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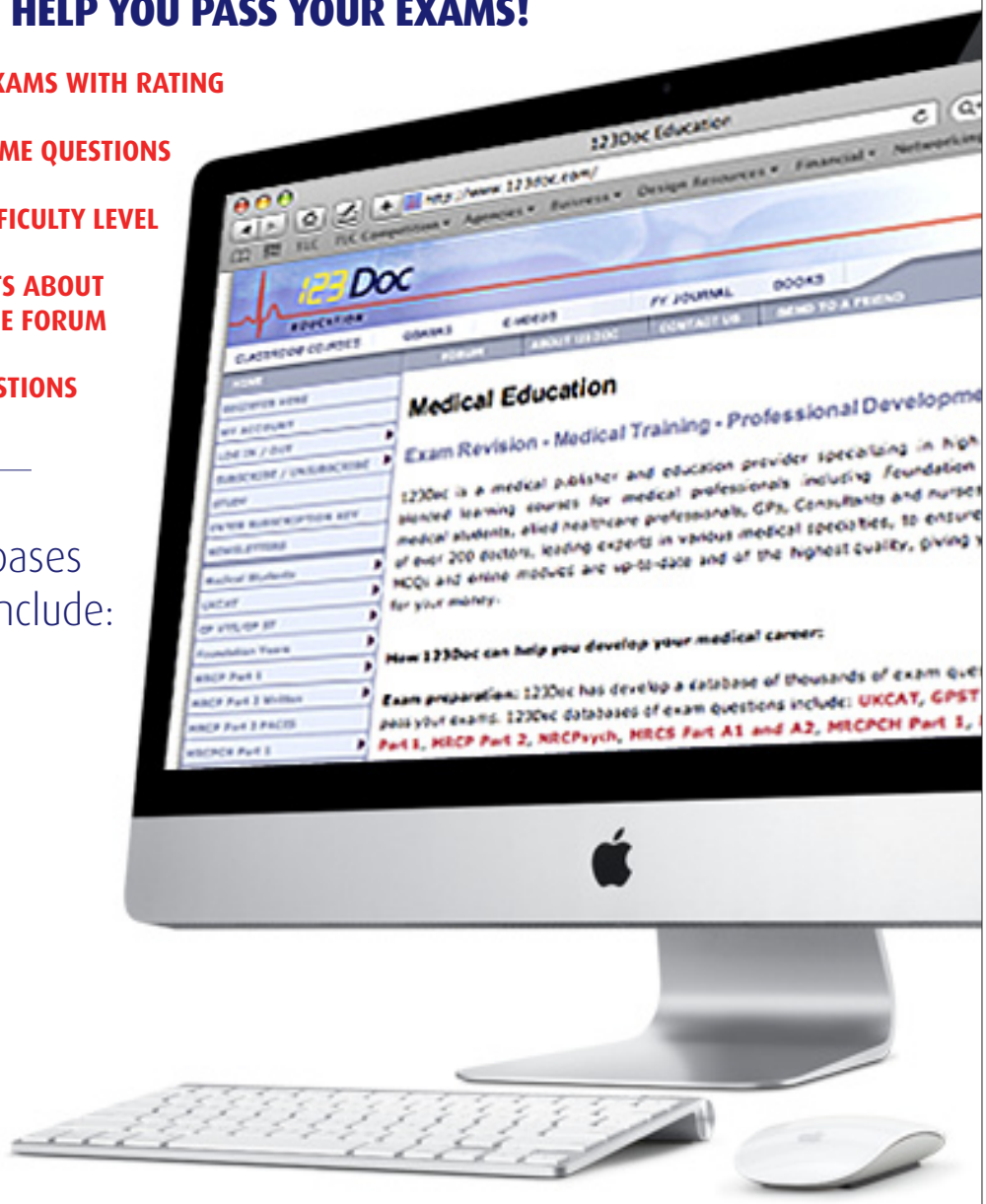

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Volume 9, Issue 7

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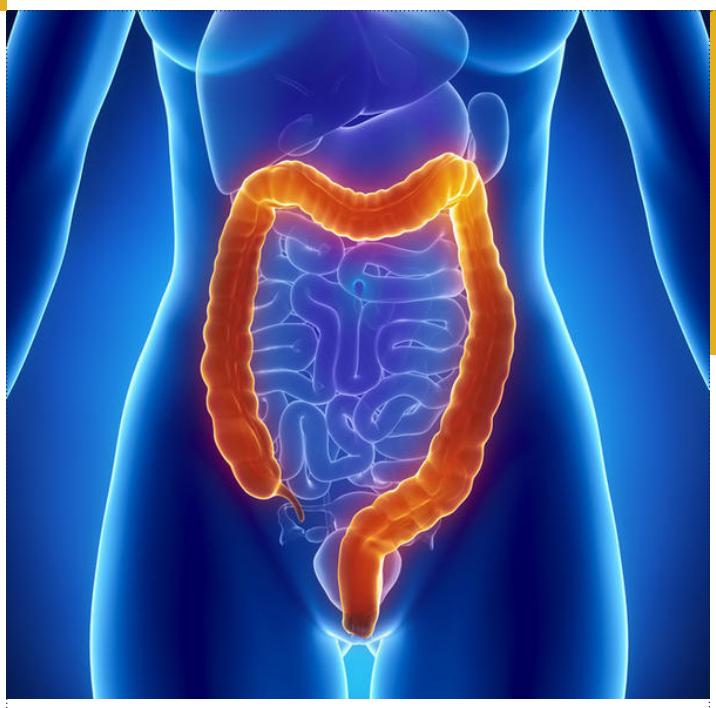
ALVARADO SCORE FOR PREDICTING ACUTE APPENDICITIS

A Choudhary, E Rawstorne, A Karim, SS Karandikar



Abstract

Acute appendicitis is a common surgical emergency and junior doctors with limited surgical experience are often first to assess patients with possible appendicitis. A diagnostic delay increases the risk of perforation, post-operative complications and longer hospitalization periods. Prompt surgical assessment and diagnosis is therefore crucial to prevent complications and reduce morbidity.

The Alvarado scoring system enables risk stratification of suspected appendicitis cases and is a useful diagnostic tool that can be implemented by the foundation year doctor. The aim of this article is to provide junior doctors with knowledge of the Alvarado scoring system including its efficacy and limitations.

Background

Acute appendicitis is the most common cause of an acute abdomen (1) and over 40,000 emergency appendicectomies are carried out annually in the UK (2). Many foundation year doctors will therefore encounter patients with suspected appendicitis; particularly during surgical on-call shifts and in the emergency department setting. However, the differential diagnosis of appendicitis can represent a diagnostic challenge to the clinician due to the spectrum of variation in presentation and physiological parameters.

Alvarado Score For Predicting Acute Appendicitis Good Clinical Care

The Alvarado scoring system first described in 1985 by Alvarado was designed to ascertain the likelihood of appendicitis and facilitate decision-making in patients with suspected appendicitis (3). It uses history, symptoms, signs and laboratory data to calculate an overall score out of ten.

Three symptoms, three signs and two laboratory data constitute the Alvarado score (see Table 1). The resultant score predicts the probability of appendicitis (see Table 2) and can be used to guide further management (see Table 3).

	Feature	Score
Symptoms	Migration of pain to right iliac fossa	1
	Anorexia	1
	Nausea	1
Signs	Tenderness in right lower quadrant of abdomen	2
	Rebound tenderness	1
	Elevated temperature > 37.3 C	1
Laboratory data	Leucocytosis	2
	Shift of white blood cells to left (left shift in ratio of immature to mature white blood cells on white blood cell differential).	1
	Total	**10**

Table 1: Alvarado Scoring System (3)

Score	Significance	Probability of Appendicitis
<4	Unlikely diagnosis of appendicitis	30%
5-6	Possible diagnosis of appendicitis	66%
7-8	Probable diagnosis of appendicitis	93%
9-10	Very probably diagnosis of appendicitis	

Table 2: Probability of Appendicitis according to Alvarado Score (3)

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Score	Recommendation
<4	Discharge if no other medical/surgical problems
5-6	Admit for regular observation
7-8	Surgical intervention
9-10	

Table 3: Recommendations regarding further management according to Alvarado score (1,3)

An acronym that can be used to memorise the components of the Alvarado scoring system is MANTRELS (Migration of pain, Anorexia, Nausea, Tenderness in right lower quadrant, Rebound tenderness, Elevated temperature, Leucocytosis, Shift of white cells to the left). Due to the popularity of this acronym the term "MANTRELS score" is often used synonymously with the Alvarado score.

Efficacy and Limitations

Ohle et al carried out a systematic review in 2011 to assess the diagnostic accuracy of the Alvarado Score for predicting acute appendicitis (1). The authors found that the Alvarado score performs well when using a cut-off score of five out of ten to rule out appendicitis (99% sensitivity overall; 96% in men, 99% in women and 99% in children) (1). However, the positive predictive value is poor when using a score of seven out of ten to confirm appendicitis and recommend subsequent surgical intervention (81% specificity overall; 57% in men, 73% in women and 76% in children) (1).

The review compared predicted events (patients diagnosed with appendicitis according to the Alvarado score) with observed events (actual number of patients with appendicitis). Men, women and children were analysed separately within three risk strata of the Alvarado score (Low, score 1 to 4; intermediate, score 5 or 6 and high, score 7 to 10). Risk ratios (RR) were calculated with 95% confidence intervals (CI). RR < 1 represents under prediction of appendicitis using the Alvarado score and RR > 1 indicates an over-prediction of appendicitis using the score.

The review established that the Alvarado score is well calibrated in men across all risk strata (low RR 1.06, 95% CI 0.87 to 1.28; intermediate RR 1.0[9], 95% CI 0.86 to 1.37 and high RR 1.02, 95% CI 0.97 to 1.08) (1).

However, the score was inconsistent in children with a non-significant over-prediction of appendicitis in the low risk category (RR 5.03, 95% CI 0.52 to 48.82) and a significant over-prediction in the intermediate and high risk categories (intermediate RR 1.81, 95% CI 1.13 to 2.89 and high RR 1.13, 95% CI 1.01 to 1.27) (1).

Additionally, Ohle et al showed that the Alvarado score results in a significant over-prediction of appendicitis in women across all three risk strata (low RR 5.35, CI 95% 2.17 to 13.19; intermediate RR 1.82, CI 95% 1.20 to 2.78 and high RR 1.14, 1.04 to 1.25) (1). Diagnosis of appendicitis in women is clinically challenging because gynaecological pathologies may present with synonymous symptoms.

Heineman carried out a separate evaluation of the Alvarado score for diagnosing appendicitis in children in 2012 (4). He concluded that the data may suggest good risk stratification in children but with wide confidence intervals it does not do this effectively and more evidence is required.

A study published in Tropical Doctor in 2011 established that the Alvarado scoring system is an effective diagnostic method, which decreases over-diagnosis of appendicitis overall and reduces the risk of missing acute appendicitis cases (5). Additionally, it determined that the score is easy to implement by junior doctors with good results5. A manuscript in Surgical Endoscopy in 2014 concurred that the score is useful for young colleagues but found clinical judgement to be more reliable (6).

Conclusion and Recommendations

The Alvarado scoring system is a useful initial diagnostic tool for predicting the probability of appendicitis; particularly for foundation doctors. The scoring system is predominantly useful in order to rule out appendicitis and guide further management. It may minimize inaccurate diagnosis of appendicitis and reduces unnecessary intervention and negative appendicectomies.

However, caution should be exercised when using the Alvarado score in women and children and additional diagnostic tools such as imaging should be considered if the diagnosis is equivocal. A low score should prompt the foundation doctor to widen their differentials and consider the other causes of right iliac fossa pain. The Alvarado score should be used as a diagnostic aid when appendicitis is suspected but simultaneous implementation of clinical judgement is imperative.

ALVARADO SCORE FOR PREDICTING ACUTE APPENDICITIS

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Questions

1) A 17-year-old male is referred to the surgical admissions unit by his GP with right-sided abdominal pain. On examination he is tender at McBurney's point. He has a temperature of 37.9 degrees celsius and his full blood count results reveal a white cell count of 15.68 x 10^9/L. What is his Alvarado score?

a. 4

b. 5

c. 6

d. 7

e. 8

2) What is the next most appropriate step in his management according to his Alvarado score?

a. Discharge

b. Commence intravenous antibiotics

c. Admit for observation

d. CT abdomen pelvis

e. Nil by mouth, intravenous fluids and surgical intervention

3) The patient is admitted for 24 hours of observation. His pain has become progressively worse with rebound tenderness and is now associated with nausea and vomiting. He also complains of a loss of appetite. His repeat full blood count shows a white cell count of 18.43 x 10^9/L and his observation chart shows that he is pyrexial despite Paracetamol administration. What should be the next management step?

a. Discharge

b. Commence intravenous antibiotics

c. Observe for further 24 hours

d. CT abdomen pelvis

e. Nil by mouth, intravenous fluids and surgical intervention

4) A 25-year-old woman presents to Accident and Emergency with pain in her right iliac fossa. On examination she is tender in the right iliac fossa and is complaining of nausea. Her blood results are unremarkable and she is apyrexial. What is her Alvarado score?

a. 2

b. 3

c. 4

d. 5

e. 6

5) What is the next most appropriate step in her management?

a. Discharge

b. Commence antibiotics

c. Admit for observation

d. Transabdominal and transvaginal ultrasound

e. Diagnostic laparoscopy

Answers and Discussion

1) b

The patient has an Alvarado score of 5 based on tenderness in the right lower quadrant (scoring 2), pyrexia (scoring 1) and raised white cell count (scoring 2). No mention is made of central pain migrating to the right iliac fossa, anorexia, nausea, rebound tenderness or shift of white cells to the left. A score of 5 indicates a possible appendicitis with a probability of 66%.

2) c

When scoring 5 or 6 on the Alvarado score the recommendation is to admit the patient for regular observation. Repeated clinical examination and laboratory data should guide further management. Note that this patient is male and a systematic review by Ohle et al showed the Alvarado score is well calibrated and reliable in men.

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3) e

The patient's clinical condition has deteriorated. If rescored the patient would now score 8 on the Alvarado score (additional 3 points for rebound tenderness, nausea and anorexia). The recommendation for a score of 7 to 10 is surgical intervention. Whilst awaiting theatre the patient should be nil-by-mouth with intravenous fluids for hydration. He should be consented and booked for the emergency theatre list.

4) b

The patient has an Alvarado score of 3 based on pain in the right lower quadrant (scoring 2) and nausea (scoring 1). There is no mention of migrating pain, anorexia, or rebound tenderness. She does not have an elevated temperature and her blood results are normal. This indicates an unlikely diagnosis of appendicitis with a probability of 30%.

5) d

A low Alvarado score of 4 or less should prompt consideration of other differentials for right iliac fossa pain. The Alvarado score must be used with caution in women and it is important to note that other intraabdominal pathologies cannot be excluded. Due to diagnostic uncertainty imaging should be considered. A transabdominal and transvaginal ultrasound scan would be appropriate in this case.

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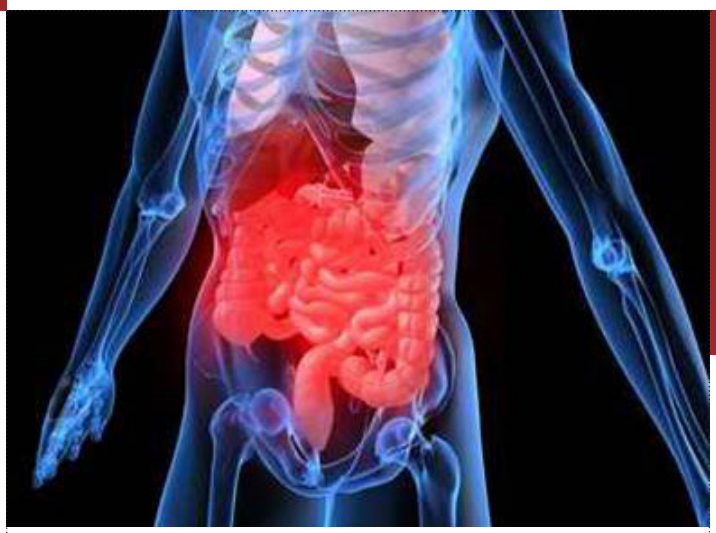
MANAGING THE ACUTE ABDOMEN

N Sihra, M Mlotshwa



Abstract

A patient presents to A&E twice in one week complaining of central abdominal pain. After re-presenting to A&E she was admitted under the gynaecology team where she went on to develop four quadrant peritonitis and underwent an emergency laparotomy. Was something missed on her first presentation?

Case history

A 54 year old female presents to A&E with a 5 day history of central colicky abdominal pain. Her pain is associated with nausea and vomiting and she denies opening her bowels for 4 days, passing small amounts of flatus only. She was discharged from A&E earlier this week with reassurance following no obvious abnormalities on imaging or blood results. Today she presents with constant sharp central abdominal pain, 9/10 in severity.

The patient is otherwise fit and well with no other medical co-morbidities. She has a history of recurrent ovarian cyst rupture and mentions that she has had similar episodes of colicky abdominal pain and distension over the past year which has always been self limiting.

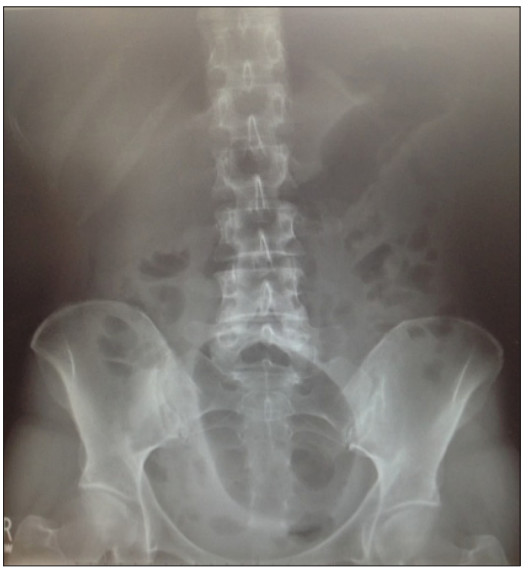
Clinically she is tachycardic (100/min), normotensive and afebrile. Bloods are unremarkable (WCC 11.1, CRP <1.0). Her abdomen is distended on palpation with generalised tenderness and scant bowel sounds. On digital rectal examination she has an empty rectum with a boggy pouch of douglas. An abdominal x-ray is performed which shows no evidence of obvious small or large bowel obstruction.

Given her unremarkable past medical history and in light of her previous ovarian accidents, she has been referred to the gynaecology team for further input.

Discussion

On the following day she went on to develop four quadrant peritonitis with features of sepsis. Blood tests show a significant rise in inflammatory markers; CRP >320 and WCC 3.4. The patient was taken straight to theatre and subsequently underwent a laparotomy, right hemicolectomy and Paul-Mikulicz stoma for a caecal volvulus and perforation. Following this she was managed on HDU where she made a good recovery.

An abdominal X-ray from her first presentation to A&E is shown below:



The gas filled stomach and gas pattern in the colon were readily identified by those assessing her in A&E. The dilated loop of bowel in the pelvis was however missed.

MANAGING THE ACUTE ABDOMEN

N Sihra, M Mlotshwa

Is this of any clinical significance?

A perforated large bowel was found at laparotomy related to the above pathology. This abdominal x-ray shows a caecal volvulus with closed loop obstruction. Over the course of the 5 days, this bowel segment underwent ischaemic insult and subsequently perforated resulting in four quadrant peritonitis.

The history of colicky central abdominal pain and near absolute constipation is suggestive of midgut obstruction. The progression of pain from colicky to constant is suggestive of imminent bowel ischaemia.

Test yourself

1. You have been called by the gynaecology SHO to review this patient who has a rigid and distended abdomen, is tachycardic and has reduced urine output. You have reviewed her history and initial presentation. What is your initial diagnosis?

a) peritonitis secondary to perforated viscous
b) appendicitis
c) pyelonephritis
d) diverticulitis
e) ovarian torsion

2. What is your key priority in managing this patient?

a) septic screen
b) resuscitation and preparing the patient for urgent surgical intervention
c) commencing IV antibiotics
d) analgesia
e) CT abdomen

3. In view of this patient's history and the x-ray above, what is your most likely differential diagnosis?

a) large bowel obstruction with imminent bowel ischaemia
b) closed loop obstruction (small bowel, caecal volvulus) with imminent ischaemia
c) perforated diverticulitis (with sentinel loop)
d) perforated appendicitis (with sentinel loop)
e) pancreatitis (with sentinel loop)

4. What feature is characteristic of small bowel that helps to differentiate it from large bowel on plain abdominal imaging?

a) haustrations
b) valvulae conniventes
c) peripheral location
d) appendices epiploicae
e) diameter

5. What is the most common predisposition of a caecal volvulus?

a) malignancy
b) embryonic development variation
c) Meckel's diverticulum
d) diet
e) adhesions

Answers

1a)

Clinically, the patient lying still with a distended, rigid and tender abdomen with scant bowel sounds is pathognomonic of peritonitis and indicates the urgency of surgical intervention.

2b)

I recognise this question is difficult to answer as most of these options are entirely appropriate. A CT abdomen may provide useful information however is likely to cause a delay in management and a subsequent adverse outcome. Initial assessment should be performed using an ABCDE approach (airway, breathing, circulation, disability and exposure). Following this, it is important to ensure that the patient has a patent airway and has supplementary oxygen in order to maintain adequate oxygenation.

When assessing circulation it is important to obtain intravenous access and fluid resuscitation and subsequently catheterisation to monitor fluid balance. A blood gas should be performed to check the patient's lactate.

If the patient is febrile, blood cultures should be obtained as part of the sepsis six pathway.

Analgesia should be prescribed as appropriate. If a perforation is clinically suspected intravenous antibiotics should be commenced within the golden hour as per local microbiology protocol for intra-abdominal sepsis.

Although all of the above is essential, if the patient is stable, it is important to liaise with the appropriate healthcare professionals; i.e. senior surgical team, anaesthetists and theatre staff in view of urgent surgical intervention as the definitive management of peritonitis.

MANAGING THE ACUTE ABDOMEN

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3b)

Note - pancreatitis with a sentinel loop is unlikely given the location of the dilatation on the plain film.

4b)

The bowel loop has no valvulae conniventes suggesting that this is large bowel. Characteristically small bowel has valvulae conniventes. These are thin circular folds of mucosa which appear to cross the full width of the lumen on a plain film. Large bowel typically has haustrations. These are sacculations formed by the longitudinal muscles (taenia coli) and circula muscles of the colon.

3, 6, 9 as a rule of thumb:

Dilatation of small bowel >3cm is abnormal

Dilatation of the colon >6cm is abnormal

Dilatation of the caecum >9cm is abnormal

5b)

A caecal volvulus is characterised by the axial twisting which can involve the caecum, terminal ileum and ascending colon. In 11% of the population it is predisposed by excess caecal mobility which is typically associated with incomplete intestinal rotation during fetal development.

The caecum is typically a retroperitoneal structure which is not susceptible to twisting however developmental variations in the degree of mesenteric fusion can result in differences in the mobility of the caecum and ascending colon. With a greater degree of mobility there is an association with potential folding or twisting on its mesentery.

Untreated and unresolved acute volvulus can be complicated by bowel necrosis and/or perforation.

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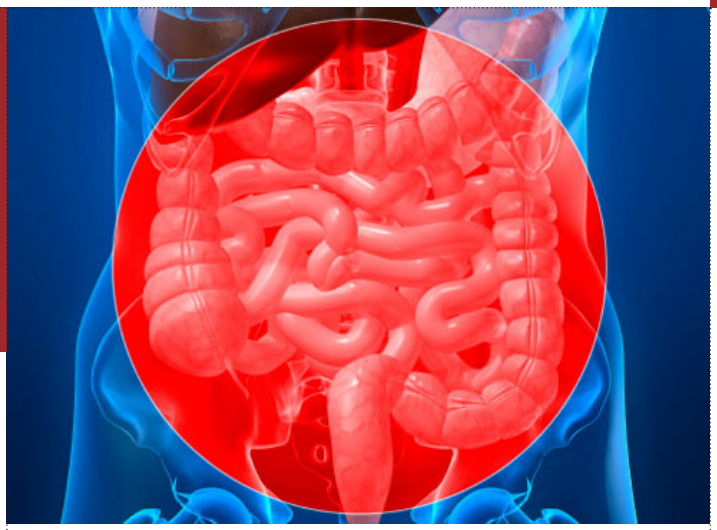
ACUTE BOWEL ISCHAEMIA

T Cash, R Ackroyd & CE Owers

Acute Bowel Ischaemia
Patient Management



Abstract

Acute bowel ischaemia is a common and potentially life threatening condition encountered by the junior doctor on the acute surgical take. We discuss the presentation, aetiology and appropriate treatment for this surgical emergency. The junior doctor should be aware of the required investigations and initial management for patients with suspected acute bowel ischaemia and understand the need to quickly escalate to more senior members of the team.

Case History

Mrs S is a 76 year old lady admitted from A+E with sudden onset abdominal pain which she describes as constant and colicky. She had a similar post-prandial episode several weeks ago that was less intense and subsided quickly. She complains of nausea and reports one loose bowel motion yesterday. Her normal bowel habit is every 2-3 days.

Past medical history includes type 2 diabetes mellitus, atrial fibrillation, hypertension and osteoarthritis. She has had no previous abdominal surgery. Her current medications are metformin, aspirin, amlodipine and paracetamol. On examination her heart rate is 108 beats per minute, blood pressure 110/72 mmHg, respiratory rate 24, oxygen saturation is 97% on air and temperature 37.5°C. Cardiovascular and neurological examination is unremarkable.

On abdominal examination, she is moderately tender in the peri-umbilical region with no guarding. The abdomen is slightly distended and bowel sounds are negligible. PR examination reveals soft stool in the rectum but no blood or masses.

Blood test results taken in A+E:

Hb	*12.1*	*Sodium*	*135*	*CRP*	*110*
WCC	*13.0*	*Potassium*	*5.4*	*LFTs*	*normal*
		Urea	*12*	*Amylase*	*214*
		Creatinine	*94*		

Arterial blood gas:

pH	*7.30*
pO_2	*11.0*
pCO_2	*3.4*
HCO_3	*18*
Lactate	*6*

Chest x-ray reveals no abnormality. There are some slightly distended small bowel loops on abdominal x-ray, but a normal gas pattern is seen throughout the colon.

The history and initial investigations raise the suspicion of acute bowel ischaemia. After discussion, the surgical registrar requests a CT with intravenous (IV) contrast.

The CT report suggests a thrombus in the superior mesenteric artery with some oedematous jejunum, supporting the diagnosis of acute bowel ischaemia. The on call consultant is informed and the patient is taken to theatre for laparotomy and bowel resection; approximately 50cm of jejunum is removed.

Good collateral blood supply is demonstrated to the remaining bowel and a primary anastomosis performed. Mrs S is transferred to intensive care postoperatively where she receives therapeutic low molecular weight heparin (LMWH) and makes an otherwise uneventful recovery.

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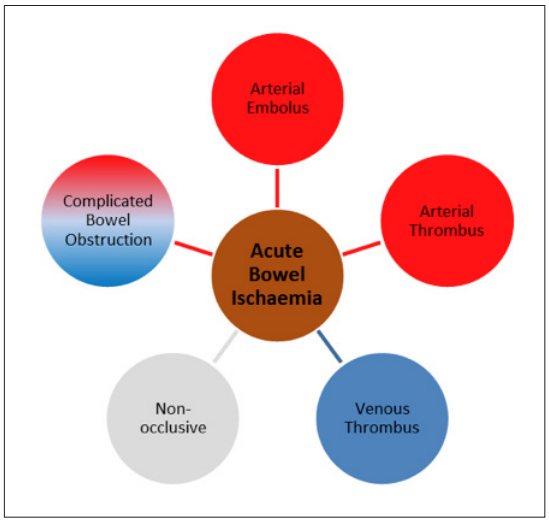
Discussion

In health the bowel receives between 10 and 35% of the cardiac output depending whether it is in the fasted or non-fasted state (1). Acute bowel ischaemia results when this blood supply is interrupted and can be caused by occlusive or non-occlusive events of both the arterial and venous blood supply.

Clinical presentation corresponds to the degree of hypoperfusion. A comprehensive history, including speed of onset of symptoms and risk factors, can help establish the likely causative mechanism. Classically, acute bowel ischaemia presents as sudden onset para-umbilical abdominal pain which is out of proportion to examination findings (2). The main causes of acute bowel ischaemia include:



Arterial embolus is the commonest cause of acute bowel ischaemia, responsible for up to 50% of cases (3). The majority of arterial emboli originate in the heart. Disturbances in cardiac function due to atrial fibrillation, myocardial infarction and endocarditis are major risk factors for the development of cardiac mural thrombi, which can dislodge and occlude the mesenteric arteries. As with Mrs S, the site most commonly affected is the superior mesenteric artery as it leaves the aorta at an oblique angle (2).

Arterial thrombus is responsible for approximately 25% of cases (3). This almost always occurs in elderly patients with advanced atherosclerotic disease (2). Operative mortality is high due to the presence of co-morbidities.

Non-occlusive ischaemia is less common and its mechanism is not fully understood (4). It is thought to occur in relation to low cardiac output and associated mesenteric vasoconstriction. It is unlikely to be encountered by a junior doctor, as patients are typically critically ill and on the intensive care unit. Risk factors include hypovolaemia, heart failure and vasoconstrictive drugs (5).

Mesenteric venous thrombosis is the least common cause of acute bowel ischaemia. It can present acutely or chronically and often has an insidious onset. It is likely to develop in patients with risk factors for venous thromboembolism; typically primary clotting disorders or pro-thrombotic states (malignancy, pancreatitis, sepsis etc.) (6).

Another common cause of acute bowel ischaemia, which will be encountered on the acute surgical take, is obstructive bowel pathologies which include; incarcerated hernia, volvulus and intussusception (7). This should be suspected in patients with clinical features of complete obstruction (although a Richter's hernia may cause ischaemia without obstruction). Patients will usually meet the criteria for systemic inflammatory response syndrome (SIRS) with fever, tachycardia and leucocytosis.

Prompt recognition and escalation of patients with suspected acute bowel ischaemia to senior colleagues is necessary to prevent bowel necrosis, perforation, sepsis and death. An arterial blood gas will typically reveal a metabolic acidosis due to raised lactate production. CT angiography is the gold standard investigation, but often impractical to perform (8).

By comparison, CT with IV contrast is routinely performed in patients with acute abdominal pain. Classic radiological features include lack of bowel wall enhancement, intramural and portal venous gas (9). However, findings are often non-specific and a high clinical index of suspicion is therefore required (10). Plain abdominal X-ray can exclude bowel perforation, but this is a late sign (11). Serial lactate measurement is of limited prognostic value but can aid diagnosis, particularly in patients who are unable to undergo CT (12).

The principle of treatment is to restore circulation as quickly as possible. Initial supportive management which should be instigated by the junior doctor includes; fluid resuscitation, correction of electrolyte disturbances and broad spectrum antibiotics (2). The presence of peritoneal signs irrespective of the underlying mechanism warrants immediate laparotomy, unless palliation is appropriate. This allows for inspection and resection of necrotic bowel as well as revascularisation where possible.

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In the presence of arterial occlusion, open surgical embolectomy is the traditional treatment method (13). However, interventional radiological approaches are increasingly being used. This approach is particularly desirable in patients with multiple comorbidities who have a higher perioperative risk. Delay in intervention increases mortality (14), therefore as a junior doctor it is imperative to quickly recognise and escalate these patients early.

By comparison surgical intervention is often not necessary in non-occlusive ischaemia and mesenteric venous thrombosis. Treatment for non-occlusive ischaemia centres on removing precipitating factors and optimising the patient's physiology. Appropriate anticoagulation is often all that is necessary to treat mesenteric venous thrombosis (15).

Despite best efforts, the outcome for acute bowel ischaemia remains poor with mortality around 50% (16). This is primarily because the modal patient is elderly and medically compromised. Patients who survive an acute episode need appropriate medical optimisation. Lifelong anticoagulation is indicated in patients with arterial embolic or venous thrombotic disease. Patients who have an arterial thrombotic event should receive targeted atherosclerotic medication including an anti-platelet and statin (17).

Multiple Choice Questions

1. In a patient with classical acute bowel ischemia what would you expect to find on arterial blood gas?

a. Metabolic alkalosis
b. Respiratory acidosis
c. Metabolic acidosis
d. Respiratory alkalosis
e. Mixed acidosis

2. In suspected acute bowel ischaemia, what is the most appropriate radiological imaging to perform?

a. AXR and erect CXR
b. MRI
c. Duplex ultrasound
d. CT with oral contrast
e. CT with IV contrast

3. A patient is found to have a superior mesenteric artery embolus. Which area of bowel is NOT supplied by the superior mesenteric artery?

a. Caecum
b. Jejunum
c. Descending colon
d. Ileum
e. Transverse colon

4. A 35 year old lady with advanced ovarian cancer presents to A+E with a 10 day history of worsening central abdominal pain and nausea. On examination the abdomen is distended but there is no guarding. Heart rate is 105 beats per minute and observations are otherwise within normal parameters. What is the most likely underlying pathophysiological process?

a. Arterial embolism
b. Arterial thrombosis
c. Non-occlusive mesenteric ischaemia
d. Mesenteric venous thrombosis
e. None of the above

5.You are called to see an elderly patient on the ward 3 days after they underwent bowel resection for acute bowel ischaemia. The patient is vomiting and they have not opened their bowels post-operatively. On examination the abdomen is soft but distended and tympanic. Chest auscultation reveals crepitations at the right base. Arterial blood gas is normal but the potassium is 2.5 mmol/L. What is the most likely cause of this patient's symptoms?

a. Post-operative collection
b. Further acute bowel ischaemia
c. Norovirus
d. Post-operative ileus
e. Obstructed incisional hernia

Answers and Teaching Notes

1c) Metabolic acidosis

A metabolic acidosis is classically seen in acute bowel ischaemia secondary to increased lactate production from anaerobic respiration.

2e) CT with IV contrast

Although CT angiography is the gold standard investigation, CT with IV contrast is more widely available. Classic radiological features include lack of bowel wall enhancement, intramural and portal venous gas. Findings are often non-specific and a high clinical index of suspicion is needed.

3c) Descending colon

The superior mesenteric artery (SMA) leaves the aorta at an oblique angle at the level of L1 and is therefore the commonest site for arterial emboli to lodge. The vascular territory of the SMA begins in the lower part of the duodenum and ends two thirds along the transverse colon. The pancreas also derives part of its blood supply from the SMA.

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4d) Mesenteric venous thrombosis

Mesenteric venous thrombosis has a slower onset of symptoms when compared to arterial causes of acute bowel ischaemia. Ovarian cancer is a risk factor for venous thromboembolism and should increase the clinical index of suspicion.

5d) Post-operative ileus

Post-operative ileus is common after laparotomy and intestinal manipulation. This patient requires insertion of a nasogastric tube and fluid balance monitoring. As the patient has right basal crepitations, a chest x-ray should be requested to look for aspiration pneumonia. Hypokalaemia may also be the cause of the ileus and should be corrected.

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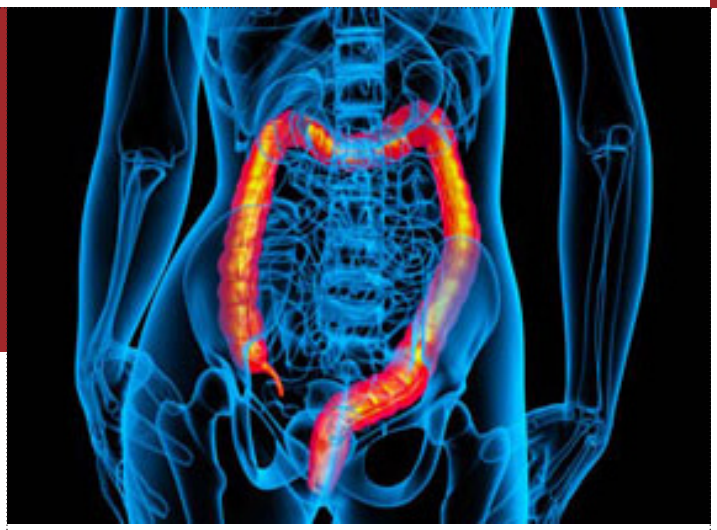
ISCHAEMIC COLITIS & BLEEDING PER RECTUM - A SURGICAL EMERGENCY

Y El-Dhuwaib, SS Prasad, A Das & N Khetan

Ischaemic colitis & bleeding per rectum - a surgical emergency
Patient Management



Abstract

Gastrointestinal bleeding (GI) is a common emergency presentation to hospitals. It accounts for about 1% to 2% of all emergency admissions. It is classified into upper and lower GI bleeding and the ligament of Treitz is traditionally considered the dividing point between the two. Any bleeding distal to its insertion (Duodeno-Jejunal flexure) is considered lower GI bleeding. Presenting history plays an important part in diagnosis and treatment plan. The authors present a 63 years old male patient with lower GI bleeding and abdominal pain due to ischaemic colitis. The article discusses the causes, investigation and management of lower GI bleed with focus on ischaemic colitis.

Case history

A 63 years old male patient was admitted as an emergency with bloody diarrhoea for 12 hours associated with left side abdominal pain and vomiting. He had previous history of atrial fibrillation (AF), though he was not on any anticoagulant. He was also known to have sigmoid diverticulosis. At presentation, he was haemodynamically stable. The abdomen was soft but tender on the left side. Digital rectal examination showed fresh blood.

His inflammatory markers were raised (C-reactive protein 34mg/l and white cell count 14500 per cubic millimetre of blood). The CT scan showed a long segment of grossly oedematous large bowel at splenic flexure with oedema of the adjacent peritoneal fat. The appearance was consistent with colitis and because of its location; a common site for ischaemic colitis (IC) a diagnosis of IC was highly suspected (Figure 1).

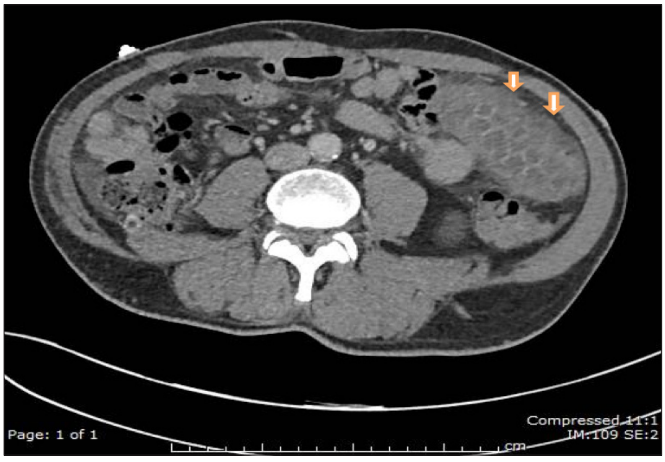


Figure 1: CT scan at day 1 showed long segment of grossly oedematous large bowel at splenic flexure with oedema of the adjacent peritoneal fat (arrows).

The scan also showed diverticular disease of the sigmoid and left colon; however, it was not inflamed. Flexible sigmoidoscopy confirmed the diagnosis of IC (Figure 2).

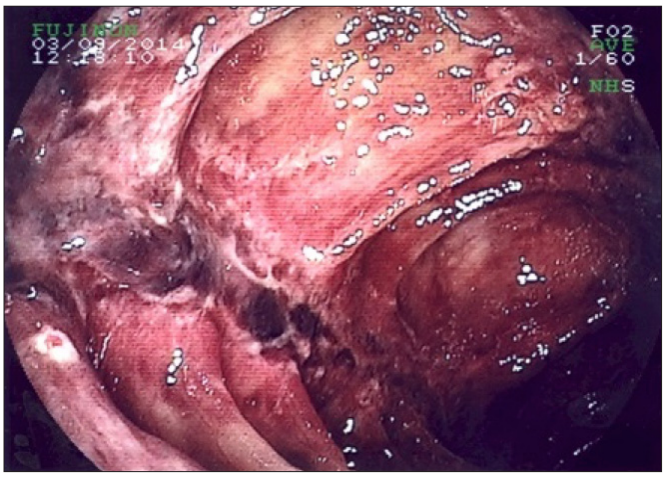


Figure 2: Flexible sigmoidoscopy showed ischemic colitis

The biopsies were taken and the histology subsequently was consistent with a diagnosis of IC. A CT angiogram was performed because of AF history and it ruled out any proximal arterial disease. The patient was treated conservatively with bowel rest, analgesics, antibiotics, low molecular heparin (dalteparin) and intravenous fluids. He made a remarkable improvement within 48 hours of admission. The bleeding stopped and he was pain free. On day five he was discharged home on therapeutic dalteparin.

The patient was reviewed in the outpatient clinic in a month's time and he remained asymptomatic. Aspirin was prescribed as a replacement of dalteparin. A colonoscopy was performed 2 months from the onset of admission and showed marked improvement of the bowel mucosa, but unfortunately, the patient developed a non- passable short stricture at the splenic flexure.

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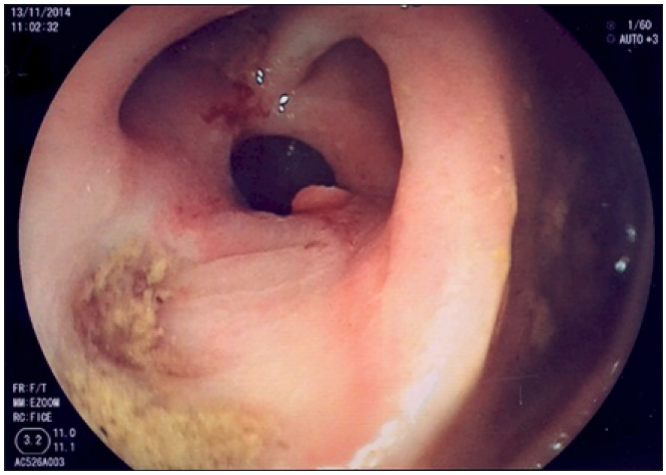

Figure 3: Complete resolution of ischaemic colitis but a stricture has developed.

A virtual CT colonograghy was performed at three months from onset of symptoms to assess the proximal colon and the stricture. It showed no proximal abnormality with complete resolution of the stricture (figure 4 and 5).

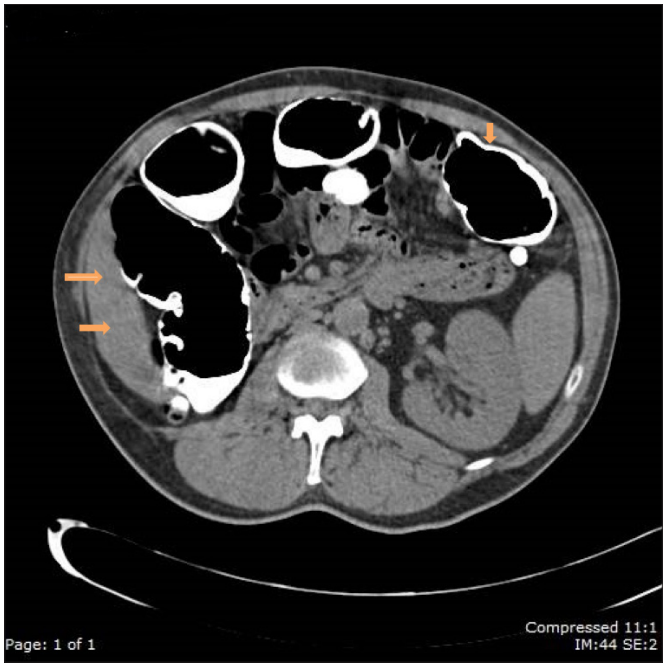

Figure 4: CT colonography showed complete resolution of the stricture at splenic flexure. One arrow shows resolution of stricture and the 2 arrows show proximal colon.

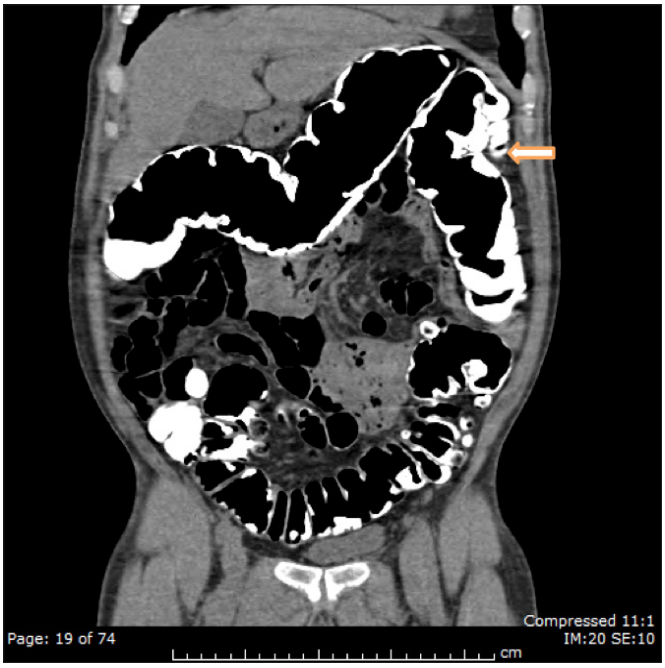

Figure 5: Coronal plane shows complete resolution of the stricture (arrow).

The patient remains asymptomatic at 6 months of follow up.

Discussion

The incidence of significant lower GI bleed requiring emergency admission is 122 event/100,000 populations per year (1). The most common causes for emergency admission with lower GI bleeding (2) are colonic diverticulosis, haemorrhoids and colorectal carcinoma. Other causes (3) are ischaemic colitis, inflammatory bowel disease, radiation colitis, angiodysplasia, and anticoagulation (warfarin).

Brisk bleeding from an upper GI source may also present as fresh bleeding per rectum. Although, emergency admission of lower GI bleed is common, most patients respond to conservative management and stop spontaneously. However, a small number of patients may require radiological or surgical intervention. The mortality rate is about 2 to 4 percent (4).

When admitting a patient with lower GI bleeding, a thorough history with complete physical examination, including digital rectal examination is necessary to initiate a diagnosis and management plan. There are certain clues in the history that can help in making provisional diagnosis. Young patients with painless fresh rectal bleeding, with normal bowel motion are almost always due to haemorrhoids or angiodysplasia.

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If it is associated with tenesmus (a spurious feeling of the need to evacuate the bowels, with little or no passage of stool) and mucous discharge, then it is likely to be colitis in young people or tumour in people above the age of 40. Lower GI malignancy should also be highly suspected if the patient has a history of recent change of bowel habit, weight loss or there is a palpable mass in the abdomen or rectum. If the bleeding is dark/ plum coloured and mixed with the stool, then the source of bleeding is more proximal in the colon. Lower GI bleeding with left sided pain in elderly patients, is usually due to diverticulosis or ischemic colitis. Patients on warfarin are infrequently admitted with bleeding per rectum.

The bleeding should be assessed whether it is life threatening or not. Signs for shock should be carefully examined. The shock index is a useful guide for assessment and it is calculated by dividing the pulse rate over the systolic pressure. In normal circumstances a result between 0.5 and 0.8 is obtained. A figure of 1 or more indicates severe shock. If the patient is in shock, he should be resuscitated immediately with fluids. Full blood count, cell reactive protein (CRP), Urea and electrolytes, clotting factors and cross match are mandatory first line of investigations. Blood transfusion should be given if necessary.

Once the patient is resuscitated further investigations are performed to diagnose the source of bleeding. Gastroscopy should be performed in all patients with suspected upper GI bleeding (upto 15% of cases(5)). The lower gastrointestinal endoscopy (colonoscopy/ flexible sigmoidoscopy) in acute setting has limited value because of difficult visualisation. In selected cases, intra-operative endoscopy may aide identify the site of bleeding in order to assist surgical resection. Once the bleeding has settled, colonoscopy helps identify the source of bleeding and provides therapeutic procedures such as polypectomy.

In recent years, CT angiogram has replaced colonoscopy as the initial investigation of choice to assess massive lower GI bleeding. The minimum bleeding rate that can be detected by CT angiogram is 0.3 ml/min(6) and sensitivity of 86% (95% Confidence Interval(CI) 78-92%) and specificity of 95% (95% CI 76-100%)(7). CT angiogram is best performed while patient is actively bleeding to maximise the chance of localizing the bleeding site. Other forms of investigation such as non-invasive imaging with technetium-99m–labelled red blood cell (RBC) or Tc-99m sulfur colloid scintigraphy can be used to detect and localize gastrointestinal bleeding (8). However, their use has diminished in the current UK practice.

In presence of severe abdominal pain and/or peritonism as well as bleeding per rectum, colitis or diverticulitis is often suspected and a CT scan is the investigation of choice (9). CT in ischaemic colitis shows the initial bowel wall thickening, thumb printing and peri-colonic stranding (10). It can also show thrombus in the arteries or blockage of the mesenteric vessels. In the presence of gas in the bowel wall (pneumatosis) and/or gas in the mesenteric veins, bowel infarction is suspected. It can also rule out other pathology such as perforation and diverticulitis.

Colonoscopy is considered the most sensitive investigation for IC. It offers direct visualisation of the mucosa and provides biopsy for histology. Findings at colonoscopy are highly dependent on the duration and severity of ischemic injury and it should be performed within 48 hours of onset of symptoms for accurate diagnosis (10).

Majority (75% -90%) (11) of lower GI bleeding patients respond to conservative management. Selected cases (20%) require radiological (embolization), endoscopic (polypectomy/argon diathermy) or surgical resection of bowel (life threatening bleeding). The treatment varies according to the aetiology. In case of haemorrhoids, rubber band ligation or examination under anaesthesia and suturing the bleeding artery is often sufficient. For bleeding diverticular disease or angiodysplasia mesenteric angiogram and embolization is often successful. Surgical resection of diverticular disease is indicated in up to 20% of cases (12, 13).

Majority of IC patients respond to conservative management. Patients should be resuscitated with fluids and bowel rest to reduce intestinal oxygen requirement. Optimization of cardiac function and oxygenation is indicated. Vasopressors should be avoided if possible. Antibiotics are given to cover aerobic and anaerobic bacteria and minimize bacterial translocation and sepsis due to mucosal injury (14). Surgical intervention is needed in about 20% of cases (15) with a mortality rate of up to 60%. Resection of the affected part and end colostomy is required.

The authors suggest a practical flowchart for management of emergency lower GI Bleeding (Figure 6).

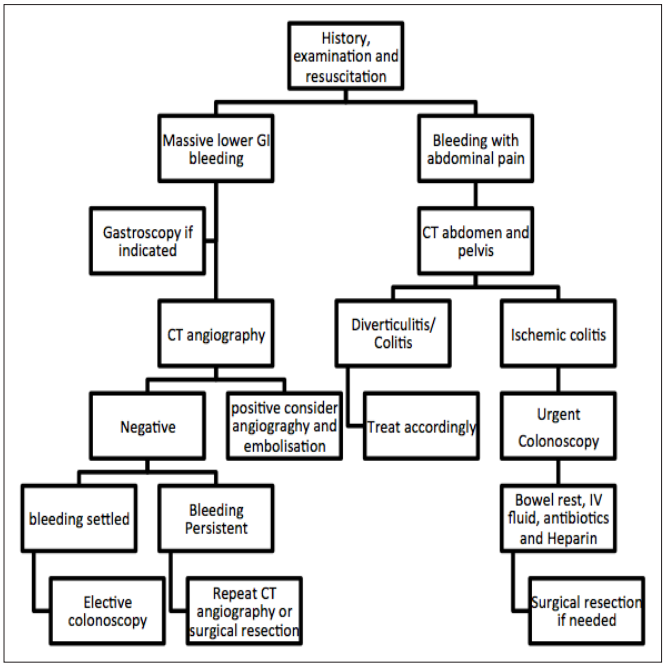

Figure6: Flowchart for management of emergency Lower GI Bleeding.

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Questions

1-5 are multiple-choice questions. Please select the best choice from the options provided in each question.

1. A patient presenting with emergency bleeding per rectum requires:

a. Complete history
b. Full physical examination
c. Digital rectal examination

Please select the best choice:

❒ *A is correct*
❒ *B is correct*
❒ *A and B are correct*
❒ *All are correct*

2. Management of patients with Lower GI bleeding include:

a. Fluid resuscitation in selected cases
b. All cases require CT angiography
c. All cases require surgical or radiological intervention
d. Majority settle with conservative management

Please select the best choice:

❒ *A is correct*
❒ *D is correct*
❒ *B and C are correct*
❒ *A and D are correct*

3. CT angiography in emergency GI bleeding is a useful test because

a. It can detect bleeding of 0.3 ml/min
b. It is non-invasive
c. It can detect bleeding of 0.1 ml/ min
d. It is always positive in a bleeding patient

Please select the best choice:

❒ *A is correct*
❒ *B is correct*
❒ *A and B are correct*
❒ *C and D are correct*

4. Common causes of emergency lower GI bleeding are

a. Diverticular disease
b. Bowel tumour
c. Angiodysplasia
d. Colitis

Please select the best choice:

❒ *A is correct*
❒ *B and C are correct*
❒ *A and D are correct*
❒ *All are correct*

5. In emergency lower GI bleeding

a. All patients require blood transfusion
b. All patients will settle with conservative management
c. Emergency colonoscopy is always diagnostic
d. It carries a mortality of 20%

Please select the best choice:

❒ *A and C are correct*
❒ *D is correct*
❒ *C is correct*
❒ *All are incorrect*

Answer Key

Q1: All are Correct

In order to assess any patient admitted with acute lower GI bleeding, a doctor should take a complete history and perform a full physical examination including digital rectal examination.

As mentioned in the discussion, there are certain clues in the history that can aid making provisional diagnosis. Physical examination is necessary to assess patient's overall condition and also provides further clues if any masses are found at the abdominal or rectal examination. The colour of the bleeding on per rectal examination can also give clue whether it is distal or proximal bleeding.

Q2: A and D are correct

All patients should be resuscitated with intravenous fluids. Majority of patients respond to conservative management and only few of them require surgical or radiological intervention. CT angiogram is indicated in patients who continue to bleed despite conservative management.

Q3: A and B are correct

CT angiogram is the first line of investigation in patients who fails to respond to conservative management. The minimal bleeding rate detected is 0.3ml/min. it should be performed while a patient is actively bleeding.

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Q4: All are correct

The most common causes for emergency admission with lower GI bleeding are colonic diverticulosis, haemorrhoids and colorectal carcinoma. Other causes are ischaemic colitis, inflammatory bowel disease, radiation colitis, angiodysplasia, and anticoagulation (warfarin).

Q5: All are incorrect

Blood transfusion should be given if a patient is profusely bleeding and the patient is in shock or the bleeding has stopped but the haemoglobin falls below 8gm/litre. Majority of patients (80%) respond to conservative management and the overall mortality rate is 2-4%.

Emergency colonoscopy can be diagnostic as well as therapeutic if a lesion is found though it has its limitation if intra-luminal blood obscures the view or the bleeding has stopped in cases like diverticular bleed or angiodysplasia. If a bleeding polyp is found, then a polypectomy is often sufficient to stop the bleeding. Argon diathermy can also be used to stop the bleeding site such as angiodysplasia.

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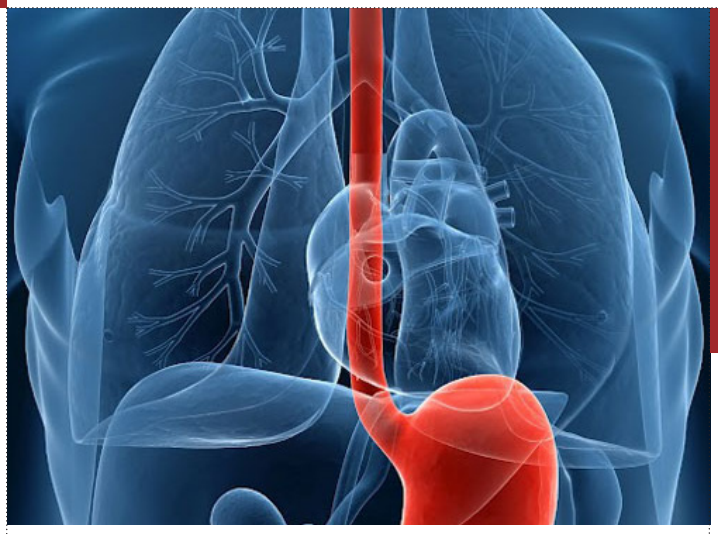
AN OVERVIEW OF MANAGEMENT OF CANCER OF OESOPHAGUS

VKH Wong, PK Jain



Abstract

Management of oesophageal cancer can be challenging. A case with oesophageal cancer discussed in detail with his management used to illustrate the treatment pathway of a locally advanced oesophageal cancer. This includes assessment of the disease and a brief overview of the treatment options of oesophageal cancer.

Case History

A 73-year-old male referred to the clinic with dysphagia and weight loss. An urgent upper GI endoscopy was organised for the patient but unfortunately, he was not able to tolerate the procedure. Subsequently, he underwent barium swallow (Figure 1) which showed a long eccentric stricture involving the middle and lower third of the oesophagus suggestive of malignant stricture.

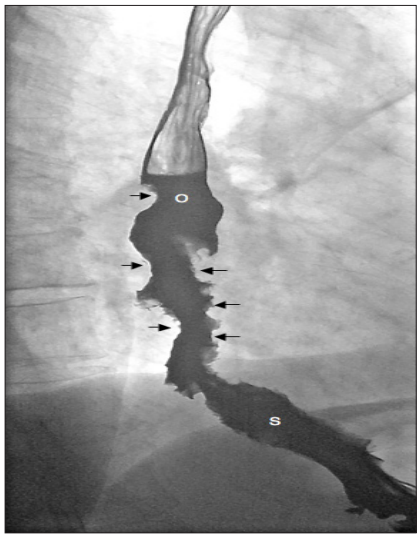


Figure 1: Barium swallow showing a long distal eccentric oesophageal cancer (black arrows highlighting the filling defect and shouldering caused by the oesophageal cancer). O - oesophagus, S-stomach.

An Overview Of Management Of Cancer Of Oesophagus
Patient Management

A CT scan of chest abdomen and pelvis (Figure 2) showed circumferential thickening of distal oesophagus and an enlarged gastro hepatic lymph node but no distant metastasis.

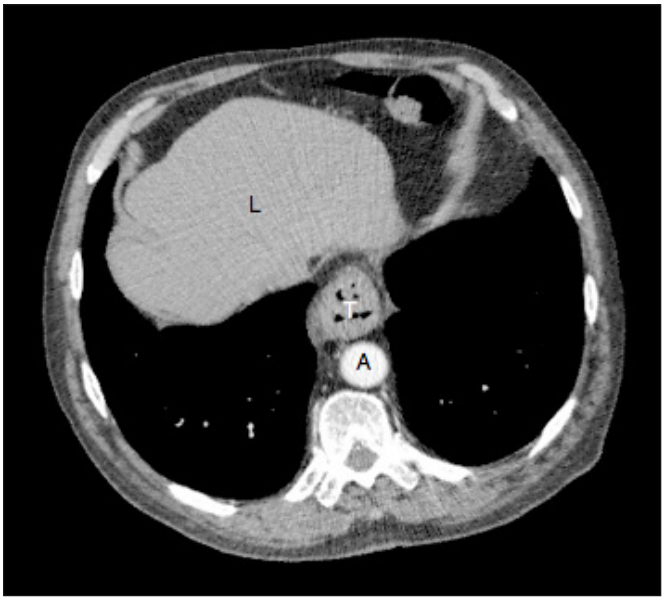


Figure 2: Axial image of the CT scan of the same patient showing the oesophageal cancer (T) and its relationship with the aorta (A) posteriorly, the liver (L) anteriorly and pleural on both sides.

The patient underwent upper GI endoscopy under general anaesthesia that showed malignant lesion involving distal third of the oesophagus. The biopsies taken during endoscopy confirmed poorly differentiated oesophageal adenocarcinoma with associated Barrett's metaplasia.

PET-CT scan demonstrated the lower oesophageal carcinoma with FDG avid node in the gastro-hepatic ligament and no distant metastasis. (Figure 3)

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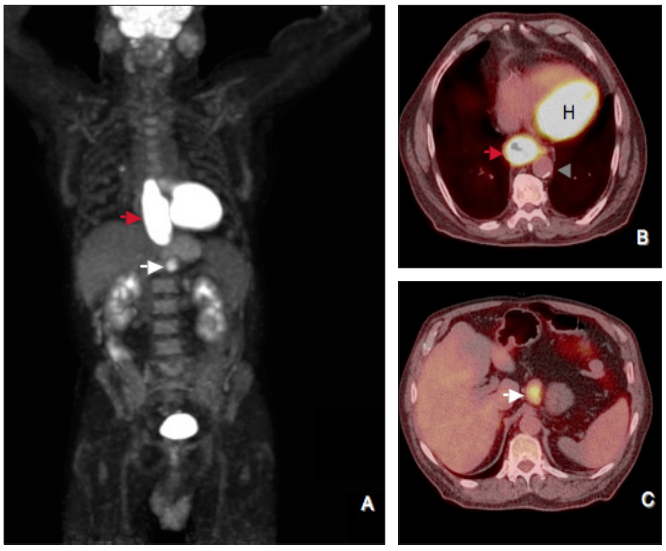
Figure 3: PET-CT scan of the same patient:



A: A coronal view of the PET-CT which showed the distal oesophageal cancer (red arrow) and the FDG avid gastro-hepatic lymph node (white arrow).

B: Corresponding transverse image of the oesophageal cancer (red arrow) with its close proximity to the heart (H) anteriorly and the aorta (grey arrow) posteriorly.

C: Corresponding transverse image of the FDG avid gastro-hepatic lymph node (white arrow) suggestive of metastases.

The patient's case discussed in Upper GI cancer multi-disciplinary team meeting (MDT) and recommendation was for patient to have neo-adjuvant chemotherapy in view of the locally advanced oesophageal cancer, prior to undergoing a two-stage oesophago-gastrectomy.

Discussion

Oesophageal cancer is the eighth most common cancer in the world. However; the incidence varies across the world with 80% of oesophageal cancer occurring in less developed regions such as Northern Iran, Central Asia and Northern Central China (1). In UK, oesophageal cancer is the eighth most common cancer for males and thirteenth most common cancer for female (Figure 4).

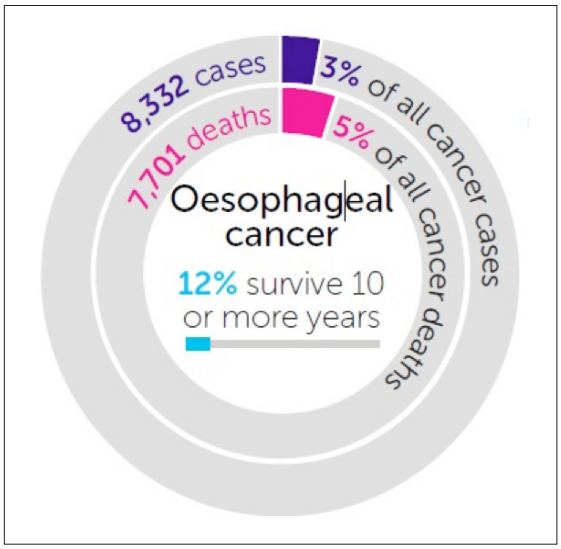


Figure 4: Key Stats oesophageal cancer in UK(Cancer Research UK).

Eight in ten oesophageal cancers occur in people aged 60 years-old and over. The overall 5-year survival for patients with oesophageal cancer is 15% (2).

There are two main types of oesophageal cancer: squamous cell carcinoma of the oesophagus (SCC) and adenocarcinoma (AC). SCC mainly occurs in the upper and middle third of the oesophagus while AC affects the lower third.

In England, AC accounts for the majority (70%) of oesophageal cancer, with a male preponderance (3). Risk factors for AC includes gastro-oesophageal reflux disease, Barrett's metaplasia, obesity, smoking and white ethnicity and smoking, alcohol, poor nutrition, ionizing radiation for SCC.

This article aims to educate the junior doctors by providing a broad overview of the management of oesophageal cancer.

The case presented is typical of a patient with oesophageal cancer. In the UK, approximately 70% of oesophageal cases are advanced oesophageal cancer as the symptoms are nonspecific in early stages. Patients may have a long-standing history of recurrent reflux symptoms and dyspepsia.

This may progress to worsening difficulty in swallowing, regurgitation, vomiting, and weight loss as the tumour becomes more advanced. Signs of locally advanced oesophageal cancer can include hoarseness of voice due to recurrent laryngeal nerve palsy, Horner's syndrome, palpable supraclavicular lymph node (Virchow's node) and rarely, broncho-oesophageal fistula.

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Assessment and Staging

The initial investigation of choice is an upper GI endoscopy. This provides direct visualization of the oesophagus and stomach, assessment of the oesophageal tumour and allows biopsies of the oesophageal cancer. In this case however, the patient was unable to tolerate the upper GI endoscopy initially and therefore, a barium contrast study was performed which showed irregularity of oesophageal lumen and shouldered margins. However, contrast study is not as sensitive as upper GI endoscopy and can miss early oesophageal cancer, and it should not be first choice of investigation for patient complaining of dysphagia.

The oesophageal cancer can spread directly to the surrounding organs such as the lungs, pericardium, bronchus and aorta, via lymphatics, and through haematogenous route to distant organs. The most common metastatic sites are non-regional lymph nodes, liver, lung, bone and brain. Tumour arising from the intra-abdominal part of the oesophagus may disseminate to the peritoneum.

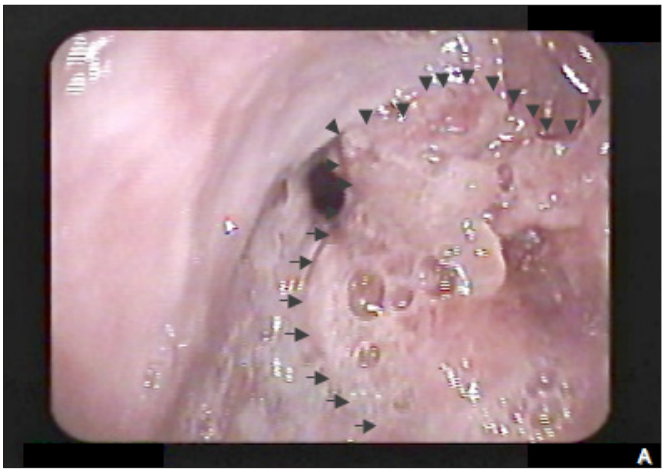
A CT scan of the chest, abdomen and pelvis will aid in delineating the local extent of the oesophageal tumour, the presence or absence of regional lymph node involvement and assessment of potential distant metastasis. This, in turn, will determine whether the oesophageal cancer is potentially resectable or not.

After confirmation of oesophageal cancer, specialist should assess patient for fitness for treatment. This includes assessment of patient's nutritional status, performance status and comorbidities. The curative option is either radical surgery or chemo-radiotherapy. Patients who are unfit for radical treatment are referred for palliative care and do not require any further investigations.

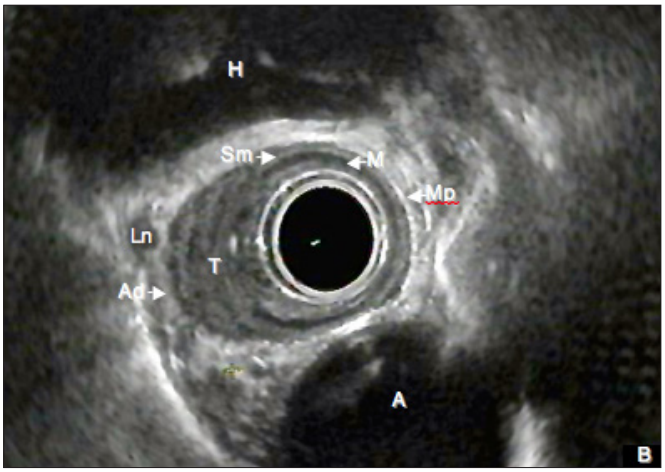
Patients who are fit for radical treatment should have further staging investigations. This includes PET scan to rule out distant metastasis (M stage), and endoscopic ultrasound (EUS) and laparoscopy to assess loco-regional stage (T and N). Laparoscopy is only indicated in patients with tumour extending below the diaphragm.

Compared to CT scan, EUS has a better accuracy in assessing the depth of tumour invasion and regional lymph nodes (4), (Figure 5). It is used to differentiate between early oesophageal cancer (T1/T2, N0) and locally advanced cancer (T3, T4a, and N1).

Figure 5: Endoscopic ultrasound of an oesophageal cancer.



A: A endoscopic screen shot of a bulky eccentric GOJ tumour outlined by arrows.



B: Corresponding EUS of the same oesophageal cancer (T) invading the mucosa (M), submucosa (Sm) and muscularis propria (Mp). It has not invaded the adventitia of the oesophagus (Ad). Note the proximity of the tumour to the heart (H) and aorta (A). An enlarged lymph node (LN) was noted.

As mentioned, PET scan is performed primarily to assess for presence or absence of distant metastasis and is more sensitive than CT in detecting distant metastasis. PET uses a radiotracer to assess for increased metabolic activity of cancer cells.

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Most commonly used radiotracer is fluorodeoxyglucose (18FDG); a glucose analogue labelled with fluorine-18. Tissue concentration of 18FDG increases with increased metabolic activity and therefore, 'highlighting' the metabolic active oesophageal cancer as well as any metastatic disease. In this case, an enlarged gastro-hepatic lymph node seen on the CT scan along with the oesophageal cancer found to be 18FDG-avid on PET-CT.

Treatment

All oesophageal cancer cases are reviewed in the MDT and recommended treatment option is dependent on the findings of the staging investigations and patient's fitness for treatment. The early oesophageal cancers (T1, T2, and N0) have single modality treatment in the form of surgery or radical chemo radiotherapy while locally advanced cancers (T1-T3, N1, N2) require multimodality approach with combination chemo/radiotherapy and surgery. The commonly used TNM 7 classification (5) summarized in Figure 6.

TNM for Cancer of Oesophagus

Tis: High-grade dysplasia
T1: Tumour invading lamina propria]T1a] or submucosa] T1b]
T2: Tumour invading muscularis propria
T3: Tumour invading beyond muscularis propria
T4a: Tumour invading adjacent structures (pleura, pericardium,diaphragm)
T4b: Tumour invading adjacent structures (trachea, bone, aorta)
N0: No lymph node metastases
N1: Lymph node metastases in 1–2 nodes
N2: Lymph nodes metastases in 3–6 nodes
N3: Lymph node metastases in 7 or more lymph nodes
M0: No distant metastases
M1: All other distant metastases

Staging for Cancer of Oesophagus

Stage 0: Tis N0 M0 — *Early Cancer*
Stage 1A: T1N0M0; Stage 1B: T2N0M0
Stage 2A: T3N0M0; Stage 2B:T1/2N1M0 — *Locally advance Cancer*
Stage3A: T4aN0M0, T3N1M0, Stage3B: T1/2N2M0; 3C: T3N2M0
Stage 4: T4aN1/2M0, T4bN0-3M0, Any T, N3M0; 4: Any T, Any N, M1 — *For palliative treatment*

Figure 6: TNM Staging for Cancer of Oesophagus (TNM7, AJCC). TNM for Cancer of Oesophagus

Neo-adjuvant therapy

Neo-adjuvant therapy is indicated for patients with locally advanced oesophageal cancer or with regional lymph node involvement. This will either be a regime of chemotherapy or a combination of chemo- and radio-therapy; followed with surgery.

Surgery

The aim of the surgery is for a 'curative' resection; resecting the segment of oesophagus containing the oesophageal cancer with clear margins and removing the lymph nodes draining the cancer.

The commonly performed operations for oesophageal cancers are:

- **two stage Ivor Lewis oesophago-gastrectomy**
- **transhiatal oesophago-gastrectomy**
- **three stage McKeown oesophago-gastrectomy**

In most UK institutions, two stage Ivor Lewis oesophago-gastrectomy is commonly performed for oesophageal cancer. The first stage involves a midline abdominal laparotomy to mobilise the stomach, excising the regional lymph nodes and dissecting the distal oesophagus. DDuring the stomach mobilisation, the left gastric artery, right gastric, left-gastro-epiploic and short gastric arteries are divided, leaving just the right gastro-epiploic artery as the main blood supply to the stomach.

For the second stage, the patient is placed in a left lateral position. An anterolateral thoracotomy is performed to gain access into the mediastinum. The right lung is then collapsed and the oesophagus dissected. In the chest, the oesophagus is in close proximity to the aorta, the pericardium, the pulmonary arteries and the main bronchi and therefore, careful dissection is vital to avoid damaging any of these structures. The oesophagus is normally divided 8-10 cm proximal to the tumour to ensure proximal margin clearance. The stomach is pulled up into the posterior mediastinum and the proximal stomach is divided using a linear stapler. This forms the distal margin of the oesophago-gastrectomy specimen (Figure 7).

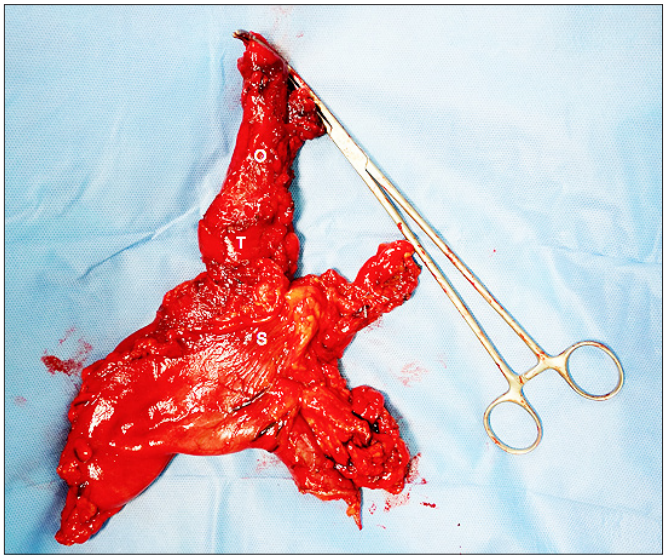

Figure 7: An oesophago-gastrectomy specimen with distal oesophageal cancer (T). Oesophagus (O) was divided approximately 8 cm proximal to the tumour. The proximal stomach (S) forming the distal margin, was excised from the level of the angularis incisura to the gastric fundus.

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Oesophago-gastric anastomosis can be performed by hand sewn single layer or by a circular stapling device to create an end-to-side anastomosis. The colon or the jejunum can also be used as an alternative conduit between the oesophagus and stomach.

In this case, history, the oesophageal dissection was challenging due to the bulky tumour invading the right pleural (T4a) and thickened fibrotic tissue due to neo-adjuvant chemo radiotherapy.

Minimally invasive & hybrid oesophago-gastrectomy

Minimally invasive oesophago-gastrectomy involves laparoscopic mobilisation of stomach and thoracoscopic resection of oesophagus. It is a technically demanding procedure but has been shown to have comparable results to open oesophago-gastrectomy in terms of oncological resection and survival (6). Another option is for hybrid oesophago-gastrectomy where the abdominal stage performed laparoscopically and an open thoracotomy for the thoracic stage. Both approaches are increasingly in use throughout UK although open oesophago-gastrectomy remains the most common operation for oesophageal cancer.

Endoscopic Mucosal Resection

This is an endoscopic technique pioneered in Japan, which is used to resect early oesophageal cancer (Tis, T1) where the tumour is confined to the mucosa or submucosa (7). In addition, the resected specimen allows an accurate assessment of the depth of tumour invasion, differentiation and presence or absence of lympho-vascular invasion. It can be curative treatment for Tis and T1a cancer followed by radio frequency ablation if Barrett's metaplasia is present. It is increasingly in use in UK for treatment of early cancer.

Palliative Therapy

Patients with stage IV cancer referred for palliative therapy. The options include chemotherapy or radiotherapy. Endoluminal stent insertion carried out to palliate dysphagia. This can be performed either under radiological or endoscopic guidance. The 5-year survival for stage IV disease is 4% but most patients have progressive disease and average survival is 12 months.

Summary

Cancer of oesophagus is difficult to diagnose in early stage. The surveillance for precancerous condition of Barrett's metaplasia can improve survival in small subgroup of patients. However, recent technological advancements and evolving treatments allow an accurate assessment of the tumour burden and tailor the treatment to individual patients with the aim of improving survival rates.

Questions

1. Regarding the indications for urgent upper GI endoscopy, which statement is false:

a) Patients of any age with progressive dysphagia

b) Patients of any age with weight loss and reflux symptoms

c) Patients over 55 years old with persistent reflux

d) Patients under 30 years old with occasional dyspepsia only

e) Patients with a palpable epigastric mass

2. Please select one or more statements about Barrett's metaplasia which is/are true:

a) Barrett's metaplasia involves the change of oesophageal mucosa from squamous cell to columnar type lining.

b) Barrett's metaplasia typically occur in the middle third of the oesophagus

c) An upper GI endoscopy diagnosis and histological confirmation are required for the diagnosis of Barrett's oesophagus

d) Patients with Barrett's oesophagus will require regular surveillance

e) There is no increased risk of developing oesophageal cancer in patients with Barrett's oesophagus.

3. A patient with oesophageal cancer underwent oesophago-gastrectomy. Histology showed no involved lymph nodes with the tumour invading the left pleural. Pre-operative CT and PET-CT scan did not show any distant metastasis. This is a:

a) T1N2M0

b) T3N0M0

c) T4N0M0

d) T4N1M0

e) T2N0M0

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4. Please select one or more blood supply to the stomach:

a) Splenic artery

b) Common hepatic artery

c) Superior mesenteric artery

d) Left hepatic artery

e) Coeliac artery

5. Complications can occur following oesophago-gastrectomy. Please select the most common immediate post-operative complication.

a) Anastomotic leak

b) Gastric conduit necrosis

c) Chylothorax

d) Infection

e) Small bowel necrosis

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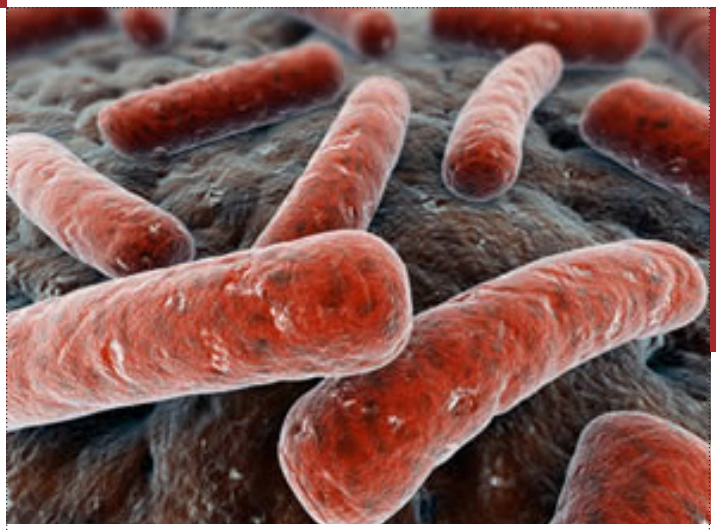
NECROTISING FASCIITIS

H Ewan, M Thornton



Abstract

Necrotising fasciitis is a life-threatening soft tissue infection that can be a diagnostic challenge due to a lack of specific signs. An awareness of the clinical features and diagnosis are vital for foundation doctors who are often the first doctor to assess patients being admitted to hospital. Early diagnosis through having a high index of suspicion and early surgical debridement of necrotic tissues are key and can be life saving. This case based discussion will highlight these aspects in detail to give the reader more confidence in managing these uncommon but difficult cases.

Case History

AA, a 42 year old male was admitted with a 2 week history of left groin and thigh pain. He was an intravenous drug user. He had last injected in this groin 2 weeks prior to his presentation. His GP had prescribed flucloxacillin but his symptoms of pain and swelling were worse. He did not have any symptoms of systemic illness.

Background

Necrotising fasciitis is a potentially fatal rare soft tissue infection characterized by rapidly spreading inflammation and necrosis of the soft tissue and fascia, sometimes involving muscles and skin, leading to severe systemic toxicity. It was first named by Wilson in 1952 but has other names include progressive bacterial synergistic gangrene, flesh-eating bug, hospital gangrene, gas gangrene, Fournier's gangrene and can be summed up by the term necrotising soft tissue infections. (1)

There are around 500 cases a year in the United Kingdom. (2) Its rarity and lack of pathognomonic sign make it difficult to diagnose but an increased awareness of the condition allows for a better detection rate by doctors. It can occur after a history of minor injuries such as cuts, injection sites, insect bites, burns, puncture wound and surgical procedures. However more than 45% occur without any preceding trauma. (1)

Necrotising Fasciitis
Patient Management

Risk factors include immunosuppression, obesity, peripheral vascular disease, intravenous drug abuse, alcohol abuse, chronic cardiac disease, cancer and age. The most common co-morbidities associated include diabetes mellitus, chronic hepatitis, cirrhosis and malignancy.

Necrotising fasciitis can also be classified based according to its microbiology. Type 1 infections are polymicrobial and more common. These yield both aerobic and anerobic organisms and are typically found in the perineum and trunk and more common in the immunocompromised and diabetic. Type 2 infections are monomicrobial and due to haemolytic group A streptococcus. These are less common, mostly occurring on limbs in healthy individuals.

Clinical Features

On examination AA was pyrexial and tachycardic. He had an injection scar in his left groin with a small amount of erythema and surrounding oedema. He was markedly tender on palpation over his anterior thigh. There was no sensory loss but he was unable to mobilise due to the pain.

The diagnosis is primarily based on the clinical findings. Early diagnosis is vital and requires a high degree of suspicion because it can often be difficult to distinguish from other soft tissue infections such as cellulitis or abscess. Initially, patient symptoms are non-specific with fever and pain.

Classical skin changes of bullae, vesicles and necrosis are seen in less than half of patients and often only after day 5 or later. Patients often seem systemically well and subsequently rapidly deteriorate with septicaemia, shock and confusion.

Clinical characteristics have been classified into 3 stages. (3)

Stage 1 (early)	Stage 2 (intermediate)	Stage 3 (late)
Tender on palpation	Blister or bullae	Crepitus
Erythema	formation	Skin anaesthesia
Swelling	Skin fluctuance	Skin necrosis – dusky
Calor		

Table showing the clinical characteristics of necrotising fasciitis in 3 stages from early to late. (3)

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Although it can often difficult to distinguish from other soft-tissue infections, there are some helpful indicators:

- *Disproportionate pain out with expected for clinical findings (most sensitive).*
- *Poorly defined and indistinct margins.*
- *Tenderness beyond the involved area.*
- *Sensory loss to overlying skin.*
- *Rapidly progressing infection despite antibiotics.*

Investigations

No investigations are diagnostic. Laboratory findings are sensitive but not specific. Patients often have high white blood cell count and low serum sodium levels as well as other deranged physiology. The most accurate diagnostic scoring system is the laboratory risk indicator for necrotizing fasciitis score (LRINEC.)

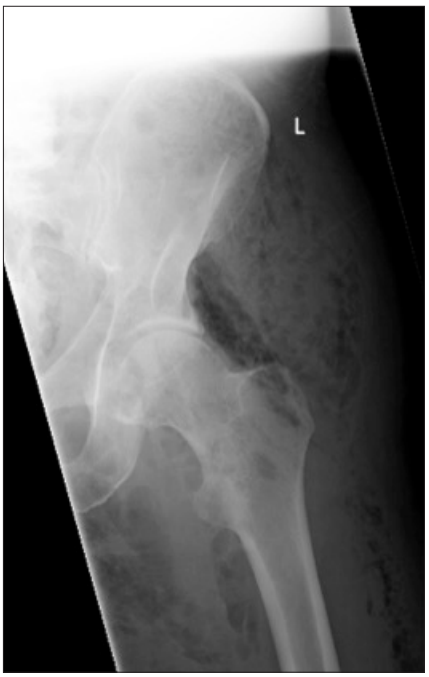
This uses 6 independent laboratory parameters (C-reactive protein, white cell count, haemoglobin, sodium, creatinine, glucose) and categorizes patients into low, intermediate or high risk, with the probability of necrotising fasciitis increasing from 50 to 75% respectively. This was devised to stratify the likelihood of infection in patients in Singapore, where a score of 6 is 93% sensitive and 92% specific in their population. (2,4) Correlation and validation in the UK is not as high as it is not routinely used in clinical practice.

In practice patients have a high C-reactive protein relatively early, reflecting the systemic inflammatory response. This is often associated with a high white blood cell count. Hyponatraemia in these patients is highly suspicious for a necrotising soft tissue infection.(2) It is important to ensure patients undergo glucose testing as many patients may have undiagnosed diabetes. Blood cultures should be taken to guide further antibiotic management, but take too long to influence immediate management.

Radiologic evaluation has been used to assist in diagnosis. Plain radiography can detect subcutaneous gas although this is rare. Computed tomography (CT) scans may also detect an increased attenuation of the subcutaneous fat with stranding, fascial swelling, inflammation and soft tissue gas. Magnetic resonance imaging (MRI) has a high sensitivity in diagnosis looking for liquefactive tissue necrosis and fascial fluid but its availability and feasibility limits its use.(2)

On admission, AA had a white cell count of 10.3 x10^9/l, C-reactive protein of 381 mg/l and a serum sodium of 126 mEq/l. The rest of his bloods were within normal parameters. He was started on intravenous antibiotics. He initially had an ultrasound of his left groin to rule out a pseudoaneurysm that showed inflammatory change around the soft tissues.

The following day he underwent further investigations. Hip x-ray showed extensive gas in soft tissue extending into his mid thigh. Subsequently, a computed tomography (CT) scan of his pelvis and upper thigh also showed extensive gas in the soft tissues of thigh, buttock and groin.



Hip x-ray

Management

Early surgical debridement is the only treatment option and can be lifesaving. The decision to surgically explore the soft tissues should be made early. Surgical incision reveals grey necrotic tissue, lack of dermal bleeding, lack of resistance on blunt dissection, loss of skeletal muscle contraction on stimulation and the classical 'dishwater' pus.

Necrotic fascia loses its adherence to surrounding tissues and therefore surgical planes open abnormally easily facilitating the 'finger sweep test'. Debridement is carried out until there is no dubiety that the tissue is healthy as evidenced by bleeding, muscle contraction and absence of the signs described above.

Multiple tissue specimens should be sent for microbiology to guide further treatment. Those patients with early exploration often require less extensive resections and reduced morbidity compared to those with later first debridement.(2) Most require a second debridement at 24-48 hours.

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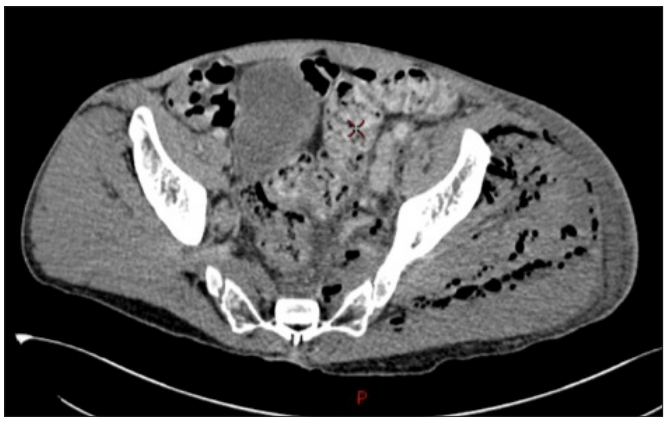
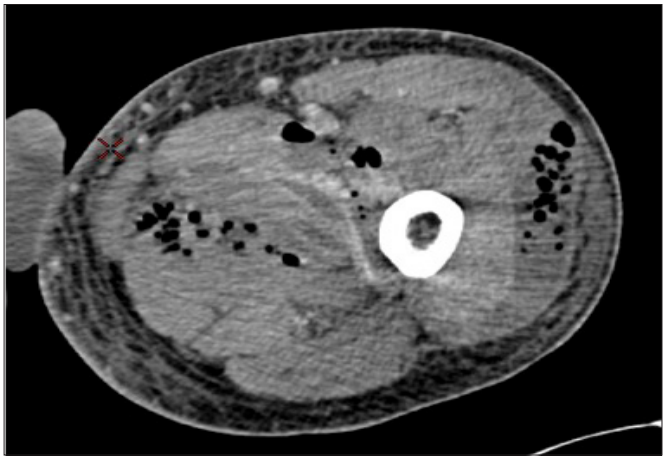


Figure 1: Hip x-ray showing extensive gas in soft tissue overlying left leg extending into mid thigh.

Patients often require systemic support in High Dependency or Intensive Care. Multi-organ failure is common and rapidly progressive. Early involvement of the microbiologist is also imperative. Necrotising fasciitis is most often caused by mixed pathogens requiring broad spectrum antibiotics according to local guidelines, blood and wound culture results. Repeat cultures should be taken at each dressing change or septic episode.

The use of IV immunoglobulins is controversial and there have been no randomised controlled trials. (4) The theory is that they block T cell activation of streptococcal and staphylococcal superantigens. Once systemically recovered, with negative wound cultures, many patients will require referral to plastic surgery unit for wound reconstruction in the form of skin grafts.

AA was taken to theatre on day 2 of admission for surgical debridement and required fasciotomies. He required a return to theatre on day 4 and day 7 for further debridement. After 15 days he had made a good recovery and was transferred to a regional plastic surgery unit for ongoing management and reconstruction.



Figures 2 and 3: CT of pelvis and left leg scan showing extensive gas in soft tissues of thigh, gluteal muscles and left groin suggestive of necrotising fasciitis.

Learning points

Foundation doctors are often the first to assess patients on admission to hospital and as such have an important role in recognising this uncommon but life-threatening condition. Early diagnosis is key and requires a high index of suspicion to distinguish necrotising fasciitis from other soft tissue infections.

AA had several risk factors that should make you consider this diagnosis: an intravenous drug user, unable to walk due to pain (out with the expected), was not responding to antibiotics, high C-RP and low serum sodium.

Glucose testing is also important to detect undiagnosed diabetes. Although his investigations were helpful in assisting with diagnosis it is important to remember that clinical diagnosis alone is sufficient. Surgical intervention is the only treatment option and perhaps should have been performed earlier in this case.

A multi-disciplinary approach is required with collaboration from microbiology to provide up to date sensitivities and advice, anaesthetists to provide higher support in a critical care environment and often partnership with plastic surgery for reconstruction following recovery.

MCQ'S

1. All of the below are risk factors for necrotising fasciitis except:

A: Trauma
B: Surgical wounds
C: Immunocompromise
D: Obesity
E: Male gender

2. The diagnosis of necrotising fasciitis is based predominately on:

A: Clinical picture
B: Blood results
C: X-ray
D: Ultrasound
E: Computed tomography

3. What is the most definitive aspect of management in the treatment for necrotising fasciitis:

A: Fluid resuscitation
B: Surgical debridement
C: Antibiotics
D: Analgesia
E: Taking blood cultures

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4. The mortality of necrotising fasciitis is:

A: 0-20%
B: 20-40%
C: 40-60%
D: 60-80%
E: 80-100%

5. Appropriate antibiotics for the treatment of necrotising fasciitis include:

A: Flucloxacillin and Benzylpenicillin
B: Gentamicin
C: Clindamycin
D: Metronidazole
E: All of the above

Answers

1. Figure 1E

Gender is not a well recognised risk factor for necrotising fasciitis. Risk factors include obesity, intravenous drug use, peripheral vascular disease, immunosuppression, cancer, age, smoking and alcohol abuse. Typically a history of minor injury or trauma to the site occurs but it is important to remember that in around 45% of cases, no definitive causative factor can be found – these are known as idiopathic necrotising fasciitis.

2. Figure 2A

Early clinical diagnosis is the most important factor in improving patient outcome. Diagnosis should be based on clinical findings rather than the use of radiological imaging which although helpful is not necessary.

3. Figure 3B

Although each of these are part of the management for necrotising fasciitis, early surgical debridement is the only definitive treatment and patients will not start to recover until this has been carried out.

4. Figure 4B

Even with surgery the mortality is 20-40%.(2) Delay in diagnosis increases mortality.

5. Figure 5E

A broad spectrum of antibiotics is initially required to cover polymicrobial infections involved in necrotising fasciitis. Local antimicrobial guidelines should be followed and early liaison with a microbiologist.

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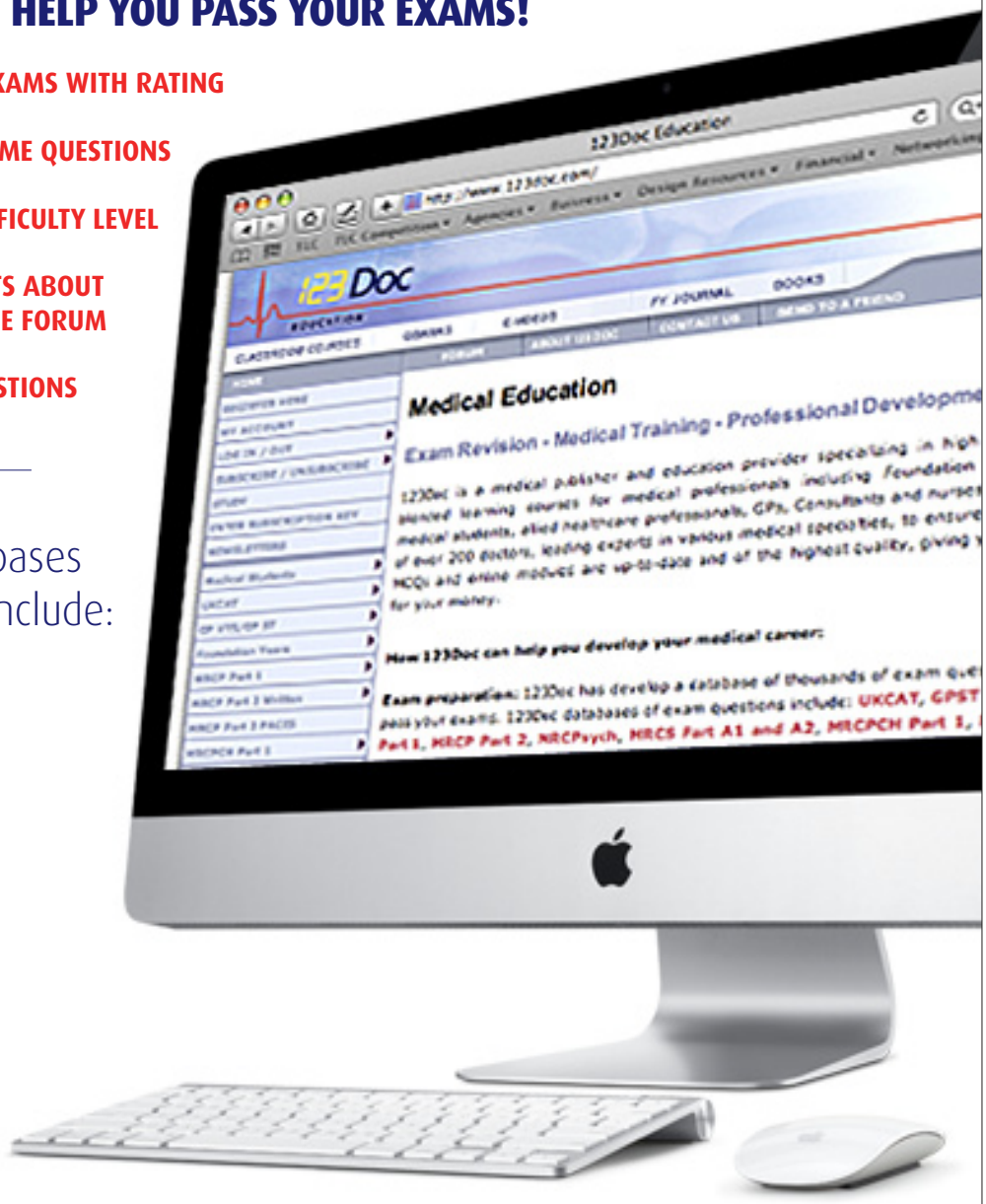

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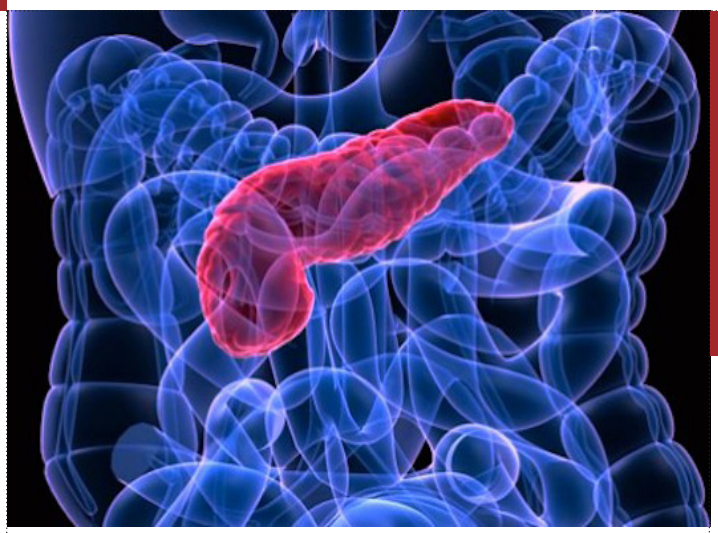
ACUTE PANCREATITIS: A REVIEW FOR THE FOUNDATION DOCTOR

C Uzoho, S Hann, R Boulton, M Saunders



Abstract

Acute pancreatitis commonly presents on the acute surgical take with an incidence of 56.5/100,000 people in the UK, and a predicted mortality of 5% (1). Effective assessment and early management of patients with pancreatitis is crucial to prevent complications and prolonged hospital admission. This paper focuses on the assessment and management of acute pancreatitis derived from current evidence-based guidelines. We will highlight pertinent aspects of the history and examination, aetiological factors, investigations and scoring systems and finally critical aspects of early management that will aid in improving morbidity and mortality.

Diagnosing acute pancreatitis

The diagnosis of acute pancreatitis relies not only on clinical assessment, but must be supported by biochemical or radiological evidence, meeting two of the following citeria (2):

1) History – acute onset of severe, persistent epigastric pain, often radiating to the back (Table 1)

2) Biochemical markers – elevation amylase three times the upper limit of normal

3) Radiological evidence of acute pancreatitis – Ultrasound (USS), Computer Tomography (CT) and Magnetic Resonance Imaging (MRI).

Acute Pancreatitis: A Review For The Foundation Doctor
Patient Management

Assessing the patient

Site	Upper abdominal pain, usually epigastric, however can be generalised with peritonism
Onset	Sudden onset, particularly in gallstone pancreatitis, following a large meal. In alcohol-induced pancreatitis onset is often more gradual, and may follow an episode of heavy alcohol consumption[3]
Character	Commonly severe and described as 'gnawing' in nature
Radiation	Typically radiates to the back, however can extend to the right upper quadrant, chest or flanks
Associated features	It is important to enquire about nausea and vomiting, fever and change in bowel habit. Nausea and vomiting affect approximately 90% of patients with acute pancreatitis[4]. Uncommonly, patients can experience shortness of breath secondary to diaphragmatic inflammation
Timing	Pain is usually continuous
Exacerbating/ relieving factors	Worse on supine position and relieved by sitting upright or leaning forward
Severity	Typically reaches a peak within the first few hours, and then plateaus thereafter; this can last for several days following an attack [5, 6]

Table 1: SOCRATES acronym for clinical history taking.

How can physical examination aid in diagnosing acute pancreatitis?

The purpose of clinical examination in suspected acute pancreatitis is two-fold: it can assist in making a diagnosis, and can give some indication as to the severity of the condition.

Depending on severity the abdomen may be mildly tender or peritonitic with guarding and rebound tenderness. Bowel sounds may be reduced with distension secondary to inflammation-induced ileus (7).

Grey-Turner sign (flank discolouration) and Cullen's sign (umbilical discolouration) are two eponymous signs indicative of retroperitoneal haemorrhage in the setting of pancreatic necrosis. Although classically associated with pancreatitis, they are only present in 3% of cases (late signs/ complications). (8)

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A full clinical examination should include a review of the patient's physical observations; fever and tachycardia are common.

It may be possible to establish aetiological clues from examination e.g. spider naevi and hepatomegaly in alcoholism, xanthomas in hyperlipiaemia and parotid swelling in mumps.

Biochemical investigations

Which investigations are required in patients presenting with suspected pancreatitis?

Routine blood tests include a full blood count, urea and electrolytes, liver function tests, lactate dehydrogenase, pancreatic enzymes (amylase and lipase), inflammatory markers, bone profile (calcium), clotting and albumin. A sample should also be sent for blood typing (Group and Save).

An arterial blood gas sample is essential as it can show early signs of Multi-organ failure and useful in accurately scoring the severity (depending on scoring system, see below). A urine sample is a useful baseline test, and in a woman of child-bearing age, a pregnancy test is mandatory as amylase can be raised in the presence of ectopic pregnancy.

Biochemically the most common investigation for acute pancreatitis is serum amylase. This rises within six to twelve hours of onset of acute pancreatitis, and normally falls after 24-48 hours (9). This is clinically significant in cases of delayed presentation, as amylase may be normal outside of this window. Furthermore, serum amylase is not specific to pancreatitis and can be raised in various intra-abdominal pathologies, such as ruptured abdominal aortic aneurysms and perforated viscus. Remember that patients who have recurrent attacks of pancreatitis (usually alcohol-related) may not develop a significant amylasemia due to chronic pancreatic damage.

Radiological investigations

In the case reported above, the diagnosis of acute pancreatitis was made from the history and raised (amylase). Further imaging is not needed to confirm the diagnosis, but is used to determine the aetiology, severity and presence of complications.

Abdominal and chest radiographs (AXR and CXR) are often unremarkable in mild disease, however, in moderate to severe disease you may see evidence of localised ileus of a segment of small intestine (sentinal loop) on AXR. (10) Up to one third of these patients have CXR findings, such as pleural effusions, basal atelectasis and pulmonary infiltrates. Presence of these findings in the first twenty-four hours suggests necrosis and organ failure. (10) Early CXR will also exclude the important differential of perforated viscus.

USS in the context of acute pancreatitis is useful for identifying underlying gallstones and common bile duct dilatation. The pancreas will appear diffusely enlarged, however, in many cases is made difficult to visualise by obscuring bowel gas in the context of paralytic ileus. USS is limited in that it cannot clearly visualise necrosis or the extra-pancreatitic extension of inflammation. (11)

Contrast-enhanced CT has over a 90% sensitivity in diagnosing acute pancreatitis. It will usually show focal or diffuse enlargement of the pancreas, and importantly can confirm both the presence and extent of necrosis, local complications and also predict disease severity (Balthazar Scoring System). A CT is not always requested immediately, particularly if the diagnosis has already been confirmed, but reserved to identify potential complications if the patient's clinical condition deteriorates.

Magnetic resonance cholangio-pancreatography (MRCP) when compared with CT is better at characterising the pancreas, bile ducts and local complications in acute pancreatitis. (12) MRCP is reported to be comparable to endoscopic retrograde cholangio-pancreatography (ERCP) in the detection of gallstones and has the advantage of being non-invasive with a reduced risk of nephrotoxicity and radiation compared to CT. (12) MRCP does however, have a longer scanning time, consequently it is less often used in the acute setting.

Causes of acute pancreatitis

The acronym that most juniors will be able to reel off and keep in the back of their minds when thinking of the aetiology is 'GET SMASHED'. It is important to remember that Gallstones and Alcohol remain the two commonest causes of Acute Pancreatitis (Table 2).

G	**Gallstones**
E	**Ethanol**
T	**Trauma**
S	**Steroids**
M	**Mumps**
A	**Autoimmune**
S	**Scorpian bite**
H	**Hyperlipidaemia/ Hypercalcaemia/ Hypothermia**
E	**ERCP**
D	**Drugs**

Table 2: GET SMASHED acronym for causes of Acute Pancreatitis.

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Determining the severity of pancreatitis

Various scoring systems have been developed to predict severity in acute pancreatitis and therefore the risk of end organ failure. These systems are fundamental in identifying patients requiring early involvement of Intensive Care, thus reducing morbidity and mortality. Commonly used scoring systems are the APACHE II, Ranson, Balthazar (CT index score) and Modified Imrie/ Glasgow Score (known best to the on-call foundation doctor by the acronym PANCREAS (Table 1). (13)

A modified Glasgow score of 3 or more, an APACHE II score of 5 or greater and a Ranson score of 3 or more equates to severe pancreatitis and indicates a need for early intensive care involvement. (13) CRP has alone been identified as an independant prognostic factor, particularly alter 48 hours. (14)

P	paO2 (60mmHg or 7.9kPa)
A	A – Age (>55)
N	Neutrophils (WCC >15)
C	Calcium (<2)
R	Renal (Urea >16)
E	Enzymes (Lactate Dehydrogenase >600)
A	Albumin (<32)
S	Sugar (Glucose >10)

Table 3: Modified Glasgow Scoring System.

Initial management of acute pancreatitis

Initial management involves an ABCDE approach, in order to stabilise the patient and assess for signs of shock. In relation to pancreatitis specifically, this should include the following:

- **Oxygen and Analgesia**
- **IV fluid replacement and catheterisation**
- **Monitoring for deterioration or complications**
- **Nutrition**
- **Antibiotics (if indicated)**
- **Early ERCP (if gallstone pancreatitis with cholangitis / CBD calculi)**

A scoring system (see above) is useful in predicting the severity of pancreatitis and need for early referral to the Intensive Care Unit (ICU) for close monitoring and organ support. (15)

IV fluid replacement

Current evidence indicates that appropriate fluid resuscitation can reduce both morbidity and mortality in acute pancreatitis by reducing the chances of multi-organ dysfunction. (16) Fluid resuscitation is particularly important within the first 24 hours of management.

The British Society of Gastroenterology have set guidelines for acute pancreatitis which suggest aggressive fluid management with crystalloids or colloids to achieve and maintain an urine output >0.5 ml/kg of body weight. (16) Fluid requirements should be adjusted at regular intervals, using clinical assessment, laboratory blood testing including urea and haematocrit, and monitoring urine output. Inadequate fluid resuscitation can lead to hypotension, acute tubular necrosis and the development of necrotising pancreatitis. (17)

Monitoring

Alongside fluid status and urine output, accurate monitoring of patients includes various other indicators: oxygen saturations, electrolytes and serum glucose levels. If there are concerns regarding oxygenation, an arterial blood sample can aid prediction of severity, and also diagnosis. Hypoxia may be secondary to a number of respiratory conditions, such as splinting of the diaphragm, atelectasis and acute respiratory distress syndrome (ARDS). Such patients may also require ventilatory support, most likely in an intensive care setting.

Electrolytes, particularly calcium and magnesium should be replaced where necessary; this is especially necessary with aggressive fluid resuscitation. Furthermore hyperglycaemia has been linked to an increased risk of secondary pancreatic infection. (18)

Nutrition

We have included an evidence-based overview of the common practices for managing patients' nutritional requirements in pancreatitis. However each trust will have its own locally agreed policy.

Patients with mild pancreatitis can often be managed with intravenous fluids only and early introduce of an oral diet in the subsequent few days since the inflammation settles quickly. In the absence of an ileus, nausea or vomiting a low residue, low fat diet can be started once the pain and inflammatory markers are improving (15). Recent studies suggest early refeeding is safe if it is tolerated and the patient is hungry, irrespective of pain or biochemical markers. (20, 21)

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Oral feeding is not usually tolerated in patients with moderate to severe pancreatitis due to nausea and vomiting or post-prandial pain as a result of gastroduodenal inflammation +/- fluid collections causing extrinsic compression and possible gastric outlet obstruction. In these patients it is ideal to commence enteral feeding via either the nasogastric (NG) or nasojejunal (NJ) routes (recent trials show no significant difference between them). (22, 23) Parental nutrition should only be used for patients not tolerating enteral feed, as this has been shown to increase mortality when used alone or as an adjunct to enteral feed. (24)

Enteral feeding is thought to maintain the intestinal barrier and reduce bacterial translocation from the gut. It is usually commenced after 48 hours when it becomes clear a patient will not tolerate oral nutrition, as there is little evidence to support a reduction in infection rates with early enteral feed (24-48 hours). (25)

Antibiotics

There is currently no consensus on the use of prophylactic antibiotics in pancreatitis, however, they can be used if an infection is suspected and in these cases broad spectrum antibiotics can be used until cultures are obtained to allow targeted antimicrobial therapy. Up to 20% of patients will develop extrapancreatic infections, with increased mortality (26).

Necrotizing pancreatitis is a recognised complication of pancreatitis. The necrotic collection is initially sterile, but can become infected in one third of patients. If suspected, early involvement of your seniors and discussion with a tertiary specialist centre is advised. The patient may require CT-guided fine needle aspiration for Gram stain and culture for targeted antimicrobial therapy or empirical antibiotics.

Endoscopic retrograde cholangiopancreatography (ERCP)

ECRP should be performed within the first 24 hours for patients with gallstone pancreatitis and cholangitis to relieve biliary obstruction and remove the gallstone. It is however, not indicated in patients with mild or severe gallstone pancreatitis without cholangitis. (27) If any doubt exists, consult with your seniors and discuss with your local interventional endoscopist. The British Society of Gastroenterology recommend patients with mild gallstone pancreatitis have definitive treatment (open or laparoscopic cholecystectomy) within two weeks or ideally before discharge. (16) In those with severe gallstone pancreatitis it is recommended that definitive surgical management is postponed until they have recovered. (16)

Systems	Complications
Local	Necrosis, haemorrhage, abscess, pseudocyst (late sign)
Pulmonary	Pneumonia with hypoxemia or/and hypoxemia, ARDS
Cardiovascular	Hypotension requiring pressure support, myocardial infarction, arrhythmias
Infection	Sepsis of any source
Renal	Acute kidney disease
Haematological	Disseminated intravascular coagulation
Neurological	Reduced Glasgow coma scale
Gastrointestinal	Paralytic Ileus

Table 4: Complications of acute pancreatitis.

Conclusion

The skills used in the assessment and management of acute pancreatitis are transferable to all critically ill patients. A thorough history and examination encompassing an 'ABCDE' approach forms the foundation of clinical assessment, enabling one to differentiate pancreatitis from other causes of abdominal pain.

As most admitting doctors will now know, patients are not always as forthcoming with relevant information as we would like and so you must be active in enquiring about risk factors, such as alcohol intake and medication. National guidelines are available through groups such as National Institute for Health and Care Excellence (NICE) and the British Society of Gastroenterologists (BSG), however, it is important to check local trust guidelines as there are several contentious areas in management of pancreatitis, such as antibiotic use and feeding protocols. One area that is uncontested is the need for aggressive fluid resuscitation. This is often an area where the foundation doctor can make most impact, particularly in the first 12-24 hours, and can help reduce complications arising from the condition. (28)

There are a number of possible complications: systemic conditions such as ARDS, or loco-regional complications such as pseudocyst formation, haemorrhage and necrosis of the pancreas (Table 4). Furthermore it is not uncommon for patients to experience multiple episodes of pancreatitis, which can ultimately progress to chronic pancreatic insufficiency.

In conclusion, a structured approach to dealing with acute pancreatitis can have a significant impact on patient mortality and morbidity; in addition the skills acquired through practice can aid in the management of other common surgical conditions that may present on the acute surgical take.

ACUTE PANCREATITIS: A REVIEW FOR THE FOUNDATION DOCTOR

C Uzoho, S Hann, R Boulton, M Saunders

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PREPARING FOR APPLICATION TO CORE SURGICAL TRAINING AS A FOUNDATION DOCTOR

G Slater, A Waghorn

Preparing for Application to Core Surgical Training as a Foundation Doctor
Teaching & Training

Introduction

To be successful in application to Core Surgical Training (CST) foundation doctors must have a comprehensive portfolio and be well prepared for interview. Applications open in November of Foundation Year 2 (FY2) year, so it is essential to begin early and to be familiar with the requirements for entry. In order to be scored for achievements such as examinations and courses you must have completed these and have portfolio evidence to bring to the interview. In 2015, these will be held from 26th January to 6th February (1).

The Core Surgery Recruitment for NHS (2) website is an excellent starting point. This is where you will find the person specification and an outline of how your portfolio will be scored. Points are available for additional degrees, clinical experience, teaching, CPD courses, research work, audit, leadership and commitment to surgery. (3) It is important to have evidence in each of these categories to score well in the short-listing of candidates. You may wish to consider preparing a draft application form early in your F1 year. This will allow you to identify areas of weakness which you will then have time to improve on.

Examinations

Once you have started work as an FY1, you are eligible to sit MRCS parts A and B. Completion of both parts is mandatory for progression to ST3 level, but sitting at least part A during foundation training helps to demonstrate your commitment to surgery. Part A is a multiple choice exam covering basic science, principles of surgery and speciality-specific clinical knowledge. The paper is sat 3 times per year (September, January and April) with the closing date for applications up to 3 months prior to the exam (4).

Part B is an OCSE style examination testing your recognition and management of clinical scenarios. Recently, the GMC announced that entry to ST3 must be no further than 7 years following completion of MRCS Part B, to ensure that trainees are up to date (5). This is worth bearing in mind if you are planning to take time out of training for research work or additional degrees. Post-graduate examinations are expensive (the current fee for Part A is £503) so careful financial planning is needed if you wish to sit these soon after graduation.

Courses

Courses such as Advanced Trauma Life Support (ATLS) and Basic Surgical Skills (BSS) are a good way to broaden your skills base. The Systematic Training in Acute Illness Recognition and Treatment for Surgery (START Surgery) is targeted to final year medical students and F1 doctors and teaches a systematic approach to the unwell surgical patient. Again, these courses can cost £500-£750 each but many deaneries provide a study budget to help trainees to pay for these.

Places on these courses are limited, and are often filled months in advance. BSS can be taken in FY1, but ATLS is only open to FY2 doctors and above (6), so plan accordingly. The Royal College of Surgeons also run courses aimed specifically at medical students (such as Basic Surgical Skills for Students), which can be good evidence of early commitment to surgery. After completing these courses, it is important to practice skills such as knot tying and suturing in your own time so that you feel confident when assisting in theatre.

Prizes

A minority of applicants will have won prizes, so if you have one it is essential to include it in your portfolio. There are a lot of relevant regional and national prizes available, with entry dates spread throughout the academic year. Your education centre will be able to provide the dates of regional meetings, many of which offer prizes for trainees. A list of essay prizes available for foundation trainees can be found on the Royal Society of Medicine and the Royal College of Surgeons careers websites (5).

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Presentations, posters and publications

Involvement in research work demonstrates that you are pro-active, can interpret and analyse data and think critically. It is not unusual for there to be a lag of months or years between starting a project and having a poster or even publication as evidence of your work, so it is important to start as early as possible. Maximum points are awarded for first author publications, or poster presentations at national and international conference. Choose your supervisor carefully; those who publish regularly will be better placed to help you design a project of appropriate scale and to guide you through the process of study design and peer review.

Audit

Completion of an audit or quality improvement project is a requirement for FY1 sign off. In order to stand out from other applicants, aim to complete a full audit cycle by implementing a change and re-auditing to assess its impact. This is easiest if you find a project early in FY1 in a department you will be able to return to easily. Again, choose an experienced supervisor who will help you to design a concise and relevant project.

Most audits can be completed within 3 months. Ideally, aim to complete 2 per year. It is also important to show you have presented your results to colleagues. Once you reach ST3 applications, you will be expected to show that you can design, implement and present an audit through a full cycle.

Teaching

Teaching juniors is an important skill in all medical specialities, and is also an effective way to consolidate your own knowledge of a subject. Ensure that you obtain evidence of all the teaching you complete throughout the year. For example, ask students to complete feedback forms (which can usually be obtained from your education centre) and reflect on their feedback in your eportfolio. Maximum points are awarded for organisation of a regional or national teaching.

There is scope to be very creative with this, for example by setting up a website or podcast on a surgical topic. Collaborating with other junior doctors on such projects can also provide evidence of management and leadership skills. If you have a special interest in medical education, you may wish to consider beginning a Postgraduate Certificate in Medical Education during FY2.

Management and Leadership

Management and leadership skills are rarely taught as part of undergraduate medical education, but become more important as your career progresses. Many candidates feel less confident when asked about these at interview. You may have examples from your time at medical school, such as holding a position of responsibility within a society or a sports club. Management outside of a medical context is also relevant, for example if you have experience of work in the voluntary sector.

There will also be seminars and conferences on medical leadership within your deanery – contact your hospital's education centre to find out when these are held.

Commitment to Surgery

Evidence from all the sections above can be used to demonstrate your commitment to surgery as a speciality. You should ensure that you have an electronic logbook, to record your involvement in as many operations as possible. Workplace based assessments (mini-CEX and Case Based Discussions) can also be used to demonstrate your interest in surgical topics. Remember that you will need evidence to back up each of your achievements, such as feedback forms for teaching. Points are also available for a clearly laid out, well organised portfolio.

Preparing for Interview

It is essential to prepare thoroughly for the interview and ensure that you are confident in expressing yourself verbally. Practising questions with friends or in front of the mirror is much more useful than writing notes. It may be worth investing in a medical interview preparation book, even one aimed at ST3 applications, as many offer useful advice on how to structure your answers to typical questions. The CST interview consists of 3 stations (clinical scenario, management and portfolio), each lasting approximately 10 minutes.

Clinical Scenario

You will be presented with 2 common clinical scenarios, the first written down before the station and the second given by the interviewer. The most likely scenarios are those commonly encountered on a surgical ward, or acute presentations such as those covered in the ATLS course. It is crucial that you demonstrate a structured ABCDE approach to the patient.

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Do not forget to consider patient factors. For example, a perforated duodenal ulcer might be managed very differently in an elderly woman with COPD and advanced dementia than it would be in a young athlete. You should be familiar with the principles of operative management for common conditions; you will not need to know the intricate details of each procedure.

Management

This station is designed to test your behaviour in challenging management or leadership situations you may encounter at work (9). Ensure you know how to escalate concerns appropriately and the duties of each person in the chain of command. You should also be familiar with the WHO guidelines for surgical safety, including the components of the WHO checklist. Plan a structure for your answers to common ethical dilemmas and communication stations (e.g. raising safety concerns about a colleague or informing a patient about a complication of their surgery).

Portfolio

During this station, you have 2 minutes to present a leadership biography, outlining your skills and how they will apply to CST. You will be asked questions related to your past achievements, commitment to a surgical career and career progression so far as outlined in your portfolio. It is important that you are familiar with the contents and can confidently discuss your contribution to each of the audits and projects you have been involved with. Be prepared to discuss any of the procedures listed in your logbook and to have specific examples for each of the leadership and clinical skills you mention.

Common interview pitfalls

• Preparing only by reading notes and preparing rote answers. Practising questions with friends provides a much more realistic experience.

• Wearing scruffy or distracting clothing, fidgeting or avoiding eye contract.

• Elaborating the truth. Ensure you have evidence to back up each of your achievements.

• Arrogance. Interviewers will be looking for candidates who can identify their own areas of strength and weakness.

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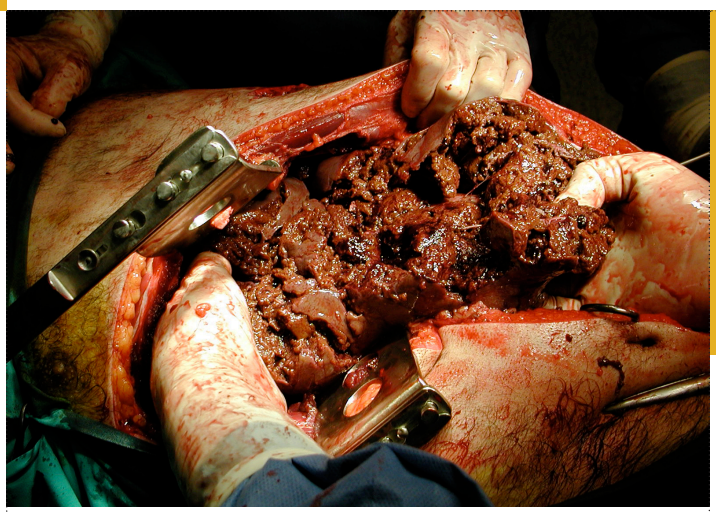
PRINCIPLES OF A TRAUMA LAPAROTOMY: A SURGICAL APPROACH

C Dawkins, S Wakefield



Introduction

Trauma is the major cause of death in young adults (1). It affects both civilian and military populations and strategies to manage these patients are continuously improving – particularly following lessons learned from recent military conflicts. All doctors should be familiar with the concept of both pre-hospital care (2) and Advanced Trauma Life Support (3).

This paper has been written to help provide an overview of the surgical approach to a patient with abdominal trauma and the principles applied to manage both blunt and penetrating injuries.

The concept of 'damage control surgery' is now accepted when life is in immediate danger – to control major bleeding, remove, contain contamination and prevent further injury, allowing patients to be stabilised before undertaking any definitive surgery (4). Only when patients are stable should a more thorough exploration of the abdomen be undertaken.

Clinical Presentation

Patients usually present through the Emergency Department. The causes and types of trauma are so varied but the underlying principle of management is uniform. FAST scan is usually accessible and stable patients can also be considered for CT if needed. Some patients however will need to undergo urgent exploratory laparotomy without these.

Surgical Approach

Ongoing anaesthetic and resuscitation requirements for these patients are not the remit of this paper. We are going to concentrate on the surgical approach required to deal with an acutely injured abdomen. Patients should be placed supine in a theatre that is equipped for major vascular and gastrointestinal surgery. Suction and cell saver capabilities should be available.

Principles Of A Trauma Laparotomy: A Surgical Approach
Good Clinical Care

Heating capabilities, in the way of heated mattress and BAIR hugger, are vital as hypothermia, along with acidosis and coagulopathy, mark the unhappy triad often signifying rapid deterioration for the patient (5).

Exposure should be from the nipples to the mid-thigh and the incision should be generous and midline. Full access to all abdominal and pelvic organs for assessment is needed, this is not a time for limited exposure.

Procedure

The guiding principles of a trauma laparotomy are threefold: to control bleeding, to contain contamination and to temporarily tamponade or close the abdomen (6).

Free blood, blood clots and contaminating fluids should be rapidly removed and any obvious areas of ongoing bleeding controlled with large gauze packs. If major vessels are clearly injured then temporary emergency clamping can be safely considered, including both the aorta and the Inferior Vena Cava (IVC).

The abdomen then requires temporary systematic tamponade with serial packing of the left upper quadrant, left paracolic gutter, pelvis, right paracolic gutter and right upper quadrant, ensuring these are placed both above, below and lateral to the liver. Having achieved haemostatic control the priority now becomes further resuscitation and stabilization of the patient.

Once your anaesthetist indicates the patient is adequately resuscitated it is time for the definitive trauma laparotomy. Packs should be removed one at a time dealing with any injuries as they present, starting in the area that is least suspicious as a source of injury.

Careful methodical examination of the abdomen and pelvis is needed and particular care must be taken to check areas know to be sites of occult injury. Each surgeon will have a standard technique that must be followed to ensure nothing is missed. It is usual to examine the compartments in sequence - supracolic, infracolic, pelvic and retroperitoneal.

PRINCIPLES OF A TRAUMA LAPAROTOMY: A SURGICAL APPROACH

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1. Supracolic

Begin with the right hemidiaphragm and right lobe of liver including gallbladder. Move past the falciform ligament to examine the left hemidiaphagm, left lobe of liver and spleen. Oesophageal hiatus, stomach and duodenal cap are next. The common bile duct is rarely injured but if needed can be accessed by duodenal mobilization (Kocker's manoeuvre) (7).

2. Infracolic

From the third and fourth part of duodenum check distally along the small bowel to caecum, appendix and colon to rectosigmoid junction. Don't forget to check for haematomas in the greater omentum.

3. Pelvic

Bladder first, uterus, ovaries and tubes next then rectum last. In the male the pelvis is inspected to the peritoneal reflection but not below this. Injuries to the seminal vesicles, ejaculatory ducts and prostate are very rare and if suspected should undergo delayed assessment and repair, they are not life-threatening. Check for any evidence of pelvic haematoma but resist the urge to explore further if this is contained.

4. Retroperitoneal

Begin with aorta and IVC, pancreas then right and left kidneys in turn – check along the lines of the ureters but do not dissect out unless there is any obvious need.

Do not be afraid to do a full second check – it is easy to miss minor injuries and this is not a time to rush. A full assessment should take less than 10 minutes in experienced hands.

Specific Injuries and their Management

Vascular Injury

Aortic trauma requires repair either by direct closure if these are small, patching if more significant, or rarely grafting when there is extensive injury. Venous injuries to major vessels including the IVC are very difficult to repair. Vein wall is thin and attempted suture often exacerbates the problem. Ligation is usually the best option and this is combined with bilateral calf fasciotomies as a precaution against compartment syndrome.

Injuries to mesenteric vessels can be repaired or bypassed, with ligation as a last resort. Renal artery injury usually requires repair. The external iliac arteries supply the lower limbs and any injury to these vessels should be patched or repaired. In extreme circumstances bypass or ligation can be considered. Internal iliac artery and vein ligation is tricky but safe (8).

Diaphragm

Diaphragm rupture (Figure 1) can cause a major problem with respiratory function. Therefore urgent repair is required to enable effective ventilation. Without this ongoing anaesthetic resuscitation is often limited.

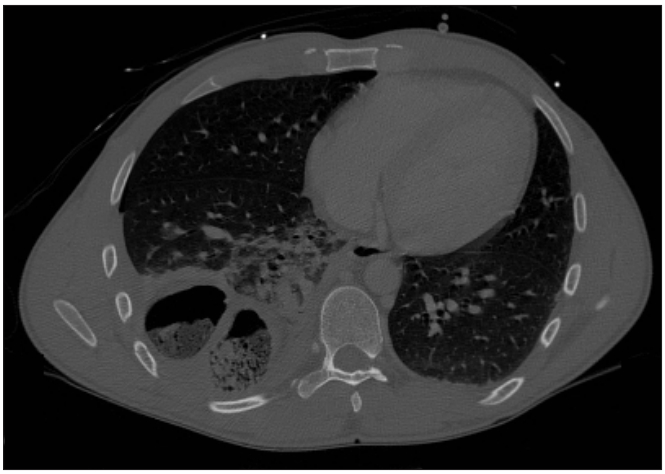

Figure 1

Splenic Injury

The spleen is injured in up to 45% of patients (9) with blunt abdominal trauma and is particularly susceptible in deceleration injuries. Injuries to this organ are classified according to the American Association for the Surgery of Trauma (10) (AAST) on either CT or laparotomy findings. This is summarized in Table 1.

I	Subcapsular haematoma <10% surface area Capsular laceration <1cm depth
II	Subcapsular haematoma10-50% surface area Intraparenchymal haematoma <5cm diameter Capsular laceration 1-3cm depth
III	Subcapsular haematoma >50% surface area or expanding Ruptured subcapsular or parenchymal haematoma Intraparenchymal haematoma >5cm in diameter or expanding Capsular laceration >3cm depth or involving trabecular vessels
IV	Laceration involving segmental or hilar vessels with major devascularisation (>25% of spleen)
V	Shattered spleen Hilar vascular injury with devascularised spleen

American Association for the Surgery of Trauma Splenic Injury Grading

Table 1: Classification of Splenic Injuries.

PRINCIPLES OF A TRAUMA LAPAROTOMY: A SURGICAL APPROACH

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Grades I-III are traditionally managed conservatively in the absence of other significant injury. Grade IV & V usually require splenectomy. Post-splenectomy patients will be at increased risk of overwhelming infection, particularly with encapsulated organisms. Immunization and antibiotic prophylaxis protocols must be followed (9).

Hepatic Injury

Similar to the spleen, the liver is commonly injured in blunt trauma (11). The liver however is an essential organ and cannot be fully removed! Liver injuries are similarly classified by the AAST as illustrated in Table 2.

I	Subcapsular haematoma <10% surface area Capsular laceration <1cm depth
II	Subcapsular haematoma10-50% surface area Intraparenchymal haematoma <10cm diameter Capsular laceration 1-3cm depth & <10cm length
III	Subcapsular haematoma >50% of surface area or ruptured haematoma Intraparenchymal haematoma >10cm diameter or expanding Capsular laceration >3cm depth
IV	Parenchymal disruption of 25-75% hepatic lobe or 1-3 Couinaud's segments
V	Parenchymal disruption of >75% hepatic lobe or >3 Couinaud's segments within one lobe Juxtahepatic venous injuries
VI	Hepatic avulsion

American Association for the Surgery of Trauma Hepatic Injury Grading

Table 2: Classification of Hepatic Injuries.

Grades I-III are usually managed conservatively. Grades IV-VI often require packing and transfer to a specialist Hepatobiliary centre.

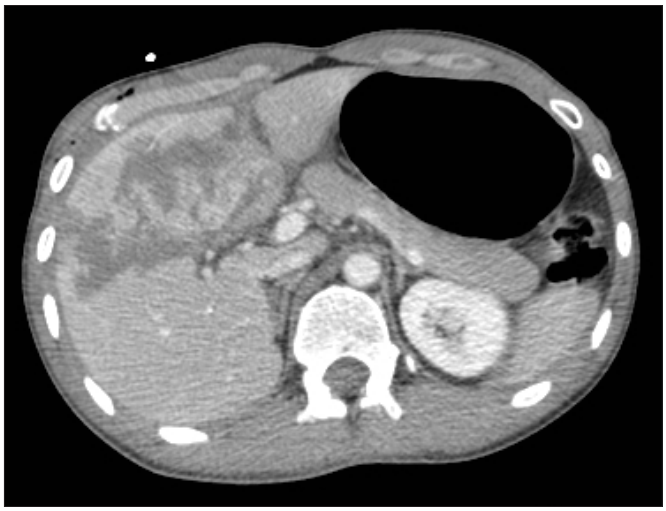

Figure 2

Temporary control of hepatic vascular inflow can be achieved by Pringles manoeuvre (12) which involves compression of the portal triad in the free border of the lesser omentum. This can be lifesaving. Careful external packing to compress an injured liver is the technique that all trauma surgeons should be familiar with.

Gastrointestinal Injury

In damage control surgery containing contamination is critical, and often the source is from an injury to the GI tract. These can occur anywhere along its length and a systematic examination is needed. As a general rule suture repair, resection with either delayed anastomosis or exteriorization are the options. Bypassing injured bowel and leaving it in situ is not practiced. The mesentery can be a source of occult injury and close examination, particularly for expanding haematomas should be carried out. If the dependent bowel shows signs of ischaemia it needs to be removed.

Renal and Urinary Tract Injury

The kidneys, bladder and urethra are susceptible to injury in both blunt and penetrating trauma. Pelvic fractures pose a particular risk to the urethra. Renal haemorrhage can often be controlled by packing alone, however with persistent bleeding or expanding pulsatile haematomas a more formal vascular approach may be required.

In extreme cases nephrectomy may be required. Partial injuries to the ureters can be managed with temporary stenting and/or repair whilst more significant injuries may require a proximal nephrostomy. Bladder injuries are usually managed by direct suture of the defect, with more formal layered closure being performed at relook laparotomy. Suprapubic catheters can be deployed in cases of suspected or confirmed urethral injury (13).

Pancreatic and Biliary Injury

Pancreatic injury is usually associated with very severe trauma and carries a poor prognosis, with a mortality of up to 34% (14). The integrity of the main pancreatic duct is the key. If this is intact primary conservative management is usually considered, if it is transected surgery is usually required. Similarly major biliary injury is rare and carries a significant mortality risk (15). Urgent discussion at a regional hepatobiliary centre is required for patients with these injuries.

Closing the Abdomen

It is acceptable, and often advisable, to either temporarily close or leave the abdomen open as a laparostomy. This reduces abdominal pressure (and risk of compartment syndrome), facilitates a second look laparotomy and aids ventilation in the immediate post-operative period. There are a number of techniques for temporary closure which can be considered (16).

PRINCIPLES OF A TRAUMA LAPAROTOMY: A SURGICAL APPROACH

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Second Look Laparotomy

Once the patient has been fully resuscitated post operatively and has returned to relatively normal physiology, ie normothermic with no remaining coagulopathy or acidosis, then they can return to theatre for a relook laparotomy and more definitive surgery. This is usually within 48 hours of the initial laparotomy.

Any remaining packs can be removed at this stage with a further full systematic inspection of the abdomen for any further injuries that may have been missed or have become apparent since the initial laparotomy. Definitive management of injuries should now be undertaken with the aim of abdominal wall closure if possible. If there is any concern at this point the abdomen can again be left open and further exploration planned.

Conclusion

This article has concentrated on the principles of a trauma laparotomy. Surgery is only one part of the process of trauma management and without rapid response and early resuscitation damage control surgery alone would invariably be futile. This is only one step of the journey that a patient with major trauma is likely to undergo. A robust and tested protocol to follow should reduce the chances of missing life-threatening injuries (17). It is therefore vital that this is led by a consultant with an appropriate level of understanding and experience.

MCQs

1. The following are associated with adverse outcome to trauma (unhappy triad)

a. Hypokalaemia

b. Acidosis

c. Low Hb

d. Time in CT

e. Hyperthermia

2. Splenic injury - the following statement is true

a. All injured spleens should be removed

b. Splenectomy patients are at risk of non encapsulated bacterial infection

c. Spleens can be classed on a Severity Scale of I-VI

d. Grade I-III splenic injuries can usually be managed conservatively

e. Splenectomy patients require BCG immunization

3. Damage control surgery. The following are appropriate in damage control surgery.

a. Resection & removal of severely injured bowel

b. Re-anastamosis of bowel in a contaminated abdomen

c. Re-anastamosis of bowel in an area of extensive mesenteric injury

d. Repair of severely injured bowel

e. Resect mesentery leaving its dependent bowel in place

4. Blunt trauma most commonly damages the

a. Ureter

b. Appendix

c. Spleen

d. Colon

e. Omentum

5. A second look laparotomy

a. Must ensure definitive closure of the abdomen

b. Identifies missed injuries

c. Should occur within 24 hours

d. Should occur to enable resuscitation

e. Must be the final surgical procedure

Answers

1. Answer b

The unhappy triad of adverse outcome to trauma is Acidosis, Hypothermia and Coagulopathy.

PRINCIPLES OF A TRAUMA LAPAROTOMY: A SURGICAL APPROACH

C Dawkins, S Wakefield

2. Answer d

The spleen can be classified by the AAST Severity Scale which runs from I to V from minor to severe. Grades I-III can usually be managed conservatively. Following splenectomy, patients are at risk of encapsulated bacterial infections, therefore should be immunized with Pneumococcal, Menigococcal and Haemophilis Influenzae Vaccinations.

3. Answer a

Optimal management is to resect and remove severely injured bowel. An anastamosis would not be appropriate in a contaminated environment, nor if the blood supply is at risk due to mesenteric injury. If mesentery is so severely injured that it requires resection then the dependent bowel must also be resected due to the risk of necrosis and subsequent contamination.

4. Answer c

The spleen is most commonly damaged in blunt trauma, especially deceleration injuries due to its ligamentous attachments. The other organs mentioned are far less commonly damaged in blunt trauma but could be more frequently injured in penetrating trauma.

5. Answer b

The primary function of a second look laparotomy is to identify and manage any missed injuries and allow more definitive surgery to be undertaken. However, this should occur once the patient has been stabilised and resuscitation has already occurred. It can be the definitive and final procedure but often requires a laparostomy and further surgical input.

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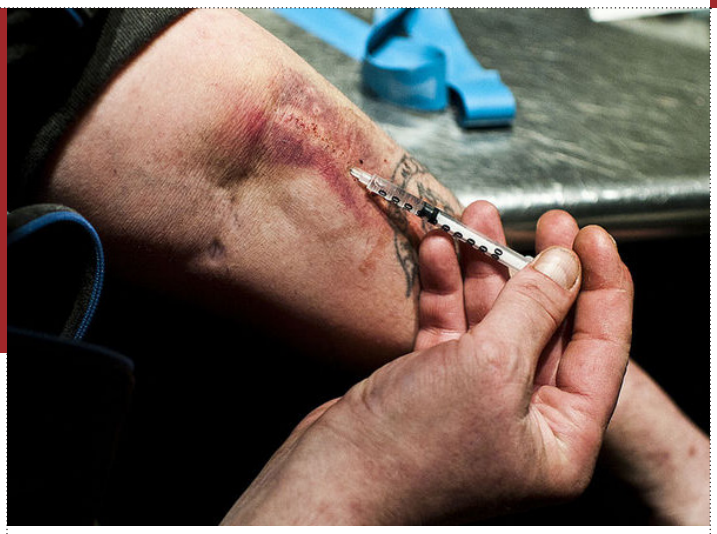
CUTANEOUS & VASCULAR COMPLICATIONS OF INTRAVENOUS DRUG USE

CR Gould, JRC Richardson, S Ghauri

Cutaneous & Vascular Complications Of Intravenous Drug Use
Patient Management



Abstract

Injection drug users (IDUs) not uncommonly present to the emergency department (ED) with cutaneous and vascular complications (1). Although numbers injecting (cocaine and heroine) have fallen (93,401 in 2010-11, 87,302 in 2011-12) (2), hospital admissions due to injection site complications have increased (3).

These complications include localised cellulitis, abscess development, pseudoaneurysm (PA) formation, compartment syndrome and necrotising fasciitis (NF) (4). The following case-based discussion exemplifies how a typical intravenous drug user may present; we discuss the important differentials and the challenges faced in managing these complications.

Case History

A 34-year-old man attends ED with a swelling in his left groin that has been present for 8 days. He complains of discomfort and rigors. He is an IDU and has needle track marks on his forearm; he denies having injected into the groin for 2 months. Examination reveals a fluctuant mass in the left groin with erythema and tenderness extending down the medial aspect of the leg. Blood tests reveal a white cell count of $21x10^9$/L and a CRP of 200mg/L. How would you manage this patient?

Overall management approach

History taking

A thorough history should be taken, including an accurate drug history and formal questions regarding use of illicit drugs and their routes of administration. Previous hospital admissions due to infections and abscesses are risk factors for NF or PA formation.

Examination

Initial examination should identify whether patients are unwell, septic and/or shocked; these presentations require early management and activation of resuscitation protocols (including oxygen, fluids and early antibiotics).

Examination of injection sites such as the groin (as in the example case) should identify erythema, induration and masses. The boundaries of erythema or cellulitis should be marked for later evaluation of signs of rapid spread. Oedema and paraesthesiae are signs of more extensive underlying inflammation or necrosis.

Digits should be closely examined for discolouration secondary to emboli from damaged areas of circulation; muscle compartments should be felt for firmness and other signs of compartment syndrome such as pain on passive stretching.

Investigations

Blood tests should be taken with strict regard to personal protective practices; in particular, hand-washing, double-gloving and safe sharps disposal. Tests should include FBC, U&E, LFT, CRP, blood cultures and hepatitis/HIV screen. Venepuncture may be a problem; IDUs often begin injecting into their prominent peripheral arm veins, however repetition of this leads to sclerosis and these vessels occlude requiring the use of increasingly hazardous sites such as the veins of the neck, groin and penis. Simple tasks such as peripheral cannulation can now prove impossible. The assistance of a senior anaesthetist with ultrasound guidance may be required to gain venous access.

Plain radiographs of the local area (groin) may identify retained needle fragments or demonstrate subcutaneous gas in severely infected cases. Ultrasound (US) (with duplex) will identify arterial/venous thrombosis, fluid collections and PA formation. Computerised tomographic (CT) angiography can further identify the extent of soft tissue involvement while also identifying proximal and distal circulatory problems, although magnetic resonance imaging (MRI) may be more sensitive for soft tissue inflammation and necrosis. An emergency surgical opinion should be sought especially when PAs or NF are suspected.

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Differential diagnoses

Cellulitis

This is a superficial infection of the skin and subcutaneous fat. It presents as a warm, tender area of erythema. Systemic upset such as malaise and fever may precede the visual findings. Most cases result from infection with streptococcus pyogenes and staphylococcus aureus from "dirty" needles and poor skin preparation (4,5). Management is with rest, elevation, analgesia and antibiotic therapy (6). Hospital guidelines and microbiology advice should be sought for the appropriate antibiotic regime. A high index of suspicion for NF should be maintained as this may present insidiously with a seemingly benign cellulitis masking a deeper tissue injury.

Abscess

An abscess is a walled-off collection of pus, accumulating secondary to a bacterial infection or localised irritation due to drug and/or toxic contaminants. Presentation is with a painful, erythematous mass. In IDUs, abscesses have been shown to be associated with "dirty" equipment (re-used needles), poor skin preparation (using saliva to clean the skin), and injection directly into the subcutaneous tissues (7). Abscesses are frequent among IDUs and many are neglected or inadequately self managed by lancing, "street" antibiotics or homeopathy (7).

They are common over the brachial and femoral vein territories; it is advisable to investigate all swellings considered to be abscesses in IDUs by US to identify/rule out associated PA and deep venous thrombosis. Abscesses can behave aggressively and erode into local structures such as arteries, nerves and joints (see Figure 1); though rare, they can induce compartment syndromes requiring extensive fasciotomies (see Figure 2). X-rays should be considered to identify foreign objects (fractured needles) within or close to the abscess cavity. Drainage is the definitive management; instrumental penetration is advised rather than digital dissection (8). The wound should be washed and packed, and healing allowed by secondary intention.

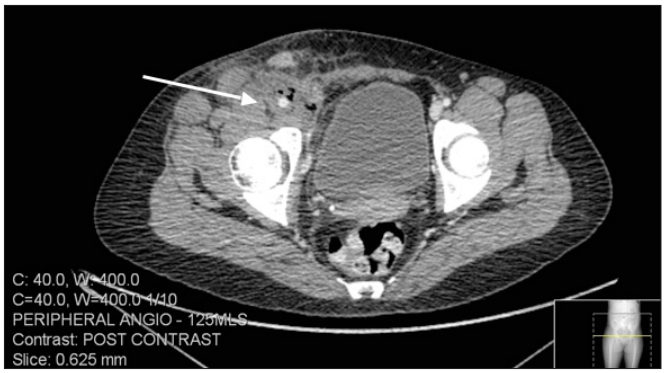

Figure 1: CT scan demonstrating a right iliacus muscle abscess extending around the common and superficial femoral arteries of the upper thigh. The abscess contains a large number of gas locules and a small fluid component.

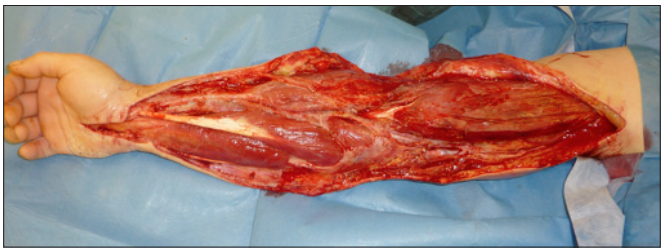

Figure 2: Post-operative image of a patient's arm following fasciotomies in the treatment of a compartment syndrome secondary to an IDU related abscess in the antecubital fossa (picture courtesy Miss A Crick, Consultant Plastic Surgeon, Salisbury).

Necrotising fasciitis

Necrotising fasciitis (NF) is a life threatening rapidly spreading inflammation of the fascia and subcutaneous tissues that develops secondary to virulent bacterial infection. Patients can present with pain out of proportion to the clinical findings, with subsequent fever progressing to septic shock. The condition is difficult to diagnose promptly as signs may be scarce early on in the course of disease.

The cutaneous features of haemorrhagic bullae, crepitus, oedema, numbness and gangrene occur late. Contaminated needles usually act as the route for the deep bacterial infection; up to half of IDUs inject at the site where NF subsequently develops (9). Factors that increase the risk of NF include immunosuppression (e.g. hepatitis/HIV), vascular insufficiency and alcohol misuse (10). US, CT and MRI may be useful in identifying gas and fascial thickening but imaging should not delay surgical exploration.

If limited bleeding, discoloured fascia and necrotic "dishwater" tissue are discovered, radical wide debridement must follow and extend to healthy tissue (11). Excised tissue should be sent for histological and microbiological analysis. Care should be transferred to an appropriate facility such as a regional burns/plastics centre. The wound must be checked regularly for further excisions and antibiotics given. There is a 10% risk of both lower limb amputation and mortality for NF in IDUs (9).

Pseudoaneurysm (PA)

Pseudoaneurysms (PA) are usually seen in the brachial and femoral arteries due to their close proximity to the respective veins which users are intending to inject (see Figure 3). Inadvertent (or deliberate) arterial puncture can cause pulsatile blood to leak from the lumen and accumulate in an encapsulating mass of surrounding tissues around the site of the defect (5).

PAs can present as a mass or an area of pain, induration and/or erythema near any injection site, they may or may not easily exhibit pulsatility (12). US is a useful first line diagnostic investigation, but CT angiography can show more detailed arterial anatomy, which is essential when considering vascular reconstruction.

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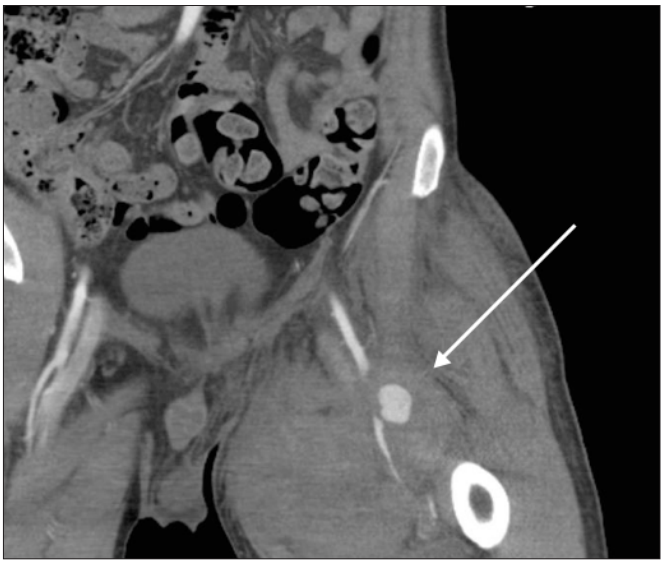

Figure 3: CT scan demonstrating a large left thigh haematoma associated with a pseudoaneurysm (PA) arising from the superficial femoral artery with probable left iliac and common femoral vein thrombosis.

Infected PAs are at great risk of rupture and catastrophic bleeding; sometimes small herald bleeds from the affected site being a warning of this impending event. The management of infected PAs within the groin (or in the forearm) is difficult and depends upon the degree of infection and the state of the arterial circulation.

Typically, wide debridement of the affected tissues is carried out alongside vessel ligation and excision. Clinical judgement guides whether or not immediate revascularisation is attempted. Collateral vessels may be sufficient to maintain vascular supply, however this risks inducing critical limb ischaemia (13).

Bypass surgery using autologous vein grafts can be performed as a concurrent or staged procedure (14). Interestingly there is no significant difference in lower limb amputation rates in those who have vascular reconstruction and those who undergo simple ligation (5, 15).

Additional considerations

All IDUs should be screened for BBVs (hepatitis B, hepatitis C and HIV); at presentation many patients are unaware of their viral status (16). Although risk taking behaviours such as needle sharing have declined, they still remain a significant factor in viral transmission (17).

Re-using needles and bending them to access difficult sites increases the likelihood of needle fracture; up to a fifth of IDUs admit fracturing needles whilst injecting (18) (see Figure 4).

Retained fragments pose a risk of infection and have been reported to migrate into the circulation, right heart and lungs (19, 20). Surgical removal is the treatment of choice; however, regular imaging to ensure further migration has not occurred may be an appropriate alternative.

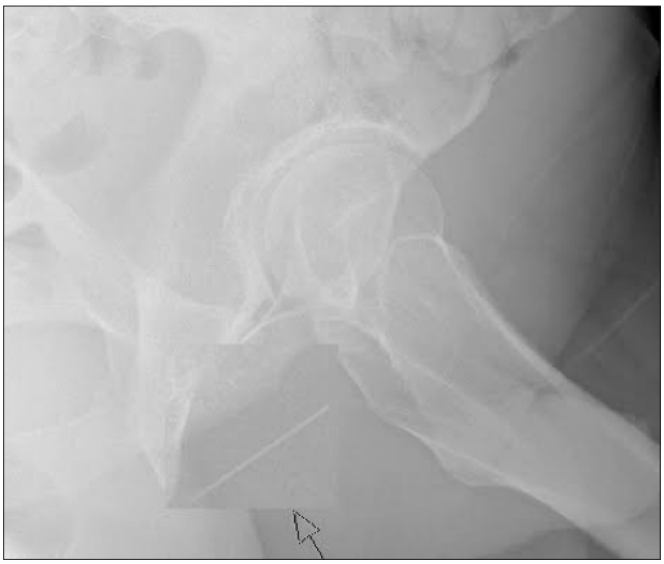

Figure 4: Radiograph demonstrating a linear metallic density projecting over the left inferior pubic ramus. This is a retained foreign body (needle).

Frequently alcohol and other substance misuse coexists in IDUs, this necessitates careful risk assessment and prescription of medication to alleviate adverse withdrawal effects. Patients can benefit from the help of other medical professionals such as alcohol and drug liaison nurses. IDUs should be informed of drug cessation programmes; an honest and frank approach is advised as these patients may be manipulative and drug seeking.

Conclusion

The management of a tender groin lump in an IDU should prompt an early referral for a surgical opinion due to the above-mentioned cutaneous and vascular complications. Other issues to consider include: personal safety, BBV exposure, difficulty gaining venous access, drug withdrawal and tendency to self-discharge.

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Junior doctors should be aware of these challenges, which may be considerable due to the lack of early engagement with healthcare services. Relevant investigations must be arranged urgently so that interventions such as drainage, debridement, fasciotomy or revascularisation can be undertaken to prevent what can rapidly evolve into life threatening consequences.

Questions

1. The optimal management of a groin swelling in an IDU is:

a) Intravenous antibiotics only
b) Needle aspiration
c) Plain x-ray and IV antibiotics and needle aspiration
d) Emergency referral to general/vascular surgeons for assessment

2. An IDU presents with an abscess in their forearm; there is marked swelling of the arm, erythema, and pain on passive movement of the wrist and elbow. Which of the following investigations are required for your further management?

a) Measurement of intra-compartmental pressure
b) Ultrasound
c) Blood tests: FBC, U&E, LFTs & CRP
d) No further investigations, take straight to theatre

3. What is the most useful first line imaging modality for diagnosing pseudoaneurysms in IDUs?

a) X-ray
b) MR angiography
c) Duplex ultrasound
d) CT angiography

4. Which of the following prominent clinicians is responsible for the term 'mycotic' aneurysm?

a) Charles Drew
b) Ambrose Pare
c) William Osler
d) James Paget

5. Features of necrotising fasciitis at surgery are (more than one applies):

a) Profuse bleeding
b) Resistance to blunt dissection
c) Dishwater pus
d) Foul smell
e) Discoloured fascial tissue

6. The definitive management of necrotising fasciitis is:

a) Observation
b) Broad spectrum intravenous antibiotics
c) Incision and drainage
d) Wide debridement

Answers

1. D

The presence of a groin swelling in an IDU should prompt early assessment from a general or vascular surgeon. If an abscess is diagnosed, consider pre-operative radiography if a foreign body is suspected within the cavity. Definitive management is with incision and drainage. The abscess cavity must be probed to break up loculations, it should then be washed out extensively. Adjunctive antibiotics are of benefit as there is usually an associated septicaemia.

2. D

This patient has compartment syndrome secondary to the formation of an abscess in their forearm. Compartment syndrome is a constellation of features that occur secondary to a rise in tissue pressure within a defined space that does not have the capacity to expand.

High pressures compromise tissue perfusion and function, resulting in ischaemia, necrosis and nerve injury. Clinically there is pain, swelling and discomfort on passive flexion and extension due to muscle ischaemia.

Measuring intra-compartmental pressures may aid diagnosis but should not be relied upon as a definitive test when clinical signs are present. Normal myofascial compartment pressure is <10mmHg. Fasciotomy is the definitive treatment – the muscles will immediately bulge on surgical fascial decompression confirming and treating the diagnosis.

3. C

Duplex ultrasound is the best first line imaging modality. The pseudoaneurysm can be assessed in terms of cavity size and communication with its respective artery. Turbulent blood flow within the aneurysmal cavity may be demonstrated on colour Doppler by a swirling flow pattern, and with pulsed Doppler with a to-and-fro signal.

It is portable, economical, quick to perform and in addition it is non invasive, avoiding ionising radiation or the use of contrast material. CT and MR angiography are secondary investigations that provide greater anatomical detail.

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4. C

William Osler first coined the term 'mycotic' aneurysm when describing the appearance of a fungal-shaped aneurysm occurring in association with (confusingly) bacterial endocarditis. It is often used to describe infected aneurysms regardless of their aetiology, though the majority are bacterial in origin.

5. C, D, E

Bleeding is sparse due to thrombosed vessels. Fascial planes are discoloured. The normally adherent tissues become lax due to putrefication, which results in a positive 'finger sweep test'. Rotting tissue has a distinctly foul odour.

6. D

Aggressive debridement of all affected regions of skin and fascia should be performed until normal tissue is reached. Patients must be advised preoperatively of the possibility of extensive skin and tissue excisions, without which mortality is almost inevitable.

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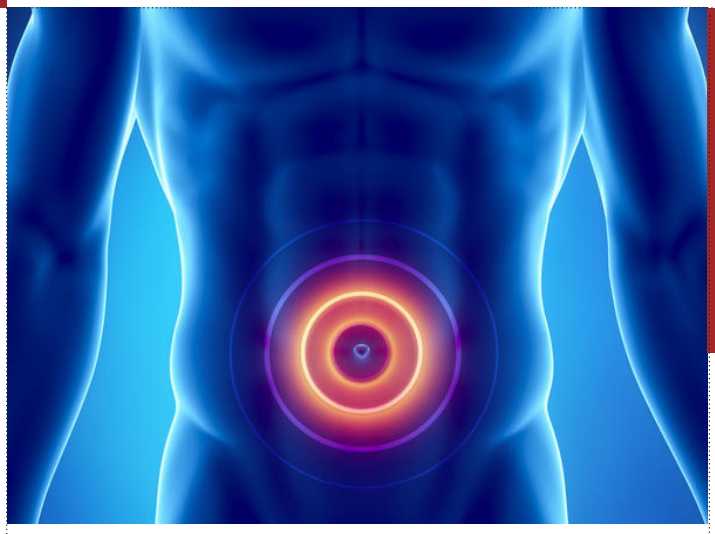
THE MANAGEMENT OF A PATIENT WITH NOT SO 'MILD' ABDOMINAL PAIN WITH REPEATED ADMISSIONS (LESSONS THAT CAN BE LEARNED): A CASE BASED DISCUSSION

O'Brien, McLaughlin, AL Widdison



The Management Of A Patient With Not So "Mild" Abdominal Pain With Repeated Admissions (Lessons That Can Be Learned): A Case Based Discussion
Patient Management

Abstract

We present the management of a 73 year old lady with acute pancreatitis (AP). She was initially predicted as having mild AP with a Glasgow score of 1 and treated with analgesia and intravenous fluids before being discharged after 7 days. She was readmitted 4 days later and diagnosed with necrotizing pancreatitis on CT scan.

Antibiotics were started and 4 days later she was discharged home. She was readmitted 6 days later septic and a repeat CT scan showed infected walled off necrosis. This was treated with a cyst-gastrostomy, debridement of necrotic tissue and cholecystectomy. She made an uneventful recovery and was discharged after 9 days. An out-patient MRCP was normal.

The cause of her AP was likely to be due to gallstones but this was not proven. We discuss the importance of measuring serum amylase, severity scoring systems, the accuracy of ultrasound, and the role antibiotics.

Case History

A 73 year old lady presented to the Emergency Department with an episode of chest pain followed by epigastric pain and vomiting. She had a history of hypertension controlled with lisinopril and amlodipine, and chronic kidney disease. There was no history of alcohol excess. Clinical examination was normal, ECG unremarkable and blood results including paired troponins and D-dimer were negative. A serum amylase and CRP were not checked. She was diagnosed with acute gastritis and discharged home later the same day.

She suffered on-going epigastric pain at home before being readmitted sixty-three days later with an acute severe exacerbation of her upper abdominal pain. On examination, she had upper abdominal tenderness with guarding. Investigations revealed amylase 1762 iu/L (normal range 28-80), WCC 13 x 10^9/L (4-11), CRP 2.6 mg/L (<5), creatinine 113 umol/L (44-80; patient's baseline 112-120) and normal liver function tests (LFTs).

Her Glasgow score was 1 and a diagnosis of mild AP was made. She was treated with intravenous fluids and analgesics. A CXR revealed a small pleural effusion. An abdominal ultrasound scan (USS) showed free fluid around the liver and spleen (Figure 1), and a thick walled gallbladder (Figure 2).

The patient had a pyrexia when admitted but the temperature was normal thereafter. Repeat blood tests showed the WCC increased to 17 on day 2, then fell to 8.7 on day 3, and remained normal until discharge after 7 days. CRP peaked at 326 on day 2 and fell to 195 by discharge. Although no gallstones were seen on the USS, gallstone AP was the likely diagnosis so an out-patient magnetic resonance cholangio-pancreatogram (MRCP) was ordered.

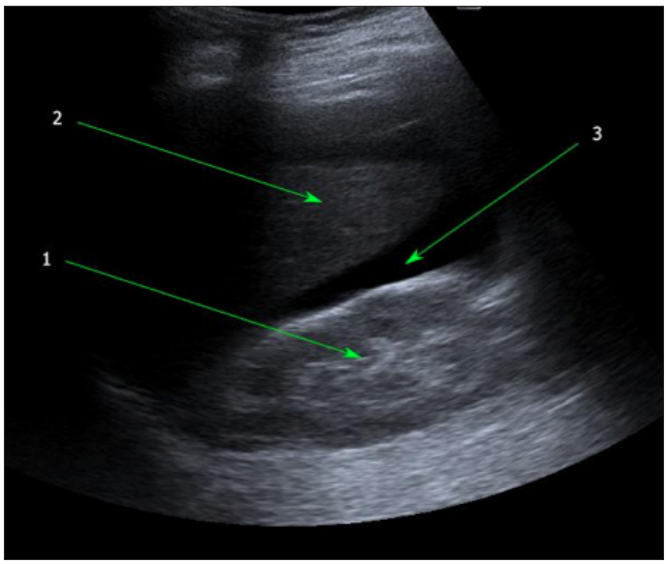


Figure 1: USS showing free fluid around the liver.

1. Right kidney
2. Liver
3. Free intra-abdominal fluid

THE MANAGEMENT OF A PATIENT WITH NOT SO 'MILD' ABDOMINAL PAIN WITH REPEATED ADMISSIONS (LESSONS THAT CAN BE LEARNED): A CASE BASED DISCUSSION

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She was readmitted four days later with abdominal pain, breathlessness and pyrexia. Serum amylase was normal at 36, WCC 17, CRP 157 and alkaline phosphatase 195 (30-130 iu/L), other LFTs were normal. A contrast enhanced CT scan showed a peri-pancreatic fluid collection, pancreatic necrosis and a large pleural effusion. Intravenous meropenem was started as prophylaxis and the pleural effusion was drained.

She improved clinically and biochemically with no further episodes of pyrexia and the CRP fell from 157 to 107. Four days later she was discharged on a course of oral co-amoxiclav and metronidazole for a total of 14 days following microbiological advice.

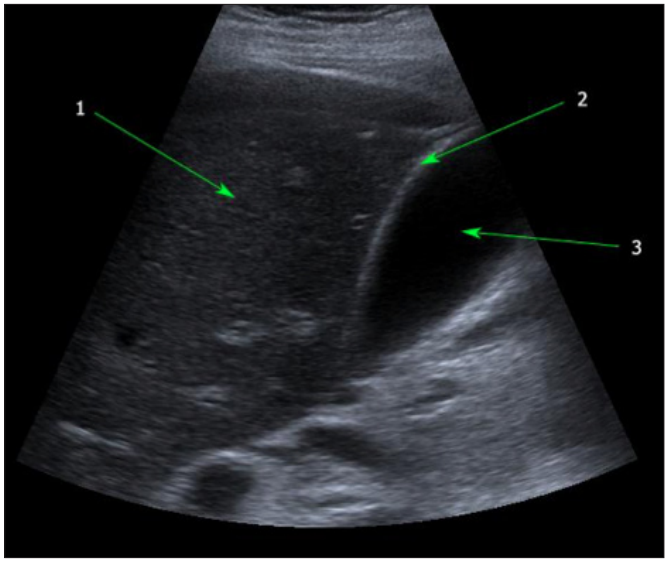

Figure 2: Ultrasound scan showing thick walled gallbladder.

1. Liver
2. Gallbladder wall (thickened)
3. Body of Gallbladder

Six days after discharge she represented with rigors and pyrexia. Serum amylase was 192, WCC 25 and CRP 189. She was re-started on intravenous meropenem and a repeat CT scan was performed that evening. This showed walled off pancreatic necrosis and extra-luminal gas indicating infection (Figure 3).

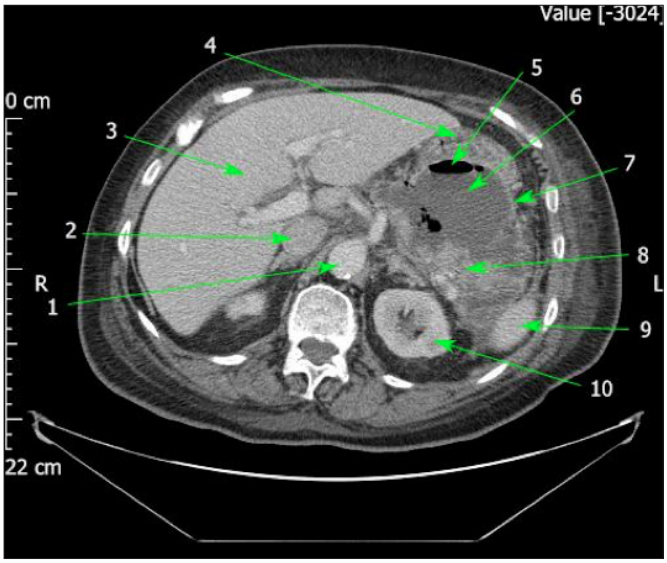

Figure 3: CT abdomen.

1. Abdominal aorta
2: Inferior vena cava
3: Liver
4: Stomach
5: Gas within walled off necrosis
6: Fluid within walled off necrosis
7: Enhancing wall
8: Necrotic tissue in pancreas
9: Tip of spleen
10: Left kidney

The following day an emergency laparotomy, cyst-gastrostomy, pancreatic necrosectomy and cholecystectomy were performed. Swab cultures taken intra-operatively grew coliforms sensitive to gentamicin and resistant to amoxicillin. Post-operatively, she spent 12 hours in the high dependency unit and was discharged home 9 days after her operation. She has made a good clinical recovery and an MRCP a month later showed resolution of the main walled off necrosis and a normal bile duct.

Discussion

AP is acute inflammation of the pancreas with variable involvement of regional tissues or remote system involvement. The incidence is 5-20/100,000/year, with a mean age of 50 years and affects males 2.5 times more than females (1). Our patient presented with the typical clinical features of AP and a raised serum amylase. In retrospect the symptoms and signs on the first admission were also typical for AP. Indeed, the patient said they were similar.

THE MANAGEMENT OF A PATIENT WITH NOT SO 'MILD' ABDOMINAL PAIN WITH REPEATED ADMISSIONS (LESSONS THAT CAN BE LEARNED): A CASE BASED DISCUSSION

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However, the amylase was not checked so the diagnosis was missed. Moreover, a normal amylase does not rule out AP because serum amylase starts to rise within 12 hours of onset of AP and is cleared rapidly from the blood, sometimes in as little as 48 hours, so it is not always raised when tested. In addition, in some patients the presentation can be atypical. Overall, approximately 30% of patients with AP are only diagnosed on CT scan or at post mortem (2).

In all cases it is important to identify the cause of AP to prevent future attacks. Gallstones and alcohol are the commonest causes but in up to 20% no cause is found (Table 1) (3).

Cause	Proportion	Notes
Gallstones	38%	Commonest causes of AP, accounting for approx. 75% cases
Ethanol	36%	
Uncertain	Up to 20%	No cause identified
Post ERCP	2-3%	Due to pancreatic duct contrast
Hyperlipidaemia	2%	Types I,II,V hyperlipidaemia. Must be very high (> 10mmol/L) to cause AP.
Medication	2%	Eg steroids, azathioprine, sulphonamides. ACE-inhibitors are a rare cause of drug-induced AP.
Pancreas divisum	<1%	Variant of normal anatomy in 5% population. Protective against gallstone pancreatitis but associated with minor papilla stenosis causing recurrent pancreatitis
Hypercalcaemia	<1%	Usually due to primary hyperparathyroidism – look for elevated calcium with normal/high parathyroid hormone levels.
Auto-immune diseases	<1%	Younger patients, often also with inflammatory bowel disease
Viral	<1%	Eg Mumps, Coxsackie
Tumour	<1%	Obstruction of the pancreatic ductal system due to pancreatic ductal carcinoma, ampullary carcinoma, cholangiocarcinoma or lymphoma
Trauma	<1%	AP develops in 5% of cases of abdominal trauma – commoner after penetrating (knives, bullets) than blunt trauma
Scorpion venom	<1%	Extremely rare in UK but the sting of the scorpion *Tityus trinitatis* is most common cause of AP in Trinidad
Hereditary	<1%	Autosomal dominant. Causes premature activation of trypsinogen to trypsin
Sphincter of Oddi dysfunction	<1%	Controversial. Causes increased pancreatic ductal pressures

Table 1: Causes of acute pancreatitis.

In our patient, gallstone AP was the likely diagnosis clinically and on USS; even though gallstones were not seen the gallbladder was thick walled. USS is operator dependent but even in the best hands is only 90% sensitive with a false negative rate of about 5% for the detection of gallstones. It is less sensitive in the obese, when there is a lot of bowel gas and for small (<2mm) non-calcified calculi (4).

MRCP is a more accurate test for the diagnosis of gallstones and bile duct stones. It can detect small stones and biliary sludge and is unaffected by obesity and bowel gas (5). Furthermore, it should be remembered that it only takes the migration of one gallstone from the gallbladder through the bile duct to cause AP.

Most patients have mild AP and make a full recovery but 25% develop severe disease with a mortality of about 35% (a third die from multi-system organ failure within a week and half from late infective complications) (1,3,6). Scoring systems (Table 2) are intended to aid early identification of patients at high risk of developing severe AP who should be managed in a critical care unit. However, many of these are unreliable as was illustrated in our patient.

	Ranson Score (1 point for each criteria met)	Glasgow Score (1 point for each criteria met)	APACHE II (Variable score for each criteria depending on result)	Revised Atlanta Criteria (Modified Marshall Score of 2 or more in any organ system to define organ failure)
Age	Age >70 years	Age >55 years	Age	-
Inflammation	WCC >18	WCC >15	WCC Temperature	-
Glucose	Glucose >12.2mmol/l	Glucose >10mmol/l	-	-
Respiratory function	PaO_2 <8kPa	PaO_2 <8kPa	PaO_2 and RR	PaO_2(kPa)/FiO_2 ratio: 0 >53.3 1 40.1-53.3 2 26.8-40 3 13.5-26.7 4 <13.5
Renal function, acid-base, and fluid balance	Within 48hrs: Base deficit >4 mEq/L Urea rise >1.8 mmol/L Fluid sequestration >4L Haematocrit fall >10%	Urea >16mmol/l	Arterial pH or venous HCO_3 Creatinine Haematocrit Sodium Potassium	0 Creat <134mmol/l 1 Creat 134-169 2 Creat 170-310 3 Creat 311-439 4 Creat >439
CVS function	-	-	HR Mean Arterial Pressure (MAP)	0 SBP>90 1 SBP<90, fluid responsive 2 SBP<90, not fluid responsive 3 SBP<90, pH<7.3 4 SBP<90, pH<7.2
LFT	AST >250 LDH >350	ALT>200 Albumin<32g/l LDH>600IU/l	-	-
Calcium (corrected)	Ca <2mmol/L	Ca <2mmol/l	-	-
Cerebral function	-	-	GCS	-
Chronic conditions	-	-	Score if chronic organ failure	-
Total	Score 3 or more for severe	Score 3 or more for severe	Add together scores for Age, Chronic conditions, and Acute physiology	**Mild:** Absence of organ failure and of local complications eg necrosis, pseudocyst **Moderate:** Local complications or transient organ failure (<48hrs) **Severe:** Persistent organ failure(>48hrs) or death

Table 2: Prognostic/severity scoring systems.

A recent review reported they have high negative predictive values but low positive predictive values for severe AP (7). The Atlanta classification, revised in 2013, is internationally recognised as the scoring system of choice (7,8). AP is classified as mild (no necrosis or organ failure), moderately severe (sterile necrosis, local complications and/or transient organ failure for <48 hours), severe (infected necrosis or persistent organ failure >48 hours) and critical (infected necrosis and persistent organ failure) (9).

It accurately predicts mortality, morbidity and length of stay (7,8). Our patient's disease was initially classified as mild on the Glasgow score, but was moderately severe according to Atlanta classification. However, this progressed to severe AP when she developed infected walled off necrosis. If this had not been treated in a timely manner multisystem organ failure would have developed.

THE MANAGEMENT OF A PATIENT WITH NOT SO 'MILD' ABDOMINAL PAIN WITH REPEATED ADMISSIONS (LESSONS THAT CAN BE LEARNED): A CASE BASED DISCUSSION

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Walled-off necrosis is a mature, encapsulated collection of pancreatic and/or peri-pancreatic necrosis that has developed a well-defined inflammatory wall. It usually occurs >4 weeks after the onset of necrotizing pancreatitis. If walled off necrosis becomes infected, as occurred in our patient, it is a life threatening complication frequently associated with multi-system organ failure. The most frequent pancreatic pathogens are coliforms, as was found in our patient, and rarely anaerobes and fungi.

The recommended antibiotics for prophylaxis in patients with severe AP are imipenem, cefotaxime and ciprofloxacin (10). Penicillins and aminoglycosides have limited penetration into the inflammatory tissue, and metronidazole may make matters worse because it kills anaerobes in the colon leading to enteric aerobic bacterial overgrowth (10). Our patient developed life threatening infection despite being on co-amoxiclav and metronidazole. Once walled off necrosis becomes infected, antibiotics alone are insufficient AND surgical drainage and debridement becomes the treatment of choice.

In summary...

- *A diagnosis of AP must be considered in all patients presenting with abdominal pain.*

- *Severity should be assessed using the Atlanta scoring system. Other early predictive scoring systems are less reliable (Table 2).*

- *Finding and treating the cause is important to prevent future attacks.*

- *Patients with severe AP should have appropriate antibiotics to reduce the risk of pancreatic infection, but once infection supervenes surgery is indicated.*

- *Pancreatic necrosis is a serious complication at risk of becoming infected.*

Test Yourself

1. A 36 year old lady presenting with abdominal pain is diagnosed with acute pancreatitis. She has Crohn's disease first diagnosed at age 20 when she had a right hemicolectomy and is currently treated with azathioprine. She drinks 4 units of alcohol each day. Investigations reveal a fasting triglyceride of 1.9 mmol/L and serum calcium of 2.6 mmol/L. What are the most likely causes of her pancreatitis?

a) Alcohol
b) Gallstones
c) Hypertriglyceridaemia
d) Drug-induced pancreatitis
e) Autoimmune pancreatitis

2. What initial investigation would you request to find out the cause of AP in this lady?

a. Contrast enhanced CT Chest, Abdomen and Pelvis scan
b. MRCP
c. ERCP
d. Ultrasound scan
e. Se HCAT bile salt malabsorption test

3. A 45 year old self-employed builder presents with a severe abdominal pain and tenderness with guarding. His admission HR was 120 bpm, BP 85 mmHg. Serum amylase was 1800 iu/L. He was given morphine iv, 15 L/min O_2 and 1 litre of Hartmann's over 1 hour. An hour later, after the fluid challenge his RR was 28/min, SaO_2 98% (on oxygen), HR 100 bpm, BP 110/50 mmHg. Bloods showed WCC 15.5x10^9/l, Hb 14.1g/dl, Urea 12.1, Creatinine 173 mmol/l, CRP 200. ABG showed PaO_2 19.9kPa, $PaCO_2$ 4.0, lactate 3.0, BD -4.4, pH 7.36. What is the likely diagnosis?

a) Mild oedematous pancreatitis
b) Severe necrotizing pancreatitis
c) Pancreatic infection
d) Pancreatic perforation
e) Ischaemic bowel

4. Please see figure 3. What complication is shown?

a) Acute pancreatic fluid collection
b) Splenic artery false aneurysm
c) Acute gastric dilatation
d) Simple pancreatic pseudocyst
e) Infected walled off necrosis

5. A 66 year old man is admitted with severe necrotizing pancreatitis. He is unwell for 2 weeks with persisting inflammation, ileus, and mild renal and respiratory failure requiring intravenous fluids and oxygen. Which of the following complications can occur?

a) Portal venous thrombosis
b) Infected pancreatic necrosis
c) Splenic infarction
d) Pancreatic ascites
e) All of the above

THE MANAGEMENT OF A PATIENT WITH NOT SO 'MILD' ABDOMINAL PAIN WITH REPEATED ADMISSIONS (LESSONS THAT CAN BE LEARNED): A CASE BASED DISCUSSION

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6. A 70 year old lady who is currently awaiting a cholecystectomy for gallstones presents with severe upper abdominal pain. The serum amylase is 3000 iu/L. Despite intravenous fluids, analgesia and 50% oxygen her HR is 130 bpm, BP 80 mmHg, SaO_2 89%, the creatinine is 450 mmol/L and the urine output is 10 mL/hour. The WCC is $2x10^9$/L and the CRP 35. What is the role of antibiotics?

a) Broad spectrum amoxicillin, gentamicin and metronidazole iv are indicated.
b) Antibiotics should be prescribed only if the blood cultures are positive.
c) Antibiotics are not indicated because pancreatitis is a chemical inflammation.
d) Treat with "second line" antibiotics such as imipenem or ciprofloxacin.
e) Prescribe antibiotics to treat infected pancreatic necrosis or an infected pseudocyst.

Answers

1. What is the most likely cause of her pancreatitis?

a) Alcohol: Although alcohol is the second most common cause of pancreatitis. It usually causes this lady drinks very little alcohol. Acute pancreatitis is usually caused by binge drinking or drinking large quantities of alcohol daily.
b) Gallstones: Gallstones are the likely cause. They are the most common cause of pancreatitis and this patient is at increased risk because the terminal ileum was previously diseased and she has had a right hemicolectomy which can disrupt the entero-hepatic circulation of bile salts increasing the risk of gallstones.
c) Hypertriglyceridaemia: The triglyceride level is only slightly raised and it would be insufficient to cause pancreatitis, which is associated with levels far higher than this.
d) Drug-induced pancreatitis: Azathioprine is one of the more common drug causes of pancreatitis it is unlikely to be the cause because she had been on the medication a long time. Statistically it is less likely than gallstones.
e) Autoimmune pancreatitis: Autoimmune pancreatitis is more common in female patients but is an uncommon cause of AP. It is usually causes chronic rather than acute pancreatitis.

2. What initial investigation would you request to find out the cause of AP?

a. A Contrast enhanced CT scan of the abdomen is indicated in patients with severe AP as part of the assessment or if the diagnosis is uncertain, but it is not usually an initial test to determine the cause and there is no indication for scanning the chest.
b. An MRCP is indicated to investigate the biliary tract and pancreas but is reserved when there is diagnostic uncertainty after an USS.
c. An ERCP is indicated when bile duct stones have been proven. Abdominal USS is the initial investigation of choice: it may show gallstones, a likely cause of AP, and will also image the bile duct to investigate the presence of dilatation or bile duct stones.
e. Se HCAT bile salt malabsorption test is inappropriate. It is indicated for the investigation of diarrhoea.

3. What is the likely diagnosis?

a) Mild oedematous pancreatitis is the likely diagnosis. He is a fit man who responded rapidly to fluid replacement. There is no evidence of organ failure.
b) It is unlikely to be severe necrotizing pancreatitis. There is no evidence of organ failure. If he were to deteriorate or remain unwell a CT scan would be indicated.
c) It is not uncommon for patients with AP to have raised inflammatory markers. These are not diagnostic of pancreatic infection which is a late complication.
d) There is no such condition as pancreatic perforation
e) A raised lactate may indicate tissue hypoperfusion in the context of a systemic inflammatory response and thus of a degree of circulatory dysfunction. However, this patient showed a good response to fluid resuscitation and their pH was normal. They are compensating for the metabolic dysfunction through hyperventilation and a low PaCO2. Although ischaemic bowel can cause a raised serum amylase this diagnosis is unlikely in a previously fit man.

4. What complication is shown?

a) The collection is walled off indicating it has been present for several weeks at least. Therefore it is not an acute pancreatic fluid collection.
b) Splenic artery false aneurysms do occur as a complication of AP and can contain gas when infected. However, they are closely related to the splenic artery which this walled off collection is not and they usually show intravenous contrast within the lumen which also is not present
c) The stomach is compressed over the walled off necrosis and not dilated
d) A simple pancreatic pseudocyst is a fluid collection within a mature wall. It is not associated with pancreatic necrosis and the gas within it indicates infection.
e) Walled off necrosis is the correct answer: the collection has a mature wall associated with pancreatic necrosis and the presence of gas is caused by infection.

5. Which of the following complications can occur?

a) Portal venous thrombosis can occur as a complication of any pro-thrombotic condition such as the inflammation caused by pancreatitis. It classically presents with signs of portal hypertension, abdominal pain and ascites, and if chronic may present with variceal bleeding.
b) Infected pancreatic necrosis is a "late" complication of severe AP. It usually presents with a deterioration of organ failure and inflammatory markers. It is diagnosed on contrast enhanced CT scan.

THE MANAGEMENT OF A PATIENT WITH NOT SO 'MILD' ABDOMINAL PAIN WITH REPEATED ADMISSIONS (LESSONS THAT CAN BE LEARNED): A CASE BASED DISCUSSION

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c) Splenic infarction is caused by splenic artery thrombosis secondary to the pancreatitis or from an embolus from a false aneurysm.
d) Pancreatic ascites is an uncommon but serious complication in which the ascitic fluid has a very high amylase content (usually >10,000 iu/L). It may follow a type of AP called "central cavity necrosis" in which the body of the pancreas is destroyed but the tail of the pancreas functions normally or a leak from a pseudocyst (eg after percutaneous aspiration).
e) "All of the above" is the correct answer

6. What is the role of antibiotics?

a) There is no evidence that any of these antibiotics are effective in preventing infective complications of AP. This may be because the penetration of penicillins and aminoglycosides, such as gentamicin, into the pancreas and pancreatic necrotic tissue is limited or because many enteric aerobes, which are the common causes of pancreatic infection, are resistant to amoxicillin. Anaerobes are infrequent causes of pancreatic infection whereas metronidazole will eradicate anaerobes from the colon leading to colonization by other bacteria likely to be more pathogenic.
b) Prophylactic antibiotics are indicated in severe AP to reduce the risk of infection. Treatment is preventative and should not await the results of blood cultures.
c) Antibiotics are not used as a treatment for AP but to reduce the risk of infection developing.
d) This is the correct answer: several studies have shown imipenem or ciprofloxacin reduce the risk of pancreatic infection in patients with severe AP.
e) Infected pancreatic necrosis or an infected pseudocyst are treated by drainage. Antibiotics are used as adjuncts but will not eradicate the infection on their own.

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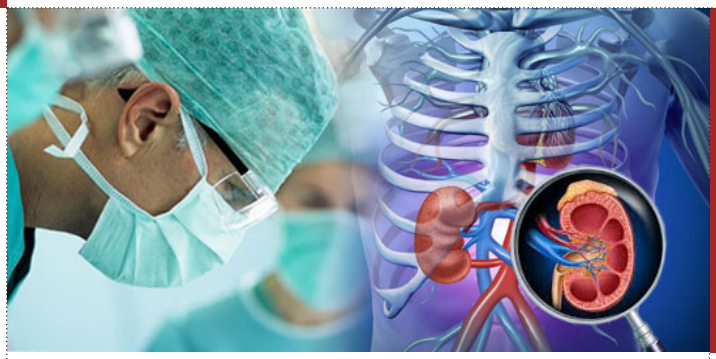
URETERIC COMPLICATIONS OF RENAL TRANSPLANTATION

E Brown, U Khalid, R Chavez



Ureteric Complications Of Renal Transplantation
Patient Management

Abstract

Kidney transplant is the best available treatment option for most patients with end-stage renal failure. Although relatively uncommon, complications affecting the ureter of the transplant kidney can cause significant morbidity. Here we discuss two cases that illustrate typical ureteric complications associated with renal transplantation.

Introduction

Renal transplantation has been established as the treatment of choice for suitable patients with end-stage renal failure. Compared to dialysis, it improves quality of life, prolongs survival and is more cost-effective.

With advances in immunosuppression in recent years, the long-term function and survival of renal transplants has also improved. Despite these improvements, the transplant operation carries a risk of complications which can cause significant morbidity. In this article we will look at complications specifically affecting the ureter of the transplant kidney.

The Kidney Transplant Operation

Renal transplants are usually implanted in an extra-anatomical location, specifically either the right or left iliac fossa. This site has many advantages, including easy access to a blood supply (the iliac vessels) and to the bladder. The transplanted kidney is also easily accessible for biopsy if required in the future.

In simple terms, a kidney transplant essentially involves joining three tubes, the artery, vein and ureter. Through a curved incision in the iliac fossa, the iliac vessels are accessed via an extra-peritoneal approach. The renal vein and artery are anastomosed in an end to side fashion usually to the external iliac vessels.

The transplant kidney ureter is anastomosed to the bladder (neocystostomy). Several techniques have been described for this, of which the most commonly used is the Lich-Gregoir technique. After filling the bladder with saline, the detrusor muscle and then mucosa of the bladder are opened. The transplant ureter is trimmed to a suitable length and spatulated.

A continuous anastomosis, mucosa to mucosa, with absorbable suture material is performed and the seromuscular layer then closed loosely over the anastomosis to produce a semi-anti-refluxing tunnel. Although not universally used, most surgeons protect this anastomosis with a stent. A double –J stent is used with one end in the bladder and the other in the transplant renal pelvis. This has been shown to reduce the incidence of ureteric complications (1). Typically this is removed two to six weeks after surgery.

Many complications of surgery can be thought of in simple terms and the same applies to renal transplantation. Many of the short to medium term complications of renal transplantation relate to these three tubes and their anastomoses. In this paper, we will present two cases that illustrate typical ureteric complications of renal transplantation.

Case 1

A 54-year old patient, Mrs A, undergoes a living donor kidney transplant. At the time of surgery, it is noted that there is a reduced amount of fatty tissue surrounding the ureter (usually this is preserved so as not to damage the blood supply). Six weeks after surgery her double-J ureteric stent is removed uneventfully. Two weeks later, she is seen in the clinic and it is noted that her creatinine is rising. An ultrasound scan of the transplant kidney shows marked hydronephrosis.

URETERIC COMPLICATIONS OF RENAL TRANSPLANTATION

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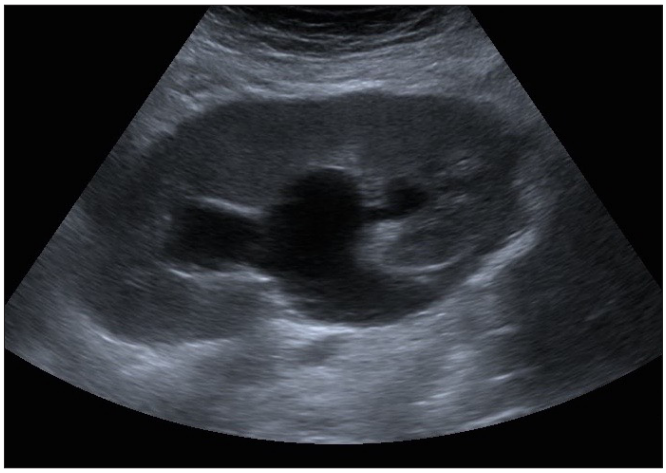

Figure 1: Image from ultrasound scan of renal transplant showing marked hydronephrosis with dilatation of renal pelvis and calyces.

The following day she has a percutaneous nephrostomy placed to relieve the hydronephrosis. A nephrostogram (contrast injected via the nephrostomy tube) which is then performed shows stricturing at the distal end of the ureter.

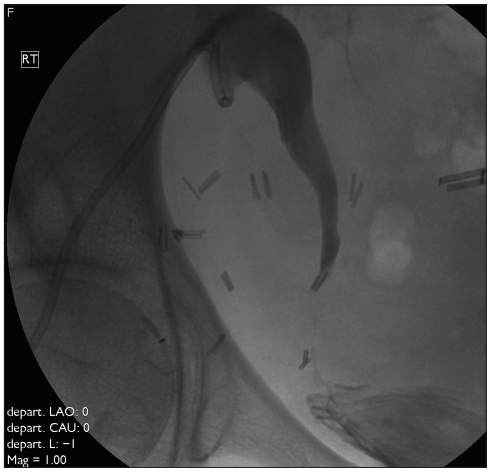

Figure 2: Nephrostogram showing a distal ureteric stricture. Contrast is injected via the nephrostomy which is seen to fill the renal pelvis and proximal ureter which then abruptly terminates.

The interventional radiologists pass a wire through the stricture and dilate it with an 8mm balloon.

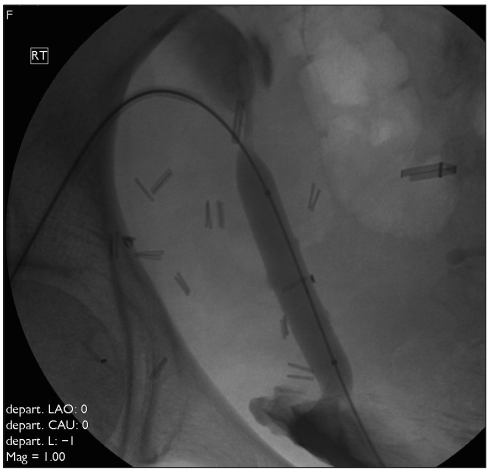

Figure 3: Balloon dilatation of ureteric stricture. The balloon is filled with contrast to show its position. In this case, a 8mm x 4cm balloon was inflated for 3 minutes.

An antegrade stent is then passed into the ureter to allow continuing drainage and the nephrostomy removed two days later. Her renal function returns to normal.

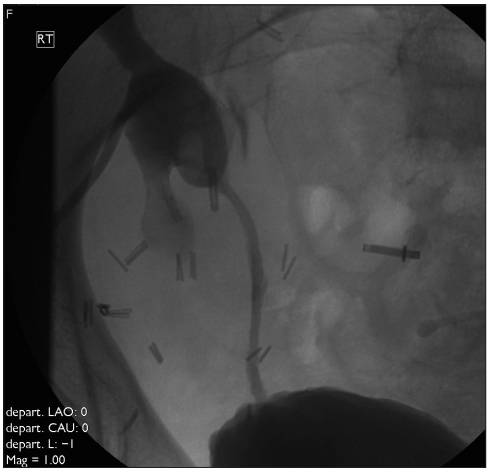

Figure 4: After balloon dilatation an antegrade stent has been performed. Its proximal end is visible in the renal pelvis and contrast is seen to pass easily down the ureter and fills the bladder.

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Three months later, the stent is removed. One week after removing the stent, the creatinine starts to rise again and a repeat ultrasound shows hydronephrosis. A second nephrostomy is performed and a nephrostogram shows recurrence of the distal ureteric structure. She is taken to theatre for definitive surgical treatment.

At surgery, the findings of a fibrotic stricture of the distal ureter are confirmed. The anastomosis between the ureter and bladder is 'taken down', the ischaemic part of the ureter excised, and a new anastomosis formed between the transplant ureter and the ureter of the native right kidney (uretero-ureterostomy). This is protected with a stent. This stent is subsequently removed without any problems and Mrs A continues to enjoy good function of her transplant to this day.

Case 1 Discussion

Ureteric strictures can occur at any point of the transplant ureter but most commonly affect its distal end. The most common cause is ischaemia. The ureter obtains its blood supply from adjacent structures as it travels into the pelvis and so when a kidney is removed for transplantation, the ureter will be dependent for its blood supply on branches from the renal artery and the distal ureter will be relatively ischaemic.

When implanting the kidney, the transplant ureter is normally kept as short as possible to minimize the risk of ischaemia. Similarly, during the backbench preparation of the transplant kidney prior to transplantation, it is recommended to avoid dissecting too far into the renal pelvis and to avoid stripping the peri-ureteral tissue as that may damage the blood supply of the ureter. In this case, the 'skeletalisation' of the ureter may have contributed to the stricture formation.

Other less common causes of ureteric obstruction can occur in transplant kidneys. In the early phase, these include technical error, obstruction with blood clot or extrinsic compression from a lymphocele. In the late phase, causes include calculi or tumour, and fibrosis resulting from rejection or opportunistic infection with BK virus.

Ureteric obstruction is usually easily identified by ultrasound scanning which will show hydronephrosis. If this is significant then it will be accompanied by graft dysfunction.

Initial management is with nephrostomy to relieve the hydronephrosis. This should be accompanied by improvement in graft dysfunction. A nephrostogram can be performed via the nephrostomy. This will delineate any areas of narrowing or stricturing.

Ureteric strictures can be treated with balloon dilatation. This utilizes an angioplasty balloon placed over a wire via the nephrostomy and is usually only considered in short strictures. Several case series have demonstrated success rates of balloon dilatation of 40-70% (2). A small case series showed success rates of 100% after 24 months follow up when utilized with laser endoureterotomy (3), although this is not in widespread use.

As this case illustrates, some strictures are resistant to balloon dilatation. Longer strictures are also considered to be unsuitable for dilatation. These should be treated surgically. This involves re-implantation of the ureter, either to the bladder if there is enough length (neocystostomy), or to the native ureter (uretero-ureterostomy). A third possibility is formation of a Boari flap –tubulisation of a flap of bladder to extend to the healthy ureter. In rare cases where surgery is not possible, treatment options include permanent stenting, extra-anatomic stenting (4) or permanent nephrostomy.

In a recent case series of more than 1500 renal transplant patients over 10 years, the rate of ureteric stricture requiring surgical repair was only 2.5% (5). All but two of these patients had successful re-implantation by either neocystostomy or Boari flap.

Case 2

A 43-year old man, Mr B, undergoes a kidney transplant. His post-operative course is not straightforward and he returns to theatre twice for exploration in the first post-operative week; firstly due to poor perfusion of the transplant due to kinking of the vessels; and secondly to evacuate a haematoma from around the kidney.

Following this second procedure, Mr B's renal function deteriorates. He is oliguric and requires ongoing haemodialysis. Ultrasound scans show adequate perfusion of the transplant kidney. A MAG-3 renogram confirms findings consistent with acute tubular necrosis (ATN). Three weeks post-operatively, an increasingly elevated output is noted from the surgical drain, associated with swelling around the surgical site. Another MAG-3 renogram is performed which now shows that the 'tracer' is being excreted by the kidney and appearing in the surgical drain. This is consistent with a urinary leak.

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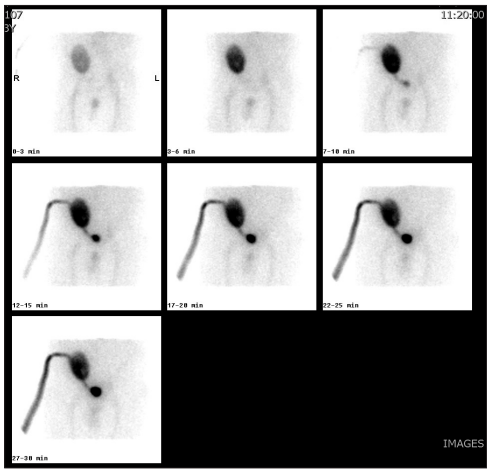

Figure 5: Image from a MAG 3 renogram with images taken at 5 minute intervals. The isotope tracer is seen to accumulate first in the kidney. Some then passes into the bladder but more passes into the surgical drain on the left side of each image, indicating a urinary leak.

Mr B returns to theatre. The entire length of the transplant ureter is noted to be necrotic with urine leak from the anastomosis site. The ureter is excised and an anastomosis is performed between the renal pelvis and the native ureter over a stent (pyelo-ureterostomy).

Two days later, Mr B continues to have high drain output and poor renal function. Biochemical analysis of the drain fluid shows that its composition is similar to urine, and a further MAG-3 renogram confirms an ongoing urinary leak. A CT scan shows a large fluid collection adjacent to the renal transplant.

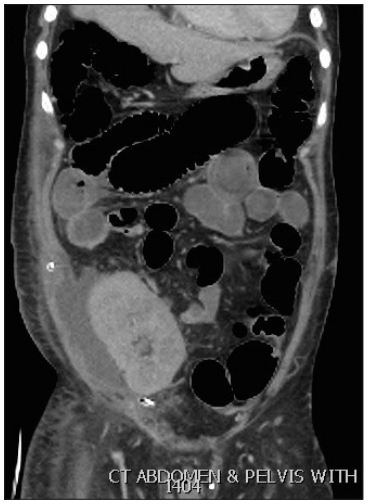

Figure 6: Coronal slice of CT scan with oral and IV contrast showing the renal transplant in the right iliac fossa with a fluid collection lateral to the kidney. In this case this fluid collection was a collection of urine from a urinary leak. The drain is visible in cross-section exiting the abdominal wall.

Mr B undergoes another operation, where it is noted that the ongoing urine leak is from the previous bladder closure. This is repaired and Mr B makes a full recovery with good renal function.

Case 2 Discussion

Urine leak is a recognised complication of renal transplantation, but its incidence is reduced by the use of prophylactic ureteric stenting (1). The most common site of urine leak is from the anastomosis of transplant ureter to bladder (neocystostomy) but it can also occur from the renal pelvis, ureter, or even from the bladder. Urine leaks almost exclusively present in the early post-operative period.

Urine leak can occur as a result of poor surgical technique or, as in this case, due to ischaemia and necrosis of the ureter. In this case, it is likely that this resulted from ischaemic insult after surgery from either kinking of the vessels or compression by the haematoma. More commonly, ischaemia of the ureter occurs due to damage to its blood supply and tends to affect the distal ureter.

Diagnosis of a urine leak is not always straightforward. Clinical features include pain and swelling or oedema of the tissues around the graft which may track into the scrotum. There will normally be graft dysfunction. If a surgical drain is in situ, drain outputs are likely to be high or increasing. Drain fluid can be sent for biochemical analysis. Urine typically has much higher potassium and creatinine values than serum.

If a urine leak is suspected, this should be confirmed with imaging. Ultrasound may show fluid collections around the graft and will exclude some other causes of graft dysfunction. In our unit, a MAG-3 renogram is the preferred modality for diagnosis of urine leak. Although less commonly used, a urine leak can also be demonstrated by cystogram.

Urine leaks occurring with a stent in situ usually require surgical repair. If a stent is not in situ, then placement of a nephrostomy and antegrade stenting may be adequate to control the leak while healing takes place.

Conclusions

Ureteric complications of renal transplantation are relatively rare but must be recognized and corrected promptly in order to preserve kidney transplant function. It has been demonstrated that prophylactic ureteric stenting at the time of surgery can decrease the incidence of major urological complications (1). Although ureteric stenting is associated with a slightly increased risk of urinary tract infection, this risk is reduced by antibiotic prophylaxis.

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Ischaemic ureteric stricture and urinary leak are the two most common ureteric complications following renal transplantation (6). Short distal ureteric strictures can be treated endo-urologically but may need revisional surgery. A urinary leak should be considered in a patient with graft dysfunction in the post-operative period, particularly if associated with subcutaneous oedema or high drain output.

Multiple Choice Questions

1. In a urinary leak after renal transplantation, analysis of drain fluid composition is likely to show:

A: Creatinine level similar to serum levels

B: Potassium level lower than serum levels

C: High potassium and high sodium levels

D: High potassium, urea and creatinine compared with serum

E: High amylase

2. True or false; ureteric stricture in renal transplant patients can be caused by:

A: BK virus infection

B: Infection with Pneumocystis carinii

C: Ischaemia of ureter

D: Hypercalcaemia

E: Rejection

3. A patient 5 days post renal transplant develops graft dysfunction and swelling around the wound. What should you do?

A: Send drain fluid for biochemical analysis

B: Check MSU

C: Arrange urgent Doppler ultrasound scan of graft

D: Arrange isotope renogram

E: All of the above

4. Which part of the ureter is at most risk of ischaemia in a renal transplant operation?

A: Proximal

B: Mid

C: Distal

D: All of it

E: None of it

5. Following a renal transplant when is the transplant ureteric stent usually removed?

A: Immediately after the ureter to bladder anastomosis

B: Within 1 week

C: Two to 6 weeks

D: 3 months

E: Never

Answers

1. D

Output from surgical drains in the case of a urinary leak is likely to have a composition similar to urine. It would be expected to have very high potassium, urea and creatinine levels and low sodium levels compared with serum values.

2. TFTFT

The most common cause of ureteric stricture in renal transplant patients is ischaemia. Ureteric stricture can also be caused by multiple episodes of rejection and by certain infections, including BK virus infection and repeated UTIs.

3. E

The clinical picture should raise suspicions of urinary leak. This can be confirmed with biochemical analysis of drain fluid and imaging including isotope renogram. Other early causes of graft dysfunction should be considered, including thrombosis of the graft (excluded by Doppler ultrasound) and urinary tract infection.

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4. C

The distal ureter is most at risk of ischaemia in a renal transplant. This is because once retrieved, the normal blood supply of the more distal ureter is interrupted. During a renal transplant operation, the ureter is trimmed as short as possible to reduce the risk of ischaemic complications.

5. C

The ureteric stent is usually removed between 2-6 weeks after surgery. Later removal carries a higher risk of complications such as urinary tract infection and encrustation of the stent.

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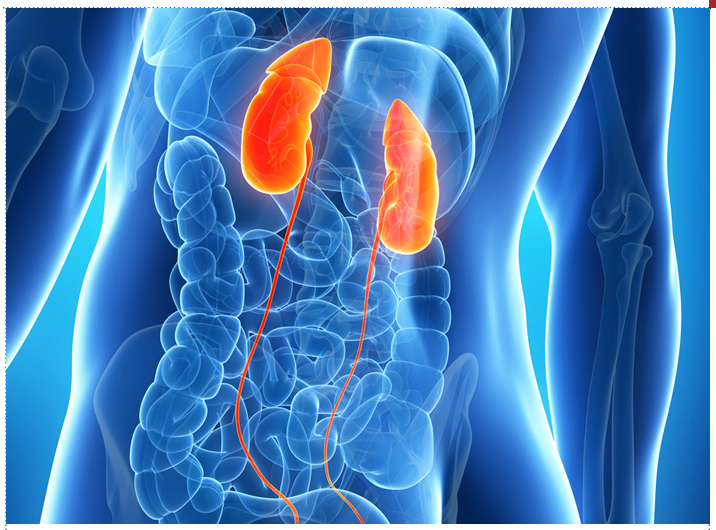

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UTILISING FOUNDATION DOCTOR RESPONSES TO IMPLEMENT A MODERN COST-EFFECTIVE SURGICAL TEACHING PROGRAMME & IMPROVE SURGICAL TEACHING

D Baglow, AF Young

Abstract

Introduction

Newly qualified doctors are often neglected when it comes to higher surgical training. We aim to use their responses to develop and evaluate a new surgical teaching programme for newly qualified foundation doctors to maintain patient safety and promote surgery as a career.

Method

A structured questionnaire was sent out to all foundation doctors undertaking surgical attachments over a 24-month period at a single centre to evaluate their experiences of the existing surgical teaching model.

A new teaching programme was devised and implemented based on the current UK surgical curriculum and questionnaire responses.

Results

A total of 60 junior doctors responded to the questionnaire (100% response rate). 16/60 foundation doctors (27%) were able to regularly attend formal teaching sessions. No foundation doctors (0%) were able to regularly attend a journal club, 8/60 (13%) received regular bedside teaching, 4/60 (7%) had time allocated to attend clinics and 20/60 (33%) were involved in departmental audit. 30/60 (50%) disagreed that their existing teaching gave them more confidence in their daily job and 22/60 (37%) strongly disagreed.

Following implementation of a revised surgical teaching programme 46/60 foundation doctors (93%, p-value < 0.01) were able to regularly attend formal teaching sessions. 60/60 (100%, p-value<0.01) regularly attended a journal club, 48/60 (97%, p-value<0.01) received regular bedside teaching and 42/60 (70%, p-value<0.01) had time allocated to attend clinics. 42/60 (70%, p-value <0.01) felt that a robust teaching programme gave them more confidence in their job.

Conclusion

Frequent job rotations and increased numbers of junior doctors working shorter hours means that surgical teaching programmes focused at foundation doctors are increasingly important. Teaching programmes must be both comprehensive and flexible in order to maximise exposure to surgery in the foundation years and maintain both quality of training and patient safety.

Utilising Foundation Doctor Responses To Implement A Modern Cost-Effective Surgical Teaching Programme & Improve Surgical Teaching
Teaching & Training

Introduction

The combined effects of the European Working Time Directive (EWTD) and Modernizing Medical Careers (MMC) have reduced the time that UK trainee doctors spend in hospital, increased the frequency with which they rotate through different departments and have reduced the time available for surgical training in theatres, clinics and on the ward. (1-3)

On qualifying from medical school UK graduates enter into a two-year Foundation doctor programme prior to entering higher specialty training, the equivalent of Internship.

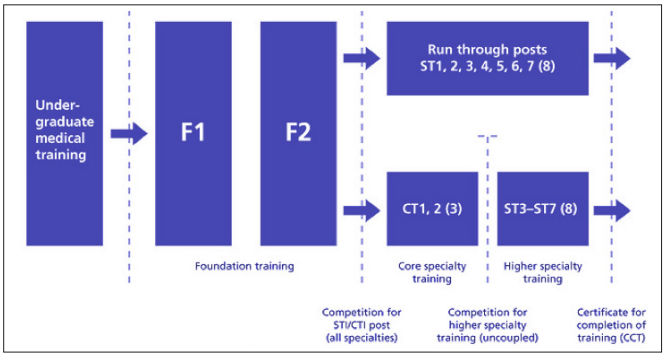

Figure 1: Basic UK surgical training structure.

In the United Kingdom selection into Core Surgical Training takes place four months into Foundation Year 2, sixteen months after graduation from medical school. Within this time applicants for surgical jobs are likely to have between four to eight months experience working in surgical jobs with the remainder of their placements spent in general medicine or general practice. The majority of foundation year general surgical jobs last four months and take place during foundation year one (FY1), the first year following graduation from medical school.

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Within this time newly qualified foundation doctors are expected to gain sufficient knowledge of surgical conditions to be able to safely manage patients on the ward and to develop basic surgical skills for their future careers. While some knowledge of surgical conditions is assumed on qualifying from medical school it is vital that surgical education is continued on the job to maximize patient safety. (4)

Surgical training in the United Kingdom is assessed using a competency-based surgical curriculum. The Intercollegiate Surgical Curriculum Programme (ISCP) distils the qualities required of a surgeon into categories based on Bloom's learning domains. (5)

While doctors selected into core surgical training receive regular teaching sessions to prepare them for membership examinations it can be more difficult to organise surgical teaching for newly qualified, foundation doctors due to ward-based commitments and shift patterns. In a reduced working time environment it is vital to not only optimise the teaching of basic surgical principles at an early stage but also to maximise opportunities for the practise of operative and practical skills. (6-7)

While surgical simulators and online resources have been shown to enhance surgical education these are often costly and are not readily available in most UK NHS trusts. (6)

Foundation year surgical jobs can be extremely busy with numerous ward commitments and little time for formalised teaching in theatre, clinics or on the wards. Based on anecdotal evidence from junior doctors and senior surgeons surgical teaching needs to adapt to work-time constraints in order to maximize teaching opportunities, maintain high standards of training and protect patient safety. (8-10)

This study aims to evaluate foundation doctors' perceptions of and access to surgical teaching and to utilise their suggestions to implement a modern, cost-effective surgical teaching programme aimed at newly qualified doctors that can be easily reproduced at other centres with minimal expense.

Methods

A questionnaire-based study aimed at foundation year one doctors in general surgical jobs was undertaken over a two-year period at a single centre in the United Kingdom.

Participants

Structured questionnaires were distributed via email and hard copy to all sixty foundation doctors who had undertaken a general surgical attachment. Following implementation of a new teaching programme the same questionnaire was then re-distributed. Incomplete questionnaires were excluded.

Study Questionnaires

Junior doctors and senior surgeons developed study questionnaires based on the Core Surgical Training Syllabus outlined in the Intercollegiate Surgical Curriculum Programme (ISCP) designed by the Joint Committee on Surgical Training (JCST) in the United Kingdom. Questionnaires were distributed to all sixty participants. Questionnaires looked at how often doctors were getting to theatre, how often they attended formal teaching sessions, how often they were given formal bedside teaching and what their expectations were from surgical teaching together with how they felt this impacted their confidence and ability to safely manage surgical patients.

The existing surgical teaching programme was ad-hoc and registrar led with no overall syllabus or timetable. The questionnaires featured direct questions regarding access to surgical teaching. In addition, statements were made regarding perceptions of current surgical teaching methods and respondents were asked to record their strength of agreement on a Likert scale from 1-4 with 'Strongly Agree' scoring 4 and 'Strongly Disagree' scoring 1. Respondents were also encouraged to add their own views on improvement in free-text boxes and highlight areas of particular concern. Data from the questionnaires was then transposed into a spreadsheet, tabulated and analyzed.

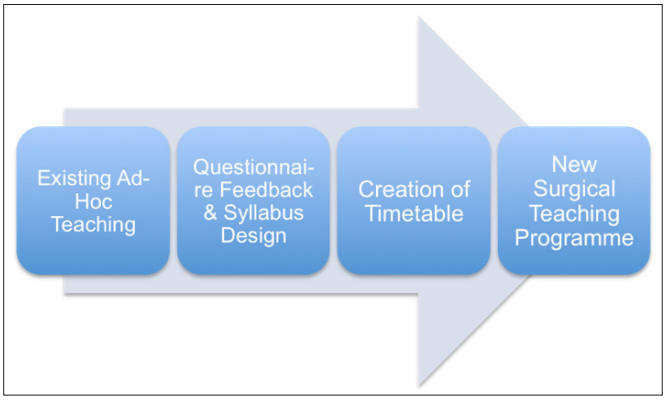

Figure 2: Process showing creation of the new surgical teaching programme.

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Devising a Revised Surgical Teaching Programme

Practical changes were implemented based on the results of the questionnaires together with discussion with senior surgeons. A new surgical teaching programme was developed. The syllabus was based on the Intercollegiate Surgical Curriculum Programme (ISCP) and featured didactic and skills sessions covering core topics.

The revised surgical teaching programme utilised the comments from foundation doctors who themselves suggested having a weekly early morning journal club and holding protected teaching sessions after ward rounds in the morning. Study questionnaire feedback revealed that teaching sessions on acute presentations, examination and fluid balance were most valuable. These topics were thus given precedence with fluid balance covered early on in the programme.

Surprisingly Foundation doctors themselves devised a rota to ensure that they were able to regularly attend theatre with other foundation doctors covering the duties of the doctor whose turn it was to assist in theatre. Also surprisingly foundation doctors felt that having formal sessions covering gloving and gowning and instrument handling was unnecessary as formalised teaching and preferred to practise this when they were in theatre.

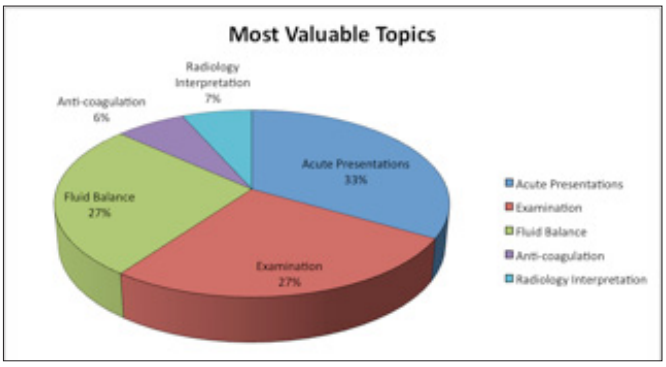

Figure 3: Most valuable topics as suggested by Foundation doctors (n=60)

Implementing a Revised Surgical Teaching Programme

Foundation doctor surgical jobs last for four months and so a sixteen-week programme was devised following the syllabus. An example weekly timetable is shown in Table 1.

The new teaching programme was led by both consultants and registrars and featured times for bed-side teaching, a journal club and protected teaching sessions covering topics highlighted by foundation doctors. Holding sessions immediately after ward rounds ensured that there was a good attendance from foundation doctors and if there was any poor attendance due to illness or leave sessions could be rearranged for a lunchtime session.

	Time	Activity	Clinic/Theatre
Monday	Post-Ward Round 0830-0900	Journal Club (30minutes)	FD 1 Clinic AM
	Lunchtime 1330-1400	Small Group: Radiology Interpretation	FD 2 Theatre PM
Tuesday	Post-Ward Round 0830-0900	Didactic: Upper GI Anatomy (30 minutes)	FD 3 Clinic AM
			FD 4 Theatre PM
Wednesday	Post-Ward Round 0830-0900	Bedside Teaching Upper GI Histories (30 minutes)	FD 5 Clinic AM
			FD 6 Theatre PM
Thursday	Post-Ward Round 0830-0900	Bedside Teaching Upper GI Examination (30 minutes)	FD 7 Clinic AM
	Lunchtime 1330-1400	Didactic: Upper GI Emergencies (30 minutes)	FD 8 Clinic PM
Friday	Post-Ward Round 0830-0900	Didactic: Fluid Balance (30 minutes)	FD 9 Clinic AM
			FD 10 Theatre PM

Table 1: Example of a Weekly Timetable (FD=Foundation Doctor).

The surgical teaching programme was explained to the cohort of foundation doctors at their departmental induction and each foundation doctor was provided with a teaching timetable together with a date for presentation at the journal club. The importance of attending theatre and clinics was discussed with the foundation doctors together with the other members of their surgical firms. Foundation doctors were encouraged to attend theatre and clinics by liaising with colleagues and sharing ward jobs so that wards were always covered if a junior doctor was in theatre or clinic.

Since teaching was held after ward-rounds, all patients were seen prior to teaching and urgent jobs completed. Sessions were limited to 30 minutes with two nurse practitioners and an on-call doctor covering the wards. No extra staff were hired and no extra costs were incurred with teaching held in a seminar room next to the surgical wards meaning that doctors were accessible should there be any emergencies from the wards to maintain patient safety.

Consultant and registrar roles were unaltered with respect to service commitments with clinic commencing at 0930 following teaching. Teaching was divided between 12 consultants and 14 registrars with radiologists and anaesthetists also teaching topics. A total of 56 person hours were required for teaching over the 16 weeks of the programme. Consultants and registrars were added to the teaching rota for set sessions and were able to create around 4 hours each over the 16-week period for foundation teaching without disruption to their existing commitments.

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Re-evaluation

Re-evaluation was undertaken to ensure that these changes had been positively implemented with the original questionnaire distributed to the next cohort of sixty foundation doctors undertaking general surgical jobs. Data from the second cohort of participants was then transposed into a spreadsheet, tabulated and analysed.

The two rounds of responses were analysed using chi-squared test with Yates correction and two-tailed p-values for statistical significance. Mean Likert scores were calculated and are presented with their standard deviation. The student t test for paired data was used to compare the responses of pre- and post-programme groups, A p-value of less than 0.01 was considered significant.

Results

Round 1: Pre- Implementation Of Surgical Teaching Programme

A total of 60 junior doctors responded to the questionnaire (100% response rate) of which 39 were female and 21 were male, mean age 24. 16 foundation doctors (27%) reported that they were able to regularly attend formal teaching sessions. Only 2 agreed that their current teaching provided as much teaching as they wanted with the majority disagreeing (32/60, 53%) or strongly disagreeing (24/60, 40%). 30/60 (50%) disagreed that their existing, sporadic teaching gave them more confidence in their daily job and 22/60 (37%) strongly disagreed. Theatre attendance was poor with 2 foundation doctors never attending theatre during their attachment.

No foundation doctors (0%) were able to regularly attend a journal club, 8/60 (13%) received regular bedside teaching, 4/60 (7%) had time allocated to attend clinics and 20/60 (33%) were involved in departmental audit during their attachment.

Qualitative Responses

In the further comments boxes of the questionnaire the main reasons for non-attendance at teaching sessions were workload on the wards and lack of formalised teaching times. Foundation doctors also commented that lack of teaching contributed to making them feel unsupported and was detrimental to their enjoyment of surgery as a specialty.

Foundation doctors highlighted that administrative work such as completing drug charts and writing discharge letters took up the majority of their time and that this time could be spent in theatre or at teaching.

Only 10/60 (17%) foundation doctors felt that they wished to pursue a career in surgery following the surgical placement with 36/60 (60%) citing lack of teaching as causative factor.

Round 2: Post-Implementation Of Surgical Teaching Programme

A total of 60 junior doctors responded to the questionnaire (100% response rate) of which 41 were female and 19 were male, mean age 24. Following implementation of a revised surgical teaching programme the number of foundation doctors able to regularly attend formal teaching sessions increased to 56/60 foundation doctors (93%, p-value < 0.001).

	Round 1 Mean Likert Score (1-5)	Round 2 Mean Likert Score (1-5)	p-value (paired student *t* test)
Able to regularly attend teaching	1.32 ± 0.70	3.46 ± 0.78	< 0.001
Provided as much teaching as you wanted	1.45 ± 0.54	3.76 ± 0.47	< 0.001
Made you feel more confident in your job	1.28 ± 0.62	3.59 ± 0.53	< 0.001

Table 2: Comparison of Foundation doctor responses to statements on teaching and confidence.

48 foundation doctors either agreed (36/60) or strongly agreed (22/60) that the updated programme provided as much teaching as they wanted (FIG 4.) with 42 either agreeing (24/60) or strongly agreeing (28/60) that the programme gave them more confidence in their role.

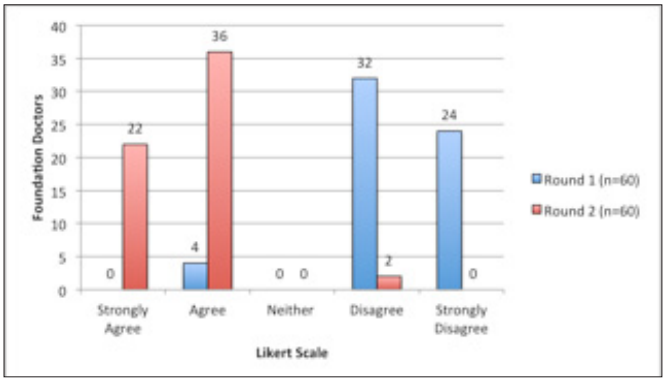

Figure 4: Number of Foundation doctors agreeing or strongly agreeing with the statement 'the surgical teaching programme provided as much teaching as you wanted' before and after implementing the new teaching programme (n=60).

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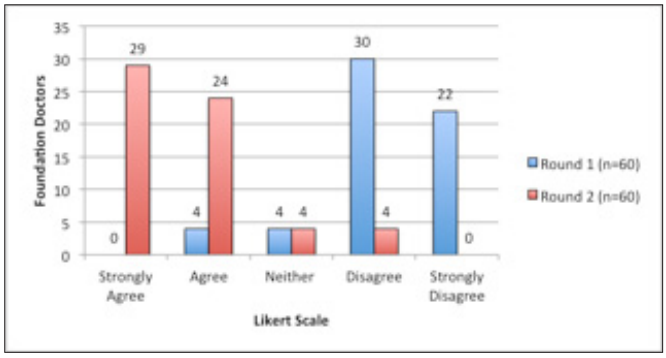

Figure 5: Number of Foundation doctors agreeing or strongly agreeing with the statement 'the surgical teaching programme made you feel more confident in your job' before and after implementing the new teaching programme (n=60).

While ward commitments necessitate that foundation doctors' priorities are patient-centred and ward-based there was also an increase in frequency of theatre attendance following implementation of the new teaching programme.

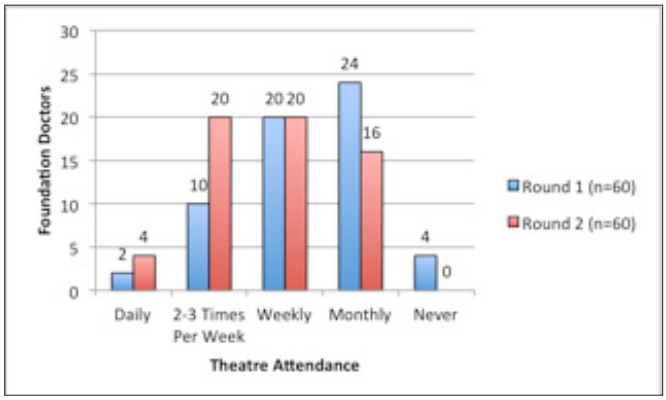

Figure 6: Comparison of theatre attendance during surgical placement before and after implementing the new teaching programme (n=60).

60/60 (100%, p-value <0.001) were able to regularly attend a journal club, 58/60 (97%, p-value <0.001) received regular bedside teaching, 42/60 (70%, p-value <0.001) had time allocated to attend clinics and 44/60 (73%, p-value <0.001) were involved in departmental audit during their attachment.

	Round 1 Mean Likert Score (1-5)	Round 2 Mean Likert Score (1-5)	p-value (paired student *t* test)
Able to regularly attend journal club	1.12 ± 0.30	3.01 ± 0.64	< 0.001
There was time allocated for bedside teaching	1.43 ± 0.44	3.36 ± 0.36	< 0.001
Able to attend clinic	1.55 ± 0.63	3.57 ± 0.54	< 0.001
Involved in audit or research	1.96 ± 0.65	3.61 ± 0.39	< 0.001

Table 3: Comparison of Foundation doctor responses to statements on access to teaching .

Following implementation of the new teaching programme 30/60 (50%, p-value <0.001) foundation doctors felt that they would like to pursue a career in a surgical specialty and 13 of these planned to sit part of the membership examinations (MRCS) during the foundation years.

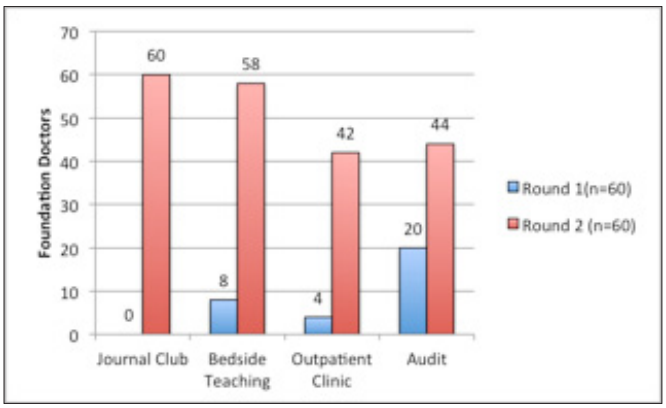

Figure 7: Number of Foundation doctors agreeing or strongly agreeing with the statements about attendance at journal club, bedside teaching, outpatient clinic and involvement in departmental audit before and after implementing the new teaching programme (n=60).

Discussion

The study demonstrates that traditional surgical teaching programmes, which relied on junior doctors being attached to a single surgical team and in hospital for 80 hours plus per week, are not perceived to provide adequate surgical education by today's newly qualified doctors. Foundation doctors themselves appreciate the fact that they must maximise learning opportunities in order to progress and to make their job more enjoyable. Doctors themselves highlight the importance of a teaching programme at this early stage of training to improve confidence and patient safety.

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The experienced-based apprenticeship model of surgical training, which relied on junior doctors being exposed to a large volume of surgical procedures and consultants having time in theatre to teach trainees, has been greatly impacted by a reduction in working hours and an emphasis on operative service provision. Service provision and theatre efficiency can vary between individual hospitals, healthcare trusts and regions meaning that junior doctors may get different levels of teaching depending on location.

While the current competency-based surgical teaching curriculum outlined by the Intercollegiate Surgical Curriculum Programme (ISCP) aims to maintain training standards for core surgical trainees and registrars in a reduced work time environment foundation doctors, who have an increasing focus on administrative tasks, are missing out on surgical teaching.

By utilising the responses of the foundation doctors themselves we have created an accessible and effective surgical teaching programme that maximises the time that foundation doctors spend on surgical attachments without increasing cost to the department or adversely impacting patient safety.

The addition of an effective surgical teaching programme to a foundation surgical attachment increases foundation doctor confidence when on the wards and when managing acute surgical conditions on-call. Correct identification and treatment of surgical conditions by juniors will also improve patient safety.

In order to maximise time available for teaching it is important that administrative tasks designated to junior doctors are kept to a minimum and their time is spent looking after patients or learning from colleagues. Nurse practitioners can facilitate the role of the foundation doctor by undertaking many of these administrative tasks and liaising with the ward staff so that foundation doctors have more time to spend learning and assessing ill patients.

For foundation doctors unsure of their career path it is important that they gain a good insight into what life is like as a surgeon and that they can confidently assess surgical problems on the wards. They should be encouraged to spend time in theatre and to consider a career in surgery.

For foundation doctors wishing to pursue a career in a non-surgical specialty this may be the only time in their training when they have exposure to surgical pathology, to learn from experienced senior colleagues and to actually see operations being performed.

For foundation doctors with a keen interest in pursuing a surgical career it is important that they maximize all available learning opportunities on the wards and in theatre. This is not just to boost their logbook numbers and surgical skills but also to ensure they can safely manage the acute surgical patient pre and postoperatively prior to selection into core surgical training.

The results after implementation of a new teaching programme demonstrate that foundation doctors are keen to attend teaching sessions and operating lists provided there is sufficient cover on the wards and that effective teaching programmes increase junior doctor confidence on the wards and exposure to surgical principles at an early stage.

Study Limitations

It is important to address the limitations of the study. We were unable to randomly assign foundation doctors and we acknowledge that our participants, in giving suggestions for a new teaching programme, may have been biased with their responses following its implementation. It would also be useful to repeat the questionnaire at a further 12-month interval to ensure sustainability of the teaching progarmme and foundation doctor attendance.

The study does not look at the quality of the teaching and it would be useful to follow-up foundation doctors to see which of them went on to pursue a surgical career and whether the teaching was helpful in their surgical membership examinations. The study was also dependent on consultants and registrars freeing up early morning and lunchtimes for teaching. While this has worked well in our center reproducibility will be limited by consultant roles and duties at other centers.

Conclusion

Although surgery is a craft, traditional apprenticeship models of training are no longer applicable in today's climate of the 48-hour workweek (12-14). Frequent job rotations and increased numbers of junior doctors working shorter hours means that foundation doctors must take every available opportunity to maximise their exposure to surgical specialties prior to selection into specialty training. Reciprocally more surgical education focus is needed on the foundation years to provide early surgical exposure to those who are keen to pursue a career in surgery. (15-16)

Due to the constraints of a forty-eight hour workweek imposed by the European Working Time Directive (EWTD) together with a reported increase in the volume of consultant service commitments and administrative work expected of UK foundation doctors on the wards it can be difficult to fully integrate foundation doctors into a surgical firm. (17-18)

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While core surgical training programmes offer protected theatre time, regional teaching days and revision sessions prior to membership examinations most foundation programmes do not.

Surgical teaching programmes aimed at newly qualified, foundation doctors are increasingly important and must be both robust and flexible in order to maximise exposure to surgery in the foundation years to maintain both quality of training and patient safety and to inspire the next generation of surgeons. (19-21)

While pre-intern and intern bootcamps have been shown to improve confidence in newly qualified doctors it is important that surgical education is an ongoing process to stimulate deep learning of surgical topics. (22-23) Implementing a robust, local surgical teaching programme for doctors at an early stage of their training is a cost-effective and well-received method of improving surgical education and patient safety that can be easily reproduced at other centres around the United Kingdom and beyond.

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