Lesions that are asymptomatic do not require any treatment. However, patients need close monitoring and good dental care to prevent the need for dental extraction and its associated complications. Lesions that become exposed and infected require treatment with antibiotics. The exposed sclerotic masses usually sequestrate slowly which is followed by healing. Partial surgical removal of large sclerotic masses is not usually helpful, and total removal requires fairly extensive surgery and should be avoided.

Acknowledgement

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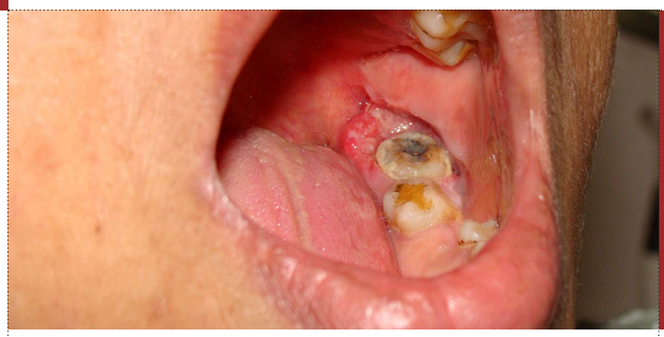
Reference

1. Brannon R B, Fowler C B. Benign fibro-osseous lesions: a review of current concepts. Adv Anat Pathol 2001; 8:126-43.
2. Hess LM, Jeter JM, Benham-Hutchins M, Alberts DS. Factors associated with osteonecrosis of the jaw among bisphosphonate users. Am J Med 2008; 121: 475-483.
3. Kos M, Luczak K, Godzinski J, Klempous, J. Treatment of monostotic fibrous dysplasia with pamidronate. J Craniomaxillofac Surg 2004; 32: 10–15.
4. Koury ME, Regezi JA, Perrott DH, Kaban LB. "Atypical" fibro-osseous lesions: diagnostic challenges and treatment concepts. Int J Oral Maxillofac Surg 1995; 24: 162–169.
5. Kramer IR, Pindborg JJ, Shear M. The World Health Organization histological typing of odontogenic tumours. Introducing the second edition. Eur J Cancer B Oral Oncol 1993; 29B: 169 – 71.
6. MacDonald-Jankowski DS. Fibro-osseous lesions of the face and jaws. Clin Radiol 2004; 59: 11-25.
7. Margo CE, Ragsdale BD, Perman KI, Zimmerman LE, Sweet DE. Psammomatoid (juvenile) ossifying fibroma of the orbit. Ophthalmology 1985; 92: 150-159.
8. Mohammadi-Araghi H, Haery C. Fibro-osseous lesions of craniofacial bones. The role of imaging. Radiol Clin North Am 1993; 31: 121-34.
9. Slow IN, Friedman EW. Osteogenic sarcoma arising in a pre-existing fibrous dysplasia: a case report. J Oral Surg 1971; 29: 126 – 129.
10.Waldron C A. Fibro-osseous lesions of the jaws. J Oral Maxillofac Surg 1993; 51: 828-35.
11.Waldron CA. Fibro-osseous lesions of the jaws. J Oral Maxillofac Surg 1985; 43: 249-62.

ORAL CANCERS – EPIDEMIOLOGY AND MANAGEMENT

M Alibhai, R Bhandari & L Cheng



Oral cancers – epidemiology and management. Patient Management.

Abstract

Oral cavity and oro-pharyngeal squamous cell carcinoma (SCC) accounts for 2% of all cancers in the UK and 90% of all oral cancers, affecting 8 per 100,000 populations.[1,2] Although the number of newly diagnosed oral cancers has shown an increase of 30%, the overall 5-year survival rates has improved over the last 2 decades.[1,2]

Traditional treatment of oral SCC involves surgery and radiotherapy and/or chemotherapy, depending on tumour staging. The management of patients with oral cavity and oro-pharyngeal SCC is multidisciplinary, led by Oral and Maxillofacial Surgeons.

This article is by no means an exhaustive text on oral SCC but aims to provide an overview of a topic that is often not covered in an undergraduate medical syllabus.

Case review

A 65-year old man presented with a 2 month history of a painless ulcer on the left lateral border of his tongue (Figure 1). He is unsure as to whether he has bitten his tongue. He has used topical preparations without any effect. He has a past medical history of hypertension and ischaemic heart disease. He lives alone. He is a retired taxi driver with a 40 pack-year smoking history. He drinks 30 units of alcohol per week.

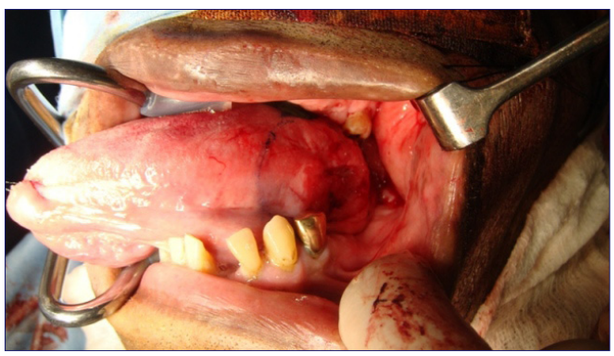


Figure 1: Left lateral tongue squamous cell carcinoma.

Clinical examination revealed a 3cm diameter ulcer on the right lateral tongue. The ulcer was indurated with rolled margins. There was a neglected dentition. There was no palpable cervical lymphadenopathy.

Incisional biopsy revealed poorly differentiated squamous cell carcinoma. Radiological assessment consisted of a CT thorax and a MRI head and neck. His case was discussed with the Head and Neck Multidisciplinary Team (MDT). He was staged as T4N0M0 (see later for TNM staging) as MRI showed invasion of the hyoglossus muscle.

Following informed consent, he underwent a surgical tracheostomy, resection of his tongue SCC with a wide surgical margin through a lip split and mandibulotomy access procedure, selective neck dissection and multiple dental extractions for unrestorable teeth. The intraoral soft tissue defect was reconstructed with a free tissue transfer from his forearm using microvascular anastomoses and his forearm defect was repaired with an abdominal full thickness skin graft.

Pathological specimens showed no evidence of metastatic spread to the regional lymph nodes but poor prognostic features within the tumour and a close deep surgical margin. He therefore received adjuvant radiotherapy to the primary site.

Anatomy of the oral cavity

The oral cavity forms a complex region with multiple anatomical regions (Figure 2). Tumours can present in any anatomical region, however the gutter areas comprising the floor of mouth, the retromolar trigone and the ventral and lateral surfaces of the tongue are the high-risk sites (Figure 3). The mucosa of the oral cavity is stratified squamous epithelium.

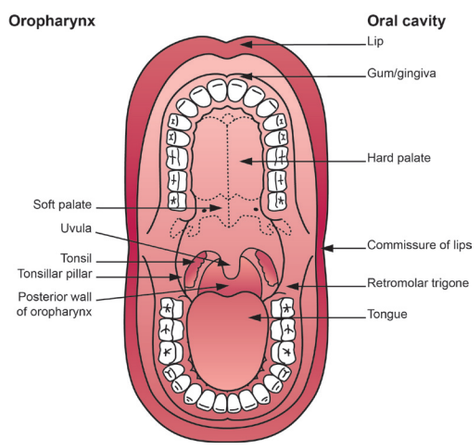


Figure 2: Anatomy of the oral cavity and oropharynx.

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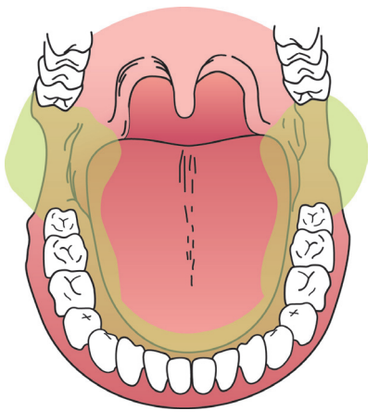


Figure 3: Shaded U-shaped high risk areas for oral carcinoma.

Epidemiology – key points

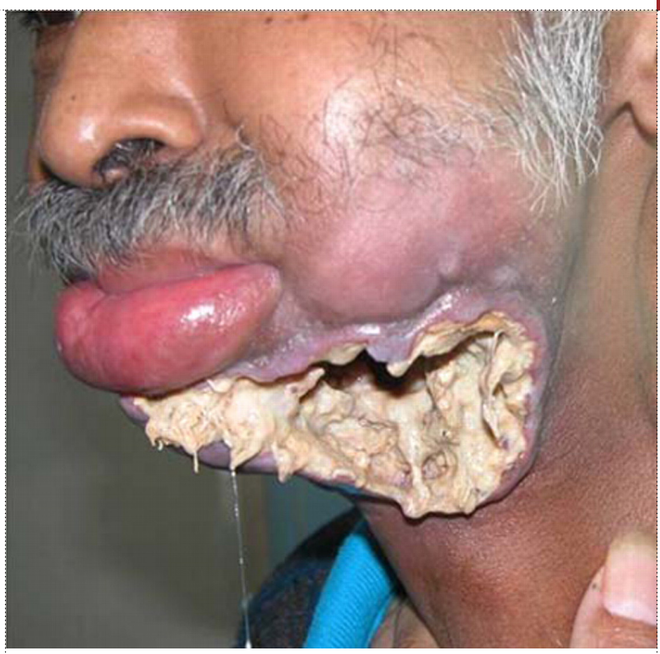
- The 10th most common cancer and the 7th most common cause of cancer-related mortality worldwide.[2]
- There is a varying incidence around the world. Less than 4% of all cancers in the Western world but nearly 40% of all cancers in the Indian sub-continent and parts of Asia.[2]
- In England, oral cancer has increased by more than 30% over the last 2 decades.[3]
- An age-related disease with a sharp linear rise with age. 85% of oral cancer occurs in patients over the age of 50 years.[2]
- An increasing trend in the Western world especially among those under 40 years.[2]
- Male to female ratio is about 3:2 (except lip SCC at 8:1 ratio).[2]
- Overall 5-year survival has improved to 56% in the last 20 years.[3]
- Death to registration ratio at 30-40%.[3]

Aetiology

i. Tobacco
ii. Alcohol
iii. Betel nut
iv. Diet and nutrition
v. Sun exposure
vi. Precancerous oral lesions and conditions
vii. Human papillomavirus
viii. Immunosuppression

- Tobacco use in all forms is the single most important risk factor in oral cancer. Smokers have a three fold increased risk of developing oral SCC compared with non-smokers with the risk being both dose and duration dependent.[4] It may take 20 years or more from cessation for the risk to revert back to that of non-smokers.[5]

- There is a high incidence of oral cancer in South Asia which transcends into migrant populations from this area. The primary reason for this is the chewing of betel quid or paan. The chief ingredients are the leaf of the vine, Piper betel, areca nut, slaked lime (calcium hydroxide) and spices. Areca nut is carcinogenic and the risk is increased when tobacco is added to the quid.[5,6]



- Epidemiological studies have shown that the oral cancer risk is increased with alcohol consumption.[7] Carcinogenic contaminants and by-products in alcoholic beverages enhance the mucosal penetration of carcinogens. Nutritional deficiencies and impaired metabolism among alcoholics affect the barrier function of oral mucosa. Hepatic dysfunction in detoxifying potential carcinogens and a suppressed immune system among chronic alcoholics can increase the risk of oral cancer. Studies have also shown a synergistic effect of alcohol and tobacco in the risks of oral cancer.[8]

- There has been concern about the use of mouthwashes with high alcoholic content, but the majority of studies show no increase in risk.[8]

- Poor diet is a risk factor for head and neck cancer. Conversely, people with a good Mediterranean diet have less than half the risk of developing oral/pharyngeal cancer.[9]

- Studies have shown a risk reduction of between 26% - 50% with each piece of additional fruit and/or vegetables.[10]

- Exposure to UV radiation is a risk factor for cancer of the lip. Lip cancer is three times more common in men than women which may be an effect of occupation, smoking and sun-exposure.[11]

- The most common precancerous oral lesions are leukoplakia and erythroplakia. Other conditions include lichen planus, oral submucous fibrosis, oral syphillis and Plummer Vinson syndrome. Leukoplakia has many clinical variants but is much less likely to progress to malignancy than erythroplakia and has a transformation rate between 3-20%.[12]

ORAL CANCERS – EPIDEMIOLOGY AND MANAGEMENT

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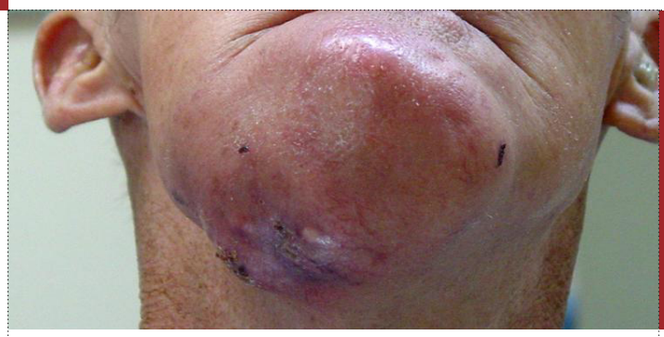

Oral cancers – epidemiology and management. Patient Management.

· Epidemiological studies in the developed world have shown a rapid increase in HPV related oro-pharyngeal cancer especially HPV subtype[16]. Sexual behaviour including the number of life time sexual and oral sexual partners is associated with the risk of developing HPV related oral cancer.[13]

· An increased risk of oral cancer has been shown in individuals with HIV/AIDS or people who have undergone organ transplants, supporting a role of immunosuppression.[14]

Clinical Presentation

Oral cancer can present in many ways depending on the site of the tumour. The most frequent site of oral cancer is the lower lip followed by the tongue. Within the mouth, the majority of oral cancers concentrate on the U-shaped area from the oropharynx and retromolar regions, lateral borders of the tongue and lingual aspect of the alveolar margins to the floor of mouth. This is where saliva and carcinogens pool before swallowing.

Oral Cancer – clinical features

· Early oral SCC are usually asymptomatic and painless

· Common presentations of early cancer – white or red patches, shallow ulcers, small exophytic growth, area of erythroplakia and speckled leukoplakia

· Local invasion leads to:
Tissue fixation - binding tissue planes together
Tissue induration – rubbery hardness, lack of mucosal elasticity
Tissue destruction – underlying bony destruction, numbness of the lip
Tissue distortion – seen with fixation and induration
Tissue dysfunction – reduced tongue mobility affecting speech and swallowing

· Common presentations of late cancer include ulcers with prominent rolled edges, induration, pain, bleeding due to mild trauma, lip numbness, difficulty in speech and swallowing, and associated constitutional symptoms.

· Metastatic spread to regional cervical lymph nodes

Management

In the UK, the management of all Head and Neck cancers falls within the remit of a MDT in a tertiary referral centre.

The stages of diagnosis and management of oral cancer follows that of head and neck cancer:

i) History and examination
ii) Special investigations, including biopsy
iii) Diagnosis and MDT discussion
iv) Definitive treatment
v) Rehabilitation
vi) Follow up/surveillance

· The history from oral cancer patients follows the standard format of questions during a medical clerking. Specific information noted is the onset symptoms and progression of the oral cancer. Swallowing, dietary intake, speech and airway are worth noting for future management. Alcohol and smoking history help to stratify risk.

· Apart from examination of the oral cavity, it is essential to exclude second primary tumours in the aero-digestive tract by palpation of the neck and face followed by examination of the nasal and pharyngeal cavity with a flexible nasendoscope.

· If a suspicious lesion is identified an incisional biopsy is performed.

· Radiological assessment of oral cancer is vital in diagnosis, localising and analysing tumour spread, staging, treatment planning and surveillance. The evolving radiological procedures have changed from plain radiographs to cross sectional imaging and functional imaging with the evaluation of tissue metabolism. This greatly enhances the planning of accurate surgical resection margins, reconstructive options and postoperative surveillance

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The imaging modalities available in tumour diagnosis and treatment are:

i. Plain radiographs
ii. Computerised Tomography (CT)
iii. Magnetic resonance imaging (MRI)
iv. Ultra-sound (US)
v. Positron emission tomography (PET)
vi. Nuclear/ Radionuclide imaging

- Orthopantomogram is a conventional jaw radiograph widely available for the initial investigation of gross dental caries, bone invasion and pathological fracture.
- With regards to staging of oral cancers, plain radiographs have been superseded by CT and MRI.
- Thin CT slices (< 1mm) can produce accurate cross sectional imaging and allow reformatting to create 3-dimensional images. It is essential for detecting bony invasion and staging the chest because 10% of oral SCC have distant metastasis and 5% have a synchronistic bronchial carcinoma.[15]
- MRI provides excellent soft tissue imaging of oral cancers and their advancing edges. There is no ionising radiation involved but some patients are unable to tolerate lying in the machine due to claustrophobia.
- US is an essential diagnostic tool in head and neck staging and it allows pathological lymph nodes to be biopsied using fine needle aspiration or fine tru-cut needles.[16]
- Information of the number, level and size of involved cervical lymph nodes are vital for staging of oral cancers.
- PET is a radiological investigation which maps the biological active tissues (for instance oral cancer) with an intravenous biological contrast. This modality is particularly useful in detecting occult primary tumours with metastatic cervical lymph nodes and recurrent cancers in the head and neck.[17]
- Radionuclide imaging allows tracers to be used with a gamma camera to allow it to show the areas of high biological activity. This is largely superseded by other forms of cross-sectional imaging.

The staging of head and neck cancers (Table 1) describes the anatomical extent of disease based on an assessment of the extent of the primary tumour, the absence or presence and extent of regional lymph node metastasis and the absence or presence of distant metastasis.

Stage	
TX	Primary tumour cannot be assessed
T0	No evidence of primary tumour
Tis	Carcinoma-in-situ
	ORAL CAVITY
T1	2cm or < 2cm
T2	> 2cm to 4 cm
T3	> 4cm
T4a	Through cortical bone, deep/extrinsic muscle of tongue, maxillary sinus, skin
T4b	Masticator space, pterygoid plates, skull base, internal carotid artery
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph nodes metastasis
N1	Ipsilateral single node 3cm or < 3cm
N2	a. Ipsilateral single node > 3cm to 6 cm b. Ipsilateral multiple nodes 6 cm or < 6 cm c. Bilateral, contralateral nodes 6 cm or < 6cm
N3	> 6cm
MX	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Table 1: TNM staging of oral cancers.

The neck is divided into six levels, I-VI (Table 2, Figure 4). Metastasis from the oral cavity to the neck nodes up stages the disease process and has a direct correlation with a poorer prognosis.

Level	Name	Boundaries and landmarks
IA IB	Submental nodes Submandibular nodes	Anterior belly of digastrics muscles anteriorly, hyoid bone inferiorly, and body of the mandible superiorly -
II	Jugulo-digastric nodes	From the level of the hyoid bone inferiorly to the skull base superiorly
III	Mid cervical nodes	From the hyoid bone superiorly to the cricothyroid membrane inferiorly
IV	Jugulo-omohyoid nodes	From the cricothyroid membrane superiorly to the clavicle inferiorly
V	Posterior triangle nodes	Anterior border of the trapezius posteriorly,m the posterior border of the sternomastoid muscle anteriorly, and the clavicle inferiorly
VI	Anterior compartment nodes	From the hyoid bone superiorly to the suprasternal notch inferiorly. The lateral borders are formed by the medial border of the carotid sheath

Table 2: Cervical lymph nodes and boundaries.

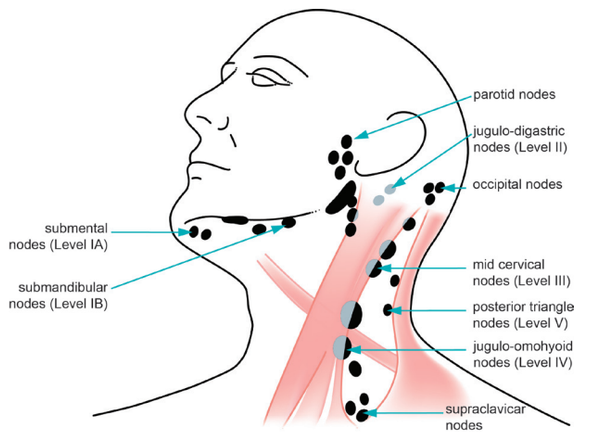

Figure 4: Cervical lymph nodes and levels.

Metastasis away from the primary site is rarely picked up on clinical examination and carries the gravest prognostic implications. The thorax is the most common site for metastasis from the oral cavity.

Treatment of oral cavity tumours can be complex. Clinical, radiological and pathological staging are discussed in multidisciplinary team meetings when patients' co-morbidities and wishes are considered during treatment planning. The treatment modalities for oral cancer are:

i) Palliative
ii) Surgical resection with/without reconstruction
iii) Chemotherapy/Radiotherapy
iv) Photodynamic therapy (PDT)

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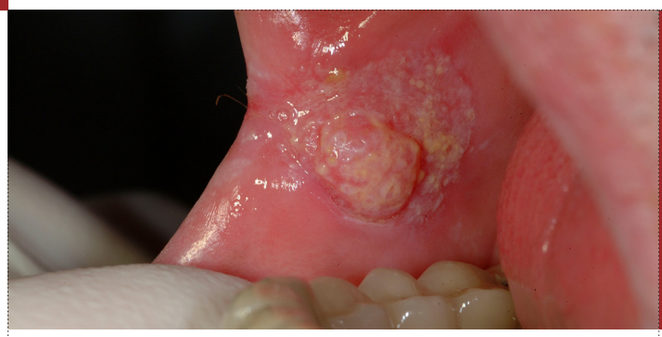

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- Palliative treatment is guided towards symptom control in both hospital or community settings to preserve some levels of quality of life.
- Surgical resection of the primary site is a mainstay of treatment. Depending on tumour size, location and nodal status, a neck dissection to remove cervical lymph nodes may be performed. For instance in the opening case report, although the patient has a clinically negative neck, he is offered a neck dissection because there is a risk of greater than 20% of occult nodal metastasis.
- Reconstruction of a large oral defect will require local, pedicled or microvascular free tissue transfer.
- Chemotherapy and radiotherapy can be given as primary treatment or used in conjunction with surgery. For advanced cancers, they can be provided as a palliative measure. Radiotherapy can also be delivered as an out-patient treatment. Recent advances in radiotherapy include Intensity Modulated Radiation Therapy (IMRT), Gamma knife radiosurgery and Cyberknife stereotactic radiotherapy for intracranial metastasis.
- PDT involves giving a light sensitive drug which targets rapidly growing tumour cells. The tumour is then exposed to intense light either delivered on the tumour surface or within the tumour. PDT can reduce symptoms as a palliative treatment and it is only provided by certain cancer centres.

In patients with head and neck cancer, 76% of recurrences occur within the first two years post-treatment, and 11% occur in the third year. Therefore patients should be seen frequently in the first 3 years.[18]

Treatment has a significant impact on quality of life. Patients may need psychological and financial support, support with diet and nutrition, speech and swallowing and with regular mouth care.

Conclusion

Oral cancer is a relatively uncommon disease but it can develop in the areas of the oral cavity which are difficult to examine. As early diagnosis is the most important factor in improving prognosis, clinicians' high level of suspicion and vigilance are vital when oral lesions persist without an identifiable cause or after the elimination of putative factors.

Cancer of the oral cavity is often difficult to treat. The treatment is often complicated due to the complex anatomy and varied functions. Patients with oral cancer tend to have a relatively high mortality rate caused by multiple risk factors including heavy smoking, drinking, and malnutrition.

In the UK, oral cancers are managed in specialised centres staffed by multi-disciplinary teams to provide a streamline management plan to optimise survival and minimise the side effects of unpleasant treatment.

Early detection and management, oral health campaigns and general health education are all essential in reducing the impact of oral cancer.

Questions

1. The most common sites for oral cancer are:

a) Palate
b) Lateral tongue
c) Floor of mouth
d) Upper lip
e) Retromolar regions

2. Please select the false statement regarding positron emission tomography (PET)

a) It is used to detect metastatic disease
b) It is helpful in detecting occult primary tumour
c) It is widely available with low radiation
d) It requires injection of a biological contrast
e) It can detect recurrent cancer in the head and neck

3. Please select the false statement regarding oral submucous fibrosis

a) It is caused by betel quid (paan) chewing
b) Treatment is total surgical excision
c) It is found among paan chewers
d) It can affect young children
e) It is a precancerous lesion of the cheek

4. Treatment of oral cancers does not include:

a) Surgical resection and reconstruction
b) Chemotherapy
c) Photodynamic therapy
d) Immunotherapy
e) Radiotherapy

ORAL CANCERS – EPIDEMIOLOGY AND MANAGEMENT

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5. Which is an aetiological factor for oral cancer?

a) Lack of vitamin K
b) Magnesium deficiency
c) Lack of vitamin D
d) Reverse smoking
e) Alcohol abstinence

Answers

1. Answer: b,c and e

Carcinogens from tobacco, alcohol or betel quid can maintain contact with oral mucosa where saliva tends to pool on the U-shaped area within the mouth before swallowing. This risky area starts from the oropharynx and retromolar regions, lateral borders of the tongue and lingual aspect of the alveolar margins to the floor of the mouth.

2. Answer: c

It is not widely available because cyclotrons are used to produce ionising radiation for PET scanning. It is expensive and the on-site facilities to produce this radiopharmaceutical are only available in major hospitals. A PET scan is often read with either MRI or CT scan in order to provide both detailed anatomical and metabolic information.

3. Answer: b

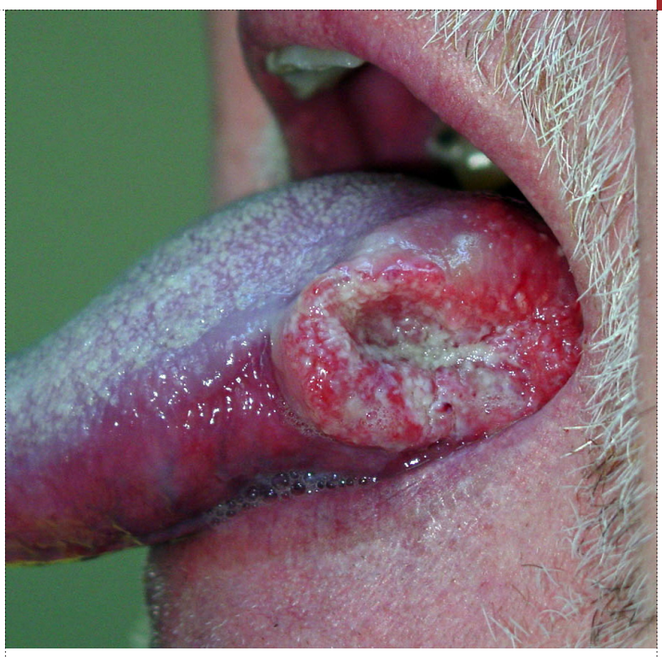
Betel quid (paan) chewing produces 2 adverse effects, namely oral cancer and submucous fibrosis. Oral submucous fibrosis undergoes malignant transformation in 4-8% of cases making it a significant contribution to the high incidence of oral cancer in the Indian subcontinent and in Asian immigrant populations. Treatment is largely ineffective because fibrosis affects not only the buccal mucosa, soft palate and lips, but also muscles of mastication. Surgical treatment may create more scarring which can lead to worsening trismus.

4. Answer: d

Immunotherapy uses the patient's immune system to reject cancer cells by providing antibodies or by immunising the patients with cancer vaccine. At present the main treatment modalities for oral cancers are surgery with or without reconstruction followed by adjuvant radiotherapy and chemotherapy. PDT is not widely available

5. Answer: d

It is well established that there is an aetiological relationship between oral cancer and tobacco smoking regardless of the type and method of tobacco consumption. The reverse smoking habit, which is smoking with the burning end inside the mouth, is popular in some parts of India. This smoking habit is associated with cancer of the palate which is one of the rare locations for oral cancer.



References

1. Moore SR, Johnson NW, Pierce AM, Wilson DF. The epidemiology of mouth cancer: a review of global incidence. Oral Dis 2000; 6(2):65-74.
2. Warnakulasuriya S. Global epidemiology of oral and oropharyngeal cancer. Oral Oncol 2009; 45(4-5): 309-16.
3. National Cancer Intelligence Unit. (January 2010) Profiles of head and neck cancers in England: incidence, mortality and survival.
4. Warnakulasuriya, S., G. Sutherland, and C. Scully. Tobacco, oral cancer, and treatment of dependence. Oral Oncol 2005; 41(3): 244-60
5. Bosetti C, Gallus S, Peto R, Negri E, Talamini R, Tavani A, Franceschi S, La Vecchia C. Tobacco smoking, smoking cessation, and cumulative risk of upper aerodigestive tract cancers. Am J Epidemiol 2008; 167(4):468-73.
6. Warnakulasuriya S, Trivedy C, Peters TJ. Areca nut use: an independent risk factor for oral cancer. BMJ 2002; 324(7341):799-800.
7. Bagnardi V, Blangiardo M, La Vecchia C, Corrao G. A meta-analysis of alcohol drinking and cancer risk. Br J Cancer 2001; 85(11):1700-5.
8. Castellsague X, Quintana MJ, Martinez MC, Nieto A, Sanchez MJ, Juan A, Monner A, Carrera M, Agudo A, Quer M, Munoz N, Herrero R, Franceschi S, Bosch FX. The role of type of tobacco and type of alcoholic beverage in oral carcinogenesis. Int J Cancer 2004; 108(5): 741-749.
9. Bosetti C, La Vecchia C, Talamin R, Negri E, Ievi F, Fryzek J, McLaughlin JK, Garavello W, Franceschi S. Energy, macronutrients and laryngeal cancer risk. Ann Oncol 2003; 14(6):907-12.
10. Freedman ND, Park Y, Subar AF, Hollenbeck AR, Leitzmann MF, Schatzkin A, Abnet CC. Fruit and vegetable intake and head and neck cancer risk in a large United States prospective cohort study. Int J Cancer 2008; 122(10):2330-6.
11. Perea-Milla Lopez E, Minarro-Del Moral RM, Martinez-Garcia C, Zanetti R, Rosso S, Serrano S, Aneiors JF, Jimenez-Puente A, Redondo M. Lifestyles, environmental and phenotypic factors associated with lip cancer: a case-control study in southern Spain. Br J Cancer 2003; 88(11):1702-7.

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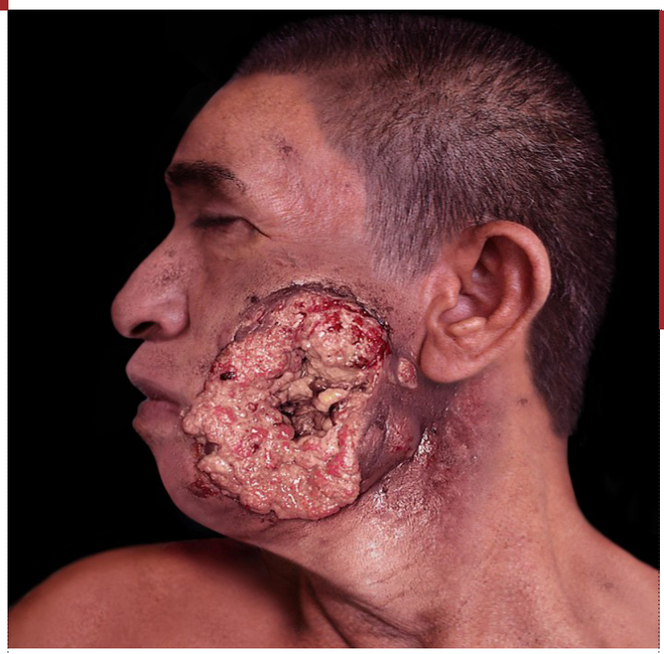

12. Ho PS, Chen PL, Warnakulasuriya S, Shieh TY, Chen YK, Huang IY. Malignant transformation of oral potentially malignant disorders in males: a retrospective cohort study. BMC Cancer 2009; 9:260.
13. Herrero R, Castellsague X, Pawlita M, Lissowska J, Kee F, Balaram P, Rajkumar T, Sridhar H, Rose B, Pintos J, Fernandez L, Idris A, Sanchez MJ, Nieto A, Talamini R, Tavani A, Bosch FX, Reidel U, Snijders PJ, Meijer CJ, Viscidi R, Munoz N, Franceschi S; IARC Multicenter Oral Cancer Study Group. Human papillomavirus and oral cancer: the International Agency for Research on Cancer multicenter study. J Natl Cancer Inst 2003; 95(23): 1772-83.
14. Grulich AE, van Leeuwen MT, Falster MO, Vajdic CM. Incidence of cancers in people with HIV/AIDS compared with immunosuppressed transplant recipients: a meta-analysis. Lancet 2007; 370(9581):59-67.
15. Haughey BH, Gates GA, Arfken CL, Harvey J. Meta-analysis of second malignant tumors in head and neck cancer: the case for an endoscopic screening protocol. Ann Otol Rhinol Laryngol 1992;101(2 pt 1):105-12.
16. Castelijns JA, van den Brekel MW. Imaging of lymphadenopathy in the neck. Eur Radiol 2002; 12(4): 727-38.
17. Ha PK, Hdeib A, Goldenberg D, Jacene H, Patel P, Koch W, Califano J, Cummings CW, Flint PW, Wahl R, Tufano RP. The role of positron emission tomography and computed tomography fusion in the management of early-stage and advanced-stage primary head and neck squamous cell carcinoma. Arch Otolaryngol Head Neck Surg. 2006 Jan;132(1):12-6.
18. Boysen M, Lovdal O, Tausjo J, Winther F. The value of follow-up in patients treated for squamous cell carcinoma of the head and neck. Eur J Cancer 1992; 28(2-3):426-30.

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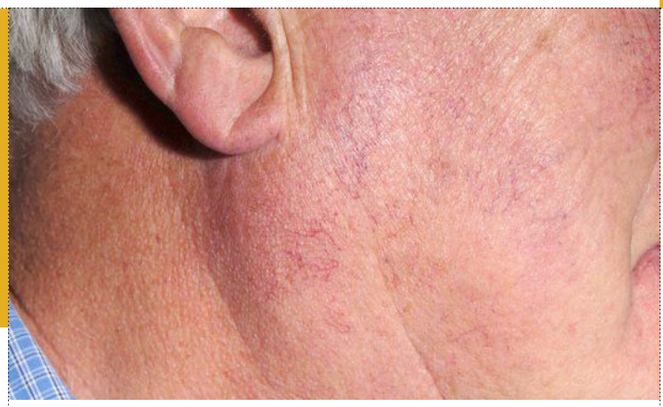
MANAGEMENT OF SALIVARY GLAND SWELLINGS

R Skinner, S Madhavarajan & L Cheng

Management of salivary gland swellings. Good Clinical Care.



Abstract

Salivary gland swellings are common but salivary neoplasms are rare and reported incidences vary from 0.4 to 13.5 per 100,000. The overall incidence for all salivary gland tumours in the UK is approximately 8 per 100,000 population (Nottingham population study). Overall, 10% of all salivary gland tumours are malignant. This case based discussion focuses on a patient referred to the Oral and Maxillofacial Surgery department with a lump below the right ear.

Salivary Gland Swellings: Overview

The salivary glands can be divided into two groups: the major and minor salivary glands. The major salivary glands (Figure 1) are the paired parotid, submandibular and sublingual glands. There are around 800-1000 minor salivary glands which are located in the labial, buccal, palatine, tonsillar, retromolar and lingual areas of the oral cavity. Salivary gland swellings are common causes for facial and intra-oral swellings. These can be divided into inflammatory and neoplastic conditions.

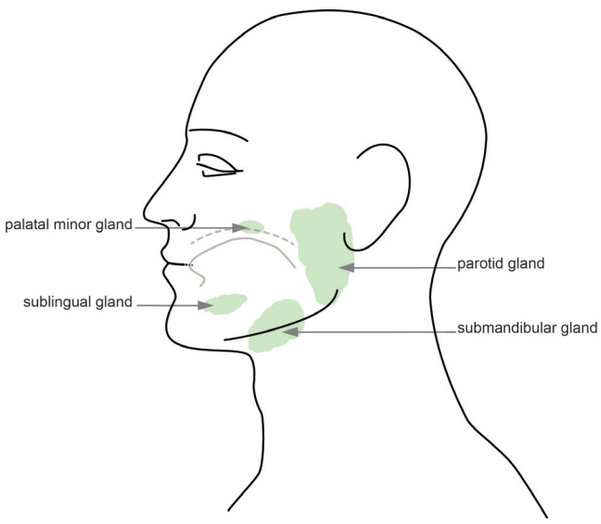


Figure 1: Major salivary glands.

Inflammatory

Acute Sialadenitis: subdivided into bacterial and viral infections

- Viral mumps (paramyxovirus) is typically a bilateral parotid swelling accompanied with malaise and pyrexia. This lasts 14-21 days and treatment is symptomatic.
- HIV may manifest with bilateral, painless parotid swelling due to benign cyst formation as a result of lymphocytic infiltration. There is a risk of neoplastic disease in this group of patients (lymphoma, Kaposi's sarcoma, adenoid cystic carcinoma) therefore this must be a diagnosis of exclusion.
- Bacterial infection: typically unilateral swelling with overlying erythema and tenderness. This is most common in the parotid gland but may also occur in the submandibular gland. Pus may be seen at the duct orifice on gently massaging the gland. Staphylococcus Aureus and Streptococci are commonly cultured. The elderly, immunocompromised and poorly controlled diabetics are at greater risk and it is commonly associated with dehydration and poor oral hygiene. Some medications causing decreased salivary flow and obstructions within the gland or duct (calculi, strictures) may also predispose to bacterial infections. Treatment is with high-dose broad-spectrum antibiotics including anaerobic cover. Underlying conditions must also be addressed.

Chronic Sialadenitis

Uncommon condition typically affecting the parotid gland and thought to be caused by stasis of salivary flow or following episodes of acute infection. Patients have recurrent episodes of pain and swelling which may require antibiotics in the acute phase.

Recurrent sialadenitis of childhood

Children typically 3 to 6 years old present with recurrent episodes of swelling, typically in the parotid, with associated discomfort. This lasts several days at a time and eventually resolves spontaneously. Treatment is symptomatic and rarely includes the use of antibiotics.

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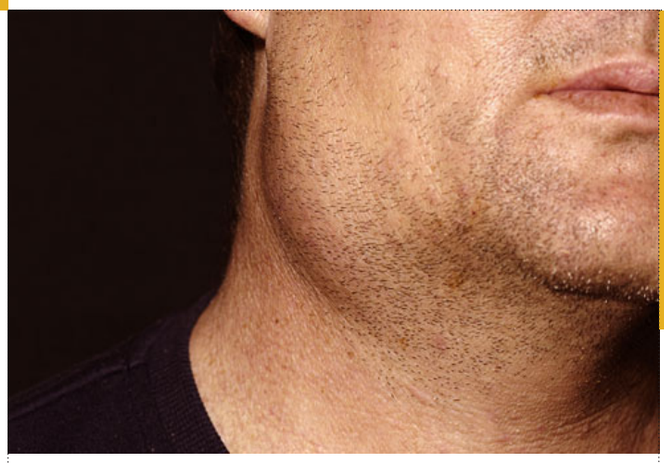

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Granulomatous disease

- *Cat scratch disease:* There is a history of a scratch from a cat or other animal transmitting gram-negative bacillus, Bartonella henselae. This causes lymph node destruction and necrosis with possible suppuration at the site of inoculation. accompanied with fever and malaise.
- *Actinomycosis:* a chronic suppurative infection caused by actinomycoses israelii infection arising from the dentition or tonsils. Patients present with swellings near the angle of the jaw and upper neck and an overlying purplish hue. They go on to develop discharging skin sinuses. Sulphur granules in pus specimens are diagnostic. Treatment is surgical debridement and drainage as well as high-dose penicillin for 4-6 weeks.
- *Sarcoidosis:* A chronic granuloma forming disease. The salivary glands may be involved presenting with tumour-like painless swellings. Heerfordt's syndrome of uveitis, parotid enlargement and facial palsy may warrant the use of steroids as well as symptomatic treatment.
- *Sjogren's syndrome:* This is an autoimmune disease causing dry mouth and dry eyes in Primary Sjogren's syndrome with the addition of another connective tissue disease in Secondary Sjogren's syndrome. Histologically there is lymphocytic infiltration into the salivary and lacrimal glands with progressive destruction of acini. They are at greater risk of developing acute bacterial sialadenitis and chronic sialadenitis.
- *Sialosis:* Uncommon painless enlargement of parotid glands which is of unknown cause but has common associations with diabetes, alcoholic liver disease, thyroid disease and bulimia. Treatment is to correct the underlying condition.

Salivary gland obstruction

- *Sialolithiasis:* Calculi form most commonly in the submandibular gland and duct (Figure 2), then the parotid and rarely form in the sublingual gland. Ductal stones of the parotid gland can be removed via an intraoral surgical approach, basketing techniques or by means of an endoscope inserted into the parotid duct. Very large calculi can be broken down using lithotripsy and are then passed spontaneously or retrieved with a basket. In rare cases the entire gland is removed.
- Ductal strictures may occur as a result of scarring .

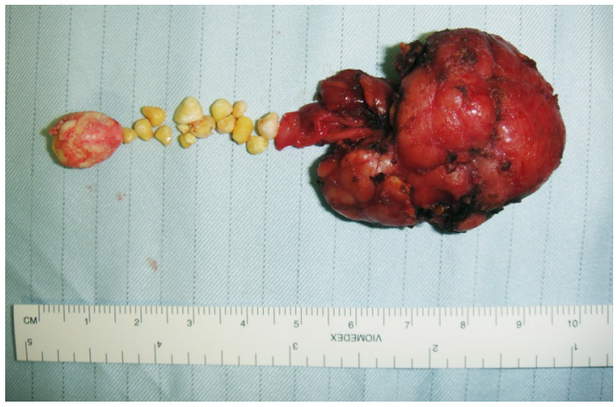

Figure 2: Submandibular gland and duct with multiple calculi.

Neoplastic

Benign

- *Pleomorphic Adenoma:* The commonest salivary neoplasm which accounts for 80% of all parotid neoplasms . Typically unilateral, slow-growing and painless with preservation of facial nerve function. Diagnosis is with imaging and fine needle aspiration cytology. Treatment is surgical with superficial parotidectomy. Facial nerve damage is an accepted risk of the surgery.
- *Warthin's tumour:* typically occurs in elderly males who develop a painless mass in the parotid.

. 10% are bilateral and treatment is as for a pleomorphic adenoma.

Malignant

Clinical appearance may be similar to benign lesions presenting with an asymptomatic mass. Features raising suspiscion of malignancy include rapid expansion, pain, fixation and changes in the overlying skin. Involvement of the facial nerve i.e. paralysis is highly suggestive of malignancy. Malignant neoplasms may invade deep structures and metastasise to adjacent lymph nodes. Imaging of choice is MRI and FNAC is a helpful diagnostic tool. If malignancy is suspected imaging of the chest is also warranted. Tumours are staged using the tumour/node/metastasis (TNM) tool. Treatment typically involves surgery with the possibility of radiotherapy and/or chemotherapy. Management is part of a multidisciplinary team approach.

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Case History A

Mrs K, a 35 year old fit and well lady was referred by her GP with a lump below her left ear. This had been present for some years. It was slowly increasing in size, but otherwise asymptomatic.

Focussed History

What are the important questions to ask when taking a history for a salivary gland swelling?

- Does the swelling fluctuate in size, increasing just before mealtimes? This is relevant as a swelling that fluctuates at mealtimes is likely to be due to blockage or stricture within the salivary duct. This phenomenon is called "Mealtime Syndrome'.
- Ask about the duration of the condition and the rapidity of growth.
- Is the swelling associated with pain? This is a feature of 'Mealtime Syndrome', inflammatory and malignant lesions.
- Is there any alteration in the movement of the face on the associated side? If the facial nerve is compromised alarm bells should ring.
- Is there any reported sensory change over the face or inside the mouth?

Does food taste as it should?
Is the lesion affecting the trigeminal, facial or glossopharyngeal nerves.

- Is there any reported difficulty in swallowing? A deep parotid lobe mass can cause deviation of the ipsilateral tonsil which may be felt by the patient, especially on swallowing.
- Does the patient report any difficulty in chewing? Is this an invasive lesion affecting the adjacent masseter muscle?
- Does she have a sensation of dry mouth indicating reduced salivary gland function?
- Check for previous history of trauma to the face or treatment under another clinician where the integrity of the salivary gland may have been compromised.
- Does she have a good fluid intake on a day-to-day basis or take any medications? Ask about anxiety and depression. Does the patient have a history of chronic excessive alcohol consumption? These can all cause xerostomia which predisposes the patient to sialolith formation and ascending bacterial salivary gland infections.
- Is there any history of radiotherapy to the head and neck area? This causes intrinsic damage to the salivary gland. With a sub-optimal salivary gland function the patient will be at greater risk of bacterial infection.
- Does she suffer with ocular dryness or an autoimmune disorder? This could be linked to Sjogren's Syndrome.
- Is there a history of diabetes? Poorly controlled diabetes can predispose to opportunistic infections.
- Is the patient up to date with immunisations including the mumps MMR vaccine.
- Does the patient have any risk factors for developing malignant lesions? Ask about smoking and alcohol drinking habits, apetite and body weight. Ask about their occupation and situation at home.
- Fever, chills and night sweats are all indicative of acute infection.

Focussed Examination

Head and neck:

There was facial asymmetry with a visible swelling over the left pre-auricular area. This was a well circumscribed 5cm x 6cm mass, firm, rubbery and non-tender. It was lobulated, mobile and lifted the right ear lobe. The skin overlying it was normal (Figure 3).

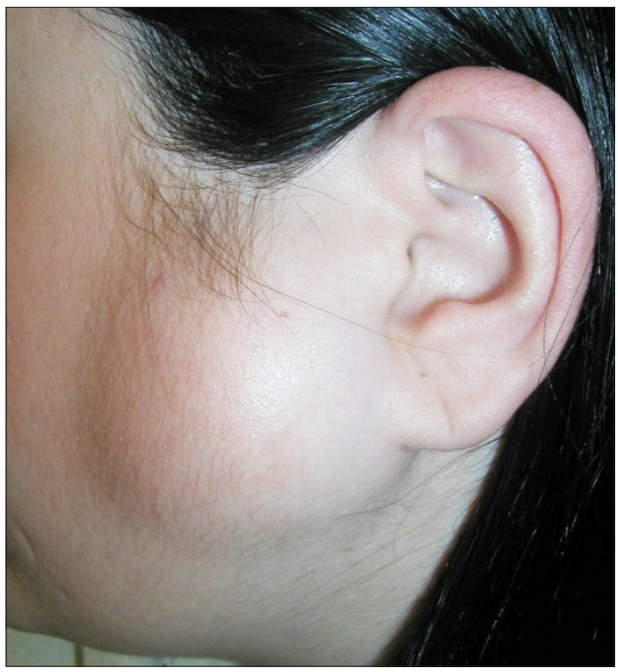

Figure 3: 6 years history of a left parotid mass.

There was no weakness of the muscles of facial expression or muscles of mastication. There was no sensory deficit over the distribution of the trigeminal nerve. No palpable cervical or submandibular lymph nodes were detected.

Discussion

This clinical picture is most likely to represent a benign tumour of the parotid gland. The most common benign salivary tumour is the pleomorphic adenoma. Other benign salivary tumours include Warthin's tumour and basal cell adenoma. Further investigations are required to confirm the clinical diagnosis.

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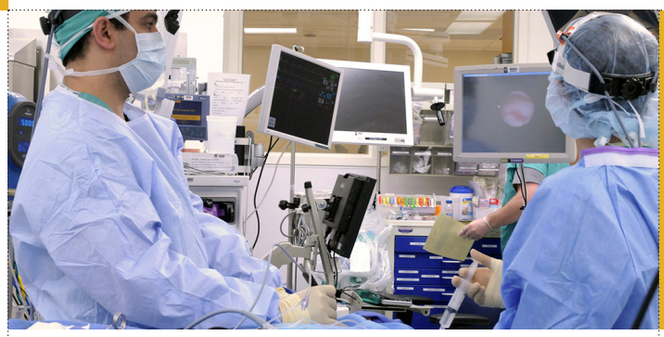

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What radiological investigations can be used to aid diagnosis and management in salivary gland pathology?

- Plain films are primarily used to identify stones (calculi) although only 80-85% of all stones are radiopaque.
- Sialography. The salivary gland is cannulated and a radiopaque dye injected into the ductal system. A series of radiographs are obtained during the procedure. This is useful for demonstrating salivary stones and gland architecture. This procedure is contraindicated in acute salivary gland infections.
- Ultrasonography (US) is a relatively simple, non-invasive investigation used to determine whether a mass lesion is solid or cystic. It is a good preliminary investigation to confirm a parotid swelling and to differentiate between glandular and ductal pathology .
- Fine needle aspiration cytology (FNAC) or core biopsy is used to identify benign or malignant cells but is not always conclusive.
- Computed Tomography (CT) is generally reserved for assessing mass lesions of the salivary glands.
- Magnetic Resonance Imaging (MRI) is used to delineate the soft tissue detail of salivary gland lesions.

The following investigations were performed. Findings are detailed below.

- MRI scan: suggestive of pleomorphic adenoma in the superficial lobe of the parotid gland.
- US guided FNAC: confirmed diagnosis of pleomorphic adenoma

Management

This patient went on to have the pleomorphic adenoma removed surgically with a superficial parotidectomy procedure under general anaesthesia. The procedure provides both the definitive diagnosis and treatment.

Follow-up

This patient's initial recovery was very good, however, years later, Mrs K returned saying she had developed some lumps in the same region. Examination revealed three discreet lumps in the region of the left parotid with no facial nerve involvement. A repeat MRI scan confirmed the lumps to be within the remnant parotid gland and FNAC confirmed recurrent pleomorphic adenoma. Further surgery and radiotherapy was utilised to eliminate residual disease.

Conclusion

Salivary gland swellings are common but salivary gland tumours are rare. There are a multitude of inflammatory salivary gland conditions which demonstrate a wide range of pathological processes. The key to making a diagnosis is through listening to the patient's history.

Neoplastic disease in the major salivary glands is more common and more likely to be benign in contrast to the minor salivary glands. Pleomorphic adenoma being the commonest overall salivary gland tumour. 80% of parotid tumours are benign and of these 80% are pleomorphic adenomas. 10% of parotid lumps are malignant with the most common being a muco-epidermoid carcinoma.

The smaller the salivary gland, the lower the incidence of neoplastic disease but the higher the chance of malignancy. Virtually all sublingual gland tumours are malignant. Management is best provided by a specialist Oral and Maxillofacial Surgery team and therefore referral is advised.

Self Assessment Questions

1. The following are options for treatment of sialoliths

a. Sphincterotomy and extraction of stone
b. Ductotomy and extraction of stone
c. Basketting and removal of stone under fluoroscopy
d. Lithotripsy
e. All of the above are true

2. Which of the following are true statements

a. 8:1:1 rule refers to the distribution of all tumours in the salivary glands
b. Tumours of the sublingual gland are more likely to be benign
c. Tumours of the parotid gland are more likely to be malignant
d. Warthin's tumour is the most common benign tumour of the salivary glands
e. 10% of Pleomorphic adenoma of the parotid gland are likely to be bilateral

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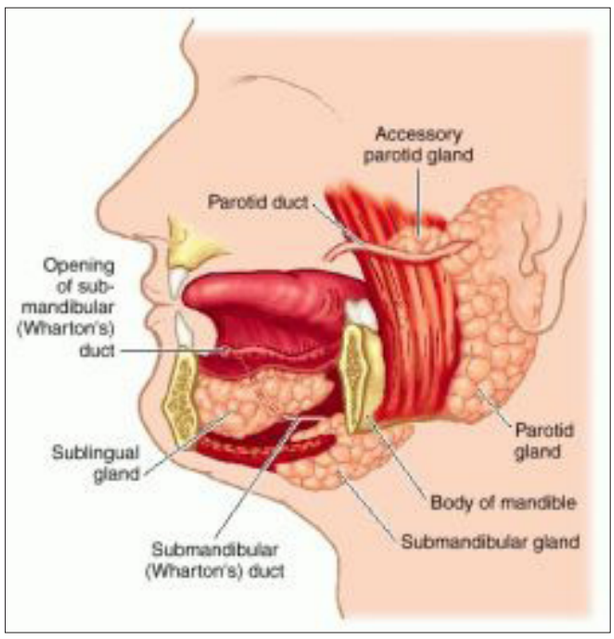

3. Imaging of salivary glands

a. Ultrasound imaging of salivary glands is a good primary investigation, but is operator dependant.
b. Sialograms are useful in diagnosing salivary gland tumours
c. MRI scans give excellent resolution images of parotid tumours.
d. Plain radiographs are not useful in diagnosis of ductal stones
e. Ultrasound of salivary glands may be useful to differentiate between inflammatory pathology and tumours

Answers to MCQs and learning points

1. (e) All of the above are true

2. (a) 8:1:1 refers to distribution of tumours in salivary glands (80% of tumours occur in parotid gland, 10% in submandibular and 10% in minor salivary glands). Sublingual gland tumours are almost always malignant. 80% of all parotid tumours are benign Pleomorphic adenoma is the most common benign tumour of the salivary glands.

3. (a), (c) & (e) are correct. Sialograms are more useful for diagnosing intraductal pathology such as ductal calculi and strictures, though occasionally they may show abnormal splaying of ducts around a tumour (this is not the primary modality of investigation of tumours). Plain radiographs such as dental X-rays (lower occlusal view), Orthopantomogam (OPG) may show salivary calculi.

References

1. Ord RA, Pazoki AE. Salivary gland diseases and tumors. In: Miloro M, Larsen P, Ghali GE, Waite P eds. Peterson's principles of oral and maxillofacial surgery, Volume 1, 2nd edn. London: BC Decker Inc Hamilton 2004; 671-7.

2. Berry RL. Sialadenitis and .sialolithiasis: diagnosis and management. Oral Maxillofac Surg Clin North Am 1995; 7:479.

3. Karavidas K, Nahlieli O, Fritsch M, McGurk M. Minimal surgery for parotid stones: a 7-year endoscopic experience. Int J Oral Maxillofac Surg 2010; 39(1):1-4.

4. Cawson RA, Odell EW. Neoplastic and non-neoplastic diseases of salivary glands. In: Cawson RA, Odell EW eds. Cawson's essentials of oral pathology and oral medicine, 8th edn. Edinburgh: Churchill Livingstone 2008; 291-313.

5. Eveson JW, Cawson RA. Warthin's tumor (cystadenolymphoma) of salivary glands. A clinicopathologic investigation of 278 cases. Oral Surg Oral Med Oral Pathol 1986; 61: 256-62.

6. Bradley M J. Salivary glands. In: Ahuja AT, Evans R, eds. Practical head and neck ultrasound. Cambridge University Press 2008; 19-33.

7. Bradley P. General epidemiology and statistics in a defined UK population. In: McGurk M, Renehan A eds. Controversies in the management of salivary gland disease. Oxford: Oxford University Press 2001; 3-12.

8. Speight PM, Barrett AW. Salivary gland tumours. Oral Dis 2002; 8(5):229-40.

MANAGEMENT OF SALIVARY GLAND SWELLINGS

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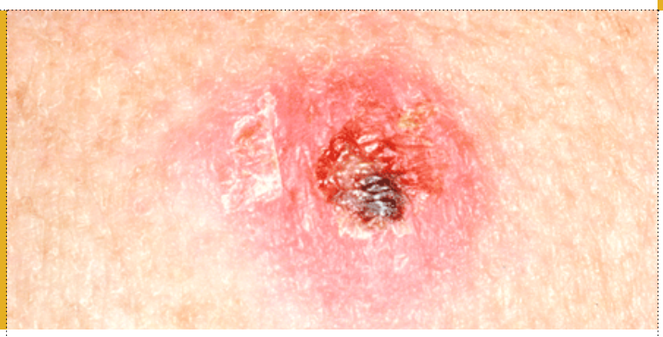
COMMON BENIGN AND MALIGNANT SKIN LESIONS OF THE HEAD AND NECK

L Cheng, M Alibhai & L Pozo-Garcia

Common benign and malignant skin lesions of the head and neck. Good Clinical Care.



Abstract

Lumps and bumps of the head and neck are a common presenting complaint and often pose a diagnostic dilemma. They are often brought to the attention of various healthcare professionals in different specialities. It can be difficult for the junior doctor to accurately differentiate benign from malignant skin lesions on the head and neck. Clinical features suggestive of malignancy include rapid growth in size, change in colour, asymmetry and bleeding. Raised suspicion should be made in individuals who present with new lumps, especially those with fair skin. Suspicious lesions should be biopsied to obtain definitive histological diagnosis.

This article aims to cover the basics of diagnosis and management of the most common head and neck lumps and bumps.

Key points

- Epidermoid cysts, seborrhoeic keratoses and pigmented melanocytic naevi are the most common benign skin lesions on head and neck.
- Most moles are harmless and benign. The presence of colour changes, enlargement or bleeding in a mole should raise suspicion and these patients need referral to the dermatologist for further assessment.
- Basal cell carcinoma (BCC), squamous cell carcinoma (SCC) and malignant melanoma (MM) are the most common types of skin cancers.
- Basal cell carcinoma is a slow growing tumour but it may cause clinically significant local destruction and disfigurement if neglected or inadequately treated.
- Actinic keratoses and Bowen's disease (SCC in situ) are the most important precursor lesions for SCC's. Ear and lip SCC's have a higher potential risk of metastases.
- Lentigo malignant melanoma is typically located on the face of elderly patients and presents as an irregular macule with variegate pigmentation.
- Conventional surgery is the treatment of choice for most skin cancers, early diagnosis is crucial.

Benign Skin Lesions

There are multiple lumps and bumps on the skin which can occur on head and face. Cystic lesions[1], seborrhoeic keratoses and moles are among the most frequent cutaneous benign lesions on head and neck.

Cutaneous Cysts

1. Epidermoid cyst

Epidermoid cysts are the most common cutaneous cysts (Figure 1). They frequently affect young and middle age adults and are twice as common in males as females. Although they can appear anywhere on the skin, face is commonly involved, in particular in patients with acneiform skin or family history of acne.

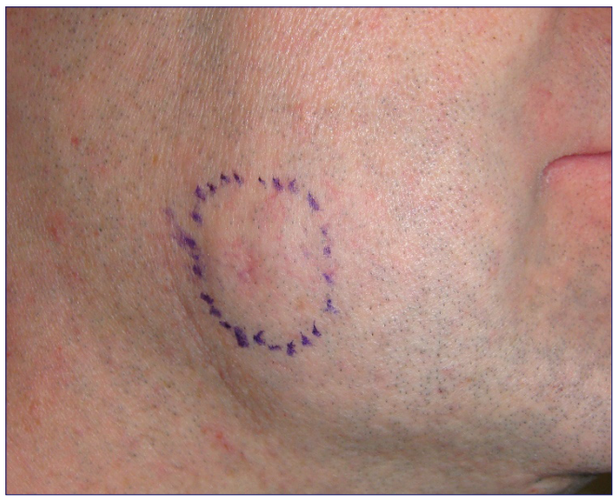


Figure 1: Epidermoid cyst of right cheek.

Epidermoid cysts are derived from the follicular infundibulum and are also known as "infundibular cysts". They may be primary or arise from disrupted follicular structures or traumatically implanted epithelium, hence the synonym of "inclusion cyst".

Clinically, they appear as firm, well-circumscribed dermal or subcutaneous nodules with a visible central punctum, representing the follicle from which the cyst is derived. They are filled with a keratinous material. These cysts can become inflamed and present as abscesses.

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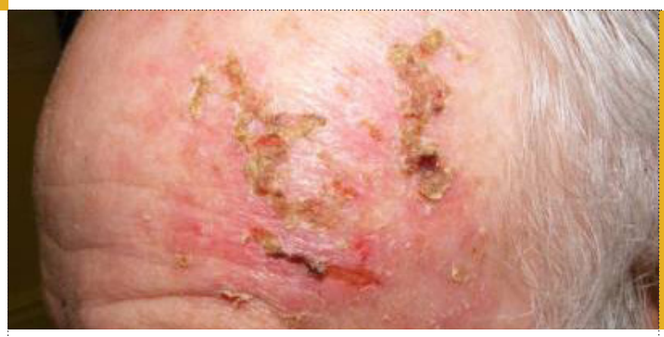

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Histology

Histological examination shows a cavity lined with stratified squamous epithelium and filled with laminated keratin.

Treatment

Removal may be performed by simple excision with an overlying ellipse of skin to include the punctum. The entire cyst wall has to be removed to prevent recurrence. Inflamed cysts may require a course of oral antibiotics. They can be treated by formal excision once the acute phase has resolved.

2. Pilar cyst

Pilar or trichilemmal cysts are common on the scalp and occur in 5-10% of the population. They arise from the outer root sheath or trichilemma of the hair follicle and can locate preferentially in areas of dense hair follicle concentration.

Clinically, 90% of pilar cysts arise in the scalp and they present as smooth, mobile swellings. Overlying hair loss may be noted.

Histology

Pilar cysts are lined by squamous epithelium without a granular layer and contain homogenous, non-laminated keratin with frequent areas of calcification.

Treatment

Treatment is by excision. Pilar cysts are often "delivered" through an incision more easily than epidermal cysts.

3. Dermoid cyst

Cutaneous dermoid cysts typically present in an infant as a discrete subcutaneous nodule on the head. They occur as a result of sequestration of skin along lines of embryonic closure. Forehead and periocular area, in particular the lateral aspect of eyebrows, are the most common locations of dermoid cysts.

Histology

From the histological point of view, cutaneous dermoid cysts are lined by stratified squamous epithelium including granular layer. They contain other normal cutaneous structures, such as hair, sebaceous lobules, sweat glands or smooth muscle.

Treatment

Complete surgical excision is the treatment of choice.

Seborrhoeic Keratoses

Seborrhoeic keratoses are the most common benign growth in older individuals (Figure 2). The aetiology is unknown but some patients have a positive family history, which may reflect a genetic predisposition. They occur more frequently in sunlight-exposed areas[2].

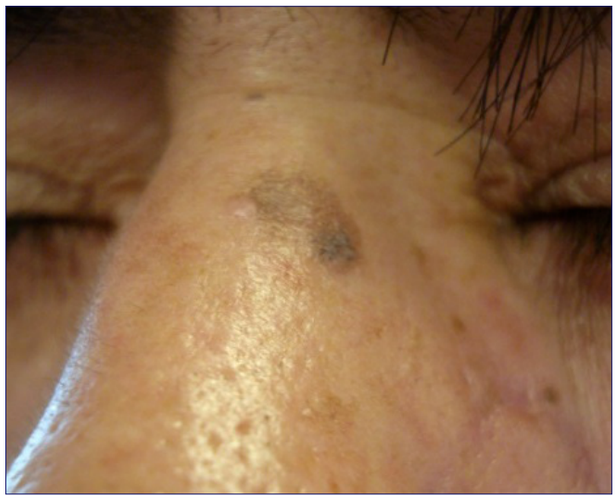

Figure 2: Seborrhoeic keratosis of left bridge of nose.

They begin as flat and sharply demarcated brown macules, which develop an uneven, verrucous or waxy surface with a characteristic stuck-on appearance in more advanced lesions. Some seborrhoeic keratoses may become reddish when inflamed and may simulate skin cancer.

Dermatosis papulosa nigra is a variant of seborrhoeic keratoses, which typically appears in individuals of African descent with darkly pigmented skin. It consists of multiple small hyperpigmented smooth papules on cheeks.

Histology

Seborrhoeic keratoses are characterized by variable degrees of hyperkeratosis, acanthosis and papillomatosis, that is, thickening of the epidermis, secondary to the accumulation of benign squamous and basaloid keratinocytes.

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Horn pseudocysts (cross-sectioned epidermal invaginations) are highly characteristic of seborrhoeic keratoses.

Treatment

Treatment of asymptomatic seborrhoeic keratoses is mainly performed for cosmetic reasons. The most common treatment modalities include shave excision, cyrotherapy, curettage and electrodessication.

Pigmented Melanocytic Naevi (Moles)

Naevi or moles are common pigmented lesions that are caused by the proliferation of melanocytes in the dermo-epidermal junction (junctional), dermis (intradermal) or both (compound naevus). Moles tend to appear during early childhood, remain relatively constant in numbers during adulthood, and decrease or regress in the elderly.

Pigmented melanocytic naevi can be classified in congenital and acquired depending on the age of onset.

- Congenital melanocytic naevi are those present from birth or appearing during the first few years of life[3]. Clinically present as brown macules, which tend to darken and thicken over time. The presence of coarse hairs on the surface is typical of these naevi. They can present anywhere in the skin, including the face and neck. Small congenital naevi have a very small risk of melanoma and should not be excised routinely.

- Acquired melanocytic naevi appear later in life anywhere in the skin, face and neck are commonly involved areas. The subclassification of acquired naevi is summarised in table 1.

Acquired melanocytic naevi	Clinical features
Junctional naevi	Flat, regular, brown macules
Intradermal naevi	Flesh coloured papules, sometimes with warty or hairy surface. Common on head and neck.
Compound naevi	Brown raised lesions, often with hyperpigmented halo.
Blue naevi	Well-defined bluish papules on dorsa of hands and feet, also common on the face and scalp.
Spitz naevi	Well-circumscribed red papule, angioma-like. Face of children.

Table 1: Subclassification of acquired melanocytic naevi.

Most moles are harmless and benign[4,5]. Malignant melanoma is exceptional in childhood. In general, regular borders, uniform colour and size less than 6 mm are benign clinical features. The majority of patients presenting for mole checking require reassurance only but it is important to detect potential changes as an early diagnosis of melanoma saves lives. Patients with atypical pigmented lesions need referral to specialists in Dermatology for assessment.

Skin cancers

Basal cell carcinoma, squamous cell carcinoma and malignant melanoma are the most common types of skin cancer located on head and neck.

Basal Cell Carcinoma

Basal cell carcinoma (BCC) is the most common malignancy in humans. It is a malignant neoplasm derived from non-keratinizing cells from the basal layer of the skin –basal cells-, hence its name.

It typically occurs in areas of chronic sun exposure and is more common in fair skinned individuals, although it can also present in dark skinned races (pigmented variant). It is a tumour more frequently found in adults or elderly patients, but the incidence is progressively increasing in younger populations[5].

Although sun exposure is the primary aetiological agent, other risk factors for BCC include exposure to ionizing radiation, chemical exposures, rare genetic conditions and immunosuppression.

There are several clinical subtypes of BCC, which include nodular, superficial, morphoeic and pigmented BCC's. These variants may exhibit different patterns of behaviour. Recognizing the clinical subtypes is important because more aggressive therapy is often necessary for higher risk variants.

- Nodular BCC is the most common subtype of BCC and is particularly frequent on head and neck (Figure 3). It is characterised by a translucent dome-shaped papule or nodule with superficial telangiectasias and pearly-white border associated with a central crusted or ulcerated area. The presence of any friable, bleeding, non-healing ulcer should raise the suspicion of skin cancer, especially in the elderly population.

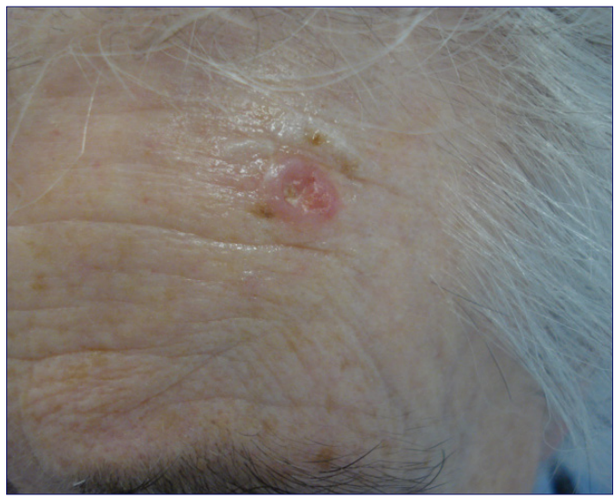

Figure 3: Nodular basal cell carcinoma of left forehead.

- Pigmented BCC clinically presents as a hyperpigmented nodular BCC due to the high melanin content. This variant is most commonly found on head and neck of dark-skinned individuals.

COMMON BENIGN AND MALIGNANT SKIN LESIONS OF THE HEAD AND NECK

L Cheng, M Alibhai & L Pozo-Garcia

· Morphoeic or sclerodermiform BCC is an aggressive growth variant of BCC, which clinically resemble a scar or small lesion of morphea (Figure 4). It appears as an ivory-white or yellowish indurated patch, most commonly on the face. It can be easily overlooked in the early stages.

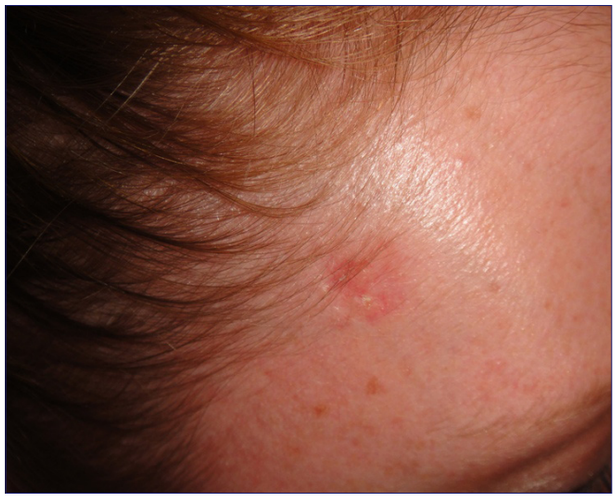

Figure 4: Morphoeic basal cell carcinoma of right forehead.

· Superficial BCC appears as an erythematous patch, usually on trunk, simulating eczema. It is unusual on head and neck.

Histology

Histopathological features vary with the subtype of BCC, but most of them share some cytological features, such as cells with large, oval hyperchromatic nuclei and scanty cytoplasm. The peripheral cells are typically arranged in a palisading pattern and there is retraction of the stroma from the tumour islands, creating peritumoral lacunae.

Treatment

The greatest danger of BCC results from local invasion. Although it is a slowly growing tumour, if left untreated it may result in significant tissue damage and local destruction. Management of BCC depends on anatomical location and histological features. Ideally, a surgical approach with a complete excision is the treatment of choice. Defects are repaired or reconstructed depending on the size of defect and the most suitable method on the reconstructive ladder. The most appropriate surgical margins and guideline for basal cell carcinoma have been produced by the British Association of Dermatologists (2008)[6]. Other therapeutic options are curettage, cryotherapy, topical preparations (imiquimod cream 5%, 5-fluoruracil cream) or photodynamic therapy in selected cases.

Mohs micrographic surgery is indicated in recurrent or aggressive forms of BCC. With the Mohs technique, a repeated series of surgically removed tissue are mapped, colour-coded and examined microscopically of all tissue margins in order to ensure the tumour is completely excised. Radiotherapy may be considered as palliative therapy in large facial tumours of frail elderly patients when surgery is contraindicated.

Squamous Cell Carcinoma

Cutaneous squamous cell carcinoma (SCC) is the second most common form of skin cancer[7]. The most important etiologic factor is chronic sun exposure, but chemical carcinogens, HPV infection, genetic syndromes, chronic inflammatory dermatoses and immunosuppresion (organ transplantation) are also risk factors for the development of SCC.

· SCC can arise de novo, or most commonly from precursor lesions, such as actinic keratoses (Figure 5). Actinic or solar keratoses have been classically considered as precancerous conditions as the atypical keratinocytes are confined to the epidermis. There is no risk of metastases until these lesions evolve into invasive carcinoma . They present on sun damaged skin and are very common on the face. The primary lesion is a rough erythematous patch with white or yellowish scale. They can be multiple and thick in advanced cases.

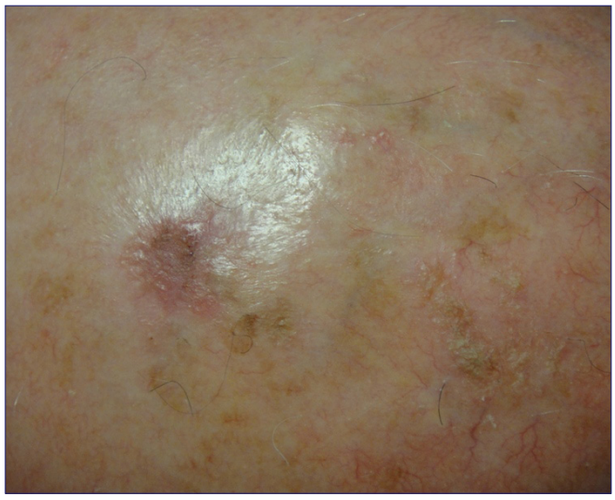

Figure 5: Actinic keratoses of the scalp.

· SCC in situ is commonly called Bowen's disease. The most common presentation is an erythematous scaling patch on sun-exposed areas of elderly individuals. Head and neck are commonly involved.

· Invasive SCC presents as a raised, pink-coloured keratotic indurated papule or plaque with variable degree of erosion and ulceration (Figures 6 and 7). Large exophytic fungating lesions may be seen in advanced or neglected cases (Figure 8). As these lesions progress, they become locally invasive and destructive, fixed to underlying and adjacent structures with potential capacity of metastasize to cervical lymph nodes and then to distant sites. Ear and lip are considered high risk locations, SCC of the ear shows the greatest risk of recurrence, while lip SCC has the highest risk of metastases.

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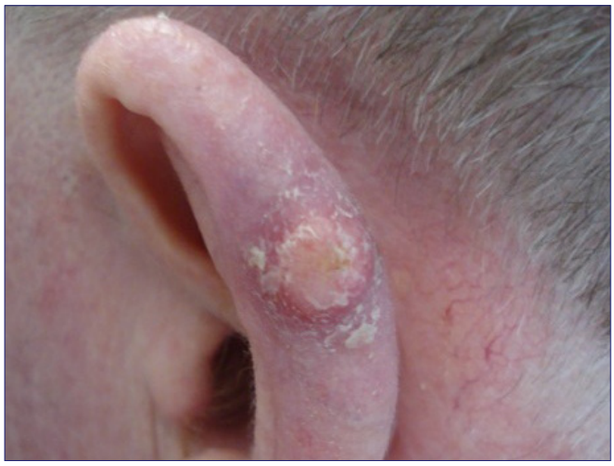

Figure 6: Early squamous cell carcinoma of left pinna.

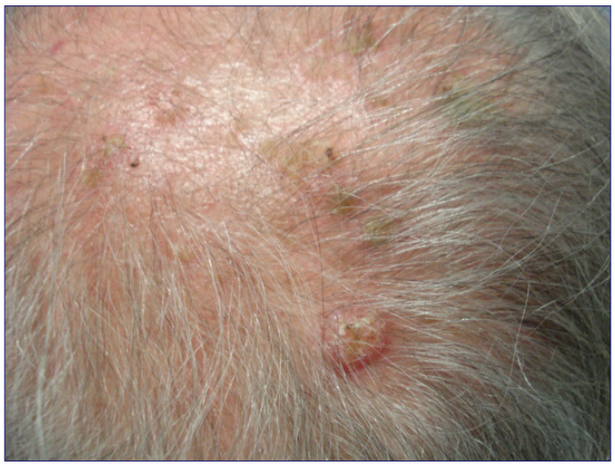

Figure 7: Early squamous cell carcinoma of the scalp.

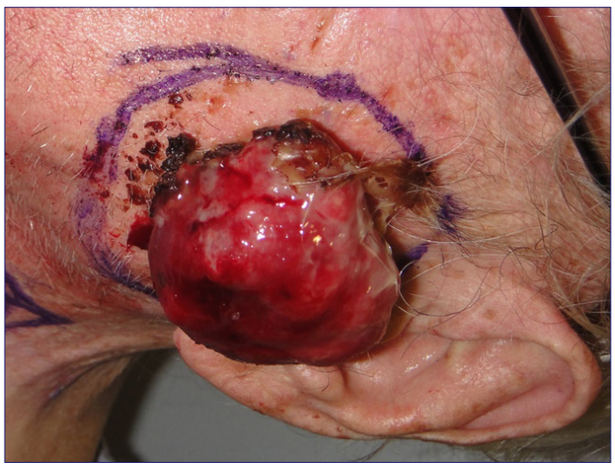

Figure 8: Squamous cell carcinoma of left cheek.

• Keratoacanthoma is a well-differentiated variant of SCC, which typically presents as a well-circumscribed dome-shaped papule or nodule with central keratotic crater[9]. They tend to grow quickly over a period of a few weeks and then may spontaneously resolve leaving an atrophic scar. Most lesions occur on the head. Although classically considered as a benign lesion due to the spontaneous regression, keratoacanthoma can be locally destructive and aggressive and is currently viewed as a clinical subtype of SCC[10].

Histology

SCC in situ shows full-thickness atypia of keratinocytes confined to the epidermis. Invasive SCC is characterized by a downward proliferation of lobules and islands of atypical keratinocytes. Perineural and/or perivascular infiltration and sclerotic stroma changes are common in poorly differentiated SCC's.

Treatment

Surgical excision with a 4–6 mm margin is the treatment of choice in most cases. Surgical margins and guidelines have been updated by the British Association of Dermatologists (2009)[11]. Cryotherapy, electrodesiccation and curettage may be a therapeutic option in small low-risk SCC's. Mohs micrographic surgery can be performed with excellent cure rates for large tumours or compromised locations. Mohs surgery is a microscopically controlled surgery, which provides histological control of the surgical margins and therefore it achieves the lowest recurrence rate with maximal preservation of the normal tissue. This is the treatment of choice for recurrent or histologically aggressive skin cancers. Neck dissection followed by chemoradiotherapy is provided for metastatic neck disease. Other treatment modalities include laser ablation, photodynamic therapy (actinic keratoses/Bowen's disease), local immunotherapy (imiquimod) and local chemotherapy (5-fluoruracil). Sun protection measures (for instance, slip, slop and slap) and regular follow up should be advised.

Malignant Melanoma

Melanoma is a malignant tumour arising from melanocytes. It accounts for a minority of all skin cancers, however, it causes the largest number of skin cancer–related deaths worldwide. The development of melanoma is multifactorial[12]. Putative factors include fair complexion, excessive childhood sun exposure (sunburns), an increased number of common and dysplastic moles, a family history of melanoma, and rare genetic syndromes involving DNA repair defects.

Classically, melanoma can be classified into four clinical types: Superficial spreading malignant melanoma, nodular melanoma, lentigo maligna melanoma and acral lentiginous melanoma. Acral lentiginous melanoma typically occurs on the palms and soles of elderly individuals.

• Superficial spreading malignant melanoma and nodular melanoma can both occur on face and neck, as variegated irregular macules or nodules (50% amelanotic).

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· The most common subtype of melanoma on the face is the lentigo maligna melanoma (Figure 9). It occurs on chronically sun-damaged skin of elderly patients, in particular nose and cheeks, as a slow-growing asymmetric brown to black macule with irregular indented borders.

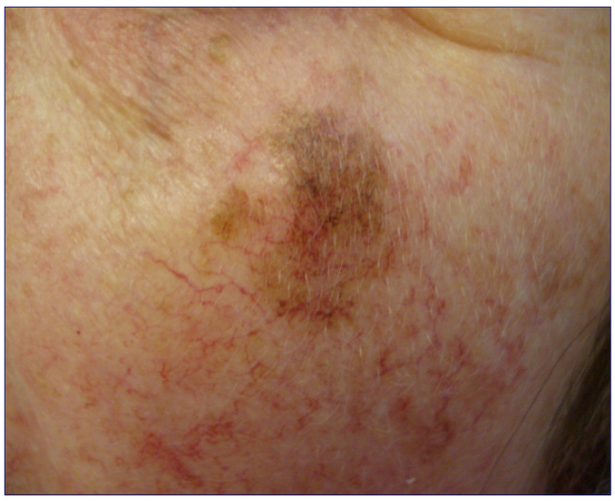

Figure 9: Lentigo maligna melanoma of left cheek.

· Lentigo maligna (melanoma in situ) is the precursor lesion of lentigo maligna melanoma. It has been estimated that approximately 5% of lentigo malignas progress to invasive melanomas. The presence of dark areas or new papules on facial lentigos should be considered as suspicious and these patients need to be referred to a dermatologist.

Early detection of melanoma is crucial to improve the prognosis and the key is public and clinician awareness. Hand held dermatoscope either contact using liquid interface or non-contact with cross-polarisation has improved the rate of melanoma detection. Biopsy and histopathology remains the gold standard for melanoma diagnosis.

Histology

Melanoma is defined by asymmetric atypical melanocytes with confluent nests extending along adnexal structures. There is no maturation of melanocytes with progressive descent into the dermis. Dermal mitotic figures appear in vertical growth phase melanomas.

It has been proposed that melanoma progresses in two phases: the first is the "radial growth phase", defined by centrifugal spread of melanocytes within the epidermis and infiltration of the papillary dermis by single cells or small nests, with minimal capacity for metastases. The second, "vertical growth phase" is characterized by the presence of dermal nests or nodules of atypical melanocytes, that are larger or cytologically distinct from their epidermal counterparts. The vertical growth phase has metastatic potential.

Prognosis

The prognosis depends on the stage at diagnosis . Stages I and II denote local cutaneous disease, stage III disease involves regional nodes, while stage IV disease is associated with distant metastases. Tumour thickness (Breslow depth) is the most important single prognostic factor in melanomas. Thickness is measured from the top of the granular layer to the greatest depth of tumour invasion. Survival is inversely proportional to the Breslow depth. Ulceration is another negative prognostic factor correlated with patient survival.

Treatment

The standard of therapy for primary cutaneous melanoma is wide local excision with appropriate margins, indicated by Breslow thickness. The surgical margins and guidelines are prepared by the British Association of Dermatologists (2010)[14]. Sentinel cervical lymph node biopsy followed by radical neck dissection for positive sentinel lymph node are performed in some specialist centres. Chemotherapy and immunotherapy have not shown overall survival benefit.

Multiple Choice Questions

1. Which is the most common subtype of melanoma affecting the face of elderly patients?
a) Superficial spreading malignant melanoma
b) Nodular melanoma
c) Acral lentiginous melanoma
d) Lentigo maligna melanoma
e) Amelanotic melanoma

2. Please select the most aggressive form of BCC among the following clinical subtypes of BCC:
a) Nodular BCC
b) Morphoeic BCC
c) Pigmented BCC
d) Superficial BCC
e) Cystic BCC

3. Please select the false statement regarding squamous cell carcinoma.
a) It is the second more common form of skin cancer
b) Chronic sun exposure is the most important etiologic factor
c) Actinic kearatoses are the most common precursor lesions for SCC
d) Bowen's disease is an invasive SCC
e) Conventional surgery with 4-6 mm margins is the treatment of choice

4. Please select the false statement regarding pigmented melanocytic lesions.
a) Malignant melanoma in childhood is exceptionally rare
b) Junctional melanocytic naevi appear as flat brown macules
c) Compound melanocytic naevi are more common on the trunk
d) Intradermal naevi present as flesh round coloured papules
e) Small congenital melanocytic naevi have a high risk of developing melanoma

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5. Keratoacanthoma is considered a variant of one of these conditions:
a) Seborrheic keratosis
b) Basal cell carcinoma
c) Squamous cell carcinoma
d) Actinic keratosis
e) Malignant melanoma

Answers

1. Answer: d
Lentigo maligna melanoma occurs on elderly patients' face due to sun exposure, and nose and cheeks are the most common areas of face affected. The precursor lesion is lentigo maligna or melanoma in situ and 5% of these lesions show malignant transformation.

2. Answer: b
Morphoeic BCC is the most aggressive and therefore requires wider surgical margins (over 5 mm) in order to achieve complete surgical excision. The recurrence rate is for morphoeic BCC is higher than other types of BCC.

3. Answer: d
Bowen's disease is the early stage or intraepidermal form of SCC. Clinically presents as asymptomatic reddish scaly patches on sun exposed areas. The prognosis of patients with Bowen's disease is excellent with appropriate therapy.

4. Answer: e
Small congenital naevi have a very small risk of melanoma and should not be excised routinely, only large congenital melanocytic naevi (more than 20 cm) are considered risk factor for developing melanoma.

5. Answer: c
This is an important lesion because it was classically treated as a benign condition. Now it is viewed as a variant of squamous cell carcinoma because of its destructive and aggressive nature. It is both recommended and a safe practice to remove the keratoacanthoma with a wide surgical margin for histological diagnosis.

References

1. Al-Khateeb TH, Al-Masri NM, Al-Zoubi F. Cutaneous cysts of the head and neck. J Oral Maxillofac Surg 2009; 67(1):52-7.
2. Yeatman JM, Kilkenny M, Marks R. The prevalence of seborrhoeic keratoses in an Australian population: does exposure to sunlight playa part in their frequency? Br J Dermatol. 1997;137(3):411-4.
3. Tannous ZS, Mihm MC Jr, Sober AJ, Duncan LM. Congenital melanocytic nevi: clinical and histopathologic features, risk of melanoma, and clinical management. J Am Acad Dermatol 2005;52(2):197-203
4. Ceilley RI, Del Rosso JQ. Current modalities and new advances in the treatment of basal cell carcinoma. Int J Dermatol 2006;45(5):489-98.
5. Rubin AI, Chen EH, Ratner D. Basal-cell carcinoma. N Engl J Med 2005; 353(21):2262-9.
6. Telfer NR, Colver GB, Morton CA. Guidelines for the management of basal cell carcinoma. Br J Dermatol 2008; 159:35-48.
7. Rudolph R, Zelac DE. Squamous cell carcinoma of the skin. Plast Reconstr Surg 2004;114(6):82e-94e.
8. Fu W, Cockerell C J. The actinic (solar) keratosis : a 21st-century perspective. Arch Dermatol 2003;139(1):66-70.
9. Schwartz RA. Keratoacanthoma: a clinico-pathologic enigma. Dermatol Surg 2004;30(2 Pt 2):326-33.
10. Karaa A, Khachemoune A. Keratoacanthoma: a tumor in search of a classification. Int J Dermatol 2007;46(7):671-8.
11. Motley RJ, Preston PW, Lawrence CM. Update of the original multi-professional guidelines for the management of the patient with primary cutaneous squamous cell carcinoma. 12th November 2009 British Association of Dermatologists **http://www.bad.org.uk/Portals/_Bad/Guidelines/Clinical%20Guidelines/SCC%20Guidelines%20Final%20Aug%2009.pdf.** Original guideline: Motley RJ, Kersey P, Lawrence CM. Multi-professional guidelines for the management of the patient with primary cutaneous squamous cell carcinoma. Br J Dermatol 2002; 146:18-25.
12. Miller AJ, Mihm MC Jr. Mechanisms of Disease: Melanoma. N Engl J Med 2006;355(1):51-65.
13. Markovic SN, Erickson LA, Rao RD, et al. Malignant melanoma in the 21st century, part 2: staging, prognosis, and treatment. Mayo Clin Proc 2007; 82(4):490-513.
14. Marsden JR, Newton-Bishop JA, Burrows L, Cook M, Corrie PG, Cox NH, Gore ME, Lorigan P, MacKie R, Nathan P, Peach H, Powell B, Walker C. Revised U.K. guidelines for the management of cutaneous melanoma 2010. Br J Dermatol 2010; 163:238-56.

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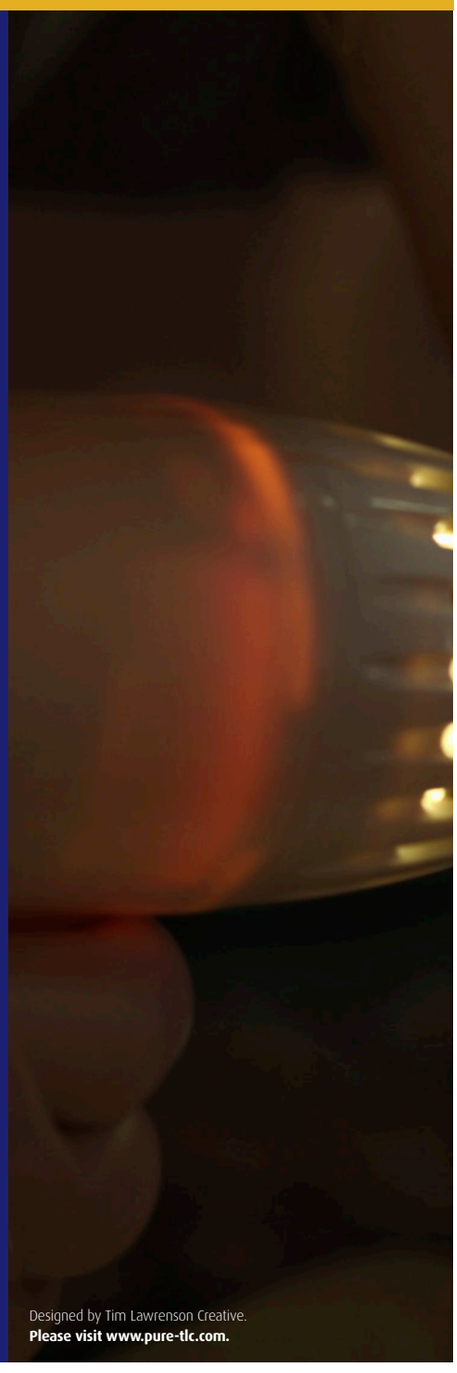

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