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CORE SURGERY JOURNAL

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Dear Prospective Authors

Thank you for considering the submission of an article to 'Core Surgery'. This is a new journal aiming to educate and inform junior surgical trainees about relevant 'core' subject topics. Each issue will cover a topic from selected subspecialty fields; General Surgery, Orthopaedics and Trauma, Plastic Surgery, Ear Nose and Throat Surgery, Neurosurgery, Urology, Paediatric Surgery and Intensive Care Medicine. Articles will be required to be broad enough to help with preparation for the intercollegiate MRCS examination but also focus on key hints and tips on becoming a higher surgical trainee. A list of core topics in each subspecialty has therefore been agreed by the editors based on a selection of key topics in the MRCS curriculum. Authors are advised to agree a topic with the editors before writing an article.

Types of Article

Manuscripts are considered under the following sections:

1) Case based discussions
2) Practical procedures
3) Audit
4) Review articles
5) Course reviews
6) Research papers

Submission of Manuscript

Submissions will only be accepted via email and must be accompanied by a covering letter. Please submit your article to **coresurgery@123doc.com.** The covering letter must include a statement that all authors have contributed significantly and accept joint responsibility for the content of the article. In addition any financial or other conflict of interest must be declared.

Manuscript Style

Submissions should follow the style of the Vancouver agreement detailed in the International Committee of Medical Journal Editors' revised 'Uniform Requirements for Manuscripts Submitted to Biomedical Journals: Writing and Editing for Biomedical Publication', as found at **http://www.ICMJE.org/**

References

All articles must be referenced appropriately. The Vancouver system of referencing should be used; details can be found at **https://workspace.imperial.ac.uk/library/Public/Vancouver_referencing.pdf.** References should be cited using numerals in brackets [eg. (1)], in the order in which they appear. The list of references should reflect this order and names of journals should be abbreviated in the style used in Index Medicus **ftp://nlmpubs.nlm.nih.gov/online/journals/ljiweb.pdf.**

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Format of Articles

Guidelines for the format of respective article types are as follows. All articles must contain an abstract of 150-250 words for indexing purposes and 3-5 keywords.

Case Based Discussions

Guidelines for the format of respective article types are as follows. All articles must contain an abstract of 150-250 words for indexing purposes and **3-5 keywords.**

Case based discussions

Should be about 1000-1500 words long and should focus on clinical assessment, differential diagnosis or treatment. The basic structure should be as follows:

Abstract:	The salient points of the case and discussion.
Case history:	Including the initial presentation, clinical setting and problem, investigation and treatment.
Discussion:	Covering the critical aspects of the management and the treatment options.

Practical Procedures

Should be about 1000-1500 words long. Although not essential it is highly advantageous if pictures and diagrams are supplied to illustrate the most salient points. Articles should be set out as follows:

- Abstract (Essential) – A summary of the article structure and salient features.
- History and pathology
- Indications and contraindications
- Gaining informed consent /explaining procedure to patient
- Equipment required
- Draping / sterile field preparation
- Patient positioning and relevant anaesthetic points
- Documentation of procedure
- Recording of complications and management of such

Audit

Articles should be 1000-1500 words long and of high quality. Each article must contain an abstract. Completed audit cycles are strongly preferred as are audits which have led to guideline development.

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Review Articles

The topic should be relevant to core surgical trainees, and a maximum of 2500 words long. The review should include an abstract, and a clinical vignette of a case relevant to the topic. The aim of including a clinical case is to provide a focus for discussion, and to ensure that the review is relevant and useful to our readership.

Course Reviews

Should be a maximum of 1000 words and review a course which is either mandatory or desirable for core trainees and junior higher surgical trainees. An abstract is required summarising the article contents and salient conclusions.

Research Papers

Although the publication of research articles is not a core aim of the journal, Core Surgery welcomes research submissions if thought to be of interest to the readership. Articles should be written using the following headings (title page, abstract, introduction, methods, results, discussion, references). They should be a maximum of 2500 words of text including abstract, 30 references, 3 illustrations or figures. The abstract should be a maximum of 250 words and use the following headings (introduction, methods, results, conclusion). The title page should contain the title of the paper, the full names of the authors, the addresses of the institutions at which the research was carried out and the full postal address, email address and telephone number of the corresponding author.

MCQs / EMQs (All Articles)

Please note that all articles should be submitted **with five multiple choice questions (MCQs) or extended matching questions (EMQs)** attached, in the style of the Member of the Royal College of Surgeons (MRCS) 'Part A' examination. These questions should have answers and brief teaching notes/discussion included. Examples of the requirements for question style can be found here: **http://www.intercollegiatemrcs.org.uk/old/pdf/samplequestions_MCQ.pdf**

Summary

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MINIMISING BLOOD LOSS IN SURGERY

KC Lee, M Jones

Abstract

The management of blood loss is an important aspect of surgery that influences the outcomes of patients and the need for the use of blood products which carry inherent risks. In this article, we review the various methods that may assist in reducing blood loss during the pre-operative, intraoperative and post-operative periods. We also touch briefly on the different blood products that are commonly utilised when coagulopathy occurs.

Keywords: blood products, surgery, coagulopathy.

Introduction

Surgery is inherently associated with blood loss and major elective and trauma surgical procedures are usually associated with significant amounts of blood loss with more than 50% of estimated blood volume deleted for lost in scoliosis surgery (1, 2).

Although the transfusion of blood and its components can be life-saving, it carries a host of potential risks such as acute and delayed reactions, incorrect blood component transfusion (ABO incompatibility), circulatory overload leading to left ventricular failure, transfusion related acute lung injury, and transfusion-transmitted infections. Some studies have also suggested that allogeneic blood transfusions can cause problems with wound healing (3), and down-modulate the immune system (4) leading to increased infections (5-7). It can even lead to further blood loss by contributing to the development of coagulopathy by the dilution of normal coagulation factors, alteration of acid-base status, hypothermia, or disseminated intravascular coagulation (DIC) (8). Furthermore, in certain situations, transfusion of blood and its components are not an option, for example in patients who are Jehovah's witnesses. Thus one of the most important principles in surgery is the achievement of haemostasis, and it is to both the surgeons' and his patients' interests that strategies are employed to minimise bleeding during surgery. In this article, we discuss the various measures that can be employed through-out the perioperative period to minimise blood loss.

Pre-Operative Period

a) Pre-operative planning

Pre-operation assessment and planning before an elective procedure can help ensure that the patient is in the best condition for the surgery. Planning extends to the surgical instruments, the team and the procedure itself. Reviewing and rehearsing the procedure can help minimise unexpected occurrences and flag up any special precautions that should be taken.

The use of technology in the form of computer aided surgery can be of help especially when complex or unusual anatomy is involved for example, by helping to avoid the breaching of medullary cavities (9) and hence blood loss. Ensuring that the right instruments and implants (and back-ups) are available can avoid valuable time wasted intra-operatively looking for the right kit and allows the operation to progress smoothly with less seeping of blood from cut tissues. Being aware and keeping track of blood loss may help keep operating surgeons vigilant and meticulous in haemostasis. Nelson et al suggested the calculation of the patient's blood volume pre-operatively, then using that figure to estimate a safe amount of blood loss. Keeping this posted in theatre can serve to increase the awareness of the safe estimated blood loss and can serve as a method to estimate the need for transfusion (10).

An acceptable method of calculating the estimated blood volume is by multiplying the patient's weight by 75ml/kg for men and 65ml/kg for women. For example, the estimated blood volume for a 60kg female would be 3900mls. Online calculators can be used for this purpose as well (11). For larger and more complex cases, having a larger operating team with 2 or more senior surgeons may be required to reduce the operating time and hence the potential for bleeding. If these cases are sufficiently complex or require a lengthy operation time e.g. bilateral joint replacements, staging of these procedures should be considered as it has been shown to potentially reduce blood loss (12).

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Reversible patient factors should also be considered to help in ensuring the success of their operations. Obese patients (BMI>30) may have an increased risk of bleeding (13-15). This may be due to a longer length of operation time due to the increased technical difficulty of the operation, and an increased surface area of subcutaneous tissues exposed and hence more arterial and venous bleeding points. Although an effort should be made to advise the patient on weight loss, operations should not be withheld on solely on the basis of obesity itself (16).

b) Haematological investigations

Identifying and treating blood dyscrasias, anaemias, and coagulopathies is important however the indiscriminate use of routine coagulation testing in the preoperative setting is probably not helpful. Borderline abnormalities are not uncommon with most being insignificant and the probability of detecting an undiagnosed and clinically important clotting abnormality is rare. This may lead to further unnecessary testing and the delay of surgery. A careful history taking should allow you to identify patients with bleeding disorders. Veen et al (17) have recommended that coagulation testing should be restricted to the below well-defined circumstances.

• Known family history of inherited bleeding disorders. Some common disorders of clotting are listed in table 1.
• Patients with a personal history of bleeding identified by structured history taking or a family history of bleeding.
• Patients with acute illnesses such as sepsis or conditions that can be associated with a coagulopathy such as chronic liver disease.
• Patients on anticoagulants medications.

	Congenital	Acquired	Drugs
Clotting factors	Haemophilia A (factor VIII) Haemophilia B (factor IX) Von Willebrands disease	Liver dysfunction, DIC Vit K deficiency	Warfarin
Platelet count	Haemopoietic disorders	DIC Sepsis ITP TTP Haemolytic Uraemic syndrome	Penicillin Rifampicin Co-trimoxazole thiazide diuretics Heparin
Platelet function	Von Willebrands disease	Uraemia Liver disease	Aspirin Clopidogrel NSAIDS GP 2B/3A inhibitors
Vessel wall	HHT Ehlers-Danlos syndrome	Vasculitis Infection	Steroids

Table 1: common disorders of clotting.

The value of pre-operative haemostasis tests may increase with comorbidity and as such, NICE recommends these tests be performed for all patients who have an ASA grade of 3 or 4 undergoing major operations i.e. grade 3 and 4 operations such as total joint replacements, radical neck dissections, colonic resections and etc (8).

Preoperative anaemia can not only increase the need for blood transfusions during the intra or post-operative period but can also lead to an increase in post-operative mortality and this holds true even if the anaemia is mild (18).

The cause of the anaemia needs to be ascertained (macrocytic versus microcytic anaemia) and then corrected as appropriate for example with the use of iron, folate, B12 supplementation or epoietin injections. 'Low dose' epoetin therapy coupled with acute normovolaemic haemodilution has been shown to be cost-equivalent to the predonation of 3 autologous blood units (19) and also more effective than pre-operative autologous blood donation in reducing the need for allogenic blood transfusion (20).

c) Medications

Another important aspect of pre-operative planning is managing the patients' medications that interfere with haemostasis. Non-steroidal anti-inflammatory drugs (NSAIDs), aspirin, warfarin, clopidogrel and medications which interfere with the coagulation process should be stopped where appropriate.

Aspirin and other NSAIDs irreversibly inhibit platelet cyclooxygenase (COX), diminish thromboxane A2 production, and diminish platelet aggregation, leading to an increase in bleeding time measurement. Increased bleeding risk has been shown with NSAIDs both intra-operatively (21) and post-operatively (22,23) in non-orthopaedic studies but the evidence in orthopaedic surgery has been found to be conflicting (24,25). Aspirin should only be stopped for major procedures where the risk of bleeding is high or patients who are not at high risk of cardiac events as it has been suggested that stopping aspirin leads to a rapid loss of protection and even rebound increased risk of ischaemic event. In such cases, there is evidence to recommend that Aspirin be stopped 5 days before surgery (26). Other different NSAIDs vary in their effect on bleeding time and this does not correlate well with their elimination half-lives thus a general recommendation is to stop most NSAIDs at least 3 days prior to surgery.

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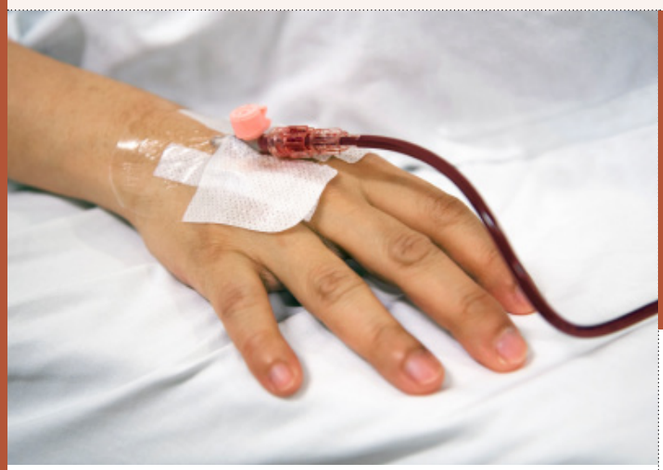

Clopidogrel on the other hand works by irreversibly inhibiting the binding of adenosine diphosphate (ADP) to its platelet receptor, thereby preventing the ADP-mediated activation of glycoprotein IIb/IIIa complex. This in turn inhibits platelet aggregation. Manufacturers and BNF guidelines currently advise that Clopidogrel be stopped 7 days before elective surgery based on studies in cardiothoracic surgery (27,28).

Both aspirin and clopidogrel should be resumed 24 hours post-operatively (29). An exception to the rule are patients who have coronary stents. In these cases Aspirin and Clopidogrel should be continued in the peri-operative period (29,30) but it is advised that surgeons liaise with the cardiology and haematology colleagues and their local trust policies on this matter.

Warfarin is commonly used to prevent thrombi in atrial fibrillation, metallic heart valves and for thromboembolic events such as a deep vein thrombosis or pulmonary embolism. It works by the inhibition of vitamin K epoxide reductase, an enzyme that recycles oxidized vitamin K to its reduced form after it has participated in the carboxylation of several blood coagulation proteins, mainly prothrombin and factor VII (31). Many trusts now have a warfarin bridging plan that gives guidance on how warfarin should be discontinued or reversed to allow for a suitable INR level for the operation to proceed safely (usually at an INR of <1.5).

An oft neglected part of drug history taking is enquiring the patients about any over the counter supplements they may be taking. Gingko (32), feverfew, Asian ginseng, and garlic (33) have all been found to increase the rate of bleeding due to their effect on platelets and can cause significantly more bleeding than would be expected (34).

Intra-Operatively

a) Positioning of patient

The positioning of the patient can help reduce the amount of blood loss. Where possible, the positioning of the operation site above the level of the patient's heart e.g. in the Tredenleburg position (or reverse Tredenleburg depending on site of surgery,) and also avoiding pressure on major veins can help reduce bleeding.

b) Tourniquet use

The use of tourniquets for extremity orthopaedic procedures such as the knees (35), feet (36) and upper limbs (37-39) to reduce intra-operative blood loss is widely accepted. The bloodless field produced also improves visualisation which helps to expedite the procedure. The most commonly used are pneumatic tourniquets which are placed distal to the operation site and inflated to a pressure minimally above that needed to stop arterial blood-flow past the cuff (limb occlusion pressure), usually around 200-300mmHg.

The timing of the tourniquet release is an important consideration in reducing blood loss. In a prospective, randomised study by Lotke et al, the least amount of blood loss occurred in patients who have had the tourniquet released after application of a compressive dressing and a protective splint without immediate continuous passive motion (40). A meta-analysis by Rama et al (35) has also confirmed that early tourniquet release for haemostasis increases the blood loss associated with primary knee arthroplasty, but it also revealed that tourniquet release after wound closure can increase the risk of early postoperative complications requiring another operation such as haematomas and a low (0.17% (41)) but important risk of major vascular damage being missed. Caution also needs to be taken for patients with sickle cell anaemia, as tourniquet use can theoretically lead to a sickle cell crisis (42). However their use has been shown to be safe in such patients provided that optimum acid-base status and oxygenation are maintained throughout anaesthesia (39).

c) Surgical skill

Training, experience and competence of the operating surgeon is crucial in reducing operative blood loss. Meticulous surgical technique including the utilisation of operative approaches through dissection planes that conserve blood and attention to bleeding points can contribute to the reduction of blood loss. The use of minimal access techniques such as arthroscopy in knee surgery e.g. synovectomy (43), minimally invasive hip arthroplasty (44,45) and minimally invasive approaches in spine surgery (46) have been shown to decrease blood loss compared to open or surgery with more extensive approaches although the resultant reduced orientation and steep learning curve have to be taken into account.

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d) Cauterisation devices

The use of diathermy (electrocoagulation) to produce a localised heating effect to seal bleeding vessels is well established and is routinely used in all surgery even though few studies have attempted to quantify the magnitude of blood loss reduced by its use (47).

The two common settings usually available are cut (whereby the water in cells is evaporated creating a cutting effect,) and coagulation (using higher amplitude with and interrupted waveform to coagulate tissues,). In unipolar diathermy the current passes from the electrode through the target tissue and the patient to reach a plate electrode usually placed on the lower limb. In bipolar diathermy the current passes between the two electrodes of the forceps and is generally deemed safer, especially in patients with pacemakers.

There have been recent innovations to cautery devices including argon beam coagulators, microwave coagulating and ultrasonic scalpels. 2 studies have showed that the use of the ultrasonic scalpel in orthopaedic surgery (minimally invasive total hip replacement and posterior spinal fusion) resulted in a less intraoperative blood loss (48, 49). As the ultrasonic scalpel achieves coagulation and tissue dissection at lower temperatures than standard diathermy by converting electrical energy into mechanical energy (oscillation of the blade,), its use could also reduce tissue damage with less smoke production with the added advantage of no electrical energy being passed through the patient (useful in patients with implanted devices such as pacemakers,).

e) Anaesthetic techniques to limit blood loss:

• *Hypotensive anaesthesia*

Hypotensive anaesthesia is the use of pharmacological agents to deliberately induce hypotension in a controlled manner to reduce blood loss and improve the surgical field, with a target reduction of systolic blood pressure to 80-90mmHg, a mean arterial pressure (MAP) to 50-65mmHg or a 30% reduction of baseline MAP (50). A meta-analysis of randomised controlled trials by Paul JE et al showed that the use of deliberate hypotension for total hip arthroplasty, and spinal fusion resulted in a significant reduction in blood loss (51).

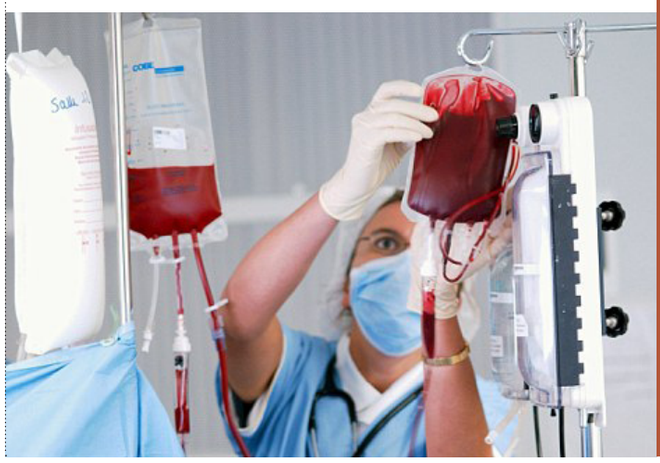
Hypotensive anaesthesia can be achieved via several different agents and they can be divided into primary agents (agents that can be used by themselves) and secondary agents (those that are used adjunctively with other agents to reduce the other agents' toxicity).

Primary agents include regional anaesthetics (such as spinal and epidural anaesthesia.), inhalational anaesthetics (such as Halothane, Sevoflurane and Isoflurane), sodium nitoprusside (SNP), prostaglandin, and adenosine. Secondary agents include ACE inhibitors and Clonidine (51). Some agents can be used alone or adjunctively such as calcium channel antagonists and beta-blockers.

Each agent has its own advantages and disadvantages and thus selection should be tailored to the patient and also the anaesthetist's experience with the agent. For example, SNP has the advantage of easy titration, a rapid onset and offset and also increases cardiac output. However there is a risk that it may induce coronary ischaemia via coronary steal syndrome, increase intracranial pressure, and the possibility of cyanide (a metabolite of SNP) toxicity in patients with hepatic dysfunction or B12 deficiencies (8).

Hypotensive anaesthesia however is contraindicated in hypoxaemia, either global or localised to a particular major organ and also where there is loss of autoregulation (10). Thus a history of cardiovascular disease (e.g. congenital heart disease, coronary artery disease and congestive heart failure), renal and liver dysfunction or severe peripheral claudication would suggest that hypotensive anaesthesia would not be suitable as the organs are less likely to have adequate perfusion during the period of hypotension. Likewise, adequate organ perfusion would also be compromised in patients with hypovolaemia or severe anaemia and unless this is corrected, they should not undergo hypotensive anaesthesia.

Hypotensive anaesthesia relies on the autoregulation of the organs to ensure their adequate perfusion. Thus patients with increased intracranial pressure, a history of significant cerebrovascular disease or uncontrolled hypertension would indicate a loss of autoregulation and thus are also unsuitable candidates for hypotensive anaesthesia.



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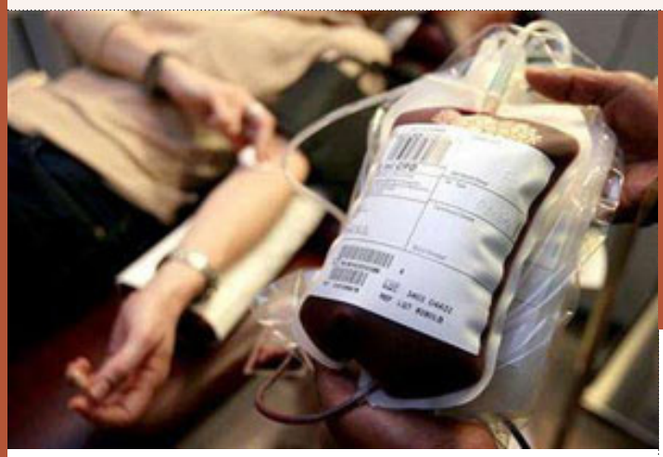

• *Haemodilution*

By making blood more dilute than normal with the use of intravenous fluid, fewer red blood cells are lost when bleeding occurs. In elective surgery, acute normovolaemic haemodilution can be performed in which some of the patient's blood is removed pre-operatively and replaced with fluids, and thus maintaining normal volume. The patients' own blood, containing all the necessary clotting factors and platelets than can be returned to them post-operatively. It can be effective in reducing the need for allogenic transfusions especially in surgeries where massive blood loss occurs such as liver resections (52, 53).

• *Maintenance of normothermia*

Induced hypothermia has been shown to be protective in patients who present with cardiac arrest and stroke but spontaneous hypothermia secondary to major trauma can lead to greater transfusion requirements and worse outcomes (54).

Both types of hypothermia however lead to coagulopathy as it decreases the enzymatic activity of clotting factors and also the number and function of platelets (55), the effect of which becomes clinically measurable when core temperature drops below 33°C. Clotting times can differ as much as 3 times when measured at a temperature of 22 degrees and 37 degrees (55).

Thus it is important for the surgical team to maintain the body temperature of the patient with measures including the use of heated blankets or mattresses, use of warmed intravenous and irrigation fluid, and limiting the area of skin exposed.

f) Pharmacological agents:

Pharmacological aids for haemostasis can be divided into topical and injectable agents. Topical agents come in a variety of forms and include surgical adhesives such as thrombin, collagen and fibrin glues. Fibrant sealants mimic the final stage of the coagulation cascade where fibrinogen is converted to fibrin, one barrel delivers the fibrinogen, fibronectin and factor XIII while the other delivers the thrombin, calcium and aprotinin required for the reaction to occur resulting in an instantaneous formation of a clot.

Desmopressin (DDAVP) can be administered intravenously, subcutaneously or intranasally. It works by increasing the release of both Factor VIII and von-Willebrand factor from endothelial storage sites and accelerating the activation of factor X (factor VIII) and mediating platelet adhesion to the vascular endothelium (vWF). Although useful in patients with acquired and congenital platelet dysfunction, its usefulness in patients with normal coagulation function is not clear. Some studies (56,57) have shown that although it does not reduce intra-operative blood loss it reduces post-operative transfusion rates while others have shown that it does neither (58,59). A review by Erstad et al concluded that desmopressin lacked efficacy (60).

Aprotinin works by inhibiting the enzymatic formation of several serine protease enzymes e.g. factor XII, plasmin, kallikrien and trypsin, and preserves platelet formation. The majority of the evidence for its use comes from cardiac surgery (61) but it has also been found to decrease blood loss and transfusion amount in major orthopaedic surgery (62). However, aprotinin is associated with potential serious side effects such as allergic reactions (5% if patient has had previous exposure in less than 6 months) (8) and renal toxicity.

Tranexamic acid is another antifibrinolytic agent, an inhibitor of plasminogen and plasmin, which has been shown to be successful in reducing bleeding. Meta-analyses by Zufferey et al and Ker et al both showed that tranexamic acid significantly reduces the need for blood transfusions. Furthermore, although tranexamic acid has been associated with some adverse effects, these have not been serious enough to warrant its disuse (63,64).

g) Blood products:

In cases of severe haemorrhage, the use of blood products may be necessary to reduce further bleeding by correcting coagulopathies due to the loss of clotting factors. Here we summarise some of the commonly used blood products.

• *Platelets*

Platelets can be given where there is a severe platelet deficiency or platelet dysfunction. They can be given prophylactically or as a treatment during haemorrhage. A count of less than 10X109/L in any patient is indicative for platelet transfusion, however in patients undergoing surgery the count should be above 50x109/L and around 100x109/L for neurosurgery (65).

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• *Fresh frozen plasma*

Fresh frozen plasma (FFP) contains many different factors. Factors V and VIII are usually unstable. It is used in situations where coagulation factors and plasma proteins are deficient or have reduced function. It can be given with massive transfusion, states with consumptive coagulopathy such as DIC and sepsis and reversal of warfarin where prothrombin complex is unavailable (66).

• *Cryoprecipitate*

Cryoprecipitate has factors VIII and vWF, fibrinogen and factor XIII. It is useful in DIC or massive haemorrhage (66).

• *Prothrombin complex concentrate*

Prothrombin complex concentrate (commonly known by its trade names Beriplex or Octaplex) is a combination of factors II, VII, IX and X. It is used commonly for the reversal of warfarin associated with significant haemorrhage (67) or patients who have developed coagulopathy secondary to massive blood loss e.g. gastrointestinal bleeding. Several guidelines now recommend the use of PCC due to its effectiveness (quote) however due to its cost, its use has to be discussed and approved by a senior haematologist.

Post-Operatively

During the post-surgical period, vigilance needs to be maintained in order to detect continued bleeding which may not be visible. Attention should be given to drain amounts and post-surgical blood test results. Any existing deficiencies and coagulopathies should be corrected.

Conclusion

There are many steps that the surgical team can take to minimise the amount of blood loss that occurs in surgical patients and the utilisation of several techniques in combination is likely to be more effective than any single method alone with not only the surgeon but also the anaesthetist playing key roles.

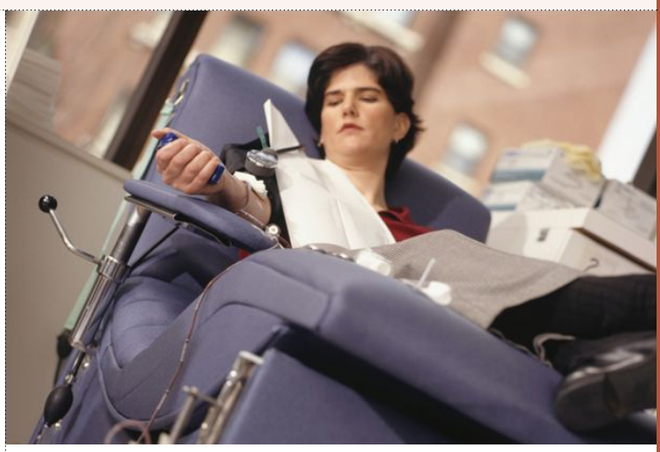
Extended Matching Questions

Theme: reversal of clotting abnormalities

Options

a) Vitamin K
b) Prothrombin complex concentrate
c) Fresh frozen plasma, cryoprecipitate and platelets
d) Fresh frozen plasma and platelets
e) Desmopressin (DDAVP)

For each of the situations below, select the single most appropriate produce to correct the coagulation abnormalities from the options listed above. Each option may be used once, more than once or not at all.



1. A 74 year old male present to A&E having collapsed shortly after developing abdominal pain radiating to his back. His medical background includes hypertension and atrial fibrillation treated with warfarin. On arrival to A&E you find the patient to be clammy and shut down with a respiratory rate of 30, blood pressure of 90/44, pulse rate of110 and with a pulsatile mass in his abdomen. A FAST scan performed in A&E confirms a 6.5 cm abdominal aortic aneurysm with likely rupture. His INR comes back from the labs as >10.

2. A 50 year old male presents with a small volume of rectal bleeding. Initially his haemoglobin is 13.0 g/dL, pulse rate is 80 and blood pressure is 130/78. On day one of his admission he begins to lose large volumes of fresh blood per rectum. Upper and lower GI endoscopy are normally and a laparotomy is required to diagnose and treat a bleeding jejunal diverticulum. During this episode his haemoglobin drops to 4.0 g/dL and he receives a transfusion of 10 units of red cells.

3. A 57 year old female presents with jaundice, fevers and right upper quadrant pain. White cell count is elevated at 25x109/L. A diagnosis of ascending cholangitis is made and broad spectrum antibiotics are started. Over the next 24 hours the patient becomes more hypotensive and tachycardic and does not respond to fluid boluses. She is transferred to ITU and placed in invasive monitoring with inotropic support. After a further 24 hours on the ITU she is noted to be bleeding around her central venous line and peripheral canulae sites. Fibrinogen is found to be 0.8 g/L.

Theme: clotting disorders

Options

a) Aspirin use
b) Liver failure
c) Haemophilia
d) Warfarin use
e) Von Willebrand's disease

MINIMISING BLOOD LOSS IN SURGERY

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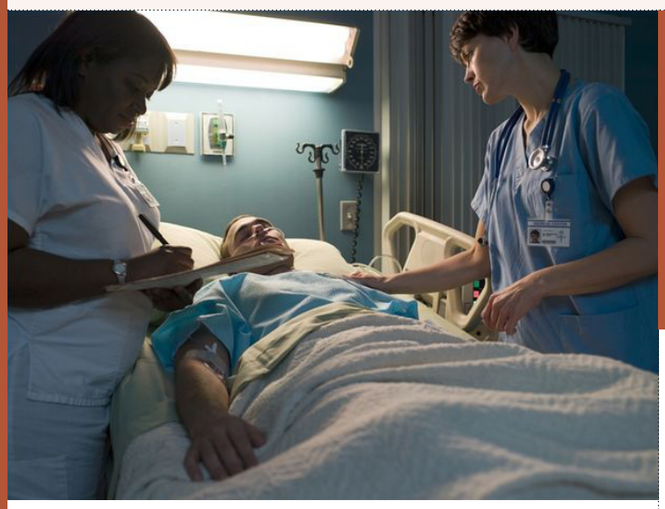

For each of the situations below, select the single most likely diagnosis from the options listed above. Each option may be used once, more than once or not at all.

4. Prolonged bleeding time

5. Prolonged PT, APTT and decreased platelets

Answers

1. b)
Prothrombin complex concentrate contains factors II, VII, IX and X. Its use is indicated in instance of congenital or acquired deficiency of these factors. When used for reversal of warfarin in cases of haemorrhage its effect is fairly rapid. Where not available FFP can be use to a lesser effect. When there is no haemorrhage vitamin K can be used to reduce the prothrombin time.

2. d)
A massive transfusion is defined as a replacement of a patient's circulating volume within 24 hours. This can lead to consumption of clotting factors and platelets. Replacement with FFP and platelets should be based on laboratory values and not as a prophylactic measure.

3. c)
Disseminated intravascular coagulation is a consumptive coagulopathy causing low platelet and fibrinogen count and an increase in PT, APTT and D-dimers. Management principles are to treat the underlying cause and provide haematological support in the form of FFP, platelets and cryoprecipitate.

4. a)
Aspirin reduces the functionality of platelets best reflected by an increased bleeding time.

5. b)
End stage liver failure will cause a decrease in synthetic function leading to reduced amount of clotting factors and functional platelets.

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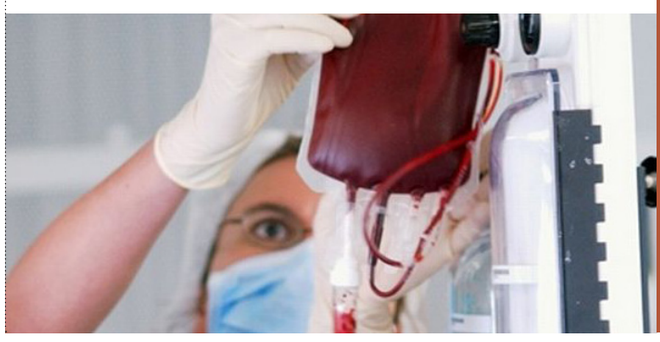
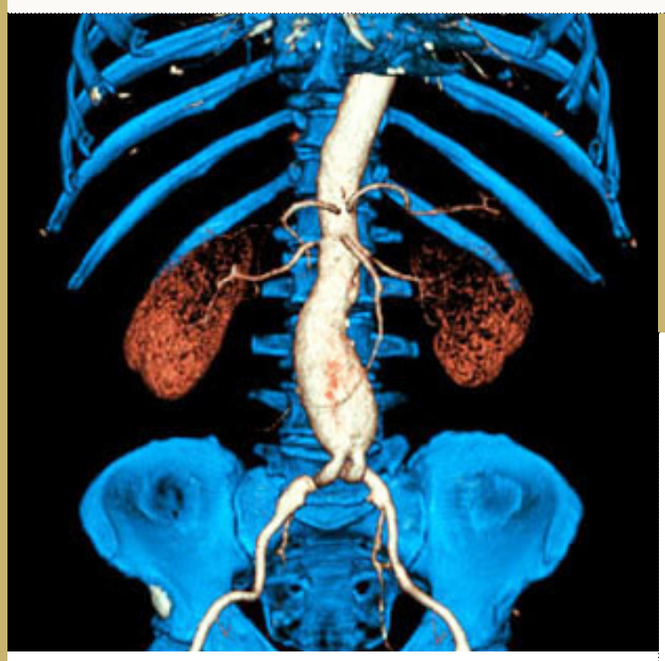


ABDOMINAL AORTIC ANEURYSM (AAA)

R Dickson-Lowe



Abstract

This review article covers clinical examination, aetiology and pathology and both the management of non-ruptured and ruptured AAA. This emergency presentation of AAA and the journey through to theatre is outlined. Important trials, the screening process and secondary prevention methods are integral to AAA management and hence comprise the article's backbone. In latter parts, a clinical vignette will help to consolidate the knowledge gained from the former parts of the article.

Keywords: AAA, EVAR

Definition of AAA

An aneurysm is a pathological, permanent, segmental dilatation of an artery to >1.5 times its normal diameter. Thus an AAA is dilatation of the aorta >3cm in diameter. (1)

Anatomical location of AAA

90% of aortic aneurysms affect the infra-renal abdominal aorta, 8% the thoracic aorta and 2% both (thoracoabdominal) with 30% involving the iliac arteries. (2,3)

Classification of aneurysms:

- **True aneurysms:**

dilatation of an artery involving all layers of arterial wall.

- **False aneurysms:**

a defect in an arterial wall, with blood outside the arterial lumen, surrounded by a capsule of fibrous tissue or compressed surrounding tissues.

Abdominal Aortic Aneurysm (AAA)
General Surgery

Epidemiology & Aetiology of AAA

Reported incidence is 4.9-9.9% (5-7). 6000 men die from a ruptured abdominal aortic aneurysm in England and Wales every year which represents about 2% of all deaths in men aged greater than 65. (8,9)

Aetiology	Example
Degenerative	Atherosclerotic
Inflammatory	Variable degrees of inflammation of aneurysm wall
Congenital	Berry aneurysm of the cerebral circulation
Mycotic	Infected emboli in endocarditis
Infective	Syphilitic aneurysms
Traumatic	Iatrogenic, blunt or penetrating injury
Connective tissue disorders	Marfan syndrome, Ehlers-Danlos syndrome, tuberous sclerosis, Takayasu's arteritis; Post-stenotic.

Table 1: AAA aetiology (1)

Risk Factors for AAA

The risk factors are complex, with contributions from both familial susceptibility and degenerative components. (10)

Modifiable	Non-modifiable
Age – incidence increases with age (6% of 65-74 men; 9% of >75 men). (3)	***Smoking*** – The association between smoking and AAA formation, aneurysm growth, and rupture risk suggests a role for smoking cessation. Smokers are seven times more likely to develop aneurysmal dilatation. Stopping smoking 4-8 weeks prior to surgery can minimise complications. (3,10-13)
Sex – affects males more than age-matched females (6:1) but women have an increased risk of rupture. (3,11,14)	***Hypertension*** – The UK Small Aneurysm Trial found an weak association between hypertension and risk of rupture, with a higher blood pressure having a higher rupture rate. (14)
Familial risk – 12-fold increased occurrence in 1st degree relatives. (1,3)	***Diabetes*** – diabetes may protect against the formation and growth of AAA but increases the risk of rupture. (3)
Ethnicity – prevalence is greater in white males than of Asian origin and Black origin. (3)	

Table 2: AAA Risk factors.

ABDOMINAL AORTIC ANEURYSM (AAA)

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Natural history of AAA

Mechanical factors are very important in progression of aneurysm. The Law of Laplace states that increased radius requires increased wall thickness to accommodate a stable wall tension at any given pressure. The wall tension in this case represents the muscular tension on the wall of the vessel. If an aneurysm forms in a blood vessel wall, the radius of the vessel has increased. This means that the inward force on the vessel decreases, and therefore the aneurysm will continue to expand until it ruptures. (13)

The natural course is one of progressive enlargement, and maximum aortic diameter is the strongest predictor of aneurysm rupture. (12,15)

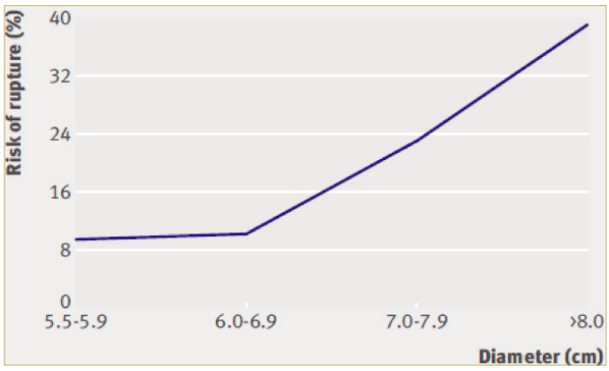


Figure 1: Association between maximum aortic diameter and annual risk of rupture (1,12)

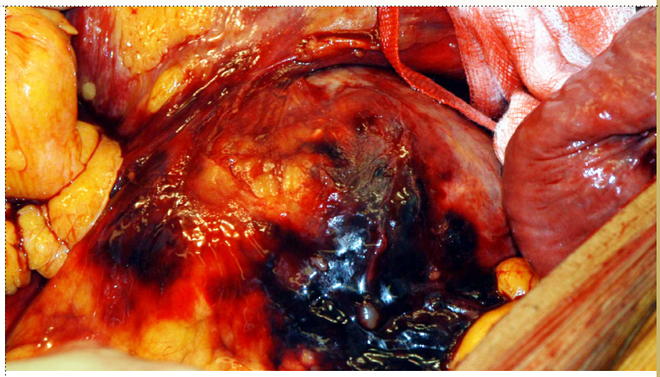
Not all growth is linear and there may be periods where expansion accelerates or stops altogether. (3) The risk of rupture can be as high as 25% per year for aneurysms with diameters greater than 6cm. (14) Generally AAA greater than 5.5cm have an annual rupture rate of about 10% and AAA <5.5cm have an annual rupture rate of 1%. (2) These thresholds aim to balance the rupture rate and risk of operative repair and lead a patient into screening or MDT meetings to discuss repair.

Presentation of AAA

• Asymptomatic
- An expansile pulsatile mass on abdominal examination
- Incidental finding on radiological examination
- Screening

• Pain
- Central abdominal and may radiate to the back or groin. Symptomatic aneurysms are usually expanding rapidly or leaking and need prompt repair. (2)

• Embolisation
- Thrombi/atheroma from the aneurysm may give rise to acute limb ischaemia.



• Rupture
- The patient presents with a hypotensive episode or collapse associated with severe central abdominal pain radiating to the back and the flanks. A pulsatile expansile mass is not always palpable. Rupture commonly occurs at the postero-lateral wall causing a retroperitoneal haematoma.

• Other
- AAA can rarely present with acute thrombosis or fistulisation into surrounding structures (e.g. IVC, duodenum, terminal ileum).

Medical management of modifiable risk factors

• Smoking cessation

• Antiplatelets - Low dose aspirin reduced significant coronary events and cardiovascular mortality. (1) European Society for vascular surgery guideline suggests starting low dose aspirin on diagnosis of AAA and continuing indefinitely. (16)

• Anti-hypertensives and statins as secondary prevention for cardiovascular disease.

• Diabetes management

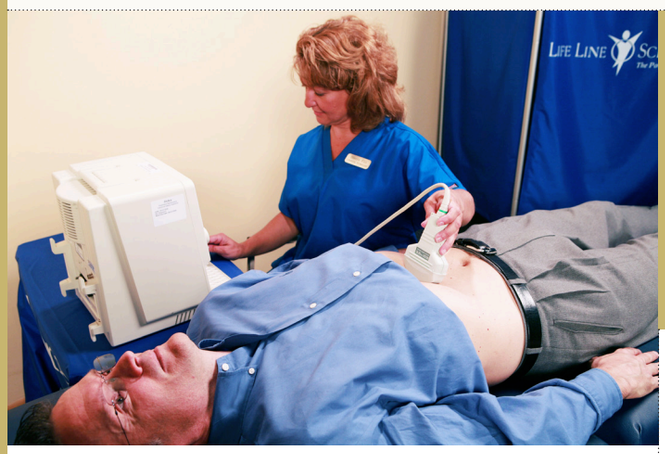
Elective AAA Repair

MDT improves decision-making and patient progress along care pathways in cancer and this principle has been applied to the management of AAA patients. (17) The National Abdominal Aortic Aneurysm Screening Programme (NAAASP) has set a deadline of 8 weeks from referral to treatment for all large AAA (over 5.5.cm in men) (9).

NICE Guidance for Endovascular Aortic Aneurysm Repair stated that "to identify patients for whom EVAR was appropriate it is necessary to take account not only of the size of the aneurysm but also of other factors such as physiological measures of the person's fitness for surgery and aneurysm morphology, and patient choice." (18)

ABDOMINAL AORTIC ANEURYSM (AAA)

R Dickson-Lowe



Pre-operative investigations for elective AAA repair

Routine pre-operative investigations are required such as routine bloods and a CXR but further assessment may involve lung function tests and echocardiography. Cardiopulmonary exercise testing (CPEX/CPET) has rapidly become a standard for major operations. This assesses a multitude of factors on an exercising patient to test anaerobic tolerance and is a good indicator of fitness for a general anaesthetic.

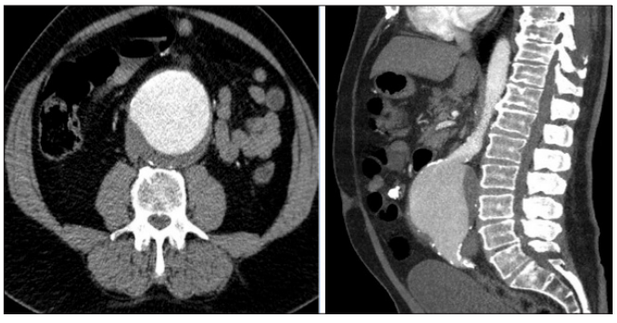


Figure 2: Shows AAA on CT scan, (A) Axial slice (B) Oblique slice.

There are 2 approaches for operative repair of AAA >5.5cm. These are open surgical repair (OSR), practiced since 1951 and Endovascular Aortic Repair (EVAR), practiced since 1986. OSR is performed for patients with AAA >5.5cm and fit for major open surgery. EVAR can be used for patients who are anatomically suitable for stenting and for those unfit for open repair. Long term complications and co-morbidities play an important role in the decision for type of repair. In the emergency patient the OSR is commonly used but some centres have initiated emergency EVAR.

Abdominal Aortic Aneurysm (AAA) General Surgery

The Open Repair

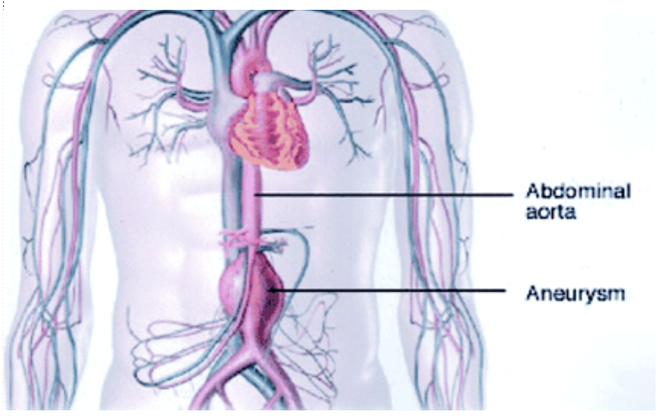
Positioning: supine, crucifix

Incision: Midline laparotomy

Procedure: Enter abdominal cavity and retract bowel and duodenum to the right. Divide posterior peritoneum and dissect around aneurysm. Define proximal neck of aneurysm and distal extension, which may be progress into the iliacs. Administer IV heparin dependent upon weight (usually 5000 units). Apply aortic clamps (distal then proximal) and secure before opening aneurysm sac. Oversew or Ligaclip lumbar arteries and IMA if they are patent. Repair is made with either inlay 'tube/straight' graft or 'Y/trouser'-graft made from Dacron or polytetrafluoroethylene (PTFE) dependent upon whether the iliac arteries are involved. If it is not possible to attach to the iliac vessels then bilateral groin dissection may be required for bypass onto femoral vessels, tunnelling the limbs of the graft under the inguinal ligaments. A synthetic, monofilament, nonabsorbable polypropylene suture is used to make the proximal and distal anastomoses. Haemostasis should be achieved prior to closure.

Closure: The aneurysm sac is closed over the graft to prevent fistulisation. Standard mass closure is used for the laparotomy wound. A laparostomy with secondary closure (24-48hrs later) may be necessary to prevent abdominal compartment syndrome.

Post-op: Patients must go to High Dependency Unit/Intensive Care Unit following theatre. (2,3)



ABDOMINAL AORTIC ANEURYSM (AAA)

R Dickson-Lowe

Morbidity & Mortality (to be included on the Consent Form) (1)

• Immediate complications:
- Haemorrhage (primary)
- Distal embolisation (ischaemic limb, trash foot)

• Early:
- Haemorrhage (reactive, secondary)
- MI
- Renal failure (especially if proximal clamp above renal vessels)
- MOF, DIC, ARDS
- Colonic ischaemia
- Pneumonia
- Stroke
- DVT/PE

• Late:
- Graft infection (causing graft thrombosis, false aneurysm or rupture)
- Aorto-enteric fistula
- Anastamotic aneurysm

Elective infra-renal repair has a peri-operative mortality rate of about 5% but returns the patient to a life expectancy equal to their age matched peers. Mortality overall for rupture is about 75% where 50% make it to the hospital alive and then 50% of those survive operation (2).

Endovascular Aortic Repair (EVAR)

NICE Guidance on EVAR concludes that "Current evidence on the efficacy and short-term safety of stent–graft placement in abdominal aortic aneurysm appears adequate to support the use of this procedure provided that the normal arrangements are in place for consent, audit and clinical governance." (20)

Endovascular repair involves relining the aorta using an endograft, which is an exoskeleton of metallic stents over a fabric lining, under fluoroscopic guidance via a catheter through a femoral arteriotomy. The 'stent-graft' is then positioned, below the renal vessels, such that when the stents are expanded the aneurysm is excluded from the circulation and thus is no longer at risk of rupture.

Stents can be straight or Y-shaped, with one or two iliac limbs. EVAR requires detailed pre-op assessment with calibrating angiograms, discussed at Vascular MDT meetings, to assess graft proximal fixation point (minimum of 1-1.5cm below the renal arteries) and, if necessary, pre-op embolization of any vessels (e.g. lumbar vessels) feeding into the aneurysm sac.

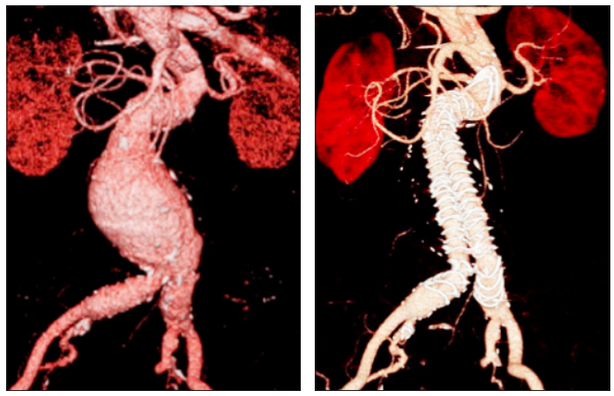


Figure 3: 3D reconstruction of (A) AAA (B) AAA with endovascular EVAR stent in situ.

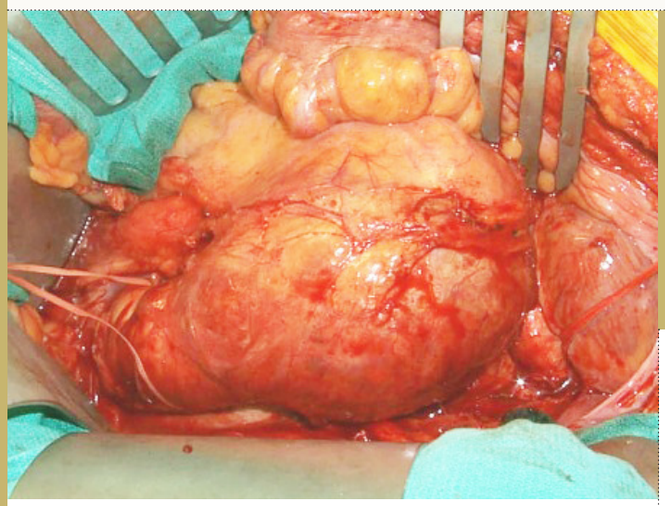
Principles of Procedure:
A bilateral groin dissection is used to expose the femoral vessels and gain control. Arteriotomies are made and guidewires are introduced through the femoral vessels under fluoroscopic control. The Endovascular stent is passed over guidewire, in the most pliant femoral artery to sit below the renal arteries above the aneurysm. A balloon is used to release the stent at infrarenal aneurysm neck, and the main body is deployed with its trouser leg deployed in the corresponding iliac artery above the bifurcation. The contralateral guidewire is then retracted and inserted into the main body of the graft. The trouser limb is passed over the contralateral guidewire and inserted into the main body of the graft. It is the deployed within the contralateral iliac and must sit above the iliac bifurcation. The graft can then be stretched to fit the aneurysm walls using a balloon catheter and a final check is performed with fluoroscopy. (19)

Advantages:
- *Avoids large chest/abdominal incisions*
- *No aortic cross-clamping and thus less physiological insult (21)*
- *Reduced blood loss*

Disadvantages:
- *High cost*
- *Annual CT surveillance*
- *'Endoleak' (two main types are type 1 with a leak at the proximal or distal attachment site and type 2 with a leak from vessels feeding into the aneurysm sac).*
- *Limb occlusion*
- *Distal embolization*
- *'Stent-graft' migration*
- *Femoral false aneurysm (at groin puncture site)*
- *Renal failure (contrast nephropathy)*

ABDOMINAL AORTIC ANEURYSM (AAA)

R Dickson-Lowe



NICE states that patients should understand risks of endovascular leaks, the possibility of secondary intervention and the need for lifelong follow-up. (18)

Morbidity and Mortality

The procedure carries a lower operative mortality but a cumulative annual 10% re-intervention rate for device failure or slippage or continued aneurysm expansion is required. Reduced infection, increased mobility, and shortened length of hospital stay have also been described. (1) EVAR confers to survival benefit over conservative management in patients unfit for open surgery. (2)

The two big trials involving EVAR are:

• EVAR trial 1 compared long term results of OSR with EVAR in large aneurysms. It concluded that although EVAR has a lower 30-day operative mortality than OSR, the long-term results are uncertain. Compared with open repair, EVAR offers no advantage with respect to all-cause mortality and is more expensive leading to a greater number of complications and reinterventions. However it does result in a 3% improved aneurysm-related survival. The continuing need for interventions mandates ongoing surveillance and longer follow-up of EVAR for detailed cost-effectiveness assessment. (22)

• EVAR trial 2 identified whether EVAR improves survival compared with no intervention in patients unfit for open repair of large aortic aneurysms. EVAR had a considerable 30-day operative mortality in patients already unfit for open repair of their aneurysm. EVAR did not improve survival over non intervention and was associated with a need for continued surveillance and reinterventions, at substantially increased cost. Ongoing follow-up and improved fitness of these patients is a priority. (23)

Where should patients have their procedures?

There is evidence that larger volume centres achieve better outcomes following AAA repair with reduced length of stay and improved survival after complications. (24) The Vascular Society recommends that centres undertaking AAA repair should perform a minimum of 100 elective interventions (Endovascular repair (EVAR) or Open surgical Repair (OR)) in a 3 year period.

Screening

The aim of population screening is to identify aneurysms before rupture, allowing elective repair of large aneurysms or surveillance of small aneurysms. Screening may also help optimise medical treatment of patients with AAA by making a positive diagnosis and driving aggressive management of risk factors. (1)

Ultrasound is the preferred method as it is reliable, non-invasive, and readily available way to establish aortic diameter, with sensitivity and specificity approaching 100%. (25) The value of this modality is highly dependent upon the user.

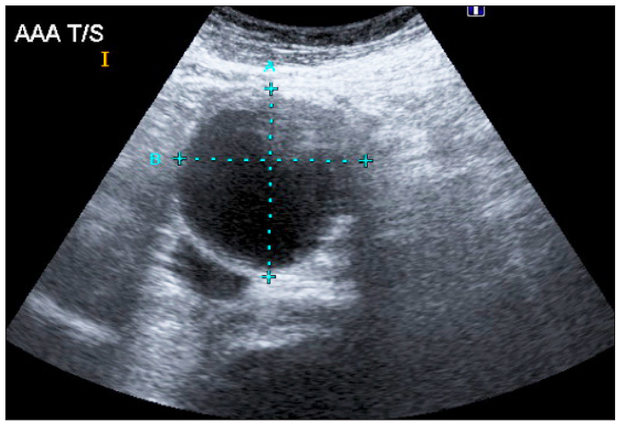


Figure 4: AAA displayed by ultrasound scan.

Evidence suggests that screening is beneficial in men over 65. Screening is associated with a significant decrease in AAA rupture and aneurysm related mortality for men aged 65-79 but not women. (5,11,25-27) There is also further evidence to suggest a reduced aneurysm related mortality from long-term follow up and a trend towards reduced all-cause mortality, presumably as a result of risk factor management. (28)

ABDOMINAL AORTIC ANEURYSM (AAA)

R Dickson-Lowe

The two big screening trials are:

• *Multicentre Aneurysm Screening Study (MASS) - The Multicentre Aneurysm Screening Study was designed to assess whether or not such screening is beneficial. It concluded that there is benefit from screening for abdominal aortic aneurysms. (5)*

• *UK Small Aneurysm Trial - The UK Small Aneurysm Trial was a multicentre, randomised controlled trial to investigate whether early elective open surgery or regular ultrasound surveillance is the best management policy for small abdominal aortic aneurysm (AAA). It concluded that small AAA less than 5.5cm in diameter can be monitored safely under surveillance until they reach 5.5cm, become tender or grow by more than 1cm/year. (14)*

Clinical Vignette – Structured questions

An 81 year old gentleman, previously fit and well came into A&E with severe central abdominal pain radiating to the back and flanks. He had collapsed at home, prior to his wife calling 999. His blood pressure was steady with a systolic of 100-110, mild tachycardia 90-100bpm. His previous medical history stated hypertension treated with amlodipine and hypercholesterolaemia treated with simvastatin. No previous operations. He was an ex-smoker of 40 pack years who had given up 25 years ago.

What are the differential diagnoses? (2)

• Abdominal Aortic Aneurysm
• Renal or ureteric pain (and in the College of Emergency Medicine 'Renal Colic' guidelines to be ruled out in those with ureteric colic over the age of 60) (29)
• Pancreatitis
• Perforated DU
• Sciatica
• Osteoporotic vertebral collapse
• Myocardical infarction

At the bedside, following your history, how would you confirm your diagnosis of ruptured AAA?

He was seen by an A&E SpR who confirmed an AAA via clinical examination of an expansile and pulsatile mass centrally in the abdomen and a FAST/Aortic USS scan.

After clinical suspicion and a confirmatory examination and aortic scan at the bedside, what would be your next course of action?

Call the General Surgeon on call who should liaise with a vascular surgeon immediately. If there are any delays do not hesitate to call the vascular surgeon on call.

What would be your investigation/management/actions following this phone call (or at the same time by one of your colleagues)?

In ruptured AAA there is often insufficient time for full pre-operative investigations. Minimal requirements for an emergency aneurysm repair include IV access with two large bore cannulas, basic bloods (mentioned above), cross-match and portable CXR. CT should only be performed if there is doubt about the diagnosis and if the patient remains stable. Do not give excessive intravenous fluids as this can increase the blood pressure and risk of further bleeding. (1)

As the Vascular SpR arrived to make an assessment, the patient was in the CT scanner, which confirmed ruptured AAA.

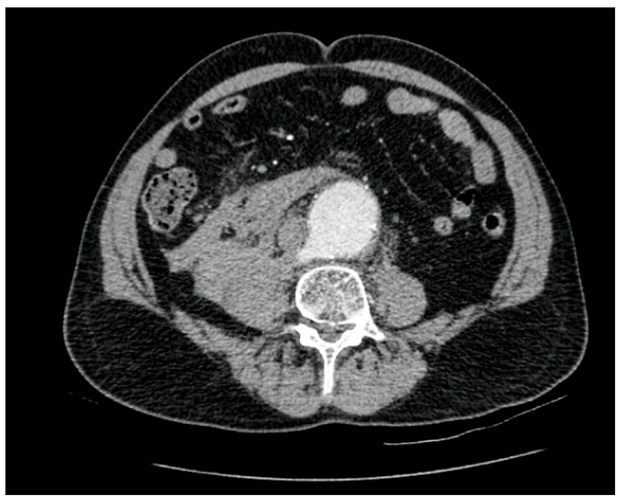
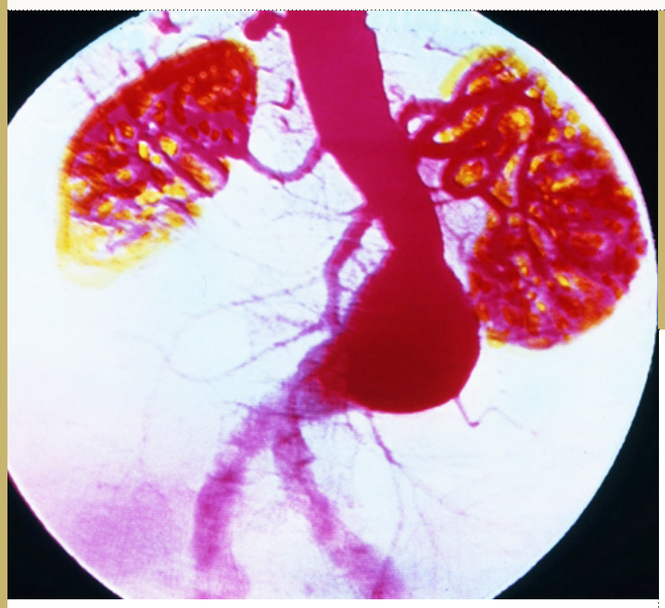


Figure 5: Transverse view of postero-lateral (to the RIGHT) rupture of infrarenal AAA on CT.

The patient was delivered to the anaesthetic room with a GCS of 15/15 that rapidly deteriorated as he lost output (cardiac arrest) during initiation of anaesthetic. Thus the abdomen was rapidly opened to access the retroperitoneum and establish the aneurysm neck in order to apply the cross clamp to stop the bleeding that was causing the cardiac arrest/hypovolaemic shock. Once this cross clamp was on, the haemorrhage stopped and control was gained. The operation saw >1000mL blood loss requiring the 'massive transfusion protocol' (including x-matched blood, fresh frozen plasma and platelets) in addition to cell-saver blood. It also required that the patient's abdomen was left open (laparostomy) to prevent compartment syndrome.

ABDOMINAL AORTIC ANEURYSM (AAA)

R Dickson-Lowe



Where is the best place to send this patient post-operatively?

All patients following a ruptured AAA repair MUST go to an Intensive care unit.

After 48hrs the patient returned to theatre for closure of his abdomen and subsequently woken up. He developed right sided weakness which persisted and was diagnosed as a stroke. He developed acute renal failure (requiring haemofiltration) and also heparin induced thrombocytopaenia (HIT), whilst on the filter, requiring the use of danaparoid in lieu of enoxaparin/heparin. The patient spent 14 days on ICU and returned to the ward to spend a further 25 days. It was suspected that he would need rehabilitation via an intermediate care bed but he made a significant recovery with the ward MDT (Physio, OT, SALT teams and was discharged home on day 39.

Multiple Choice Questions

1. Who's Law applies when considering risk of rupture of a AAA?
a. Henry's Law
b. Courvoisier's Law
c. Laplace's Law
d. Boyle's Law
e. Saint's Law

2. Risk factors for atherosclerotic AAA include all but one of the following
a. Age
b. Family History
c. Smoking
d. Syphilis
e. Being male

3. After resuscitation using ABCDE, you confirm AAA rupture, what is your first course of action?
a. Discuss with ED consultant
b. Discuss with Radiology consultant
c. Arrange a CT scan
d. Take bloods
e. Discuss with a vascular surgeon

4. Why is the remnant aneurysm sac closed over the synthetic Dacron/PTFE graft following anastomosis with adequate haemostasis prior to closure?
a. To protect the graft from injury
b. To prevent aorta-enteric fistulae
c. To tamponade any bleeding from the anastomoses
d. To prevent collections
e. In case the graft fails

5. The aorta is crossed anteriorly by:
a. Neck of pancreas
b. 4th part of the duodenum
c. RIGHT renal vein
d. Splenic vein
e. Adrenal gland

Answers

1. (c) Laplace's Law.
It states that increased radius requires increased wall thickness to accommodate a stable wall tension, at any given pressure. Thus with an increased radius, the inward force on the vessel decreases, and therefore the aneurysm will continue to expand until it ruptures. Henry's and Boyle's Laws are gas laws, Courvoisier's Law is an indicator for pancreatic cancer, and Saint's triad (rather than Law) describes cholelithiasis, hiatus hernia and diverticular disease in one patient.

2. (d) Syphilis.
The question asks about atherosclerotic aneurysm of which all the others are risk factors. Syphilis can be, albeit rare, an infective cause of AAA.

ABDOMINAL AORTIC ANEURYSM (AAA)

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3. (e) Discuss with a vascular surgeon.

Regardless of whether your diagnosis is correct, this patient has needed resuscitation and you suspect a ruptured AAA and needs urgent discussion with a vascular surgeon as the patient will undoubtedly need immediate surgery. Even if the diagnosis is unclear it is better to suspect than to miss it.

4. (b) To prevent aorto-enteric fistulae.

The graft will be retroperitoneal and once fixed in place with a good anastomosis will not need protection, should not need tamponading as haemostasis should be achieved prior to closure and it will not prevent collections from occurring.

5. (d) Splenic vein.

The aorta is crossed anteriorly by splenic vein, body of pancreas, 3rd part of the duodenum, and the left renal vein.

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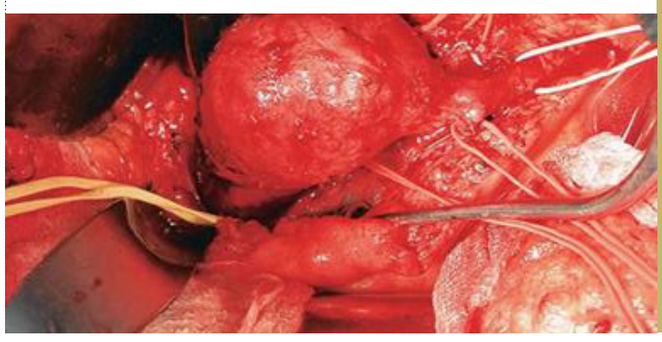
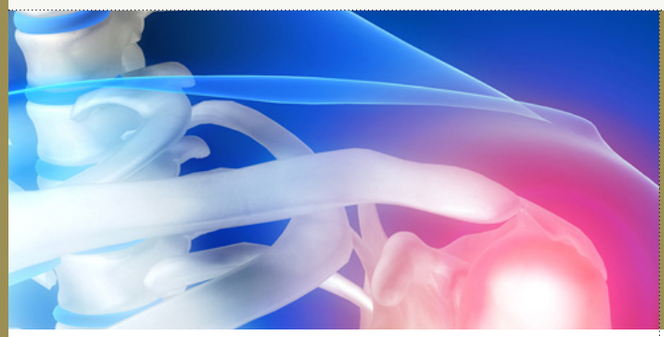


REVIEW: ANTERIOR SHOULDER DISLOCATION

R Gogna



Review: Anterior Shoulder Dislocation
Trauma & Orthopaedic Surgery

Abstract

The shoulder is the most commonly dislocated joint in the body, with the vast majority of dislocations occurring anteriorly. Young, active males are the group with the highest risk of dislocation and recurrent dislocation. It is important to perform a thorough clinical assessment as nerve and vessel injuries, rotator cuff tears and greater tuberosity fracture can frequently accompany dislocation. There are several methods of reduction described in the literature including the Stimson technique, scapular manipulation, external rotation method, Milch technique and traction-countertraction. Each has its own merits and should be attempted after taking into account patient factors, staffing and equipment availability. With regards to post-reduction care there are two distinct groups; the young active patient who often requires surgical intervention to prevent the high incidence of recurrent dislocation, and the slightly older population who respond well to cuff strengthening exercises and rehabilitation. Despite early arthroscopic Bankart repair, instability and recurrent dislocation may still occur, requiring the need for more complex procedures to be performed.

Keywords: Shoulder, Glenohumeral Joint, Dislocation, Instability, Anterior

Introduction

The shoulder is the most commonly dislocated joint in the body, with 98% of dislocations occurring anteriorly.(1) The incidence is between 1 and 2% or 8.3 to 12.3 per 100,000 per year. Studies have shown that male patients aged 21 to 30 years are the population with the highest risk.(2)

The shallow glenoid compromises stability for range of motion, predisposing the joint to dislocation. Stability is conferred by soft tissue restraints, with the inferior glenohumeral ligament complex providing the most significant anatomical constraint to anterior shoulder dislocation.(3-4)

Dislocations can be classified into two distinct groups; Traumatic Unilateral dislocations associated with a Bankart lesion, often requiring Surgery (TUBS). A Bankart lesion (figure 1) is an avulsion of the inferior glenohumeral ligament-labral complex.(3) These injuries usually occur in young males with normal shoulder anatomy and are the result of significant trauma. The high recurrence rates (50-90%) mean surgical intervention is often required.(1, 5)

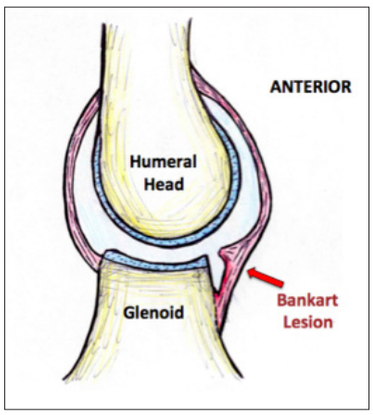


Figure 1: Bankart Lesion – avulsion of the inferior glenohumeral ligament-labral complex.

The other group is the Atraumatic Multidirectional Bilateral shoulder dislocation/subluxation, which often responds to Rehabilitation and sometimes an Inferior capsular shift is required (AMBRI). These patients have abnormally lax soft tissues; therefore a minimal amount of trauma can result in dislocation.

Complications of anterior shoulder dislocation include axillary nerve and brachial plexus injuries, greater tuberosity fracture and rotator cuff tears. Very rarely there is damage to the axillary artery.(6) Hence it is important to correctly identify and perform a thorough clinical assessment of these patients.

Clinical Features

Acute shoulder dislocation can be an extremely painful injury, and hence patients will often resist all movements and hold the arm in adduction. They often use the contralateral hand to support the affected limb below the elbow.

The shoulder may appear 'squared-off' with a loss of the usual contour, and on palpation there is often a hollow space beneath the anterior edge of the acromion where the humeral head is usually located.

REVIEW: ANTERIOR SHOULDER DISLOCATION

R Gogna

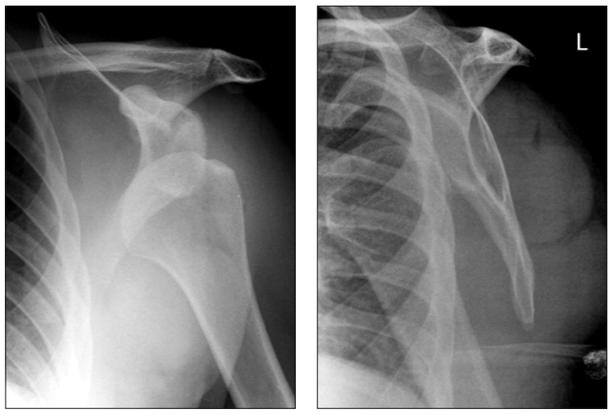
It is vital to perform a full neurovascular assessment of the affected limb. The most common neurological complication is axillary nerve palsy. This must be assessed and documented prior to reduction of the shoulder, as inadequate recovery will render the shoulder useless and there can be significant medico-legal implications. The axillary nerve can be evaluated by assessing pinprick sensation over the 'regimental badge' area of the lateral aspect of the proximal humerus. Usually the shoulder is too painful to assess deltoid activity.

Although rare, it is important to exclude an injury to the radial portion of the posterior cord by testing active wrist dorsiflexion. Distal pulses and capillary refill should also be assessed to rule out any potential axillary artery involvement. (6) This is more common in the elderly population. Any patient with a good capillary refill but absent pulses must be managed as a vascular injury until proven otherwise.

Radiographs

The next step is to obtain radiological evidence to confirm that the shoulder joint has been dislocated. The majority of anterior dislocations are evident on a standard AP radiograph where the humeral head is incongruent with the glenoid.

If the diagnosis is unclear, it is essential to obtain alternative radiographic projections. This may be an axial lateral projection (Y-view) or an apical oblique or translateral projection.



Figures 2-3: Radiographs showing anterior shoulder dislocation.

Anterior shoulder dislocation can be associated with a fracture of the greater tuberosity. Although this poses no immediate complication to reduction, intervention may be required at a later date if displaced by greater than 1cm.

Treatment - Indications

Reduction in the emergency department should be limited to young healthy patients who are neurovascularly intact.

There are however some absolute and relative contraindications to joint reduction in the emergency department as shown in the table below (Table 1). Although the relative contraindications do not prohibit reduction, they require prompt reduction and avoidance of multiple attempts. In these situations reduction may be difficult, hence should ideally be done in theatre with x-ray guidance, muscle relaxant and the ability to open the shoulder if necessary.

Contraindications		
Absolute	*Subclavicular or intrathoracic dislocation*	Contact senior orthopaedic surgeon
	Asssociated humeral neck fracture	Risk of avascular necrosis
Relative	*Nerve injuries / Neuropraxia* - brachial plexus or axillary nerve	Resolves in weeks to months
	Suspected arterial injury	Urgent Angiography
	Hill-Sachs deformity - Compression fracture of posterolateral humeral head	
	Bankart Lesion - Detachment of anterior glenoid rim	Arthroscopic Bankart repair
	Greater Tuberosity fracture - Avulsion	Require intervention if displaced >1cm

Table 1: Contraindications to reduction of acute shoulder dislocation in ED.

Treatment - Anaesthesia

The key to an effective reduction is pain control and muscle relaxation. Prior to sedation, patient consent needs to be obtained and the procedure should be fully explained. The most commonly used intravenous agents for procedural sedation are opiates (morphine sulphate), benzodiazepines (midazolam) and propofol. Dose should be adjusted depending on patient factors and titrated to response.

Whilst under sedation it is vital to continually monitor cardiovascular and respiratory function and assess the patient for potentially unwanted side-effects. Reversal agents for opioids (eg. Naloxone) and benzodiazepines (eg. Flumazenil) must be readily available.

REVIEW: ANTERIOR SHOULDER DISLOCATION

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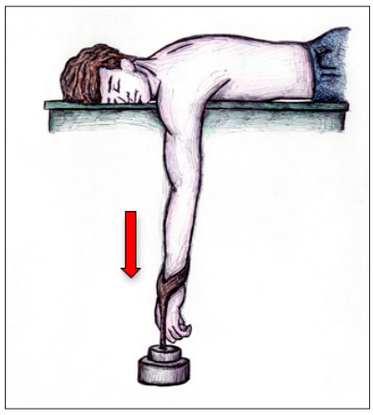
Review: Anterior Shoulder Dislocation
Trauma & Orthopaedic Surgery

Treatment – Methods of Reduction

Stimson Technique

Position the patient prone on an elevated trolley. The affected shoulder should hang downward off the edge of the bed with adequate weights attached to the patients' wrist. Eventually once the shoulder musculature relaxes, the humeral head will reduce into the glenoid. This technique can be combined with scapular manipulation (see below).



Advantages:

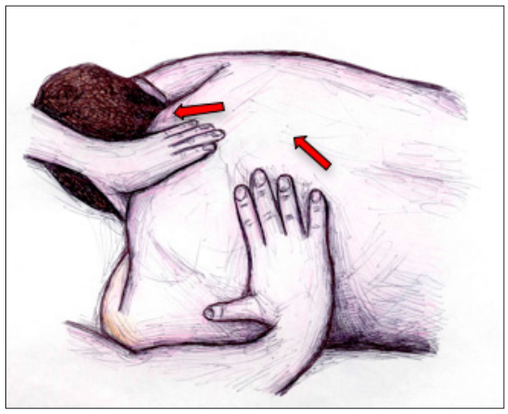
✓ No assistance required
✓ Shoulder is reduced with minimal force

Disadvantages:

× Continual patient monitoring
× Equipment required
× May take relatively longer to achieve reduction (15-20min)

Scapular Manipulation

Whilst in a prone position, stabilise the scapula with two hands as shown in the figure below. Using both hands, rotate the superior aspect of the scapula laterally, whilst rotating the inferior tip medially. This moves the glenoid fossa back into its anatomical position.

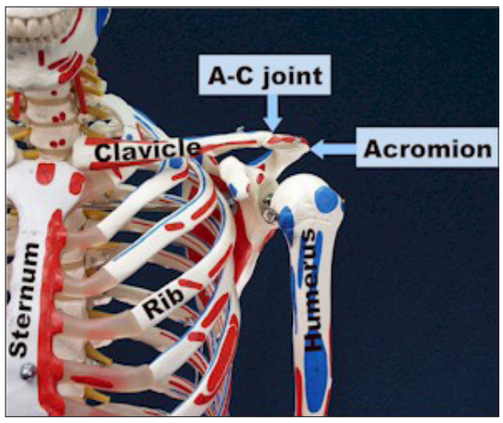


Advantages:

✓ Well tolerated by patients, minimal force required
✓ 85% success rate

Disadvantages:

× Difficult to palpate in obese patients
× Assistance may be required

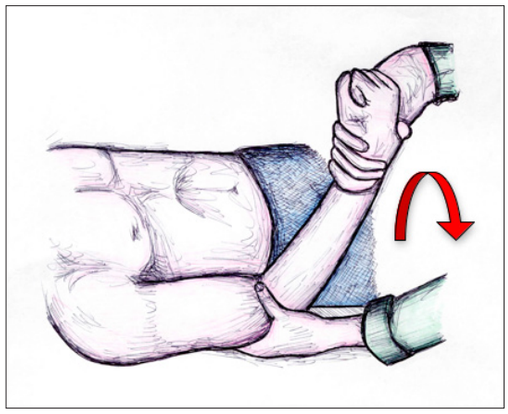


REVIEW: ANTERIOR SHOULDER DISLOCATION

R Gogna

External Rotation (Kocher's) Method

Get the patient to hold the affected arm in adduction and flex the elbow to 90o. Whilst holding the patients wrist, slowly externally rotate the arm. If pain or resistance is felt then take some time to pause and allow the muscles to relax before continuing. Reduction occurs between 70º and 110º of external rotation.



Advantages:

✓ Well tolerated by patients
✓ No assistance required, quick and easy to perform
✓ No force or traction necessary

Disadvantages:

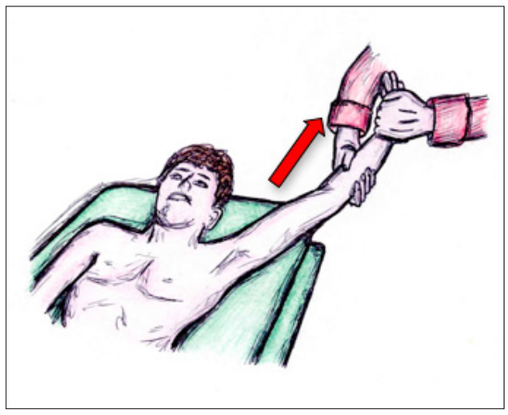
× Approximately 80% success rate
× Risk of proximal humerus fracture in elderly/osteoporotic patients (hence use with extreme caution)

Milch Technique

With the affected arm placed in overhead abduction, gently apply longitudinal traction and external rotation with one arm. Use your thumb to push the humeral head over the glenoid rim to successfully reduce the joint.

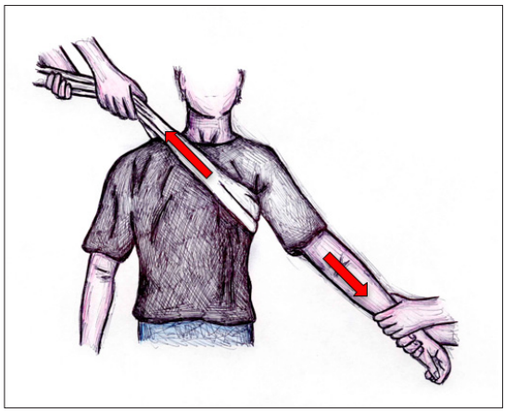
Advantages:

✓ Well tolerated by patients, sedation not required
✓ No assistance required
✓ 70-90% success rate



Traction-Countertraction

Wrap a sheet under the axilla of the affected arm and get your assistant to apply countertraction from the other side of the bed. Hold the patients arm flexed to 90o at the elbow and apply traction by leaning backwards with your full body weight. Apply gentle traction until the dislocation is reduced.



Advantages:

✓ Traditional method, well recognised
✓ High success rate – useful if severe muscle spasm

Disadvantages:

× Procedural sedation required due to substantial force applied
× Assistance is required
× Prolonged force and endurance

Treatment – Post-reduction care

If successfully reduced, a 'clunk' may be felt and the patient should be able to touch the contralateral shoulder with the affected arm. It is important to recheck the neurovascular status of the limb and document examination findings.

The patient should be sent for post-reduction radiographs to confirm that the glenohumeral joint has been successfully reduced and to identify any previously missed fractures.

REVIEW: ANTERIOR SHOULDER DISLOCATION

R Gogna

Traditionally a broad arm sling is used to support the arm in adduction and internal rotation. However, a recent literature review found that some authors believe immobilisation in external rotation may reduce the risk of recurrence in first-time shoulder dislocations.(7) Young, active patients should be immobilised for 3-6 weeks, with early orthopaedic follow-up to assess whether surgical stabilisation is required. Due to the high rate of recurrent dislocation (80-90%), many authors advocate early arthroscopic Bankart repair.(1, 8)

For patients older than 40 years, the shoulder should be immobilised for 1-2 weeks before commencing pendular exercises to increase mobility and reduce the risk of developing a frozen shoulder. During follow-up, it is important to assess the integrity of the rotator cuff, as 10-15% of anterior shoulder dislocations are associated with cuff tears. The patient should be able to abduct their arm to 90 degrees, and if there is any suspicion of a rotator cuff tear then an ultrasound scan should be arranged. The patient should be advised to avoid overhead activities that involve abduction and external rotation of the shoulder, as this predisposes to recurrent dislocation.

Instability and Recurrent Dislocation

Instability and recurrent dislocation have been shown to develop in 56% of shoulders within the first two years after primary dislocation and increases to 67% by the fifth year.(5) As for primary dislocation, young male patients are most at risk of instability. The most significant risk factors for recurrent dislocation include:

- **Initial high-energy injury**
- **Neurological deficit**
- **Associated large rotator cuff tear**
- **Associated fracture of the glenoid rim**
- **Associated fracture of the greater tuberosity (9)**

Review: Anterior Shoulder Dislocation
Trauma & Orthopaedic Surgery

Arthroscopic Bankart repair involves reattachment of the labrum and inferior glenohumeral ligament complex to the glenoid. This has now become the 'gold standard' treatment for young patients with traumatic shoulder dislocation.(1) Several studies have shown high success rates in preventing recurrences, with low surgical morbidity.(3, 10)

Unfortunately arthroscopic repair can also fail, with studies quoting failure rates of 8.1 - 13.3%.(11) Recurrent shoulder dislocation after failed surgery, and also primary dislocations with glenoid bone loss are most commonly treated with a Bristow-Latarjet coracoid transfer. This involves transfer of the coracoid with its muscle attachments to the front of the glenoid, providing additional muscular support and preventing further dislocations.

Summary

We have discussed the epidemiology, clinical features and management of anterior shoulder dislocation. As highlighted, there are several methods for reduction of the dislocated shoulder joint. Each method has its advantages and disadvantages so patient factors, staffing and equipment availability should be considered before deciding on the most suitable method of reduction.

MCQ's

Q1.) A 27-year-old rugby player sustains a closed dislocation of his shoulder during a game that reduces spontaneously. An MRI will most likely reveal which of the following?

A.) Supraspinatus tear
B.) Anteroinferior labral tear
C.) Humeral avulsion of the glenohumeral ligaments
D.) Long head of the biceps tear
E.) Superior labrum anterior to posterior tear

Q2.) After an anterior shoulder dislocation, what nerve is most frequently injured?

A.) Axillary
B.) Median
C.) Musculocutaneous
D.) Radial
E.) Suprascapular

REVIEW: ANTERIOR SHOULDER DISLOCATION

R Gogna

Q3.) Which of the following is an absolute contraindication to reduction of the dislocated shoulder joint in the emergency department?

A.) Brachial plexus injury
B.) Hill-Sachs deformity
C.) Humeral neck fracture
D.) Greater tuberosity fracture
E.) Suspected arterial injury

Q4.) Post-reduction, the shoulder should initially be immobilised in a broad arm sling. What is recommended as the most stable position in which the shoulder should be immobilised?

A.) Axial traction in adduction
B.) Adduction, external rotation
C.) Adduction, internal rotation
D.) Abduction, external rotation
E.) Extension, internal rotation

Q5.) What is the most significant risk factor for recurrent instability following a traumatic anterior shoulder dislocation?

A.) History of contralateral shoulder dislocation
B.) Family history of shoulder instability
C.) Dislocation of the dominant shoulder
D.) Age < 25 years at time of dislocation
E.) Low-energy injury

Answers:

1) Preferred Response: B

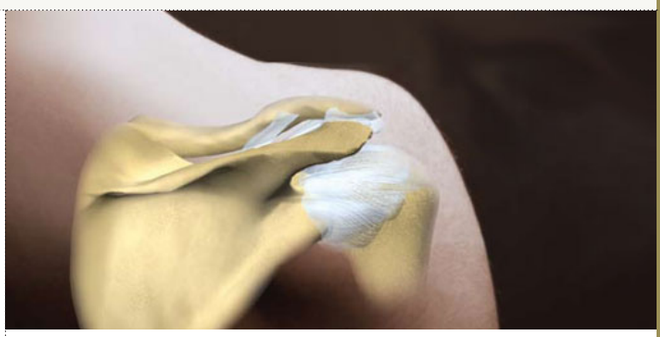
Acute traumatic shoulder dislocations in young athletes are associated with a high rate of anteroinferior labral tears. However older patients (above 40 years) are at increased risk of an associated rotator cuff tear.

2) Preferred Response: A

Anterior dislocation of the shoulder is the most common shoulder dislocation. Nerve injury results from traction of the nerve being stretched over the humeral head. The axillary nerve is the most commonly injured nerve during an anterior shoulder dislocation due to its course around the surgical neck of the humerus. It can be assessed by checking deltoid activity and sensation to the regimental badge area of the proximal humerus.

3) Preferred Response: C

An associated humeral neck fracture is an absolute contraindication to performing a reduction under sedation. This is because there is increased risk of avascular necrosis of the humeral head. The case should be discussed with a senior orthopaedic surgeon. All of the other options are relative contraindications; they require prompt reduction and avoidance of multiple attempts.



4) Preferred Response: C

The classic position of anterior shoulder instability is abduction and external rotation, hence when immobilising the joint it should be kept adducted and internally rotated. Although studies from Japan showed decreased recurrence rates when the shoulder was immobilised in external rotation, subsequent studies have refuted their initial findings and their results have not been reproducible.

5) Preferred Response: D

Regardless of the duration of immobilization, a rehabilitation cuff strengthening program or activity restriction, recurrent dislocation rates remain high. Patient age is the only consistent predictor of recurrence, with an 80-90% recurrence rate in younger patients. Family history confers a 34% risk of recurrence, while dislocation of the contralateral shoulder is seen in 25% of recurrently unstable patients.

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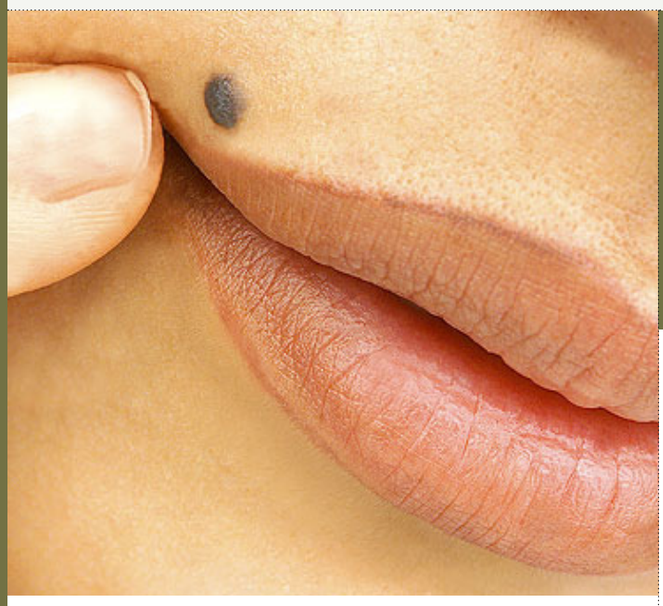
BENIGN NAEVI OF THE SKIN

B Green, A Scott, D Nikkhah



Abstract

In this review, we will be discussing common benign naevi of the skin, both congenital and acquired and the surgical and non-surgical management of these lesions.

Keywords: naevus, skin lesion, melanocytic.

Case study

A 1 year old boy presents to the outpatient clinic in the plastic surgery department with a red lesion on his right cheek. The lesion appears to be vascular in nature and was present from birth. An ultrasound was performed to demonstrate the depth, extent and flow through the region. A superficial venous malformation was diagnosed and Laser therapy was used to treat the lesion.

Introduction

Congenital naevi or birthmarks are skin markings that develop before or shortly after birth. Benign developmental skin lesions that occur later in life are referred to as 'acquired' naevi.

Naevi may be derived from the outer layers of the skin (epithelial naevi) or from the deeper layers (dermal/subcutaneous naevi). Naevi are further classified based on the cell type involved. Melanocytic and vascular naevi are the most common types of birthmarks.

Vascular naevi result from clustering blood vessels, melanocytic naevi are due to clusters of melanocytes, and epidermal naevi are derived from keratinocytes but can also be derived from epidermal appendages such as sebaceous glands (sebaceous naevi) or from a proliferation of hair follicles, keratinocytes and melanocytes (Becker naevi). The exact cause of why these lesions occur is unknown but it may relate to localised abnormalities of certain genes.

Epithelial naevi	Dermal/Subcutaneous naevi
Melanocytic naevi • Congenital melanocytic naevi (CMN) • Acquired naevi (moles) • Blue naevi • Halo naevi • Spitz naevus • Mongolian spot	**Vascular naevi** • Infantile haemangioma • Capillary malformations • Venous malformations • Lymphatic malformations • Arteriovenous malformations
Epidermal naevi • Epidermal naevi • Becker naevus • Sebaceous naevus	

Table 1 summarises the types of benign naevi.

Melanocytic naevi

Congenital melanocytic naevus (CMN)

These present as brown or black moles at birth as a result of a benign proliferation of cutaneous melanocytes. The incidence varies depending upon the size of the lesion, from 1in 100 for small naevi (1) to 1 in 20,000 for giant lesions (2). Giant lesions, are defined as more than 20 cm diameter at adulthood, or 9 cm on the scalp and 6 cm on the trunk in newborns (2). Giant CMN can have accompanying satellite lesions. CMN increase in size as the skin expands. There is probably only a significant increased risk of melanoma arising in giant CMN, which has been reported as being 0.7-2.4% (2-7). The risk is related to the size of the lesion so, the bigger the lesion, the increased risk of developing a melanoma (2).

The decision to excise a CMN depends upon the size of the lesion, the location, the risk of malignancy and the patient's choice. Serial photos and monitoring in clinic is an alternative management option (8).

Acquired naevi

These arise as a result of a proliferation of melanocytes and are benign in nature, but a malignant melanoma can arise within a previously benign naevus (9). The clinician would be suspicious of a malignant melanoma if the lesion were asymmetric, with an irregular border, variegated colour pigmentation and a diameter > 5mm (ABCD) (9-10). It is generally noted that the possession of a large number of naevi is a risk factor for developing melanoma. The history is crucial in diagnosing melanoma.

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Concerning aspects in a history include; a change in a mole such as bleeding, increasing size, itching or a new mole appearing. If a melanoma is suspected then the mole should be excised with a 2mm margin (9-11).

Spitz naevus

These are melanocytic proliferations that contain epithelioid or spindle melanocytes. They were first described in 1948 and were initially termed "juvenile melanoma" but they are benign. The most common age of presentation is under 10 with a well-defined, smooth symmetric, red or reddish brown nodule about 5-6 mm in diameter. They arise most commonly on the face and neck but can also occur on the extremities. Generally Spitz naevi are excised due to the difficulty in clinically differentiating them from a malignant melanoma (12-14).

Halo naevus (Sutton naevus)

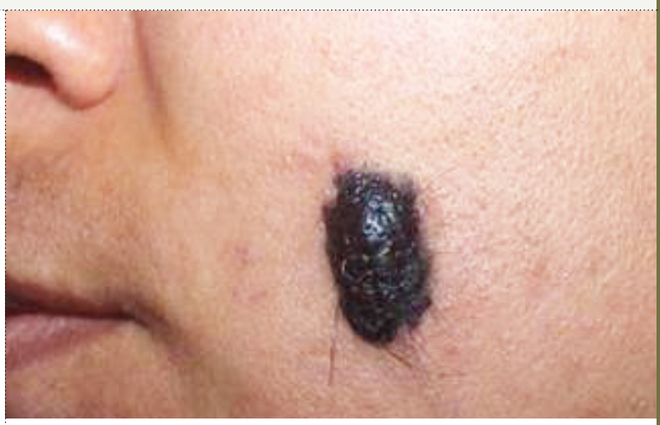
Halo naevi present as a melanocytic naevus surrounded by a ring of depigmentation. These occur in around 1% of the population usually before adulthood and have no predilection for race or sex. There is an association with vitiligo. Halo naevi can persist for years and the appearance of the halo suggests that the naevus is regressing but most patients' fear that its malignant melanoma. There are four stages in the development of a halo naevi; the appearance of the halo; loss of pigmentation within the central naevus; disappearance of the naevus and finally the disappearance of the halo (15). When examining a halo naevus it is important that you examine it like you would any other naevus; assessing for an irrugaulr border, incraseing size, colour variegation. Unless there are any atypical features the management of these naevi is conservative (16).

Mongolian spot

Mongolian spots are typically found on babies in the lower back and buttock region. They are considered a form of birthmark and are referred to as congenital dermal melanocytosis. They are the result of an entrapment of melanocytes in the dermis and that have not reached their correct position within the epidermis. These are commonly seen in patients of South-East Asian, Micronesian and Polynesian races (17).

Blue naevus

Blue naevi are benign, dermal, dendritic, melanocytic proliferations that appear as single or multiple, firm papules or nodules. They are blue, blue-gray or blue-brown in colour and are seen predominantly on the extremities or face but can also be found in other sites such as the conjunctiva, genitalia and oral cavity. Blue naeviare more common in females and can present at any age. The management can be either conservative or surgical excision (18,19).



Epidermal naevi

Epidermal naevus

They are also known as organoid naevus and are believed to originate from the pluripotential germinative cells in the basal layer of the embryonic epidermis. It is these cells that give rise to keratinocytes. These naevi are commonly found on the scalp and neck but can also be found on the trunk and limbs. They appear as papillomatous hyperkeratotic plaques. They generally present at birth and rarely enlarge after adolescence (20,21).

Becker naevus

This is an uncommon acquired skin condition that consists of the development of a unilateral hyperpigmented patch that in time develops a verrucous, slightly elevated surface. Hypertrichosis is often a feature, mainly in male cases. The prevalence is 0.25-2.5% occurring more frequently in males. Currently, there is little understanding behind the disorder although one suggestion is androgen sensitivity. Becker's can be left alone but often patients find these lesions cosmetically unacceptable. Surgical excision is an option but this is often challenging due to the size of the lesion. Ng:Yag and Q- switched ruby laser can be affective for reducing the degree of hyperpigmanetation (15, 22-24).

Sebaceous naevus

These are benign lesions that are predominantly seen on the scalp, but can also present on the face, preauricular area and neck. This is a hamartoma that contain epidermal, apocrine, follicular and sebaceous elements. They are verrucous, granulated yellow-orange plaques that can be round, linear or crescentic in shape. It has been suggested that there are three stages in the natural history of this naevus. The first stage is a papillomatous epithelial hyperplasia with underdeveloped hairs; the second stage occurs at puberty and is characterised by the maturation of apocrine glands, epidermal verrucous hyperplasia and large development of sebaceous glands. The most widely accepted hypothesis is that sebaceous naevi result from genomic mosaicism in stem cells that expand in the lines of Blaschko. HPV has also been implicated in the aetiology and was found in 82% of specimens studied. Surgical excision is the preferred treatment but alternatives options include photodynamic therapy, CO2 laser resurfacing and dermabrasion but there is a risk of recurrence (25-27).

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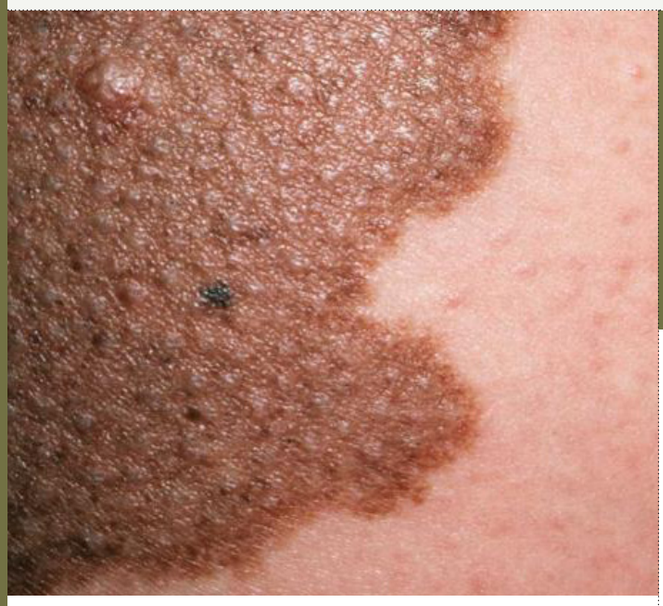

Haemangioma

Infantile haemangiomas (IH), or more commonly known as strawberry naevi, are the most common type of tumour in infancy, arising in 10%-30% (28, 29) of the Caucasian population. It is characterised by rapid proliferation during infancy followed by slow spontaneous resolution over 10 years (29). Risk factors are prematurity, female sex, and Caucasian ethnicity. The pathogenesis has three possible hypotheses: embolisation of placental endothelial cells: increased angiogenic and vasculogenic activity or tissue hypoxia. If infantile haemangiomas are ulcerated, impairing function or located in an area associated with a poor cosmetic outcome such as the nose, the first line treatment is oral propanolol (30-32) Propanolol results in vasoconstriction due to nitric oxide release within the first three days of treatment, during the intermediate period there is blocking of pro-angiogenic signals and long term, there is the induction of apoptosis in proliferating endothelial cells (29). Pharmacological treatments previously used include corticosteroids (if life-threatening) (33, 34) and vincristine (35). Captopril has also been investigated as a drug treatment but further investigation is needed (36). Pulse dye laser is an alternative treatment to propanolol.

Capillary malformations

These are made up of post-capillary venules within the papillary and reticular dermis. The presentation is of flat, pink macules that darken with age leading to a cobblestone appearance and are commonly found on the nape of the neck, the glabellar region or the eyelids. These arise from altered neural modulation of the papillary plexus (28, 37, 38). They can be seen in isolation or as part of a syndrome such as Sturge-Weber or Klippel-Trenaunay. The rate of progression can be induced by hormones and trauma and are seen in 0.3% of children.

Pulsed dye laser is the treatment of choice. Two-six treatments are needed and the response rate varies from 4-100% and that generally they recur within 2-3 years (40%). This may be the result of the pathogenesis of the malformation (28).

Venous malformation

They are characterised by an abnormal collection of veins, which do not have any mitotic activity in endothelial or pericyte cells and often lack a uniform smooth muscle layer (28). The clinical features depend on size and age. The presentation is of a soft, compressible lesion that enlarges in size when there is an increase in venous pressure. The diagnosis is made clinically together with an ultrasound to assess depth and Doppler used to assess flow. Treatment is dependent on the extent, depth and anatomical location (28).

Treatment	Explanation (28)
Laser	• Nd: YAG laser is used for treating a superficial lesion or a superficial component of a compound lesion • Destroys the superficial component and for a deeper lesion, provides an avascular plane for an access flap • Repeated treatments are common
Sclerotherapy	• Used for larger malformations • The most commonly used injectable agents are 100% ethanol and Ethibloc® • Recurrence not uncommon
Excision	• Difficult to perform, often done in combination with other treatments • Residual or recurrent lesions treated by other modalities

Lymphatic malformation

These are congenital embryological malformations of the lymphatic system that consist of channels and cystic spaces of varying size (39). There is an accumulation of fluid, beneath the skin. Lymphatic malformations are believed to originate from the sequestration of lymphatic tissue during the development of lymphaticovenous sacs, which do not communicate with the venous or lymphatic system. This results in dilatation of the sequestered lymphatic tissue appearing as the cystic lesions (40).

Lymphatic malformations are most likely to occur during childhood with an incidence of 1 in 20,000, whereas in adults, it is 1 in 100,000. These account for about 5% of benign tumours presenting in paediatric practice. Around 66% are found in the head and neck region before the age of 2 and can in some cases, extend to the mediastinum. They are commonly seen in the tongue, buccal mucosa and on the neck (39, 40).

Histologically, they are categorised as being macrocystic or microcystic but both types can arise in the same lesion. They are lined by a single endothelial layer and can have lymphocyte involvement with germinal centres being reported. Their presentation in childhood is thought to be the cause of trauma or infection but can present at birth. If they present in the adult, it is either the result of trauma or an upper respiratory tract infection (41, 42).

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The indications for treatment are recurrent infection, cosmetic disfigurement and compression of structures such as the airway or major blood vessels. The diagnosis is made clinically and is a generally soft, compressible, non-tender, transilluminable mass with no bruit. CT and MRI imaging are useful in identifying the proximity to other anatomical structures but ultrasound is used first as an imaging modality showing a multicystic lesion with septations and no blood flow is seen on colour flow Doppler (39).

Treatment of lymphatic lesions is complex and therefore needs to be discussed at a multi-disciplinary team meeting. There are reports of spontaneous regression in children, so treatment should not start until the age of 2-3 years (39).

Treatment	Explanation (39)
Aspiration (43,44)	• This is performed under ultrasound guidance • Objective is to reduce pressure symptoms temporarily • Lesions produce a sticky, serous, straw-coloured fluid when aspirated with a wide-bore needle
Radiofrequency ablation (45)	• A non-invasive technique • Causes increased temperature in tissue leading to denaturing of local proteins • The rise in temperature due to vibrations within the cells • Precise treatment of specific areas and useful if lesion is proximal to vital adjacent structures • Reduces amount of pain and morbidity post-treatment
Laser (46)	• Mainly used to treat small, microcystic and superficial, mucosal-based lesions in the larynx and oral mucosa • Point destruction so minimize damage to adjacent structures • CO_2 laser most commonly used
Sclerotherapy (47)	• Various agents used such as ethanol, doxycycline and hypertonic glucose • Fluid must be removed prior to treatment for optimal effect • The lesion has to be compressed to maximize contact and prevent extravasation
Chemotherapy (48)	• Agents include bleomycin and pingyangmycin • The mechanism of action is not known • Produces a non-specific inflammatory process • Can be repeated every 3-4 weeks, but if no response other treatments to be used
OK-432 (49)	• Sclerosing agent from *Streptococcus pyogenes* • Has fewer complications than chemotherapy • Mechanism is induction of an acute inflammatory response with endothelial damage • Like other sclerosing agents, need ultrasound guidance • Advantage is minimal risk of damage to anatomical structures • Disadvantage is need for more than one injection and fever can be induced with erythema and swelling
Surgery (50)	• Main treatment for macrocystic lesions • Must be considered if risk of compression of vital structures or aesthetic reason • Risk of bleeding, infection, nerve damage and recurrence • Alternative techniques include endoscopic cautery with post-operative intraluminal pressure

Arteriovenous malformations

These are the least common of all the vascular malformations and the most common sites of presentation are the mid-face, especially the cheek and ear (27). There is a considerable amount of arteriovenous shunting and these shunts develop and increase as the child grows. The lesions are generally deep but if close to the surface, a palpable thrill may be present. They are firm and do not empty as readily when compressed. Although present at birth, the presentation can be delayed until adulthood. The pathogenesis is not understood, although the primary abnormality is a nidus or bed of dilated capillaries and the hypertrophied arteries and dilated veins are secondary phenomena due to increased flow across the nidus (27).

There is also a suggestion of a pre-capillary sphincter regulates the flow of blood through the capillary bed and it is the abnormality in the sphincter causing the malformation. Diagnosis is made clinically together with duplex ultrasound and other radiological investigations such as MRI. The aim of the treatments listed is to eradicate the nidus.

Treatment	Explanation (28)
Embolisation (28)	• Useful for larger malformations • Can be used as first line treatment or as an adjunct • Various agents can be used such as onyx, polyvinyl alcohol, gelfoam and silicone fluid
Surgery (51)	• Gold standard • Aim is to remove nidus and any structures that support it • Recurrence rate lower than with embolisation

Summary

Clinical recognition of these naevi is needed before any investigations are carried out. Many of these naevi present in children but in some cases occur in adults too. There is increasing consensus that non-invasive treatments should be used prior to excision. The naevi described in this review are common and there are rarer naevi not mentioned as they are beyond the scope of this introductory teaching article.

Questions

1. If a CMN is considered "giant" what is the diameter of the lesion?

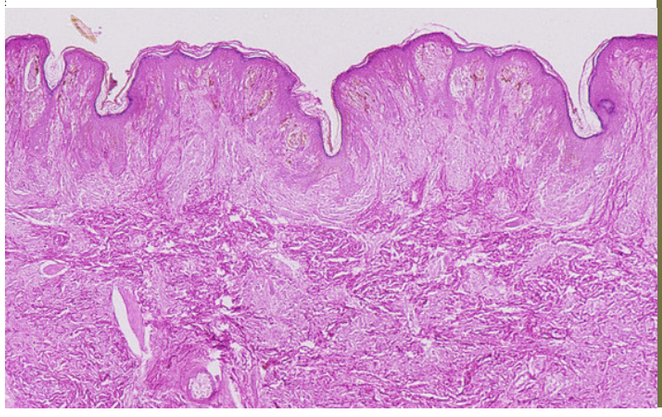
A - 5cm
B - 10cm
C - 15cm
D - 20cm
E - 25cm

2. When does a Mongolian spot present?

A - At birth
B - During childhood
C - During adulthood

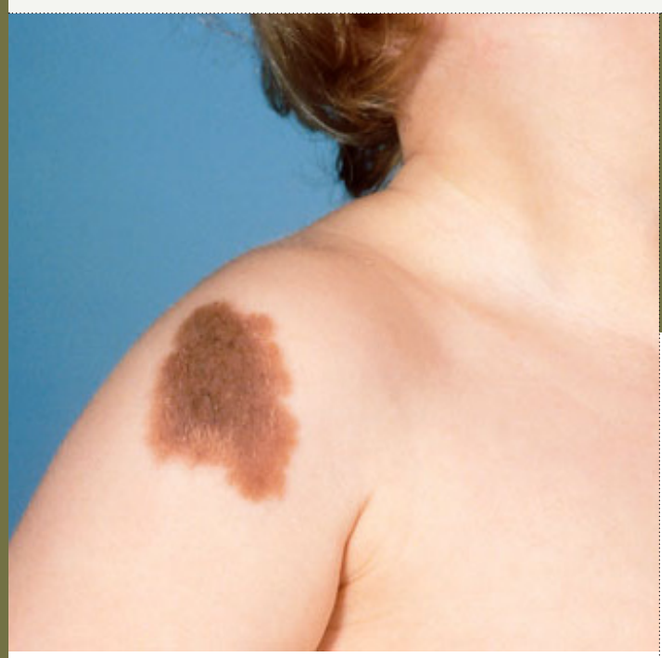
3. Which sex is Blue naevi more commonly associated?

A - Females
B - Males
C - Equal



BENIGN NAEVI OF THE SKIN

B Green, A Scott, D Nikkhah



4. Where are sebaceous naevi most commonly found?
A - Scalp
B - Torso
C - Limbs

5. What is the favoured treatment for capillary malformations?
A - Excision
B - Laser surgery
C - Conservative

6. What is the clinical finding of a lymphatic malformation?
A - Soft, compressible, non-tender, transillumable
B - Hard, tender, not illumable
C - Hard, non-tender, transillumable

Answers

1 (D)
2 (A)
3 (A)
4 (A)
5 (B)
6 (A)

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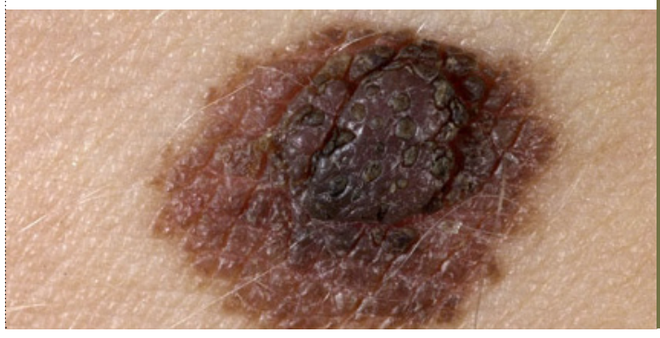

CHEST AND NECK SWELLING POST PLEURAL CATHETER INSERTION

KH Yap, WC Lin, PT Lee, P Krysiak

Chest and Neck Swelling Post Pleural Catheter Insertion
Cardiothoracic & Critical Care

Abstract

This is a case of a patient who suffers from surgical emphysema after a pleural catheter is inserted for malignant pleural effusion. The Chest X-Ray shows the abnormalities revolving surgical issues of pleural diseases secondary to lung cancer. This article describes the management of malignant pleural effusion and surgical emphysema based on the British Thoracic Society guidelines and the latest evidence. Due to lack of management guidelines, the authors took the opportunity to summarize the steps of management of surgical emphysema based on literature review.

Keywords: Surgical emphysema, pleural catheter, chest-X-ray, pleural effusion, lung malignancy

Clinical Presentation

A 69-year-old lady presented to Accident and Emergency with sudden onset of right-sided neck and chest wall swelling two days after having a right-sided pleural catheter insertion for recurrent malignant pleural effusion due to pulmonary adenocarcinoma.

The swelling was generalized, surrounding the anterior and posterior surface of the right-sided chest wall and right side of her neck. It was increasing in size slowly over a few hours. It was associated with pain and her voice was also noted to be high-pitched. She was not breathless and no history of trauma was reported.

She was diagnosed with right pulmonary adenocarcinoma with recurrent pleural effusion two years ago and chemotherapy was administered. The tumour was unresectable. She was trouble-free from pleural effusion during chemotherapy. Unfortunately, the right-sided pleural effusion developed again after the completion of chemotherapy.

On examination, she looked comfortable and not in respiratory distress. Her saturations were 98% on air, respiratory rate was 18 per minute, heart rate was 86 beats per minute, blood pressure was 130/90 mm Hg. Her respiratory examination revealed a generalized swelling in her right chest and neck. It felt soft and was tender on palpation. Her pleural catheter seemed to be secured and in the correct position. Her trachea was central and air entry was decreased over the whole right chest. A chest radiograph (Figure 1) was performed.

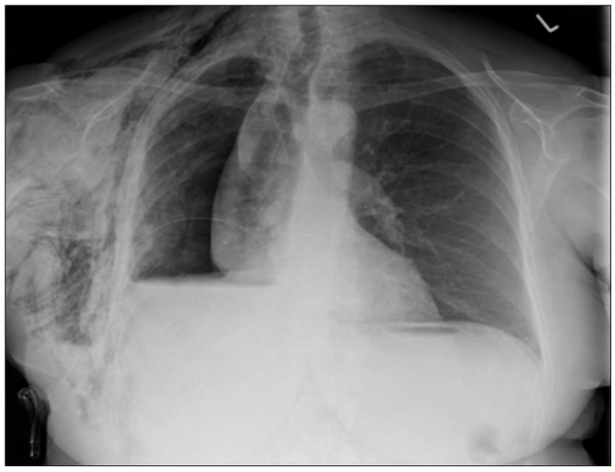

Figure1: Chest X-ray of the patient on admission to Accident and Emergency.

Department

Discussion

• As a surgical SHO, you are called to see this patient. What is the most likely diagnosis?

Taking into account the acute onset, character of swelling, lack of history of trauma and recent pleural drain insertion, this is a case of extensive surgical emphysema. This is a complication of pleural drain insertion as the procedure provided a portal for gas to enter the subcutaneous tissue. Aberrant air causing surgical emphysema here originates from pneumothorax and dissecting into the soft tissue of the face, neck, upper chest and shoulder (1). Dysphonia is caused by air dissecting into the larynx.

Clinically symptomatic surgical emphysema (SE) is defined as air under the skin that was perceptible by the clinician, patient, or family member, or dysphonia (1), therefore a clinical diagnosis. SE most often is self-limiting and does not pose much clinical significance. However, in severe cases, it can be a distressing cosmetic deformity and cause much anxiety in patients (1). It leads to serious comorbidities and even mortality.

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SE can lead to upper airway obstruction, acute respiratory failure due to chest wall compression, intracranial hypertension, tension pneumomediastinum which could lead to circulatory failure, and pacemaker malfunction. It can also lead to difficulties in the interpretation of chest radiographs, echocardiography, ultrasound, and electrocardiograms (2).

• What are the changes that you can see on the chest radiograph (Figure 2)?

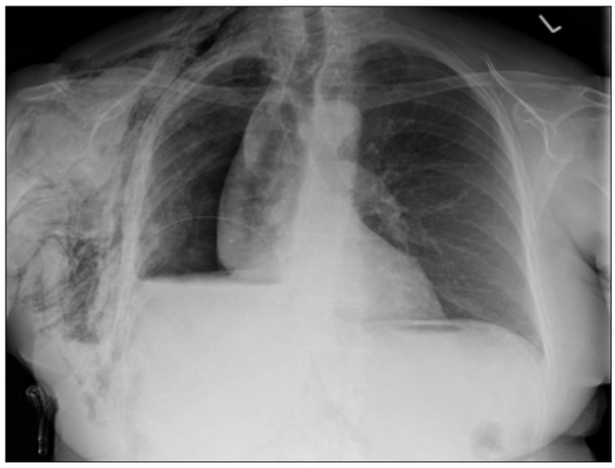


Figure 2: Changes on chest radiograph.

A. Pleural catheter seen here is inserted via the 6th intercostal space. This is an appropriate position with it being at the base of the right hemithorax for drainage of effusion. In contrast with chest drain insertion at the apex for pneumothorax.
B. Small amount of effusion is present in this erect CXR as demonstrated by the horizontal fluid level.
C. The right lung is almost completely atelectatic leaving the right hemithorax air-filled.
D. This phenomenon is named lung entrapment caused by combination of visceral pleural restriction secondary to malignant pleural membrane, endobronchial obstruction or chronic atelectasis (3).
E. The atelectatic lung also resulted in the ipsilateral shift of mediastinum exemplified by position of the trachea.
F. Extensive surgical emphysema is demonstrated in the diffuse opacification in the soft tissue swelling on the right chest extending into the neck.

• One of the patient's relatives would like to speak to you because she wonders what the indications for pleural catheter insertion were.
The presence of malignant cells in pleural fluid and/or parietal pleura signifies advanced cancer disease and a reduced life expectancy ranging from 3 to 12 months. According to the British Thoracic Society (BTS) Guidelines for the management of malignant pleural effusions 2010, treatment options for malignant pleural effusions are determined by symptoms, performance status of the patient, the primary tumor and its response to systemic therapy, and the extent of lung re-expansion following pleural fluid evacuation (4).

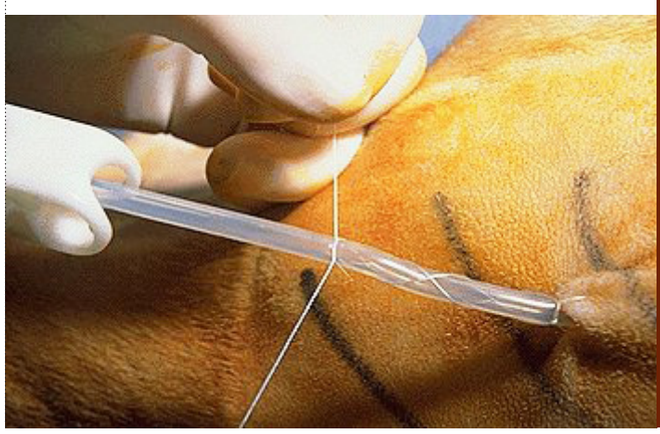
If the patient is asymptomatic and the tumor type is known, observation is recommended. Symptomatic patients should be referred for specialist opinion from a member of respiratory medicine.

Pleural effusions treated by aspiration alone are associated with high recurrence rate. Moreover, repeated aspiration may limit the scope for thoracoscopic intervention owed to the formation of adhesions between two layers of pleura. Therefore, the option of aspiration alone is not recommended for patients with life expectancy > 1 month (4). Transient relief of symptoms and hospitalization avoidance of frail patients with a very short life expectancy can be achieved by repeat therapeutic pleural aspiration.

On the other hand, small-bore intercostal chest drain insertion followed by pleurodesis should be performed in patients with good prognosis to prevent recurrence. Patients with confirmed significant trapped lung are an exception to this. A chest radiograph can assess the lung re-expansibility after fluid drainage. Pleurodesis may still be attempted if only partial lung expansion or pleural apposition is achieved after drainage. In patients with failure of pleurodesis, BTS guideline recommends repeat pleural fluid drainage with either repeat pleurodesis or insertion of indwelling pleural catheter. The presence of trapped lung is the consideration factor. Insertion of indwelling pleural catheter is recommended in cases of complete lack of pleural apposition e.g. trapped lung.

The therapeutic role of thoracoscopy has been extensively evaluated and it is deemed to be a safe procedure with low complication rate. Its ability to obtain diagnosis, drain the effusion and perform a pleurodesis during the same procedure is very beneficial to patients and clinicians. It also allows a direct visual confirmation of the presence of trapped lung thus allowing the decision of talc pleurodesis or pleural catheter insertion to be made. Besides, it is also used to break up the adhesions or loculations hence facilitate the lung re-expansion followed by pleurodesis.

Studies have shown that an indwelling pleural catheter is an effective option to control recurrent and symptomatic malignant effusion when length of hospitalization can be kept to minimum. The algorithm of management of malignant pleural effusion is available on the BTS guidelines (4).



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- **How should this patient be managed?**

Although SE is a relatively common condition, its treatment has been poorly studied probably due to its non-lethal nature (1). SE may be secondary to a pneumothorax, pneumomediastinum or other perforated hollow viscus. SE is also a recognized complication of chest tube drainage, which may form a portal for gas to enter subcutaneous tissues (1). In patients with post Video Assisted Thoracoscopic Surgery (VATS) or thoracotomy, part of the air-leaking lung that adheres to the intercostal space that had been previously opened may cause SE (1).

The first step of management of SE in patients without an existing chest drain is to identify the primary cause especially pneumothorax. If a pneumothorax is identified, then insertion of a chest drain is the first line treatment. This will usually drain the pneumothorax followed by settling of the SE (5).

In patients with existing chest tube, the first step is to ensure the drain is patent, properly secured in place, not kinked, clogged or clamped. Patients with large leak (e.g. voice change secondary to SE dissecting into the larynx) mandate the addition of suction (1). This treatment had been used in this patient.

Patients with massive SE may require intubation or tracheostomy to protect the airway and assist with ventilation (5).

SE that does not resolve with a chest drain with additional high wall suction warrants further managements. Different techniques have been described but the optimal one is yet to be identified (1). They include placing a new intrapleural chest drain, creating a small supraclavicular incision to help evacuate the air, placement of a subcutaneous fenestrated catheter with sequential massage to evacuate air into an underwater seal system (6), placement of a Penrose drain covered with a colostomy bag (5), and placement of a Penrose catheter with sequential massage (7). RJ Cerfolio and colleagues suggest that SE in patients who undergo lobectomy is best treated with VATS by breaking the adhesions of the leaking lung to the previously opened intercostal space. This directs the air leak back into the pleural space instead of leaking into the subcutaneous tissue, hence reducing the duration of SE and hospitalization (1). This technique is clearly not suitable for patient in this case because of the trapped lung.

In conclusion, the optimal treatment of surgical emphysema needs to be identified through further studies. The understanding of the primary cause of SE in each individual patient and the experience of the clinician play major roles in management.

Patient Outcome

This patient suffered from surgical emphysema due to the chest drain insertion and was readmitted to the hospital. Her chest drain was not blocked and not misplaced. Therefore, the drain was connected to an underwater seal system with suction. Her surgical emphysema resolved with this measure and she was discharged home.

Single Best Answer MCQ

1. Which part of the pulmonary system is responsible for pleural fluid production and reabsorption?

A. Lung Parenchyma
B. Bronchus
C. Parietal Pleura
D. Visceral Pleura
E. Pulmonary Vein

2. Which of the followings complicate chylothorax?

A. Malnutrition
B. Respiratory difficulty
C. Opportunistic infection
D. Sub-therapeutic level and effects of amiodarone
E. All of the above

Extended Matching Questions

A. Median Sternotomy
B. Posterolateral Thoracotomy
C. Anterior Thoracotomy
D. Clamshell Incision
E. Anterior Mediastinotomy
F. Sub-xiphoid window
G. Thoracoabdominal incision

CHEST AND NECK SWELLING POST PLEURAL CATHETER INSERTION

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3. A 26-year-old woman presented with diplopia and fatigue. She has been diagnosed with Myasthenia Gravis. She was found to have a mass in the anterior mediastinal compartment. Biopsy was performed and surgery was recommended. Which is the most appropriate for the surgery?

4. A 40-year-old gentleman with 3 weeks history of right-sided pneumonia complicated by empyema was referred to a thoracic surgical center. He had a large bore chest drain in place and has been on broad-spectrum antibiotics in the last 2 weeks. However, his inflammatory markers were still deteriorating. A surgery has been recommended. What is the most appropriate incision for the surgery

5. A 30-year-old male patient was admitted to Accident & Emergency Department after a road traffic accident. He sustained a penetrating injury on the left chest. His presented with severe hypotension, high jugular venous pressure and muffled heart sounds. It was decided that emergency thoracotomy should be carried out. What is most appropriate thoracic incision?

Mcqs Answers

1. Answer: C

Production: Pleural fluid is produced at parietal pleural level mainly in the less dependent regions of the cavity. Reabsorption: Pleural fluid is reabsorbed by parietal pleural lymphatic in the most dependent part of the cavity, on the diaphragmatic surface and in the mediastinal regions. When production exceeds maximal pleural lymphatic flow, pleural effusion occurs (8).

2. Answer: E

Chylothorax is a rare condition that caused by thoracic duct damage with chyle leakage from the lymphatic system into the pleural space. Chyle contains large amount of cholesterol, triglycerides, chylomicrons, fat-soluble vitamins, immunoglobulin, enzymes, white cell count and digestive products. Therefore, immunosuppression results from chyle leakage may predispose patients to opportunistic infection, sub-therapeutic effects of certain drugs and malnutrition. Respiratory difficulty occurs as the results of filling of fluid in the pleural space (9).

3. Answer: A

The patient has thymoma. Thymoma and Myasthenia Gravis are commonly associated. Median sternotomy gives good direct access to mediastinum. It is also used in majority of cardiac procedures, lung transplantation, lung reduction surgery and in cardiothoracic trauma (10).

4. Answer: B

The patient has empyema and lung decortication has been recommended. This incision is used for lung resection, oesophageal surgery, decortication and other pleural surgery. The rib space accessed is dependent on the structures you wish to approach. Apical structures and aortic isthmus are best approached through fourth space. The fifth space gives good access to the hilum. Some surgeons use 6th or 7th space for lower lobectomy or oesophageal procedure (10).

5. Answer: C

The patient suffers from cardiac tamponade. The anterior thoracotomy is commonly used for obtaining an open lung biopsy and in trauma. It can be extended to a clamshell incision (bilateral thoracosternotomy) in an emergency situation if necessary. Clamshell incision gives access to all chest cavities and requires minimal equipment (10).

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TESTICULAR CANCER: REVIEW

KE Murphy, JM Trotter

Abstract

Testicular cancer is the most common malignancy in males aged 18-40(1). Common tumour types are Seminoma and Non-seminomatous germ cell tumours (formerly 'Teratoma'). Lymphoma is rare. Treatment is based on radical inguinal orchidectomy and following this, on a combination of chemo-radiotherapy.

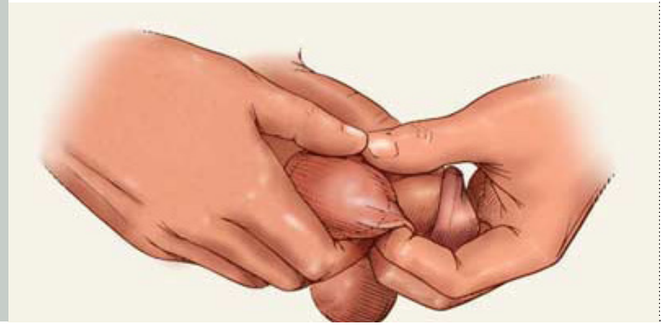
Keywords: Seminoma, Non-seminomatous germ cell tumour, orchidectomy

Case Vignette

'A 22 year old student is referred from the GP to the Outpatients Department with a six week history dull ache in the right testis, associated with a firm, non-tender swelling. He has previously been treated with a two week course of oral Doxycycline and Ciprofloxacin by the GP, however his symptoms have not improved with this treatment. On more detailed questioning, he admits that he may have had the swelling for more than three months, but had attributed it to a 'knock' whilst playing football.

The patient states he has lost weight and does look thin. On examination, you find a firm, non-tender, irregular mass in the right hemi-scrotum. There is no associated lymphadenopathy. You are able to confirm by Ultrasound scanning the presence of a suspected tumour, and inform the patient of the diagnosis, and the next steps for management.

He is referred to the Urology Cancer Multidisciplinary team meeting, after which he has a staging CT scan, and has a right sided inguinal orchidectomy performed on the next available operating list. His histology confirms non-Seminomatous germ cell tumour (teratoma), and his CT does not show any evidence of metastatic spread.



Background

Testicular cancer is the most common malignancy in males aged 18-40. It is uncommon below 15 and over 60 years. Increased levels of awareness have made significant strides towards earlier detection, although for some it remains a taboo subject for discussion. Its incidence is increasing (7 per 100,000 in the UK), and this may be due to public campaigns encouraging testicular self examination (TSE).

Embryology

The testicles form from the primitive genital or gonadal ridges, with germ cells appearing at about 6 weeks gestation. Shortly before and during the arrival of primordial germ cells, the epithelium of the genital ridge proliferates and penetrate the underlying mesenchyme. Here they form the primitive sex cords. It is impossible to differentiate the male and female gonad, hence is known as the indifferent gonad.

If the embryo is genetically male, under the influence of the SRY gene on the Y chromosome, the primitive sex cords continue to proliferate and form the testis. During further development the tunica albuginea separates the testis cords from the surface epithelium. Interstitial Leydig cells from the primitive gonagal ridge begin to produce testosterone at about 8 weeks and begin sexual differentiation and the development of the external genitalia.

Surgical Anatomy

The term testicle includes both testis and epididymis. The testicle lies in front of the testicle, slung from the external inguinal ring by the spermatic cord. The tunica vaginalis, a remnant of peritoneum almost completely surrounds the testis. The epididymis is a coiled tube lying behind the testicle which continues as the vas deferens.

Blood supply comes from the testicular artery, a branch of the aorta around the level of the renal arteries, passing in front of the ureters, and slinging around the inferior epigastrics to enter the inguinal canal. The vas deferens runs along the back of the spermatic cord, curling around the inferior epigastrics and courses in front of the ureter, passing through the inner and outer zones of prostate and entering the common ejaculatory duct, and opening into the prostatic urethra on the verumontanum.

The veins draining the testicle join together to form the pampiniform plexus, which enters the inferior vena cava (IVC) directly on the right; and the renal vein on the left.

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Lymphatic drainage of the testes follows the testicular arteries back to the para-aortic lymph nodes, while lymph from the scrotum drains to the inguinal lymph nodes.

Structure

Each testicle is made up of sets of tubules arranged in loops, which empty into the rete testis, which then drain into the epididymis via a dozen vasa efferenta. The testicular tubules contain both germinal cells and Sertoli cells, in between which there are packed Leydig cells. The germ cells divide into spermatocytes, which ultimately become spermatozoa by mitosis and meiosis.

Risk factors for testicular cancer:

The dramatic increase in the incidence of testicular cancer has led to an intense search for its causes, but, as yet, no preventable risk factors have been found. Both environmental and genetic factors are likely to be involved (4).

Risk factors include

Cryptorchidism: A common congenital abnormality in males - at birth 6% of all male babies have undescended testes. Most of these descend spontaneously by 3 months at which time only 1.6% of babies still have one or more undescended testes, or maldescent testicle (MDT). MDT is known to increase the risk of testicular cancer. Correction of cryptorchidism is possible through a procedure known as orchiopexy.

According to a large Swedish study, cryptorchidism is associated with a two-fold increased risk of testicular cancer in men who underwent orchiopexy before the age of 13, but risk of testicular cancer is more than five times higher for men treated at the age of 13 or later(6).

Orchiopexy: Through an incision over the internal ring, the external oblique is opened and the testicle is mobilised, once the testicular artery and the vas have been visualised. The testicular vessels are followed up behind the peritoneum and mobilized allowing the testicle to be placed in the sac, between dartos muscle and skin. Orchidopexy can also be performed laparoscopically if the testis is found to be in the intra-abdominal cavity.

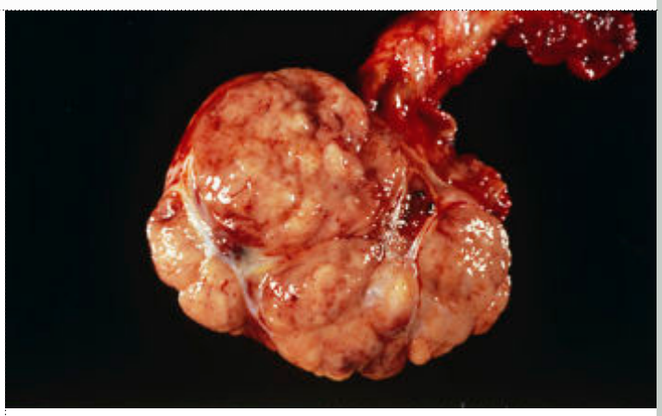
Inguinal hernia: Has been associated with a 63% increased risk of testicular cancer(7).

Hypospadias: Associated with an 88% increase in risk(7)

Caucasian: White males have a higher risk of testicular cancer than men of other ethnicities, however the reason for this appears unclear(8).

Previous testicular cancer: A previous diagnosis of testicular cancer increases the risk of developing a subsequent (metachronous) testicular tumour by around 12 times(9).

Other risk factors include sub-fertility, age 20-40, and smoking although there is a difference of opinion on this.



Staging of testicular cancer:

There are 5 T stages for testicular cancer;

· TIS in situ, means that cancerous cells have been found but they have not moved into surrounding tissues in the testicle
· T1 means the tumour is contained within the testicle and epididymis
· T2 means there are signs that the cancer has grown into blood vessels or lymphatics
· T3 means the tumour has grown as far as the spermatic cord (and may also have grown into blood vessels or lymph vessels)
· T4 means the tumour has grown into the scrotum

There are four lymph node categories in the TNM classification for testicular cancer. The bigger the lymph nodes, the higher the stage. The four categories are;

· N0 means there are no lymph nodes containing cancer cells
· N1 means that one or more lymph nodes contain cancer cells but they are smaller than 2cm across
· N2 means at least one of the lymph nodes is bigger than 2cm, but smaller than 5cm across
· N3 means at least one of the lymph nodes is bigger than 5cm

There are 3 categories of tumour spread in the TNM classification for testicular cancer;

· M0 means there are no signs of metastases
· M1a means there is cancer spread to the lungs OR there are cancer cells in lymph nodes a long way away from the testicles
· M1b means there is spread to other body organs such as the liver or brain
TNM staging for testicular cancer also looks at the levels of particular testicular proteins in the blood. It calls this the S stage. The proteins are called markers.
· S0 means markers are at normal levels
· S1 means markers are slightly raised
· S2 means markers are moderately raised
· S3 means markers are very high

TESTICULAR CANCER: REVIEW

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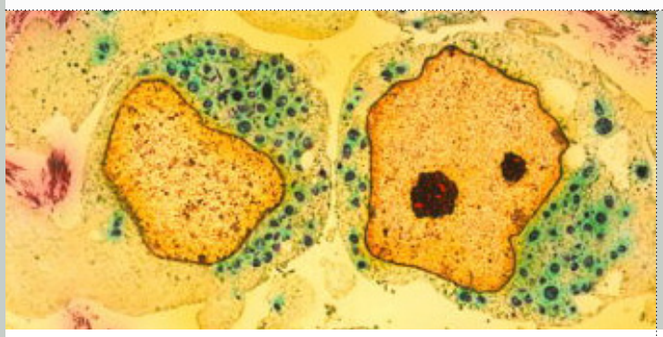


The markers usually checked are;

· *AFP or alpha feta protein*
· *HCG or human chorionic gonadotrophin*
· *LDH or lactate dehydrogenase*

Treatment of Testicular Cancer

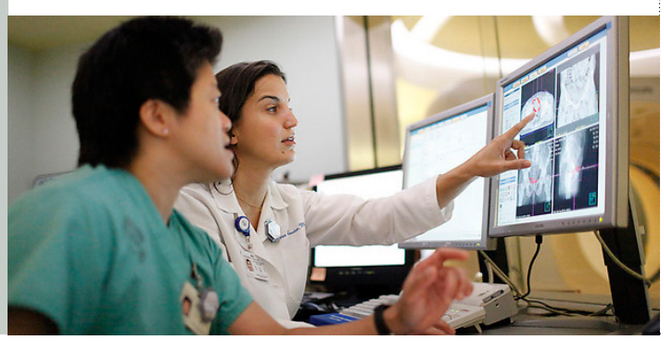
The mainstay of testicular cancer treatment remains orchidectomy. This must be done through the inguinal approach. As well as providing the definitive primary treatment it also informs the surgeon and the extended oncology team about the tissue type. Orchidectomy is curative in around 80% of cases.

Orchidectomy: This involves excision of the testis, epididymis and the cord, as well as their coverings, through the inguinal incision.

The whole groin and scrotum should be prepared and draped with appropriate antiseptic solution. After groin dissection to identify the cord and other structures, the cord is clamped, and then transfixed with a strong (eg Vicryl™) suture. The testis is then manipulated into the groin wound. The testis is removed, and it may be appropriate to place a silicone prosthesis back into the scrotum at this time.

Adjuvant therapy: Adjuvant therapy is provided on a patient by patient basis according to disease stage, tissue histology and extent of metastatic spread. Both chemotherapy and radiotherapy are used in combination.

Summary: Our 22 year old student should have been cured based on the clinical and histological findings, as well as the finding of no metastatic disease. He should be closely followed up for the next few months, and should subsequently see a uro-onocolgist based on local policy guidelines for follow up.



Testicular Cancer: Review
Urology

Extended Matching Questions

1. A 45 year old man is referred as a 2 week wait from his GP with a lump in the left testis. Clinically the appearances suggest a varicocele and a prompt ultrasound testes confirms this.

Further action should be

a) Reassurance
b) Surgery to correct varicocele
c) Renal Imaging
d) Varicocele embolisation
e) Discharge

2. A 30 year old man is referred to urology clinic with bilateral inguinal lymphadenopathy and night sweats. On examination he has palpable inguinal lymph nodes and on further assessment has a couple of palpable cervical lymph nodes.

Likely Diagnosis

a) Seminoma
b) Non-seminomatous germ cell tumour
c) Tuberculosis
d) Lymphoma
e) Mumps

3. A 27 year old surgical SHO is referred to urology clinic by his GP after noticing a Lump in his right testicle. When a detailed history is taken in clinic he admits to symptoms of gynaecomastia. Possible explanations of his gynaecomastia are:

a) Alcoholic secondary to stress causing liver cirrhosis
b) Klinefelter's Syndrome
c) Non-seminomatous germ cell tumour (Teratoma)
d) Seminoma
e) Hyperthroidism

TESTICULAR CANCER: REVIEW

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4. A 30 year old man has undergone a right radical orchidectomy for what was found to be a Non-seminomatous germ cell tumour. Histology and imaging are complied to give a staging result. This shows tumour growing into blood vessels and lymphatics and is present in the spermatic cord. There was one lymph node found to contain cancer cells of those dissected, which was 12mm in size. No distant metastasis were found and LDH was mildly raised pre operatively.

What is the stage of this cancer on the TNM scale?

a) T3N1M0S0
b)T3N2M0S1
c) T2N1M0S1
d) T3N1M0S1
e) T2N0M0S0

5. Seminomas are generally sensitive to

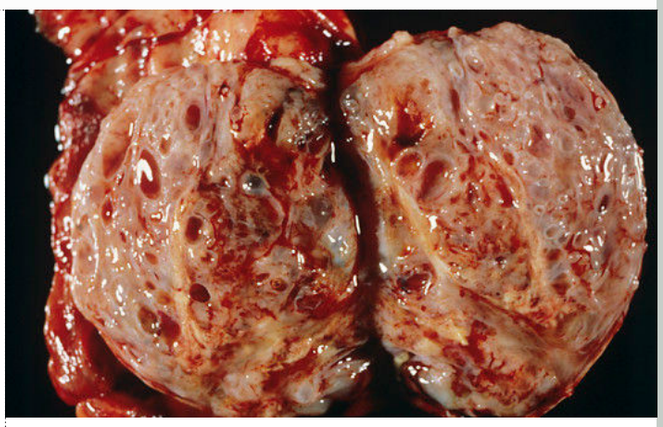
a) Radiotherpay
b)Chemotherapy
c) Both
d) Neither

Answers

1-c) Renal Imaging
A left varicocele can represent a blockage of the venous drainage of the left testicle at the left renal vein possibly due to renal malignancy. Detailed knowledge of the anatomy of the testicles including their blood supply and lymphatic drainage is needed for proper assessment.

2-d) Lymphoma
Testicular lymph drainage follows its blood supply and drains into the para-aortic nodes. Understanding of the lymphatic drainage of the testes is needed for proper assessment. This patient requires a FBC and ESR, as well as USS guided biopsy of his enlarged lymph nodes.

3-c) Non-seminomatous germ cell tumours
All are possible but with the addition of testicular lump a tumour secreting B-hcg is the most likely. Seminomas produce B-hcg in up to 30% of cases and Non-seminomatous germ cell tumours in 60% of cases making this the most likely diagnosis.



4-d) T3N1M0S1

5-c) Both
Seminomas are extremely radio and chemo sensitive and prophylactic doses of both do reduce recurrence rates even in stage 1 disease. However survival rates are not altered by the use of prophylactic therapy if adequate surveillance is done post surgical treatment. If relapse is detected, treatment with radiotherapy or chemotherapy, still gives close to 100% survival in stage 1 disease.

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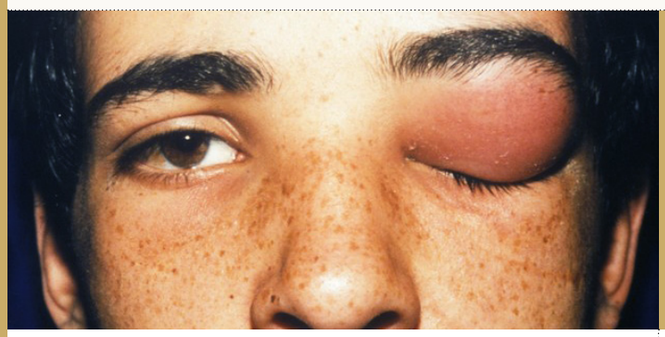
MANAGEMENT OF PERIORBITAL & ORBITAL CELLULITIS

B Dhillon



Management Of Periorbital & Orbital Cellulitis
Otorhinolaryngology & Neck Surgery

Abstract

Periorbital and orbital sepsis (including cellulitis and abscess) is a potentially sight-threatening condition and needs to be identified early and treated aggressively. The most common cause is acute rhinosinusitis however other non-ENT causes must be considered. Management and assessment is best considered on a shared care basis with Ophthalmology and Paediatrics (in children). Full and continual assessment of the eye is mandatory. Antibiotic therapy has dramatically improved the morbidity and mortality from this condition.

Surgical decompression needs to be considered when there is optic nerve compromise or evidence of abscess. This can be approached endoscopically or externally. CT scanning remains the imaging modality of choice as it demonstrates bony and sinus pathology well and is easily accessible out-of-hours. MRI scanning can be considered if intracranial complications are suspected. CNS problems tend to be late signs and carry worse prognosis.

Keywords: orbital cellulitis; periorbital cellulitis; sinusitis

Clinical Vignette

A 12 year-old boy attended the Emergency department with a swollen left eye that has been progressively worsening over the past 48 hours. He complains of coryzal symptoms for the past week, and has only been on simple analgesics and anti-pyretics. On examination both upper and lower eyelids are tender, erythematous and oedematous, you are unable to open the eyelids fully to examine the pupils, but the patient admits to being able to see light when you are examining.

Anterior rhinoscopy shows obvious nasal mucosal inflammation with pus in the left nostril. The child is pyrexic at 38°C but is haemodynamically stable. He has no other relevant medical history, no known allergies and is not on any regular medication.

Introduction

Orbital and periorbital sepsis are well-recognized complications of sinusitis which carry a significant morbidity. The reported incidence of sub-periosteal abscess in orbital infections has been reported at 15% (1). Furthermore, 15-30% of these patients will develop various visual sequelae despite aggressive management (2). It is far more common in the paediatric population and young adults - up to 76% of affected patients are under the age of 20 years (3) and up to 50% under the age of 6 years (4).

Frequently it is secondary to acute sinusitis, however one must consider alternative causes which include odontogenic infection, trauma, insect bites, conjunctivitis, dacrocystitis and blepharitis. Some other rarer causes that have been reported include lymphoma, malignancy and some tropical infections such as leishmaniasis which should be considered in atypical presentations of the problem.

In cases of acute sinusitis, infection commonly spreads from the ethmoid air cells through the paper-thin lamina papyracea. Infection can also spread from the floor of the frontal sinus and from the roof of the maxillary antrum (5). The valveless communication between the orbital/frontal venous plexuses with the cavernous sinus leads to indirect spread of infection and subsequent cavernous sinus thrombosis.

Relevant Anatomy

The paranasal sinuses are very closely associated with the orbit and periorbita. Figure 1 (a, b, c) demonstrates the locations of the frontal, maxillary and ethmoid air cells in relation to the orbit.

MANAGEMENT OF PERIORBITAL & ORBITAL CELLULITIS

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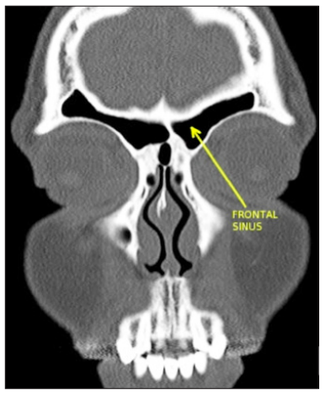
a)



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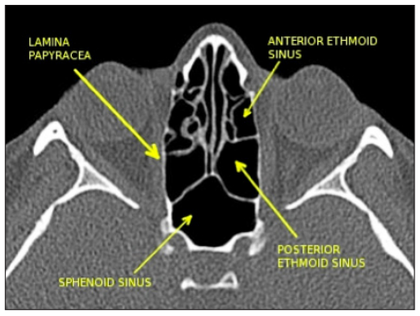

c)

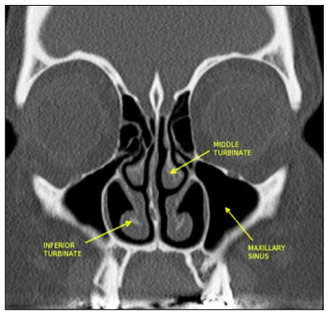

Figure 1: Frontal (a), ethmoidal (b) and maxillary (c) sinuses as shown on computerised tomography (CT)

The anterior and posterior ethmoid air cells lie immediately medial to the orbital compartment and are separated from the orbit by the lamina papyracea which is paper-thin. The frontal sinuses lie superior to the orbital compartment and are separated by a thicker shelf of bone. The maxillary sinuses lie inferior to the orbital compartment. The sphenoid sinuses lie posteriorly in the nose and are closely associated with the optic nerve.

Clinically, periorbital sepsis is usually discussed as being either pre-septal or post-septal. The orbital septum was identified by Chandler et al (6) as an important landmark and is described as a fascial extension of the orbital rim periosteum that extends to the tarsal plates of the upper and lower eyelids (7). It is the only barrier that impedes the spread of infection from the eyelids to the orbit (5). This anatomical relationship is schematically represented in Figure 2.

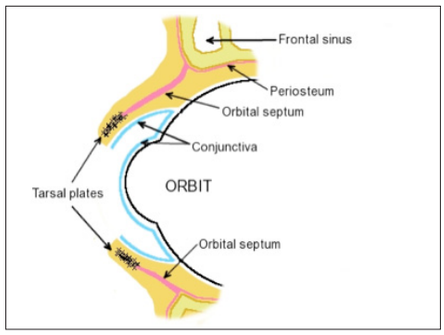

Figure 2: Schematic representation of the orbital septum.

The orbital septum and bony orbit form a rigid box; hence any uncontrolled swelling in post-septal cellulitis effectively creates an orbital compartment syndrome contributing to a higher risk of ocular morbidity (8).

Clinical Presentation And Assessment

Patients will present with obvious unilateral eyelid oedema which may also be tender, firm and erythematous (Figure 3). Usually there is also a concurrent or preceding upper respiratory tract infection with nasal obstruction and nasal discharge. The patient may also be septic.

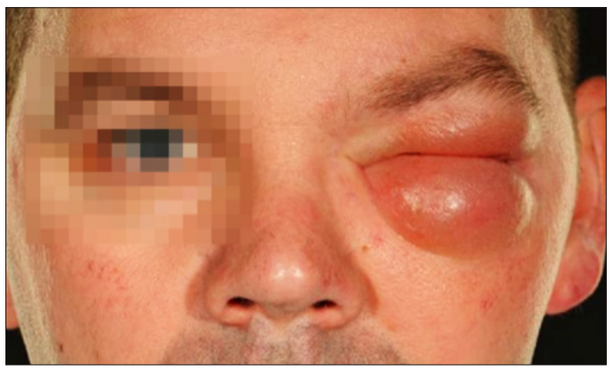

Figure 3) Clinical presentation or periorbital cellulitis.

History taking should be focused on duration of symptoms, recent treatment, history of URTI and trying to ascertain any other causes apart from acute sinusitis. Important features which are suggestive of acute sinusitis include nasal obstruction, nasal discharge, headache, facial pain and pyrexia.

A full ENT and eye examination is mandatory. ENT examination should include nasal examination to look for purulent discharge from the sinus drainage pathways and nasal mucosal oedema (Figure 4). You must look for and document eye examination findings to include diplopia, visual acuity, colour vision, pupillary reflexes, proptosis and ophthalmoplegia. Snellen charts (for acuity) and Ishihara charts (for colour vision) are useful tools to standardize documentation. Central nervous signs (CNS) signs such as drowsiness, vomiting, headache, seizure or cranial nerve lesions imply intracranial extension and are late and worrying signs.

MANAGEMENT OF PERIORBITAL & ORBITAL CELLULITIS

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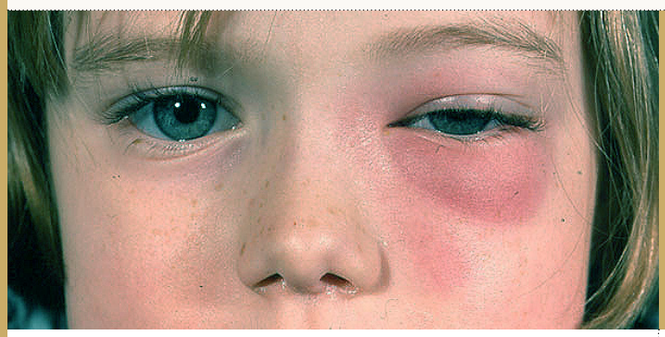

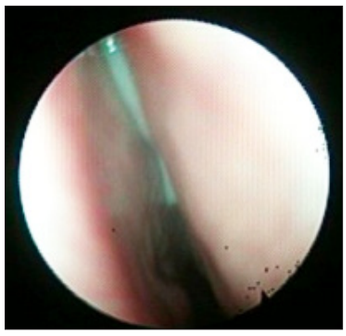

Figure 4) Mucopus evident within the nasal cavity on nasendoscopy.

Chandler's classification (6) is widely used to distinguish between the types of periorbital inflammation and infection (Table 1). While there is clearly a graduation between stages, it is important to note that one stage does not necessarily lead to the next (5). Furthermore, there is no clear correlation between the extent of spread and the clinical signs, making determining between the stages difficult (5). Often the classification is confirmed only after imaging.

Stage	Description	Features
1	Inflammatory oedema (pre-septal disease)	Swelling of eyelid, remains anterior to orbital septum, usually non-tender with no other ocular signs or symptoms
2	Orbital cellulitis	Posterior extension aross orbital septum; oedema of orbital contents, proptosis, chemosis, ophthalmoplegia ± visual disturbance
3	Subperiosteal abscess	Collection of pus between orbit and periosteum, potential visual loss and proptosis
4	Orbital abscess	Complete ophthalmoplegia, visual loss, marked proptosis
5	Cavernous sinus thrombosis	Bilateral proptosis and periorbital oedema with ophthalmoplegia, loss of vision and meningism (late signs)

Table 1: Chandler classification with summary of important features

Management

Admission

According to Howe and Jones (5) the only patient who is safe for discharge and outpatient management with oral antibiotics should have minimal upper lid oedema, normal eye examination and is systemically well with no CNS signs or symptoms. All other patients and including those in whom it is not possible to examine the eye should be admitted (5). The indications for admission as advised by Howe and Jones are summarized in Table 2.

Majority of patients with periorbital swelling
Proptosis
Diplopia or ophthalmoplegia
Reduced visual acuity
Reduced light reflexes or abnormal swinging light test
For those in whom a full eye examination is not possible
Toxic or systemically unwell
Central nervous system signs or symptoms

Table 2: Criteria for admission (5)

Primarily these patients are managed under the care of ENT when the cause is sinogenic. In other causes, an ENT opinion is often sought to rule out sinusitis.

When the cause is sinogenic, the current consensus supports shared care to include the Ophthalmologists and Paediatricians as appropriate (5). Ophthalmology opinion should be sought as an emergency in situations where the eye examination is difficult or findings are inconclusive. Eye observations should be continued twice daily (or more frequently if needed) during the inpatient stay, and include testing for colour vision (using Ishihara charts) and acuity (using Snellen charts), eye movements and pupil reflexes.

MANAGEMENT OF PERIORBITAL & ORBITAL CELLULITIS

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Investigations

Baseline investigations would be the same as for any patient with sepsis. This includes full blood count, urea and electrolytes and C-reactive protein.

Blood cultures have been reported to have a variable rate of positive yield. In children, there is a reported yield of between 0% and 42% (9, 10). The positive yield is even poorer (5%) in adults (11). Still blood cultures are considered more likely to be positive in younger patients and if taken prior to administration of antibiotics (5). Certainly, antibiotic therapy is commenced empirically before results are available.

Other methods of pathogen isolation that have been tried include culture of eyelid aspirates or eye secretions, sinus aspirates, and swabs from mouth, nose or oropharynx. The evidence to support these methods is poor, and there is a high rate of commensal contamination with these methods (12). Lumbar punctures are only to be considered with CNS involvement and when raised intracranial pressure is excluded.

Imaging

Plain radiographs were used historically prior to the advent of computed tomography (CT). While they can show sinus opacification, they do not provide information on orbital collections.

Contrast enhanced CT scanning is a useful diagnostic aid (8) and is the imaging modality of choice as it can define the extent of periorbital oedema and collections, as well as visualise the sinuses simultaneously (see Figure 5, 6 and 7).

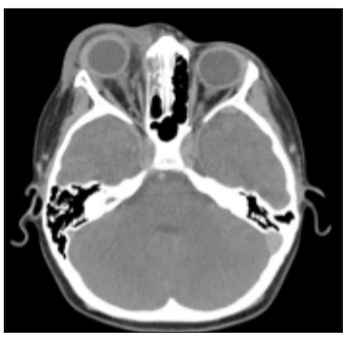
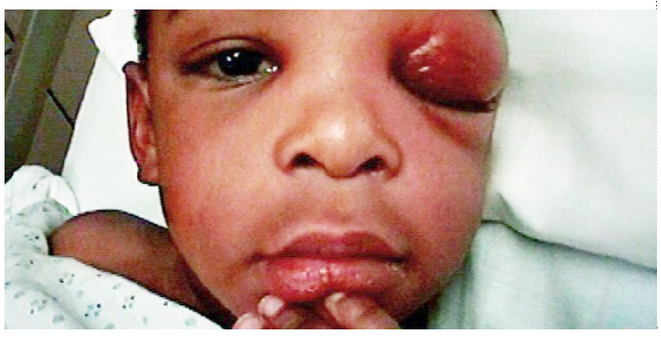


Figure 5 : Axial CT Figure showing eyelid oedema with proptosis of the right eye. Note the opacification within the adjacent ethmoid air cells.



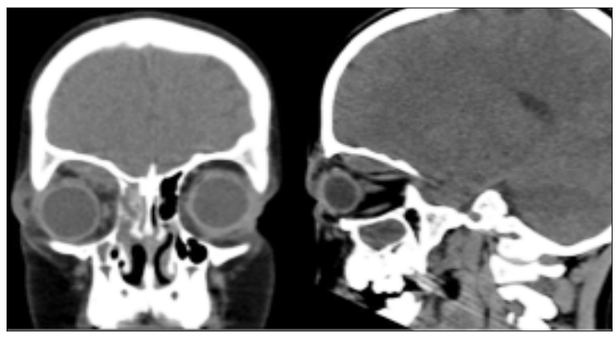


Figure 6 : Coronal and sagittal CT Figures showing a right sided supero-medial orbital abscess. There is also adjacent ethmoid air cell opacification and breach of the lamina papyracea. The eye is obviously displaced laterally as well.

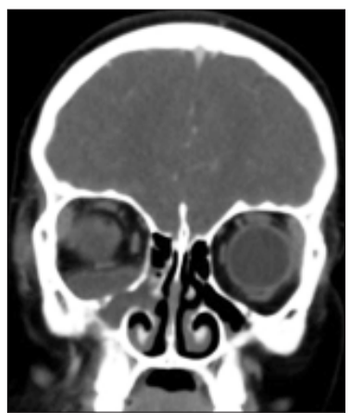


Figure 7: This coronal CT Figure demonstrates an inferior orbital abscess with adjacent maxillary sinus opacification. There is a breach in the orbital floor which has allowed the infection to track into the orbit. The inferior rectus muscle is displaced superiorly.

CT imaging also helps to differentiate abscesses from orbital cellulitis (13). The point of contention regarding imaging is about when to Figure and when to repeat imaging. Howe and Jones (5) advise some main indications for scanning that are summarized in Table 3. Imaging is certainly advocated in the emergency setting if adequate examination is difficult or there are CNS signs. In patients who are stable with no worrying eye signs, CT can be considered if there is no improvement or deterioration after 24 hours of treatment.

The appropriate imaging request would ensure best quality of Figures - CT orbits / sinuses are the protocol required with additional CT head if suspecting intracranial complications. The disadvantage of CT is the need for radiation exposure and especially in children the added risk of sedation which is why it is not considered a first line investigation in all cases of orbital and periorbital sepsis (5).

MANAGEMENT OF PERIORBITAL & ORBITAL CELLULITIS

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Magnetic resonance imaging (MRI) is mainly considered to be superior to CT in the evaluation of intracranial complications such as cerebritis and cavernous sinus thrombosis (14). It does not however have much of a place in the emergency imaging of periorbital cellulitis owing to the difficulty in accessing MRI services out of hours and it takes significantly longer to perform than CT. MRI also does not provide adequate bony definition and demonstration of osteitis in relation to sinusitis. Diffusion-weighted imaging (DWI) shows some promise in differentiating inflammatory conditions from abscess but due to small numbers in reported series and the lack of access to this specific modality of MRI it again has less of a place in the emergency management of periorbital cellulitis (15).

Orbital ultrasound has been described as a technique that is well tolerated by children and in one small series was able to demonstrate collections when CT was inconclusive (16). Ultrasound is however very operator dependent, and orbital imaging may not be as ubiquitous as CT or MRI and is not able to visualize the posterior 1/3 of the orbit. Similarly it will not provide much information on the bony anatomy and sinus involvement.

CNS signs
Unable to accurately assess vision
Gross proptosis, ophthalmoplegia, bilateral oedema
No improvement or deterioration at 24 hours
Swinging pyrexia not resolving within 36 hours
Deteriorating visual acuity or colour vision (only image if it would not delay operative decompression which is a priority)

Table 3: Indications for CT scanning (5)

Medical Management

Antibiotics

The common organisms implicated in orbital and periorbital sepsis are Streptococcal species such as Streptococcus milleri, pyogenes, viridans and pneumoniae, as well as Staphylococcus aureus, Staphylococcus epidermidis, and Klebsiella pneumonia (3, 5).

Anaerobic organisms are also significantly isolated - in up to 24% of orbital collections, and 45% of intracranial collections reported in one series (17). Haemophilus influenza B is implicated less now since the advent of the HiB vaccine (17), but is still seen on occasion especially in the very young (2, 5).

The use of antibiotics is not in dispute, but there is inconsistency in the choice, route of administration and duration in the literature and clinical practice.

The emergence of resistance has prompted a move to using broad spectrum cephalosporins (5). There is strong evidence that anaerobic cover from the outset is useful which is why Howe and Jones advocate the use of metronidazole in combination with cefuroxime (5).

In patients requiring admission, an intravenous route of administration would be beneficial in maximizing systemic absorption. The antibiotic choice should be tailored to the culture results once these are available. It is best practice to consult your local hospital's antibiotics policy or involve the Microbiologists to determine the most suitable choice of antibiotic.

Other medications

Nasal decongestants aim to reduce mucosal oedema and are inferred to improve drainage of the sinus pathways affected by disease. Howe and Jones advocate using Ephedrine 0.5% nose drops (5).

One study looking at the use of systemic steroids could not show any significant benefit with its use (18).

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Surgical Management

Surgical drainage is required in the presence of radiologically demonstrated orbital abscess or acute evidence of optic nerve compromise (reduced visual acuity or loss of colour vision). Optic nerve compromise is a surgical emergency and needs decompression urgently - seconds count to avoid irreversible blindness! Do not wait to Figure these patients.

The method of decompression or surgical management varies based on surgeons' experience and location of the abscess, but overall is divided into endoscopic and open approaches.

Endoscopic approach

The main surgical objective is to drain the pus adequately, release the pressure on the orbit and obtain a culture (5). Endoscopic sinus surgery is performed intranasally using rigid nasendoscopes and appropriate instruments to open the sinus cavities and improve drainage (Figure 8). It is useful in dealing with ethmoid sinusitis as well as medially placed abscesses, which can be dealt with by removal of the lamina papyracea and an ethmoidectomy (19).

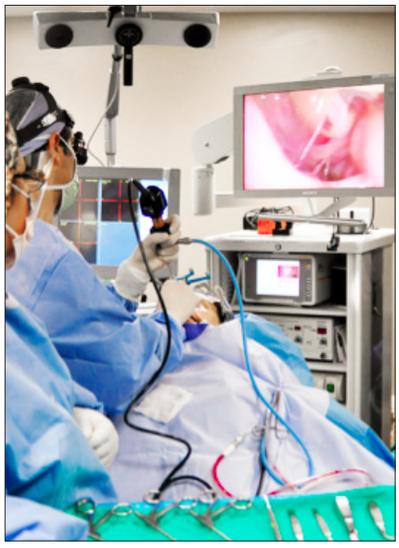

Figure 8: Functional endoscopic sinus surgery using radiology navigation assistance (© Photographer - James C. Mutter / Otolaryngologist Mani Zadeh, MD) [Online] Available from http://commons.wikimedia.org/wiki/File:Mani_Zadeh_MD_Endoscopic_Sinus_Surgery.jpg [Accessed 29 July 2012]

There remains controversy about the extent of resection required and whether a full sinus dissection should be carried out at the same time. One study from Egypt suggested that with medially placed orbital abscesses there was equal benefit from either limited or wide excision of the lamina papyracea (20). Further case series reporting small numbers advocate anterior and posterior ehtmoidectomy, uncinectomy and middle meatal antrostomy in addition (21). Similarly the extent of dissection of the ethmoid air cell complex is debated with some authors advocating limited (22) or full dissection (23).

This method does lead to some difficulty as the inflamed nasal mucosa is prone to bleeding intraoperatively which can significantly obscure the surgical field. Furthermore, in children there is limited maneouvering space

which makes surgery challenging for the less experienced surgeon (21).
The endoscopic route will also not deal with the more laterally or superiorly based abscess where instead an external or combined approach may need to be considered (5, 24, 25).

External approach

The traditional approach to drain orbital collections externally is via the Lynch-Howarth incision (8). The incision is made on the side of the nose, medial to the orbit (Figure 9).

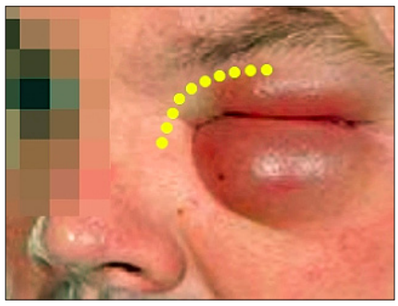

Figure 9: Location of Lynch-Howarth incision for external approach.

Elevation of the orbital periosteum will expose the medial orbit and abscess cavity. The lamina papyracea can also be fractured to create a drainage passage. Extension of the initial incision can also allow access to any superior collections. Drains can be considered either opening externally or intranasally, however there is very little evidence for this in the literature (8).

External drainage has the advantage over endoscopic drainage in that it avoids the risk of intranasal adhesions or stenosis of the frontal recess (5). It does however result in a scar over the face that can be complicated by disfiguring webbing (8). The extent of this type or surgery is very dependant on the comfort level and experience of the surgeon.

MANAGEMENT OF PERIORBITAL & ORBITAL CELLULITIS

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Management Of Periorbital & Orbital Cellulitis
Otorhinolaryngology & Neck Surgery

Prognosis

In the pre-antibiotic era, there was significant morbidity and mortality from orbital and periorbital sepsis. 17% of cases resulted in death and 20% resulted in blindness (7, 26). Today fortunately intracranial complications and blindness are rare events occurring in less than 1% of patients (7).

Conclusion

Care of patients with orbital and periorbital sepsis is best managed in a multi-disciplinary setting. Full assessment of the eye and ENT systems are mandatory from time of presentation and should continue to be observed during the inpatient admission. Antibiotics have significantly improved the morbidity and mortality associated with this condition. However there is still a place for urgent surgical intervention when the optic nerve is at risk.

Key Points

• Full assessment of the eye is mandatory

• Seek help if you are unable to assess the eye

• Reduction in visual acuity / colour vision requires urgent surgical decompression to avoid blindness

• Aggressive broad spectrum antibiotics should be commenced early, preferably with advisement of local microbiology

• Involve your seniors in the management from early on

MCQs

1. Which of the following would be the earliest sign of optic nerve involvement in someone with periorbital cellulitis?

a. Proptosis
b. Loss of colour vision
c. Reduced visual acuity
d. Ophthalmoplegia
e. Retro-orbital pain

2. You are assessing a child with periorbital cellulitis in A&E. On examination of the eye, the colour vision appears to be reduced in the affected eye. What would be the next step in the management of the child?

a. Admit for IV antibiotics
b. Arrange a CT scan of the orbits
c. Contact Paediatricians / Ophthalmologists for review
d. Contact seniors immediately for consideration of surgical drainage / decompression
e. Re-examine after 15 minutes

3. In the management of a patient with periorbital cellulitis, which of the following is UNTRUE?

a. Topical nasal decongestants should be commenced immediately
b. Patients should be managed in a multidisciplinary / shared care setting
c. Ophthalmology opinion should be sought if unable to examine eye properly in A&E
d. All patients should be admitted under ENT
e. Severe headaches are a delayed sign

4. Regarding imaging, which of the following is UNTRUE?

a. There is no Gold Standard investigation
b. MRI is good at assessing sinusitis
c. CT scanning can provide good information on intracranial extension of the abscess
d. Orbital ultrasound is well tolerated by children
e. Plain X-rays should be performed in A&E to assess for collection

MANAGEMENT OF PERIORBITAL & ORBITAL CELLULITIS

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5. Which of the following bacteria are implicated in periorbital sepsis?

a. Streptococcus pyogenes
b. MRSA
c. Moraxella cattarhalis
d. Klebsiella pneumonia
e. All of the above

Answers

1. b (colour vision is affected before visual acuity - always check for red-green colour vision when assessing eye)

2. d (any delay will lead to irreversible optic nerve damage)

3. d

4. b, e (plain X-rays do not provide any useful information and expose to radiation unnecessarily)

5. e (all these pathogens can cause rhinosinusitis)

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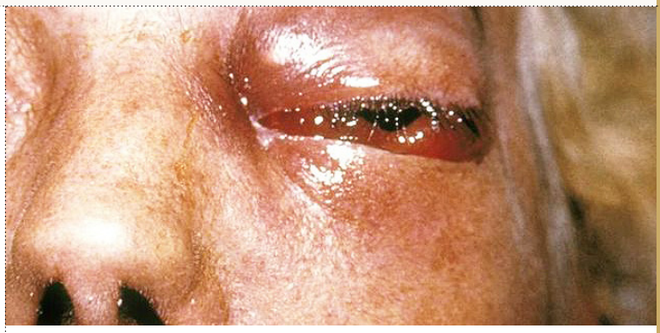

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PYLORIC STENOSIS

J Ashraf, A Mahomed

Pyloric Stenosis
Paediatric Surgery

Abstract

Hypertrophic pyloric stenosis (HPS) is a common cause of gastric outlet obstruction in infants and presents as one of the most common surgical conditions of infancy. The pylorus part of the stomach becomes thickened and presents as gastric outlet obstruction. Cause of HPS is still debated but genetic and environmental factors appear to play a major role in the pathophysiology of this condition. Ultrasound has become the benchmark technique for the diagnosis of HPS whereas surgical pyloromyotomy remains the standard treatment with an excellent outcome. This review article includes diagnostic workup including physical examination, bloods and imaging, preoperative resuscitation, various surgical approaches used, intra and postoperative complications.

Key words: Pyloric stenosis; Infant; Resuscitation; Pyloromyotomy

Introduction

Hypertrophic Pyloric Stenosis (HPS) is the commonest cause of surgical vomiting in infancy (1). Fabricious Hildanus first described HPS in 1627 with later accounts given by Blair in 1717 and Beardsley in 1788 (2). It was not until 1888 when the Danish paediatrician Harald Hirschsprung's described the post mortem clinic-pathologic findings and introduced the term congenital pyloric stenosis, that the condition became well known (3). He described the key features based on two infants who died from the disease in 1888.

Epidemiology

Incidence of HPS is about 1- 4 per 1000 live births in western populations and is less common in the African and Asian population (4,5,6,7), with male-to-female ratio of 4:1. HPS appears to be more common in first born male babies, bottle fed infants, in rural populations, younger mothers, where there is a positive family history and with a link to maternal feeding patterns (6, 9, 10) The condition typically presents at post natal age of 3 to 6 weeks and is rare beyond 4 months of age. Premature infants are diagnosed with HPS later than term or post- term infants (7).

Aetiology

The aetiology of HPS is still debated but genetic and environmental factors appear to play a major patho physiological role. Variations in HPS incidence over time along with the type of feeding (breast vs. formula), maternal erythromycin use, seasonal variability and trans pyloric feeding in premature babies suggests a possible impact of environmental factors. Genetic predisposition is suggested by race discrepancies, higher incidence in males, and increased incidence in first born male babies with a positive family history. There is a 7% incidence of HPS in children of affected parents with a 4 fold risk if the mother is affected (7, 8).

Clinical Presentation

HPS is characterized by hypertrophy of the inner circular muscle layer with consequent lengthening and narrowing of the pyloric canal manifesting in gastric outlet obstruction. Typical presentation of HPS is non bilious, projectile vomiting in a term infant between 2 to 8 weeks old. At first, vomiting is infrequent but gradually emesis becomes regular and projectile. Vomitus consists of recent feeds but occasionally contains 'coffee grounds' due to altered blood secondary to gastritis. In preterm infant symptoms can be quite atypical.

Differential Diagnosis

Various other causes for non bilious vomiting should also be kept in mind including gastro oesophageal reflux, gastroenteritis, raised intracranial pressure, congenital adrenal hyperplasia, metabolic disorders, antral web, foregut duplication cysts, gastric tumour or even extrinsic antral compression by a tumor.

Physical Examination

Gastric peristalsis may be observed in the left upper quadrant. However, palpation of the hypertrophied pyloric mass or 'olive' in the epigastrium or right hypochondrium (usually around the lateral margin of the right rectus abdominis below the liver edge) is key to clinical diagnosis with a 99% positive predictive value. To palpate the olive, baby must be relaxed so hips should be flexed to loosen the abdominal wall while examining from the left, utilising a pacifier such as sugar water. A distended stomach obscures the tumour so it is easier to feel the olive after the stomach has been decompressed with an 8 Fr nasogastric tube. However, if after a physical examination, the diagnosis is still unclear, then radiological evaluation is warranted.

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Investigations

Repeated vomiting due to gastric outlet obstruction leads to loss of hydrogen (H+), sodium (Na+), chloride (Cl-), potassium (K+) and water, leading to dehydration and hallmark metabolic derangement of hypochloraemic, hypokalaemic metabolic alkalosis.

Bloods: Serology usually demonstrates, low (Đ) Na+, Đ K+, ĐCl-, ĐUrea, ĐCreatinine, ĐHCO3-, ĐpH. Reference values for HCO3- are as follows: mild <25 mmol/L range, moderate 26-35 mmol/L range, severe> 35 mmol/L ranges.

Ultrasound: Ultrasound is non- invasive and quick, without radiation exposure and has become the standard technique for diagnosing HPS. The accuracy approaches 100% if performed by an experienced radiologist (sensitivity of 99.5%, specificity of 100%). Diagnostic ultrasound dimensions of a pyloric tumour are muscle thickness >4 mm with pyloric canal length > 16mm (10) (Figure 1). A muscle thickness of more than 3 mm is considered positive if the neonate is younger than 30 days of age (11). Real time observation of failure of relaxation of the pyloric canal is also an important feature.

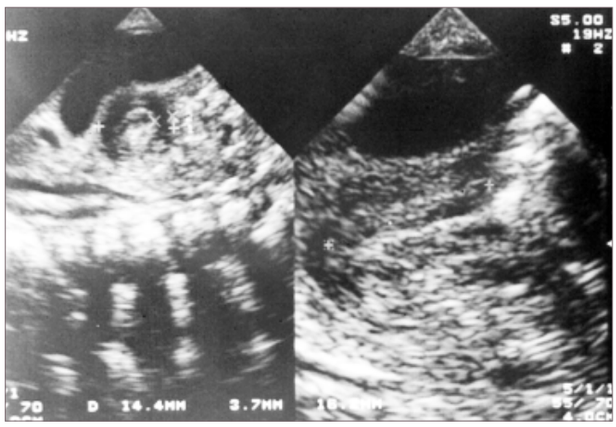

Figure 1: Transverse (L) and longitudinal (R) sections of enlarged pylorus as seen on Ultrasound.

Upper Gastrointestinal Contrast: If ultrasound findings are equivocal then an upper gastrointestinal contrast can help in making the diagnosis, but is not warranted as a routine. Diagnostic features of pyloric stenosis include; 'string sign' of narrowed and elongated pyloric canal, (Figure 2) double track in the pyloric canal due to infolding mucosa, delayed gastric emptying, gastric hyper peristalsis or the mushroom effect in the duodenal cap because of proximal indentation by the pyloric tumor. Aspirating the contrast agent post study is critical to avoid tracheal aspiration and disastrous pulmonary complications.

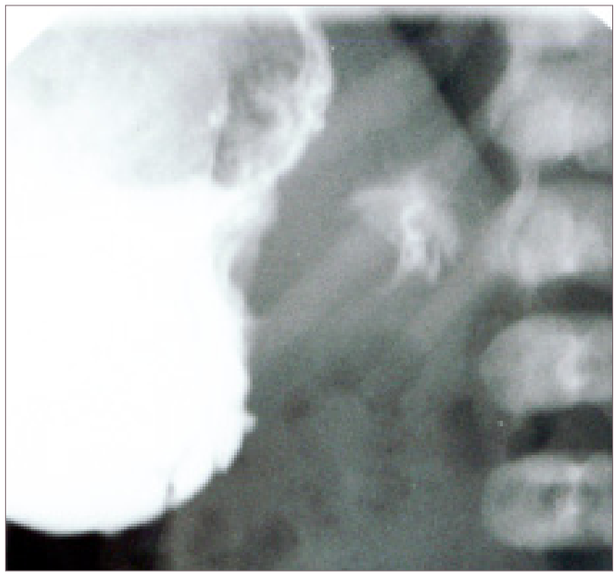

Figure 2: Contrast study demonstrating 'string sign' and shouldering effect of pyloric stenosis.

Treatment

Resuscitation

HPS is not a surgical emergency but rather a medical one and initial resuscitation is critically important. The degree of dehydration is estimated by clinical examination, urine output and serum chloride and bicarbonate estimates. Surgery should be deferred until the baby is appropriately resuscitated by intravenous fluid replacement usually consisting of 5% dextrose in 0.45% saline at 1.25 to 2 times the maintenance level. Severely dehydrated babies should initially receive 10 to 20 ml/kg fluid boluses with isotonic normal saline. After an adequate urine output has been demonstrated, 10 to 20 mEq/L of potassium chloride can be added to the fluids. Assessment of adequate resuscitation is determined by normalising serum electrolyte levels, moist mucous membranes, good skin turgor and an adequate urine output. Blood glucose monitoring is also important.

Once the diagnosis of HPS is made, the patient is kept nil by mouth and a nasogastric tube inserted for gastric decompression. Most infants are ready for surgery in 24 hours but prolonged vomiting may involve resuscitation for a couple of days. For a safe anaesthetic, serum bicarbonate less than 28 mEq/dl, serum chloride over 100mEq/dl and potassium of 4.5 to 6.5 mmol/L are generally acceptable.

Medical Treatment

There are reports of medical treatment with atropine but patients require prolonged hospitalisation and with results rather unpredictable is of historic and research interests only (12-16).

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Surgery

Open Surgery

The first surgical treatment performed by Löbker in 1898 was a gastroenterostomy bypass of the pyloric obstruction (17). Extramucosal pyloroplasty was demonstrated by Dufour and Fredet in 1908, and then modified to solely a muscle-splitting procedure by the early 20th century. Extramucosal pyloromyotomy became the procedure of choice when German military surgeon, Conrad Von Ramstedt, described two successful cases in 1912 (18,19). Historically various other surgical approaches have been used including midline laparotomy (Fredet 1908), oblique right upper quadrant incision (Robertson 1949), right upper abdominal transverse muscle splitting incision (Rickham 1940). Tan and Bianchi presented their cosmetically pleasing omega shaped circum-umbilical approach for pyloromyotomy in 1986. (Figure 3) Various modifications to improve this technique including the addition of squeeze technique in delivery of the pylorus have subsequently been described (20).

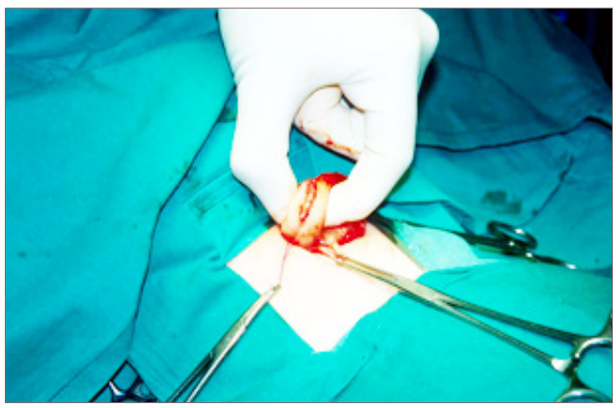

Figure 3: Trans umbilical pyloromyotomy.

Regardless of incision utilised, the pyloric tumour is pulled out through the wound, a longitudinal serosal incision is made on the anterior surface of the pylorus approximately 2 mm proximal to the junction with the duodenum and carried proximally onto the anterior gastric wall for approximately 5 mm. Muscle fibres are then spread completely till mucosal protrusion is seen.

Complete pyloromyotomy can be assessed by demonstrating mobile myotomy margins. Mucosal integrity may be checked by instilling air into the stomach through the nasogastric tube whilst instilling saline over the exposed pyloric mucosa and observing for bubbling. Unrecognized mucosal perforation will lead to peritonitis and sepsis. Air should be sucked out if no leak is found. Post pyloromyotomy the tumour is returned to the abdomen and the incision is closed in layers.

Laparoscopic Surgery

Laparoscopic Pyloromyotomy was first described by Alain and co-workers in 1991 (21), since then this procedure has gained popularity amongst paediatric surgeons and has become the standard technique in many centres. Recent randomized prospective trials have shown acceptable complication rates compared to the open approach (22,23). An umbilical incision is used to introduce either a 3 mm or 5 mm camera port into the abdomen. Two further stab incisions in right and left upper quadrant is made before introducing a bowel grasper and arthrotomy knife. The duodenum is held firmly just distal to the pylorus with grasper and myotomy executed with blade as for the open technique. A laparoscopic pyloric spreader is then used to complete the myotomy. (Figure 4)

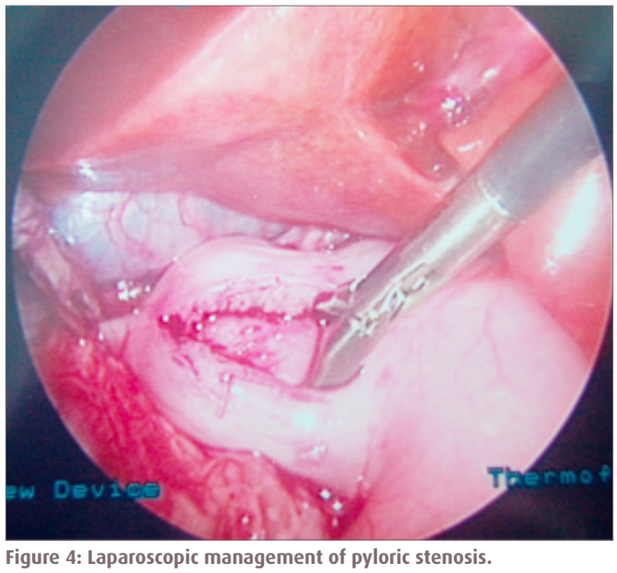

Figure 4: Laparoscopic management of pyloric stenosis.

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Post Operative Care

Postoperative care is similar for both open and laparoscopic techniques. Recent studies have suggested ad libitum feeding in the early postoperative periods as it results in a faster time to discharge (24, 25). Vomiting maybe seen postoperatively and feeds may need to be intermittently stopped and then re started. Babies requiring prolonged resuscitation to correct metabolic derangements take longer to tolerate full feeds (26). Most babies go home on the first postoperative day. If duodenal perforation occurs in theatre then nasogastric aspiration with intravenous antibiotic cover is continued for at least 48 hours.

Complications

Reported complications of pyloromyotomy include mucosal perforation (1 – 2%), wound infection (1-2%) (22, 23), incisional hernia (1%) (22), prolonged postoperative vomiting (2 to 26%), incomplete myotomy (0-6%) (23) and injury to duodenum (27). If mucosal perforation is at the duodenopyloric junction, it can be closed with interrupted absorbable sutures. If the perforation is large or in the middle of the myotomy, then the myotomy should be closed and a new incision made 90 to 180 degrees from the original. Persistent postoperative vomiting is usually caused by too rapid advancement of postoperative feeds, gastro oesophageal reflux, residual pyloric edema or even incomplete pyloromyotomy. The requirement for re-pyloromyotomy is around 2% (27).

Outcome

Mortality from pyloric stenosis was quite high in the past approaching 50% however currently it is nearly zero owing to much improved resuscitation, anaesthesia and surgical technique. Morbidity also has dropped significantly, now ranging between 1 – 2% of overall complication rate (27).

Clinical Vignette for MCQs

A 4 week old male baby to the Accident & Emergency of the hospital with repeated vomiting. Baby was born at term without any antenatal concerns. He is exclusively breast fed, vomiting began last week. Initially he was vomiting some feeds but now it's occurring after every feeding. There is no blood, bile or mucous in the vomit. Baby does not have any diarrhoea or fever but he seems a bit lethargic though still keen to feed despite the non-bile-stained vomiting. He had also lost weight and had few wet nappies.

MCQs

1. The differential diagnosis of non bilious vomiting at this age includes:

A. Overfeeding
B. Gastroesophageal Reflux
C. Pyloric Stenosis
D. Raised intracranial pressure
E. All of the above

2. In blood investigations, the changes in the electrolytes are as follows: Sodium 136, Potassium 2.8, Chloride 89, Bicarbonate 35, Urea 8.5 mmol/l, Creatinine 62 umol/l. This electrolyte changes may be explained by:

A. Gastric outlet obstruction leading to loss of stomach hydrochloric acid.
B. Renal failure.
C. Severe alkalosis because of dehydration secondary to vomiting.
D. Drug for example aspirin overdose
E. All of the above

3. Fluid resuscitation for a baby with pyloric stenosis includes:

A. D5 ½ normal saline with added potassium chloride
B. Lactated ringers solution as bolus.
C. Normal saline bolus until capillary refill is normal and then D5 ½ normal saline with 40 mEq/L of potassium chloride at maintenance rate.
D. Colloids like albumin as bolus.
E. Normal saline by bolus until capillary refill is normal and continued as maintenance fluid as well.

PYLORIC STENOSIS

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4. Most common biochemical abnormality in congenital pyloric stenosis:

A. Hyperkalemic metabolic acidosis
B. Hypokalemic metabolic acidosis
C. Hyperkalemic metabolic alkalosis
D. Hypokalemic metabolic alkalosis
E. None of the above.

5. Pyloric stenosis:

A. Is more common in boys.
B. Surgery is the definitive treatment.
C. Infants usually present at birth.
D. Can have a positive family history.
E. An ultrasound is usually performed to confirm the diagnosis.

Answers

1. E

Overfeeding is a diagnosis by detailed history. These babies usually have excellent weight gain and not particularly dehydrated. Gastroesophageal reflux is very common cause of non bilious vomiting is this age group but tends to cause vomit since birth but still can present like this baby. Raised intracranial pressure secondary to hydrocephalus or an intra cranial mass can present with vomiting.

2. A

Electrolyte abnormalities in this baby are typical for pyloric stenosis, hypochloremic, hypokalemic, metabolic alkalosis. This happens secondary to the pyloric obstruction leading to vomiting of hydrogen and chloride ions. Renal failure causes acidosis with hyperkalemia and increased urea in this baby is due to dehydration. Acute dehydration causes acidosis because of production of lactate. Also drug overdose by aspirin cause metabolic acidosis not metabolic alkalosis.

3. C

Initial fluid bolus consists of normal saline only without added potassium because rapid infusion of potassium is dangerous. Potassium is later added to the maintenance fluids when urine output is evident and renal failure is excluded. Lactated Ringers solution in this setting of hypochloremic alkalosis is not appropriate solution as it contains only 109 mEq/L of chloride and also it has added lactate which gets metabolized to bicarbonate worsening already present alkalosis.

4. D

Hypokalemic metabolic alkalosis. Repeated vomiting due to gastric outlet obstruction leads to loss of hydrochloric acid (HCL), sodium (Na^+), chloride (Cl^-), potassium (K^+) and water, leading to dehydration and hallmark metabolic derangement of hypochloraemic, hypokalaemic metabolic alkalosis.

5. A, B, D, E

Pyloric stenosis is more common in boys with a male to female ration of 4:1. Pyloromyotomy is the definitive treatment. Babies usually present at post natal age of 3 to 6 weeks after birth. Babies with pyloric stenosis may have a positive family history with a reported 7% incidence in children of affected parents. Ultrasound has become the standard technique for diagnosis of Hypertrophic pyloric stenosis.

PYLORIC STENOSIS

J Ashraf, A Mahomed

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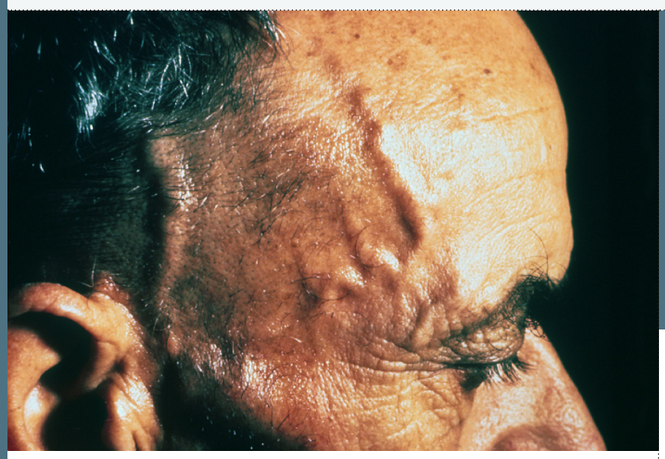
PRACTICAL PROCEDURE: TEMPORAL ARTERY BIOPSY

P Orchard, V Robba



Abstract

Temporal arteritis is a disorder caused by chronic inflammation of large and medium arteries most commonly affecting the superficial temporal artery. Although relatively uncommon, it is the most frequent type of vasculitis which left untreated, can lead to non-reversible complications such as blindness and stroke. This article covers the recognition of this condition and describes the practical procedure used to aid diagnosis.

Keywords:Temporal artery, vasculitis, biopsy.

History and pathology

Introduction

Temporal artery biopsy is a surgical procedure performed to aid in the diagnosis of temporal arteritis (TA), otherwise known as giant cell arteritis. Patients with suspected temporal arteritis will usually present to and be managed by the medical team or be referred directly to a rheumatologist, however, the biopsies are performed by a variety of surgeons (general surgery, vascular, ophthalmology, maxillofacial) depending on the hospital. The operations take place on either the emergency list or on an urgent inpatient list, it is therefore a key procedure for trainees to be aware of. It is also a common question in the MRCS part B exam. In order to understand the procedure itself, it is important to be familiar with the anatomy and pathology of this condition.

Epidemiology

Temporal arteritis is the most common primary vasculitis (1). The incidence of the disease varies amongst different reports, from 22.2 to 200 per 100,000 per year at the age of 50 (1,2). However all reports are in agreement that females are affected more than males and as the population age increases so does the incidence of the disease (1,2,3,4)

The first case to be reported in the literature of temporal arteritis was described in 1890 in a man who was unable to wear his hat as it was too painful against his temporal region – his temporal arteries were found to be hard and pulseless. However, it was not until 1932 that the first 2 pathological cases of temporal arteritis were confirmed (2)

Practical Procedure:
Temporal Artery Biopsy
Neurosurgery

Aetiology and pathology

Temporal arteritis is granulomatous arteritis of unknown aetiology which affects large to medium sized vessels, especially the branches of the external carotids. The most susceptible vessel is the superficial temporal branch (1, 2, 5, 6). Other vessels which can be targeted include the aorta and its branches.

TA presents with both local and systemic features. The local features are due to arterial inflammation causing endovascular damage, stenosis and occlusion which unlimitedly leads to ischemia and necrosis of tissue (2). The inflammatory cells which are involved in the ischemic process also bring about systemic features such as malaise, fever and weight loss (2, 7).

Temporal arteritis is associated with muscle diseases, in particular polymyalgia rheumatica.

Histology

In temporal arteritis the vessels are found to be thickened with intimal hypertrophy. The intima and sub intima contain a variety of inflammatory cells, mainly T-lymphocytes but also histocytes and giant cells (which give Temporal arteritis its alternative name). These giant cells are related to fragments of elastic lamina which have broken off from the vessel wall. It is the degenerated and altered elastin found in theses arterial walls which are targeted by the immune system which is thought to bring about the immune reaction.

Clinical presentation

Temporal arteritis can present with a variety of symptoms which can mimic other diseases therefore making diagnosis difficult. Classically symptoms are described as headache, tenderness over one or both temples and feeling generally unwell. However studies suggest that jaw claudication, visual changes and systemic features such a fever and malaise are more specific than headache (1,9).

Clinical examination is often unremarkable in patients with temporal arteritis but it is important to look out for systemic signs, temporal artery changes and complications of arterial occlusion.

PRACTICAL PROCEDURE: TEMPORAL ARTERY BIOPSY

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Systemic	Temporal artery changes	Complications of arterial occlusion
Pyrexia Recent weight loss Polyrheumatica - joint pain - stiffness - reduced ROM	Tenderness Reduced or absent pulsation Erythema Swelling Nodules in the artery	Ischemic changes to scalp and tongue Changes to optic disc including pale or swollen disc Retinal artery occlusion

Table 1: Clinical features of temporal arteritis

Investigations

If TA is suspected, it is important to diagnose and initiate treatment swiftly due to the risk of irreversible symptoms.

The American College of Rheumatology (ACR) has developed a 5 point scoring system to aid in diagnosis (8):

1. Age over 50 years
2. ESR > 50mm
3. Superficial artery tenderness
4. Temporal headache
5. Positive histology on a temporal artery biopsy

Each point has an equal rating and a score of 3 or more is highly accurate (sensitivity 93.5 and specificity 91.2%) in diagnosis (5).

A temporal artery biopsy is gold standard for the diagnosis of the disease, however it is not essential in the diagnosis of TA (as seen above). However a positive biopsy is diagnostic and histopathlogical changes often correlate with clinical features of severity (1). Diagnostic features include granulomatous panarteritis with mononuclear cell infiltrates and giant cell formation within the arterial wall. This confirms the diagnosis of TA in biopsy specimens of the temporal artery.

A negative biopsy does not exclude the disease; it is possible to get a false negative result. Factors which can increase the likelihood of this include (1):

- presence of a skip lesion
- pathological sectioning technique
- duration of steroids

It has been suggested that ultrasound may be of value in the diagnosis of temporal arteritis. Using ultrasound it is possible to examine for halos around the arteries (due to oedema), which can be either help direct the biopsy to avoid false negatives, or if it is a convincing history can initiate treatment and avoid an invasive form of investigation (10,11).

Treatment

If a diagnosis of TA is suspected then high-dose steroids are started even before a temporal artery biopsy is performed due to consequences of delayed diagnosis on vision. Once diagnosis is confirmed the steroids are continued long term with a tapering dose.

Biopsy: Indications & Contraindications

Indications	Contraindications
TA clinically suspected To aid diagnosis	Previous negative biopsy Prolonged treatment with steroids (>30 days)

Table 2:Indications and contraindications for temporal artery biopsy

Gaining informed consent

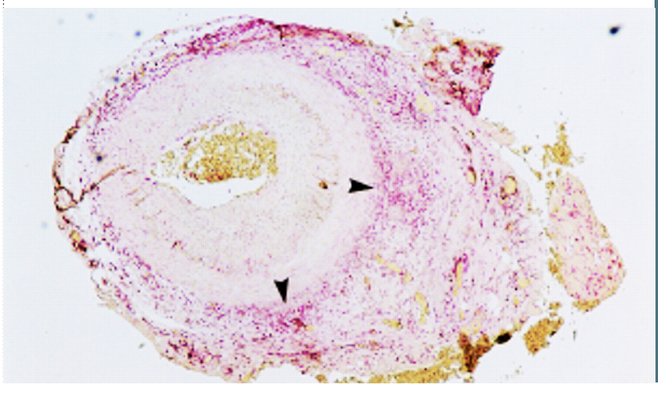
As with every surgical procedure, a discussion with the patient covering the stages of the procedure, the risks, and the potential benefits is paramount.

The complications of temporal artery biopsy are generally rare (see table) but need to be explained thoroughly to the patient.

General complications	Procedure specific complications:
- bleeding - haemorrhage - haematoma - infection - scaring - pain	- False negative results due to skip lesions (1-2cm vein is taken to avoid this) - Entrapment of hair in the scar resulting in foreign body reaction - Alopecia or skin loss (reduced blood flow at site of biopsy) - Numbness or drooping of the brow (damage to auriculotemporal or facial nerve branches) - Removal of the vein - Stroke

Table 3: Complications of temporal artery biopsy

The patient needs to be made aware of the prognosis if the proposed biopsy is rejected. This includes worsening of symptoms, including blindness if arteritis is present and not treated.



PRACTICAL PROCEDURE: TEMPORAL ARTERY BIOPSY

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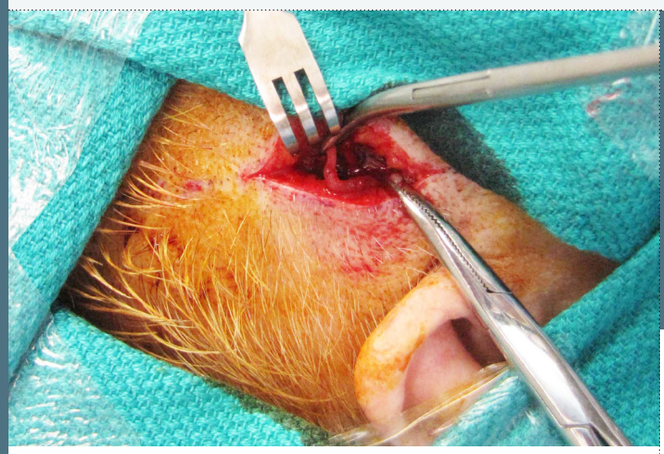

Practical Procedure: Temporal Artery Biopsy
Neurosurgery

Positioning

Positioning of the patient:

- Supine position
- Patients head turned laterally, facing away from the surgeon
- Bed elevated to 45° to present venous congestion at the site of surgery

Relevant Anaesthetic Points

- Performed under local anaesthetic – preference of performing surgeon
- Ensure you have enough time to allow anaesthetic to take effect
- Adrenaline can be used to help reduce bleeding from the skin edge, however should be avoided close to the artery due to the risk of arterial spasm.

Procedure

Relevant Anatomy

The maxillary artery and the superficial temporal artery are the two terminal branches that bifurcate superiorly from the external carotid artery, the latter being the smallest.

Superficial Temporal Artery		
Course	Relations	Landmarks
1. Begins behind neck of mandible, within parotid gland. 2. Passes superficially over posterior root of zygomatic process 3. 5cm above zygomatic process, divides into frontal and parietal branch	As it crosses zygomatic process • Covered by auricularis anterior muscle and dense fascia • Crossed by temporal and zygomatic branches (facial nerve) • Auriculotemporal nerve lies posteriorly Anastomoses • Supraorbital artery • Internal carotid artery	Pulse is palpable anterior and superior to tragus (superior to the zygomatic arch)

Table 4: Anatomy of the temporal artery

Technique:

- The path of the temporal artery should be identified and mapped by palpation of the pulse anterior to tragus and aided by a hand held doppler. It should be marked anterior to the tragus and extend into the hairline
- Once marked the area should be prepared with hair within a 2cm radius shaved (patient should be pre-warned about this)
- Once the area is marked the patient should be drapped ensuring not all of their face is covered to prevent claustrophobia
- The surgical incision should be made over the identified artery approximately 3-5cm in length, ensuring the underlying artery is not damaged.
- The artery can be found in the temporoparietal fascia following blunt dissection through subcutaneous fat.
- 3-5cm of artery should then be exposed and identified by its pulsation (to avoid removal of temporal vein) it is then excised and placed in Formalin solution to allow histological examination
- Once haemostasis has been achieved the subcutaneous layer and then skin layer should be closed.

It is important to remember to ensure the specimen is delivered to the lab with a request card containing adequate clinical information including if the patient has received steroid therapy and the length of the course.

It is also important to inform the referring team that the biopsy has been performed.

Post operative Management

The case should be performed as a day case procedure, although it will usually occur on an emergency list. Simple analgesia should be enough to control post operative pain.

If there is concern regarding haematoma formation then a pressure dressing can be applied for around 24 hours to minimise this risk.

The patient should be advised to continue with their steroid treatment until they are reviewed in clinic (by the referring doctor) when biopsy results should be available and a diagnosis of TA has been confirmed.

PRACTICAL PROCEDURE: TEMPORAL ARTERY BIOPSY

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Multiple choice questions:

1. Which two arteries are the terminal branches of the external carotid artery?

a) Posterior auricular
b) Superficial temporal artery
c) Superior thyroid
d) Facial
e) Maxillary

2. Which of these is not a recognised complication of temporal artery biopsy?

a) Drooping of the brow
b) Drooping of the mouth
c) Alopecia of surgical site
d) Stroke

3. Which of these is not included in the ACR scoring system for aiding the diagnosis of TA?

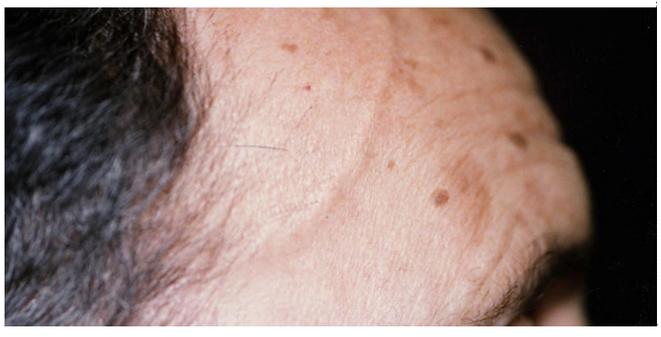
a) positive histology on a temporal artery biopsy
b) age > 50
c) raised white cell count
d) raised ESR
e) temporal headache
f) superficial artery tenderness

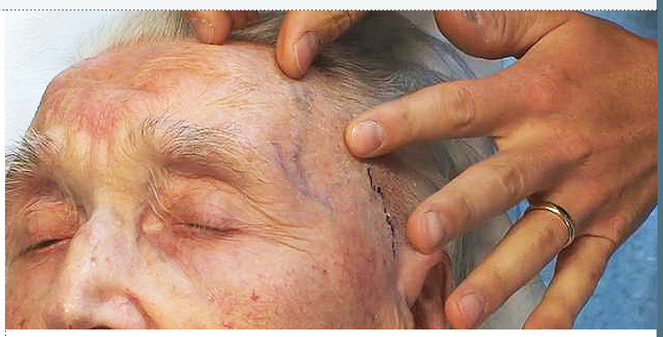
4. Which of these statements is false:

a) A previous negative biopsy is a contraindication to performing a temporal artery biopsy
b) High-dose steroids should be started even before a temporal artery biopsy is performed if temporal arteritis is suspected
c) A negative biopsy does not exclude the disease.
d) The likelihood of a false negative can be increased by the presence of skip lesions
e) Prolonged treatment with steroids (>30 days) is not a contraindication to performing a temporal artery biopsy

5. Which of these statements is false: The temporal artery:

a) Is in close relation to the auriculotemporal nerve which lies posteriorly
b) Runs posterior to the zygomatic arch on it's way to the scalp
c) divides into frontal and parietal branch around 5cm above the zygomatic process
d) Runs on the surface of the deep fascia
e) Is a branch of the external carotid artery





6. Which statement is true with regard to the temporal artery biopsy procedure:

a) The path of the temporal artery should be identified and mapped by palpation of the pulse posterior to the tragus and aided by a hand held doppler.
b) The procedure should be carried out under local anaesthetic and with adrenaline.
c) The surgical incision should be made over the identified artery approximately less than 3cm in length, ensuring the underlying artery is not damaged.
d) The artery can be found in the temporoparietal fascia following blunt dissection through subcutaneous fat.
e) 5-10 cm of artery should then be exposed and excised and placed in formulin solution to allow histological examination

Answers

1-B, 2-B, 3-C, 4-E, 5-B, 6-D.

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COURSE REVIEW: TEACH THE TEACHERS

M Singh

There are currently a lot of formal teaching courses available to doctors and it can be difficult to know which one is best. Teach the Teachers, offered by ISC Medical, is one such course. It's aim is to show candidates how best to teach to different audiences, ranging form small groups to large auditoriums. As formal training in teaching becomes more desirable for candidates trying to enter higher surgical training, I believe courses such as this will only gain popularity.

What stage would you do it?

This course is designed to benefit trainees of almost every grade. The one I attended had trainees ranging from foundation year 1 to ST3. The principles and skills taught would be beneficial to any trainee involved in teaching, whether it be informally by the bedside or lecturing at international conferences. With greater pressure on surgical trainees to present at national and international level, the development of effective teaching skills is essential and so would benefit all surgical trainees from foundation year 1 onwards.

Why did you do it?

I have always had a keen interest in teaching and have taught from the bedside to presenting at international meetings. I felt that having some formal training in how to teach and present would only be of benefit and improve the impact of any future presentations.

How is it structured?

It is a two day course with a maximum of twelve candidates and two instructors. Courses are run on almost a monthly basis, with the venue based in central London.

The course teaches evaluation of learning styles, including those of candidates by way of a questionnaire. I found this was extremely useful and now that I'm aware of how best I take in information, has actually changed the way I study!

Candidates are taught how to deliver effective teaching catering for all learning styles and also, how to provide feedback and constructive criticism to colleagues. On the second day, candidates have to present a short teaching presentation, often using Microsoft PowerPoint, to the group, which is also recorded so that candidates can see themselves presenting.

Course Review: Teach the Teachers
Career Focus

The topic can be anything, including non-medical subjects, as it is the style of teaching that is evaluated. They are then given detailed feedback on their teaching, and constructive comments that will hopefully help in future presentations.

How are candidates assessed?

There is no formal examination/evaluation at the end of the course. However, there is a short five to ten minute presentation given to the rest of the group on the second day. This can be prepared in advance or done after the first day. The topic can be anything and certainly, a common one in my group was "How to make a paper aeroplane!"

How much did it cost?

£395 for two days

Was it worth it?

This was an extremely useful course. It covers some very interesting aspects of learning and candidates have the opportunity to discover their best method of learning by completing an in-course questionnaire. The feedback sessions are very informative and always done in a constructive way. I would highly recommend this course to anyone involved in teaching, whether that be formally or informally, such as teaching by the bedside.

Further information

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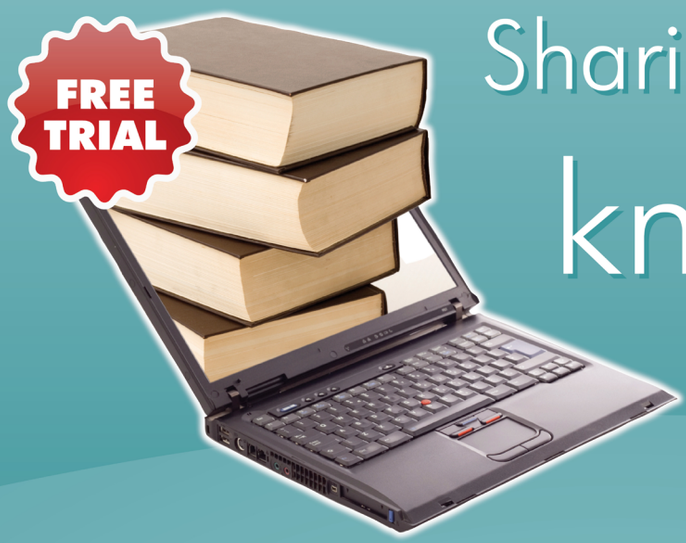

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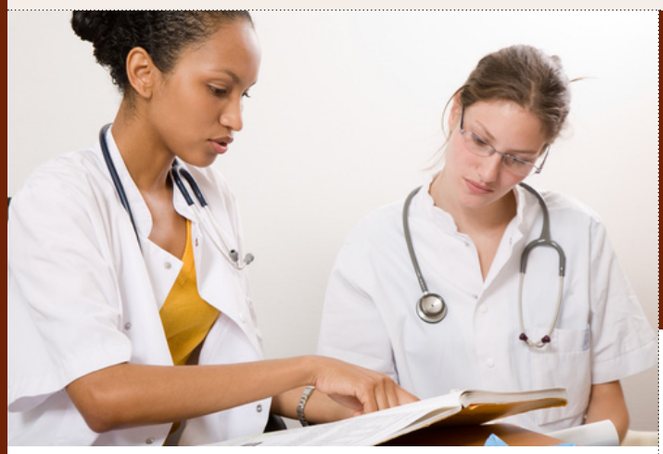
SENIOR HOUSE OFFICERS 'ACTING UP' AS REGISTRARS

A Harrison, J Risley



Abstract

It is well-recognized that as SHOs progress and become more senior, they start to bridge the gap in experience and knowledge between themselves and that of a registrar (albeit a junior registrar). In such circumstances, they may find themselves placed onto the SpR on-call rota and hence fulfilling the duties of an SpR whilst still an SHO. Although this represents an opportunity to make a gradual step upwards in terms of responsibility and experience, some trainees may be concerned with regards to their liability if something were to go wrong. It is the aim of this article to provide some guidance on the above, and provide an outline of the issues that a trainee should consider prior to 'acting up' as a registrar.

Key Words: Standard of Care, Acting up, Medical Defence Organisations, Good Medical Practice.

It is well-recognized that as SHOs progress and become more senior, they start to bridge the gap in experience and knowledge between themselves and that of a registrar (albeit a junior registrar). In such circumstances, they may find themselves placed onto the SpR on-call rota and hence fulfilling the duties of an SpR whilst still an SHO. Indeed they may be asked to step into the breach and cover for unexpected absences or during an emergency.

Although this represents a great opportunity to make a gradual step upwards in terms of responsibility and experience and, although usually provided with the best of intentions, some trainees may be concerned with regards to their liability if something were to go wrong.

It is the aim of this article to provide some guidance on the above, and provide an outline of the issues that a trainee should consider prior to 'acting up' as a registrar.

The areas that we will consider are those relating to the duty and standard of care owed in such situations, the GMC's Good Medical Practice guidance, contractual obligations and indemnity. The MDU and MPS have also kindly provided some excellent advice to consider in such situations.

Standard of Care Owed

The previous article in this series provided an introduction to negligence, including the concept of duty of care, and importantly the standard of care that is owed when treating patients.

It will be recalled that ***Wilsher v Essex Area Health Authority*** confirmed that the standard of care expected of a professional is to be determined by considering the nature of the 'post' they occupy and the tasks it involves, which is to be distinguished from their 'rank' or 'status'. Thus, if a junior doctor were filling a 'post' involving the performance of tasks usually undertaken by a senior doctor, the junior would be judged by exactly the same standards as the senior doctor unless they seek and act upon the opinion of a senior colleague, who would then assume responsibility for any actions they advised.

GMC: Good Medical Practice

It is also important to recognize that even if a doctor's actions do not amount to negligence within the common law, they may still be accountable to the GMC and hence be vulnerable to disciplinary proceedings. It is thus important to be aware of the GMC's guidance as provided in Good Medical Practice.

This is the key guidance provided by the GMC, which covers all areas of medical practice and contains certain paragraphs relevant to the issue at hand, including:

SENIOR HOUSE OFFICERS 'ACTING UP' AS REGISTRARS

A Harrison, J Risley

2. Good clinical care must include:

c. referring a patient to another practitioner, when this is in the patient's best interests

Thus, if you do not feel that you can fulfil the role, you should refer the patient to a colleague to be managed.

3. In providing care you must:

a. recognise and work within the limits of your competence

i. consult and take advice from colleagues, where appropriate

11. In an emergency, wherever it arises, you must offer assistance, taking account of your own safety, your competence, and the availability of other options for care.

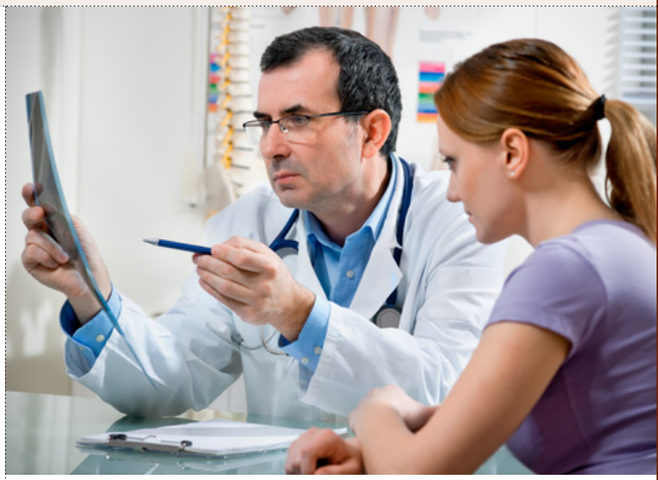
30. If a patient under your care has suffered harm or distress, you must act immediately to put matters right, if that is possible. You should offer an apology and explain fully and promptly to the patient what has happened, and the likely short-term and long-term effects.

Thus if, while acting outside your area of competence an adverse event occurs, the above would apply.

34. You must take out adequate insurance or professional indemnity cover for any part of your practice not covered by an employer's indemnity scheme, in your patients' interests as well as your own

41. Most doctors work in teams with colleagues from other professions. Working in teams does not change your personal accountability for your professional conduct and the care you provide. When working in a team, you should act as a positive role model and try to motivate and inspire your colleagues. You must:

c. make sure that your patients and colleagues understand your role and responsibilities in the team, and who is responsible for each aspect of patient care.



Hence if acting up, you should ensure that those around you and in your team are aware of the situation. It is also important to consider whether you are being expected to act as both SHO and Registrar simultaneously, and if not, whether the F1 is thus being asked to act up as an SHO to cover you. Attempting to cover both posts at the same time is potentially a very dangerous situation, and indeed it is unlikely that an F1 would technically be allowed to act as an SHO.

Contractual Considerations

It is important to consider the contractual obligations that may apply. There are clearly different situations in which being asked to act up may occur, from the unplanned situation to cover for unexpected illness, to the essentially fixed commitment such as if the Registrar is regularly elsewhere (for example doing a clinic at a peripheral hospital). In any event, if asked to do so and the SHO does not feel fully comfortable with the situation, they need to either decline to do it or, if they feel unable to do that, insist that they receive a written disclaimer to the effect that they will not be held responsible or accountable for any adverse incidents arising out of it.

Medical Defence Organisations

Both the MDU and MPS have been kind enough to provide advice on this matter. We have provided the the advice in full so as not to alter the context in which it has been given:

Dr Natalie Hayes, MDU Medicolegal adviser:

"When doctors in training are asked to 'act up' and to undertake the tasks and responsibilities of more senior colleagues, there are a number of issues to consider before accepting. Broadly speaking, these fall into two main areas:

Firstly, it is important to check with the HR department that this is a practice that is acceptable to the hospital and that there are no contractual terms that may prevent you from accepting. It may be wise to seek external advice concerning any contractual implications and it is also important to check that the Trust's NHS indemnity arrangements will extend to cover this additional level of responsibility.

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Secondly, if you are asked to undertake an additional role, you should bear in mind the GMC's advice that in providing care, you must "recognise and work within the limits of your competence." (Good Medical Practice, 2006, paragraph 3).

The GMC also states that 'good doctors make the care of their patients their first concern; they are competent (and) keep their skills up to date....'. (paragraph 1) As such, if asked to act up, you must firstly consider whether or not you are competent to undertake the extra responsibilities and tasks the role entails. If you feel that you are not competent or sufficiently experienced to take on the work of a more senior colleague, you should not agree to do so and should explain why.

If you were to take on the role of a more senior colleague and it was beyond your level of competence and expertise, patient safety may be compromised and you may have difficulty justifying your actions if later asked to do so. In such cases, the GMC states that 'if you have good reason to think that patient safety is or may be seriously compromised by inadequate premises, equipment, resources, policies or systems, you should put the matter right if that is possible. In all other cases you should draw the matter to the attention of your employing or contracting body.' (paragraph 6) In practice, this would mean discussing the situation with your educational supervisor or supervising consultant in the first instance.

Exceptionally, doctors in training may be asked to act up in an emergency situation where there is no one more senior who can assist, such as in a major incident, or if the hospital experiences exceptional levels of staff illness such as a pandemic, for example. In these emergency situations, you may have no choice but to step in to help out although the GMC says when doing so you should take account of your own safety, your competence and the availability of other options for care. (paragraph 11)

In summary, when asked to act in a more senior role, doctors in training must carefully consider what they are being asked to do, whether or not they are competent to undertake it, and what alternative arrangements can be made if they are not; as always, patient safety must be the first priority."

Senior House Officers 'Acting Up' as Registrars
Current Training Issues

Dr Tom Lloyd, MPS Medicolegal Adviser:

"From a medicolegal point of view no doctor should work beyond their expertise and experience.

If this person is being asked to act up then they must not practice or make decisions beyond their competence. This is important as some trainees may feel an inherent pressure to cope, having been given the extra responsibility and therefore could delay seeking assistance.

Equally they should be able to state to any consultant their limitations if they are being expected to work beyond their limits.This would be in line with standards set by the GMC in Good Medical Practice and accompanying guidance. If they are acting up they should contact their indemnity provider to ensure they have adequate indemnity."

Indemnity

As per paragraph 34 of Good Medical Practice (above) therefore, doctors must take out adequate insurance or professional indemnity cover for any part of their practice not covered by their employer's indemnity scheme. Doctors working in hospitals will be indemnified by their Trust to an extent, via the NHS Litigation Authority (NHSLA), which provides indemnity to employees in respect of clinical negligence claims.

Since 1990, the NHS has had financial responsibility for negligence attributable to medical staff employed in hospitals. Most doctors employed by the NHS are covered for the duties listed in their contract by the Hospital and Community Health Services indemnity scheme (often called NHS or Crown Indemnity), and are not obliged by law to take out additional medical defence cover (1).

It is, however, in both a doctor's and their patients' best interests, to take out additional indemnity cover from one of the medical defence organisations (MDOs), since health service indemnity schemes do not provide support for complaint handling, Trust disciplinary issues, coroner's inquests, criminal charges or referrals to the General Medical Council.

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This highlights the many and various potential pitfalls of a negligent clinical act, something the MDU term 'multiple jeopardy' i.e. if a negligent act is performed, the consequences for the practitioner may include a complaint from the patient or their 'health and welfare lasting power of attorney', a civil action, Trust disciplinary proceedings, GMC disciplinary proceedings and, if a patient death has sadly resulted, possible criminal proceedings and/or a coroner's inquest.

Another important motivation for hospital practitioners to take out supplementary indemnity cover with one of the MDOs or provide themselves with other personal indemnity insurance, in addition to the above reasons and in order to comply with GMC guidance, is because the NHS indemnity scheme ONLY covers medical negligence claims which arise from contracted NHS duties. The following are examples of eventualities and activities which are not covered (2):

- **defence of medical staff in GMC disciplinary proceedings for stopping at a roadside accident, and other good Samaritan acts not listed in your contract;**
- **clinical trials not covered under legislation;**
- **work for any outside agency on a contractual basis;**
- **work for voluntary or charitable bodies; and,**
- **work overseas.**

Hence, as Dr Hayes of the MDU stated, "It may be wise to seek external advice concerning any contractual implications and it is also important to check that the Trust's NHS indemnity arrangements will extend to cover this additional level of responsibility."

Conclusion

In conclusion, although a request to 'act up' is naturally quite flattering and the thought of gaining higher level experience exciting, we would advise any SHO asked to work at the grade of SpR by their employing Trust, to consider several things before agreeing to do so.

These include being mindful of the GMC's guidance in Good Medical Practice, considering the contractual implications of such a request, ensuring there will be adequate senior cover to whom you can turn for advice, and informing your MDO so that they can increase your personal indemnity cover if necessary. If all of these are taken into account and addressed, there should be no reason why an SHO cannot 'act up' and enjoy the extra experience potentially afforded by SpR level work.

References

(1) http://bma.org.uk/practical-support-at-work/immigration/doctors-new-to-the-uk/medical-defence-and-indemnity
(2) Ibid.

Appendix A: Checklist

1. Can you fulfil the requirements of the post of Registrar? You will be judged by the 'post' which you are working in with regards to standard of care owed.

2. Have you considered the relevant sections of Good Medical Practice?

3. Are there any contractual obligations preventing you from doing so?

4. Will there be senior colleagues to ask advice if needed?

5. Have you informed your MDO?

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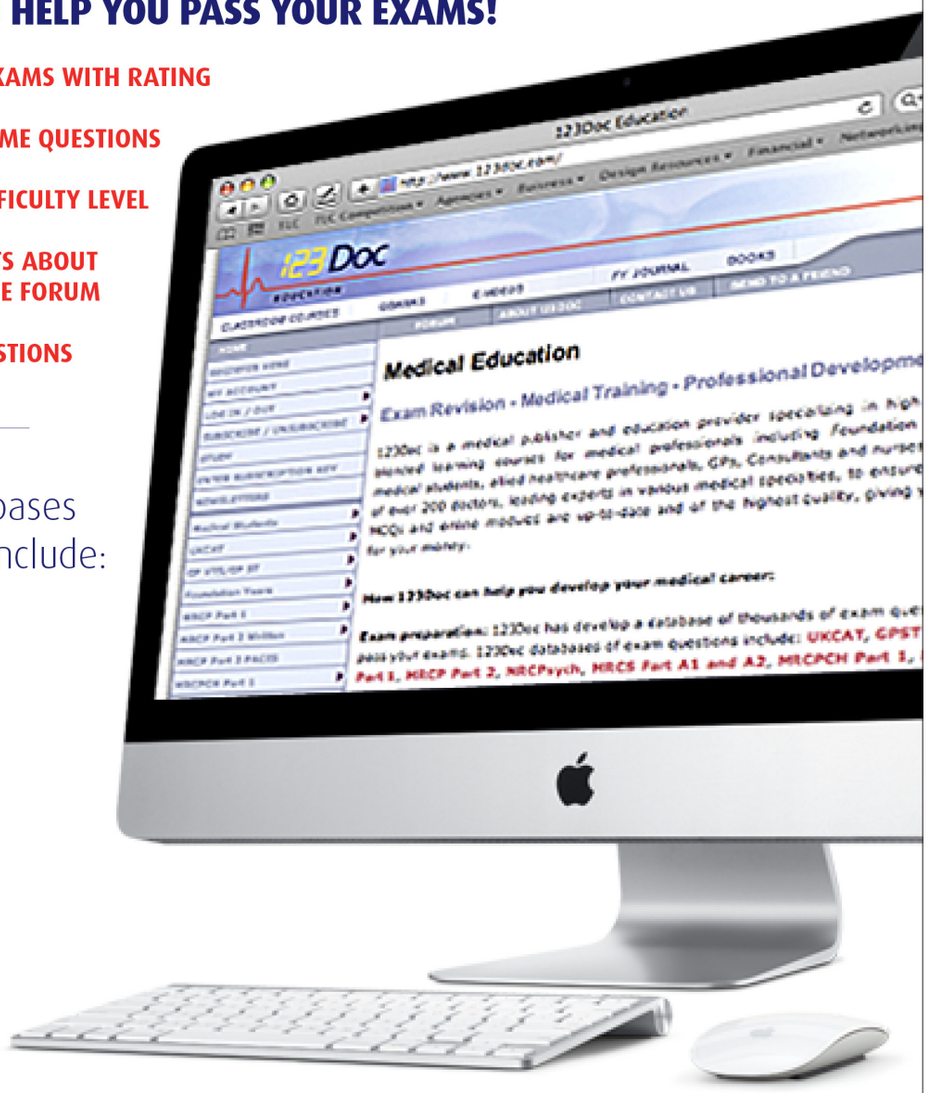

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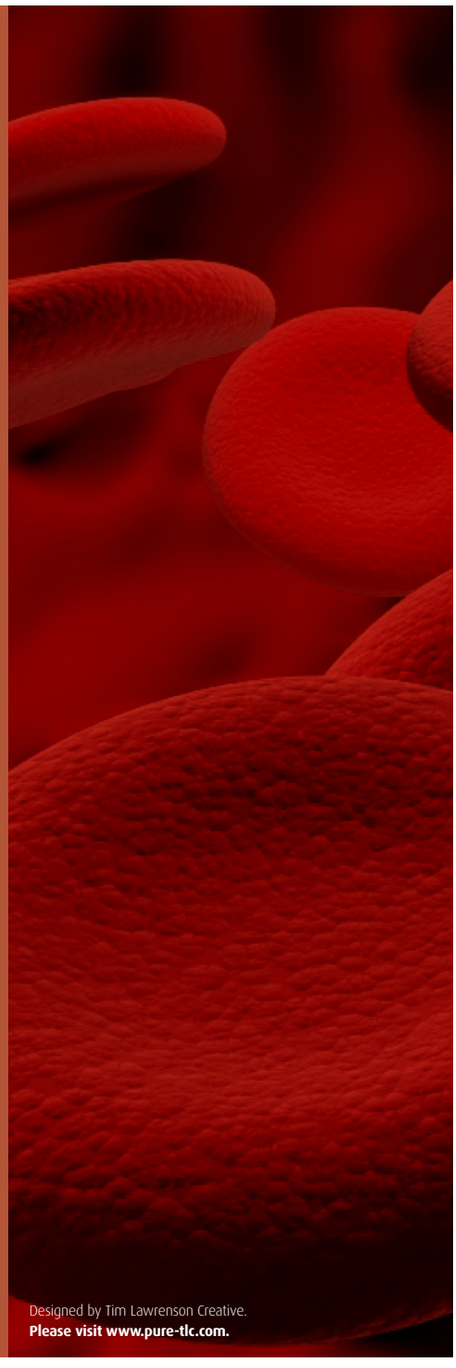

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