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FOUNDATION YEARS JOURNAL 2018

Volume 12

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Editor in chief

Publisher's office

Sophie Wood (Managing Editors)
123 Library, 72 Harley Street, London, W1G 7HG
Tel: +44 (0)207 253 4363 | Email: sophiewood@123doc.com

Editorial board

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Volume 12

Foundation years journal

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'CLOSTRIDIUM SEPTICUM ATRAUMATIC NECROTIZING FASCIITIS IN A PATIENT WITH COLORECTAL CANCER: A CASE REPORT & REVIEW OF THE LITERATURE'

R Jones, J Little

Abstract

Introduction

Necrotizing fasciitis (NF) is a rare but life threatening mono or poly-microbial infection of the skin, subcutaneous tissues, fascia and even muscle which progresses rapidly and has the potential to cause extensive tissue necrosis. It can be classified as traumatic or atraumatic.

Case

A 57 year old male presented to our DGH emergency department with a 24 hour history of pain and altered sensation in his right arm with no history of trauma. The patient had recently been diagnosed with a T4N2 caecal tumour.

Clinical examination demonstrated a 15cm area of erythema and swelling over the right scapula and posterior deltoid with a 2.5cm area of duskiness. There was associated subcutaneous crepitus over the right scapula, axilla and deltoid.

A plain radiograph of the right shoulder demonstrated air in the subcutaneous tissues and a CT Thorax confirmed subcutaneous gas in the right humerus and soft tissues of the shoulder and thorax. Blood cultures taken from the patient on admission grew Clostridium Septicum.

Discussion

Clostridium septicum is a gram positive, spore forming anaerobic bacillus. It is a rare subgroup of the clostridia family which has the ability to cause atraumatic myonecrosis and gas gangrene leading to a rapid, fulminant and most often fatal necrosis of human muscle. Numerous studies have found a strong association between Clostridium septicum infections and malignancy and immunosuppressed states.

Most cases of Clostidium septicum associated with colorectal cancer are related to tumours of the ascending colon. This case report demonstrates a severe and fatal case of Clostridium septicum atraumatic myonecrosis in a patient with newly diagnosed caecal tumour.

It aims to highlight the association of Clostridium septicum with atraumatic myonecrosis and gas gangrene with malignancy (most notably colorectal and haematological) and to emphasise the importance of a high index of clinical suspicion leading to early diagnosis and prompt and aggressive treatment.

Introduction

Necrotizing fasciitis (NF) is a rare but life threatening mono or poly-microbial infection of the skin, subcutaneous tissues, fascia and even muscle which progresses rapidly and has the potential to cause extensive tissue necrosis(1).

Most commonly NF is caused by a toxin producing bacteria and can be distinguished from other soft tissue infections by its extremely rapid progression and significant tissue necrosis. Advancement of infection within the tissue can reach a potential rate of 2-3cm/hour and clinical progression to septic shock is typically measured in hours.

Diabetic, obese and immunocompromised patients have been found to have higher rates of NF and diabetes mellitus has been found to be the most common co-morbidity amongst patients diagnosed with NF. Advancing age, increases in serum creatinine and lactate, a low INR (decrease of anti-thrombin III), acidosis and hypoalbuminemia at presentation have been suggested as markers of poorer prognosis(2).

Hippocrates first described necrotizing soft tissue infections in 5th century BC and later gas gangrene was described by an American army surgeon Joseph Jones in 1871, he reported a mortality rate of 46%. Today, NF is still associated with very poor outcomes.

Varying mortality rates have been suggested however it is likely that mortality rates often exceed 30% with optimal treatment and approach 100% if left untreated/undiagnosed despite modern advances in surgical and intensive care treatment. Multiple studies have consistently proven that delays in diagnosis and therefore management are directly associated with poorer outcomes both in morbidity and mortality(3).

Case Study

A 57 year old male presented to our DGH emergency department with a 24 hour history of pain and altered sensation in his right arm. There was no history of trauma and he did not report any further symptoms. A few weeks prior to admission the patient had been found to have iron deficiency anaemia on routine diabetic blood tests completed by his GP.

Following further investigations he was found to have T4N2 caecal tumour and was awaiting a colonoscopy. Past medical history included type two diabetes mellitus and hypercholesterolaemia. The patient smoked 20 cigarettes per day and had an alcohol intake of around 30 units per week. Medications included metformin, gliclazide, atorvastatin, ramipril and ferrous sulphate.

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On examination there was a 15cm area of erythema and swelling over the right scapula and posterior deltoid with a 2.5cm area of duskiness. There was associated subcutaneous crepitus over the right scapula, axilla and deltoid.

Peripheral blood tests demonstrated a marked inflammatory response with a white blood cell count of 15.1 x 10^9/L, a C reactive protein of 350 mg/L and a lactate of 3.3mmol/L. Renal and liver function tests were unremarkable.

Plain radiographs of the chest and right shoulder excluded a pneumothorax, and demonstrated significant air in the subcutaneous tissues (Figure 1).

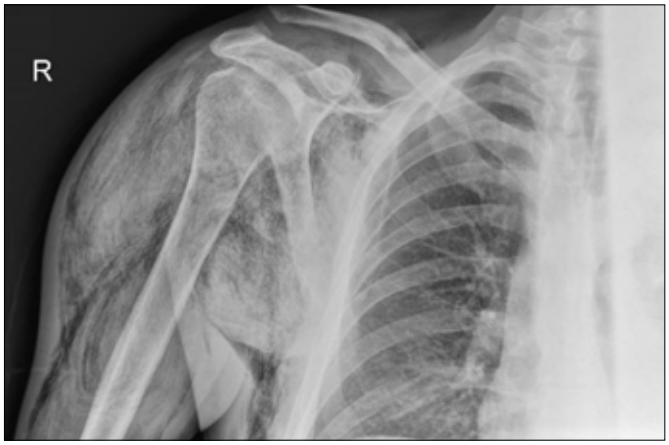

Figure 1: Plain radiograph of right shoulder. Warrington Hospital.

Initial management included empirical antibiotic cover: Piperacillin-Tazobactam, Clindamycin and Metronidazole, blood cultures and urgent orthopaedic referral.

Upon orthopaedic review a CT chest and right shoulder were requested. This demonstrated gas in the subcutaneous tissues likely secondary to necrotizing fasciitis in the right humerus and soft tissues of the shoulder and thorax.

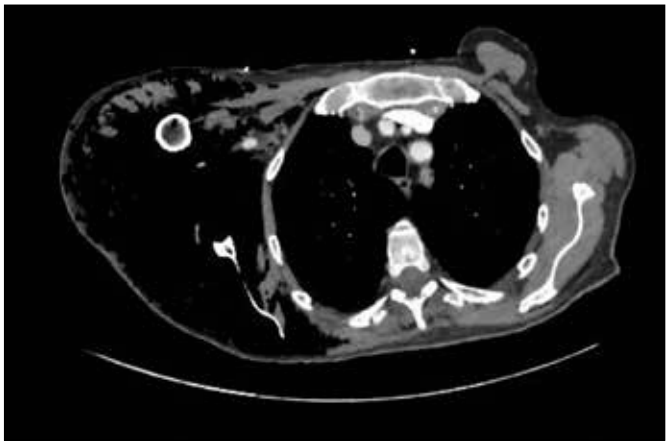

Figure 2: Transverse section of CT Thorax. Warrington Hospital.

Subsequently, a senior orthopaedic review was undertaken, at which time there was progression of clinical signs with swelling, erythema and subcutaneous crepitus covering the lower back, right wrist and right side of thorax.

The patient was taken to emergency theatre for debridement, where upon surgical exploration it was found that there were extensive areas of tissue necrosis including all muscles of the chest wall.

At this point it was agreed by both the consultant orthopaedic and consultant general surgeon that amputation of the right arm would be inadequate and that the disease was expected to be un-survivable. The surgical procedure was abandoned with a plan for repeat surgical exploration in 24 hours if the patient was sufficiently stable.

The patient was transferred to a level three ICU bed post-operatively. He required extremely high levels of inotropic support and intravenous immunoglobulins were commenced.

Despite medical intervention, the patient remained critically unwell and haemodynamically unstable with ever increasing inotropic requirements. Following further orthopaedic review, it was decided that due to the severity and extent of disease and the patient's unstable condition any further surgical intervention would be futile. Following a discussion with the patient's family, treatment was withdrawn and the patient died later that day.

Blood cultures taken on admission grew Clostridium septicum sensitive to penicillin and metronidazole.

Aetiology

NF infection can be classified as traumatic (inoculation of the pathogen into the subcutaneous tissues via a break in the epithelium or mucosa) or atraumatic (haematogenous spread from distant sites in the absence of skin trauma). NF can be further classified via bacterial aetiology; this is an important classification as it alters clinical presentation and aspects of management.

Type I NF is a poly-microbial infection, commonly including Clostridia bacteria. Type 1 NF is typically atraumatic affecting older patients with multiple co-morbidities. Clostridial species are particularly difficult to isolate from blood cultures and so negative blood cultures should not rule out a diagnosis of Clostridial NF if there is clinical suspicion. Clostridial NF infections are traditionally labelled as 'gas gangrene' because they present with subcutaneous gas and tissue crepitus; gas production is mediated via production of toxins(3).

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Clostridium septicum is a gram positive spore forming anaerobic bacillus. It is a rare subgroup of the clostridia family which has the ability to cause atraumatic myonecrosis and gas gangrene resulting in a rapid, fulminant and most often fatal necrosis of human muscle.

These effects are mediated by the release of exotoxins, most notably Ð toxin by Clostridium septicum which is a pore forming potent cytotoxin. This is hypothesised to cause programmed necrosis of cells via intra-cellular calcium ion influx, deregulation of mitochondrial activity and reducing intra-cellular levels of ATP(4).

The alpha toxin also acts as a platelet agonist causing thrombus formation, potentiating ischaemia at the site of infection and causing increasingly acidic conditions which favour bacterial replication and contribute to neutrophil dysfunction, impaired phagocyte function, intravascular haemolysis and impairment of endothelial cell integrity. The septic process ultimately leads to suppression of cardiac function and decreased vascular tone(3).

Numerous studies have found a strong association between Clostridium septicum infections and malignancy and immunosuppressed states. A review of 162 cases of Clostridium septicum by Kornbluth et al found that 81% of patients had an associated malignancy, most commonly haematological or colorectal. Most cases of Clostridium septicum associated with colorectal cancer are tumours of the ascending colon(5).

It is thought that the acidic and hypoxic environment created by anaerobic glycolysis resulting from a tumour outgrowing its own vascular supply increases germination of Clostridium septicum spores which can propagate into the bloodstream via mucosal ulceration causing distant, atraumatic myonecrosis and fulminant sepsis, septic shock and multi-organ failure. Translocation of Clostridium septicum spores is further increased in patients with impaired host immunity from diabetes mellitus, atherosclerosis, alcohol abuse, neutropenia and steroid use.

A high index of suspicion for Clostridium septicum NF is essential for early diagnosis as prognosis for fulminant Clostridium septicum sepsis remains very poor with a greater than 60% mortality rate(6) and Clostridial infections have been shown to be independent predictors of limb loss and mortality(7). Group A beta-haemolytic streptococci alone or in combination with Staphylococci are the causative agents for type II NF infections.

Type II NF infections have a characteristic aggressive local spread and systemic toxicity. There is more often a history of trauma, surgery or intravenous drug use. Streptococci mediate their toxicity via modulation of M proteins which allow bacterial to adhere to tissues, evade phagocytes and bypass the antigen presenting complex of the major histocompatibility complex II molecule. The result of this is massive cytokine release and an inflammatory cascade which quickly manifests clinically as shock.

Diagnostics

Diagnosis of NF infection can be difficult as clinical presentation is often non-specific and there are no single biochemical parameters which point to a diagnosis of NF. Typical presentation includes erythema, localised pain out of proportion to cutaneous findings (fascial involvement occurs before superficial cutaneous involvement) and swelling.

Later symptoms include bullae, necrosis, gas in the tissues and cutaneous anaesthesia with advanced disease presenting as extensive necrosis with septic shock and multi-organ dysfunction. Differentiating NF from cellulitis can prove difficult for clinicians but a faster rate of progression, skin oedema and necrosis should alert clinicians to the diagnosis of NF. A high clinical index of suspicion and awareness of which groups of patients are at risk is commonly needed for early diagnosis(8).

Wong et al developed a laboratory risk indicator for necrotizing fasciitis score (LRINEC) to distinguish NF infections from other soft tissue infections. This score uses C-reactive protein, total white blood cell count, haemoglobin, sodium, creatinine and glucose measurements. The study reported 96% PPV and 96% NPV with a score of six or above, however this has not been validated and criteria used in this score remain non-specific. Diagnosis should not be made solely on the basis of this score(9).

The role of imaging in NF infections remains controversial, as any imaging will likely cause a delay in the ultimate management of the patient. Plain radiographs may demonstrate subcutaneous emphysema however this rarely occurs outside of Clostridial infections. CT is a more sensitive modality than radiographs however can only display non-specific findings and as aforementioned will delay treatment. It is widely accepted that the gold standard for the diagnosis of NF is surgical exploration(3).

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Management

Early diagnosis is key in the management of NF as this allows for prompt surgical intervention which remains the definitive management of NF. Once NF has been diagnosed initial management should include appropriate resuscitation and early and empirical antibiotic therapy(10).

Antibiotics have a significant role in adjuvant therapy alongside surgical intervention; alone they provide little benefit as local ischaemia and hypoxia impair delivery of antibiotics to the site of infection. It is widely recommended that empirical antibiotics that cover gram negative, gram positive and anaerobic bacteria be commenced as early as possible.

This is generally includes a penicillin, aminoglycoside and clindamycin. Clindamycin is a relatively broad-spectrum antibiotic which has been found to reduced Clostridial alpha toxin release, reduce Streptococcal M protein activity and to decrease production of tumour necrosis factor-α by monocytes.

As for any use of anti-microbials, treatment should be adjusted depending on patient allergies, renal function and local anti-microbial guidelines. Following results of microbial cultures, anti-microbial treatment should be targeted to resulting organisms on senior microbiological advice. Antibiotics should be continued until no further surgical debridement is required and the patient is haemodynamically stable.

Early, wide and aggressive surgical debridement which extends to healthy bleeding margins is the mainstay of the management of NF. Delays in and inadequacy of debridement is associated with poor outcomes. If extremities are affected, amputation is often required. NF frequently requires multiple surgeries and a second debridement is often performed within 24 hours of the initial debridement(3).

Following radical surgical debridement, patients are often haemodynamically unstable and severe protein and fluid loss requires intensive care support. Haemodynamic and nutritional support are key aspects of management of NF in the ICU setting(2).

Intravenous immunoglobulin (IVIG) has been hypothesised to play a role in the management of NF. Human donor IVIG is thought to enhance bactericidal activity of serum and may bind endotoxins released by Streptococcal and Staphylococcal bacteria reducing the systemic inflammatory response. There are however no randomized controlled trials demonstrating the efficacy of IVIG in NF and so they are limited to critically ill, haemodynamically unstable patients(11).

Hyperbaric oxygen therapy has also been suggested for the management of NF. Some studies have shown elevated tissue oxygen levels to reduce oedema, stimulate fibroblast growth, inhibit bacterial endotoxin release and enhance antibiotic efficacy. Despite this, multiple studies have failed to find any significant differences in mortality, furthermore any attempts at this may delay surgical debridement and so routine use of hyperbaric oxygen therapy in NF is not recommended(3).

Ongoing aftercare of NF is an important part of the long term management of NF but is beyond the scope of this article.

Conclusion

This case report demonstrates a severe and fatal case of Clostridium septicum atraumatic myonecrosis in a patient with newly diagnosed caecal tumour. It aims to highlight the association of Clostridium septicum with atraumatic myonecrosis and gas gangrene with malignancy (most notably colorectal and haematological) and emphasise the importance of a high index of clinical suspicion leading to early diagnosis and prompt and aggressive treatment as outlined above.

MCQs

1. How can necrotizing fasciitis be distinguished from other soft tissue infections?

a. Pyrexia

b. Rapid progression

c. History of trauma

d. Pain at site

e. Odour

2. In necrotizing fasciitis why does pain out of proportion to clinical findings occur?

a. Initial fascial involvement

b. Joint involvement

c. Trauma

d. Compartment syndrome

e. Nerve damage

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3. What is the gold standard for diagnosis of necrotizing fasciitis?

a. MRI

b. CT

c. Plain radiographs

d. Clinical examination

e. Surgical exploration

4. What is the mainstay of management for necrotizing fasciitis?

a. Antibiotics

b. Blood transfusion

c. Early surgical debridement

d. Fluid resuscitation

e. Analgesia

5. How should antibiotic prescribing be guided?

a. Nature of trauma

b. Site of necrotizing fasciitis

c. Haemodynamic state of patient

d. Trust microbiology guidelines

e. Clinician choice

Answers

1. = b.

Necrotizing fasciitis is characterised by rapid progression to fulminant septic shock, potentially within hours. Pyrexia, history of trauma, pain and odour, although do occur, can also occur with many other soft tissue infections and so cannot distinguish between less severe soft tissue infections. The key here is that any suspicion of necrotizing fasciitis should be dealt with as an emergency with early senior and surgical input.

2. = a.

Pain out of proportion to clinical findings is a hallmark of the clinical presentation of necrotizing fasciitis and should always raise suspicion of a necrotizing fasciitis. This occurs due to deep fascial involvement prior to subcutaneous signs. As explained above, not all cases occur due to trauma.

3. = c.

Surgical exploration is the gold standard for diagnosis of necrotizing fasciitis and this should not be delayed for other investigations if there is a clinical suspicion. Plain radiographs and CT can demonstrate non-specific features of necrotizing fasciitis however are not diagnostic alone.

4. = c.

Early surgical debridement is key in the management of necrotizing fasciitis and should not be delayed for other treatments. Antibiotics, fluid resuscitation and analgesia are key in the management however alone are not proven to improve outcomes. Blood transfusion should only be used if clinically required.

5. = d.

Antibiotic therapy should be guided by trust microbiology guidelines until culture results are available. Early discussion with a consultant microbiologist is also advised where possible. Early antibiotics should be given no matter the haemodynamic state of the patient but should not delay surgical intervention.

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Authors

Dr Rebecca Jones
Intensive Care Department
Warrington Hospital
Lovely Lane
WA5 1QG

Dr Jeff Little
Intensive Care Department
Warrington Hospital
Lovely Lane
WA5 1QG
Jeff.Little@whh.nhs.uk

Corresponding Author

Dr Rebecca Jones
ra.jones@doctors.org.uk

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HARD TO STOMACH: PYLORIC STENOSIS IN THE ADULT

N Yang, D Graham, BH Mohamed

Abstract

Acute abdominal pain is one of the most common presentations seen in emergency departments. The cause of abdominal pain is often unidentified which can lead to a high re-admission rate. Pyloric stenosis is an exceedingly rare cause of abdominal pain in adults and similarly it is a diagnosis that is infrequently considered and often missed.

In adults it is associated with significant primary pathology such as abdominal malignancy and severe peptic ulcer disease. The decision to admit and investigate a patient should focus on the chronicity and severity of clinical presentation irrespective of patient age and response to initial management even if commoner differentials seem unlikely at first.

Case vignette

A 24 year old man with no other pertinent medical problems represents to Accident and Emergency (A&E) with abdominal pain and vomiting having been discharged twice within the last week with similar complaints. He reported sharp epigastric pain without radiation for the last 10 days without significant improvement.

This was accompanied by numerous episodes of vomiting and an absolute inability to tolerate solids and increasing difficulty keeping fluids down. He denied fevers, diarrhoea, rectal bleeding or any urinary symptoms. Investigations within the department in the week leading up to admission had been grossly normal except for some constipation demonstrated by abdominal plain film; after his last attendance he was sent home with oral laxatives.

On examination the patient appeared dehydrated but otherwise well with a soft, non distended, non tender abdomen with no rebound or percussion tenderness and normal bowel sounds. Digital rectal examination was also unremarkable. Blood tests including amylase, full liver screen and markers for coeliac and inflammatory bowel disease were unremarkable.

The only deranged biochemical parameters discovered were suggestive of a mild inflammatory response with C-reactive Protein (CRP) at 52 mg/L and White Cell Count at 13.1 x 10 Ð/L. Plain films of the chest and abdomen revealed no acute pathology.

The patient had a similar episode 7 months prior to this admission after returning from a family holiday in Egypt. On this occasion he was thoroughly investigated for an infectious source. An abdominal MRI was performed to look for an anatomical cause of this vomiting, but did not yield a clear cause for his symptoms.

This episode was self limiting and outpatient Upper Gastrointestinal Endoscopy (OGD) demonstrated Campylobacter-like organism (CLO) positive gastritis for which he was treated with a course of proton pump inhibitors (PPI) and antibiotics and remained symptom free until this recurrence.

The patient was treated symptomatically and underwent an inpatient OGD which demonstrated the following:

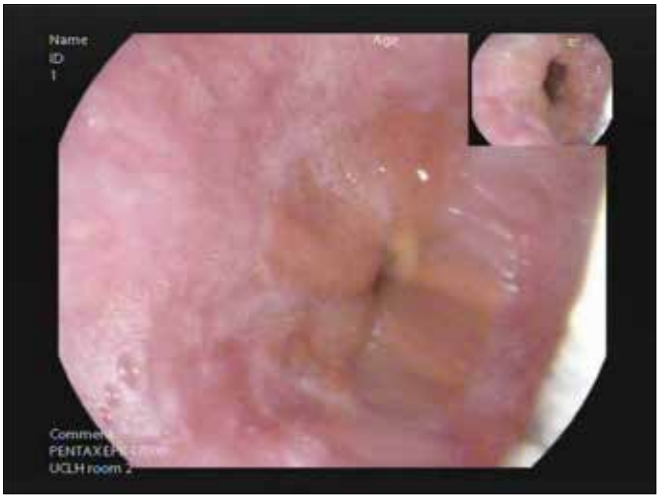

Figure 1) Appearance of a severely stenotic and deformed pylorus.

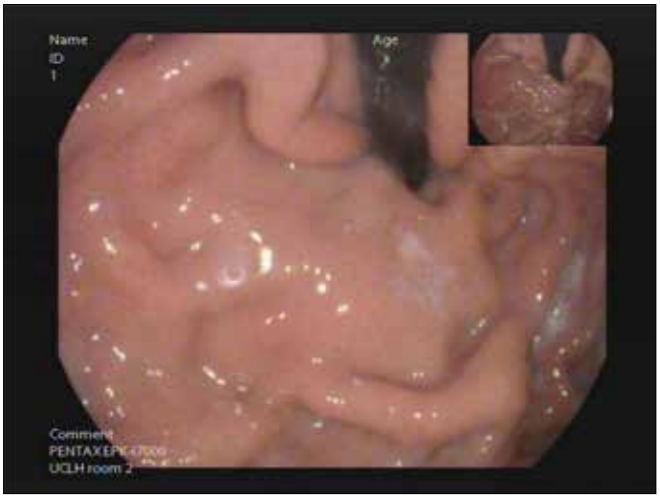

Figure 2) Inflamed antral mucosa.

HARD TO STOMACH: PYLORIC STENOSIS IN THE ADULT

N Yang, D Graham, BH Mohamed

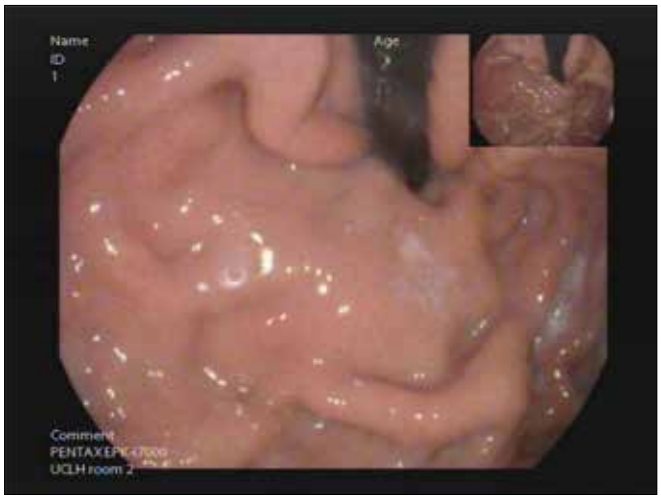

Figure 3) Antral mucosa bleeds very easily when touched.

Initial treatment with intravenous PPI cover was given and urgent CT chest abdomen and pelvis was performed to search for primary malignant pathology. The CT mirrored the endoscopic findings of the pylorus without evidence of further pathology within the body. He improved symptomatically with treatment and was discharged when able to tolerate fluids and soft diet for 5 days. He later had an elective endoscopic dilatation procedure and remains well.

Discussion

Pyloric stenosis is a rare presentation that we are more accustomed to expecting in the Paediatric population. It is a serious condition that is seen in approximately 2-5 live births per thousand and classically within the first 8 weeks of life (1).

Rarer still do we see the adult counterpart of this condition in clinical practice; in fact, it is so infrequent that we do not have reliable epidemiologic data and the literature surrounding this presentation is limited to a selection of individual case studies (2). It has been suggested that adult onset pyloric stenosis may be divided into three subtypes (3)

1. Late onset infantile hypertrophic pyloric stenosis

2. Adult idiopathic hypertrophic pyloric stenosis

3. Hypertrophic pyloric stenosis secondary to other abdominal pathology (peptic ulcer disease, malignancy, hiatus hernia and other inflammatory diseases)

Of which by far the most common is the latter, emphasising the need to accurately discern the primary pathology in such patients.

Whilst the above case illustrates that adult onset pyloric stenosis is a differential diagnosis that is often omitted in the consideration of acute abdominal pain, I must emphasise that this condition one that is vanishingly rare.

The point of this article then, is not to encourage young clinicians to go hunting after Zebras, but to safely and appropriately manage abdominal pain in A&E even when there doesn't appear to be overwhelming evidence to suggest a specific pathology.

Abdominal pain is by far one of the commonest presenting complaints to the emergency department in both adult (4) and paediatric populations (5). Therefore, that the key skill of the clinician is in differentiating between those who require prompt admission and those who may be safely discharged to be managed in the community. Whilst some cases will be clearer cut, there is a vast grey area in which we are susceptible to diagnostic uncertainty, especially given the pressures of a busy emergency department.

It has been shown that up to 40% of abdominal pain seen and treated in the emergency setting can result in reattendance within a short time span and that many of these patients do not have a definitive diagnosis by discharge (4).

Younger adult patients can often be overlooked as their symptoms resolve swiftly with supportive care and are considered less likely to have serious underlying pathology. The above case highlights the importance of objective consideration of symptom severity in abdominal pain of unclear aetiology, even in lower risk population groups.

MCQs

1. Which of the following is not considered a risk factor for infantile pyloric stenosis?

A – Male gender
B – Genetic predisposition
C – Exposure to macrolides in the first 2 weeks of life
D – Foveolar Cell Hyperplasia (FCH)
E – None, they all contribute to the risk of pyloric stenosis

2. Approximately what proportion of abdominal pain presenting to the emergency department remains without clear diagnosis by discharge?

A – 10%
B – 20%
C - 25%
D – 40%
E – 60%

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3. Which of the following scoring systems is the least likely to be useful in the emergency department setting?

A – Alvarado
B – Blatchford
C – Truelove and Witts
D – Modified Glasgow
E – Rockall

Answers

1. Answer D

Male gender is a well established risk factor with the condition being seen in 4 times as many males as females (6). Numerous susceptibility loci have been uncovered based on molecular studies, there is some evidence to suggest dysfunction of genes regulating innervation of smooth muscle cells is implicated in the pathogenesis of pyloric stenosis (7).

Early exposure to macrolides, particularly erythromycin has been associated with the subsequent development of pyloric stenosis and caution is advised with its use (8). FCH in itself is not a risk factor for subsequent development of pyloric stenosis but is associated with persistent vomiting postoperatively (9).

2. Answer D

Although this type of data is cumbersome to accumulate and difficult to interpret, the article by Graff et al (4) suggests it may be as high as this.

3. Answer E

With the exception of the Rockall score, all of the above scoring systems need only clinical and blood test parameters meaning they can easily be utilised in the acute setting. Much of the Rockall score depends on endoscopic findings and the pre-endoscope version has been shown to have little value in identifying low risk patients who are suitable for conservative management (10).

Authors

Dr Narisu Yang
Accident and Emergency SHO
University College Hospital
235 Euston Roadd
Fitzrovia, NW1 2BU

Dr David Graham
Gastroenterology Consultant
University College Hospital
235 Euston Road
Fitzrovia, NW1 2BU
david.graham14@nhs.net

Dr Besher Helmy Mohamed
Gastroenterology SpR
University College Hospital
235 Euston Road
Fitzrovia, NW1 2BU
besher.helmymohamed@nhs.net

Corresponding Author

Dr Narisu Yang
narisu.yang@nhs.net

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SPONTANEOUS INTRACRANIAL HYPOTENSION

P Reddy

Abstract

Mrs B, a 39 year old woman presented to the Accident and Emergency department with a 12 hour history of severe diffuse headache. For one month prior to presentation, she had been suffering from persistent frontal and occipital headaches, at times associated with nausea and vomiting. Lying down slightly relieved the headaches.

She also complained of bilateral tinnitus and a sensation of fullness in her ear. Her General Practitioner had been treating her symptoms as tension-type headaches. She had an unremarkable past medical and social history. Examination findings were normal. A computerized tomography head (CTH) scan showed shallow bilateral chronic subdural collections.

What are your differential diagnoses? How would you further investigate and manage this patient? This paper will focus on the relatively uncommon but clinically significant condition of Spontaneous intracranial hypotension (SIH) highlighted by a real case example. By the end, readers should have a broad understanding of the condition and be able to answer the questions posed above.

Introduction - What Is Spontaeous Intracranial Hypotension?

Spontaneous intracranial hypotension (SIH), also known as primary intracranial hypotension, is defined as an idiopathic syndrome characterised by symptoms of low-pressure headache secondary to low cerebrospinal fluid (CSF) pressure (1,2). Although SIH has become a clinically recognised entity, it still remains an uncommon and probably under-diagnosed cause of headache. Existing evidence suggests a female preponderance (F:M, 2:1) with an estimated incidence of 2-5 per 100 000 per annum and a peak age of presentation at 40 years (1,3).

Pathophysiology

Intracranial hypotension commonly results from CSF leak anywhere along the neuro-axis via a breach in the dura mater (1,3). In SIH, there is no identifiable history of preceding trauma or iatrogenic procedures that could explain the source of fluid loss (e.g. lumbar puncture, pituitary surgery, ventriculo-peritoneal shunt surgery). Most leaks are thought to occur within the spine, and a minority from the skull base (1,3,4). The majority of leaks (when identified) are found in the thoracic spine or cervico-thoracic junction (1,4).

The Monro-Kellie doctrine states that a dynamic equilibrium is maintained between the non-compressible components (brain tissue ~1400mL, CSF~150mL and blood~150mL) within the rigid cranium (5). The sum of the volumes of these remain constant, thus an increase in one causes a compensatory decrease in the other two. The clinical symptoms and imaging findings can be attributed to this theory.

A decrease in intracranial CSF volume (the primary pathology in SIH) may cause a postural headache (aggravation of pain when upright) in two ways: 1) the downward displacement of brain causing meningeal traction – the meninges are extremely pain sensitive structures, 2) compensatory dilatation of the vascular spaces due to low intracranial pressure, leads to subdural effusions or haematomas (3,5).

Are There Any Pre-Disposing Risk Factors For Sih?

SIH is commonly observed in patients with connective tissue disorders such as Marfan syndrome, Ehlers-Danlos syndrome and polycystic kidney disease, conditions associated with dural abnormalities such as diverticuli or meningoceles – CSF filled dural out-pouchings (1,3,4). These are areas of weakness subject to spontaneous tears/dehisence (1). However, most patients have no identifiable risk factors (1). Table 1 outlines some causes of primary and secondary low-pressure headaches.

SPONTANEOUS INTRACRANIAL HYPOTENSION (PRIMARY)	SECONDARY INTRACRANIAL HYPOTENSION
1. Idiopathic 2. Connective tissue disorders a. Marfan syndrome b. Ehlers-Danlos syndrome c. Polycystic kidney disease 3. Neurofibromatosis 4. Congenital focal absence of dura 5. Degenerative dural tears a. From vertebral osteophytic bone spurs	1. Lumbar puncture 2. Overshunting (excess CSF drainage) via Ventriculo-peritoneal shunt device 3. Trauma 4. Many neurosurgical interventions

Table 1: Causes of intracranial hypotension.

What Are The Clinical Features Of Sih?

Although there are the no pathognomonic features of SIH, the most prominent clinical symptom is headache. It can be either gradual or thunderclap in onset. A "generalized" or "throbbing", occipital or frontal headache that rapidly increases in intensity within minutes of being upright and settles on lying down is supportive (1).

This feature may become less obvious with chronicity of symptoms, therefore, it is important to elicit the initial nature of the headache at the very beginning of symptoms (7). Some patients may describe a delayed response to changes in posture, with symptoms peaking after several minutes to hours. In the history, secondary causes of a low pressure headache need to be excluded (such as recent lumbar puncture). More often than not, the clinical examination is normal, although stigmata of connective tissue disorders can sometimes be appreciated (1,6). Some associated features are outlined in Table 2 (1,2,6). The prevalence of these has not been studied.

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ASSOCIATED SYMPTOMS OF SIH
1. Nausea and Vomiting
2. Neck Stiffness & interscapular pain
3. Visual:
a. Diplopia
b. Blurred vision
4. Auditory:
a. Tinnitus
b. Altered hearing

Table 2: Common symptoms associated with SIH.

Due to variation in symptom severity and presentation, the diagnosis remains difficult, and many patients with mild headaches may be incorrectly or undiagnosed. The International Headache Society developed the 3rds diagnostic criteria to aid detection of SIH, Table 3 (1,7,8).

DIAGNOSTIC CRITERIA FOR SIH
A. Low CSF pressure<60mmH20/or/Evidence of CSF leakage on imaging / imaging consistent with Intracranial hypotension / or / both
B. Headache has developed in temporal relation to the Low CSF pressure or leakage / or / has led to its discovery
C. Not better accounted for by another diagnosis in the ***International Classification of Headache Disorders*** (3rd edition)

Table 3: Diagnostic Criteria for SIH.

The differential diagnoses for SIH are similar to those of any patient presenting with headache. A focused history and examination will identify distinguishing features – outlined in Table 4; although this list is not exhaustive. Investigations should be targeted accordingly.

DIFFERENTIAL DIAGNOSES	DISTINGUISHING FEATURES
Migraine	Preceding aura, photophobia/phonophobia, **No postural element**
Chronic Daily/ Tension Headache	Pressure/tight band-like sensation around head Possible history of chronic analgesia use **No postural element**
Subarachnoid Haemorrhage	Sudden onset severe headache, neck stiffness Nausea & vomiting **No postural element**
Venous Sinus Thrombosis	Progressive or sudden onset headache Oral contraceptive pill in drug history Dehydration **No postural element**
Meningitis	Rash, fever, photophobia/phonophobia Neck stiffness **No postural element**

Table 4: Important differential diagnoses and some distinguishing features.

What Investigations Are Useful In The Diagnosis Of Sih?

1) Imaging

Routine blood tests are valuable in ruling out differentials and obtaining baseline measurements, however, imaging is the essential diagnostic tool in confirming SIH. An active site of CSF leak is not always detected and imaging is largely focused on identifying signs compatible with SIH. The modality of choice is magnetic resonance imaging (MRI) of the brain with gadolinium contrast.

Loss of CSF volume in the non-collapsible rigid cranium is compensated by development of subdural CSF collections (hygromas) and increased venous return (1,6,9). Table 5 lists radiological findings on MRI brain, a combination of which is attributed to SIH (1,3). An MRI venogram can rule out sinus thrombosis and depict venous engorgement. It is important to note that individually these signs are not specific to SIH, and 20-30% of patients may have a normal scan (10).

MRI BRAIN IMAGING FINDINGS ASSOCIATED WITH SIH
1. Subdural collections (most commonly noted feature)
a. Subdural Hygromas
b. Subdural haematomas
2. Pachymeningeal thickening (Image 3)
a. Diffuse smooth dural enhancement pattern seen with contrast administration
3. Enlarged pituitary gland/ pituitary hyperaemia
4. Engorgement of cerebral venous sinuses
a. Increased blood volume in the high compliance venous system
5. Reduced CSF Volume leading to:
a. 'Sagging' brainstem towards foramen magnum
b. Mild degree descent of cerebellar tonsils through foramen magnum
c. Downward drooping of the corpus callosum splenium

Table 5: Imaging findings on MRI associated with SIH.

Although not as sensitive as MRI, computerised tomography head (CTH) is likely to be the most common primary imaging and may also demonstrate some of the above features (3). Image 1 illustrates the bilateral shallow concave hypodense lesions seen on the CTH of Mrs B. These were later confirmed on MRI brain (Image 2) in addition to a prominent pituitary gland and mild drooping of the corpus callosum splenium. Image 3 highlights some other MRI appearances associated with SIH (1). CT myelography or MRI whole spine may be conducted if the diagnosis is in doubt or localisation of the leak is required for directed treatment (3,4).

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2) Other Investigations

A CSF pressure <60mmH2O (70-200mmH2O) favours intracranial hypotension, however, lumbar puncture (LP) is not necessary if patients have positive MRI findings and a consistent history. LP results are non-specific; the opening pressure may not always be low, and CSF analysis may reveal pleocytosis or elevated protein count (4,6).

What Are The Management Options?

There exists no guideline on optimal management and patients often recover spontaneously. Despite evidence being largely anecdotal, the consensus is to follow an initial trial of conservative measures in the form of hydration, bed rest, regular analgesia and anti-emetics (1,9).

This may suffice for some, but in the event of failure of symptomatic relief, the next step is autologous epidural blood patch (EBP) (1,3,4,10). The process involves instilling approximately 20mL of the patient's own blood into the epidural space – usually a 'blind' injection into the lumbar spine regardless of the location of the leak (1,4). Some patients report immediate symptomatic relief, the underlying theory is the result of increased CSF pressure due to increased CSF volume within the spinal canal and a tamponade effect on the dural defect (1).

Nevertheless, there are no controlled studies for efficacy of EBP and the response rate is variable with up to 50% of patients requiring repeated EBPs due to refractory SIH (1,10). The location of the leak may be identified on CT myelography and the EBP can be directed at the corresponding spinal level. A lack of response to the above measures makes management even more complex. Other experimental treatment options (despite no firm evidence base on positive outcomes) include CT-guided EBP with fibrin glue injection and neurosurgical intervention via repair of identified leaks (1,11).

What Is The Likely Prognosis?

Outcomes vary from complete resolution to recurrent leaks with unremitting symptoms. Described complications include rebound intracranial hypertension after successful treatment, subdural haematomas complicating subdural hygromas (through rupture of bridging veins) and rarely cerebral venous sinus thrombosis (6). Re-presentation or an acute change in nature of symptoms should prompt re-investigation.

Mrs B - Case Continued

Mrs B's symptoms had been present for one month. On further questioning, she admitted to experiencing intensifying occipital headaches on sitting upright or standing. She also described a 'whooshing' sensation in both ears, suggestive of tinnitus. Examination showed no neurological deficit. Blood tests were normal. Her radiological investigations (Image 1 & 2) along with her clinical symptoms were consistent with a diagnosis of SIH.

She was admitted under the neurosurgical team for monitoring and conservative management. After 2 days, symptoms had settled and Mrs B was discharged with self-care guidance and advised to attend her local emergency department (ED) if symptoms worsened or reappeared.

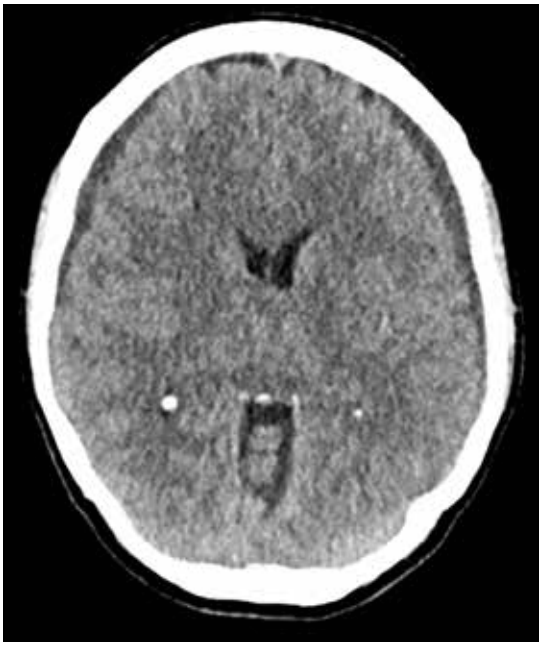

Image 1: CTH of Mrs B showing bilateral shallow hypodense subdural collections (5mm) in a patient with SIH.

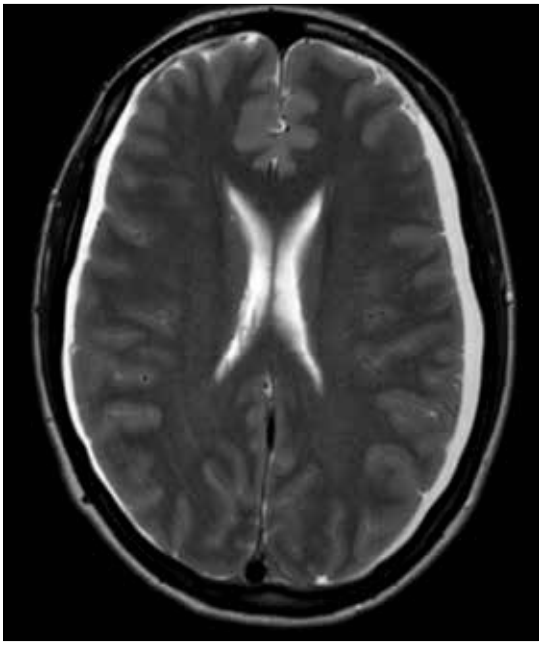

Image 2: Axial MRI T2 of Mrs B showing bilateral hyperintense subdural collections.

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Three weeks following this, Mrs B presented to ED in a similar fashion. She was admitted under the neurologists and underwent two blind EBPs before being discharged again. She reported instant but only temporary relief of symptoms following the procedures. One week later, Mrs B returned to hospital with a severe constant headache which had now lost its postural element. An MRI Brain scan showed that her bilateral subdural collections had increased in size with haemorrhagic transformation into haematomas, thus creating a mass effect. She was referred to the neurosurgeons.

Management involved bilateral burrhole evacuation of the haematomas followed by 4 days of strict bed rest. An MRI whole spine didn't identify a source of leak. Mrs B described immediate symptomatic relief following surgery and remained clinically stable. She was discharged following a period of observation and in-patient rehabilitation, with a plan for routine follow up. This is an example of a case with no identifiable cause for SIH and a high probability of recurrence.

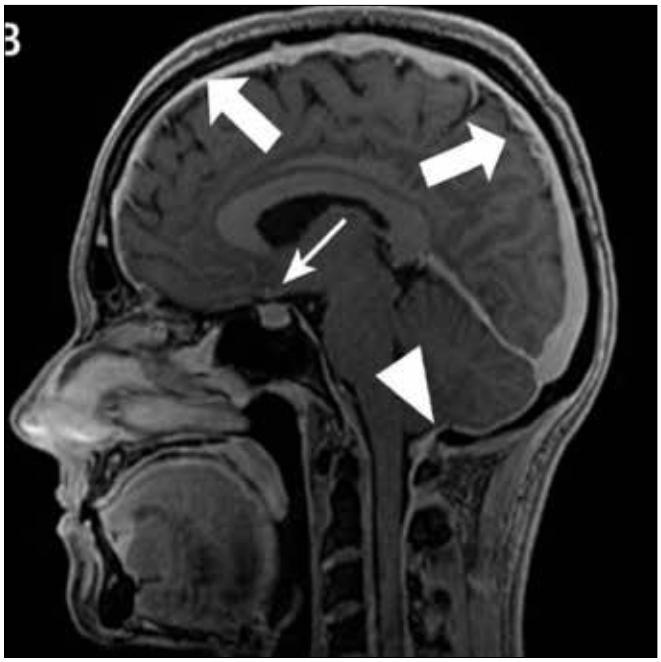

Image 3: Sagittal MRI wiith gadolinium showing: a) Diffuse pachymeningeal enhancement + dural thickening (thick arrows), b) Prominent homogeneously enhancing pituitary gland (thin arrow) (1)

Conclusion & Learning Points

1. SIH is an under-appreciated but important clinical syndrome that poses diagnostic and treatment challenges in the patient who presents with headache to ED.

2. Increasing awareness of clinicians in recognising characteristic features in the history and investigations will aid timely diagnosis and management.

3. MRI Brain with gadolinium contrast is the ideal investigation to support a diagnosis of SIH.

4. Conservative management is the mainstay of initial treatment, but appropriate discussions with the neurosciences team can help identify those who will benefit from invasive options like EBP or primary repair of identified defects.

5. Further controlled trials are required to investigate the benefits of various proposed treatment strategies for refractory cases.

Self-Assessment Questions - MCQ Best Of 5

1. A possible cause of SIH can be

a) Following spinal anaesthesia
b) Following insertion of a ventriculo-peritnoeal shunt device for long standing intracranial hypertension
c) Associated with a past medical history of migraine
d) Following trauma to the cervical spine
e) Associated with osteoarthritis affecting the vertebral column

2. Supportive features of SIH

a) Always associated with positive findings on MRI Brain
b) Headache is always worse immediately after sitting upright or standing
c) Auditory and visual symptoms may be present
d) Males and females are equally affected
e) An epidural blood patch (EBP) is considered first line management option for suspected SIH

3. Differential Diagnosis of SIH include:

a) Meningitis
b) Subarachnoid Haemorrhage
c) Migraine
d) Venous sinus thrombosis
e) All of the above are possibilities

4. Brain imaging findings include all except:

a) Enlarged ventricles
b) Bilateral subdural collections
c) Prominent pituitary gland
d) Meningeal enhancement
e) Degree of downward descent of the brainstem through the foramen magnum

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5. A patient previously diagnosed and treated conservatively for SIH represents with a constant severe headache unrelated to posture. Which of the following statements are false? Select the 2 correct options

a) Repeat imaging of the brain is not always necessary
b) This presentation could suggest a possible complication of SIH such as acute intracranial bleeding i.e. Subdural haematomas
c) Venous sinus thrombus is a possible complication of SIH and can cause presentation as described above
d) A repeat EBP is almost always indicated
e) Rebound intracranial hypertension is a noted complication of EBP-treated SIH

Answer

1. e)

Osteoarthritis (OA) affecting the vertebral column: OA of the spine can cause osteophytes or vertebral body spurs. These are rarely associated with an increased risk of dural tears with minor trauma. All the other options listed are secondary causes of low-pressure headaches.

2. Answer: a)

Patients may describe blurred vision, tinnitus or a sensation of fullness in the ears. MRI imaging including the other forms described in the article can be normal. With chronic symptoms, the postural element of the headache can become less prominent. SIH is associated with a female dominance. Conservative management is always first line, and invasive options such as EBP or surgical repair are only considered following failure to respond.

3. Answer: e)

All of the above are possibilities: It is important to remember that 'common things are common'. The above differentials will need to be ruled out in any patient presenting with recent onset headache. A careful history and examination will help identify defining features of SIH – in particular, the postural nature of the headache.

4. Answer: a)

Enlarged ventricles: All the other options are findings seen in patients with intracranial hypotension. In the context of headache presentations, enlarged ventricles are commonly associated with hydrocephalus or benign intracranial hypertension.

5. Answer: a) & d)

Repeat imaging of the brain is always indicated when a patient presents with a change in the nature of the headache from previous occasions. Imaging can help identify potential complications; SDH, rebound intracranial hypertension and cerebral venous sinus thrombus. Management should be targeted according to an accurate diagnosis, therefore EBP should not be the default treatment option.

Authors

Priyanka Reddy
SHO
Southmead Hospital
Southmead Road
Westbury-on-Trym
Bristol
BS10 5NB
pri.reddy03@gmail.com

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TRAUMATIC BLADDER RUPTURE

RM Warner, S Tang, C Bastianpillai, JSA Green

Abstract

We report the case of a 45 year old man who suffered an intraperitoneal bladder rupture, necessitating open surgical repair, secondary to blunt trauma from falling down the stairs whilst under the influence of alcohol. Bladder rupture is an important differential diagnosis in the presentation of pain following abdominal trauma, particularly if there is accompanying visible haematuria, and difficulty passing urine. We present the key management steps including the correct choice of imaging, identifying the site of injury and when to call the urology team, as well as some common pitfalls to avoid as a non-specialist such as when not to catheterise and missing a small rupture or additional injuries. The full management pathway of different rupture types is described.

Case History

A 45 year old gentleman was reviewed in the Accident and Emergency (A&E) department following a fall down the stairs. Whilst heavily intoxicated, he tripped and fell forward hitting his abdomen on the stairs before sliding to the floor. He was able to get up independently. After a couple of hours he developed worsening lower abdominal pain and noticed haematuria. An ambulance was called.

In A&E further history taking revealed no loss of consciousness or head injury, no chest, neck or back pain and that he had been walking around without any apparent neurological issue prior to calling the ambulance.

He had a past medical history of type 2 Diabetes Mellitus which was diet-controlled, no history of previous abdominal surgery and no previous urological issues.

The patient was assessed using the Advanced Trauma Life Support (ATLS) principles. Primary survey demonstrated no airway, cervical spine, or breathing abnormalities with a normal chest x-ray. Assessment of circulation and haemorrhage identified a heart rate of 106 beats per minute and he was normotensive. There were signs of generalised peritonitis with guarding, percussion and rebound tenderness (although the patient did not exhibit severe pain) and blood at the urethral meatus. There was no evidence of pelvic or long bone tenderness. His Glasgow Coma Score was 15/15.

A Focussed Assessment with Sonography for Trauma (FAST) scan was performed by the A&E registrar. This demonstrated fluid in the hepato-renal and spleno-renal recesses and a bladder volume of 70ml.

Initial management included intravenous access and fluids, analgesia and antibiotics. Bloods were sent including a full blood count, urea & electrolytes, clotting and group & save. A CT scan of the head, abdomen and pelvis was performed.

The CT head demonstrated no acute pathology. CT of the abdomen and pelvis with contrast in the portal-venous and urographic phases demonstrated an intraperitoneal bladder rupture with a defect in the dome of the bladder and large amount of free intra-abdominal fluid (Figure 1). The kidneys and other intra-abdominal organs were normal.

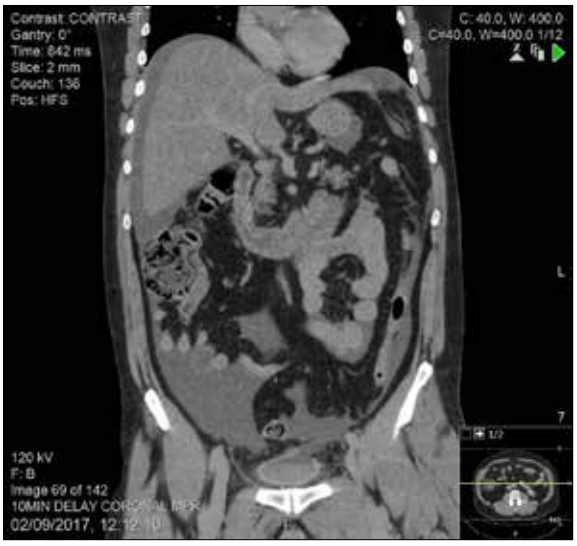

Figure 1: 1(a) Coronal CT with contrast showing bladder rupture. Yellow arrow shows site of perforation. Red arrows show free fluid.

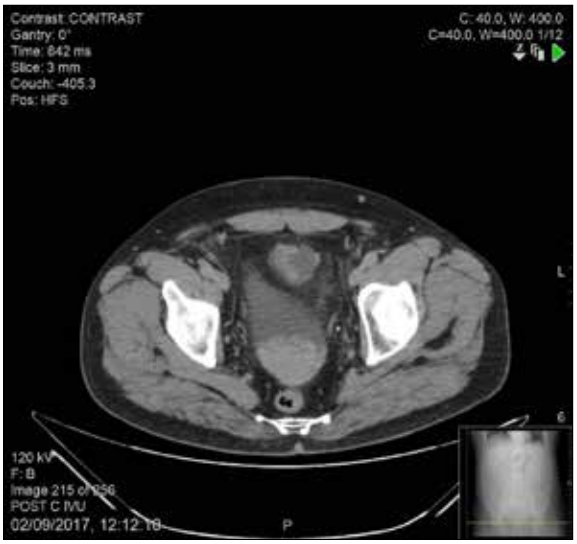

Figure 1(b): Axial CT with contrast showing bladder rupture. Yellow arrow shows site of perforation.

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A secondary survey revealed no other injuries. The patient was catheterised and taken to theatre by the urology team.

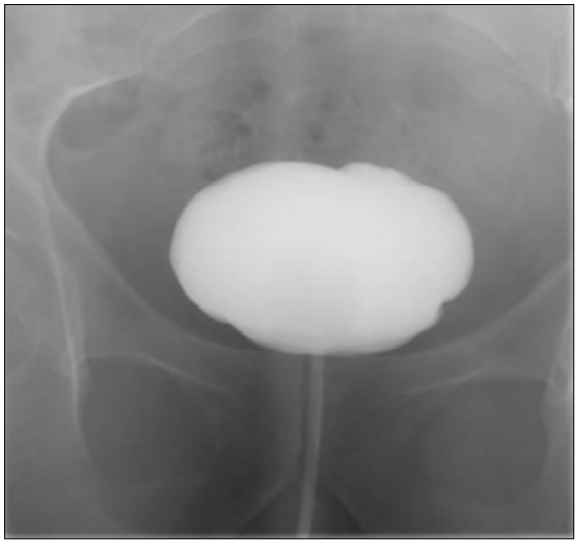
In theatre a diagnostic cystoscopy demonstrated a large 5cm tear in the bladder which had appeared to have sealed itself off. The bladder mucosa was otherwise normal with no evidence of malignancy and both ureteric orifices were uninvolved. No urethral injury was present. Lower midline laparotomy was then undertaken.

A 3-layer closure with absorbable sutures was performed and integrity tested by filling the bladder. No other injury was found during the laparotomy. A standard abdominal wall closure was executed with clips for the skin and a large Robinson's drain left with tip adjacent to the bladder repair.

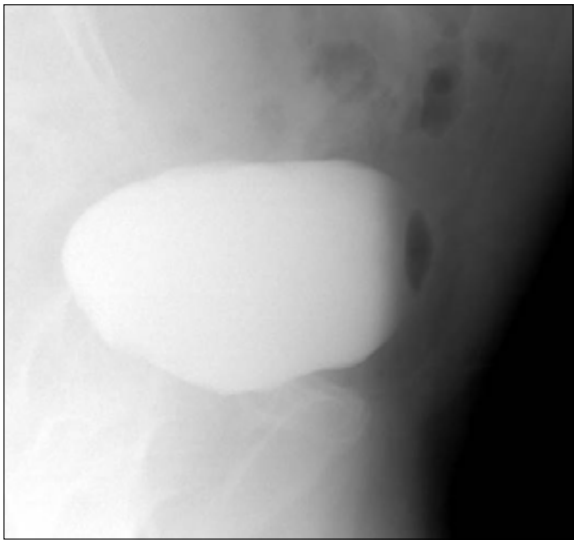
Post-operatively, the patient had an excellent recovery. The drain was removed after approximately 36 hours and he was discharged 2 days after his surgery eating and drinking normally, fully ambulant and with urethral catheter in situ.

After 10 days his wound clips were removed by the nurse at his GP surgery. A retrograde cystogram was performed via the catheter at 3 weeks post-surgery which demonstrated no leak of urine (Figure 2) and he underwent a successful trial without catheter (TWOC) following this.

Figure 2: Retrograde cystogram with (a) AP view and (b) lateral view demonstrating no extravasation of contrast.



(a)



(b)

Discussion

Bladder rupture is an important differential diagnosis in the work-up of a trauma patient with abdominal pain. Classic descriptions of bladder rupture following blunt trauma include where there is pelvic fracture, rapid deceleration injuries for example a seatbelt injury with a full bladder and, as in this case, the intoxicated patient with a full bladder falling on to the abdomen.

Drinking large volumes of alcohol can promote a diuresis leading to a very full bladder. If a bladder rupture is suspected the key questions to answer are: firstly, whether it is intraperitoneal (urine leaking in to the peritoneal cavity) or extraperitoneal (urine remains contained within the extraperitoneal space), and secondly, whether any other injuries have occurred. Answers to these will alter management.

Aside from lower abdominal pain, features that may help you to suspect a bladder injury include visible haematuria, blood at the urethral meatus and disruption to urinary voiding. This can range from intermittent interruption of the stream to the total inability to pass urine. Previous series' of bladder trauma patients suggest that visible haematuria is almost always present (1) and therefore should always prompt further imaging. Approximately 80% of bladder ruptures following blunt trauma are associated with pelvic fracture (2) and thus this must be excluded.

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It is also important to identify whether a concomitant urethral injury has occurred. This happens in 10-20% of cases (3), and is seen more commonly in instances of extraperitoneal rupture and pelvic fracture rather than cases such as that presented here.

Importantly, urethral injury can present with similar features to an isolated bladder rupture including the inability to void and blood at the meatus. It is vital that this possibility is identified before blindly placing a urethral catheter and is a potential pitfall for the inexperienced clinician to fall in to.

An absence of blood at the meatus does not rule a urethral injury out (4). Specialist input is required early on. If the patient is in urinary retention, current European Association of Urology (EAU) guidelines recommend that a single attempt at urethral catheterisation in a partially disrupted urethra could be carried out in experienced hands (5).

Where not possible, caution should be taken by juniors and non-specialists before considering percutaneous suprapubic catheterisation because of the possible presence of pelvic haematoma often associated with pelvic fractures. An open surgical approach to diverting the urine is usually required in these circumstances.

To confirm the diagnosis of a bladder rupture and determine whether it is intraperitoneal or extraperitoneal imaging is required. EAU guidelines recommend that CT Cystography is the gold standard imaging modality (5).

Alternatively, a fluoroscopic retrograde cystogram can be performed. CT has the advantage of diagnosing more complex injuries and trauma to other organs. Both of these methods involve retrograde filling of the bladder with contrast via a catheter in the urethra. The more commonly performed CT with intravenous contrast and excretory phase scan is may not be adequate because the bladder may be insufficiently filled to demonstrate a leak.

Standard management of an extraperitoneal bladder rupture is conservative with catheter drainage. Exceptions include complex injuries and where surgical management of other injuries is required. The catheter is then usually removed following a cystogram confirming no extravasation of urine, typically after 10-14 days (6).

Intraperitoneal ruptures, as in this case, are managed surgically. This is usually performed with an open approach and closure in 2 layers. More recently, single layer closures (2) and laparoscopic approaches (7) have been used in isolated intraperitoneal bladder ruptures, however, these are not common practice and are perhaps better suited to the repair of iatrogenic ruptures noted at the time of injury.

Finally, it is useful to measure the serum urea and electrolytes. Intraperitoneal ruptures can cause an apparent acute kidney injury with a rise in the serum urea and creatinine. This is in fact brought about by the reabsorption of the leaked urea and creatinine, rather than renal impairment, and would not usually be seen to such an extent in an extraperitoneal rupture.

In conclusion, we present a classic description of intraperitoneal bladder rupture following a fall on to the abdomen in an intoxicated patient with a full bladder. The trauma can often appear inconspicuous in these situations and therefore the admitting doctor must have a low index of suspicion for a bladder injury. Following immediate assessment and resuscitation, early imaging is required to make the diagnosis and categorise the injury. The urology team should be involved from the point of diagnosis to decide on a conservative or operative management approach.

Test Yourself (Best of 5 Questions)

1. A trauma patient with blood at the urethral meatus and an earlier report of visible haematuria is unable to pass urine. What would be your first step in attempting to relieve the urinary retention?

a) Try urethral catheterisation yourself

b) Ask your A&E registrar to attempt urethral catheterisation

c) Ask the urology team to insert a percutaneous suprapubic catheter

d) Ask the urology registrar to attempt urethral catheterisation

e) Prepare the patient for the urology team to take them to the operating theatre immediately

2. You have reviewed a trauma patient in A&E resus who you suspect has a bladder rupture. You need to make the diagnosis so that you can instigate management and refer to the correct speciality. What is your imaging modality of choice?

a) Bladder scan

b) Ultrasound

c) CT abdomen and pelvis with intravenous contrast

d) Plain cystogram (fluoroscopic)

e) CT cystogram

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Answers

1. The correct answer is d)

The concern here is that the patient may have a bladder and/or urethral injury. EAU guidelines recommend a single attempt at urethral catheterisation by an experienced hand is the appropriate first step. This should not be attempted by a foundation doctor or A&E registrar. Percutaneous suprapubic catheterisation, even with ultrasound guidance, is not recommended unless the bladder is full and any pelvic haematoma has been ruled out. If urethral catheterisation is not possible, open cystostomy is the best option.

2. The correct answer is e)

A bladder scan is a non-invasive means of assessing current bladder volume using ultrasound. It is unable to identify whether there is a bladder rupture and in circumstances of pelvic trauma is unreliable in assessing volume as it may pick up pelvic haematoma. In skilled hands ultrasound may be able to identify free fluid either in the abdomen or in the extraperitoneal perivesical space. However, it is not a conclusive investigation and not recommended in guidelines.

A CT with IV contrast will usually pick up a bladder rupture and determine whether it is intraperitoneal or extraperitoneal. However, passive bladder filling during this study is sometimes not enough to exclude a rupture as a larger volume is needed. A plain cystogram is a correct answer and if performed correctly has a very high sensitivity and specificity. However, because of the added benefits with performing a CT such as identifying complex injuries involving the bladder neck and trauma to other organs, a CT cystogram is considered the gold standard.

Authors

Mr Ross M. Warner
Urology research and clinical fellow
Department of Urology
Whipps Cross University Hospital
London
E11 1NR

Miss Sarah Tang
Urology ST6 registrar
Department of Urology
Whipps Cross University Hospital
London
E11 1NR

Mr Christopher Bastianpillai
Urology research and clinical fellow
Department of Urology
Whipps Cross University Hospital
London
E11 1NR

Mr James S. A. Green
Consultant Urologist
Department of Urology
Whipps Cross University Hospital
London
E11 1NR

Corresponding Author

Mr Ross M. Warner
ross.warner@nhs.net
rossmark.warner@bartshealth.nhs.uk

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ACUTE LIMB ISCHAEMIA - CASE & TOPIC REVIEW

A Bharmal, K Varty

Abstract

Acute limb ischaemia (ALI) is an emergency that a foundation doctor should be confident in recognising and initiating early management. ALI is a sudden reduction in limb perfusion within 14 days. In as little as 6 hours there can be extensive and rapid tissue necrosis resulting in the loss of limb and life (1). It is an emergency to consider when working in all specialities because it can present as a complication or side effect of multiple conditions and/or treatments - our case study will provide an example of this. Our review gives a foundation doctor a framework to understand ALI by discussing: pathogenesis, presentation and management.

Case Report

A 66 year old female presented to the Emergency Department (ED) with abdominal pain and vomiting for 10 days. On taking a full history it was noted that for the last 3 days she had developed severe right leg pain, weakness and numbness. Relevant past medical history included colorectal cancer (T3 N2 Dukes C2) which she had a right hemicolectomy the previous year and was currently on her first cycle of adjuvant chemotherapy with curative intent.

On examination she was in pain and had a tender weak leg. Her examination findings revealed a regular heart rate at 90 bpm and a blood pressure of 110/80mmHg. Her right leg had signs consistent with ALI compared to her normal left leg, these were:

- pallor
- reduced distal power with a weak dorsiflexion and plantar-flexion
- impaired sensation distally, with no sensation on the dorsum of her foot (L5/S1 dermatomal distribution)
- absent distal, popliteal and femoral pulses in the right leg.

A prompt Vascular Consultant review was sought within 2 hours of her presentation and a CT Angiography (CTA) confirmed her diagnosis of delayed presentation of an acute ischaemic limb. She had biochemical abnormalities with a raised Creatinine Kinase (CK) > 18,000 (normal range 25-200 U/L) compatible with skeletal muscle damage and necrosis.

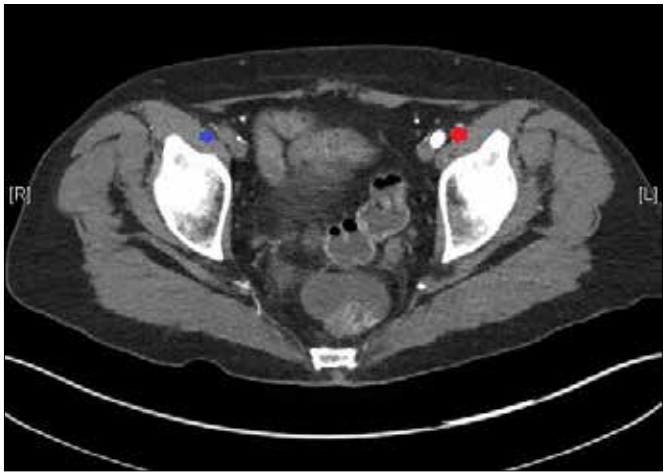

Figure 1: CTA confirming occlusion of right external iliac artery (blue arrow) vs. patent left external iliac artery (red arrow)

The CTA confirmed occlusion of the right distal external iliac artery with no distal reconstitution due to embolic occlusion.

Initial management was with fluid resuscitation, intravenous heparin 5,000 Unit bolus dose and analgesia. Within 5 hours of her presentation to the ED she had an emergency femoral and popliteal embolectomy under a general anaesthetic. A 4 compartment fasciotomy was performed at the same time as the embolectomy because she was at high risk of developing re-perfusion syndrome due to her delayed presentation and raised CK. Immediately following the procedure, she had an improvement in her circulation with a palpable dorsalis pedis pulse.

Post-operatively she developed multiple complications requiring organ support. Due to the extensive tissue necrosis, she developed rhabdomyolysis (CK peaking at 2,632) causing an acute kidney injury (AKI). Her AKI required continuous venous haemofiltration. Despite attempted correction of her biochemical and electrolyte abnormalities with haemofiltration, she developed fast atrial fibrillation (AF) which was controlled with digoxin. Her initial presenting complaint of diarrhoea and vomiting settled after cessation of her chemotherapy.

She was maintained on intravenous heparin infusion post-operatively for 24 hours, during which time her leg was closely monitored to assess for adequate re-circulation. Once there was no immediate need for further surgery she was maintained on low molecular weight heparin until her long-term anticoagulant, oral warfarin, was within a therapeutic INR > 2.5.

Her fasciotomy wounds healed well during her admission with the use of vacuum-assisted closure (VAC) dressing. VAC dressings apply negative pressure to aid healing of deep wounds and she was discharged with Plastic Surgery follow-up for further skin grafting.

Despite her lengthy admission and organ support requirement, she is now living an independent life with only a foot-drop as remaining evidence of her right acute ischaemic limb. She benefited from prompt vascular treatment and intensive care support. She has now chosen to not have any further chemotherapy trials because of the pro-thrombotic state that occurred.

Review

What is acute limb ischaemia?

Acute limb ischaemia (ALI) is when limb perfusion is suddenly reduced. This is defined as an onset of symptoms and signs within 14 days (2). Chronic ischaemia is due to the gradual onset of peripheral arterial disease (PAD) with the atherosclerotic narrowing of arteries slowly resulting in reduced distal perfusion. If this becomes severe it can lead to chronic critical limb ischaemia (CLI). This should not be confused with acute ischaemia, the presentation and management are different. In CLI there can be pain in the foot at rest, ulcers, and patches of gangrene, with a history greater than 14 days – often many weeks.

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Acute limb ischaemia has an incidence of approximately 1 in 12,000 people per year (3) and patients who have a delay in receiving treatment have the poorest prognosis (4). The amputation rate can be up to 40% and the 30-day mortality be as high as 30% (3).

Acute limb ischaemia has two common causes. The first is "in-situ" thrombosis where there is significant new thrombosis in the leg on top of pre-existing atherosclerotic disease. Pathologically this can be thought of as similar to a myocardial infarction (MI) in the leg i.e. plaque rupture, extension and thrombotic occlusion. This causes approximately 50-60% of cases of ALI (5).

The second common cause of ALI is embolism, mainly from the heart, often with atrial fibrillation or after a recent MI. This causes approximately 30% of cases of ALI. The remaining 10% of cases are due to less common causes which include: trauma, iatrogenic injury, aortic dissection, popliteal artery aneurysm thrombosis and phlegmasia cerulea dolens complicating a DVT (5).

Risk factors for developing ALI can be focussed on the likely underlying cause. Thrombo-embolic risk factors are: older age, AF, other cardiac history and aortic aneurysm. In-situ thrombotic risk is increased by: age > 50 years, smoking history, diabetes mellitus and hypercoagulable stables (5). Hypercoagulable states can either be secondary to acquired causes e.g. dehydration and cancer; or due to congenital causes e.g. thrombophilia (5). However it is important to remember that patients can develop acute limb ischaemia who do not have any of the risk factors and therefore it is always a diagnosis to consider if a patient has the signs and symptoms associated with it (6).

How to recognise it?

Acute limb ischaemia should be a differential that a foundation doctor should suspect when a patient acutely (within minutes to days) develops leg pain with loss of pulses and pallor.

The key symptoms and signs in acute limb ischaemia can be remembered by the six "P's". These are: pain, pulseless, pallor, paralysis, paraesthesia and perishingly cold. Some of these features may be present in chronic limb ischaemia e.g. absent pulses and pallor however it is the onset of the symptoms that is the key in differentiating between the two conditions.

ALI can present differently depending on whether it is caused by an embolus or thrombosis. ALI due to an embolus is typically more profound with fine reticular blanching because there is no other collateral circulation. Whereas acute thrombosis will often have a more insidious onset because often the patient will have a background of chronic ischaemic symptoms.

Remember to check for any sensory or motor changes because these are particularly useful to assess the severity of ischaemia and viability of the limb. An early indicator of more severe ischaemia is tenderness in the calf to palpation or passive stretching.

Symptoms and Signs of ALI	
Pain	May not always be present
Pulseless	It is recommended to palpate and use a Doppler probe to assess pulses
Pallor	The colour of the limb can change as the duration of ischaemia progresses. Initially it is marble white and then a light blue/ purple with a reticular skin pattern which is when the limb is still salvageable. If the limb changes to a darker non-blanching colour it may no longer be salvageable.
Paralysis	Inability to move
Paraesthesia	Loss of light touch
Perishingly cold	

Figure 2: Six features of acute limb ischaemia

How to diagnose it?

In addition to feeling for pulses in both legs, use a hand held Doppler to listen for signals, which provides an audible recording of arterial flow. If present (they may be completely absent) use the ankle pressure to measure the strength of the flow.

Ankle-Brachial Pressure Index (ABPI)

ABPI is a simple non-invasive bedside test that can provide useful information alongside the clinical suspicion of ALI.

ABPI is calculated by dividing the highest ankle systolic pressure by the highest brachial systolic pressure. The ABPI value can act as a guide as to the severity of the disease. Corresponding values are:

- Normal value: 1.0 – 1.4.
- Peripheral artery disease: < 0.9
- Critical limb ischaemia: < 0.4

Figure 3 depicts how to measure ABPI by using a Doppler probe to assess whether the pulse is audible and to record the pressure when it can be first heard.

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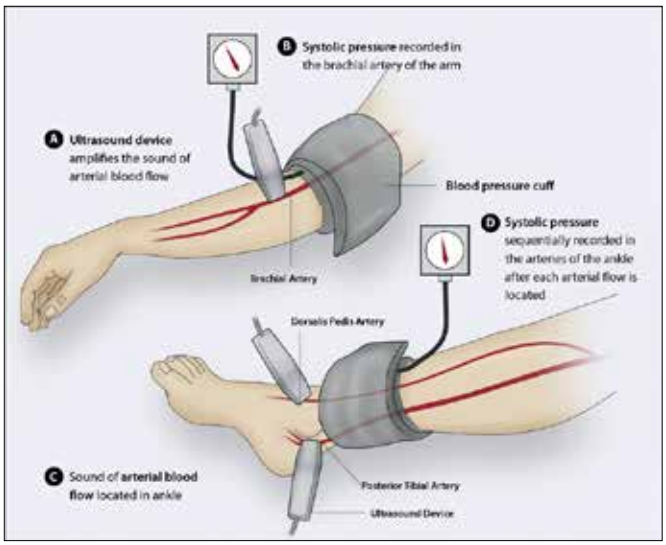

Figure 3: Calculating ankle-brachial pressure index (7)

In true acute limb ischaemia the ABPI would be very low (< 0.4) or un-recordable. The value of a better ABPI reading is that it will reassure you the leg is not imminently threatened.

Imaging

Imaging is beneficial to diagnose acute limb ischaemia however it should not be arranged if it will delay a vascular opinion and definitive management.

Colour Duplex Ultrasound can be an invaluable non-invasive tool to assess the anatomic location of the occlusion in ALI. Duplex combines two-dimensional ultrasonography with colour mapping of flow based on directional Doppler (8). However the disadvantages of Duplex ultrasound are: it does not provide full arterial imaging, it is observer dependent and can be limited by patient factors e.g. more challenging in obese patients.

More detailed imaging techniques such as CT angiography (CTA), which is the gold-standard, and MR angiography (MRA) can provide a "roadmap" of the vascular system which can aid further management planning. Both modalities depict the extent, severity, location and upstream/ downstream status of the ischaemia.

The disadvantages of CTA are radiation dose and contrast-induced nephrotoxicity in patients with severe renal failure. The nephrotoxic effects can be limited by adequate pre and post-hydration (8). MRA cannot be used in patients with: pacemakers, metal implants and patients who suffer with claustrophobia (8).

Management of Acute Limb Ischaemia

It is key to remember that early vascular opinion should be sought if there is a suspicion of ALI as this may be a key determinant in overall prognosis (1). The Rutherford classification system is based on the clinical presentation to guide overall management (9).

Pre-Operative management

It is important to medically optimise a patient with acute limb ischaemia and prepare for likely intervention. In patients with an embolus, likely secondary to fast atrial fibrillation, they may need their heart rate controlling with input from cardiology. Rehydration is an early and simple intervention to reduce the pro-thrombotic state.

Adequate intravenous fluids and catherisation to measure an accurate fluid-balance must be arranged promptly. It is also important to ensure that patients' have adequate analgesia. Analgesia should be based on the WHO pain ladder where regular paracetamol in combination with a weak opioid e.g. codeine or stronger form e.g. morphine or oramorph should be offered depending on the patient's pain score and renal function (10). ALI is typically very painful and therefore it is often necessary to titrate up to morphine.

Unless contra-indicated intravenous heparin is beneficial by limiting propagation and protecting collateral circulation. Intravenous heparin requires monitoring of the dosage by APTT ratio.

Treatment of Acute Limb Ischaemia

Treatment depends on the severity of the ischaemia and viability of the limb. In less severe cases, heparinisation alone may be sufficient if the leg remains viable and their symptoms resolve quickly (within a few hours).

If there is any concern about limb viability the definitive treatment is to re-vascularise the ischaemic limb and this can be achieved by either endovascular intervention or surgically. If it is not possible to re-vascularise the limb, then an amputation may be required.

Endovascular re-vascularisation

Endovascular re-vascularisation can be considered providing the limb is not immediately threatened. Lysis can take more than 12 hours to clear the occlusion. A diagnostic angiography is performed initially which provides useful information of the nature and length of the thrombosis as well assessing the inflow and outflow of the arteries.

A guidewire and subsequent catheter is inserted which allows direct delivery of thrombolytic agent into the thrombus. The thrombolytic drugs, for example recombinant tissue plasminogen activator or streptokinase, convert plasminogen to plasmin which degrades fibrin.

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It is possible to aspirate clots and there are endovascular catheter devices for clot disruption and extraction. These can speed up the time for endovascular clearance of any occlusion but they are currently specialised and not widely available. If there is an underlying arterial stenosis identified after lysis, it should be treated with angioplasty +/- stenting to improve flow and reduce the risk of recurrent thrombosis.

Lysis is contra-indicated for: non-viable limbs, immediate limb threat, infected bypass graft, or if the patient has any contra-indications to thrombolysis. Important contra-indications to be aware of for thrombolysis are: haemorrhage/at risk of haemorrhage, recent major surgery, stroke < 3 months and intracranial lesions.

Surgical re-vascularisation

Surgical re-vascularisation is often achieved by either thrombo-embolectomy with a balloon catheter or bypass surgery, depending on the cause and anatomical location of the ischaemia. Bypass surgery is used for cases with acute-on-chronic thrombus where underlying disease prevents the passage of a balloon catheter so clot removal is not possible and the underlying disease (stenosis or aneurysm) also requires treatment. Thrombo-embolectomy is typically used for embolic ALI.

Post-Operative Management

Following acute limb ischaemia it is important to initially monitor limb status, particularly over the first 24 hours with regular limb observations, as re-perfusion may be incomplete.

Long-term, the patient will require anticoagulation. Typically warfarin is used once within a therapeutic INR range or a newer direct oral anticoagulant could be considered. An anti-platelets can be used in conjunction or in special circumstances as a single agent e.g. if the patient has a high bleeding risk or there has been correction of the underlying

ALI aetiology. It is important to manage any cardio-vascular risk factors with pharmacological therapy, including statins and anti-hypertensives, and non-pharmacological interventions for example smoking cessation advice and regular physical exercise.

Follow-up care should be arranged regarding the cause of the acute ischaemic limb, particularly if it was an embolic event. Initial investigations include 24 hour Holter and Echocardiogram to assess for atrial fibrillation and mural thrombus respectively.

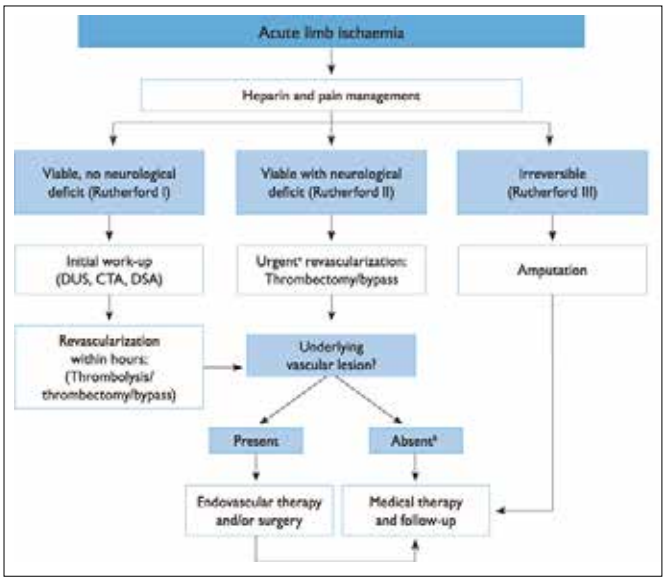

Figure 4: Management of ALI (8)

What are the complications of acute limb ischaemia?

Post-ischaemic syndromes refer to a group of complications caused by reperfusion injury (RPI). RPI causes local and systemic complications which often require Intensive Care support.

Reperfusion injury is caused by the reintroduction of blood to a limb that has been ischaemic and therefore the metabolism converts back to aerobic respiration. Subsequently there is a generation of oxygen free radicals causing a downstream cascade of activating neutrophils and proteases. The activated neutrophils adhere and damage endothelial cells which increases the permeability of the capillaries.

Local effects

Compartment syndrome

Compartment syndrome is a localised complication of reperfusion injury. There is increased permeability of the damaged capillary endothelial cells thus causing oedema and increasing the interstitial pressure of muscles that are confined within a tight fascial boundaries. Although there is re-established arterial flow the rising pressure can impair venous drainage and cause further muscle necrosis.

Patients who develop compartment syndrome present with exquisite pain, which would be greater than expected, and swelling. The diagnosis is primarily made by clinical findings, however it can be confirmed if the pressure is greater than 30mmHg. The treatment is a fasciotomy in order to release the fascial boundaries and lower the interstitial pressure.

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Chronic pain syndromes

Peripheral nerve injury can occur due to ischaemia which can cause severe neuropathic pain. Medical management includes use of various neuropathic agents e.g. gabapentin, or this can be surgically treated with sympathectomy.

Systemic effects

Depending on the extent of ischaemia, the reperfusion injury can be fatal. Damaged cells leak myoglobin, urate, potassium and phosphate which causes severe biochemical insults.

The myoglobinaemia can cause an AKI due to acute tubular necrosis. Depending on the severity of the AKI the patient may require renal replacement therapy. The leakage of electrolytes from the cells, exacerbated by the AKI, can cause cardiac arrhythmias. If the vascular permeability becomes systemic it can lead to gastrointestinal endothelial oedema and endotoxic shock.

Key learning points

1. Early recognition and management of ALI is the key determinant on the overall prognosis of the patient.

2. Clinical features of ALI are: pain, pulseless, pallor, perishingly cold, paraesthesia and paralysis.

3. Initial treatment include rehydration, analgesia and IV heparin infusion.

4. Definitive treatments can either be endovascular re-vascularisation or surgical re-vascularisation by thrombo-embolectomy or bypass surgery.

5. Reperfusion injury is due to the re-introduction of blood causing generation of oxygen free radicals that damage endothelial cells. It causes rhabdomyolysis, compartment syndrome, acute kidney injury and arrhythmias.

MCQS

1. What is the most common cause of an acute limb ischaemia?

a. Acute on chronic thrombus

b. Aortic dissection

c. Deep venous thrombosis

d. Embolic origin

e. Popliteal aneurysm

2. Which of the following is not a feature of the ALI?

a. Cold

b. Painless

c. Pulseless

d. Reduced power

e. Reduced sensation

3. Which of the following would be the most appropriate test to monitor unfractionated heparin infusion?

a. APTT ratio

b. Creatinine kinase

c. INR

d. PT

e. Xa level

4. What is the mechanism of action of thrombolytic drugs?

a. Activate neutrophils

b. Conversion of plasminogen to plasmin

c. Conversion of plasmin to plasminogen

d. Fibrin conversion to plasminogen

e. Fibrin conversion to plasmin

5. "Pain and swelling in a limb with an interstitial pressure > 30 mmHg". Which syndrome best matches this definition.

a. Acute limb ischaemia

b. Chronic limb ischaemia

c. Compartment syndrome

d. Deep venous thrombosis

e. Post reperfusion syndromes

ACUTE LIMB ISCHAEMIA - CASE & TOPIC REVIEW

A Bharmal, K Varty

MCQ answers

1. A: Acute on chronic thrombus

Acute on chronic thrombus is the most common cause of ALI, approximately 50-60% of cases. This is when a new thrombosis forms on top of an artery which has pre-existing atherosclerosis.

2. B: Painless

The six features of ALI are: pain, pulseless, pallor, paralysis, paraesthesia and perishingly cold.

3. A: APTT ratio

Intravenous heparin is an important step in the initial management of ALI. An initial 5000 units bolus is given followed by a continuous maintenance IV infusion. APTT ratio must be taken 4 hours after the start of the infusion, if this is out of range, there should be a rate adjustment and subsequent re-check of the APTT ratio. Standard target APTT ratio is 1.5 -2.5 however this may differ depending on the clinical scenario. It is important to always check your local hospital guidelines on how to prescribe, monitor and adjust heparin infusions.

4. B: Conversion of plasminogen to plasmin

Thrombolytic drugs activate plasminogen which forms plasmin. Plasmin degrades fibrin which is the molecule providing structural integrity of the thrombus.

5. C: Compartment syndrome

Compartment syndrome is a localised complication of reperfusion injury which is due to cellular oedema that occurs during ischaemia and is exacerbated by reperfusion. There is an increase in interstitial pressure in muscles that are confined within a tight fascial boundaries causing extreme pain and swelling.

Authors

Dr Aamena Bharmal
Foundation Doctor 2
Addenbrookeís Hospital NHS Foundation Trust
Cambridge
CB2 0QQ

Mr Kevin Varty
Vascular Consultant
Addenbrookeís Hospital NHS Foundation Trust
Cambridge
CB2 0QQ
kevin.varty@addenbrookes.nhs.uk

Corresponding Author

Dr Aamena Bharmal
aamena.bharmal@gmail.com

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CAROTID ARTERY STENOSIS

P Kamalathevan, M Kanapathy, A Cheng, T Richards

Abstract

Approximately one in three of all strokes are thromboembolic in origin. Carotid artery stenosis is the single most common cause of thromboembolic strokes. Atherosclerosis is the primary aetiology for carotid artery stenosis. Duplex ultrasound is the first line imaging modality to investigate both symptomatic and asymptomatic patients with high risk atherosclerotic features.

Management is dependent on patient symptoms, degree of stenosis and fitness for surgery. Surgical management involves carotid artery endarterectomy or carotid artery stenting.

We present the case of a 73-year-old gentleman who presents with a stroke with symptoms of right arm hemiparesis. Duplex US confirmed severe stenosis of the left internal carotid artery. The patient went on to have a successful left carotid endarterectomy.

Case history

A 73-year-old gentleman with a background of hypertension, type 2 diabetes and ischaemic heart disease presented to the Emergency department with symptoms of sudden right arm weakness. The patient noticed he was unable to lift his right arm whilst he was shopping. The patient denied any sensory loss, vertigo, loss of consciousness, biting of tongue or any postictal symptoms.

The vital signs in the emergency department were as follows: blood pressure 153/88, pulse rate 84min, temperature 36.2, oxygen saturations 100%, respiratory rate 18.

Physical examination revealed right arm weakness distally particularly in the extensors. Power in the lower limbs was normal bilaterally and there was no sensory deficit. The patient's coordination was difficult to test due to his weakness, but his gait was normal.

There was no abnormality in the chest x-ray and electrocardiogram (ECG) revealed normal sinus rhythm. CT head showed an ill-defined low attenuation area extending to the cortex in the superior aspect of the left superior frontal gyrus raising strong suspicion of acute infarction (Figure 1).

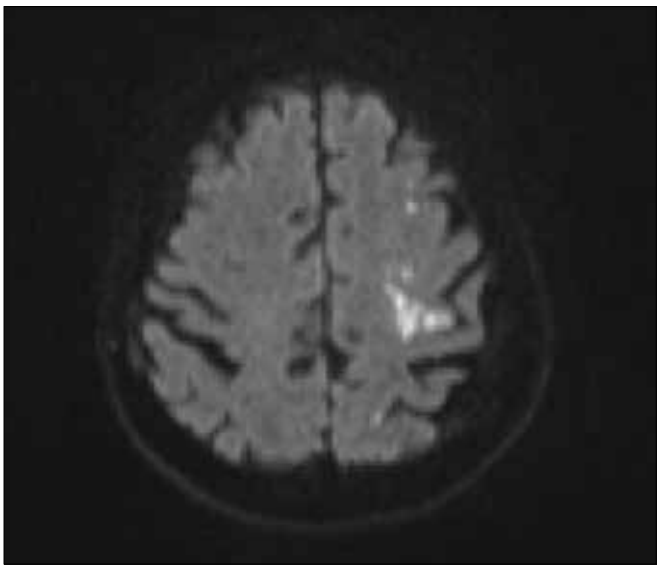

Figure 1: CT Head showing acute infarct in the left superior frontal gyrus.

Patient was reviewed by the vascular surgical team and sent for carotid Duplex assessment. The right common carotid artery (CCA) and internal carotid artery (ICA) appeared mildly calcified with no haemodynamically significant stenosis. The right external carotid artery (ECA) was patent.

The left CCA was also mildly calcified with no haemodynamically significant stenosis. However, there was a smooth surfaced, echogenic plaque in the left proximal ICA causing 70-79% stenosis. A CT Angiogram of the left ECA was then performed to further delineate the extent of carotid disease. (Figure 2).

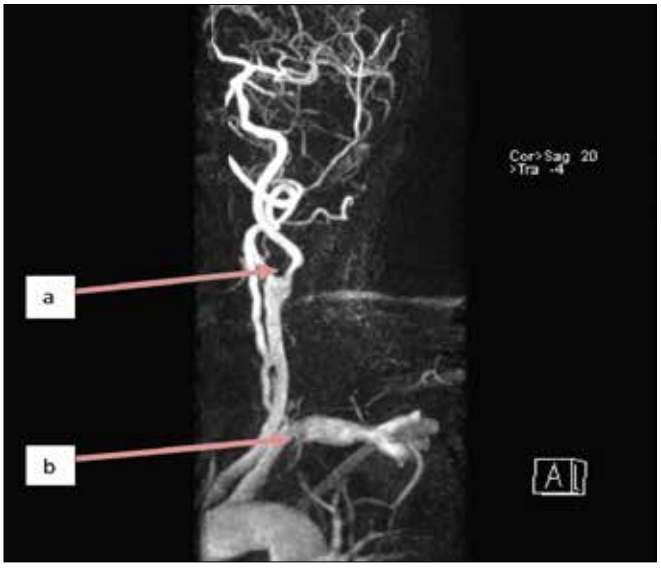

Figure 2: CT Angiogram- Significant disease in the left carotid bifurcation and proximal internal carotid with near complete occlusion of a short segment of the vessel(a). Ulcerated plaque in the proximal left subclavian (b).

CAROTID ARTERY STENOSIS

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This confirmed mild stenosis of the left common carotid with a soft ulcerated plaque at the carotid bifurcation with near complete occlusion of a short segment of the origin of the left internal carotid.

In addition, an ulcerated plaque was identified in the proximal left subclavian. A further cerebral MRI confirmed acute infarcts affecting the left-hand motor area, left middle frontal gyrus, left frontal supplementary motor area and a single focus of restriction in the left occipital lobe.

The appearances were consistent with border-zone infarction secondary to large vessel occlusion. The patient was discussed at the stroke multidisciplinary team meeting attended by stroke physicians and vascular surgeons and a decision was made to perform a left carotid artery endarterectomy (CEA) under general anaesthetic. The procedure was uncomplicated and the patient was sent home the next day on dual anti-platelet agents.

Discussion

Stroke affects over 15 million people worldwide each year. Around one in three of all strokes are thromboembolic in nature (1). Carotid artery stenosis secondary to atherosclerosis is the single most common contributor to thromboembolic strokes (2). Atherosclerosis is a chronic inflammatory condition characterised by the build-up of fibrous-lipid particles within the intimal arterial wall.

The consequence of plaque build-up is either plaque rupture or embolization to occlude another vessel; commonly the retinal vessels or the intra-cranial arteries. In asymptomatic patients, moderate-to-severe carotid artery stenosis occurs in approximately 2-5% of European women and 5-8% of European men aged over the age of 60 (3).

The development of atherosclerotic plaques is dependent upon hydrodynamic factors, low wall shear stress and hypercholesterolaemia. Other rare causes include fibromyalgia dysplasia, carotid dissection, trauma, radiation exposure (in head and neck tumours). Re-stenosis following previous endarterectomy is another recognised cause.

Atherosclerosis is characterised by the accumulation of lipid and fibrous particles in the arterial wall, usually at the carotid bulb (4). The inflammatory process involves the adherence of monocytes to the endothelial wall at points of fatty streaks. Subsequently, monocytes migrate into the intimal layer where they proliferate and differentiate into macrophages which phagocytose lipoproteins to form foam cells. The foam cells eventually release their lipid filled contents to the necrotic core of the atherosclerotic plaque.

Carotid artery stenosis can be further classified by symptomatic status or the degree of stenosis

Symptomatic status (6)(7)

- Symptomatic: *patients experience focal neurological symptoms that includes stroke (symptoms last > 24 hours), transient ischaemic attacks (symptoms resolve within 24 hours) or amaurosis fugax (transient loss of vision, usually in one eye, indicating stenosis of the ipsilateral carotid artery).*

- Asymptomatic: p*atients have no focal neurology. The diagnosis is suspected in such patients by delineating the presence of atherosclerotic high-risk factors. These high-risk factors include age >65 with the presence of either hypercholesterolaemia, hypertension, peripheral artery occlusive disease, coronary artery occlusive disease or a history of smoking. Asymptomatic patients are usually detected incidentally from imaging or on work-up for coronary artery bypass grafting. Carotid disease is most often detected in the contra-lateral carotid.*

Degree of stenosis (6)(7)

- Mild stenosis: (< 50% stenosis)

- Moderate stenosis (50% -69% stenosis)

- High grade stenosis (70%-79% stenosis)

- Critical stenosis (80%-99% stenosis) or a very tight and long stenosis, usually extending from the carotid bifurcation to the base of the skull.

- Near Occlusion/Trickle flow – tight atherosclerotic stenosis in which the carotid artery beyond the stenosis is collapsed.

- Complete occlusion.

The mainstay investigation for carotid artery stenosis in both symptomatic and asymptomatic patients is Duplex ultrasound (8). Duplex ultrasound can identify carotid artery stenosis of >70% with a sensitivity of 90% and a specificity of 94% (5). Once carotid artery stenosis has been identified the next stage step is to grade the degree of stenosis. The North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria is used to quantify the degree of stenosis (9).

CAROTID ARTERY STENOSIS

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If Duplex ultrasound is either non-diagnostic or more vascular detail is required, then a computed tomography angiography (CTA) or a magnetic resonance angiography (MRA) can be organised. These investigations are usually ordered to better define the extent of the disease.

This is especially the case for high carotid bifurcations where the carotid arch anatomy needs to be delineated for planned stenting. Furthermore, CTA and MRA helps define the degree of stenosis when Duplex ultrasound views are poor due to underlying heavy calcification or tortuous arteries.

The management approach to carotid artery stenosis is dependent upon the patient's symptoms (symptomatic vs. asymptomatic), patient's fitness for surgery and underlying carotid anatomy. Asymptomatic patients with < 70% carotid artery stenosis are usually managed medically (6)(7)(10). The criteria used to select patients for carotid revascularisation is based upon the degree of stenosis, patient co-morbidities and surgeon specific outcomes. Medical management usually involves the use of an anti-platelet agent, most often aspiring or clopidogrel (11).

Although more centres are now using clopidogrel as a first-line anti-platelet agent. Atherosclerotic risk factors including smoking, hypertension and hypercholesterolemia must also be addressed according to respective guidelines. Lifestyle modifications involves increased exercise.

Symptomatic patients with an ipsilateral stenosis of 50-69% benefit most from surgical intervention if it is done within the first 2 weeks (6)(7). Therefore, in this category it is recommended that carotid revascularisation is done as an urgent procedure within 2 weeks.

This is because the risk of recurrent stroke is early, and this risk outweighs the slightly increased risk of early surgery (6)(7). The patient must also be referred urgently to a specialist at the onset of the neurological event. There is still ambiguity over the decision to treat low risk symptomatic stenosis; but this will be answered by the European Carotid Surgery Trial (ECST) 2 trial (update May 2017: 247 patients randomised at 28 centres) (12).

Carotid artery stenting (CAS) offers an alternative to carotid endarterectomy for symptomatic and asymptomatic patients with carotid artery stenosis. However, the uptake of CAS has been low in the United Kingdom due to several reasons. Firstly, only a small number of operators are being trained.

More importantly, emerging evidence from major trials have suggested higher incidence of minor stroke rates in patients being treated for symptomatic disease (13). In addition, CEA confers lower stroke and death rates compared to CAS in patients with asymptomatic carotid artery stenosis (13). It is also worth noting that CEA can be safely undertaken under either local or general anaesthesia (14).

Conclusion

This article has highlighted a typical case of a patient presenting with sudden onset unilateral symptoms with no obvious cardio-embolic source, suggesting carotid artery stenosis to be the cause. This directed relevant investigations leading to the surgical management of this case of carotid artery stenosis.

Questions

1. Which of the following statements is not true regarding carotid artery disease?

A) Carotid artery disease is associated with peripheral arterial disease.

B) Carotid bruit suggests a stenosis of at least 70%.

C) Stenosis of the external carotid frequently results in a stroke.

D) Atherosclerosis of the carotids is usually bilateral.

E) Ultrasound is the best initial imaging modality.

2. Ipsilateral collateral filling of a proximal occluded internal carotid artery (ICA) is most commonly achieved by which of the following collateral arteries:

A) Posterior cerebellar artery collaterals.

B) Posterior cerebral artery collaterals.

C) Ophthalmic artery collaterals.

D) Middle cerebral artery collaterals.

E) Superior cerebellar artery collaterals.

CAROTID ARTERY STENOSIS

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Answers

1. C) Stenosis of the external carotid frequently results in a stroke.

Explanation:

Carotid artery disease is often associated with peripheral arterial disease and carotid artery disease. External carotid artery stenosis can result in a stroke but this is very rare.

2. C) Ophthalmic artery collaterals.

Explanation:

Collateral filling from an occluded internal carotid artery is most commonly accomplished via ophthalmic artery collaterals, therefore connecting the external carotid artery to the internal carotid artery.

Authors

Dr Pragash Kamalathevan (1)
Clinical Research Fellow
UCL Division of Surgery & Interventional Science
University College London
NW3 2QG
pragash.kamalathevan.12@alumni.ucl.ac.uk

Dr Muholan Kanapathy (1)
Core Surgical Trainee
Royal Free Hospital
Pond Street
Hampstead
London
NW3 2QG
muholan.kanapathy.13@alumni.ucl.ac.uk

Dr Amanda Cheng (2)
Clinical Research Fellow
UCL Division of Surgery & Interventional Science
University College London
NW3 2QG
amanda.cheng@nhs.net

Professor Toby Richards (1)
Professor of Surgery and Consultant Vascular Surgeon
UCL Division of Surgery & Interventional Science
University College London
NW3 2QG
toby.richards@ucl.ac.uk

Corresponding Author

Dr Pragash Kamalathevan
pragash.kamalathevan.12@alumni.ucl.ac.uk

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