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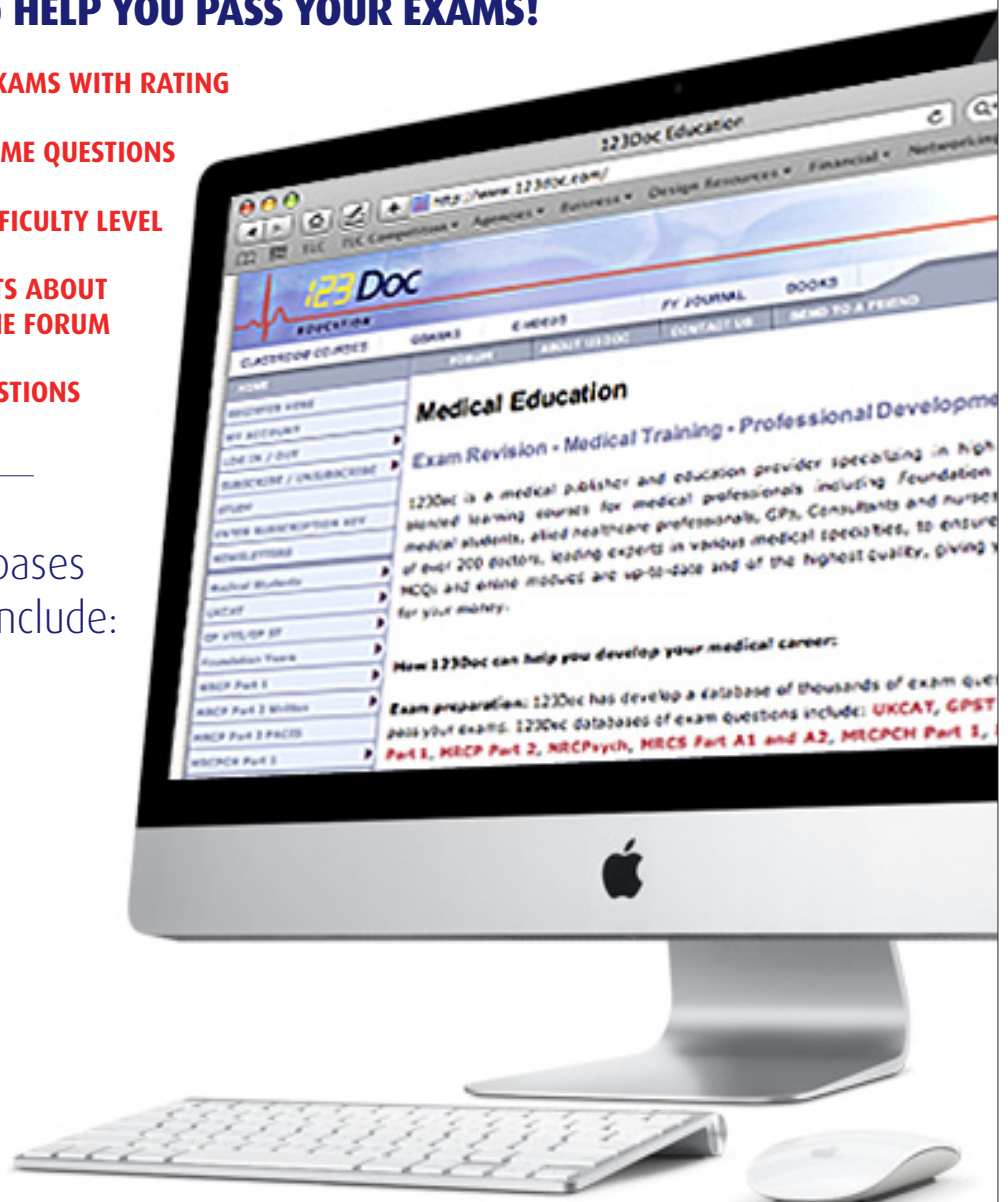
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FOUNDATION YEARS JOURNAL 2016

Volume 10

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CASE BASED DISCUSSION; INITIAL MANAGEMENT OF ACUTE KIDNEY INJURY (AKI)

T Shalaby, J Robin



Case study

A 71 year old man; was found on the floor unconscious; by his neighbour. The last time he was seen was two days previous in his garden. From the ambulance, GP notes and the collateral history it was noted that he had a past medical history of benign prostatic hypertrophy, type 2 diabetes mellitus and hypertension. His medications at the time were Metformin 1 g BD, Sitagliptin 100 mg OD, Ramipril 10 mg ON, Bendroflumethiazide 2.5 mg OD and Tamsulosin 400 mcg OD. He had recently started Ibuprofen for back pain and it was noted that he was allergic to Penicillin.

Medical history was a week history of diarrhoea and vomiting after eating at a Chinese restaurant. Biochemistry showed urea to be 52 and creatinine of 412, creatinine clearance of 22, C-reactive protein (CRP) of 220, White Cell Count (WCC) of 16, neutrophils. Creatine Kinase (CK) was 600, urine dipstick showed leucocyte of 3+, nitrites positive, Blood ++, Protein ++, the patient also had a low-grade temperature. Biochemistry the previous month (requested by the GP) showed urea of 4 and creatinine of 105.

The patient had a non-contrast CT head and neck and x-ray pelvis to rule out stroke or fractures due to un-witnessed fall and being found unconscious. He was alert but confused in A&E. The patient was admitted into a side room, initial impression being pre-renal AKI secondary to viral gastroenteritis (confirmed by presence of norovirus in stool) plus urine infection.

Case Based Discussion; Initial Management Of Acute Kidney Injury (AKI) Patient Management

A fluid flow chart was commenced upon initiation of crystalloids and a catheter was inserted to monitor urine output, which found a residual volume of 50 mls dark coloured urine. A renal ultrasound was requested to rule out obstruction and/or mass (haematuria) USS reported as within normal limits. A vasculitis screen was requested (active sediment-protein and blood), nephrotoxins stopped and his mean arterial pressure was kept above 65 to maintain good perfusion to kidneys.

The patient was prescribed Ciprofloxacin for three days, as per microbiology advice. On day four, biochemistry showed a creatinine of 130 and urea of 5. Due to the improvement in renal function his medications were recommenced. The vasculitis screen was negative.

The patient was discharged home under the care of his GP with advice to monitor his renal function (risk of diabetic nephropathy and obstructive uropathy).

Abstract

Acute Kidney Injury (AKI) is a very common clinical presentation, this is a serious yet often avoidable condition. The reported incidence of AKI is between 13-18% of all patients admitted to hospital with older adults being particularly affected (1). Most important to note is the reported inpatient mortality rates of AKI that varies depending on severity; in the UK this can vary between 25-30% (1). If medical teams put appropriate measures in place patients can receive the care and treatment required.

The financial impact of AKI on the NHS is estimated to be between £434 million and £620 million per year, this is reported to be more than the associated cost of breast, lung and skin cancer combined (1).

CASE BASED DISCUSSION; INITIAL MANAGEMENT OF ACUTE KIDNEY INJURY (AKI)

T Shalaby, J Robin

Evidence states

"30% of patients who died from AKI had predictable and avoidable AKI". The publication also highlighted "20% of the patients who developed AKI following admission subsequently died"(2, 3).

Discussion

Using the Kidney Disease Improving Global Outcome (KIDGO) scale to stage AKI helps provide a universal staging criteria (please see table 1).

Stage	Serum creatinine (SCr) criteria	Urine output criteria
1	Increase ≥ 26 $\mu\text{mol/L}$ within 48hrs or Increase ≥ 1.5 to 1.9 X reference SCr	<0.5 mL/kg/hr for > 6 consecutive hrs
2	Increase ≥ 2 to 2.9 X reference SCr	<0.5 mL/kg/ hr for > 12 hrs
3	Increase ≥ 3 X reference SCr or Increase ≥ 354 $\mu\text{mol/L}$ or commenced on renal replacement therapy (RRT) irrespective of stage	<0.3 mL/kg/ hr for > 24 hrs or anuria for 12 hrs

Table 1: KIDGO AKI scale.

Why staging is important?

This scale is used for patients presenting with renal failure and provides medical professionals an easy to use tool to simply identify the level of AKI of their patient. This scale is linked to the possibility of mortality and morbidity with the understanding that a rising creatinine is directly proportional to mortality and morbidity. (1, 2, 4)

Summary of initial management

1. Early detection; high-risk patients should be identified as early as possible. The following list identifies who are classified as high-risk:

- Chronic Kidney Disease (CKD) eGFR <60 .
- Heart failure.
- Liver disease.
- Diabetes.
- History of AKI.
- Dehydration/oliguria.
- Patients taking nephrotoxins.
- Use of iodinated contrast agent within the past 7days.
- Symptoms or history of urological obstruction.
- Sepsis.
- Over 65years of age.
- Surgical patients-especially intra-peritoneal surgery.

2. Fluid assessment and therapy

Adequate fluid therapy requires a number of steps. A volume status assessment is used to assess if the patient is under filled or overloaded by checking heart rate, blood pressure and postural blood pressure, mucous membranes and Jugular Venous Pressure (JVP). Achieve euvolaemia by administration of Crystalloids by boluses and then maintenance is guided by urine output, blood pressure and fluid balance.

3. Monitoring

This is achieved by frequent observational monitoring through fluid chart, daily weight and the use of the Early Warning Score (EWS). Urinary catheter and monitoring output is essential.

4. Investigation

The following investigations should be requested:

- Urine dip stick – to look for infection (Nitrites or leucocytes). If proteinuria then protein creatinine ratio and send urine for microscopy (casts) plus autoimmune screen if haematuria + and proteinuria (active sediment) with rash and joint pains.
- Bone profile /liver function/CK-to rule out rhabdomyolysis – usually CK in thousands.
- Myeloma screen (normocytic anaemia, raised calcium, bone pains).
- If platelets are low perform microangiopathy screen (blood film, LDH and reticulocytes).
- Ultrasound - within 24 hours if no identified cause of AKI or risk of urinary tract obstruction. This should be requested as urgent if there is suspicion of hydronephrosis.
- Treat sepsis – if present, guided by finding a focus of infection, fever, raised inflammatory markers and lactate.
- Stop nephrotoxins (NSAID/ACEI/ARB/diuretics).
- Avoid hypotension to maintain adequate perfusion to the kidneys.
- Consider Proton Pump Inhibitor (PPI) (platelet dysfunction and ensure pharmacist input for drug monitoring).

CASE BASED DISCUSSION; INITIAL MANAGEMENT OF ACUTE KIDNEY INJURY (AKI)

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5. Onward referral to nephrology should be made within 24 hours if the likely diagnosis of AKI may need specialist treatment (vasculitis, glomerulonephritis, myeloma). Other reasons for referral to nephrology include AKI with no clear cause, when there is inadequate response to treatment, persistent complications (persistent hyperkalaemia, pulmonary oedema, or acidosis), when the patient is in stage 3 AKI, history of renal transplant or Chronic Kidney Disease (CKD) stage 4 and 5.

MCQs

1. The likely cause of renal failure is:

- a. Dehydration
- b. Ibuprofen and other nephrotoxins
- c. Rhabdomyolysis
- d. Sepsis
- e. All of the above

2. Best initial management:

- a. Intravenous Fluid
- b. Urinary catheter
- c. Stop nephrotoxins
- d. Treat sepsis
- e. All of the above

3. Which drugs should be stopped:

- a. Ibuprofen
- b. Ramipril
- c. Bendroflumethiazide
- d. Metformin
- e. All of the above

4. Urine Culture and sensitivity showed Enterococcus sensitive to all the below, what will be your choice?

- a. Trimethoprim
- b. Nitrofurantoin
- c. Amoxicillin
- d. Gentamicin
- e. Non of the above

5. Urine dipstick showed active sediment (protein and blood), which tests should be ordered?

- a. ANCA
- b. Anti GBM
- c. ANA
- d. Immunoglobulins
- e. All of the above

CASE BASED DISCUSSION; INITIAL MANAGEMENT OF ACUTE KIDNEY INJURY (AKI)

T Shalaby, J Robin

Answers

1. Answer: A

History of diarrhoea, vomiting and disproportionate urea to creatinine level points towards pre-renal failure.

2. Answer: A

Rehydration with crystalloids is the best first initial management in patients with pre-renal failure. Catheter is essential if urinary retention is present and useful for monitoring urine output

3. Answer: E

All the above are nephrotoxins, A, B cause a reduction in glomerular filtration pressure, diuretics are contraindicated in renal failure and metformin is contraindicated if eGFR <30 as risk of lactic acidosis.

4. Answer: E

Trimethoprim is contraindicated if eGFR <30, Nitrofurantoin is contraindicated if eGFR <60, Patient is allergic to penicillin and Gentamicin carry a risk of toxic tubular necrosis.

5. Answers: E

Active sediment urine (i.e. proteinuria and haematuria) may indicate glomerulonephritis secondary to vasculitis.

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AN OVERVIEW OF THE TRAUMA TEAM & MANAGEMENT

V Ameh



An Overview of The Trauma Team & Management Teaching & Training

Pre-hospital information & sequence of events

When receiving major trauma patients, the following information relayed by the ambulance personnel is vital for adequate planning and preparation for receiving the patient;

- Nature of incident
- Number, age and sex of casualties
- The injuries sustained and priorities
- Airway, ventilatory and circulatory status
- The level of consciousness
- Treatment provided and response
- Estimated time of arrival (ETA)

This information can either be received by the team leader or by a designated trauma coordinator. If received by the trauma coordinator, it needs to be relayed immediately to the team leader.

The team leader then briefs the rest of the team and allocates relevant tasks. It is essential that each and every member of the team is familiar with his or her role and responsibility in order to ensure a smooth coordination of events.

Roles & tasks

The doctor team

This comprises the team leader (usually the Emergency Department Registrar or Consultant), Anaesthetist, ED Registrar, General Surgery Registrar, Orthopaedic Registrar, Obstetrics & Gynaecology Registrar (if a pregnant woman is involved) and Paediatric Registrar (if a child is involved) and a scribe.

Nurse Team

This comprises a nurse team leader, airway nurse, 2 circulation nurses and a "relative support nurse".

Introduction

Trauma is a significant public health issue. It is the leading cause of death among individuals aged 1 to 45 years. It causes about 17,000 deaths in the UK annually. There are approximately 720,000 hospital admissions and 6 million ED visits per year (1).

Trauma patients are difficult to assess due to multiple and occult injuries. Early diagnosis and treatment is vital for patient survival and reduction of morbidities.

Major trauma is best managed by a trauma team in a major trauma centre or unit; using a pre-determined plan for urgent assessment and resuscitation. It is also very resource intensive.

The Trauma Team

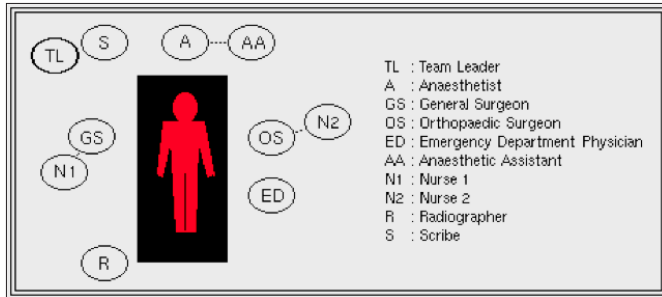
A trauma team is a multidisciplinary group of healthcare personnel who collectively work together on the assessment and treatment of those who are severely injured (2).

The team typically consists of doctors, nurses and radiographers. It normally consists of approximately 6 to 7 doctors, 3 to 5 nurses and one radiographer. The team can get very large therefore proper organization is required in order to avoid a chaotic scene.

AN OVERVIEW OF THE TRAUMA TEAM & MANAGEMENT

V Ameh

Position plan



(Courtesy trauma.org)

Details of roles & tasks

Team leader

- Coordinates the medical team.
- Ensures that the team members wear personal protective equipment (PPE), allocated roles are clear and makes personal introductions.
- Assesses the chest and may carry out particular procedures such as pericardiocentesis, thoracostomy and focused assessment sonography in trauma (FAST) scan.
- Assimilates information and lists investigations and treatment in order of priority.
- Liaises with other specialists and obtains relevant information from the ambulance staff.
- Talks to relatives.
- Dismisses and debriefs team members.

ED Registrar

When not acting as team leader, undertakes;

- Primary and secondary survey.
- Obtains intravenous access and blood samples.
- Performs thoracostomy or thoracotomy.
- Inserts urinary catheter.
- Documents in the notes.
- Relays findings to the team leader and scribe.

Anaesthetist

- Clears and secures the airway and C-spine control.
- Undertakes ventilation.
- Inserts central and arterial line if required.
- Monitors fluid and drug administration.
- Communicates airway issues with the team leader.
- Documents in the notes.

General Surgeon

- Focuses on the assessment of the abdomen.
- Assists in log roll.
- Inserts urinary catheter if necessary.
- Documents in the notes.

Orthopaedic Surgeon

- Assesses the long bones, spine and pelvis.
- Applies external fixator or pelvic sling.
- Undertakes dressing of open wounds and stabilisation of fractures.
- Documents in the notes.

Scribe

This is usually a separate doctor (but could be delegated to the trauma nurse). Records the following information in the standard trauma documentation sheet;

- Time of arrival.
- Mechanism of injury.
- Physical signs.
- Vital signs, urine out put and GCS.
- X-rays and other investigations.
- Fluid and drugs administered.
- Past medical history.
- Summary of injuries.
- Disposal of patients.

Radiographer

- Takes standard X-ray films as required- Usually chest and pelvis.
- Takes a more selective approach in penetrating trauma.

Nurse team leader

- Coordinates the nursing team.
- Records clinical findings, laboratory results, IV fluids, drugs and vital signs as called out by the circulation nurse.
- Prepares sterile packs for procedures.
- Assists the circulation nurse and brings extra equipment as necessary.

Airway nurse

- Assists in securing the airway and stabilising the cervical spine.
- Establishes a rapport with the patient in the resuscitation room.

Circulation nurse

- Assists in the removal of the patient's clothes.
- Assists in starting intravenous fluids, inserting chest drain and urethral catheterization.
- Monitors fluid balance.
- Measures vital signs and connects the patient to the monitor.

"Relatives" nurse

- Provides support for the patient's relatives.
- Provides and obtains information from relatives.

In order to avoid chaos and disorganisation, no more than 5 people should be touching the patient. Other team members should stand well back.

AN OVERVIEW OF THE TRAUMA TEAM & MANAGEMENT

V Ameh

Major Trauma Alert Triggers

Every hospital that deals with major trauma should have a major trauma alert trigger procedure. This is usually in the form of a standardised protocol. The ambulance services also use a similar protocol to triage patients to the most appropriate Centre.

The trigger can be classified into anatomical, physiological, clinical, mechanism of injury and others;

Anatomical

- Unmanageable airway.
- Unsupported airway.
- Uncontrollable haemorrhage.

Physiological

- Respiratory rate of less than 9 or more than 30 per minute.
- Systolic BP of 89 mmHg or less.
- GCS of 12 or less.

Clinical

- Flail chest.
- Penetrating trauma.
- Fracture of 2 or more long bones (humerus or femur).
- Amputation proximal to wrist or ankle.
- Crushed, mangled or degloving injuries.
- New onset motor or sensory deficit (whole limb or partial).
- Major burns.

Mechanism

- Falls over 5 meters (2 storeys).
- Vehicular entrapment.
- Complete or partial ejection from vehicle.
- Death in the same passenger compartment.

Others

- Older adult (older than 65 years).
- Significant co-morbidities.
- Pregnancy of 20 weeks or more.
- Other clinical concern.

Key investigations in Major Trauma

Early and targeted investigations in trauma patients have been shown to correlate with a very good outcome for these patients. Plain X-ray of the chest and pelvis, focussed assessment sonography in trauma (FAST) and organ-specific computerised tomography (CT) are the usual investigations undertaken in these patients. This is also recommended by the Advanced Trauma Life Support (ATLS) protocol (3,4). This protocol has however, been shown to lead to a misdiagnosis of some potential life-threatening solid organ injuries and is time consuming.

More recently, especially with the introduction of multi-slice helical CT (MSHCT), an increasing number of trauma centres are now using whole-body CT (WBCT); defined as CT scan of the head, neck, chest, abdomen, pelvis and spine. Huber-Wagner and colleagues reported that WBCT is associated with a lower mortality in trauma patients regardless of their haemodynamic status (5,6,7).

FAST (Focussed Assessment Sonography in Trauma) is another investigative tool that has become very popular in the evaluation of trauma patient in the Emergency Department. Ultrasound has significant advantages over CT scanning for the detection of free intra-peritoneal fluid. It is rapid, portable, non-invasive, does not involve radiation and can be repeated if required. The average time to perform a FAST in experienced hands is 5 minutes. However, there are limitations to the use of ultrasound; image interpretation is operator dependent but this improves with increasing experience. Furthermore, ultrasound is poor at identifying solid organ damage. Excess subcutaneous fat and bowel gas obscures the images obtained with ultrasound.

Several studies reviewing the use of FAST scan for blunt abdominal trauma have shown it to have a high specificity but a relatively low sensitivity. (8,9,10) Therefore it can be a useful decision making tool for identifying the need for laparotomy in hypotensive patients (systolic BP < 90 mmHg).

Scoring Systems For Trauma

Several scoring systems for trauma have been devised. This is helpful for audit, research and standardisation of trauma care across institutions. Commonly used ones are the Abbreviated Injury Score (AIS), Injury Severity Score (ISS) and the Revised Trauma Score (RTS).

Abbreviated Injury score (AIS)

This is an anatomical-based coding system devised by the Association for the Advancement of Automobile Medicine. It is used to clarify and describe the severity of injuries. (11) This gives a score for each injury the patient has sustained. Scores range from 0 to 6. For example (table 1)

Injury type	Severity	Score
Shoulder sprain	Minor (no injury)	0
Wrist sprain	Minor	1
Tibial fracture (closed, undisplaced)	Moderate	2
Head injury with LOC for less than 60 minutes	Moderate	3
Incomplete transection of the thoracic aorta	Severe	4
Complex liver laceration	Critical	5
Brain stem laceration	Incompatible with life	6

Table 1

(Abbreviated Injury Score)

AN OVERVIEW OF THE TRAUMA TEAM & MANAGEMENT

V Ameh

Injury Severity Score (ISS)

This is calculated from the Abbreviated Injury Score (AIS). An ISS score of greater than 15 is considered major trauma. It is usually calculated retrospectively but the extent of the injury can be predicted from the mechanism of the injury, distribution of the injuries and the physiological parameters. (See trigger list). The body is divided into six "AIS body regions"; head and neck, face, chest and thoracic spine, abdomen, extremities, external (skin). The squares of the 3 highest scores are added. Maximum score is 75 (5x5 +5x5+5x5).

An AIS of 6 in one region automatically scores 75 (these are usually unsurvivable injuries).

For simplicity, the AIS90 dictionary is used for the calculation. (12,13)

Revised Trauma Score (RTS)

The revised trauma score is a tool used to assess the physiological disturbance of the trauma patient. It is calculated from the respiratory rate, systolic BP and the GCS (Glasgow Coma Scale). Each parameter is allocated a value to which a weighting factor is applied. The three scores are then added together to give the revised trauma score. (14)

Revised Trauma Score (RTS)

Physiological parameter	Coded value	Weighting factor
Respiratory rate		0.2908
10-29	4	
>29	3	
6-9	2	
1-5	1	
0		
Systolic BP		0.7326
>89	4	
76-89	3	
50-75	2	
1-49	1	
0	0	
GCS		0.9368
13-14	4	
9-12	3	
6-8	2	
4-5	1	
0	0	

Table 2

Triss Methodology

This refers to the Combined Trauma and Injury Severity Score (TRISS). It assesses the degree of physiological derangement and anatomical injury and determines the probability of survival.

It combines the ISS and RTS plus a weighting factor to determine score. It also takes the age of the patient into consideration.

Patients who survive with a probability of <0.5 are unexpected survivors and those who die with a probability of >0.5 are unexpected deaths.

It may be used to compare the performance of one center against the national standards.

Key Summary Points

- **Survival is better in trauma patients if they are managed in a major trauma centre. Trauma units have a role to play in stabilising these patients prior to transfer.**
- **Optimum management relies on a well-organised trauma team.**
- **Treatment priorities are based on the ATLS protocol.**
- **Trauma scoring systems are useful for audit, research and surveillance and aid the organisation of trauma services.**

Multiple Choice Questions

Choose only one answer.

1. A 24-year-old male motorcyclist was involved in a high-speed road traffic accident. He collided head-on with a car. He was brought in to your ED, which is a trauma unit. He sustained a head injury with a loss of consciousness for 30 minutes. His GCS was 14, Pulse 120/min, BP 80/40mmhg, Respiratory rate 35/min. He was noted to have a bruise over his upper abdomen. What is the most appropriate course of action?

- Send him for immediate whole body CT scan and then transfer to a trauma centre.
- Put out a trauma team call, secure the airway, ensure adequate ventilation, commence fluid resuscitation, transfuse blood if required. Arrange FAST and/or whole body CT scan. Then organise transfer to a major trauma centre.
- Secure the airway, commence fluid resuscitation. Then admit to the ED observation ward and observe overnight.
- Transfuse 4 units of whole blood, send patient for whole-body CT scan, and then admit under the Surgeons.

AN OVERVIEW OF THE TRAUMA TEAM & MANAGEMENT

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2. History as in Question 1. Limb X-rays showed a closed right midshaft humeral fracture. The CT scan showed small right subdural haematoma and a complex liver laceration. There was blood in the peritoneal cavity. What is the Injury Severity Score (ISS)?

- a. 10
- b. 38
- c. 34
- d. 25

3. With regards to FAST scan, which of the following is true?

- a. FAST scan is able to detect the specific site of organ damage in trauma patients.
- b. A negative FAST scan is sufficient grounds to discharge patients home.
- c. A positive FAST scan in a haemodynamically unstable trauma patient warrants immediate laparotomy.
- d. ED-performed FAST scan has a very low sensitivity and specificity.

4. Which of the following is true of the trauma team?

- a. The team leader should ideally not touch the patient except to undertake specific procedures.
- b. The trauma team leader must be a Consultant.
- c. The scribe is not an essential part of the trauma team.
- d. The Obstetrics and Gynaecology doctor must be part of the trauma team in trauma involving all female patients.

5. A 70 year old man was travelling at 65mph when he lost control of his car and collided with the central reservation of the highway. He was brought to the ED by the paramedics on a spinal board and a cervical collar in-situ. He was able to localise pain with his left hand but was only able to flex his right elbow. He was making incomprehensible sounds. He could only open his right eye to voice. What is his Glasgow Coma Scale (GCS)?

- a. 7
- b. 8
- c. 9
- d. 10

Answers to Questions

1: The correct answer is d.

You need to follow the ATLS guideline for initial assessment of such patients. Ensure adequate ABC (airway, breathing, circulation) and Cervical-spine control. The patient is hypotensive and tachycardic; indicating ongoing blood loss. The patient needs to be stabilised with IV fluids (crystalloid) and blood transfusion if required. In general, he should be receiving blood transfusion after the second unit of crystalloid. Do not transfer such patients for CT scan unless they haemodynamically stable.

2: The correct answer is d.

The patient's injury severity score (ISS) is 38. This is calculated by adding the squares of the three highest scores. (closed humeral fracture = (2×2) + Head injury with LOC less than 60 minutes = (3×3) + Liver laceration = (5×5)

3: The correct answer is c.

A positive FAST scan in a haemodynamically unstable patient warrants a referral for immediate laparotomy. FAST scan is not a rule-out investigation; therefore a negative scan cannot be used to rule out an intraabdominal injury; especially in symptomatic patients. A period of observation is required.

AN OVERVIEW OF THE TRAUMA TEAM & MANAGEMENT

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4: The correct answer is a.

The Trauma Team leader (TTL) should not be getting involved in the physical handling of the patient, except to undertake specific procedures for which no other skilled personnel is available. The TTL does not have to be a Consultant; it could be another doctor with sufficient skills, experience and seniority to be able to undertake the role.

The obstetrics and gynaecology doctor is only required when a pregnant female is involved. The management of the pregnant female patient is directed at the resuscitation and stabilisation of the mother; since survival of the foetus is dependent on the survival of the mother.

5: The correct answer is d.

The Glasgow Coma Scale (GCS) is 10. This is calculated as follows;

- Eye opening to voice = 3
- Best verbal response (Incomprehensible sounds) = 2
- Best Motor response (localising pain) = 5

(Note that only the best response is included in the calculation)

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HOW TO DO A FASCIA ILIACA BLOCK FOR A FRACTURED NECK OF FEMUR

SJ Trehane, J Nelson, P Deol



How To Do A Fascia Iliaca Block For A Fractured Neck Of Femur Patient Management

It provides long lasting regional analgesia which facilitates transfer of patients from the ED to the ward and ultimately to the operating room. It also reduces the need for opioid analgesics in the elderly which can lead to side effects including nausea, vomiting and respiratory depression.

Contraindications

- Competent patient refusal
- Anticoagulation (INR >1.5 Platelets <100)
- Previous femoral vascular surgery
- Inflammation or infection over injection site
- Allergy to local anaesthetics
- Clinical suspicion of compartment syndrome

Abstract

An 80-year-old gentleman presents to the Emergency Department (ED) following a fall. He has severe pain in his right hip which is shortened and externally rotated. He is fast tracked for an X-ray. An X-ray of the hip shows a subcapital fracture neck of femur (NOF). We decide to perform a fascia iliaca block for long lasting analgesia before the patient is transferred to the ward.



Figure 1: X-Ray of right subcapital fractured neck of femur.

Indications

Fascia iliaca blocks are relatively easy to perform and are administered to patients with a fractured neck or shaft of the femur. The fascia iliaca compartment contains the femoral nerve, lateral cutaneous femoral nerve and the obturator nerve. The fascia iliaca block reaches the femoral nerve and lateral cutaneous femoral nerve; however the obturator nerve is rarely blocked.

Equipment

- Tuohy needle
- Antiseptic wipe or solution for cleaning the skin
- Sterile pack
- Approximately 5 mls of 1 % Lidocaine
- 2x 20ml syringes
- Gauze
- 30-40ml of long-acting local anaesthetic: 0.25-0.375% Bupivacaine or Levobupivacaine. If 0.5% Bupivacaine available dilute using 0.9% saline to make a 0.25% concentration. Ensure safe dose of local anaesthetic is calculated and adjusted according to patients' weight. For example: Bupivacaine 0.25% maximum dose is 2.5mg/kg.
- Ultrasound machine with a high frequency US probe 13-16MHz linear array if injection is to be administered by ultrasound guidance



Figure 2: Equipment.

HOW TO DO A FASCIA ILIACA BLOCK FOR A FRACTURED NECK OF FEMUR

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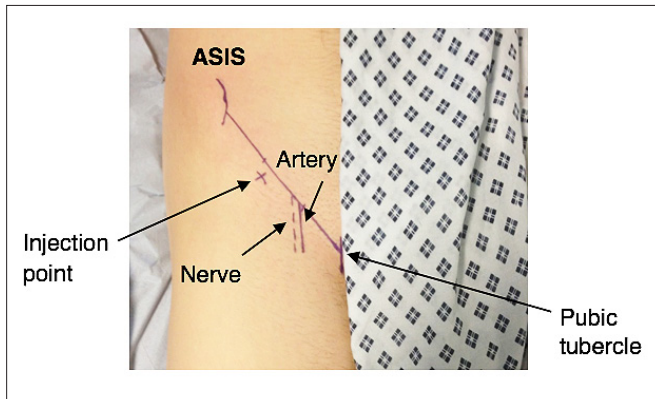


Figure 3a: Surface anatomy.

Explaining the procedure to the patient

- The procedure should be explained to the patient and informed consent should be gained and documented.
- Potential complications of the procedure should be communicated to the patient.

Complications

- Local anaesthetic toxicity
- Block failure
- Intravascular injury
- Damage to the nerve
- Allergy to agent used
- Infection

The procedure

- The patient should be lying supine with the groin of the affected side exposed.
- Aseptic technique should be adopted.
- Ensure no contraindications are present.

Landmark technique

- Draw a line between the Anterior Superior Iliac Spine (ASIS) and pubic tubercle (on the affected side).
- Divide the line into thirds.
- Find the junction of the lateral and middle third, and then go 1cm caudally; this is the injection site.
- Keeping a finger on the ipsilateral femoral pulse during the procedure prevents inadvertent intravascular injection.

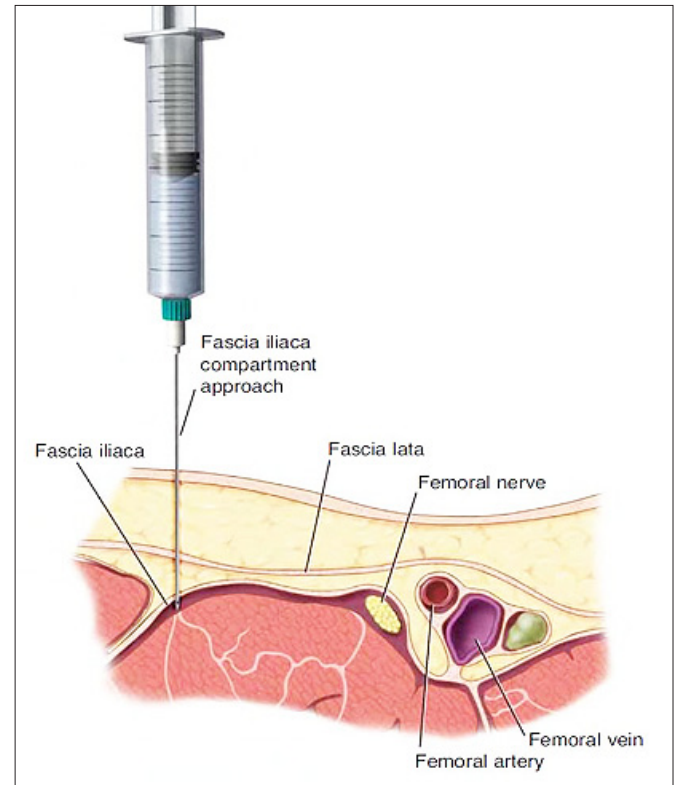


Figure 3b: Fascia iliaca technique (1).

- Clean the injection site using antiseptic solution or wipes.
- Infiltrate the skin and tissues with 1-2mls of 1% Lidocaine.
- Using the Tuohy needle enter the skin at a 90 degree angle to the skin surface.
- Advance the needle, there should be two 'pops' as the needle passes through the fascia lata and then the fascia iliaca (the latter is a more subtle 'pop').
- Aspirate prior to injecting.
- Check nothing is aspirated then slowly inject the anaesthetic: 30-40 mls of 0.25% Bupivacaine.
- Resistance should not be felt during injection.
- If resistance is experienced the needle may be in the iliaca muscle, in which case slightly withdraw the needle.
- The patient should not experience pain or paraesthesia whilst the injection is being given.
- Equipment should be carefully disposed of with particular care of sharps.
- Ensure that the procedure is clearly documented.
- Observations should be taken pre and post procedure.

HOW TO DO A FASCIA ILIACA BLOCK FOR A FRACTURED NECK OF FEMUR

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Ultrasound guided approach:

Place the ultrasound probe under the inguinal ligament in a transverse direction over the anterior aspect of the thigh. Identify the femoral artery and the iliacus muscle lateral to it, covered by the fascia iliaca. Advance the needle until the tip is underneath the fascia iliaca, confirm negative aspiration, then inject the local anaesthetic.



Figure 4: Ultrasound probe.

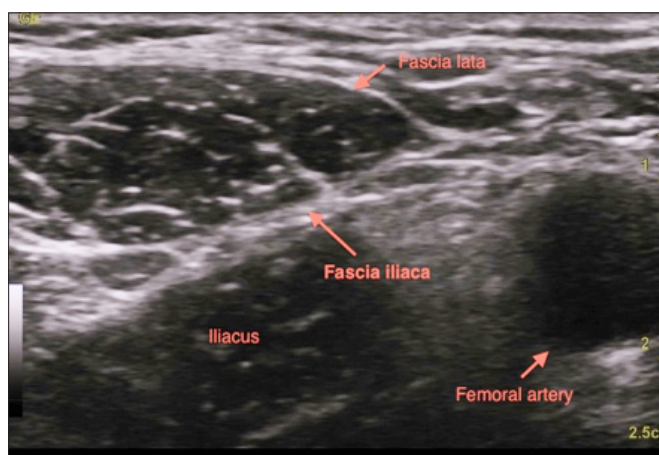


Figure 5: Ultrasound image.

MCQs

1) An 80 year old lady begins to feel unwell five minutes post fascia iliaca block. You notice that you have used 40mls of 0.5 % Bupivacaine rather than 0.25%. Which of the following is NOT a common symptom of local anaesthetic toxicity?

1. Vomiting
2. Paraesthesia
3. Confusion
4. Drowsiness
5. Lip swelling

2) You decide that she may have local anaesthetic toxicity. Which of the following is NOT a common sign of local anaesthetic toxicity?

1. Hypotension
2. Grand mal seizures
3. Widened QRS complexes on ECG trace
4. Tachycardia and arrhythmias
5. Jaundice

3) You decide that she has local anaesthetic toxicity. Which one of the following drugs would be most helpful in managing local anaesthetic toxicity?

1. Glucagon
2. Paracetamol
3. Intralipid
4. Naloxone
5. Penicillin

4) Having given a fascia iliaca block and waited for 15 minutes, the patient states they are still in significant pain in their hip. You decide that the block has failed. Which of the following is the best way to proceed?

1. Repeat the nerve block with 40mls of 0.25% bupivacaine
2. Perform a sciatic nerve block
3. Give an alternative form of analgesia intravenously
4. Ask the patient to take nitrous oxide to help pain relief
5. Use a Thomas splint with traction

HOW TO DO A FASCIA ILIACA BLOCK FOR A FRACTURED NECK OF FEMUR

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5) Your patient is from a nursing home and is known to have dementia and the carers tell you he is pleasantly confused. When you ask for consent to perform the procedure he declines but you are aware he is still in significant pain. Which of the following is the best way to proceed?

1. Carry on and perform the procedure regardless of the patients wishes
2. Consider a psychogeriatric opinion before administering analgesia
3. Perform a capacity assessment and try to involve family members and senior doctors in the decision to perform the block
4. Sedate the patient in order to perform the procedure
5. Ask the pain team to review the patient

Answers

1) Answer 5

Paraesthesia, confusion, vomiting and drowsiness are all symptoms of local anaesthetic toxicity. Lip swelling may present as an allergic reaction to local anaesthetic but is not a symptom of toxicity. Symptoms of local anaesthetic toxicity are most common in the first 15 minutes following administration.

2) Answer 5

Local anaesthetic toxicity can present with a spectrum of signs from seizures, which need to be managed with benzodiazepines, through to cardiac arrest. Although one needs to be vigilant for this, fascia iliaca block is a very safe procedure and risks of toxicity are low.

3) Answer 3

The patient should be assessed using an ABCDE approach using ALS protocol. Intralipid emulsion 20% can be used as an adjunct to cardiopulmonary resuscitation. This is given as a 1.5ml/kg bolus followed by an infusion of 0.5ml/kg/min for 30-60 minutes (2).

4) Answer 3

If nerve block fails it is not recommended to repeat but to try an alternative analgesic such as IV morphine or paracetamol. Reconsider using ultrasound approach for future attempts.

5) Answer 3

This is not an uncommon scenario. Pain relief is an important part of management and should be delivered promptly. It is important to ascertain whether the patient has capacity to make the decision to give consent. If they do not have capacity it is important to involve family members and senior members of staff to help make a strategy of best dealing with the patient's pain.

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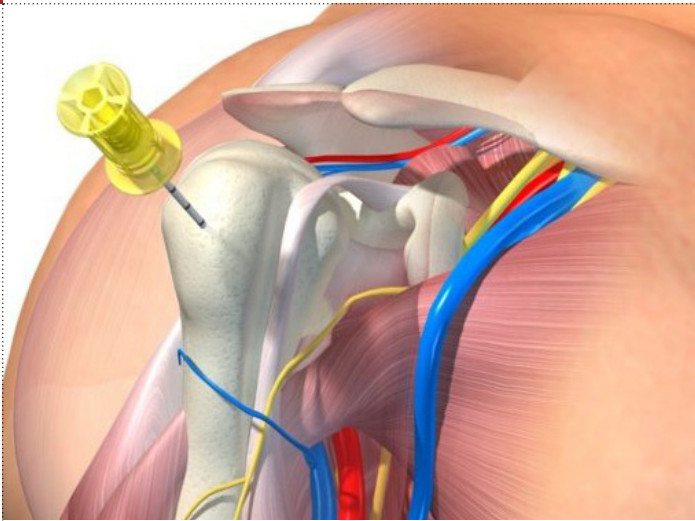
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INTRASOSEOUS ACCESS IN THE EMERGENCY SETTING

D Eddy, A Vaghela



Abstract

Intraosseous access is becoming increasingly popular in the emergency department setting and is now first line alongside intravenous access in resuscitation scenarios in both adults and children. It is important that all junior doctors are familiar with this simple procedure and feel confident to implement it in the emergency setting.

History

An eight year old child was admitted to the emergency department in cardiac arrest of 45 minutes duration. Intravenous (IV) access had been attempted by the paramedics and one twenty-three gauge venflon was inserted. However on arrival in the emergency department this was found to no longer be working. Further peripheral IV access was attempted by the paediatric registrar and after two failed attempts intraosseous access was achieved in the left tibia with the use of an intraosseous EZ-IO® device. Whilst no aspirate was gained, fluids were delivered via the intraosseous route and as the patient was haemodynamically optimised, further peripheral IV access was able to be obtained.

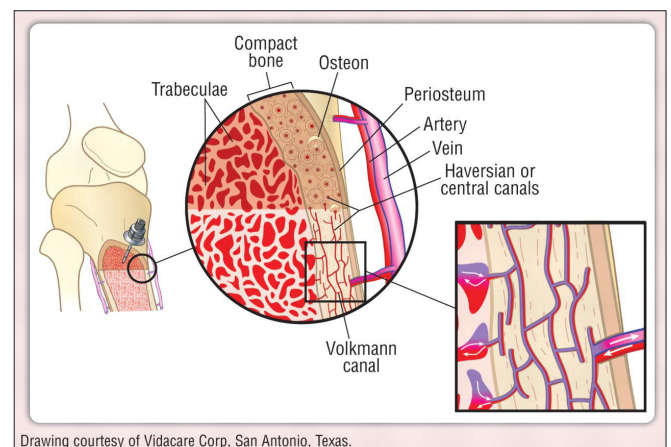
Intraosseous access in the emergency setting

Where patients are haemodynamically unstable, it is essential to be able to gain quick and secure access in order to deliver life-saving interventions. Whilst IV access is the traditional route of choice, intraosseous access provides a reliable alternative in both adults and children (1). This has been recognised by both the European Resuscitation Council (ERC) and Resuscitation Council UK (RCUK) in their latest guidelines for both adults and children (2,3,4,5) with the recommendation that intraosseous access should be used if IV access is difficult or impossible.

Intraosseous Access In The Emergency Setting Patient Management

Despite a recent surge of interest in intraosseous (IO) access, the method has been around since 1922 where Drinker et al. described this as a way to deliver fluids and other medications to mammals (6). It went on to be used extensively throughout World War II for fluid resuscitation of injured soldiers, but as the war ended its popularity declined (7). Research over the past five years is now driving forward once more the use of intraosseous access, particularly in the emergency setting for both adults and children (1,8).

What is intraosseous access?



Drawing courtesy of Vidacare Corp, San Antonio, Texas.

Figure 1: Intraosseous space and blood supply.
Image courtesy of Vidacare Corp.

Intraosseous access is the process where by a needle is drilled in to the intraosseous space through which fluids and medications can be delivered. Whilst peripheral veins can collapse when a patient is haemodynamically compromised, the intraosseous space (Figure 1) is non-collapsible (8). Once access is gained into the marrow cavity, there is good venous blood supply providing a reliable route into the venous system.

INTRASKELETAL ACCESS IN THE EMERGENCY SETTING

D Eddy, A Vaghela

The benefits and complications of IO

When comparing IO to IV access, drug delivery is comparable. Infusions via the IO route have been found to reach the central circulation within one second (9), a speed equivalent to IV access. The process of IO access takes around one minute in comparison to IV access which takes an average of ten minutes (10). All medications which are safe to be administered via the IV route are also safe via the IO route meaning IO access doesn't restrict the medications able to be given.

There are some rare complications of IO access which include the risk of dislodgement, extravasation, compartment syndrome, fracture of targeted bone, infection and pain on use with extravasation being the most commonly documented (8). It should also be remembered that all medications or fluids delivered via the IO route will need to be given under pressure to optimise flow rates (10).

Indications & Contraindications

Indications for intraskeletal access (4, 5, 12)

- Difficult or impossible IV access

Contraindications for intraskeletal access (12)

- Fractured bone at site of insertion
- Previous surgery on targeted bone
- Infection in local area of insertion
- Absence of bony landmarks
- IO in targeted bone in last 48 hours

Consent

If possible, all patients should be verbally consented to the procedure. However in many situations this is not possible, in which case it will be placed in best interest of the patient. In life-threatening situations, IO access should not be delayed in order to seek consent. With children, if the parent/carer is present they should be informed as to what you are about to do in order to avoid distressing them. If you are able to consent the patient or parent/carer, you should inform them that there is a risk of the previously mentioned complications.

Methods used

Manual IO access is less commonly used since the introduction of battery powered devices but can still be found in some areas (Figure 2). However, in adults these can be particularly difficult to use due to increased bone thickness.



Figure 2: Cook® Manual intraskeletal device

EZ I-O® guns are now commonly stocked in emergency departments across the UK (Figure 3). They are battery driven and quick to use.



Figure 3: EZ-I-O® device with needle set, dressing and extension set. Red needle 15mm; Paediatric, Blue needle 25mm; Adolescents/Adults, Yellow needle 45mm; Adults. Picture courtesy of Teleflex®

INTRASOSEOUS ACCESS IN THE EMERGENCY SETTING

D Eddy, A Vaghela

Equipment needed

- Manual IO device with a connector device or EZ-IO® Power Driver, Needle

Set and Extension Set

- Non-sterile gloves
- Cleansing agent of choice
- Luer lock syringe with sterile Normal Saline flush (5-10 mL for adults, 2-5 mL for infant/child)
- Sharps container
- 10ml syringe

Depending on the consciousness level of the patient you may also need

- 2% preservative & epinephrine-free lidocaine (intravenous lidocaine)
- Intravenous fluid

Adults	Paediatrics
<ul style="list-style-type: none"> • Proximal tibia • Proximal humerus • Distal tibia 	<ul style="list-style-type: none"> • Proximal tibia • Distal Femur • Proximal humerus • Distal tibia

Table 1: Sites of insertion for IO access (12)

The most common and popular site of insertion is the proximal tibia due to its ease of location and higher first attempt success rate¹³. Other sites are listed in Table 1. In this article the proximal tibia insertion site will be described.

Location of insertion site



Figure 4: Site of tibial tuberosity.

Adults; Extend the patient's leg. The insertion site is approximately 2 cm medial to the tibial tuberosity (Figure 4), or approximately 3cm below the patella and approximately 2 cm medial, along the flat aspect of the tibia.

Paediatrics; Extend the patient's leg. Pinch the tibia between your fingers to identify the medial and lateral borders. Insertion site is approximately 1 cm medial to the tibial tuberosity, or just below the patella (approximately 1 cm) and slightly medial (approximately 1 cm), along the flat aspect of the tibia.

Manual IO Technique

1. Locate the landmarks for insertion as above
2. Aim the needle set at a 90-degree angle to the bone
3. Push the needle into the skin down to the bone
4. Whilst pushing turn the needle clockwise and anticlockwise until a give is felt
5. Remove needle leaving catheter behind and place dressing around site to secure catheter
6. Attach catheter to connection device such as a smart site and flush with 0.9% normal saline

Battery driven or EZ I-O® technique

1. Locate the landmarks for insertion as above
2. Push the needle set tip through the skin until the tip rests against the bone. The 5 mm mark must be visible above the skin for confirmation of adequate needle set length
3. Gently drill, advancing the needle set approximately 1-2 cm after entry into the medullary space or until the needle set hub is close to the skin. You should feel a give as you enter the medullary space.
4. Hold hub in place and remove needle from catheter
5. Place dressing on site to secure line
6. Prime extension with 0.9% saline and attach to catheter. Flush line and catheter with 10mls saline.

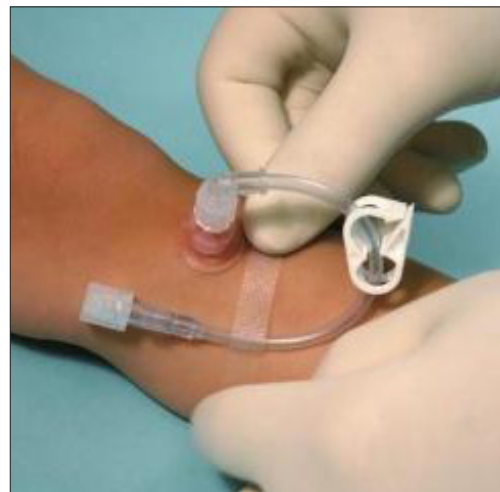


Figure 5: Intraosseous access in the proximal tibia.

INTRAOSSUEOUS ACCESS IN THE EMERGENCY SETTING

D Eddy, A Vaghela

Insertion of an IO in a conscious patient

Where possible IV access should be used in patients responsive to pain as insertion is less painful. However where IO access has had to be gained, pain can be minimised by following these steps;

Adult

1. Prime the extension line with 2% intravenous lidocaine
2. Slowly infuse lidocaine 40 mg IO over 120 seconds
3. Allow lidocaine to dwell in IO space 60 seconds
4. Flush with 5 to 10 mL of normal saline
5. If needed, slowly administer an additional 20mg of lidocaine IO over 60 seconds

Paediatrics

1. Calculate maximum dose of lidocaine for child's weight according to local guidelines
2. Prime extension set with lidocaine or for small doses of lidocaine, consider administering by carefully attaching syringe directly to needle hub (prime extension set with normal saline)
3. Slowly infuse lidocaine over 120 seconds
4. Allow lidocaine to dwell in IO space 60 seconds
5. Flush with 2-5 mL of normal saline
6. Slowly administer subsequent lidocaine (half the initial dose) IO over 60 seconds.

Repeat PRN; consider systemic pain control for patients not responding to IO lidocaine

Removal

IO access is not recommended to be used for greater than 24 hours.

1. Remove all extensions and dressings
2. Attach luer lock syringe to needle
3. Maintaining alignment, twist clockwise and pull straight out whilst avoiding rocking the syringe
4. Dispose in a sharps container
5. Apply pressure and a dressing to the site

For further demonstrations on how to insert IO access, videos are available providing detailed instructions (14).

A junior doctors reflection on intraosseous access.

Intraosseous access is becoming more and more popular across the UK due to its ease of use and speed. It is an easily achievable skill and all junior doctors should feel comfortable in gaining IO access. As a junior doctor, you are a valuable member of the resuscitation team, especially when it comes to gaining access. Junior doctors are well placed to be suggesting intraosseous access when IV access is seen as being difficult as often it isn't thought of soon enough, such as the scenario detailed above.

MCQ Questions

1. Which of the following are not recommended IO insertion sites?

- a) Proximal tibia
- b) Sternum
- c) Distal tibia
- d) Proximal Humerus
- e) Distal Femur

2. Which of the following is not a contraindication to IO insertion?

- a) Fractured femur
- b) Cellulitis overlying targeted area
- c) Osteogenesis imperfecta
- d) Obesity
- e) Previously failed IO access at same site

3. What is the correct procedure for pain relief in adults?

- a) Inject 2% lidocaine in skin surrounding the insertion site
- b) Inject 10mls 2% lidocaine after flushing line with normal saline
- c) Inject 10mls 2% lidocaine with adrenaline immediately after insertion
- d) Inject 40mg lidocaine STAT then flush with normal saline after 60 seconds
- e) Inject 40mg lidocaine over 120 seconds then allow to dwell for 60 seconds before flushing with normal saline

4. How do you remove the IO catheter?

- a) Using forceps pull at tip of catheter
- b) Rock catheter from side to side to loosen and then pull
- c) Attach a luer lock syringe and pull IO catheter directly away from site
- d) Pull directly at IO catheter away from site
- e) Attach a standard syringe and pull at IO catheter

INTRAOSSUEOUS ACCESS IN THE EMERGENCY SETTING

D Eddy, A Vaghela

5. Which of the following is the most common complication of IO insertion?

- a) Fat embolus
- b) Infection at site
- c) Bleeding
- d) Fracture of bone
- e) Extravasation

Teaching notes

1. C

The sternum is not recommended due to its proximity to major vessels and the position obstructing ongoing cardio pulmonary resuscitation.

2. D

Obesity is not a contraindication for IO access. Longer needles are available for these scenarios.

3. E

It is important to flush the line with lidocaine to minimise pain and allow it to dwell in the cavity to ensure maximum efficacy.

4. C

It is important not to rock the needle from side to side as this risks breaking the catheter. Attaching a luer lock syringe is the safest way to remove the catheter.

5. E

The most common complication of IO is for the IO needle to be in the wrong position leading to extravasation.

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FEBRILE & OFF LEGS - WHERE DO YOU BEGIN IN THE ED

A Dimakopoulou, W Howard, K Vithian

Febrile & Off Legs - Where Do You Begin In The ED Patient Management

Abstract

A 47-year old female presented to Accident and Emergency department with recurrent falls, confusion and pyrexia. She was recently discharged from Intensive Care, where she had a prolonged stay and received treatment for pneumonia and *Escherichia coli* bacteraemia. During her second admission she was treated for another episode of sepsis but the origin was not easy to identify. A detailed physical exam revealed signs in a well protected anatomical area and led to definitive management.

Introduction

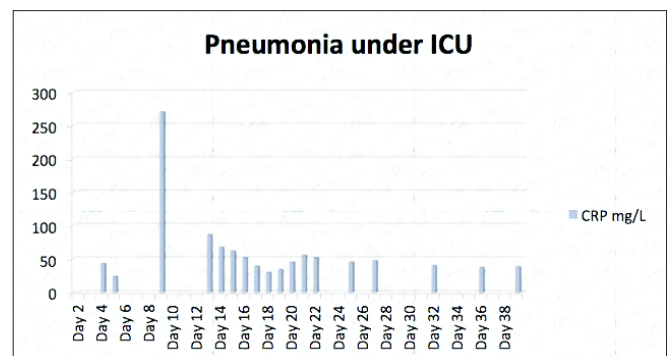
Fever of Unknown Origin is a challenge for physicians in general internal medicine. The list of differentials is broad and significant amount of time may be required before a final diagnosis is made. The two most critical features in management of such cases is a careful history combined with a detailed clinical examination, both of which may have to be performed several times before a conclusion is reached.

Physical examination can occasionally be demoted as it is perceived as “less reliable” compared to laboratory results and modern imaging studies. In our case, history taking was not very informative due to confusion. Clinical progress was made after a thorough physical exam. We would like to present this case to highlight the role of clinical examination, a fundamental diagnostic tool in clinical practice.

Case Presentation

A 47-year-old female patient was admitted to our hospital with multiple falls and recurrent episodes of sepsis. She had history of hypertension, bipolar disorder, fatty liver disease and alcohol misuse.

Approximately two months prior her presentation, she had a prolonged stay in Intensive Care Unit (ICU) with mixed overdose and poor conscious level. She was septic with acute kidney injury and metabolic acidosis. Chest X-Ray findings were suggestive of aspiration pneumonia and a prolonged course of antibiotics was completed. C-reactive protein settled within fifteen days and that is shown on the following graph.



Graph 1: CRP response to initial antimicrobial treatment for pneumonia.

Pyrexia Of Unknown Origin: Commonest Causes & Investigations

Fever higher than 38.3°C and raised C-reactive protein persisted for more than three weeks after our patient’s hospital admission. As a result she was investigated for pyrexia of unknown origin. The commonest causes are outlined in the following table.

Diagnostic categories	Most Prevalent diagnoses of Fever of Unknown Origin
Infections	Endocarditis Syphilis Mycobacterial Abdominal sepsis ie hepatobiliary sepsis or abscess
Neoplasms	Lymphoma Multiple myeloma Solid tumor
Multisystem	Rheumatic fever Sarcoid Lupus Rheumatoid arthritis Giant cell arteritis
Miscellaneous	Drug Fever Factitious Fever
No diagnosis	

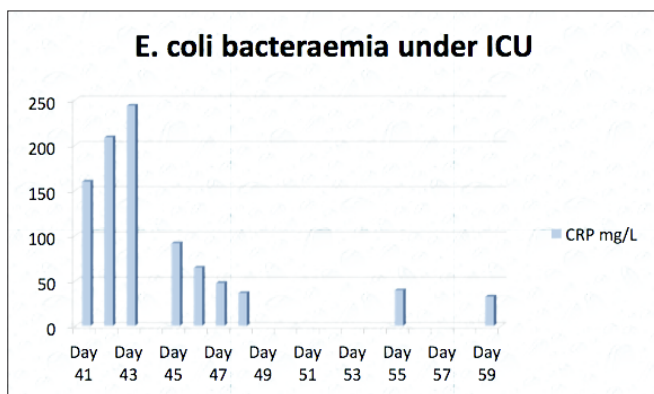
Table 1: Commonest causes of Fever of Unknown Origin.

FEBRILE & OFF LEGS - WHERE DO YOU BEGIN IN THE ED

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Anaemia and leukocytosis could have been supportive of lymphoma, but there was no lymphadenopathy or hepatosplenomegaly. Blood films as well as immunoglobulins were normal and multiple myeloma was excluded. Hepatobiliary sepsis was considered in view of raised alkaline phosphatase, but liver ultrasound was unremarkable. Hepatitis, HIV testing and autoimmune screen were also negative.

New blood cultures were taken and were found to be positive for *Escherichia coli*. In the context of a prolonged febrile illness and central vein catheterization, echocardiography was performed and ruled out endocarditis. CT scan imaging excluded intra-abdominal collection. Finally, urine cultures grew mixed coliform organisms and the most likely source of infection was considered to be the urinary tract. A second course of antibiotics was completed and the downward trend in C-reactive protein is shown below.



Graph 2: CRP response to antimicrobial treatment for *E. coli* bacteraemia.

Diagnostic evaluation tests in cases of fever of unknown origin are listed in Table 2.

Diagnostic Evaluation to establish that a patient has Fever of Unknown Origin
<ul style="list-style-type: none"> • History • Physical examination • Complete blood count, including differential and platelet count • Blood cultures (three sets drawn from different sites with an interval of at least several hours between each set; in cases in which antibiotics are indicated, all blood cultures should be obtained before administering antibiotics) • Routine blood chemistries, including liver enzymes and bilirubin • Urinalysis, including microscopic examination, and urine culture • Chest radiograph
Additional minimum diagnostic evaluation <ul style="list-style-type: none"> • Erythrocyte sedimentation rate or C-reactive protein • Serum lactate dehydrogenase • Tuberculin skin test or interferon-gamma release • HIV antibody assay and HIV viral load for patients at high risk • Rheumatoid factor • Creatine phosphokinase • Antinuclear antibodies • Serum protein electrophoresis • CT scan of abdomen • CT scan of chest

Table 2: Diagnostic evaluation in Fever of Unknown Origin.

Final Presentation & Treatment

Two days after discharge, the patient presented to our Accident and Emergency department with recurrent falls and required a second hospital admission. She suffered from delirium as a result of sepsis and it was impossible to obtain a clear history.

A detailed clinical examination was essential to establishing a diagnosis. An area of erythema in the gluteal region was revealed and an ultrasound confirmed a 10cm gluteal abscess. A CT scan of the lower pelvis confirmed a 10 x 4.5cm subcutaneous abscess in medial portion of the right buttock, which contained gas loculi.

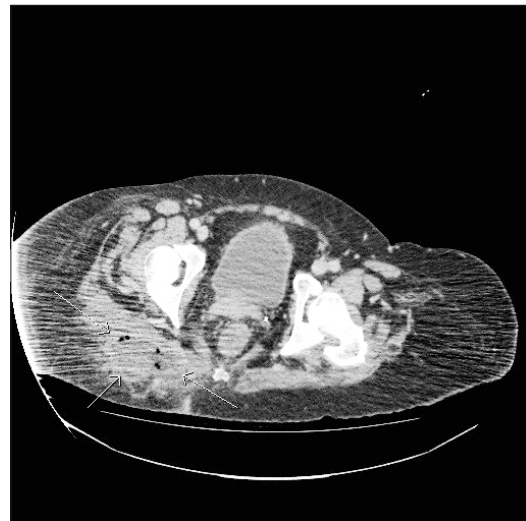


Figure 1: CT scan pelvis subcutaneous abscess in medial portion of the right buttock containing gas loculi.

Gluteal abscesses are difficult to diagnose because they develop in a well-protected location. The clinical picture may be non-specific and further difficulties may occur if there is preceding antibiotic therapy. The principal symptom of anorectal disease is pain. High abscesses have few local symptoms but significant systemic symptoms such as sepsis. In contrast low abscesses are associated with local swelling and cellulitis. Our patient's presentation included symptoms of sepsis with no history of pain affecting the gluteal area. She had several courses of antibiotics which suppressed the inflammatory process without eliminating it.

The diagnosis of a gluteal abscess prompted further microbiology testing. The patient had an intrauterine contraceptive device and high vaginal swab was sent. This grew *Candida*, coliform organisms and skin flora. Additional wound swabs from the gluteal region also grew coliform organisms.

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Definitive therapy was provided by the surgical team with incision and drainage of the abscess. An MRI pelvis one week later showed a shallow 10 cm deep collection in the right gluteal region medially.

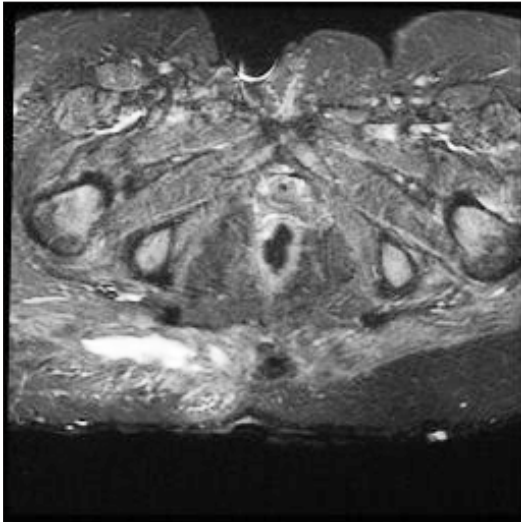


Figure 2: MRIa pelvis one week after incision and drainage shallow deep collection in the right gluteal region medially.

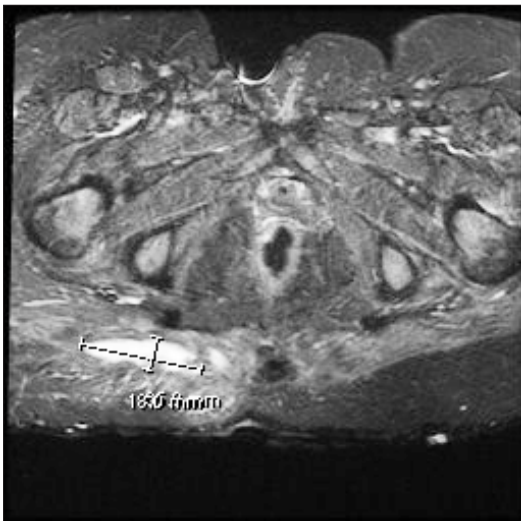
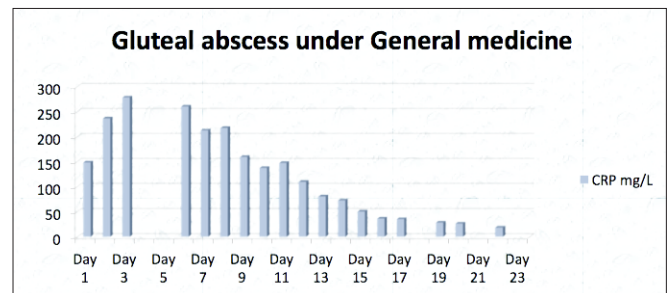


Figure 2: MRIb pelvis one week after incision and drainage shallow deep collection in the right gluteal region medially.

Outcome

As the the gluteal abscess was incised and drained the patient made a good recovery. Haemoglobin and liver functions tests came back to baseline. Inflammatory markers normalised.



It is not clear which factors and when precipitated formation of a gluteal abscess in this case. Our patient had an intrauterine device and high vaginal swab was positive for candida and coliforms. Urinary cultures were also positive for coliforms. It has been reported that diabetic patients with previous inflammatory pelvic disease are prone to develop gluteal collections, but our patient did not have diabetes.

Ultrasound can be used in the first instance to detect gluteal collections. Patients should be best scanned in the prone position. CT and MRI imaging can display anatomical details. Full clinical remission is achieved after drainage of the fluid collection. Management of gluteal abscess includes antibiotics based on cultures, incision and surgical drainage. Needle aspiration under CT control for diagnostic and therapeutic purposes is an alternative established procedure for management.

Physiotherapy was the final management step so that the patient could gain confidence and independence in daily activities.

Discussion

The first definition for Pyrexia of unknown origin was provided by Petersdorf and Beeson in 1961 and included fever higher than 38.3°C for at least three weeks, with uncertain diagnosis after one week of study in the hospital. Petersdorf and Beeson divided FUO into four major etiologies: infection, malignancy, multisystem including collagen vascular disorders and miscellaneous other causes, such as drug fever. The percentage of fever with no identifiable cause was only 7% in this study. Although new imaging techniques, microbiology investigations and laboratory assays have been introduced since 1961, the percentage of patients with fever of unknown origin increased.

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An observational study was conducted by Reilly in 2003 to demonstrate the importance of clinical examination in the care of medical inpatients, and the results were extremely supportive. Hospital records were reviewed retrospectively to determine whether physical findings led to changes in the medical management. Pivotal findings, clinical signs that involve active collaboration of a physician and lead to changes in medical management, were demonstrated in 26% (one in four) patients.

Another prospective study conducted in 2011 by Paley et al. also demonstrated that 4 out of 5 internal medicine patients could be accurately diagnosed close to their admission on the basis of little other than traditional clinical information. History alone was a potent diagnostic tool and was considered useful in establishing the correct diagnosis in 20% of cases. History in combination with physical examination was felt to be useful to establish a diagnosis in 40% of cases.

Case presentations have become complex and multiple medical conditions may co-exist. Physical examination remains an essential diagnostic tool. It is the most reliable guide to modern laboratory and imaging methods. It is not costly, can be repeated as many times as required and can also enhance the doctor patient relationship. A number of studies have shown that physical examination facilitates early diagnosis and remains the standard of care.

MCQs

1. A 47 year old female patient is admitted to A&E with recurrent falls, pyrexia and confusion. She was discharged from hospital 48h ago after a prolonged stay in Intensive care, where she was treated for sepsis due to pneumonia and E. coli bacteraemia. What is the next most appropriate step that is likely to reveal the source of sepsis?

- a) Organise a CT chest-abdomen as soon as the patient is stable.
- b) Contact the next of kin to obtain a collateral history.
- c) Take 3 routine blood cultures drawn from different sites over a period of at least several hours without administering antibiotics, if not already performed.
- d) Ensure a full physical examination is performed.
- e) Perform a lumbar puncture as the most likely cause of confusion and fever in an adult patient is meningococcal meningitis.

2. Pivotal findings are clinical signs that involve active collaboration of a physician and lead to changes in medical management. According to an observational study published in the Lancet in 2003, pivotal findings were demonstrated in:

- a) 1 in 4 patients after retrospective review of the hospital records
- b) 1 in 8 patients after retrospective review of the hospital records
- c) 1 in 10 patients after retrospective review of the hospital records
- d) 1 in 20 patients after retrospective review of the hospital records
- e) 1 in 50 patients after retrospective review of the hospital records.

3. Gluteal infection may be associated with all of the following, EXCEPT:

- a) Parenteral drug administration by buttock injection,
- b) Spread of infection from the chest to the subgluteal region
- c) History of diabetes and inflammatory pelvic disease
- d) Necrotic metastatic neoplasm to psoas muscle
- e) Infected pelvic hematoma following trauma

4. Which of the following statements regarding gluteal abscesses is CORRECT:

- a) Diagnosis of a gluteal abscess is usually obvious, as patients present with fever and pain in the affected area.
- b) Gluteal abscess can be localized based on presenting complaint and symptoms can be immediately relieved after needle aspiration at the site of maximum pain
- c) Obesity, injuries after trauma and previous antibiotic therapy do not alter the clinical picture in patients presenting with gluteal pathology
- d) 'Soft tissue gas' is a hallmark sign for diagnosis of a gluteal abscess, when identified on plain radiographs
- e) Ultrasound can be used to detect a gluteal fluid collection but the patient should be scanned in the prone position.

Answers

1. Answer: d)

History taking is essential to the diagnostic process; however the next of kin is not always available. A radiologist can assist the diagnostic process if he is guided by clinical suspicion – otherwise imaging can be non conclusive. Three routine blood cultures may take a long time before a causative pathogen is revealed. Confusion and fever may be the manifestation of any pyrexial illness and not just meningococcal meningitis. A detailed clinical examination is the first step to elicit signs that can guide investigations.

2. Answer: a)

Physical examination can have a substantial effect on the care of medical patients. According to an observational study published in the Lancet in 2003, among 100 patients 26 (1 in 4) had pivotal physical findings that led to important changes in clinical management. These findings might have important implications for medical educators and quality improvement initiatives.

FEBRILE & OFF LEGS - WHERE DO YOU BEGIN IN THE ED

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3. Answer: b)

The most common cause of gluteal infection is needle contamination during injections in the buttock area. There have been some case reports in the literature regarding gluteal region infection which resulted from pelvic sepsis spreading via the sciatic foramina.

This is the mechanism postulated for subgluteal infections occurring in patients with diabetes and previous inflammatory pelvic disease. Similar cases presenting as psoas abscesses have been described. Spread of infection from the chest to the gluteal region is extremely unlikely. Haematomas can develop after trauma or surgery, especially hip operations, and these can become infected leading to gluteal abscesses.

4. Answer: e)

It is noted that gluteal abscesses can be identified by ultrasound scanning but is advisable that the patient is scanned in the prone position. The gluteal area is well protected, therefore abscesses can be difficult to diagnose radiologically. CT scan can effectively demonstrate the presence and extent of these lesions. Only 50% of patients with a gluteal abscess have soft tissue gas demonstrated on plain films.

Clinical presentation can be vague as pain is often the only symptom and may be felt in the hip, back, legs, perineum or abdomen. Needle aspiration at the site of maximum pain does not always reveal pus. Further difficulties in diagnosis may relate to obesity, anatomical deformities after trauma and previous antibiotic therapy. Antibiotics can suppress the inflammatory process and patients can present with chronic ill health or fever of unknown origin.

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CHEST PAIN IN THE EMERGENCY DEPARTMENT: THINKING LATEROALLY

R Slade, H Shufflebotham, R Kinston



Chest Pain In The Emergency Department: Thinking Laterally Good Clinical Care

Her medications included Omeprazole, Sertraline, Creon and Vitamin D tablets. She had a history of excessive alcohol intake but stated she drank approximately 16 units per week at present, and had an 80-pack year smoking history. She lived at home with her husband and mobilised with a frame.

On examination, the patient looked pale but well. Her observations on arrival were heart rate of 120 beats per minute, blood pressure 120/85 mmHg, a respiratory rate of 18 per minute, oxygen saturations were 95% on air and a temperature of 36.9 degrees Celsius. Her cardiovascular and respiratory examinations were unremarkable. She complained of some mild left upper quadrant pain on abdominal palpation. An electrocardiogram (ECG) on admission showed a sinus tachycardia with non-specific T wave changes. Chest radiograph and urine dipstick testing were unremarkable. Pre-hospital glyceryl trinitrate (GTN) was administered with no relief of symptoms. She had some continuing pain in the ED that was mildly relieved by paracetamol and codeine phosphate.

Initial blood tests revealed an elevated Troponin I (2549, normal range is <4.0), normal blood counts, amylase and renal function. As such, a provisional diagnosis was non-ST-elevated myocardial infarction (NSTEMI) and the patient was managed with medical therapy according to hospital guidelines: Aspirin 300mg, Clopidogrel 300mg and Fondaparinux 2.5mg. The patient was transferred to the Coronary Care Unit on cardiac monitoring for subsequent angiogram.

A coronary angiogram showed that the coronary arteries were unobstructed but there was persistent slow flow in the left anterior descending artery. A left ventricular angiogram showed apical ballooning, in the entire ventricle except the most apical segment, which retained contraction. A diagnosis of Takotsubo Cardiomyopathy was given.

Abstract

We present a case based discussion of a 65-year-old female who attended the Emergency Department with acute onset chest pain, who was subsequently diagnosed with Takotsubo Cardiomyopathy. The case highlights the importance of taking a complete history and thinking laterally. We discuss Takotsubo Cardiomyopathy – or broken heart syndrome, its presentation, diagnosis and management.

Case presentation

A 65-year-old Caucasian woman with a background of severe agoraphobia presented to the Emergency Department (ED) with left sided chest pain. The patient was experiencing episodic chest pain for 2 weeks prior to her admission, but did not seek medical attention due to her agoraphobia. On the day of presentation, she was found by her husband slumped in a chair clutching her chest. The presenting episode was described by the patient as tight and severe, on the left side of her anterior chest with no radiation and was not pleuritic in nature. It lasted approximately 15 minutes with some associated clamminess and sweatiness. There was no shortness of breath, vomiting, dizziness or collapse. There had been no trauma to the chest, no haemoptysis and no recent travel abroad.

On further questioning, the patient stated a history of severe agoraphobia, having not left the house for two years, hypertension and necrotising pancreatitis secondary to alcohol abuse.

CHEST PAIN IN THE EMERGENCY DEPARTMENT: THINKING LATERALLY

R Slade, H Shufflebotham, R Kinston

The patient remained stable during her inpatient admission with no signs of left ventricular compromise. However the patient subsequently self-discharged, declining cardiac rehabilitation. She was discharged on Aspirin 75mg, Atorvastatin 40mg, Bisoprolol 5mg and Ramipril 2.5mg. A follow up transthoracic echocardiogram and clinic appointment were arranged to monitor for left ventricular function and resolution of the apical ballooning.

Discussion

Takotsubo cardiomyopathy (TC) is an acute form of cardiac failure, with a characteristic and reversible ventricular dysfunction, which is associated with normal coronary artery circulation (1). TC has a number of synonyms that are used in the literature of the condition; these include broken heart syndrome, stress cardiomyopathy and left ventricular apical ballooning syndrome (2). Many of these names relate to the condition having an association with stressful situations preceding the onset of the pathophysiological changes.

The term Takotsubo cardiomyopathy was first described by Sato et al in 1990 (3) due to the characteristic cardiac abnormality that resembles a Japanese octopus trap, which has a round bottom and a narrow neck (4). Statistics from the United States demonstrate a frequency of 5.2 per 100,000 for women and 0.6 per 100,000 for men, with a higher frequency in Caucasian patients compared with Afro-Caribbean and Hispanic patients. TC has a worldwide distribution; with cases reported in 6 continents however there have been no large scale studies on global incidence or racial differences (1).

Clinical presentation

As described in the above case, the most common presentation of Takotsubo cardiomyopathy is acute onset cardiac pain, mimicking acute coronary syndrome (ACS). The most common presenting symptoms are chest pain and new onset dyspnea, but patients can also develop acute left ventricular failure (5).

One factor that may influence the diagnostic possibility of TC, is the patient's age and gender. Over 90% of those who develop the condition are female, and the majority of cases occur in postmenopausal women between 61 and 76 years (6). A prospective cohort study suggested that the incidence of TC in postmenopausal women initially diagnosed with ACS could be as high as 6% (7). Thus in this subset of patients presenting with chest pain, particularly after a stressful life event, a diagnosis of Takotsubo cardiomyopathy should be considered.

Evidence of triggers

Throughout the medical literature there are an abundance of psychosocial triggers that have been described for TC including: bereavement, illness, severe arguments, depression, car accidents and personal assault (5). The case described is unusual as there was no apparent isolated causative factor, however severe agoraphobia has been described in numerous case reports as a potential inducing factor for TC (8). An important point to emphasise is that almost one in three patients with TC have no recognised trigger and therefore the lack of a precipitative factor should not exclude the diagnosis (5).

Pathophysiology

The pathophysiology of TC is not fully understood but it is thought the condition arises after a transient raised catecholamine state that leads to cardiac damage. However it is not clear how the myocardial tissue becomes damaged, with proposed mechanisms including coronary artery spasm with spontaneous recanalisation or direct myocardial toxicity from catecholamines (9). These mechanisms reflect the normal coronary vasculature observed in patients with TC. Catecholamine-secreting tumours, such as phaeochromocytomas also induce a hyper-adrenergic state and can cause cardiac damage, so these need to be excluded for diagnosis of TC (as seen in the Mayo Clinic criteria, figure 1).

As the majority of patients with a diagnosis of Takotsubo cardiomyopathy fully recover and cardiac biopsy is not required for diagnosis, the histological findings of TC are not well evaluated. However case reports from post-mortems of TC cases show epicardial lesions in the left ventricle, which are uncommon in coronary artery disease. These lesions include myocyte necrosis, rupture and contraction band formation with interstitial haemorrhage (10,11).

Diagnostic criteria

The most commonly used diagnostic criteria for Takotsubo cardiomyopathy was produced by the Mayo Clinic, based upon radiological evaluation of patients.

- | |
|---|
| 1. Transient hypokinesia, akinesia, or dyskinesia of the left ventricular mid-segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always present. |
| 2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture. |
| 3. New electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin. |
| 4. Absence of Phaeochromocytoma or Myocarditis |

Figure 1: Mayo clinic diagnostic criteria for Takotsubo cardiomyopathy/ Apical ballooning syndrome (12).

CHEST PAIN IN THE EMERGENCY DEPARTMENT: THINKING LATERALLY

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Investigations

Patients presenting with acute onset chest pain are always evaluated systematically, with ECG and cardiac biomarkers used routinely. There is no characteristic ECG pattern seen in TC. One case series from the Minneapolis Heart Institute showed that ST elevation was present in 56% of patients, 17% had widespread T-wave inversion, 10% had previous evidence of an anterior MI (abnormal Q waves or abnormal R wave progression) and 17% had nonspecific changes (13). Thus ECG is not helpful in distinguishing between ACS and TC. As with ECG changes, cardiac biomarkers are not specific and are commonly raised in TC reflecting the myonecrosis present in Takotsubo (14).

As discussed TC typically mimics acute coronary syndrome (ACS), so these patients commonly receive coronary angiogram as an initial diagnostic imaging. Angiography reveals normal coronary vasculature, which differentiates the condition from ACS (2). The characteristic ventricular wall abnormalities are clearly seen on left ventriculography, with classical images of hypokinesia/akinesia/dyskinesia of the apical and mid ventricular segments, with basal hyperkinesia (figure 2).

Care needs to be taken to determine that the coronary arteries are not obstructed and the wall motion abnormalities extend beyond that of a single artery, as obstruction of the left anterior descending artery can also produce a similar ventricular abnormality (4). These findings can also be easily demonstrated on cardiac MRI and echocardiogram.

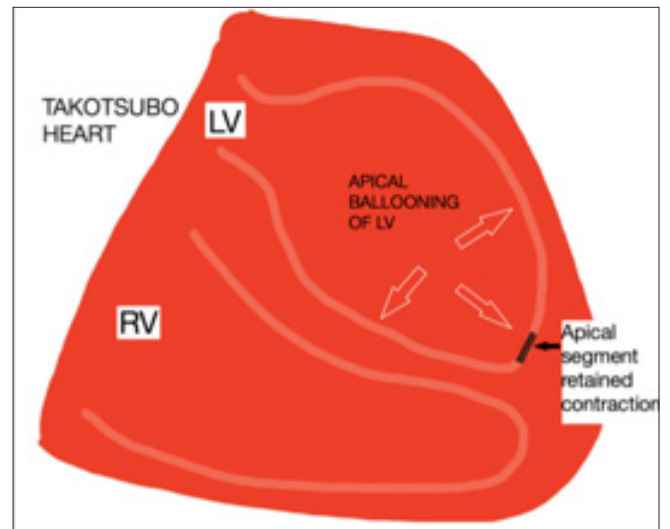
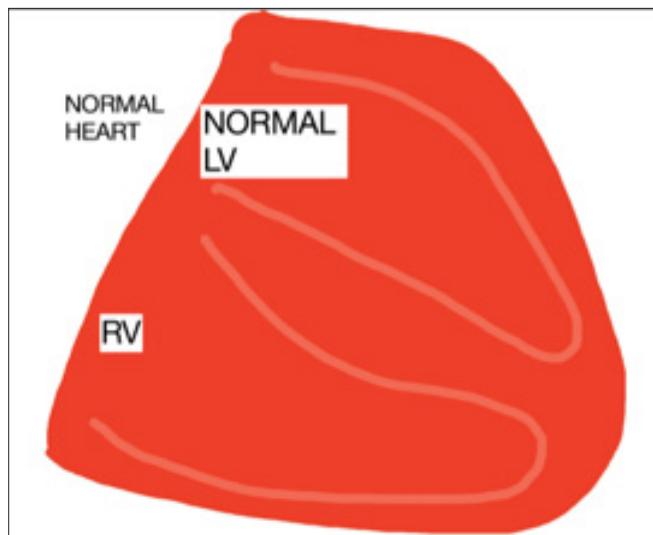


Figure 2: A pictorial representation of the changes in the left ventricle occurring in Takotsubo cardiomyopathy. There is a distinctive pattern of wall motion abnormalities including apical or midventricular akinesia/hypokinesia with ballooning and basal hyperkinesia.

Treatment

Takotsubo is generally managed with supportive therapies to maximise cardiac function. In the acute setting ACE inhibitors and beta-blockers are usually started unless contraindicated. Patients with a severely reduced left ventricular function should be managed in an intensive care or high dependency unit and in cases of cardiogenic shock an intra-aortic balloon pumps can be utilised to maintain cardiac output (2).

The use of inotropic support is usually contra-indicated as it can potentially worsen LV outflow tract obstruction and exacerbate catecholamine damage of the myocardium (2). A repeat echocardiogram should be performed several months after the initial event, to monitor for resolution of left ventricular function and apical ballooning (4).

Prognosis

Patients with Takotsubo cardiomyopathy generally make a complete recovery, but patients should be monitored for signs of left ventricular compromise which is a significant early complication that can affect mortality. The in-hospital mortality rate of the condition was shown to be 4.2% in a large retrospective study of nearly 25,000 cases of TC.

CHEST PAIN IN THE EMERGENCY DEPARTMENT: THINKING LATERALLY

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While males comprise the minority of cases they have a higher mortality rate (8.4%) compared to females (3.4%) (15). Although the condition is generally thought to be transient and reversible, cohort studies have shown a 10% recurrence rate in a 4-year follow up period (16).

MCQs

1. What is the pathophysiology of TC believed to be?

- a) An increase in free radicals causing direct damage to the myocardium
- b) A blunt traumatic injury to the chest
- c) A raised catecholamine state that results in cardiac damage
- d) As a direct result of excessive alcohol intake over a prolonged period of time
- e) Cardiac toxicity from inappropriate drug intoxication

2. What is the treatment for Takotsubo Cardiomyopathy in a patient with compromised left ventricular function?

- a) CRT-D (cardiac resynchronisation therapy device)
- b) Supportive therapies +/- intra-aortic balloon pump
- c) CABG
- d) Supportive therapies +/- inotropes
- e) Drug eluting stents via coronary angioplasty

3. In the UK, what is the most appropriate investigation that should be used to ensure the resolution of the left ventricular function over time?

- a) Transthoracic echocardiogram
- b) Cardiac MRI
- c) Chest radiograph
- d) MIBI scan
- e) Repeat ventriculogram

4. How is Takotsubo cardiomyopathy diagnosed?

- a) Brain natriuretic peptide (BNP) levels
- b) Chest radiograph and Troponin I levels
- c) Troponin I levels and ECG
- d) Ventriculography
- e) Clinical examination

5. What is the in hospital mortality rate associated with TC?

- a) 90-95%
- b) <10%
- c) 100%
- d) 50%
- e) 15-20%

Answers

Question 1 - Answer: (C)

It is believed that the condition results from a transiently raised catecholamine state that directly leads to cardiac damage. Catecholamine-secreting tumours, such as pheochromocytomas can also cause cardiac damage, so these need to be excluded before a diagnosis of TC can be appropriately made.

Question 2 - Answer: (B)

TC is managed with supportive therapies such as ACE inhibitors and beta-blockers to maximise cardiac function. If the left ventricular function is compromised but gradually improving, an intra-aortic balloon pump is often used as bridging treatment to maintain cardiac output until the left ventricle regains better function.

Question 3 - Answer: (A)

The most common investigation performed several months after the initial presentation is transthoracic echocardiogram. This is a simple, non-invasive test that is able to image the heart to ensure resolution of left ventricular function and apical ballooning.

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Question 4 - Answer: (D)

TC is most commonly diagnosed with left ventriculography, which reveals a characteristic dilatation of the apical segment (hypokinesis/akinesis/dyskinesis) and a narrow neck. These findings can be seen also on cardiac MRI and echocardiogram. However as these patients are usually managed as having acute coronary syndrome, cardiac angiography typically is performed first. There is no evidence that ECG or cardiac biomarkers can diagnose TC, as these can be abnormal in both acute coronary syndrome and TC.

Question 5 - Answer: (B)

Patients with TC normally make a good recovery. The mortality rate was shown to be 4.2% in a large cohort study, with males having worse outcomes than females.

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TIME TO REFOCUS? AN OBSERVATIONAL STUDY EVALUATING THE EXTENT OF REDUNDANT MEDICAL PRACTICE IN TWO “EFFICIENT” UK EMERGENCY DEPARTMENTS

S Smith, M Beresford



Time To Refocus? An Observational Study Evaluating The Extent Of Redundant Medical Practice In Two “Efficient” UK Emergency Departments

Patient Management

Introduction

Emergency department (ED) crowding is a pervasive issue of international significance. This relates to its negative impact on clinical outcomes including mortality, time to treatment and error rate, as well as its effect on patient and staff satisfaction(1-4). Understanding the flow of patients through ED is a pre-requisite for developing strategies to alleviate crowding. It can be conceptualised as resulting from the interplay between input, throughput and output factors(5).

Many Emergency Medicine (EM) physicians believe that input factors, such as the access to primary care, and output factors, such as the level of occupancy of inpatient beds, likely outweigh throughput factors in terms of their influence on metrics of crowding(6). Unfortunately, for the most part, these lie outside the remit of EM and thus the preponderance of research into ED crowding has focused on throughput.

Throughput interventions fall broadly into two categories: changes to ED process and changes to ED structure(7-10). Process interventions involve changes to the way in which actions are sequenced. Examples in ED include consultant-led triage, streaming patients based on severity and the institution of standard protocols for the treatment of common conditions, all of which have been shown to ameliorate crowding(11-13).

Structural interventions involve changes to the ED environment. Examples include shortening lab turnaround times, which has been shown to alleviate crowding, and increasing ED capacity, which, counter-intuitively, seems to reduce efficiency(14, 15). Identifying suitable throughput interventions has been facilitated by the deployment of business improvement methodologies such as “lean thinking”. This approach, which attempts to eliminate “waste” by means of employee-driven iterative process redesign (“kaizen”), has been shown to consistently assuage crowding and improve clinical outcomes, however a positive publication bias likely exists(16).

This systems engineering based approach, which has proved very useful in reducing metrics of crowding, has formed the basis of most studies evaluating efficiency in ED to date. Unfortunately, this seems to have had the secondary effect of drawing EM into the axiomatic belief that all the processes that occur are useful, and “if only we could do them quicker”. Rather, based on anecdotal experience, we believe there is likely to be lots of redundancy in the ED process itself.

Abstract

Emergency department crowding negatively impacts patient care and patient satisfaction. To date, interventions aimed at alleviating crowding have sought to reduce wasteful “downtime” between medical engagements through process reorganisation and department restructuring, ignoring the possibility that redundancy likely exists within process steps themselves. Our objective was to quantify this under-researched aspect of redundancy.

Methods

For 108 patients arriving at two UK “A&E” departments, a senior ED consultant evaluated whether medical interventions (e.g. medical assessment, blood investigations, imaging) were clinically indicated at the time of occurrence, and then retrospectively, if each intervention ultimately affected management.

Results

As expected, patient assessment was frequently repeated (mean 2.36 per patient), but surprisingly, this was often by staff of the same decision-making level. Furthermore, while frequently requested (n=31), senior review only materially affected management on one occasion. The ordering of unnecessary blood tests was also found to be commonplace (clotting studies (26% indicated), liver function tests (19% indicated), and CRP (30% indicated)).

Discussion

A profound degree of redundancy exists within the process steps that constitute normal ED flow. While clinical uncertainty necessitates that some interventions inevitably fail to impact patient outcome, our prospective analysis of clinical indications for the medical interventions that make up patient journey reveals the actual degree of redundancy to be far beyond this. Addressing the complex aetiology underlying this form of redundancy could represent a novel approach for alleviating ED crowding.

Key Words: *Emergency Departments, redundancy, efficiency.*

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Crucially, this area has yet to be studied in detail and could represent a fruitful area for anti-crowding interventions. To this end, we decided to chronicle the passage of patients through ED, from point of entry to discharge, recording the number of medical assessments performed, any investigations ordered and any treatments given. In an attempt to identify redundancy, we evaluated the clinical indication for each intervention at the time it occurred and then considered retrospectively whether the intervention had materially altered the management plan.

On a separate note, it is important to emphasise that ED crowding is under-researched in the U.K. - a recent review identified only 5 primary papers - with the bulk of studies conducted in Australia and the US (8). Given the notable operational differences separating U.K. “Accident and Emergency” departments, we believe that this literature base desperately needs expanding (17, 18). Another objective of this observational study was to provide an insight into the operation of two typical UK EDs.

Methods

This was an observational study that prospectively followed the care given to patients in two Emergency Departments. Both departments belong to the same organisation (a United Kingdom National Health Service Trust), but administratively, are largely separate. One is a large University teaching hospital, one a small district general hospital.

We chronicled the journey of each patient in our cohort using a standardised pro-forma, which was completed at the time of patient assessment in ED by a senior consultant, who simply observed, and did not contribute to clinical decision-making. Using this pro-forma, the observing ED consultant recorded the number of times each patient was assessed and the type of medical professional conducting the assessment, as well as any investigations that were performed.

Investigations were deemed clinically indicated if conducted as part of a standardised presentation-based ‘care-set’ or if on interview, the medical professional could offer a clinical explanation for why the result would potentially alter management. Furthermore, the reviewing consultant recorded any treatment that was provided, including the administration of drugs, wound care and the application of medical devices such as splints and dressings. We did not record administrative tasks such as ‘booking in’ and patients were not followed-up after discharge.

The sample was a convenience sample, depending on assessor availability, with data collection starting from 0800 hours on weekdays, and finishing when a minimum of 50 cases had been collected at each site. Our sample comprised consecutive presentations to the respective Emergency Departments, and as such included a mixture of clinical presentations and triage categories.

We selected this time window as, in general, this is the least busy time of the day, and thus is likely to represent the baseline for efficiency, as it is known that increased workloads lead to increasing inefficiency. The data are reported as raw numbers. No patient identifiable data was collected, and this was not an interventional trial, which meant that ethical approval was not required.

In the two centres we studied, patients follow a pre-ordained care pathway, which begins with assessment by a nurse, who measures the individual’s ‘vital signs’ and orders investigations based on a presentation-based protocol (‘care sets’). The patient is then assessed by a clinician of decision-making level, (either a medical practitioner or less frequently, a nurse practitioner) who may request further investigations (e.g. radiographs, CT, occupational therapy assessment) and initiate treatment. If the patient is referred to an in-patient admitting team then the patients will be assessed further by junior members of the admitting team.

Results

We reviewed the care episodes of 108 patients (hospital 1=57, hospital 2=51), from point of entry to point of discharge from ED (see Table 1).

Patient inclusion depended only on observer availability with no exclusions made on the basis of clinical or demographic variables. For perspective, the average daily attendances for each department are 100 and 220 patients, respectively.

The patients in our cohort were subject to a total of 255 assessments, with 171 of these including some form of physical examination. Blood was taken for analysis on 53 occasions, and 31 patients had radiographic investigations. 37 patients received physical treatments, the remainder receiving advice only. Our aim was to expose redundancy in two guises; first, interventions performed without clinical indication i.e. those without an anticipated impact on patient management; second, replicated interventions i.e. those obtaining information already acquired elsewhere.

As expected, each patient underwent multiple assessments (mean=2.36). These were typically performed in a hierarchical manner with nurse assessment followed by junior doctor ‘clerking’ and then ‘senior assessment’. Of note, Figure 1 indicates that staff of the same decision-making level frequently carried out repeat assessments. For example, the number of junior assessments far exceeds the total number of patients.

The reviewing consultant also collected information regarding the outcome of any assessments performed, most notably the impact of ‘senior assessment’ on patient management. Strikingly, as depicted in Figure 1, ‘senior assessment’ altered management on only one out of 19 occasions. Undoubtedly senior assessments can be important in providing reassurance to juniors regarding diagnosis and management. In these cases, review is indicated even though there is no measurable effect on patient outcome. That said, anecdotally, the majority of ‘senior assessments’ observed in this study failed to meet even this threshold of clinical utility.

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		Hospital 1 (n=57)	Hospital 2 (n=51)
Patient Assessment	History	158	97
	Examination	109	62
	Observations	19	31
Blood investigations			
	FBC	23	30
	U&Es	23	26
	Troponin	7	11
	LFTs	18	19
	Amylase	0	6
	CRP	18	9
	Clotting Studies	17	10
	VBG	11	30
	ABG	1	2
	Blood Culture	6	0
	Misc (alcohol, BHCG, TFTs)	3	3
Other Investigations			
	ECG	19	12
	Urinalysis	5	5
	Xray	29	29
	CT	4	0
Treatments			
	Drugs	12	12
	Fluids	5	0
	Wound care/dressing	7	2
	Procedural Sedation	1	0
	OT assessment	5	0
	Misc (catheterisation, foreign body removal)	3	0
Referral			
	Admission	12	9
	Outpatient	4	0

Table 1: List of all interventions performed (n=108 patients).

Key: ECG: electrocardiogram, FBC: full blood count, U&E: urea and electrolytes, LFTs: Liver Function Tests, CRP: C-reactive protein, VBG/ABG: venous/arterial blood gas, CT: computerised tomogram ("CAT scan")

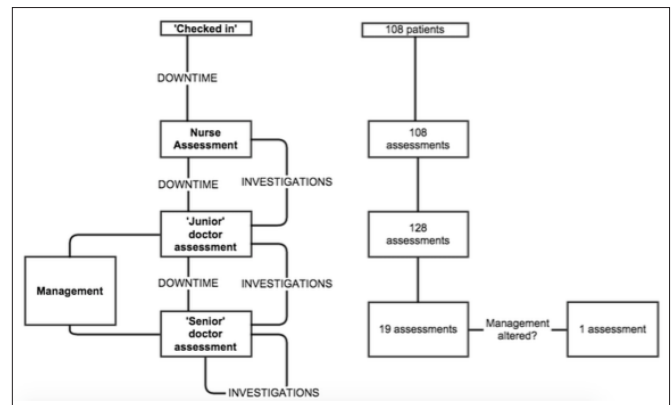


Figure 1: Patient journey through Emergency department. (A) typical patient journey, (B) patient journey, including absolute number of medical assessments and outcome of senior reviews, for 108 patients attending 2 Emergency Departments studied on 2 separate days.

When blood was taken, there was always clinical indication for at least one blood test, however many patients had additional blood tests performed that were not indicated. As demonstrated in Figure 2, the most common 'unnecessary' blood tests were clotting studies (26% indicated), liver function tests (19% indicated), and CRP (30% indicated). In addition, our analysis failed to capture redundancy introduced by performing different blood tests with the same indication. For example, U&Es and VBG both provide information on electrolyte status; it is pointless performing both these tests for this reason in a single patient, despite both being technically 'indicated' (and listed so in Figure 3). This occurred quite frequently.

In relation to radiological investigations, 14% of radiographic examinations were deemed unnecessary (Figure 2). This included 'routine' chest radiographs for admitted patients, and a small number of appendicular radiographs not fitting strict criteria (such as the Ottawa ankle rules).

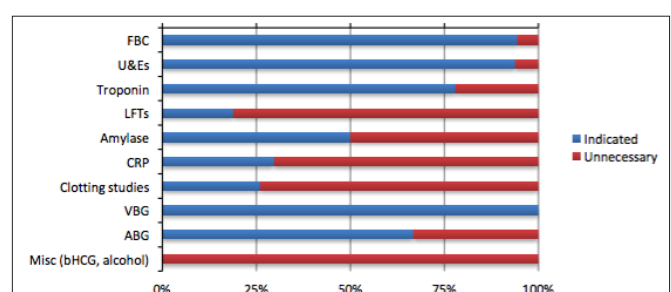
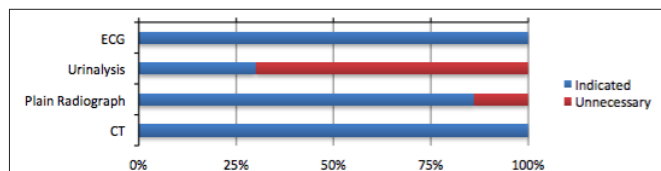


Figure 2: Proportion of unnecessary and clinically indicated investigations as a percentage of total number ordered for 108 patients attending the 2 emergency departments (hospital 1, n=57; hospital 2, n=51) studied on two separate days as determined by observing senior ED consultant. (A) Blood investigations, (B) Other investigations.

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In summary, our results reveal a considerable degree of superfluous activity occurring throughout the patient journey through ED. This includes unnecessary assessments performed by medical staff, the widespread request for blood tests without clinical indication, and the ordering of diagnostic imaging without compliance with inclusion criteria.

Discussion

As for the majority of UK Emergency Departments post-4 hour wait directive, the hospitals included in this study have been required to scrupulously examine and refine throughput to maximise efficiency and alleviate crowding. Despite this, when reviewing the patient journey through these departments, there remains a significant amount of redundant activity, both in the form of replicating information gained elsewhere and acquiring information that is not clinically useful.

Patient Assessment

Our results reveal that patients are assessed multiple times in ED: we believe this to be a profound source of redundancy. We acknowledge that different clinicians will ask patients different questions and therefore repetition increases the probability that pertinent details will be identified. That said, there is a clearly a trade-off between improving patient safety through repeated assessment, and clinical efficiency in terms of money and time expended. We argue that the data presented here demonstrates that this compromise is not being properly addressed.

For example, it is difficult to justify repeated assessment by junior staff, which intuitively is unlikely to confer a patient safety benefit. This redundancy equates to just under 4 hours of medical practitioner time over half a day in each of these departments, assuming that an ‘efficient’ assessment takes approximately 20 minutes (history and examination).

As discussed previously, while a change in management is not a pre-requisite for a ‘useful’ senior assessment, it is important that reviews are not requested unnecessarily. Rather, the data presented here indicates that this practice may instead be commonplace. This is particularly significant from a crowding perspective as waiting for senior assessment often incurs a significant time delay. Furthermore, senior ‘time’ is particularly costly.

Going even further, one may suggest (and many have) that if senior assessment is frequently required, it may instead be preferable to ‘front load’ senior decision makers or experienced doctors, eliminating the need for the redundant ‘junior history’. This comes however with the disadvantage that senior decision makers would have to perform tasks that did not utilise their unique skill-sets (e.g. ‘clerking’ the patient rather than decision making), likely rendering this arrangement highly cost-inefficient despite being time-efficient.

Investigations

Our results also reveal that many of the investigations performed in ED lack clinical indication or replicate information gained elsewhere i.e. they are redundant.

We believe our assessment of ‘clinical indication’ in this study to be relatively robust for two reasons. First, the assessment was based on whether the clinician ordering the test could justify test inclusion to a senior ED consultant. This consultant observed the entire patient journey, which, along with their unique expertise, made them ideally placed to make an accurate judgement of whether management was likely to be affected. Secondly, the observing consultant assessed the investigations prospectively, avoiding the issue that many investigations fail to alter management despite being clinically indicated at the time of ordering, which would have been a problem with retrospective analysis.

One possible explanation for the redundancy reported here is that tests may occasionally be ordered on a ‘just-in-case’ basis. For example, the large number of redundant requests for ‘clotting studies’ may reflect a belief that they should be requested ‘just in case’ a D-Dimer is subsequently needed. This form of redundancy is understandable given the potential delay associated with ‘adding tests on’. In reality however, many of the redundant blood investigations were simply protocol violations, performed because blood had been drawn for one test, and it is simple to order additional tests.

As unnecessary blood tests were always requested where blood needed to be taken for at least one clinically indicated test, reducing the number of superfluous tests would be unlikely to save much time. By contrast, reducing the number of redundant tests would undoubtedly produce a significant cost saving. Using national reference costings, we estimate this to be approximately £1,136 pounds for the patients in this study alone, even before we include the human resource cost of ordering, taking and checking the results(19). This cohort of patients equates to less than a third of the daily total for these departments.

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Of note, senior decision makers rely less on investigation use and it follows therefore that "front-loading" may have the additional benefit of reducing the frequency of redundant investigations(20). Furthermore, the introduction of near patient testing has been shown to produce a concomitant reduction in the number of laboratory investigations ordered(14). Interestingly, near patient testing also reduces crowding metrics such as ED-length of stay, however the time saving associated with bypassing a laboratory altogether far exceeds that associated with reducing the number of investigations ordered(14).

Limitations

There are a number of aspects of redundancy we failed to address in the study. Firstly, our data demonstrated that redundant practice was much more common on the non-ambulant area of the department (often called 'majors' in UK EDs). These patients tend to have greater medical needs and are therefore more complicated in terms of 'processing'; hence intuitively a greater degree of 'wastage' during the care episode is to be expected.

Crucially, inclusion in our study was determined only by the time of arrival (with no exclusions made on the basis of severity), and had we instead only included 'majors' cases, it is likely we would have found a greater degree of redundancy. Furthermore, within the 'majors' cohort some presentations are managed according to pre-ordained 'care pathways' e.g. MI, while others are dealt with in a more ad hoc manner e.g. syncope. One would expect that including only the latter would also increase the amount of redundancy exposed.

Secondly, while it is often assumed that treatments are by definition not redundant, the efficacy of these interventions is rarely assessed, only the need for them in the first place. It may be that, for example, a particular analgesic medication given, fails to materially affect patient's pain, and as such is 'redundant'.

Another big limitation of this study was the methodological drawback of using a single ED physician to review the patient journey. In this respect, we attempted to maximise reliability by excluding the observing physician from patient management, by recording each patient journey using a standardised pro-forma, and by interviewing medical practitioners using a standardised set of questions.

Summary

To date, research into ED crowding, mainly conducted overseas, has focussed on process re-organisation and improving the structures that facilitate individual process steps(8). In this study, we have instead examined the nature of constituent process steps and attempted to evaluate their necessity in relation to patient outcome. Despite an intense focus on ED throughput in recent years, we have shown in this study that a significant amount of process redundancy still exists, with repeat patient assessment commonplace, for little apparent benefit, and unnecessary investigations frequently ordered without clear clinical indication.

While we acknowledge that the a priori nature of decisions is such that an inherent redundancy exists, the actuality is that the true degree of redundancy far exceeds this collateral amount. We believe that in future it will be important to lend greater focus to the interventions that occur as part of ED flow, rather than simply the way in which these are sequenced, delivered and facilitated. It will also be important to address practices that are convention rather than evidence-based.

'Best of five' Questions for the paper

1. Regarding Emergency Department overcrowding, the following is true:

- A: Overcrowding only affects patient satisfaction, and length of stay
- B: Overcrowding is due to mismatch between workload and resources within the Emergency Department
- C: Overcrowding can be prevented by improvement in efficiency
- D: Overcrowding affects patient mortality rates
- E: Overcrowding is normal in Emergency Departments

2. If clinical redundancy is defined as 'activity that does not directly benefit patient care' then which of the following are true?

- A: All redundant activity should be eliminated from the process of patient care
- B: Some redundant activity is necessary, so all redundant activity should be tolerated
- C: Redundant activity is not harmful to the patient, so may be tolerated
- D: Pragmatically it is difficult to reduce redundant activity, so it should be tolerated
- E: Quality and Performance Improvement plans should identify and reduce unnecessary redundant activity

3. Which of the following are the key performance indicators defined by the Department of Health for Emergency Departments in England?

- A: increase ambulatory care, unplanned re-attendances rates, Stoke and cardiac thrombolysis, patient feedback, total time in Emergency department, time to initial assessment, time to treatment
- B: increase ambulatory care, reduction in community violence, left without being seen rates, patient feedback, total time in Emergency department, time to initial assessment, time to treatment
- C: increase ambulatory care, unplanned re-attendances rates, left without being seen rates, patient feedback, total time in Emergency department, time to initial assessment, time to treatment
- D: reduction in community violence, unplanned re-attendances rates, Stoke and cardiac thrombolysis, patient feedback, total time in Emergency department, time to initial assessment, time to treatment
- E: sepsis care bundle, unplanned re-attendances rates, analgesia, staff satisfaction, total time in Emergency department, time to initial assessment, time to treatment

TIME TO REFOCUS? AN OBSERVATIONAL STUDY EVALUATING THE EXTENT OF REDUNDANT MEDICAL PRACTICE IN TWO "EFFICIENT" UK EMERGENCY DEPARTMENTS

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4. In which of the following scenarios is there NO redundant activity?

- A: Computerised Tomography of the head in an epileptic who has had a seizure and has fully recovered
- B: Blood cultures in an afebrile patient with a recent 'flu like illness
- C: Abdominal X-rays in a patient with suspected appendicitis
- D: Clotting studies in a non-warfarinised patient with abdominal pain
- E: Repeat ECG in a middle aged male with chest pain

5. Clinical Quality measures in Emergency Departments often include some time-based measures. Which of the following is true?

- A: According to the Goodhart principle, when quality measures become targets they lose utility as measure of quality
- B: Time based measures are always simple to collate
- C: Targets are amenable to 'gaming' by reporting organisations, and as such should be viewed with caution
- D: Waiting times are not necessarily important to patients
- E: Reduction in time based measures of quality is easily achieved

Answers

1. Answer: D.

There is good published evidence that overcrowding in the ED affects many aspects of patient care, notably mortality. The Royal College of Emergency Medicine document 'Crowding in EDs' provides a good summary of causes and possibly solutions.

Crowding is multifactorial, with both pre and post ED factors, as well as intra-departmental factors. The 'Theory of Constraints' model would suggest the prime cause of a process blockage is an upstream rate-limiting factor, in this case often bed availability. A single 'magic bullet' solution does not therefore exist, although increased efficiency may help.

2. Answer: E.

Some activity which is 'redundant' as far as individual patient care is concerned are actually important from a wider perspective; it is easy to think of examples of some clinical governance activity, and some teaching activity is 'redundant' by this definition, but is important from either patient protection or training perspective. While it is difficult to reduce redundant activity, this should not deter clinicians from attempting to reduce it; there are efficiency and cost savings possible. Some redundant activity may be harmful; the Academy of medical Royal Colleges 'Choosing Wisely' campaign lists clinically unnecessary radiology investigations as an example.

3. Answer: C.

The seven clinical quality indications chosen by the Department of Health (DoH) and implemented in 2011 were: increase ambulatory care, unplanned re-attendances rates, left without being seen rates, patient feedback, total time in Emergency department, time to initial assessment, time to treatment. Definitions of measurement and parameters are given in the DoH guidance: A&E Clinical Quality Indicators: Implementation guidance and data definitions. Available at: http://webarchive.nationalarchives.gov.uk/20130107105354/http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_122868.

Stroke and cardiac thrombolysis times are important national targets, but not necessarily ED based. Sepsis care bundle compliance and analgesia re subject to national audit, but are not defined clinical quality indicators by the DoH. Data is commonly shared with community partners, but this is not a defined quality indicator. Staff satisfaction levels are often measured, but not a performance indicator, as not directly related to patient care.

4. Answer: E.

There is good published evidence to show that routine CTs after seizure when the patient has returned to normal, blood cultures in well normo-thermic patients, non-anti-coagulated patients who are not septic, abdominal films in patient where obstruction or foreign body is no suspected are all clinically unnecessary. Initial ECGs are known to have low sensitivity in myocardial infarction, and repeating ECGs (in any chest pain patient who is not very low risk of acute coronary syndrome) is good practice from a governance perspective as it increases the sensitivity.

5. Answer: A.

This is a controversial and widely discussed topic with Emergency Medicine. Patients expect and deserve care that is timely, as well as of high standard. Since the implementation of the '4 hour standard' in English EDs, it has become clear that achieving and maintaining this standard is not simple. While systems may make measurement of timings along the patient journey simple, this is not always so. All targets can be 'gamed', but this does not diminish the importance of the target, or the significance of results. Charles Goodhart was an economist who formulated a law originally stated as "As soon as the government attempts to regulate any particular set of financial assets, these become unreliable as indicators of economic trend", but has come to be popularly stated in a format as above.

TIME TO REFOCUS? AN OBSERVATIONAL STUDY EVALUATING THE EXTENT OF REDUNDANT MEDICAL PRACTICE IN TWO "EFFICIENT" UK EMERGENCY DEPARTMENTS

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A NOVEL ACUTE CHILDHOOD RASH - A CASE BASED DISCUSSION

C Page, N Roberts



Abstract

This article describes the clinical presentation of a new viral illness. The case illustrates the importance of taking a full history to make the correct diagnosis and the implications of the diagnosis for clinical carers as well as the family. The differential diagnosis will be explained and other conditions that share a similar association will be discussed.

Case History

A 1 year old girl presented to the emergency paediatric department with a two day history of fever and the rash illustrated in figures 1 and 2. On direct questioning her parents reported that she had lost her appetite and was "not herself" and was complaining that her hands were sore. She did not have diarrhoea. Her parents reported that she was not moving her hands but this seemed to be due to pain in her skin rather than any muscle pain. She had a past history of mild eczema but this had not been active for several months. She was otherwise well until this illness.

A Novel Acute Childhood Rash - A Case Based Discussion Patient Management



Figure 1: Discrete papules and erosions periorally with some early secondary impetiginisation.



Figure 2: Papules, vesicles and bullae on the hand associated with swelling. Note similar discrete lesions on the leg. They were also present on her feet and both legs.

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Examination

The rash on the girl's hands, feet and legs consisted of papules (small, circumscribed, superficial solid elevation of the skin less than 1 cm diameter) (1) and vesicles (small circumscribed elevations of the epidermis containing a serous fluid less than 1 cm in diameter) (1) with erosions (loss of the epidermis, shallow moist lesions) (1) on an erythematous (red) background. On inspection she had mouth ulcers (full thickness loss of epidermis and dermis). (1) She was also febrile.

Investigations and management

Given that this little girl was "off her food" she was mildly dehydrated. She was admitted for observation and swabs were taken for viral isolation. Her management included analgesia, an intravenous infusion of Aciclovir, antiseptic soaks and the application of emollients. The next day she was afebrile but was eating and drinking very little. However after 48 hours she had significantly improved and was discharged home. She continued a 5 day course of oral Aciclovir in the community and was followed up in Dermatology Outpatients at three weeks. The child's mother reported that her rash had resolved completely but that one nail had become dystrophic. Her mother also reported new blisters on her own fingers.

Discussion

In approaching this clinical case, one needs to consider the differential diagnosis of a young child presenting with fever and rash and in addition oral ulceration. See tables 1 and 2.

Viral (e.g. Varicella Zoster Chicken Pox)
Bacterial (e.g. Staphylococcus Scalded Skin Syndrome, Tuberculosis)
Parasitic (e.g. Malaria)
Leukaemia
Lymphoma
Juvenile Idiopathic Arthritis
Kawasaki Disease
Sarcoidosis
Haemophagocytic lymphohistiocytosis
Systemic Lupus Erythematosus
Erythema Multiforme major

Table 1: Rash & fever in children – Differential Diagnosis List.

Viral infection (e.g. Measles, Epstein Barr, Varicella Zoster, HHV 6, HSV, HIV)
Bacterial infection (e.g. Syphilis, Tuberculosis)
Coeliac Disease (dermatitis herpetiformis)
Inflammatory Bowel Disease (Pyoderma gangrenosum)
Behcet's syndrome
Tropical sprue
Kawasaki Disease
Wegener's Granulomatosis
Pemphigoid
Epidermolysis Bullosa
Systemic Lupus Erythematosus
Reiter's Syndrome
Lichen Planus
Erythema Multiforme major
Steven's Johnson Syndrome/ Toxic Epidermal Necrolysis
Langerhans Cell Histiocytosis

Table 2: Rash & Oral Ulceration – Differential Diagnosis List.

The distribution of this rash (hand, foot, and mouth but with involvement of the torso and extremities, i.e. a widespread exanthema) (2) is key to reaching the diagnosis. In addition, knowing that the child has a past history of eczema is also an important finding. The clinical diagnosis for this child's symptoms and signs is Eczema Cocksackium, which is a new viral illness due to the enterovirus Cocksackie A6 that has a predilection for areas of eczema (2).

The manifestations of classical hand, foot and mouth disease, due to Cocksackie A16 are well known. In contrast, Eczema Cocksackium is due to Cocksackie A6 (3) and has been recognised as a cutaneous eruption with a more varied presentation – with 4 distinct morphologies (see table 3) (2) and tends to occur in the Autumn and Winter rather than the Spring preponderance of Cocksackie A16. (2)(4) Whilst our patient did not have myalgia and diarrhoea these are frequent, associated features. (3) 62% of patients in one series had a pre-existing skin condition, 82% of whom had atopic eczema. (2) As in other reported cases the perioral and limb involvement shown in figures 1 and 2 are typical of this condition. (2) (5)

Widespread Vesiculobullous and erosive lesions extending beyond the palms and soles
An eczema herpeticum like eruption: termed Eczema Cocksackium
An eruption similar to Gianotti Crosti Syndrome
A petechial or purpuric eruption

Table 3: Morphology of Cocksackie A6 Infection (2).

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In cases where petechiae or purpura are present, a full blood count becomes mandatory as the differential diagnosis for a young child presenting with such symptoms in the context of fever and oral ulceration must include an underlying haematological malignancy. Viral isolation for PCR rather than culture (2)(6) is the best means of confirming the diagnosis of an enterovirus infection and swabs can be taken from vesicles and respiratory secretions. (2)(5).

When instigating a management plan for children who present with a fever, rash and oral ulceration it is important to consider a number of factors.

The first essential management decision is to consider whether this scenario of a child with fever and rash requires infection control measures. In fact, this child had a new viral infection, first reported in the UK in 2014 (7). The importance of this is that the emergency department staff and the parents are at risk of clinical infection, as they have no prior immunity (8).

There have been documented cases of adults becoming infected with Coxsackium A6 (8) and the child's mother in our case did in fact develop a similar rash. It is therefore imperative that good cross-infection prevention measures are instigated as soon as this possible diagnosis is entertained, e.g. arranging for an isolated cubicle for the child and advising close contacts to adhere to infection control guidance.

The management of Eczema Coxsackium can be remembered by the 4As: Analgesia, Aciclovir, Antiseptic soaks and the Application of Emollients. In our child's case, paracetamol proved sufficient pain relief, however, the ward staff were aware that such patients can need stronger analgesia and that pain relief is an important part of management. In order to treat the Coxsackie virus, intravenous infusions of Aciclovir are given (9).

The child's rash tends to initially improve within 24 hours and completely resolves within 5 days (9). To prevent secondary bacterial infection of the erosions associated with Eczema Coxsackium, antiseptic soaks of Benzalkonium Chloride 0.5% bath oil are used. Finally, this child had the application of 50:50 white soft paraffin: liquid paraffin emollient as it was tolerated the best given its fairly liquid formulation that glided over the sore, eroded skin lesions.

Thus the clinical management is similar to Eczema Herpeticum. However, grouped clusters of vesicles, which are characteristically umbilicated before eroding, distinguish the rash of Eczema Herpeticum and in this illness Herpes Simplex Virus is isolated by PCR (10). Both viral illnesses can be complicated by serious cardiovascular, neurological and respiratory disorders and deaths have certainly been reported with enterovirus infections but to date this seems to be linked to co-infection with Coxsackie and enterovirus 71. (2)(4) (5)(11) Coxsackie A6, despite being a more debilitating illness than Coxsackie A16 infection has not to date been associated with an increased risk of these complications. (2)(11)

5 MCQs (best of fives) with answers

1. Which of the following is least likely to occur in children with eczema?

1. A disseminated rash with herpes simplex
2. A disseminated rash with coxsackie
3. Alopecia areata
4. Prolonged molluscum contagiosum
5. Cutaneous Crohn's

2. Involvement of mucous membranes makes which viral infection less likely?

1. Epstein barr virus
2. Enterovirus
3. Mumps
4. Measles
5. Chickenpox

3. Prognosis is poorest with which of these infection related syndromes?

1. DRESS syndrome
2. Toxic Epidermal Necrolysis
3. Kawasaki disease
4. Staphylococcal Scalded Skin Syndrome
5. Shingles

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4. The most common presentation of Eczema Cocksackium is?

1. A boy with purpuric blisters
2. A boy with purpuric blisters and nail involvement
3. A girl with Gianotti Crosti Syndrome like papules and papulovesicles
4. A girl with purpuric blisters
5. A girl with purpuric blisters and nail involvement

5. In classical hand foot and mouth disease the following is typical?

1. The route of transmission is often faeco-oral
2. Relatively well child with grey white oval vesicles occur on the hands, feet and buttocks
3. Less than 5% body surface area is affected
4. Ill child with pharyngeal ulceration
5. Incubation period of 21 days

Answers

1. The correct answer is 5.

Children with eczema are not more prone to Crohn's disease. There is an association between Psoriasis and Crohn's disease (12) but not with eczema. Children with eczema are more prone to all viral infections and typically develop a disseminated rash with both Herpes Simplex Virus and Cocksackie virus. Herpes Simplex Virus can present with clusters of vesicles, which often erode and can become widespread – so called Eczema Herpeticum (10).

As with eczema Cocksackium, the lesions in eczema Herpeticum usually involve current or previous sites of atopic eczema. In addition, children with eczema are also more prone to alopecia areata (13). In the context of eczema, molluscum contagiosum can persist for up to two years (14).

2. The correct answer is 3.

Mumps is not typically associated with mouth ulceration. (15) However, pharyngeal lesions are typical of enterovirus infection, (2) chickenpox is associated with rose spots and measles causes Koplik spots. Epstein Barr Virus is a cause of recurrent and sometimes prolonged aphthous ulceration, which can precede B cell lymphoma. (16)

3. The correct answer is 2; Toxic Epidermal Necrolysis.

DRESS syndrome -Drug Rash with Eosinophilia and Systemic Syndrome has recently been linked to re-activation of HHV6 and has a 10% mortality. (17) It can be precipitated in children by the administration of medicines. Amoxicillin is thought to facilitate re-activation of HHV 6 and anticonvulsants are commonly the culprit (1 in 1000 to 1 in 10,000 exposures) (17). 6 cases were reported in 2014 following the anti-psychotic Ziprasidone leading to a warning being added by the FDA to its drug information leaflet. (18).

Toxic Epidermal necrolysis is the most sinister skin condition with widespread skin fragility and loss and a high mortality; up to 35% of children. (19)

The aetiology of Kawasaki disease remains unknown and although an infection seems a likely cause the causative organism has not been identified. It has a 0.1-0.3% mortality rate. (20)

Staphylococcal Scalded Skin Syndrome is more common in newborns and has a 4% mortality. (21)

Shingles is due to a reactivation of chickenpox, which is caused by varicella zoster virus, which is a member of the alpha herpes group (22).

4. The correct answer is 3; Cocksackie A6 infection can produce petechiae and purpura and in a recent case series affected 18% of children. (2)

Eczema Cocksackium has been found to be more common in females (70:30%) in a USA cohort of patients aged between 4 months and 16 years, mean 1.5 years. (2) Children with Eczema Cocksackium present with fluid filled blisters; both vesicles (<1cm in size) and bullae (>1cm size). (2) In 50% of cases it can present as Gianotti Crosti Syndrome (asymptomatic dark red lichenoid papules and papulovesicles that predominantly affects the legs then arms then finally the face) (2).

In the same case series, nail changes were observed in 25% of children with Eczema Cocksackium. The study described Onychomadesis (separation of the proximal nail plate from the nail matrix and nail bed) and Beau's lines (horizontal ridging of the nail plate), which typically occurred 3-8 weeks after the acute illness. (2)

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5. The correct answer is 2.

The incubation period of typical hand, foot and mouth disease is 3-5 days (as is for the atypical coxsackievirus) (23). Chicken pox incubation period is usually 21 days.

Hand, foot and mouth disease typically occurs in the under 5s with transmission occurring from vesicles, by faeco-oral route or by respiratory secretions. (2) Involvement of the buttocks is common (5) in addition to hands, feet and mouth but involvement of other body sites is unusual so that the total body area involved is generally less than 5%. By contrast, 60% of children with Eczema Coxsackium had over 10% body surface area involvement. Children are relatively well and have pharyngeal ulceration in classical hand, foot and mouth disease.(2)

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SHINGLES

J Sharif, A Wright

Shingles Patient Management

Abstract & Case Overview

You are the junior doctor on call and you are called to review a patient with a rash on his lower back. He is an 83 year old gentleman with a history of ischaemic heart disease and hypertension. Nursing staff are concerned the patient may have Shingles. This case based discussion will give an overview on how to assess and confirm a diagnosis of Shingles, when to initiate management including the management of post herpetic pain. This aim of this case base discussion is to assist the Foundation Year Doctor in the diagnosis and clinical decision making in the commonly encountered scenario of Shingles. It discusses some of the infection control issues to consider with Shingles.

What are the salient features would you look for to confirm a diagnosis of Shingles?

Assess for pain

This is usually a burning pain preceding the rash following the distribution of one or more sensory dermatomes. It most frequently involves the T5 or T6 dermatome. When cranial nerves are involved it is most commonly the ophthalmic division of the trigeminal nerve. The pain may precede the rash for two to three days and can often be confused for cardiac, gastrointestinal or pleural pain.

Shingles is also known as Herpes Zoster. It is the result of reactivation of the virus from the dorsal root ganglion in a patient who has previously had chickenpox (Varicella Zoster).

Assess for systemic signs

These can be headaches, fever or general malaise. Sometimes patients have associated regional lymphadenopathy

Look for the rash

Vesicular rash arising in the region of painful skin. Initially it appears as a crop of grouped red papules in one dermatome or adjacent dermatomes. The vesicles then evolve becoming pustular and later crusting. New lesions can appear for several days.

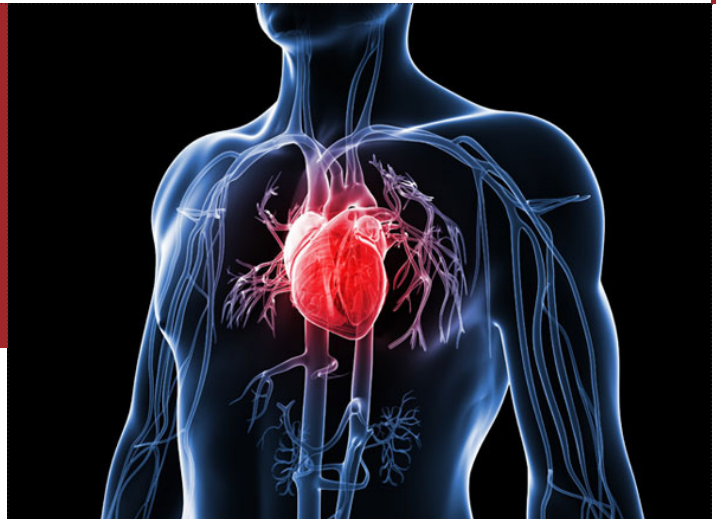


Figure 1: Illustrates the typical features of acute shingles in the L5 dermatome.



Figure 2: Crusted vesicles in a dermatomal distribution on the lower leg.

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What would you do to confirm your diagnosis?

Take a swab from the affected area. It is important to remember to take a viral swab and send it for viral PCR.

How would you treat this patient?

There are three things to consider when approaching the management of Shingles. This includes the management of the virus, the relief of acute pain and the management and prevention of post herpetic neuralgia.

Antiviral therapy

Evidence suggests that antiviral therapy can help reduce the duration of the outbreak and have a role in reducing pain. However, it is only effective if used within the first 72 hours and does not have a role if the rash has crusted¹. Aciclovir a DNA polymerase inhibitor is the most commonly used antiviral drug. It can either be given as an oral tablet or intravenous solution. Immunosuppressed patients require special consideration; immunosuppressed patients should always receive antiviral therapy. Patients who are severely immunocompromised should be considered for intravenous therapy.

Causes of immunosuppression to consider

- *Patients on chemotherapy*
- *Patients with underlying malignancies - for example up to a quarter of patients with Hodgkins lymphoma will develop Shingles (2)*
- *Patients with HIV - up to 15 times increased risk (3)*
- *Patients on long term steroid therapy*
- *Elderly patients*

As this gentleman in the case is 83 years old most physicians would start aciclovir treatment.

Management of acute pain

Appropriate pain management is essential. It should be escalated as per the World Health Organisation pain ladder. Some patients' pain may be so severe that it necessitates opiates for pain control. Topical treatment can also be useful in pain control. Calamine lotion can have a soothing effect. Once the lesions have begun to crust topical treatment with capsaicin or lidocaine can be considered.

There is variable evidence about the use of oral steroid therapy in the acute phase of Shingles. In combination with aciclovir there is evidence to suggest prednisolone can reduce the acute pain⁴. The evidence behind the benefit of steroids in reducing post herpetic neuralgia is mixed; some studies suggest a benefit (5) and some show no decrease in the incidence of post herpetic neuralgia (4).

Post Herpetic neuralgia

This is the commonest sequelae of Herpes Zoster. It is defined as pain that persists for more than one to three months after the rash of Shingles has resolved⁶. Post herpetic neuralgia increases in incidence and severity with age. It is more likely to occur if patients experienced dermatomal pain prior to the rash, if the acute pain was severe and if the rash was prolonged.

The pain is generally neuropathic in nature and patients will describe it as burning or shooting in nature. Another distressing feature is allodynia, defined as the sensation of pain in response to a normally innocuous stimulus. Allodynia is present in up to 90% of patients. The pain can be very severe and is often distressing and disabling for patients.

NICE suggests following the guidelines for management of neuropathic pain when treating post herpetic neuralgia. Effective analgesic medications are as follows: amitriptyline, duloxetine, gabapentin or pregabalin. In the first instance the patient is offered one of these agents. If unsuccessful the dose should be increased before switching to an alternative agent, it can take weeks to get to an effective dose. Stronger opiates such as tramadol should only be used as rescue therapy and should be avoided in the long term. Topical capsaicin cream can be used as an alternative if oral therapy is not effective.

The patient's granddaughter comes to visit. She is 3 months pregnant and after seeing her granddad expresses concerns that she has come into contact with Shingles. The nurses ask you to come and give her some advice. What would you do?

Ask about her exposure

The first step is to assess if the patient has had significant exposure. This would include being in the same room for a period of 15 minutes or longer, face-to-face contact and contact in the setting of an open ward housing an individual with Shingles.

Shingles is infectious two days before to five days after the onset of the rash. People who have not previously had chickenpox can therefore contract the disease if they have significant exposure during this period.

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Ask her if she has previously had chickenpox?

Maternal varicella infection can be associated with a variety of fetal abnormalities; the risk is highest if varicella is contracted in the first trimester. Complications occur in about 2% of maternal varicella infections. Such complications include; central nervous system and ocular defects, and limb hypoplasia. Neonatal death has occasionally been reported.

If a pregnant woman does not recall previously having chickenpox the first step is to check her blood for evidence of previous varicella infection. If she is IgG positive then this suggests she has previously had chickenpox and therefore is no longer at risk. If she is IgG negative and either IgM positive or negative, this suggests no previous exposure or immunity. As such the mother is at risk of developing maternal varicella and there is a risk to the foetus. In such instances the mother should receive zoster immune globulin (VZIG). This should be given at any stage of pregnancy.

Are there any other considerations in shingles?

Ophthalmic involvement

This is a special consideration as eye involvement can be serious and result in mucopurulent conjunctivitis, episcleritis, keratitis and anterior uveitis. It is important if this is suspected that the patient is referred urgently to ophthalmology.

Secondary Bacterial infection

This is a common complication of Herpes Zoster. After settling the rash may become increasingly crusted and exudative, the patient may begin to feel systemically unwell. It is important to recognize and treat secondary bacterial infection promptly. If suspected a bacterial swab should be sent and antibiotics to cover skin organisms should be started.

Test yourself Questions

1. You are in General practice, you see a 54 year old lady with a rash on her chest, she has no other past medical history. You take a history and think it is shingles, she states the rash started 5 days ago. You examine her and see a crop of crusted lesions in region of one dermatome. How would you manage this patient?

- Start aciclovir tablets
- Admit to hospital her for IV aciclovir
- Assess her pain and offer appropriate analgesia
- Start oral antibiotics
- None of the above

2. You see a woman who is 5 months pregnant in your GP surgery. She is concerned as her nephew has got chickenpox; she attended his birthday party yesterday. The patient does not recall having chickenpox in the past. What would you do?

- Reassure the patient and take no further action
- Ask her to monitor for signs of a rash and return if she develops one
- Offer her aciclovir treatment
- Admit her to hospital to IV aciclovir
- Check her antibodies to varicella zoster, if there is no evidence of previous immunity arrange varicella IVIG

3. You are the FY1 on call. You have successfully diagnosed shingles in a patient and have started the appropriate medical management. The patient is in an open bay on the ward, the ward sister asks you about isolation. What steps do you take?

- Reassure sister that no further isolation or precaution is needed
- Leave the patient in the main bay but advise that health care professionals should wear personal protective equipment (gloves and aprons) for all contact with the patient
- Leave the patient in the main bay and advise that no pregnant people or immunosuppressed individuals should have contact with the patient
- Isolate to a side room but advise no personal protective equipment needs to be worn
- Isolate to a side room and take barrier precautions

4. You are on a General practice placement and you see a patient with post herpetic neuralgia. They have tried amitriptyline for one week and there has been no improvement in their pain. What do you do?

- Increase the dose of amitriptyline and explain to the patient that it can take weeks to achieve pain relief
- Stop the amitriptyline and switch to an alternative agent for treatment of neuropathic pain such as: duloxetine, gabapentin or pregabalin
- Continue with amitriptyline and add in another agent such as: duloxetine, gabapentin or pregabalin
- Refer to pain clinic
- Add in capsaicin in addition to the amitriptyline

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5. You are a Foundation Year Doctor in A and E. You diagnose shingles affective the ophthalmic division of the trigeminal nerve, in anotherwise well patient. Which of the following is the best course of action?

- Refer to medics for admission for IV aciclovir
- Start oral aciclovir and refer to dermatology for urgent assessment
- Start oral aciclovir and refer to ophthalmology for urgent assessment
- Start oral aciclovir and discharge
- Reassure the patient that the condition is self-limiting and no further management is required.

Answers

1c

The patient no longer has vesicles and the rash started more than 72 hours ago therefore acyclovir therapy is unlikely to be of any benefit in this patient

2e

In the absence of a history of chickenpox it is essential to establish any evidence of immunity. If there is no evidence of previous immunity the patient will need varicella IIG.

3e

It is important that all patients with shingles are isolated to a side room and barrier precautions are followed they are a significant infection risk to other patients on the ward. Healthcare staff and relatives who do not recall having chickenpox should avoid contact with the patient.

4a

As per NICE guidelines the dose should be increased and continued for a number of weeks before considering an alternative agent.

5c

This patient should be started on aciclovir and referred urgently to Ophthalmology to assess for any ocular involvement.

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PARANEOPLASTIC PEMPHIGUS MASQUERADING AS POSSIBLE TOXIC EPIDERMAL NECROLYSIS: A DIAGNOSIS THAT MAY GRADUALLY EMERGE & MUST NOT BE MISSED

S Paget, R Groves, J Setterfield

Paraneoplastic Pemphigus Masquerading As Possible Toxic Epidermal Necrolysis: A Diagnosis That May Gradually Emerge & Must Not Be Missed Patient Management

Abstract & Case Overview

Over the course of three years an Afro-Caribbean man, in his sixth decade of life, presented on three separate occasions to different hospitals with oral ulceration, painful conjunctivitis, widespread cutaneous bullae and dermatitis. He was on numerous medications and was initially diagnosed with toxic epidermal necrolysis. However, his symptoms and signs persisted and worsened despite cessation of these drugs and in the context of underlying chronic lymphocytic leukaemia further investigations were undertaken to look for paraneoplastic pemphigus. We look at this challenging process and examine the importance of continuing to evaluate the working diagnosis when managing a complex case such as this.

Case History

A sixty-four year old Afro Caribbean man with a background of stable chronic lymphocytic leukaemia (CLL), prostate cancer and chronic Hepatitis B infection presented hypotensive with a widespread erythematous rash, painful oral ulceration and painful eyes. On full examination some areas of blistering and erosions were seen. A working diagnosis of toxic epidermal necrolysis (TEN) was made and he was transferred to the intensive care unit (ITU) for careful supportive treatment with potent topical steroids to affected areas, regular emollients and although not standard treatment for TEN, a trial of intravenous (IV) hydrocortisone.

A skin biopsy taken at this time supported the diagnosis of a suspected drug reaction displaying interface dermatitis (inflammation predominantly seen at the dermo-epidermal junction). The culprit drug for initiating this episode was not clear, but most likely candidates included co-trimoxazole, allopurinol and lamivudine which were all discontinued. He was noted during admission to be intermittently neutropenic, possibly related to sepsis. Blood cultures grew staphylococcus aureus, enterococcus and pseudomonas, thought to have originated from infected skin erosions, and multiple courses of antibiotics were administered. His skin and mucosal erosions slowly improved and after a two month hospital admission he returned home.



Eighteen months later he re-presented to a different hospital with extensive painful oral, lip and tongue ulceration with dysphagia and bullae on his arms. He had been given a three week course of fluconazole for suspected oral candida during a haematology clinic and at the time it was felt this was a further severe adverse drug reaction consistent with Steven Johnson syndrome (SJS). He was treated with sixty milligrams of prednisolone as a weaning course and potent topical steroids.

His condition slightly improved enabling him to return home in 10 days. At review in clinic a month later, however his oral ulceration was no better and he had developed penile erosions as well and widespread dermatitis. A diagnosis of paraneoplastic pemphigus (PNP) was suspected secondary to CLL and he was admitted again for intravenous immunoglobulin, antibiotics and potent topical steroids and was referred for review in a specialist clinic in a different hospital.

Two months later, a serum sample was tested by indirect immunofluorescence (IMF) and showed intercellular IgG antibodies on Monkey oesophagus and binding to rat bladder epithelium with intercellular and basement membrane binding (Figure 1). The same serum sample was tested for pemphigus antigens using Enzyme-linked immunosorbant assay (ELISA) and revealed anti-desmoglein 3 antibody (anti-DSG3 Ab) positive and Anti-DSG1 antibody negative. The combined intercellular and linear basement membrane positivity on rat bladder supported a diagnosis of paraneoplastic pemphigus. In light of this and with the intention of treating the mucosal and cutaneous findings, he received two cycles of chemotherapy for his CLL in the form of fludarabine, cyclophosphamide and rituximab (FCR).

PARANEOPLASTIC PEMPHIGUS MASQUERADING AS POSSIBLE TOXIC EPIDERMAL NECROLYSIS: A DIAGNOSIS THAT MAY GRADUALLY EMERGE & MUST NOT BE MISSED

S Paget, R Groves, J Setterfield

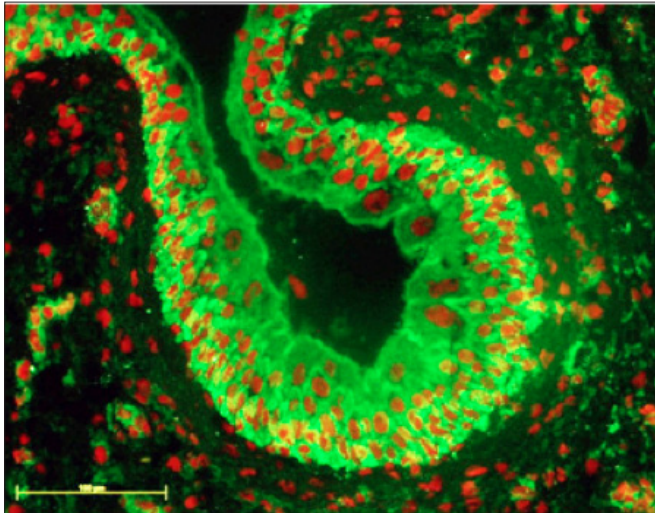


Figure 1: Intercellular IgG antibodies binding to rat bladder epithelium confirming the diagnosis of PNP.

Later, further immunosuppression was achieved by introduction of mycophenolate mofetil (MMF) enabling the prednisolone to be weaned down to 10mg. During this period he developed painful vesicles over his left buttock and thigh in a dermatomal distribution consistent with shingles and he was admitted for treatment with acyclovir. Concurrently he had a worsening of his oral ulcerations during this period. However with treatment of his CLL his symptoms began to improve. He demonstrated healing of the majority of his oral and penile ulcerations which was mirrored by a gradual reduction in the anti-anti-DSG3 antibody titre Ab titre and loss of rat bladder binding on indirect immunofluorescence.

Unfortunately two years later the patient re-presented with extensive blistering, erythema and bilateral conjunctivitis, suspected initially to be a further episode of TEN due to recent re-exposure to fluconazole in the community. His diagnosis of PNP was not available at the time of admission. His illness was complicated by *Staphylococcus aureus* bacteraemia requiring numerous courses of IV antibiotics. He received supportive treatment again and his prednisolone was increased to 60mg daily. After a month long admission he returned home.

Two weeks later he sustained a traumatic head injury after a fall at home resulting in a subarachnoid haemorrhage and a possible extradural haematoma. He was admitted for further observation but neurosurgical intervention was not felt to be appropriate.

Although his cutaneous erosions had resolved he continued to display widespread dermatitis. His oral ulcerations at this stage were extensive and haemorrhagic with a panstomatitis and his penile ulcerations were also severe despite continuing his prednisolone. Further serum samples were taken for indirect IMF and were negative for anti-DSG3, but clinically his signs were consistent with active PNP.

Discussion was made with the haematology team regarding further treatment for the CLL, but concurrent sepsis prohibited agents such as rituximab being a viable option. He developed numerous episodes of bacteraemia during admission receiving antibiotics for these, but then contracted *Clostridium difficile*, which was treated with vancomycin. He was transferred to ITU for support and began to improve. However, despite this after a month long admission he gradually deteriorated and after discussion with his family active treatment was withdrawn and within a few days of best supportive end of life care he died.

Discussion

This patient's case highlights some very important aspects of medical care in critically unwell patients. It demonstrates the difficulty in reaching a clear diagnosis in the case of mucocutaneous disease in the acute setting. It highlights the challenges in maintaining seamless care when patients present to different hospitals. When drugs are considered to be a possible aetiological factor the precise time course is essential in trying to establish how likely they are to be relevant. Thus obtaining an accurate history and close liaison with the GP and other involved teams is essential.

Wherever there is a blistering rash there are numerous potential differential diagnoses (see table 1 and Figure 2 and 3). Certain clinical features in this case help to narrow this: particularly the location of the bullae and erosions. Widespread bullae suggest a systemic process and involvement of mucosal surfaces with haemorrhagic erosions point towards SJS/TEN or pemphigus. Given this patient's exposure to new medications and sudden onset of skin symptoms SJS/TEN was initially felt more likely.

This is a very rare (incidence 0.4 per million per year⁽¹⁾) and potentially life threatening severe adverse drug reaction characterised by epidermolysis: where the epidermis shears off with lateral pressure (the Nikolsky sign). If the patient survives they are frequently left with scarring, which can be particularly severe in the cornea and may cause strictures of mucosal surfaces. However, it does not display a chronic form with longstanding erosions persisting. Therefore in this case, where the oral erosions and generalised dermatitis continued to be evident (albeit less severe) over a year later the initial diagnosis of TEN had to be challenged.

PARANEOPLASTIC PEMPHIGUS MASQUERADING AS POSSIBLE TOXIC EPIDERMAL NECROLYSIS: A DIAGNOSIS THAT MAY GRADUALLY EMERGE & MUST NOT BE MISSED

S Paget, R Groves, J Setterfield

Category	Diagnoses	Features
Immunobullous (an autoimmune group of disorders characterised by autoantibody deposition to specific target antigens in skin and or mucosa)	Bullous Pemphigoid	Tense bullae rarely mucosal involvement
	Pemphigus foliaceus	Superficial erosions on face and upper trunk
	Pemphigus Vulgaris	Shallow painful erosions and occasional superficial bullae with frequent mucosal involvement
	Paraneoplastic pemphigus	Polymorphic lesions– see table 2
	Mucous membrane pemphigoid	Predominantly mucosal blisters and erosions, may cause scarring
	Linear IgA	Classically round or oval blisters arranged in ring (annular) patterns. May arise around old blisters. Affects skin and mucous membranes
	Dermatitis herpetiformis	Intensely itchy vesicles classically at extensor sites. Association with anti-tissue transglutaminase antibodies.
Infectious	Staphylococcal scalded skin syndrome	Superficial blisters and erosions with thin sheets of desquamation and occasional pustules. Usually presents in infancy
	Herpes simplex	Momomorphic vesicles in small clusters often recurrent
	Varicella: chicken pox	Widespread itchy vesicles and crusted papules
	Varicella: shingles	Dermatomal distribution of painful vesicles
Drug reactions	Bullous drug reactions: widespread or fixed SJS/TEN	Superficial bullae Nikolsky positive bullae with epidermolysis. Mucosa usually involved.
	Trauma	Burns
Vascular	Venous stasis Oedema	Dependant oedema and superficial tense bullae.

Table 1: Differential diagnoses for a blistering rash.



Figure 2a) Pemphigus vulgaris. Shallow painful erosions on the buccal mucosa.



Figure 2b) Mucous membrane pemphigoid: typically affects the gingivae with prominent erythema and ulceration.

Paraneoplastic pemphigus is a very rare immunobullous condition driven by an underlying malignancy – most commonly of haematological origin: particularly Hodgkin's lymphoma, CLL, Castleman's and also thymoma(2). Clinically signs can be polymorphic (see table 2), as in this case, making diagnosis difficult. Further complicating the diagnosis of PNP is the variable immunoprofile of causative antibodies seen in the serum of affected patient. Indirect IMF (on monkey oesophagus and rat bladder) combined with ELISA may identify IgG antibodies targeting different intercellular and basement membrane proteins.



Figure 3) Paraneoplastic pemphigus: Painful oral ulceration affecting multiple sites including the tongue and buccal mucosa.

PARANEOPLASTIC PEMPHIGUS MASQUERADING AS POSSIBLE TOXIC EPIDERMAL NECROLYSIS: A DIAGNOSIS THAT MAY GRADUALLY EMERGE & MUST NOT BE MISSED

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Even more confusingly they may be completely negative in PNP and symptoms and signs may precede both antibody detection and underlying malignancy by months(3). Nevertheless, it is important to investigate with skin biopsies sent for histology and direct IMF, a serum sample for indirect IMF using rat bladder as a substrate and preferably ELISA of autoantibodies. The rationale for using rat bladder is that it does not contain the usual pemphigus antigens Dsg 1 or Dsg 3 but does contain periplakin, desmoplakin and envoplakin all known targets in PNP. In addition careful clinical, haematological and imaging assessment for underlying malignancy must be undertaken if not already identified.

Symptoms	Painful eyes and mouth - later dysphagia May have painful genital erosions Itchy or painful skin
Signs:	
- Oral	Limited cheilitis to start or ulcerative stomatitis – later persistent painful haemorrhagic erosions with panstomatitis and pharyngeal involvement leading to dysphagia
- Eyes	Scarring conjunctivitis
- Skin	Polymorphic: white topped papules: lichen planus-like, dermatitis or papular: graft versus host-like, bullous pemphigoid-like, targetoid lesions: Erythema multiforme-like, Shallow erosions: pemphigus vulgaris-like
- Genitals	Painful Erosions
- Respiratory	Alveolitis, pulmonary fibrosis, bronchiolitis obliterans, nasal cavity involvement
Histopathology	Suprabasal loss of epidermal adhesion; interface dermatitis
Direct IMF	Anti-epithelial cell surface IgG and C3 deposit at DEJ
Indirect IMF	Bind to Monkey oesophagus, Rat/Monkey bladder
ELISA	Desmoglein 3, Desmoglein 1, periplakin/Envoplakin, desmocollin, BP230

Table 2

PNP is often resistant to conventional immunosuppression, but high dose prednisolone is still usually first line, as was the case with this patient. Anti-B cell agents such as MMF or rituximab are also effective – as they reduce the production of the autoantibodies. Involvement of haematology or oncology teams is essential as treatment of the underlying malignancy is vital in reducing PNP activity.

However, the decision to use potent immunosuppression or chemotherapy agents is complicated by the increased risk of sepsis from impaired skin barrier function. Therefore the relative benefit of treating the underlying malignancy, thereby treating the driver of the disease process, must be weighed up against concurrent risk of impairing host response to infection. This is a large contributor to the high mortality rate associated with PNP approaching 90%(4). Discussion with the patient and their family are important and in the final week this patient and his family agreed that best supportive end of life care was preferable.

In this case, by the principle of Occam's razor, it is likely that the diagnosis of PNP explains all of his skin and mucosal signs and that those features which were initially consistent with SJS/TEN were part of the polymorphic spectrum of cutaneous findings of PNP all along. It highlights the challenging nature of diagnosing PNP and underlines the importance of continuing to reconsider the diagnosis if the patient is not responding to treatment.



Figure 4: Paraneoplastic pemphigus showing a generalised dermatitis rather than a blistering eruption.

Teach yourself

1. Which malignancy is most commonly associated with Paraneoplastic pemphigus (PNP)?

- Hodgkin's lymphoma
- Melanoma
- Colorectal cancer
- Prostate cancer
- Pancreatic cancer

PARANEOPLASTIC PEMPHIGUS MASQUERADING AS POSSIBLE TOXIC EPIDERMAL NECROLYSIS: A DIAGNOSIS THAT MAY GRADUALLY EMERGE & MUST NOT BE MISSED

S Paget, R Groves, J Setterfield

2. Which sign applies a shearing force to the edge of a blister extending the separation and blister indicating epidermolysis?

- a. Hutchinson's sign
- b. Kussmaul's sign
- c. Nikolsky's sign
- d. Darrier's sign
- e. Litten's sign

3. Which B-cell maker does rituximab attach to?

- a. CD3
- b. CD20
- c. CD19
- d. CD4
- e. CD22

4. In PNP anti-plakin antibodies are occasionally found in the serum. Which substrate expresses the highest concentration of plakins and is therefore used to bind anti-plakin antibodies via Indirect Immunofluorescence?

- a. Guinea pig oesophagus
- b. Mouse stomach
- c. Rat bladder
- d. Monkey oesophagus
- e. Mouse skin

5. Which clinical sign and symptom are most suggestive of PNP?

- a. Chronic panstomatitis and dysphagia
- b. Itchy monomorphic vesicles
- c. Chronic extensor plaques
- d. Pruritic flexural pustules
- e. Isolated tender nodule on fingertip

Answers

1. Hodgkin's lymphoma.

Haematological malignancies particularly Hodgkin's lymphoma, chronic lymphocytic leukaemia and Castleman's are most commonly associated with paraneoplastic pemphigus, although any malignancy may be implicated – especially thymoma.

2. Nikolsky's sign.

Hutchinson's sign is where vesicles arise on the tip of the nose or at the side of the nose preceding the development of ophthalmic herpes zoster. Kussmaul's sign is where there is increased jugular distension on inspiration suggestive of increased right sided heart pressure. Darrier's sign is where stroking of the skin induces urticarial – as seen in systemic mastocytosis or urticarial pigmentosa. Litten's sign is presence of cotton wool exudate in the retina suggestive of infective endocarditis.

3. CD₂₀.

Rituximab is an anti-B cell agent which targets CD20 – an ubiquitous B cell marker. It is used as part of chemotherapy regimens in a number of B-cell lymphomas and is also used as an immunosuppressant in certain autoimmune conditions where auto-antibodies drive the disease process, such as paraneoplastic pemphigus.

4. Rat bladder.

Rat bladder expresses high concentrations of plakin proteins. Therefore binding of a patient's serum to this substrate, as shown by indirect immunofluorescence, suggests presence of anti-plakin antibodies such as anti-periplakin or anti-envoplakin antibodies. Although not always found these auto-antibodies are specific for paraneoplastic pemphigus.

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5. Chronic panstomatitis and dysphagia is most characteristic of paraneoplastic pemphigus from this list.

Other clinical features of paraneoplastic pemphigus include: initially limited cheilitis and haemorrhagic erosions of oral mucosa; polymorphic cutaneous features: white topped papules (lichen planus-like), dermatitis or papular exanthema (graft versus host-like), tense bullae (bullous pemphigoid-like), targetoid lesions (Erythema multiforme-like), Shallow erosions (pemphigus vulgaris-like); scarring conjunctivitis, genital erosions, nasal cavity erosions or respiratory involvement with alveolitis, pulmonary fibrosis or bronchiolitis obliterans.

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PATIENT MANAGEMENT OF SYPHILIS

B Meeajun, V Jolliffe

Patient Management Of Syphilis Patient Management

Abstract

A previously healthy 41 year old man with a background of well controlled HIV, presents with a worsening generalised skin eruption over a period of 4 weeks, associated with fever, weight loss, and lethargy.

We discuss the approach to reaching the diagnosis and consider the wide differential diagnosis. This case highlights the importance in taking a comprehensive history in-conjunction with good clinical acumen in obtaining a diagnosis in the dermatology clinic.

Case history

A 41 year old male publican presented to the emergency dermatology clinic with a spreading rash in the last 4 weeks. The rash was not pruritic but slightly sore to palpate. He reported weight loss, pyrexia, anorexia and lethargy. He was known to have well controlled HIV.

He had not been abroad in the last 1 year or sustained any insect bites. He was taking atripla for his HIV. He denied any suggestion of recreational drug use. He did not admit to any unprotected sexual activity with new or casual partners in the last 6 months.

The rash consisted of scaly erythematous nodules and papular lesions, which started on his left hip and subsequently spread to involve his limbs, back, chest, abdomen and dorsum of feet (Figure 1-5). Some of these lesions had ulcerated and exuded pus.

There were some lesions in his natal cleft. He had a small papule on his nose. The rash was not in a photosensitive distribution. The palms of his hands and soles of feet were also affected with this rash. There were no oral lesions seen. His nails and scalp were also not involved.



Figure 1: Back.

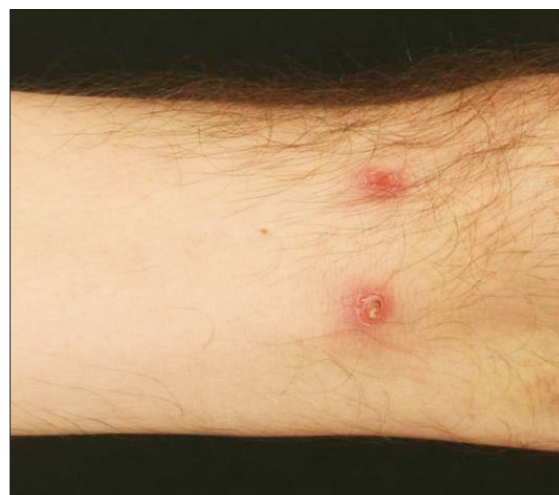


Figure 2: Arm.

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Figure 3: Close up of arm.



Figure 4: Abdomen.

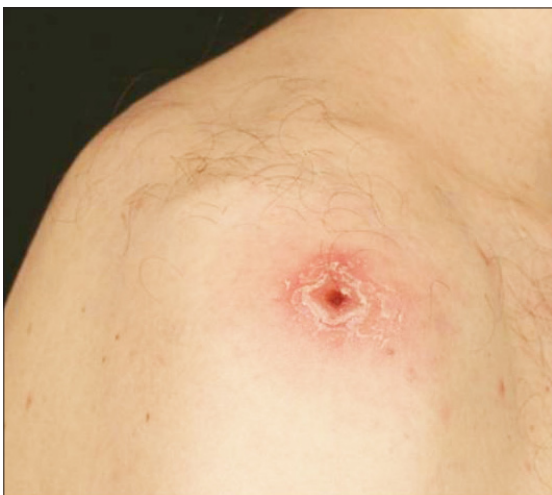


Figure 5: Shoulder.

Differential diagnosis

The symptoms of a relatively sudden onset of widespread and generalised rash, associated with fever, lethargy and weight loss, produces a wide differential. The investigations considered must help to refute or confirm the diagnosis.

We considered from a dermatological aspect of using a surgical sieve to collate a differential diagnosis. In this case, the most likely cause would be due to an infection, neoplastic or inflammation. This can be further subdivided into more specific conditions, as outline in table 1.

Causes	Examples	Investigations
Infections	Bacterial eg ecthyma	FBC, U&E, LFT, CRP, Skin swab for bacteria & virus, Urine dipstick for blood & protein
	Tuberculosis (systemic disease)	TB Elispot Culture for atypical mycobacteria
	Sarcoidosis	Serum ACE and calcium, CXR
	Syphilis	Syphilis serology, Hepatitis B and C, Skin biopsy
Neoplastic	Haematological malignancy	Blood film, Immunoglobulins, Staging CT scan
	Lymphoma	FNA of lymph node Staging CT scan
Inflammatory	Systemic Lupus Erythematosus	ANA, dsDNA, ENA, skin biopsy
	Pityriasis rosea	Skin biopsy
	Guttate psoriasis	ASOT
	Lymphomatoid papulosis	Skin biopsy

Table 1

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Results

He had a normal full blood count. The CRP was moderately elevated at 56 which reflected an infection. His hepatitis serology was negative. His urine dipstick was negative. There was no evidence of sarcoidosis or tuberculosis. He was however identified to be positive for syphilis.

The histology of his skin biopsy was that of a lymphohistiocytic infiltrate characteristically rich in plasma cells. It was also positive for a silver stain, Warthin Starry, which aids identification of the spiral shaped *Treponema pallidum* bacterium. Immunohistochemistry stain is another test which may also be used (1) to identify this infection.

Management

The patient returned back to clinic shortly afterwards to discuss his results. He was shocked to learn that he was diagnosed with syphilis. He eventually admitted to having unprotected sexual activity with three men in the last 6 months. Counselling was arranged. He was also advised to inform his sexual partners of his diagnosis so that they could be tested and any necessary treatment would be arranged for them.

He was seen urgently by the infectious diseases team who promptly treated him with IM benzathine penicillin G 2.4 million units.

Discussion

Syphilis is a bacterial infection caused by the spirochete, *Treponema pallidum*. It is transmitted by sexual activity and exchange of bodily fluids. The incidence of this disease is on the rise. It has increased by 46% in men who have sex with men (MSM) (2). It is also being seen more commonly in adults above the age of 50 years old.

It is easily treated with antibiotics if diagnosed early. In the primary phase, a chancre, a painless ulcer, usually present in the mouth or genital area may go unnoticed, which is likely to have been the case with this patient. It heals spontaneously and the disease remains in the latent phase. Latent periods are when the patient remains infected but does not show any signs or symptoms. Thus the patient may not know if they have been infected with syphilis.

When the syphilis is re-activated it may develop into secondary syphilis, as in this case. This condition can present in many ways, but most commonly as an erythematous scaly rash consisting of shallow papules or plaques. The palms of hands and soles of feet are characteristically involved. It is unusual to see nodular areas, some of which are papulo-necrotic. This type of syphilis is known as 'Lues Maligna'. We are seeing an increasing number of this type of presentation especially in MSM.

If syphilis is not treated in the primary and secondary stages, then tertiary syphilis may ensue. This may affect the brain, spinal cord, eyes, heart, bone as well as skin. It is less frequently seen in the UK.

Syphilis has been coined a 'great mimicker' as it has such a varied presentation. It should always be considered in patients who have a rash which appears like pityriasis rosea (3), particularly if they do not give the typical history of initially developing a herald patch, followed by the skin eruption in a 'christmas tree' distribution on the back, which is classically seen in this condition.

Another inflammatory condition which may masquerade as syphilis is guttate psoriasis (4). This usually presents as an eruption of scaly erythematous plaques with the appearance of 'rain drops'. These patients would expectedly have had a streptococcal throat infection prior to the onset of the rash. If however that patient had too few of these lesions and were middle-aged, that would be an unusual presentation, and one must consider syphilis again.

Fever, weight loss, lethargy and a new rash are concerning symptoms and underlying malignancy and granulomatous disease must be excluded. The investigations he underwent confirmed a normal FBE, U&E, LFTs, calcium, serum ACE, normal CD4 count and negative TB-Elispot. In the context of a positive diagnosis of syphilis, it would make malignancy unlikely.

In the majority of patients the rash caused by syphilis, can have similar appearance in both those infected with HIV infection and those who are not. However in a small number of cases it can present in a more atypical or aggressive manner (5), and this may be the case with our patient.

The treatment for syphilis is the same regardless of HIV status. Patients are highly infectious when they have either primary or secondary syphilis and it is paramount that they are aware of this to reduce the spread and also to inform their sexual partners to be tested and treated accordingly. Syphilis can be transmitted by pregnant women to their unborn child if they are not treated.

Our patient is responding well to the treatment. His rash is stable and is slowly improving. He has been in contact with all the individuals with whom he has had sexual activity and they have all been tested and treated accordingly. He is being followed up by both the dermatology and infectious disease teams.

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This case highlights the importance of taking an accurate and thorough history, as well as having good clinical acumen. This is especially key in situations where patients are initially not as forthcoming with information as they could be.

The patient may not always tell you what you need to know, but what they would like you to know. Another significant fact to mention is that it is always worth routinely checking HIV status in patients, as there is a high prevalence of concurrent HIV and syphilis infection (6). Ultimately early diagnosis of these two diseases is crucial, as it may improve prognosis, and decreases the risk of further complications and spread arising.

5 MCQs (best of 5)

1. Which of the following investigations would not be used to diagnose syphilis?

- a) Venereal disease research laboratory test (VDRL)
- b) Rapid plasma regain (RPR)
- c) Fluorescent treponemal antibody absorbed test (FTA-ABS)
- d) Dark field microscopy
- e) P24 antigen

2. Which of the following drugs would not be used to treat syphilis?

- a) Benzathine penicillin G
- b) Doxycycline
- c) Ceftriaxone
- d) Tetracycline
- e) Methotrexate

3. Which of the following of not symptoms of tertiary syphilis?

- a) Gummas
- b) Chancre
- c) Neurosyphilis
- d) Argyl Robertson pupil
- e) Tabes dorsalis

4. Which of the following is not a manner that syphilis can be spread /transmitted?

- a) Blood transfusion
- b) Oral sex
- c) Sharing needles
- d) Coughing
- e) Via the placenta in infected women who are pregnant

5. Which of the following are not seen in congenital syphilis?

- a) Hutchinson's teeth
- b) Bowed sabr shins
- c) Interstitial keratitis
- d) Rash
- e) Hepatomegaly

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Answers

1. (e) p24 is used to test HIV.

All the other tests can be used to diagnose syphilis. Dark field microscopy is used to diagnose primary syphilis. RPR and VDRL can be used for all stages, but best for secondary syphilis. FTA is also good for secondary and tertiary syphilis diagnosis.

2. (e) Methotrexate is not a treatment for syphilis.

IM benzathine penicillin G is the treatment of choice. However the other antibiotics mentioned are also efficacious. Some of these are suitable if they are allergic to penicillin.

3. (b) Chancres (oral / genital) are typically seen in primary syphilis, and sometimes in secondary syphilis.

4. (d) Coughing is not a recognised form to transmit syphilis.

In the UK, all blood transfusions are screened for syphilis. However this does not happen in all countries, and is a possible way to contract this disease.

5. (e) Hepatomegaly is not a feature of congenital syphilis.

The rash which can be seen may consist of blisters and later becoming scaly and resembling the rash seen in secondary syphilis.

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TWO CASES OF RASH, MYOSITIS & SYSTEMIC SYMPTOMS

AL Manley, JM Fawcett, JE Sansom



Abstract

This article describes two different cases of patients who presented to dermatology outpatient clinic with skin eruptions. On direct questioning, both patients describe systemic symptoms. This article highlights the cutaneous features of dermatomyositis and some of the recognised associated systemic complications. Investigations and treatment are determined by the underlying aetiology which can be associated with autoimmunity or malignancy.

Case 1

A 47-year-old man presented with a five-month history of eyelid rash and swelling. This was thought to be related to new machine oil in his workplace, a tooling company. The rash had progressed to the neck, upper chest, arms, knees, ankles and buttocks. He also felt stiff and fatigued with leg weakness, the latter which he particularly noticed when dog-walking. Other symptoms included breathlessness on exertion, weight loss and difficulty swallowing.

He had initially noticed food getting “stuck” and at presentation reported a cough on swallowing liquids. Due to his symptoms he had been unable to work for the previous six weeks. His past medical history included hypertension and medication comprised perindopril, atenolol and simvastatin. He was an ex-smoker and his alcohol intake was minimal. Examination and investigations:

On examination, there was a florid purple erythema of his eyelids with associated swelling (Fig. 1). The rash extended to his face, neck and chest. His hands showed periungual erythema and ragged cuticles with nail-fold telangiectasia (Fig. 2). On neurological examination of the limbs he was noted to have proximal muscle weakness in his legs with power 4/5 on the

Two Cases of Rash, Myositis & Systemic Symptoms Patient Management

Medical Research Council (MRC) scale.

Full blood count, liver function tests, urea and electrolytes were normal but creatine kinase (CK) was significantly raised at 1110 IU/L (normal range: 24-195 IU/L). Autoimmune profile, complement levels and extractable nuclear antigen screen were also all normal. Serum tumour markers and a computerised tomography (CT) of chest, abdomen and pelvis were negative for malignancy. A skin biopsy showed changes consistent with dermatomyositis.



Figure 1



Figure 2

TWO CASES OF RASH, MYOSITIS & SYSTEMIC SYMPTOMS

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Discussion

A history of rash and systemic symptoms, as in this case, should raise suspicion of a connective tissue disease and therefore precipitate a thorough history and examination. This is a classical presentation of dermatomyositis, and typical features include hallmark skin lesions, myopathy, interstitial lung disease and gastro-intestinal disease. The rash is described as violaceous, often oedematous, and mostly occurs in sun-exposed areas such as the face, shoulders and upper chest.

Eyelid involvement as in Figure 1 is described as "heliotrope," and the purple papules on knuckles are known as "Gottron's papules". Ragged cuticles and prominent nail-fold capillaries as seen in Figure 2 are also typical. Myositis affects the proximal muscles causing weakness and problems with mobility and activities of daily living. Sometimes affected muscles ache or can be tender to touch. The myopathy usually develops after the rash, but it can arise concomitantly, or even prior to the rash, and 6% of patients with dermatomyositis will have no rash at all (1). Dysphagia is also a frequent and serious complication.

Blood tests should include those to detect raised circulating muscle enzymes: CK, aspartate aminotransferase (AST) and lactic dehydrogenase (LDH). Autoimmune profile including antinuclear antibody (ANA) can be requested along with myositis-specific antibodies such as Anti-Jo-1, Anti-SRP and Anti-Mi-2. Anti-Jo-1 antibodies are the most common myositis-specific antibodies, but new antibody recognition means that this is a rapidly evolving and expanding field.

Biopsy of an affected muscle can be considered as well as skin biopsy. Skin biopsy is rarely diagnostic, but can be helpful in supporting the diagnosis. Importantly, immunofluorescence to complement and immunoglobulins is negative in dermatomyositis, unlike in systemic lupus erythematosus, which may be one of the main differentials. Other investigations to consider include: Nerve conduction studies (NCS), electromyography (EMG) and magnetic resonance imaging (MRI) of the muscle, or ultrasound.

Underlying cancer is a significant risk and should be considered in all patients with dermatomyositis. Initial investigations should be led by clinical findings, but a CT scan of the chest, abdomen and pelvis would be a reasonable initial screen.

Patient management

Initial management of this patient included oral steroids (prednisolone 80mg daily) and admission for ongoing care. The myopathy and dysphagia continued to deteriorate and he required nasogastric tube feeding for the first month of treatment. No malignancy was detected on CT investigation and there was no deterioration in his lung function.

Further treatment included azathioprine, hydroxychloroquine and two courses of intravenous immunoglobulin. He required physiotherapy and regular dietician input. He was discharged after a five-week admission and made a gradual improvement over the following six-to-twelve months. Over a nine-year period, he has had some relapse, but he is now in remission with no evidence of malignancy.

Discussion

This patient had a diagnosis of dermatomyositis made on the grounds of a typical skin rash, symmetrical proximal muscle weakness and elevated CK. No autoantibodies were positive, but it is worth noting that only 60-80% of patients with dermatomyositis have a positive autoantibody on screening (1, 2). Fortunately this patient had a good response to immunosuppression. The most serious and disabling feature of disease for this patient was the dysphagia, which improved with treatment. Dysphagia can be a poor prognostic indicator, and is one of the commonest causes of fatal infections in these patients.

Case 2

A 56-year-old woman presented with malaise, weight loss and rash. She described a six-week history of a rash, which started on her arms extending to her forearms and upper trunk. She reported myalgia and stiffness affecting her arms and thighs and had noticed particular difficulty climbing stairs. She also reported food and drink 'sticking' in her throat and an intentional five stone weight loss over the preceding twelve months.

Her GP had prescribed a one-week course of oral prednisolone which improved her symptoms significantly, however they had recurred with cessation. This patient had multiple co-morbidities including type 2 diabetes, hypothyroidism and recurrent venous thromboembolism. Drug history included warfarin, propranolol, citalopram, amlodipine, folic acid and levothyroxine.

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On examination, she had a symmetrical confluent violaceous papular eruption affecting her chest, trunk and the extensor surfaces of her arms and legs with sparing of light-protected sites (figs. 3&4). On the hands she had proximal nail fold erythema with dilated vascular loops and 'ragged' cuticles. She had a symmetrical proximal weakness of the arms and legs with power 4/5 on the MRC scale. Systemic examination revealed a large, firm tethered lump in her right breast.

Blood tests showed a raised CK of 211 IU/L and normal immunoglobulin and complement levels. Anti-nuclear antibodies (ANA) were positive but low titre, and specific autoantibody screen was negative. A skin biopsy showed interface dermatitis, supporting a diagnosis of dermatomyositis. As initial management, this patient was started on prednisolone 100mg daily. An urgent breast biopsy was arranged and she was diagnosed with Grade III invasive ductal carcinoma of breast. She underwent neo-adjuvant chemotherapy followed by mastectomy and axillary node clearance followed by oral anastrozole.

The dermatomyositis cleared eight months following initial breast cancer treatment, but there was a relapse in symptoms requiring further treatment with prednisolone, methotrexate and hydroxychloroquine. Despite the relapse of dermatomyositis, there has been no recurrence of breast cancer to date.



Figure 3



Figure 4

Discussion

The second case highlights the importance of systemic examination in patients presenting with dermatomyositis. There is a strong association between dermatomyositis and malignancy. Approximately 15% of adults presenting with dermatomyositis will either have a malignancy at presentation or will develop one in the future (1). Rapid clinical detection of malignancy in this case allowed for appropriate treatment and management pathway to be instituted without delay.

The field of specific autoantibodies associated with dermatomyositis has been rapidly expanding in recent years, even in cancer-associated dermatomyositis, which was previously considered an antibody-negative subset of the disease. There are no diagnostic criteria for dermatomyositis, but diagnosis is usually made with evidence of the typical skin rash of dermatomyositis in addition to either or both of the following: Symmetrical proximal muscle weakness and elevation of skeletal muscle enzymes.

With the expanding field of autoantibody detection, there is a push to move away from the clinical and pathological criteria of diagnosis to adopting a clinicoserological classification of diagnosis (2), this is for the purposes of prognosis and therapeutic guidance. Both cases presented here were seronegative at the time of diagnosis, but testing has not been repeated since the advent of newer serological markers.

Treatment

Despite the absence of placebo-controlled trials, corticosteroids provide the mainstay of initial treatment for dermatomyositis. Most patients with dermatomyositis are likely to have a good response to corticosteroids, but a high proportion will go on to require further immunosuppression. First-line steroid-sparing agents are methotrexate or azathioprine.

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AL Manley, JM Fawcett, JE Sansom

Key learning points

1. Dermatomyositis is a multi-system inflammatory disease with serious pulmonary and gastro-intestinal complications.
2. Underlying malignancy is an important consideration and full systemic examination is essential as well as imaging. Dermatomyositis can pre-date malignancy by several years.
3. Dysphagia may present insidiously, but must be taken seriously and Speech and Language therapy (SALT) review considered, avoiding the potentially life-threatening complication of aspiration pneumonia.
4. Expert opinion should be sought on appropriate screening for serological markers in this expanding field.

Test Yourself

MCQ 1: A 44-year-old man presents with a violaceous papular rash affecting his eyelids and arms over the last few weeks and more recently, proximal muscle weakness of his arms and legs. He also describes significant dysphagia and a cough after drinking. What is the most important next step in this patient's management?

- a) Check autoantibody screen
- b) Make the patient "nil by mouth" and request SALT assessment
- c) Request an EMG
- d) Take a muscle biopsy
- e) Take a skin biopsy

MCQ 2: Which of the following is least helpful in making the diagnosis of dermatomyositis?

- a) Heliotrope rash and Gottron's papules
- b) Raised CK
- c) Skin biopsy
- d) Symmetrical proximal myopathy
- e) Autoantibody screen

MCQ 3: A steroid sparing treatment is used in the management of Case 1 with autoimmune dermatomyositis. Which investigation must be checked prior to the introduction of azathioprine?

- a) Chest X-ray
- b) Creatinine clearance
- c) Liver ultrasound scan
- d) Pro-collagen III peptide (PIIINP)
- e) Thiopurine methyl transferase (TPMT) enzyme activity

MCQ 4: In a patient presenting with dermatomyositis with significant muscle involvement, which of the following treatments will have the most immediate beneficial effect?

- a) Azathioprine
- b) Cyclophosphamide
- c) Hydroxychloroquine
- d) Methotrexate
- e) Prednisolone

MCQ 5: Autoantibodies are frequently positive in dermatomyositis. Which of the following is most strongly associated with underlying malignancy?

- a) Anti-Jo-1 (antihistidyl transfer RNA [t-RNA] synthetase) antibodies
- b) Anti-La/SSB antibodies
- c) Anti-Mi-2 antibodies
- d) Anti-Ro/SSA antibodies
- e) Anti-transcription intermediary factor (TIF)-1 β

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MCQ Answers & Teaching Notes

MCQ 1: answer: b)

Dysphagia can present insidiously in patients with dermatomyositis. This important symptom must be noticed and acted upon with urgency to avoid serious and life-threatening complication of aspiration pneumonia. Diagnostic tests will help to make the diagnosis but patient safety should take precedence over diagnostic tests.

MCQ 2: answer: c)

The diagnosis of dermatomyositis can usually be made without tissue biopsy in patients a presentation, which is characteristic of this disorder, such as patients with a typical skin rash, symmetrical proximal muscle weakness and elevation of muscle enzymes. In patients with a non-specific or atypical presentation, then the next most useful investigations would include myositis-specific antibodies, muscle biopsy and EMG. Skin biopsy can be helpful for supporting a diagnosis, or excluding differentials, but features are rarely diagnostic.

MCQ 3: answer: e)

Azathioprine is a thiopurine. TPMT enzyme activity determines the rate of breakdown of toxic metabolites of azathioprine. Approximately 10% of UK individuals have a polymorphism that means they have low enzyme activity level and 1 in 300 have absent levels. Those with absent levels may develop profound bone marrow suppression.

MCQ 4: answer: e)

Oral corticosteroids will have the most immediate effect in this situation. The other systemic medications will have a much slower onset of action.

MCQ 5: answer: e)

The autoantibody directed against a 155-kd protein known as anti-p155/140 or anti-transcription intermediary factor (TIF)-1D, has been associated with malignancy in dermatomyositis but can be found in patients without malignancy. Anti-Jo-1 antibodies are associated with pulmonary involvement and 'mechanic's hands'; anti-Mi-2 antibodies are associated with classic onset dermatomyositis but are only found in 25% patients. Anti-Ro and anti-La antibodies may be positive in cutaneous lupus.

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WHAT IS A PSYCHODERMATOLOGY CLINIC: CASE BASED DISCUSSIONS ON THE MANAGEMENT OF TWO PATIENTS WITH PSYCHOCUTANEOUS CONDITIONS

S Shinhmar, A Bewley

What Is A Psychodermatology Clinic: Case Based Discussions On The Management Of Two Patients With Psychocutaneous Conditions Teaching & Training

Abstract

Psychodermatology is an emerging subspecialty of dermatology integrating psychiatry, psychology and dermatology. Psychodermatology can be considered two sides of the same coin. One side is primarily psychiatric disease presenting to a dermatologist (for example delusional infestation, dermatitis artefacta, body dysmorphic disorder). On the other side is long standing skin disease (for example eczema and psoriasis) in which there are large psychosocial co-morbidities such as anxiety and depression. (1,2) This article aims to enable foundation trainees to be aware of the dual nature of psychodermatology and introduce the multidisciplinary approach adopted in our clinics through two case based discussions. (3)

Case 1

A 68 year old Caucasian lady was referred by her General Practitioner (GP) to our psychodermatology clinic as she believed she had infestations on her body. These infestations were described as thread like fibres growing out of her skin and spread to her home. Her elder sister with whom she lived and who had accompanied her to clinic also believed that she had fibres in her skin. Both sisters experienced widespread crawling sensations and itchiness.

Despite extensive bathing and even hiring pest control professionals to treat their home, all methods to clear away these fibres were unsuccessful. Further questioning revealed that it was the elder sister who was first to see these fibres. Both sisters brought samples of these fibres to our clinic and insisted these should be tested in the laboratory. Physical examination of both women revealed extensive excoriations. Blood tests (full blood count, urea and electrolytes, liver function tests, thyroid function tests) and a urinary recreational drug screen to rule out possible organic causes of their symptoms, were normal and no causative organisms were found on analysis of fibre samples.

We made a diagnosis of delusional infestations (DI). In this case the index patient (the person who first developed the delusion and held the belief of infestations more strongly) was the eldest sibling. However it was the youngest sister who shared the delusion and who presented to her GP initially as she had the motivation to seek medical attention.



We encouraged the elder sister to also attend our clinic and subsequently prescribed her aripiprazole 5mg to minimise her crawling sensations. Topical antiseptic emollients were also prescribed to improve skin barrier function. After six months of treatment the eldest sister's DI disappeared, her younger sister also simultaneously improved with no recurrence to date.



Figure 1: Patients with DI commonly present to psychdermatology clinic with extensive excoriations secondary to pruritus stimulated by associated paresthesia such as crawling and tingling sensations.

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Figure 2: 'Specimen sign'-sample of inanimate specimen bought in by DI patient of what was thought be an infestation. Laboratory analysis did not identify any causative organism.

Case 2

A 40 year old woman was referred to our psychodermatology clinic by her local dermatologist with a one year history of pulling out her hair. She had a background of severe atopic eczema treated with topical mometasone ointment and oral ciclosporin. Examination of her scalp revealed patches of hair loss (with individual hairs at different lengths in asymmetrical patches). She also had lichenification on her limbs.

Underlying organic causes of alopecia were ruled out. Our patient was diagnosed with trichotillomania (TTM). On closer questioning, we believed that her atopic eczema had caused her to feel very anxious about her appearance and that this anxiety had been the main precipitant for her TTM. She was prescribed 20 mg fluoxetine to reduce her anxiety and referred to the clinical psychologist for habit reversal therapy to reduce her hair pulling. Her symptoms improved with 12 months of combination therapy with no further reports of reoccurrence.



Figure 3: Patches of non-scarring alopecia observed in a patient with TTM.

Discussion: How does a psychodermatology clinic work?

Management of patients with psychocutaneous disease involves a multidisciplinary approach. Close liaison between a dermatologist, psychiatrist, clinical psychologist, paediatrician, geriatrician, nurses, and general practitioners is crucial. Referral to a psychodermatology clinic (where available), provides a pathway in which patients are not stigmatised with psychiatric illness and patients are more likely to be adherent to their agreed treatments (1,2). But the exact configuration of a psychodermatology clinic is usually determined by local funding and availability of local expertise.

The psychosocial impact associated with chronic dermatological conditions is becoming increasingly recognised. Several well validated tools have been developed to measure this. These include:

- 1) Non-speciality specific tools e.g. Hospital Anxiety and Depression Scale (HADS)
- 2) Speciality specific tools e.g. Dermatology Life Quality Index (DLQI)
- 3) Disease specific tools e.g. National Institute of Mental Health Trichotillomania Severity Scale (NIMH-TSS)
- 4) Tools for use in psychodermatology clinics e.g. Derriford appearance scale (DAS)

A key question to ask when clerking patients is 'what do you think is going on?'(4). This allows the treating physician to find out if the patient has insight into their condition and allows the patient an opportunity to express their concerns. Organic causes of psychodermatological disease, such as a history of recreational drug use and alcohol intake, must be explored as treatment of the organic disease then becomes the priority.

Even where psychological symptoms appear less severe, a explicit enquiry about suicidal ideation and self-harm may be necessary. (4) Also, it is often necessary to consider the impact of the disease on the patient's surrounding members of family and friends. In particular if there are concerns that dependents such as children or vulnerable adults are being put at risk, a referral to the local safeguarding team may need to be made after an initial discussion with that team. (5)

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Delusional Infestation

DI is a fixed false belief that there is an infestation of the skin despite objective medical evidence to the contrary. (6) Our first case illustrates folie a deux, whereby DI is shared between two individuals. The index patient is the patient who has the delusion and the others are individuals (usually family members) who share that delusion. As illustrated in case 1 it is necessary to identify the index patient. The index patient is treated, usually with antipsychotics, and the relative who shares the delusion usually get better spontaneously. (5)

Trichotillomania (TTM)

TTM is an obsessive/compulsive hair pulling disorder which can involve any part of the body. It is usually preceded by an increase in tension around the hair which is relieved once the hair is pulled. Precipitating triggers may include anxiety and depression. In some cases the patient may not be aware of their actions. (7) In case 2, the patient's eczema was a source of heightened anxiety which, we believe, precipitated her hair pulling. Our patient's eczema eventually improved with the combination of ciclosporin and mometasone ointment given by her local dermatologist. But her TTM was treated with fluoxetine and habit reversal therapy (a talk therapy, or cognitive behavioural therapy, which involves identifying and reversing habits such as hair pulling).

Conclusion

Management of patients with psychocutaneous disease is often complex and there is increasing evidence that the most effective way of treating such patients is in a psychodermatology clinic. Despite huge demand, provision of this service across the UK is intermittent and teaching for trainees is limited. (1,2) Psychodermatology is a relatively new and exciting subspecialty in dermatology and there are lots of opportunities to participate in novel research. Foundation trainees wishing to explore this field further are welcome to attend our clinic and witness our holistic approach first hand.

Test yourself section: Questions and answers

1) Which of these tools can be used to assess psychological impact of Dermatological disease?

- A) Psoriasis Area Severity Index (PASI)
- B) Dermatology Life Quality Index (DLQI)
- C) Hospital Anxiety and Depression Scale (HADS)
- D) Brown Assessment of Belief Scale (BABS)
- E) Eczema Area and Severity Index (EASI)

2) What proportion of DI's are due to secondary causes?

- A) 10%
- B) 30%
- C) 90%
- D) 60%
- E) 50%

3) Which of these can cause DI?

- A) Cocaine
- B) Depression
- C) Dementia
- D) Cannabis
- E) Stroke

4) Which of these cause scarring alopecia?

- A) lichen planus
- B) OCP
- C) Radiotherapy
- D) Iron deficiency
- E) Alopecia areata

5) Which of these cause non-scarring alopecia?

- A) Trichotillomania
- B) OCP
- C) Radiotherapy
- D) Systemic Lupus Erythematosus
- E) Basal cell carcinoma

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Answers

Question 1: Answer: B, C.

The DLQI is used to measure how skin disease impacts a patient's quality of life. The HADS score directly measures the level of anxiety and depression associated with disease.

Question 2: Answer: D.

60 % of DI's are due to secondary causes. (8)

Question 3: Answer: A, B, C, D, E.

The secondary causes of DI can be divided as follows:

Drugs - substance misuse (Cocaine, amphetamine, cannabis) and prescribed drugs (morphine)

Medical conditions-delirium, dementia, stroke

Psychiatric disorders-depression and schizophrenia. (8)

Question 4: Answer: A, C.

In scarring alopecia, the follicles are replaced with scar tissue causing permanent hair loss. Other causes are discoid lupus, burns, benign tumours (sebaceous naevus), malignant tumours (basal cell carcinoma) (9)

Question 5: Answers: A, B.

Non-scarring alopecia is potentially reversible causes of hair loss. Other causes include androgenic alopecia, drugs, Iron deficiency, thyroid disease, alopecia areata and telogen effluvium. (9)

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