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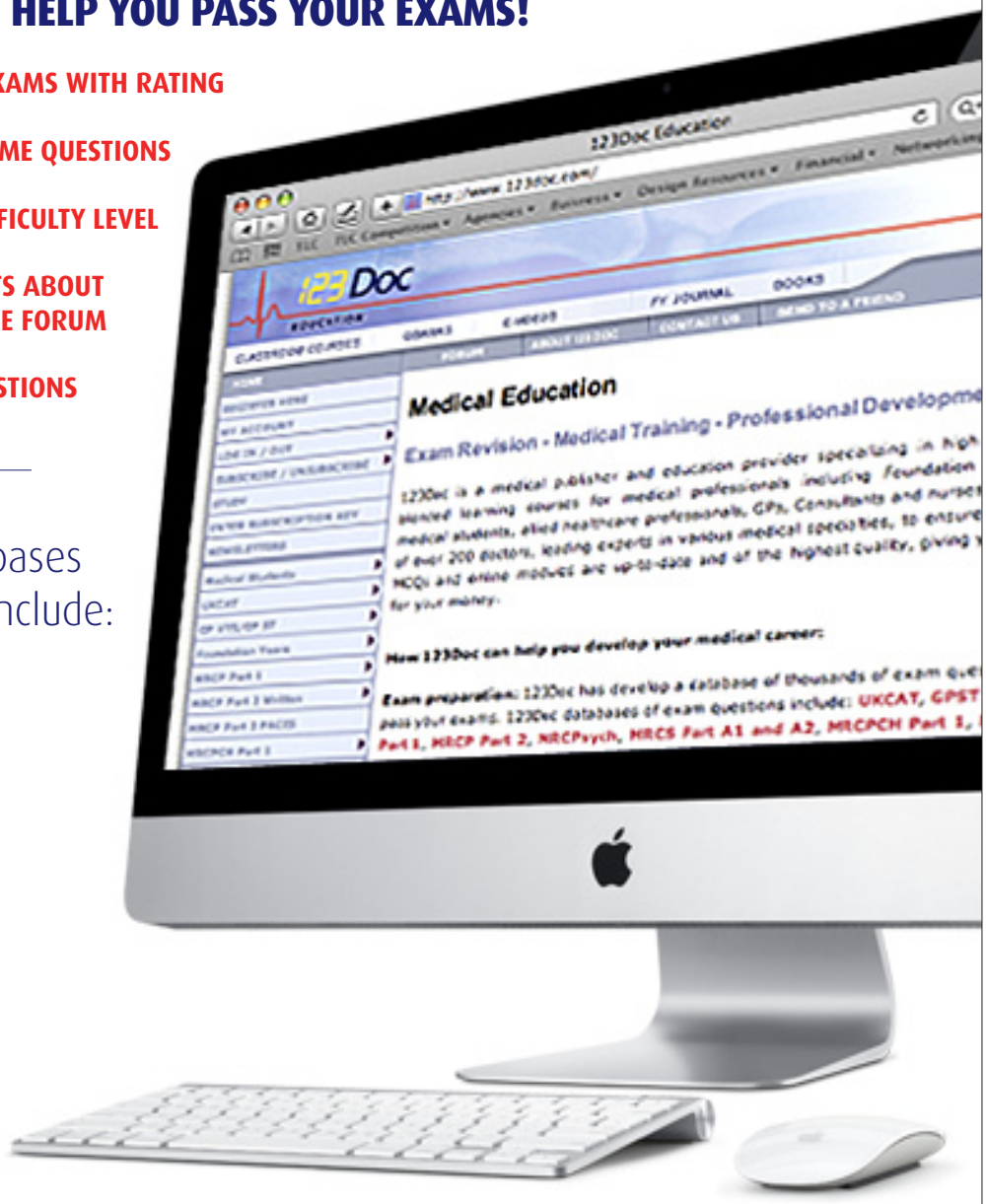

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Volume 10

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ENDOVASCULAR REPAIR (EVAR) OF A SCREENING DETECTED ABDOMINAL AORTIC ANEURYSM (AAA)

J Houghton, D Urriza-Rodriguez, M Brooks

Abstract

Abdominal aortic aneurysm (AAA) is a common disease with a high mortality if left untreated. We present the case of a 64 year old man with a screen-detected AAA who underwent successful elective endovascular aneurysm repair (EVAR).

This case highlights the importance of AAA screening in men and the role of EVAR in the treatment of AAA. We describe standard EVAR technique for an infrarenal AAA using images from the case, the early and late complications of the procedure, as well as detailing the post procedure surveillance.

Case History

A 64 year old gentleman presented to clinic with a Public Health England abdominal aortic aneurysm screening programme detected 6.1cm abdominal aortic aneurysm (AAA). He had untreated hypertension, was a non-smoker and had no other significant past medical history. His brother also had an aortic aneurysm, below treatment threshold, under ongoing surveillance.

A contrast enhanced computer tomogram (CTA) (Image 1) revealed a maximal diameter of 6.8cm with a neck length of 3.3cm (distance between lowest renal artery and the start of the aneurysm), a moderate thrombus burden and healthy common iliac arteries.

Endovascular Repair (EVAR) Of A Screening Detected Abdominal Aortic Aneurysm (AAA)
Patient Management

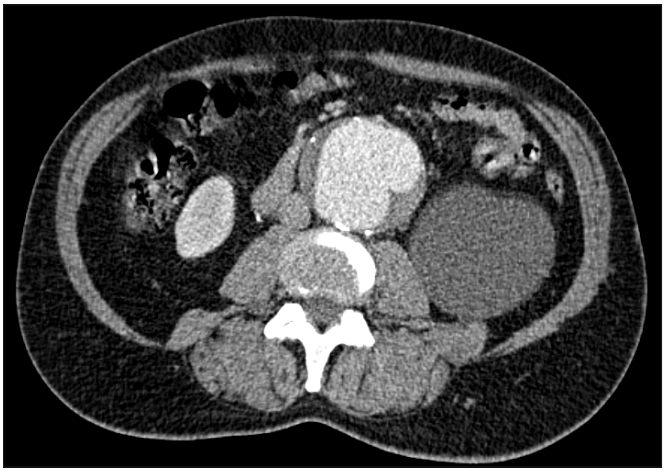

Image 1: CT angiogram showing a 6.8cm diameter infra-renal abdominal aortic aneurysm with moderate mural thrombus. Incidental large left simple renal cyst is also shown.

The patient underwent a cardio-pulmonary exercise testing (a supervised test on a static exercise bicycle to determine fitness for surgery) and was reviewed by a consultant anaesthetist. He was felt to be at low risk of major post operative complications or death for open AAA repair (a surgical 'tube' graft). After discussion at the vascular MDT it was decided that he would also be suitable for endovascular repair (insertion of a stent graft). The patient was offered the choice of procedures and opted for endovascular repair. He was commenced on Aspirin, Atorvastatin, Amlodipine and Bisoprolol prior to surgery.

The patient's was admitted and an elective EVAR was performed under general anaesthesia as a joint case between a Consultant Vascular Surgeon and Consultant Interventional Radiologist (Gore C3 Stent Graft). Post operatively he went to the vascular ward. The patient was discharged home on day 3 post op, having been kept in only due to acute urinary retention following removal of urethral catheter. The patient will undergo follow up with a CT aortogram at 6 weeks with a subsequent review in clinic and then will be entered into our EVAR surveillance programme.

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Discussion

Screening

Screening for abdominal aortic aneurysm (AAA) in men aged 65 years or above, by self-referral, has been in place in England since 2013. The aim of this screening programme is to reduce mortality from AAA rupture by 50% (1). This will be achieved by detecting asymptomatic AAA and intervening when it is considered that the risk of rupture significantly exceeds the risk of operative repair. The screening test consists of a single abdominal aortic ultrasound (US) measuring the maximal anterior-posterior & transverse diameters of the aorta (2). Table 1 indicates the outcomes of the screening test.

Maximal aortic diameter (cm)	Outcome
<3cm	No aneurysm. No further follow-up.
3.0-4.4cm	Small aneurysm. Annual US surveillance.
4.5-5.4cm	Medium aneurysm. 3 monthly US surveillance*.
≥5.5cm	Large aneurysm. Refer to vascular surgeon.

Table 1: AAA screening outcomes based on maximal aortic diameter (3).

**A rapidly expanding aneurysm ≥4.5cm increasing in size by ≥0.5cm in 6 months should also be referred to a vascular surgeon for consideration for repair.*

The UK Small Aneurysms Trial showed that for AAA < 5.5 cm in maximal diameter surveillance is as safe as operative intervention provided the aneurysm is asymptomatic and does not expand by more than 0.5cm in 6 months. (3) Aneurysms of this size have an annual rupture rate of less than one percent. (5)

Options for treatment

Whilst smoking cessation (if required) and treatment of hypertension are beneficial in management of AAA in slowing the rate of expansion and risk of rupture, no evidence exists for the medical therapy alone. (6) Therefore definitive treatment is by interventional procedure in patients fit enough to undergo surgery. In addition all patients with AAA should be treated with an anti-platelet and statin to reduce their risk of future cardio-vascular events. Statin therapy has in addition been shown to reduce peri-operative risk.

Open surgical repair (OSR) was first described in 1950 and has been the mainstay of AAA repair until recent years. Elective OSR is a major operation that carries with it up to a 5.5% 30-day mortality (5). Patients typically spend 1-2 days on ITU, a further week in hospital and anything from 3-6 months before fully recovered. It carries significant risk including limb ischaemia, bowel ischaemia, major haemorrhage, DVT/PE and significant cardio-respiratory risk. Patients are at high risk of post op ileus and hospital acquired pneumonia (6,7).

In recent years endovascular abdominal aortic aneurysm repair (EVAR) has become the preferred intervention of choice over open repair (6). EVAR is less invasive, as it avoids the need for a laparotomy and is performed through bilateral small groin incisions or via a percutaneous approach (with a 'closure' device used to close the artery).

Advantages of EVAR over OSR include avoiding arterial cross clamping, reduced post-operative pain, shorter recovery, lower risk of post-operative pneumonia, reduced length of both ITU and overall hospital stay and reduced blood loss. Thirty day mortality is lower for EVAR compared to OSR however recent meta-analysis shows no significant difference in all-cause mortality at 4 years post op (6,7).

Standard EVAR technique (Infra-renal AAA)

EVAR is usually performed under general or epidural anaesthesia. Antibiotic prophylaxis is given. Patients are consented as to the risks of the procedure and the need for life long surveillance. Surgically, bilateral groin excisions are performed to expose and control the common femoral arteries.

Usually the right common femoral artery is punctured and an angiogram performed. The main body of the graft is then introduced and deployed with the proximal end of the graft as close distally to the origins of the renal arteries as possible to get a good seal and prevent migration of the graft (Image 2 & 3).

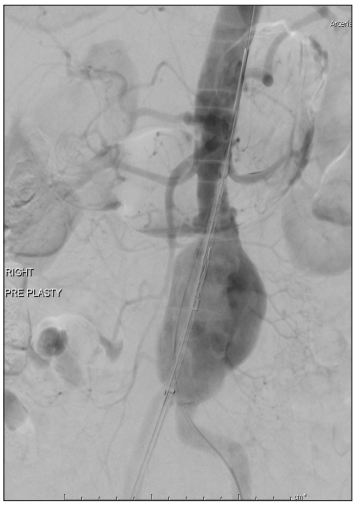

Image 2: Intra-operative angiogram showing adequate positioning of the stent graft, shown constrained within it's delivery system, just below the left renal artery prior to deployment.

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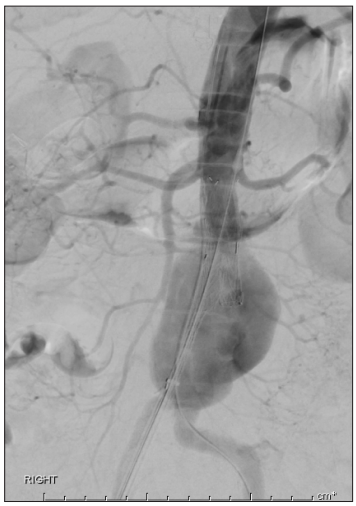

Image 3: Intra-operative angiogram showing good positioning of the top of the stent graft immediately below the renal arteries to give a long seal within the infra-renal aortic neck above the aneurysm. The contra-lateral limb is shown opened out ready for cannulation from the left side and the right stent graft limb remains constrained within the delivery system.

The top end of the graft has barbs to fix it in place to the inside of the aorta. The left common femoral artery is then punctured and the opening for the left limb of the stent graft (the 'contra-lateral' limb) is cannulated with a guide wire before introducing and deploying the left limb of the graft. This extends the stent graft into the common iliac artery aiming to land it close to, but not covering, the internal iliac artery (Image 4).

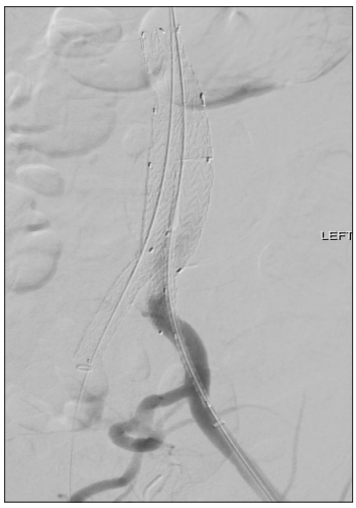

Image 4: Intra-operative angiogram showing the completed right stent graft limb and left limb being inserted up through the contra-lateral limb prior to deployment. The left limb was advanced further prior to deployment as in the position shown it would cover the internal iliac artery.

The right limb (in continuity with the main body) is then fully deployed. This limb seals to the inside of the right common iliac artery or may require an extension as performed on the left side. The graft and components are ballooned to ensure that they are fully expanded. A final angiogram is then performed to check that the stent graft has successfully excluded blood flow from the aneurysm sac (Image 5).

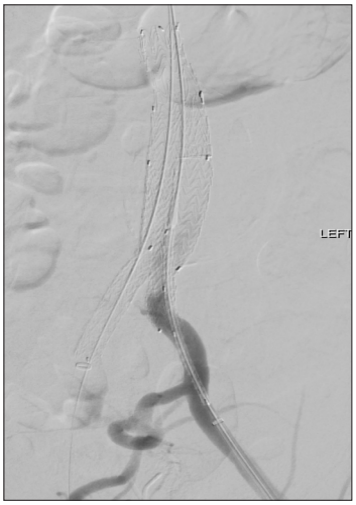

Image 5: Completion angiogram showing good flow through the graft with no visible filling of the aortic sac, a technically successful result.

Complications of EVAR

The risk of death within 30 days 2-3%, which is 2/3 lower than the risk of open repair in the randomised controlled trials (7). There is a 1-10% incidence of surgical wound morbidity post EVAR such as infection, haematoma and seroma (lymph collection). Injury to the common femoral artery can occur in up to 3% of patients with complications including thrombosis, dissection and pseudoaneurysm. As angiography is required to perform EVAR contrast induce nephropathy is a common complication particularly in difficult, lengthy procedures as higher doses of contrast are required (8).

There is a risk of both early and late ischaemic complications of EVAR. Early complications include limb ischaemia, bowel ischaemia, and renal artery occlusion. Limb ischaemia is more common when the stent graft has to be extended down into the external iliac artery. Latest generation grafts are more flexible and are less likely to kink, and as such have a lower rate of occlusion of less than 5%. Late limb occlusion may be caused by graft migration causing turbulent flow leading to graft thrombosis (8).

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Graft infection is a rare but serious complication as removal of infected grafts challenging, high-risk surgery. Graft infection may be caused by peri-procedural contamination so strict asepsis is always observed and prophylactic antibiotics are routinely given. More commonly graft infection is secondary to other intra-abdominal sepsis such as peritonitis or formation of an aortoenteric fistula (7).

By far the most common complication of EVAR is so called 'endoleak' which may involve up to one in five patients. Endoleak is defined as "persistent blood flow outside of the endograft but within an aneurysm... being treated by the device" (9). Endoleaks are classified into 5 types (Table 2) (10).

Endoleak type	Cause
I	Failure of graft to aortic seal. Ia – proximal; Ib – distal
II	Branch vessels (e.g. lumbar arteries, IMA). IIa – 1 vessel; IIb – 2 or more
III	Graft failure. IIIa – junction leak; IIIb graft rupture (minor <2mm; major >2mm)
IV	Graft porosity: This is observed with some stents < 30 days post placement
V	**'Endotension'** Aortic sac expansion with or without proven increased intra-sac pressure with no source of endoleak visualised on imaging (Duplex or CTA).

Table 2: Classification of endoleak after EVAR (10).

The presence of an endoleak can be detected using Duplex ultrasound or Contrast enhanced CT scan. In some cases the use of contrast enhanced Duplex or intra-arterial angiography are required to confirm the type of leak. The most common endoleaks are type II, which can be safely monitored provided the aortic sac is stable (no change in size).

Type II leaks may require treatment by coil embolization if persistent but usually spontaneously thrombose. All Type I and Type III endoleaks require reintervention which usually can be another endovascular intervention (aortic cuff, Limb extension or stent graft re-lining) but occasionally requires conversion to open. Endotension (Type V endoleak) may require conversion to open repair or re-doing EVAR (9,11).

Long term EVAR surveillance

Due to the risk of late endoleak or graft limb occlusion, patients post EVAR should undergo lifelong surveillance. There is no consensus on the optimal surveillance strategy. Many units have replaced regular CTA with Duplex ultrasound (Sac size and presence of endoleak) and Plain film examination (stent graft fracture or migration). Our current programme comprises a CT at 1 months and 12 months if higher risk post EVAR followed by annual Duplex ultrasound. Some authors recommend 5 yearly CT in addition (12).

MCQs

1. You are an F2 on a GP rotation. A 65 year old male patient with no prior medical history comes to see you after attending an AAA screening test where he was told he had a 4.7cm diameter abdominal aortic aneurysm. What medications would you consider prescribing?

a) Statin and a beta blocker

b) Statin and anti-platelet agent

c) Statin, rivaroxaban and ACE inhibitor

d) ACE inhibitor only

e) None of the above

2. You are an F2 on a GP rotation. A 69 year old gentleman who is a lifelong smoker comes to see you after his younger brother was diagnosed with a 6.8cm aneurysm. What would you do?

a) Offer smoking cessation advice and refer him to a vascular surgeon

b) Offer smoking cessation advice and suggest he refers himself to AAA screening

c) Reassure him and take no further action

d) Offer smoking cessation advice only

e) Offer smoking cessation advice and organise an urgent CT aorta

3. You are an F2 on a GP rotation. You had arranged a renal tract ultrasound for a 67 year old lady with microscopic haematuria. The report showed normal renal tract bilaterally but an incidental finding of a 5.7cm AAA was noted. What would you do?

a) Refer the patient to AAA screening

b) Organise a CT aortogram

c) Routinely refer to a Vascular Surgeon

d) Urgently refer to a Vascular Surgeon

e) Both b) and d)

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4. You are an F2 covering the surgical wards on a weekend. You are asked to see a patient who is day one post EVAR to see if he is suitable for discharge. Which blood test must you perform prior to discharge?

a) U&Es

b) FBC

c) LFTs

d) CRP

e) Clotting screen

5. A 72 year old man 3 years post EVAR presents to ED in with profuse upper GI bleeding. Which of the following 5 options is the most likely diagnosis?

a) Mallory Weiss tear

b) Small bowel angiodysplasia

c) Aorto-enteric fistula

d) Type III endoleak

e) Perforated duodenal ulcer

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Answers

1. Answer: b)

Whilst no level I evidence (randomised controlled trials) exists for benefit of statins and anti-platelet agents in AAA patients they are generally advised as they have other cardioprotective benefits and a low side effect profile. Studies have shown the high rate of cardio-vascular events in AAA patients, even after treatment, and are protective against peri-operative events.

Beta blockers have been shown to have no benefit. Patients with AAA do not need anticoagulation even if they have mural thrombus on a CT scan (if this causes embolization they require high dose statins and anti-platelet agent or repair of their aneurysm). An ACE inhibitor should be considered in patient who is hypertensive but so can other classes of anti-hypertensive medication.

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2. Answer: b)

He is at increased risk of having an AAA (smoking history and first degree relative with AAA) and warrants screening. It would be most appropriate for him to refer himself to the NHS AAA Screening Programme and be advised to stop smoking.

3. Answer: (d) or (e)

The rupture risk for a 5.7 cm AAA is around 3.5-5% per annum and urgent referral for consideration of surgical repair is advised. The vascular surgeon will want a CT scan to assesses the aneurysm anatomy and suitability for endovascular repair (EVAR). The CT is therefore helpful but not essential, and should not delay referral, as we know that overall around one third to one half of patient referred with AAA do not actually undergo AAA repair.

5. Answer: a)

Acute contrast induced nephropathy is a recognised complication of EVAR. Occlusion of one or both renal arteries is a rare but serious early post op complication of EVAR. Patients with a mild drop in renal function will need a repeat with their GP in the next few days but those with more significant renal impairment may warrant further investigation.

6. Answer: c)

A secondary aorto-enteric fistula caused by erosion between the stent graft within the aorta and an overlying bowel loop with secondary infection. This is a rare but serious late complication of EVAR. He should be appropriately resuscitated and urgent CT aortogram organised with immediate involvement of a vascular surgeon.

A Mallory Weiss tear follows vomiting, Small bowel angioplasia is rare, and a Type III endoleak does not cause external bleeding. A bleeding duodenal ulcer is a common cause of GI bleeding and would need excluding, a perforated ulcer however presents with peritonism not blood loss.

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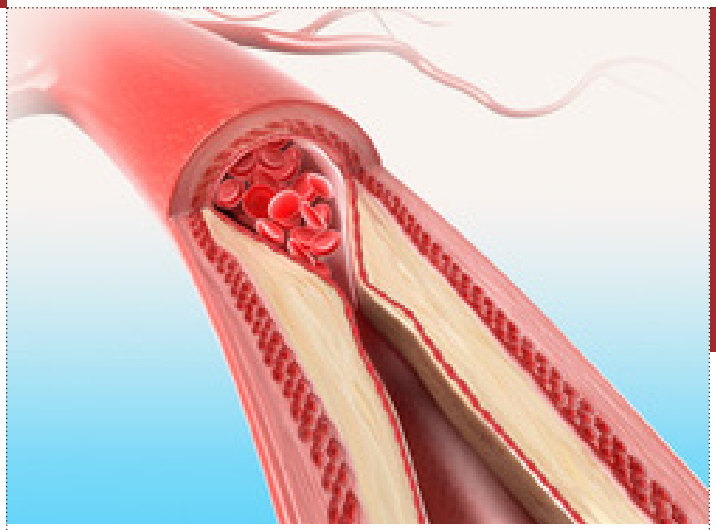
ACUTE LIMB ISCHAEMIA

T Barakat, Y Ahmed, G Stansby



Abstract

Acute limb ischaemia (ALI) is one of the major vascular emergencies which if missed and not acted on promptly could lead to catastrophic consequences in the form of permanent disability, amputation and even death.

It can affect both lower and upper limbs; however it more commonly affects the legs. The incidence of ALI is approximately 1 per 6000 population per year with the two main causes being embolization and thrombosis *in situ*.

In this article we look to focus on the key elements surrounding acute limb ischaemia that are vital for a foundation doctor to be aware of as to prevent delay in diagnosis and potentially limb-saving/life-saving interventions.

It is vitally important that if an ALI is suspected, then an immediate vascular team opinion should be sought!

We will discuss the classical teaching presentation of these patients ('The 6 P's') and look at how this may contrast slightly to actual presentation. Systemic examination with findings, further investigations and management will be detailed in the article.

Acute Limb Ischaemia
Patient Management

Causes

1. Cardiac embolic source

Patients with atrial fibrillation or underlying endocarditis can develop vegetations on the heart valve lesions and these can embolise. These emboli tend to lodge where the artery bifurcates. When an embolus lodges in the aortic bifurcation it is called a Saddle embolus.

2. Thrombosis in situ

If the patient already has a stenosis and due to any reason there was sluggish flow over the stenosis (e.g. dehydration), this can lead to thrombosis of the artery.

3. Dissection: arterial dissection can cause ischaemia and organ malperfusion

This is commonly seen in cases like extensive type B thoracic aortic dissection where there is an intimal tear in the arterial wall and the intima gets separated and causes arterial occlusion hence distal ischaemia.

4. Aneurysm thrombosis or embolization

These can be anywhere in the arterial tree (mainly aortic or popliteal aneurysms).

5. Trauma to the artery

EG. supra-condylar fractures in children or any fractures which could cause contusion or even transection of the artery. In such cases these patients tend to be young and fit and they do not tolerate ischaemia very well as there has no time to develop collaterals.

6. Other causes for example hypercoagulable states, graft occlusion and iatrogenic puncture.

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Presentation

The classical teaching is that patients present with the 6P's which are pain, pale, pulseless, paraesthesia, perishing cold, paralysis (figure 1). Of course these are a mixture of symptoms and signs. Pain is a very important symptom. It is highly unlikely that a patient with no pain will have an ALI.

So although we are taught that ALI presents with the 6 P's, in practice this is very rarely the case. Patients will present with acute onset pain with inability to stand or walk on the leg or they may present with tingling and numbness of the foot (nerve ischaemia) and paralysis or weakness (nerve and muscle ischaemia) or combination of those.

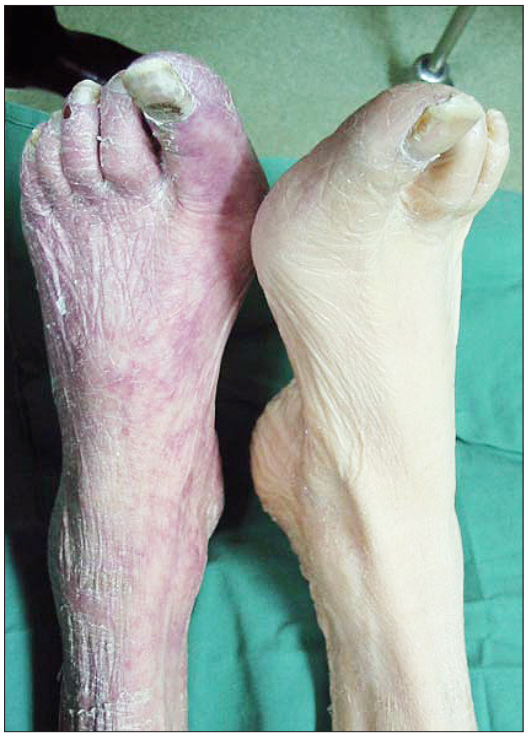

Figure 1

History

A full clinical history is key. It is important from the history to know the duration of symptoms and the possible cause. This will give you an estimate of the time which you have to restore the circulation to the limb. After six hours of ischaemia the tissues can develop irreversible damage. The patient should be assessed for any significant comorbidities and if they are on anticoagulants.

On examination

As with any other system assessment, the patient should be examined systematically.

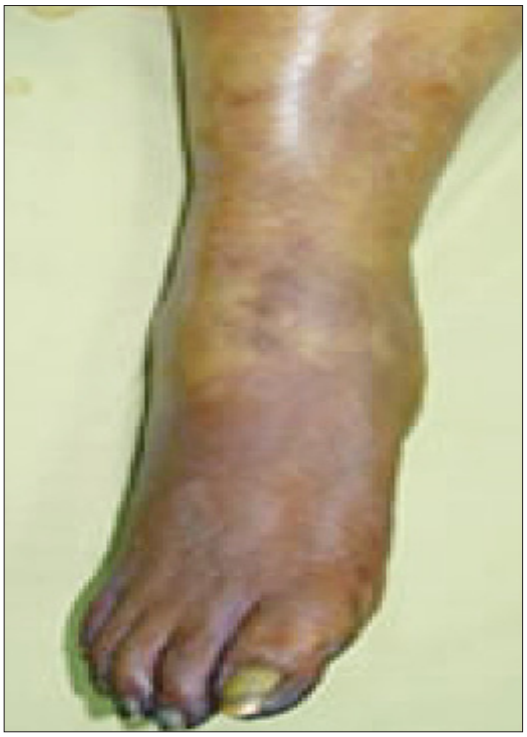

Figure 2: Fixed mottling & cyanosis.

Check the radial pulse and its character and document BP. Check the heart for any murmurs. Examine the abdomen to rule out an abdominal aortic aneurysm. Remember expansile vs pulsatile mass!

Examination of the lower limb should be methodical.

- *Firstly, inspection to look for pallor and/or scars which could be from previous bypass surgery (is the bypass still functioning or has it blocked?) Secondly, you should assess for deformity. Can the patient move the toes, foot and the leg overall?*

- *Palpation is next for assessment of temperature and capillary refill time and to assess the pulses, starting from the femoral pulse down to popliteal, dorsalis pedis and posterior tibial pulses. Sensation status showed also be checked and documented which will help in serial examinations to monitor if his/her neurological status is intact or deteriorating.*

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It is good to check for calf tenderness as it may indicate that the ischaemia is very severe and that the muscles are starting to become non-viable. A patient with severe calf tenderness will probably need a fasciotomy when they are re-vascularized.

Always check and examine the other leg if it is there!!Make comparison with the symptomatic leg.

- *Check ankle brachial pressure index (ABPI).*

The two most important of the 6Ps which vascular surgeons take very seriously are paraesthesia and loss of motor power. These indicate that the limb is either already non-viable or very close to becoming so! A further bad sign is fixed mottling which usually indicates irreversible changes.

Investigations

FBC as it is important to know if the patient is anaemic or not and the platelet count. Renal function is important as patient may need a CT scan with IV contrast and the contrast can worsen renal function further and lead to complete renal failure.

Also, if the patient has started to develop muscle necrosis then potassium levels can become elevated as damaged cells release potassium and myoglobin release can also damage the kidneys. An ECG should be performed to check cardiac status and evidence of AF.

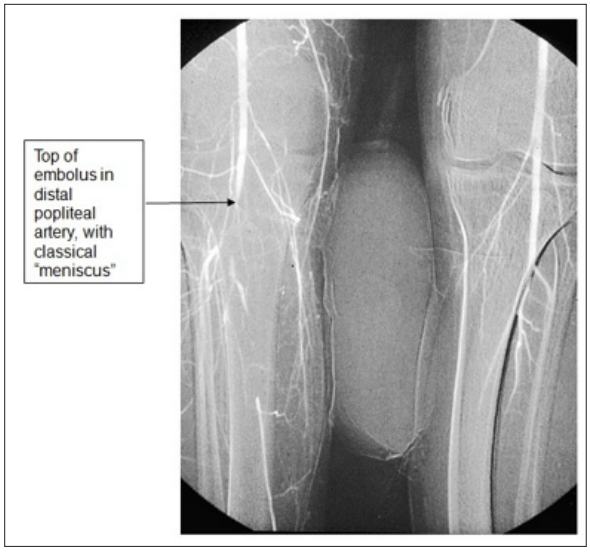

Figure 3

Duplex USS is useful to visualise the infra-inguinal vessels (i.e. If on examination the femoral pulse is palpable!) as it is non-invasive and it does not involve any contrast. Magnetic resonance angiography or CT angiogram is a very good way of imaging the arterial tree as it can be used to visualise the whole of the aorta and give more information than the duplex especially supra-inguinal vasculature.

Catheter angiography can also be performed but usually nowadays only if an intervention is going to be performed.

Management

1. Analgesia!!! ALI is usually extremely painful! Make sure the patient has adequate analgesia on board and pain is well controlled.

2. Oxygen.

3. Rehydration.

4. Call the Vascular Registrar and seek urgent input! Don't let it wait!!

5. Start intravenous heparin infusion to prevent propagation of the thrombus.

It also reduces the incidence of cardiovascular events and improves prognosis.

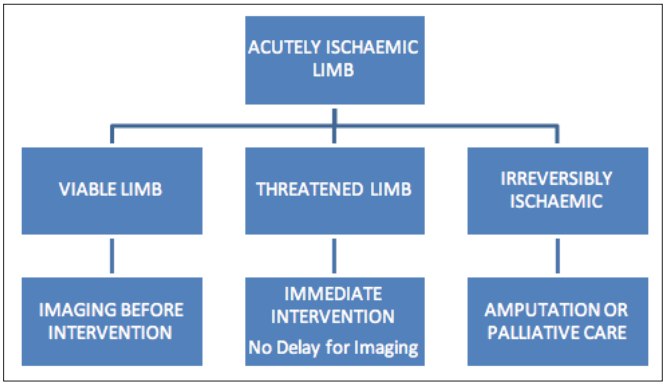

Figure 4

The various available treatment options are:

1. Surgical revascularisation

2. Surgery in the form of amputation

3. Endovascular including thrombolysis

4. Palliative care

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It is important to establish if the limb is viable or not. Changes like fixed mottling and very high CK which suggest that is a non-viable limb.

If the limb is non-viable then primary amputation or palliation become the only management options and it is decided according to comorbidities and the fitness of the patient and not to forget patient wishes.

If the limb is salvageable then it is important to establish the level of the blockage so that treatment can be planned. Occlusion of the popliteal artery tends to have a profound effect as it is the only artery crossing the knee joint. Occlusion of one of the three run off vessels (anterior tibial, peroneal and posterior tibial) tends not to have a real effect as the others can efficiently supply the leg.

If the limb is viable and the ischaemia is acute,catheter thrombolysis is an option if there are no contraindications. The drawback is that thrombolysis takes time and if the limb is extremely ischaemic it might not survive that long. Embolectomy is usually the procedure which performed.

If intra-operatively, flow is not established then another option may be to perform a bypass and here, preoperative detailed imaging (CT scan or MRI) comes into play as it gives detailed information about the state of the inflow and outflow arterial circulation.

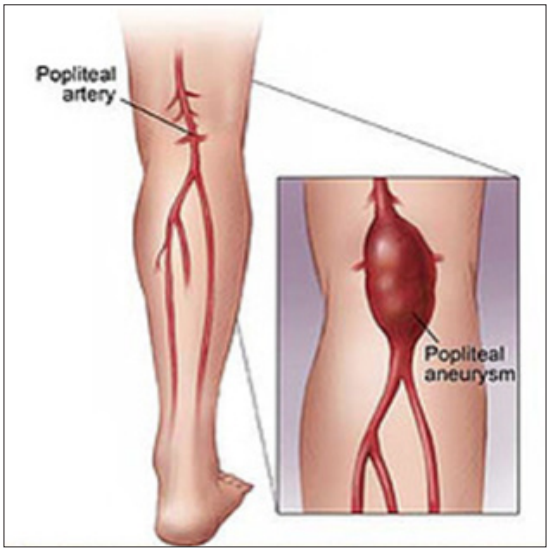
On revascularization the calf muscles tend to swell up and the patients may develop compartment syndrome so the classical teaching is that if you think the patient may need a fasciotomy then it is better to do it at the same time of the revascularization and it will save the patient another trip to theatre.

Postoperatively the patients should be anticoagulated with low molecular weight heparin and then commenced on warfarin for at least 6 months depending on the cause.

Popliteal Aneurysms

Popliteal aneurysms are usually asymptomatic but they can present with embolization distally or even thrombosis which can lead to profound ischaemia of the leg. Very rarely popliteal aneurysm can present with rupture. The risk of limb loss after thrombosis of a popliteal aneurysm is about 50%. They can be mistaken for a Baker's cyst or popliteal cyst.

About 50% are bilateral and about 30% are associated with an abdominal aortic aneurysm.



Elective repair is indicated if the aneurysm is about 2cm or more. Other indications for repair if it is symptomatic i.e. (distal embolization, thrombosis and pain). The gold standard is surgical repair by doing a bypass and ligating the aneurysm proximally and distally.

Some surgeons prefer to put an interposition graft through a posterior surgical approach. Endovascular repair in the form of stenting is still on its infancy but is recognised as an option for treatment. It is probably advocated for people who are not fit with good run-off vessels.

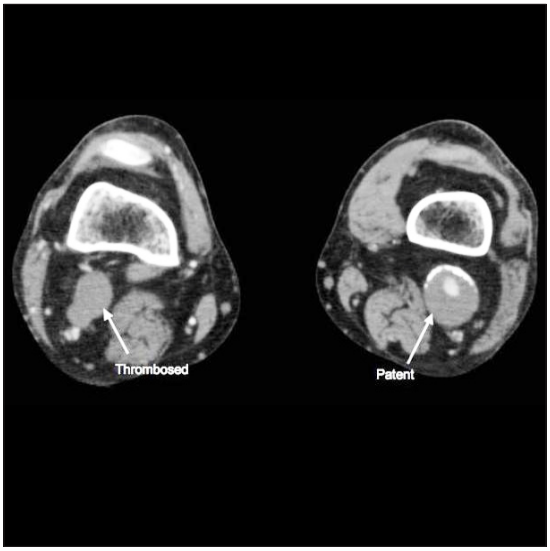


Figure 5: Showing bilateral popliteal aneurysms with thrombosis of the right side.

ACUTE LIMB ISCHAEMIA

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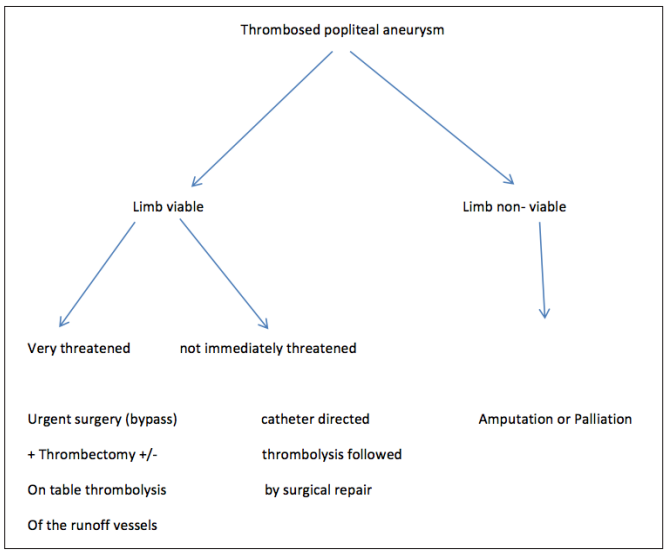
If patient presents as an emergency with a thrombosed popliteal aneurysm, they can have profound ischaemia and urgent intervention may be required. The diagram below summarises the approach in such cases. All cases of acute limb ischaemia should be referred urgently to a vascular centre with the appropriate set up to deal with such challenging cases.



MCQ's

1. Acute lower limb ischaemia:

a. Should be treated by embolectomy.
b. Is characterised by the presence of micro-infarcts on the foot.
c. Is initially managed with heparin intravenously and observation
d. Requires immediate referral to a vascular surgeon.
e. Is defined as an Ankle Brachial Pressure Index of less than 0.5.

2. A 98 years old lady with dementia and significant comorbidities presented with acute limb ischaemia present for 48 hours with fixed changes. On further questioning she is bed ridden with a very poor quality of life. The most appropriate management plan is:

a. Bypass.
b. Amputation.
c. Palliation
d. Thrombolysis.
e. Embolectomy.

3. A patient had a liver resection for an inflammatory mass 5 days ago. The foundation doctor was called urgently as the patient developed severe leg pain with difficulty walking over the last 3 hours. On examination, the patient could hardly move his toes and foot. The F1 correctly diagnosed acute limb ischaemia and organised a CT angiogram which showed an embolus in the superficial artery + generalized atherosclerosis. The best treatment option is:

a. Thrombolysis.
b. Thrombolysis + angioplasty.
c. Urgent Embolectomy.
d. Bypass.
e. Conservative with heparin infusion.

4. A 56 year old male who had a recent stroke presented to the accident and emergency with a thrombosed popliteal aneurysm. His limb was viable and he had no rest pain but short distance claudication. Best treatment option for him is:

a. Thrombolysis.
b. Endovascular repair (stenting).
c. Thrombectomy.
d. Elective bypass.
e. None of the above.

5. Thrombolysis is contraindicated in:

a. Acute limb ischaemia.
b. Patients who had recent surgery.
c. Patients with haemophilia.
d. Recent stroke.
e. Acute ilio-femoral DVT.
f. B, C, D

Answers

1. [Best Answer = d]. Explanation:

Acute lower limb ischaemia is mostly due to embolism (often from the heart) or thrombosis at the site of pre-existing atheroma. For embolus surgery is usually embolectomy using a Fogarty catheter but for thrombosis an emergency bypass may be needed. It causes impairment of muscle and nerve function (remember the 6 "P's" – Pain, pallor, pulseless, paralysis, paraesthesia and perishing cold!).

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Infarcts are a late sign. All cases should be given heparin initially to prevent further extension of the thrombosis but observation is not correct. They should also be referred urgently for vascular surgery as the limb may be lost in 3-6 hours without intervention. It is not defined by the ABI, which will usually be unrecordable in acute ischaemia

2. [Best Answer = C]. Explanation:

This lady is not a good candidate for revascularization. She has a very poor quality of life and this is a terminal event.

3. [Best Answer = C]. Explanation:

This is a threatened limb and needs urgent intervention. The gold standard operation in such situations is a femoral embolectomy. Thrombolysis is not indicated here as it for cases in which the limb is not on imminent danger. Another reason which precludes thrombolysis is that the patient had recent surgery. Bypass is only performed if no good flow could be established distally due to an occluding thrombus which could not be removed.

4. [Best Answer = D]. Explanation:

This is not a critically ischaemic leg at the moment so no need for an urgent treatment. The treatment should be planned and provided in an elective setting. This patient should have detailed imaging in the form of a CT or MR angiogram to assess his proximal arteries and distal runoff. His long saphenous vein should be scanned and assessed as a conduit for a bypass. Patient cannot have thrombolysis as he had a recent stroke which is a contraindication.

5. [Best Answer = F]. Explanation:

Thrombolysis is contraindicated in:

- *Active bleeding*
- *Cerebrovascular accident within 2 months*
- *Recent GI bleeding*
- *Pregnancy*
- *Neurosurgical procedure within 2 months*
- *Vascular surgical procedure within 2 weeks*
- *Abdominal surgery within 2 weeks*
- *Bleeding disorder*
- *Extensive trauma*

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MANAGEMENT OF A LARGE HEPATIC ANEURYSM PRESENTING AS LOWER ABDOMINAL & BACK PAIN: A CASE REPORT

MS Goonoo, RR Goel, N Husnoo, B Rawshdeh, RA Salaman

Management Of A Large Hepatic Aneurysm Presenting As Lower Abdominal & Back Pain: A Case Report
Patient Management

Abstract

Hepatic aneurysms are rare and constitute 20% of all splanchnic aneurysms. Here we report the case of a patient referred to our centre after being investigated for chronic lower abdominal and back pain. Computed tomography revealed a 47mm common hepatic artery aneurysm.

The treatment modality was an open aneurysmectomy with vascular reconstruction using venous conduit. At surgery the aneurysm was incised and end to end anastomosis with vein graft was performed. Diagnosis of HAAs is of clinical importance because of the high mortality rate associated with rupture. All hepatic aneurysms ≥2cm require prompt referral to a surgical team for definitive management.

Case presentation

A 74- year- old Caucasian man with chronic lower abdominal and back pain of three months' duration, put down to osteoarthritis of the spine, had a CT scan of the abdomen and pelvis to exclude intra-abdominal causes. This led to the finding of an aneurysm of the common hepatic artery measuring 40 x 47 x 41mm, partly calcified and partially thrombosed (figure 1).

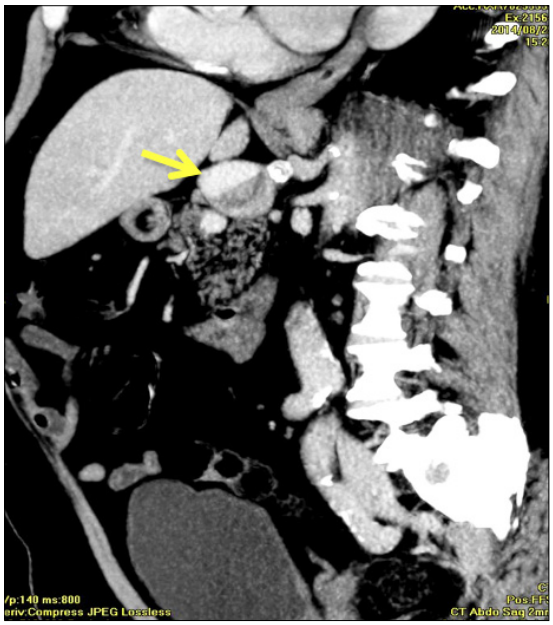

Figure 1

He was then referred to the hepato-biliary and vascular teams at our centre for management. Relevant past medical history included high BMI, hypertension, ischaemic heart disease with previous CABG and diverticulitis. His case was discussed at the multidisciplinary meeting and he was deemed unsuitable for endovascular intervention. There was no major clinical finding on examination and all blood tests were within normal range. Therefore, an elective open repair of aneurysm was done with vascular reconstruction using vein interposition graft harvested from a long saphenous vein.

A midline laparotomy was performed, the sub hepatic area exposed and aneurysmal sac revealed (figure 2).

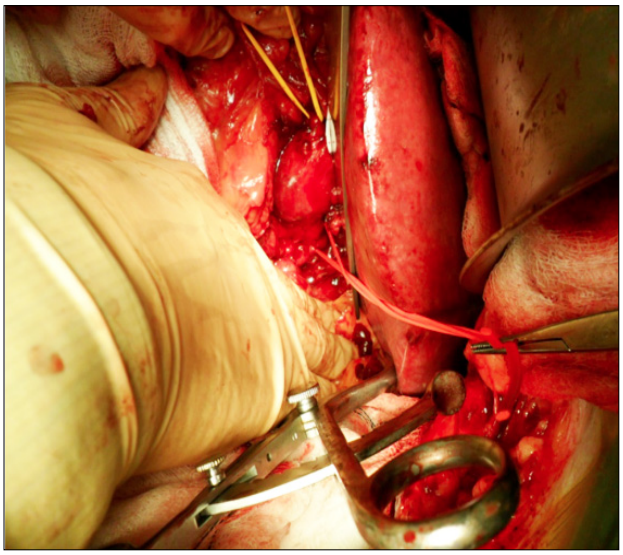

Figure 2

The sac was incised, clots scooped out and the vein graft harvested from the long saphenous vein was anastomosed distal to the origin of the gastroduodenal artery in reverse fashion within the sac. The two flaps of the sac were approximated with 2-0 polysorb.

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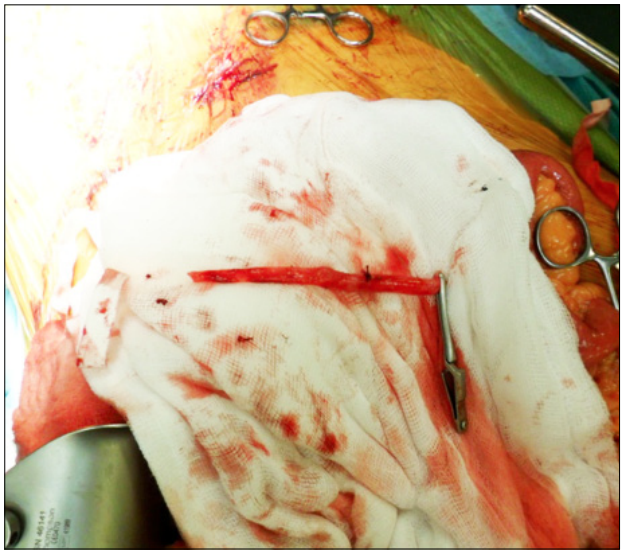

Figure 3

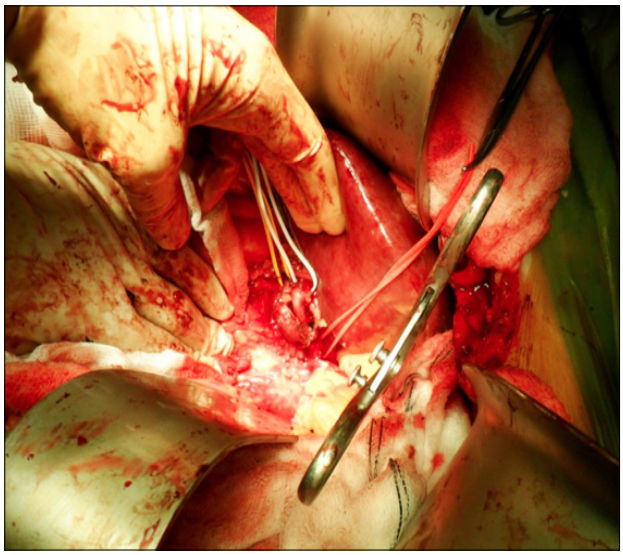

Figure 4

Post operatively the patient was admitted to the critical care unit. His recovery was slightly complicated by an initial drop in haemoglobin and platelets, and subsequently a haematoma at the site of long saphenous vein harvest. He was safely discharged 6 weeks later with no evidence of deranged liver function and now awaiting for a CT abdomen and pelvis in three months' time.

Discussion

Hepatic artery aneurysms are rare and constitute about 20% of all visceral aneurysms (1, 2, 3, 4). It was first described by Wilson in 1809 and is the second most frequent aneurysm after splenic aneurysms. Their incidence varies between 0.002% and 0.4% (1). 75-80% of the diagnosed HAA are extra-hepatic, mainly solitary in nature, (5) predominantly seen in men at mean age of 60 (2). Mortality rate, associated with aneurysm rupture, ranges from 21 to 35% (2, 6).

Hepatic aneurysms are difficult to diagnose as they are mostly asymptomatic. Their presentation varies and can present as epigastric pain, right upper quadrant pain with radiation to the back or GI bleeding. In our case the gentleman presented with chronic lower abdominal with back pain for which the diagnosis was initially put down to osteoarthritis until CT imaging was carried out. Less than a third of patients present with Quinke's triad: epigastric pain, haemobilia and jaundice (1, 2, 3).

The main aetiological factors in the development of HAA are atherosclerosis (32%), medial degeneration (24%), trauma (22%), infections(10%) and more rarely due to connective tissue disorder such as fibromuscular dysplasia or polyarteritis nodosa (1, 2). There has been a significant rise in number of iatrogenic cases following biliary procedures such as cholecystectomy (1, 2).

CT angiography has become the most common investigation but US Doppler and angiography can still provide additional details. The first radiological imaging of HAA was produced by Kirkin in 1955 via angiography (1, 2, 5, 6). This provides a definite diagnosis with details on size, shape and location to aid intervention. MRI can be of value in assessing the flow performance after any endovascular or surgical repair (2).

HAA has remarkable clinical value because most of the cases are asymptomatic with high mortality after rupture. Lack of studies on the safe and definitive treatment often means that management decision is based on local or individual preference (7). Indications for treatment of common hepatic aneurysms include: symptomatic aneurysms, non-atherosclerotic aneurysms, multiple hepatic aneurysms and aneurysms ≥ 2 cm in diameter (8). However, it is noteworthy that there is no correlation between diameter and rupture (8).

Aside from local preference, treatment strategies are also influenced by the patient's general health, morphological characteristic of the lesion (saccular or fusiform with or without ample neck) and anatomical location of the lesion respective to the gastroduodenal artery (1,2,5,6). The first aneurysm resection and reconstruction was done by Taylor in 1943 (2). With new breakthroughs in interventional radiology, less accessible areas or high risk cases can be managed using endovascular therapy.

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MS Goonoo, RR Goel, N Husnoo, B Rawshdeh, RA Salamana

Percutaneous embolization is the most commonly used technique for treating mainly intrahepatic aneurysms using spirals, microspheres or acrylic glue (2). Sugihara et al reported a case treated by embolization of the PHA without any hepatic dysfunction, bearing in mind the 22 possible routes of hepatic collateral pathway formations (9). Endovascular therapy is now the emerging treatment modality to manage visceral aneurysms (4). It is used in clinical emergencies, electives case with considerable comorbidities, in anatomically difficult cases and where it is imperative to maintain blood flow through the affected artery (10, 11).

Different types of stents have been used such as covered stent grafts, PTFE coronary stent graft, Jostent coronary stent graft and the novel Cardiatis Multilayer Flow Modulator (CMFM). Downer et al reported the case of a CHA aneurysm, managed with a covered stent graft, a fracture in which led to a recurrence 12 months later. This was treated with embolization after confirmation of hepatic collaterals (11). The PTFE stent graft, the use of which has been reported in a case report by Hiroyuki et al, is suitable for curvilinear and small vessels due to its flexibility (7).

Jostent coronary stent also showed efficacy to preserve an orthotopic liver transplant graft in a patient who developed a hepatic aneurysm post operatively, as coil embolization in this situation should be avoided (12). However C.Shick et al elaborated on the use of remodelling technique to perform coil embolization of a liver transplant graft to avoid displacement of the coil into the aorta or occlusion of the gastroduodenal artery (13). CMFM is a self-expandable stent with two interconnected layers without covering, which allows exclusion of an aneurysm from the circulation, thus reducing flow speed inside the sac promoting shrinkage while preserving collaterals.

It can be used in the presence of a short landing zone and frequent bifurcations. Balderi et al reported two cases treated with CMFM where embolization was difficult in distal branches with few collaterals and covered stent rigidity in small distal tortuous vessels would increase risks of liver ischaemia. (14, 15) Surgical treatment consists of ligation or revascularization of the hepatic artery. The first ligation was done by Kehr in 1903 (1). The CHA can be safely ligated provided there is patent gastroduodenal or pancreatico-duodenal artery to provide collaterals.

Aneurysms distal to the CHA should be treated by revascularization using prosthetic graft or venous conduit. Any large fusiform shaped lesion with short ample neck at the origin of the gastroduodenal artery could be surgically managed by aneurysmectomy and revascularization (2).

Jaunoo et al in his report purported that there is no significant difference in cardiac risk in patients undergoing vascular surgery within 6 months of an MI (3). Surgical robotics is seen as the future of complex surgical procedures and in his report Salloum et al who treated a 43mm HAA, and simultaneously performed a cholecystectomy, by robotic resection discussed the reduction in tremor effect from surgeons and increased dexterity of movement of instruments (16).

Follow-up post-intervention is usually done by US Doppler flow studies and CT angiogram. The use of alpha-glutathione-S-transferase (alpha- GST) in addition to routine liver function test to monitor sudden hepatic dysfunction in the long term has also been reported (3).

Our patient was treated by aneurysmectomy and revascularisation due to its close proximity to the celiac axis, the size and fusiform shape of the aneurysm. He was followed up by a CT angiogram at 3 months; he remained well and will be followed up by a repeat CTA at 6 months.

Conclusion

HAAs are rare but may present incidentally or a cause of chronic abdominal pain. Diagnosis is mostly by imaging techniques such as CT angiography and US Doppler. We recommend quick referral to a surgical team for definitive management.

Abbreviations

CTA: Computed tomography angiography; HAA: Hepatic artery aneurysms; CHA: Common hepatic artery aneurysms; PHA: Proper hepatic artery;

CMFM: Cardiatis Multiplayer Flow Modulator; PTFE: polytetrafluoroethylene; alpha-GST: alpha-glutathione-S-transferase; MI: myocardial infarction; US:

Ultrasonography.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interest

The authors declare that they have no competing interests.

Authors' contributions

Conception and design: MSG, RRG, NH, RAS. Data collection: MSG, RRG, BR, RAS. Writing the article: MSG, NH. Critical revision of the article: RRG, BR, RAS. Final approval of the article: MSG, RRG, NH, BR, RAS. Overall responsibility: RAS

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MANAGEMENT OF A LARGE HEPATIC ANEURYSM PRESENTING AS LOWER ABDOMINAL & BACK PAIN: A CASE REPORT

MS Goonoo, RR Goel, N Husnoo, B Rawshdeh, RA Salamana

Self-Assessment Questions

(Only consider single best answer)

1. What do you understand by Quinke's Triad?

a) epigastric pain, haemobilia and jaundice
b) fever, jaundice and right upper quadrant pain
c) haemobilia, jaundice and vomiting
d) jaundice, pedal oedema and right upper quadrant pain

2. Which is the most important aetiological factor for aneurysms?

a) Infections
b) Vasculitis
c) Atherosclerosis
d) Connective tissue disorders

3. Which is the most common investigation used for diagnosing hepatic aneurysms?

a) US Doppler
b) CT Angiogram
c) Angiography
d) MR Angiogram

4. Which is the commonest modality for treating intrahepatic aneurysms?

a) Percutaneous embolization
b) Open laparotomy
c) Stenting
d) Liver transplant

5. Treatment strategies for hepatic aneurysms depend on which of the followings:

a) Patient's general health
b) Morphological characteristic of the lesion
c) Anatomical location
d) All of the above
e) None of the above

Answers

1. a

About less than a third of patients present with Quinke's triad: epigastric pain, haemobilia and jaundice, however most hepatic aneurysms are difficult to diagnose as they are mostly asymptomatic.

2. c

The main aetiological factors in the development of HAA are atherosclerosis (32%), medial degeneration (24%), trauma (22%), and infections (10%) and more rarely due to connective tissue disorder such as fibromuscular dysplasia or polyarteritis nodosa. There has been a significant rise in number of iatrogenic cases following biliary procedures such as cholecystectomy

3. b

CT angiography has become the most common investigation but US Doppler and angiography can still provide additional details. CT angiogram is rapid and gives information to the surrounding tissues as well. Angiography alone provides details on size, shape and location to aid intervention

4. a

Percutaneous embolization is the most commonly used technique for treating mainly intrahepatic aneurysms using spirals, microspheres or acrylic glue. However endovascular therapy is now the emerging treatment modality to manage visceral aneurysms mainly in anatomically difficult cases.

5. d

All aneurysms ≥ 2 cm in diameter must be treated. Aside from local preference, treatment strategies are influenced by the patient's general health, morphological characteristic of the lesion (saccular or fusiform with or without ample neck) and anatomical location of the lesion respective to the gastroduodenal artery.

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MANAGEMENT OF MYCOTIC ANEURYSMS - A CLINICAL CASE REPORT

J Follows, K Daniel, K Murphy, S Nawaz

Management Of Mycotic Aneurysms - A Clinical Case Report
Patient Management

Abstract

An aneurysm is an abnormal, permanent, localised dilatation of a blood vessel compared to its expected diameter (1). Mycotic aneurysms are a consequence of degradation of the layers of the arterial wall with resultant aneurysmal defect.

Mycotic aneurysms have a significant associated morbidity and mortality. Complications can include peri-vascular infection, infection resistant to antimicrobial medical therapy, and vessel rupture leading to major haemorrhage, which can threaten life and limb (2,3).

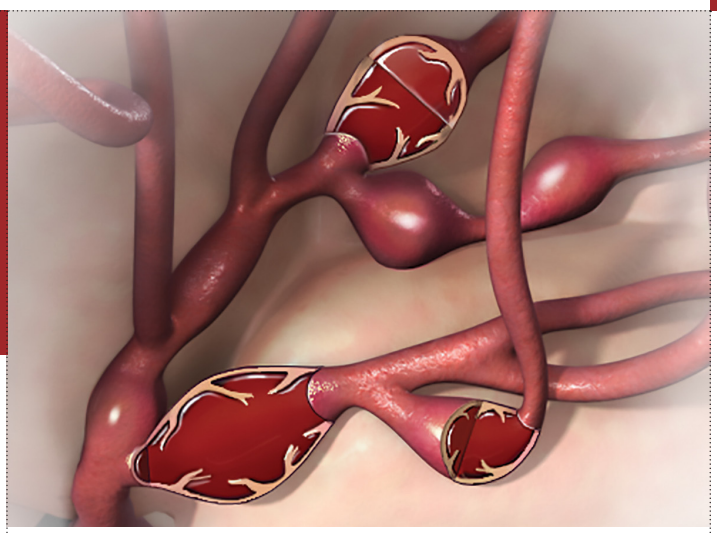
The aim of this article is to learn about the difficulties of investigating and diagnosing a mycotic aneurysm, and the practicalities of their surgical management. Additionally, this article seeks to emphasise the potential complexity of patients with mycotic aneurysms, and gives examples of optimisation prior to surgical intervention.

Case history

A 45 year old previously well man presented to his General Practitioner with dysphagia, weight loss, malaise, vomiting, fever and night sweats. He was found to have a murmur on auscultation which was new. He was admitted for investigation including routine blood tests and blood cultures. The patient self-discharged before any further investigations or treatment could take place.

The patient re-attended the Emergency Department fourteen days later with a sudden onset of swelling of his right calf. He had no previous history of intermittent claudication or ischaemic rest pain. He denied any precipitating trauma and had no risk factors for deep vein thrombosis (DVT). He denied recreational or intra-venous drug use or recent travel abroad.

On clinical examination he was found to be haemodynamically stable, there was no palpable abdominal aortic aneurysm. His right calf was found to be swollen and hot compared to the left. There were no features of acute limb ischaemia. There was evidence of good perfusion of both legs with preserved foot pulses. No neurological symptoms were reported.



Bloods

PT 12.5s, APTT 25.7s, Fibrinogen 7.8/L, Hb 93g/L, MCV, 85.4fl, White Cell Count (WCC) 25.7x109/L, Neutrophils 20.8x109/L, Platelets 537 x 109/L, Normal urea and electrolytes, Total protein 72g/L, Albumin 31g/L, Globulin 41g/L, Total Bilirubin 8umol/L, Alkaline Phosphatase 234IU/L. ALT 36 IU/L, C reactive protein (CRP) 171.6mg/L.

Discussion

Presentation of patients with mycotic aneurysms.

William Osler first described aneurysms as mycotic in 1885, due to their 'mushroom shape appearance'. The most common aetiology of these aneurysms are bacterial, rather than fungal as suggested by Oslers' description. Mycotic or infected aneurysms make up approximately 3% of all aneurysms (4,5).

Patients with mycotic aneurysms often present with non-specific symptoms, including lethargy and malaise, and often without focal examination findings (3). One study (4) found that 79% of patients have elevated WCC or CRP but less than 50% displayed a fever. 24% of the patients in the study had positive blood cultures prior to surgery. 76% complained of pain which was local to their aneurysm.

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Case history continued

The patient underwent a venous duplex scan. This scan was negative for DVT, but demonstrated a 1.8cm aneurysm arising from the origin of anterior tibial (AT) artery. The patient was clinically septic, supported by biochemical and haematological evidence of infection, so further imaging was undertaken to delineate the false aneurysm by magnetic resonance angiogram which demonstrated a 1.8cm aneurysm arising from the AT origin with a short occlusion (see figures 1 and 2).

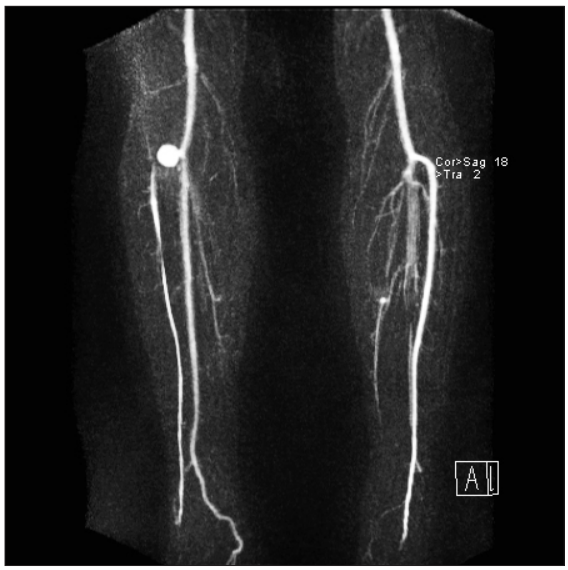

Figure 1

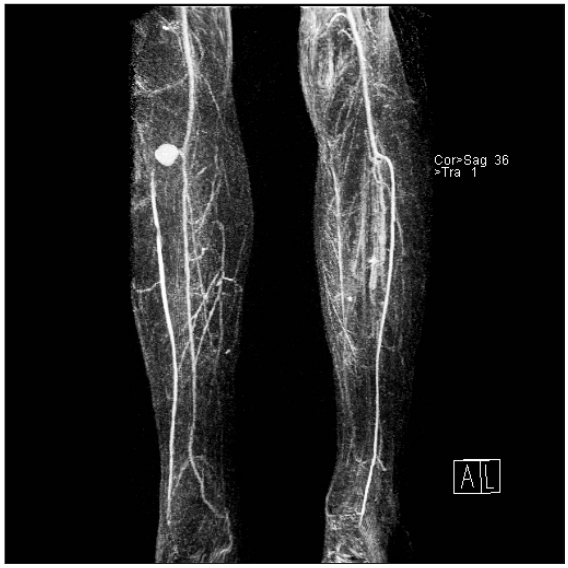

Figure 2

A trans-thoracic echocardiogram (TTE) confirmed mitral valve endocarditis with associated moderate mitral regurgitation. A vasculitic screen including cytoplasmic and perinuclear ANCA, IgA, IgG and IgM, anti nuclear antibodies, complement C3 and C4 and rheumatoid factor were all within the normal range. A bloodborne virus screen was negative.

Seven separate sets of blood cultures were taken over several days before starting antibiotics. All pre-antibiotic blood cultures (anaerobic and aerobic) isolated *Aggrega actinomycetemcomitans;* a gram negative rod which is part of the HACEK group. After the unifying diagnosis of infective endocarditis complicated by septic emboli and a mycotic aneurysm was made, the patient was started on intravenous ceftriaxone.

Discussion: aetiology and epidemiology

The most common bacterial pathogens associated with mycotic aneurysms are *Staphylococcus aureus* and *Salmonella* species although there are recorded instances of more unusual bacterium such as *Treponema pallidum, Mycobacterium tuberculosis* and *Listeria monocytogenes* (2).

The most common sites for finding mycotic aneurysms are the abdominal aorta and the common femoral arteries; the latter being particularly associated with intra-venous drug use (1,6). Other sites include intracranial vessels; the AT artery is an unusual site. Risk factors for development of mycotic aneurysms are summarised in table 1 (7).

Risk Factors
• Bacteraemia
• Infective endocarditis
• Intravenous drug use
• Immunosuppression
• Iatrogenic aortic trauma
• Pre-existing atherosclerotic disease
• Prosthetic arterial devices (stents, grafts)

Table 1 (7)

Discussion: pathogenesis

There are various potential mechanisms of pathogenesis of mycotic aneurysms. An artery may become exposed to pathogens either by trauma or medical instrumentation(6). Infection may spread from a local source (e.g. abscess) to an arterial wall(8).

Septic emboli may embed themselves in an arterial wall(8), as in the case of our patient; these are likely to have been from the mitral valve vegetations. In patients with bacteraemia, weaknesses in the tunica intima may lead to microbial seeding in the arterial wall(8). Patients with pre-existing aneurysms may also develop infective changes, in particular patients who are immunosuppressed.

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Case history continued: pre-operative optimisation

The patient underwent a *transoesophageal echocardiogram* (TOE) to monitor the size of the vegetation on his mitral valve; this demonstrated increasing vegetation size (>1cm) on the mitral valve anterior leaflet. In this context, anticoagulation in the form of unfractionated heparin was commenced to reduce the embolic risk.

Considering the patient's clinical picture, in conjunction with his new TOE findings and static inflammatory markers, a decision was made to switch to an alternative antibiotic regime following consultation with local microbiology and infectious disease consultant specialists. An amoxicillin infusion with intravenous gentamicin and oral ciprofloxacin was prescribed.

In view of his severe mitral valve regurgitation, the decision was made between a consultant anaesthetist, cardiothoracic and vascular surgeon to proceed to mitral valve replacement first. This was uneventful and the patient made a good recovery.

Following the valve replacement he underwent a *computerised tomography* (CT) positron emission tomography (PET) scan re-assess the false aneurysm and to look for further sites; this revealed progression of the false aneurysm with abscess formation. False aneurysms often enlarge more quickly than true aneurysms; the increasing size of the aneurysm in this case prompted urgent intervention. A subsequent duplex scan demonstrated multiple emboli in the profunda femoris artery.

Discussion: surgical management

Once medical management has been optimised, and certainly in the case of ongoing infection or abscess, the principle behind treating infected aneurysms includes ligation proximal and distal to the aneurysm and performing a surgical bypass if required, usually with a reversed autologous vein. The abscess cavity requires surgical drainage and debridement to ensure removal of unhealthy tissue.

The distal popliteal artery is approached below the knee with a medial incision posterior to the tibia, and heading anterior to the gastrocnemius muscle to approach the neurovascular bundle. In order to access the more distal anterior tibial artery it is frequently required to divide the soleal arch to achieve control of the vessel distally. Where the vessel is thrombosed with reasonable collateral flow into the foot, the need for a vein bypassing is significantly reduced as there are two other vessels (the peroneal and the posterior tibial) which can continue to supply the foot, as well as the aberrant AT artery in this case.

Potential complications from this procedure that need to be discussed with the patient at the time of obtaining consent are summarised in table 2.

Risks Associated With Surgery
· Bleeding
· Infection
· Injury to surrounding structures
· DVT
· Pulmonary embolism
· Limb loss
· Risk to life

Table 2

In cases such as these, there is normally little room for endovascular management due to sepsis and the need to remove the septic focus by surgical debridement. Stent grafting in this area is known to have poor medium and long term patency and its use in frank infection is not recommended.

Case history conclusion

The patient underwent ligation of right infected AT aneurysm, debridement of the aneurysm cavity and vein patch repair of the popliteal artery. He had an uneventful surgical recovery and continued on a long course of antibiotics for graft protection and to complete treatment for infective endocarditis.

Self assessment (only consider single best answer)

1) What are the most common bacterial pathogens found in mycotic aneursyms?

a) Treponema pallidum and mycobacterium tuberculosis

b) Mycobacterium tuberculosis and streptococcus

c) Staphylococcus aureus and salmonella species

d) Salmonella species and listeria monocytogenes

e) Staphylococcus aureus and streptococcus

MANAGEMENT OF MYCOTIC ANEURYSMS - A CLINICAL CASE REPORT

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2) Which of the following is not a risk factor for developing mycotic aneurysms?

a) Intravenous drug use

b) Pre-existing aneurysms

c) Vasculitis

d) Infective endocarditis

e) Immunosuppression

3) Which one is not a recognised potential mechanism for pathogenesis of mycotic aneurysms?

a) Microbial seeding in the arterial wall due to weakness in the tunica media in a bacteraemic patient

b) Artery being exposed to pathogen by trauma

c) Septic emboli embedding in an arterial wall

d) Infection spread from a local source

e) Iatrogenic exposure of an artery to a pathogen

4) Regarding presentations of mycotic aneurysms, which of the following statements is true?

a) All patients with mycotic aneurysms will have elevated white cells and CRP

b) One study found that only 32% of patients complained of pain

c) Presentations of mycotic aneurysms tend to be specific

d) One study found that 24% of patients had positive blood cultures

e) Fever is the most common presenting complaint

5) What modality of imaging is demonstrated in figure 3 (7)?

a) Computerised tomography

b) Magnetic resonance angiogram

c) Digital subtraction angiogram

d) Duplex ultrasound

e) CT angiography

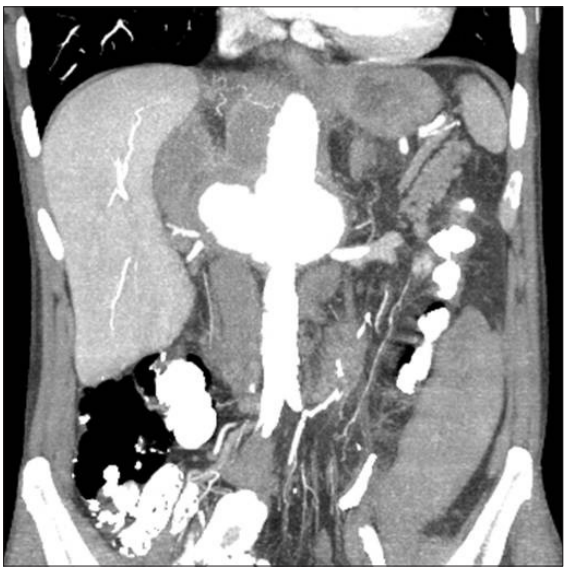

Figure 3

Answers

Answer 1: C

The most common bacterial pathogens associated with mycotic aneurysms are Staphylococcus aureus and Salmonella species(4)

Answer 2: C

Vasculitis. Bacteraemia, intravenous drug use, immunosuppression, infective endocarditis, pre-existing aneurysms & iatrogenic causes are all risk factors for developing mycotic aneurysms(8).

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Answer 3: A

In bacteraemic patients, weaknesses in the tunica intima may lead to microbial seeding in the arterial wall(8). An artery may become exposed to pathogens either by trauma or iatrogenically(8). Infection may spread from a local source (e.g. abscess) to an arterial wall(8). Septic emboli may embed themselves in an arterial wall(8).

Answer 4: D

One study(2) found that 24% pf patients had positive blood cultures prior to surgery. Presentations for mycotic aneurysms are often non-specific(5). One study(2) found that 79% of the patients have elevated white cells or C reactive protein but less than 50% displayed a fever. 76% complained of pain which was local to their aneurysm.

Answer 5: E

Figure 3 (7) is a CT angiogram demonstrating an aortic aneurysm. Contrast can be seen in the arterial tree, there is no evidence of digital subtraction in this image. This is not a magnetic resonance angiogram or a duplex.

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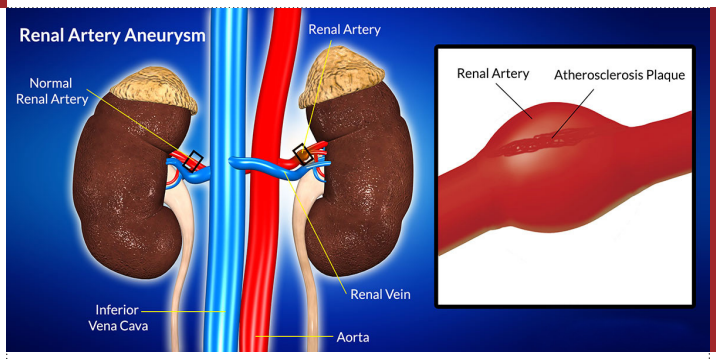
THE PRINCIPLES & MANAGEMENT OF ANEURYSMS OF THE VISCERAL & RENAL ARTERIES

ER Faulconer, AD Fox



The Principles & Management Of Aneurysms Of The Visceral & Renal Arteries
Patient Management

Abstract

Arterial aneurysmal disease within the abdomen is mainly focused on that of the abdominal aorta. With the increased use of different imaging modalities, incidental aneurysms of other arteries are being reported. We present two emergency cases of aneurysms of branches of the abdominal aorta, one true and one false, and discuss presenting symptoms and potential complications of these and other visceral arteries. We discuss the current indications for elective intervention and the different management options available.

Case history 1

A 78 year old female hypertensive collapsed at home with severe abdominal pain. Her past medical history was unremarkable. Current medication included Amlodipine and aspirin.

On examination the patient was in pain and confused. She was pale with a sinus tachycardia of 120 and blood pressure 90/60 mmHg. Oxygen saturation on pulse oximetry was 92%. Her chest was clear but abdominal examination revealed a pulsatile and exquisitely tender abdominal mass within her epigastrium.

What would your initial management be?

- *Complete a target history and informative examination*
- *Institute immediate management with supplemental oxygen.*
- *Establish intravenous access and fluids*
- *Bloods -FBC, U & E, Clotting profile, cross match, glucose.*
- *Early escalation to senior*
- *Consider immediate transfusion*
- *Analgesia as required*
- *ECG*
- *Catheterisation to aid fluid resuscitation*
- *Keep nil by mouth*

The patient was haemodynamically unstable and remained confused despite supplemental oxygen. Arterial blood gases confirmed a metabolic acidosis. You make a clinical diagnosis of a ruptured abdominal aortic aneurysm. You are aware that your patient is in a critical position. Senior review confirms your differential diagnosis and the lady is taken directly to theatres for a laparotomy. She is considered to be too unstable to perform any form of imaging to clarify the diagnosis.

A laparotomy was performed and a large 8 cm diameter aneurysm of her splenic artery was identified that had ruptured into the lesser sac. The aorta was normal. The aneurysm was opened, the feeding splenic artery (both afferent and efferent vessels) within the aneurysm were sutured, haemostasis was secured and resuscitation continued within the ITU department. She made a slow but uneventful recovery.

Case history 2

A 75 year old male diabetic (Type 2) presented to his GP with intermittent right upper quadrant pain following meals. His past medical history included a coronary bypass surgery following a heart attack, a stroke with full recovery and hypertension. His GP diagnosed gallstones disease and confirmed the diagnosis with an outpatient ultrasound scan. His blood tests were deranged: Hb 12.5g/dl, WCC 13 x 109/l, CRP 187, alkaline phosphatase 244, Bilirubin 18.

He was referred to the gastroenteologists who suspected common bile duct (CBD) pathology and organized an ERCP. During the procedure the patient became profoundly hypotensive and complained of abdominal pain. Cannulation of the common bile duct was technically challenging and the procedure was abandoned.

He was treated with fluids and recovered in the department being discharged later that same day. He subsequently underwent a successful laparoscopic cholecystectomy. Follow up some months later revealed ongoing abdominal pain of unknown cause. Examination was suggestive of a pulsatile fullness within the right upper quadrant.

What would you do next?

- *Complete a target history and informative examination*
- *Bloods -FBC, U & E, LFTs, Lipids, glucose*
- *Consider imaging options: ultrasound or CT scan*

An urgent CT scan was performed revealing an aneurysm originating from the gastroduodenal artery (figure 1). Appearances were unusual and thought to be secondary to an iatrogenic wire injury associated with the abandoned ERCP. You discuss the report with the vascular MDT (radiology and surgery). The aneurysm is considered to be a significant rupture risk to the patient and a decision is taken to attempt arterial embolisation to prevent life threatening haemorrhage.

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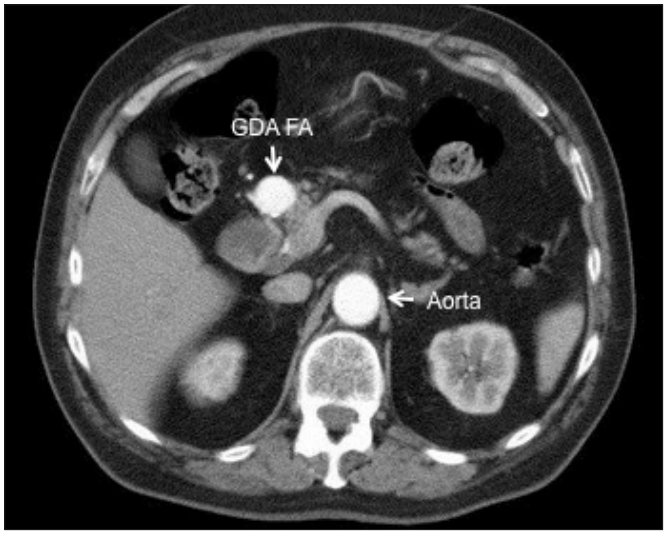

Figure 1. Contrast CT scan demonstrating contrast within false aneurysm arising from gastroduodenal artery (GDA FA).

A microcatheter was passed beyond the aneurysm into the right gastroepiploic artery and embolised with numerous microcoils. A single coil was then placed into the aneurysm itself and then the gastroduodenal artery (figure 2a -b). Completion angiography reveled a small amount of residual filling of the aneurysm and plans were made to perform a CT in 6 weeks. Following this a second attempt at embolisation was made to the inferior pancreaticoduodenal artery (figure 2c) and thereafter further follow up confirmed thrombosis and a reduction in size of the aneurysm.

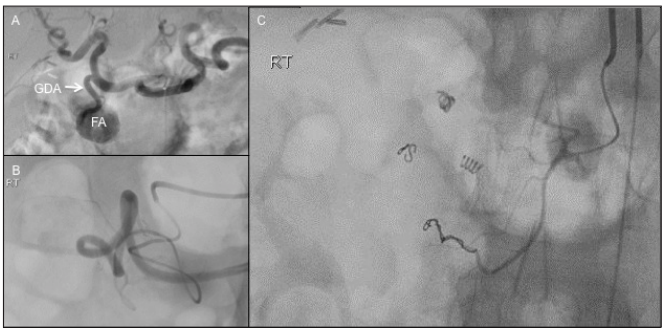

Figure 2. Selective angiogram of ceoliac axis (2A) demonstrating aneurysm (FA) arising from gastroduodenal artery (GDA). Microcatheter directed from vascular sheath to guide placement of embolisation coils (2B). Completed view following embolisation in all feeding and exit vessels showing coils (2C).

Overview of Management

An aneurysm is an abnormal, localised dilatation of a blood vessel of more than 50% that of the non-dilated vessel (1). The most common abdominal arterial aneurysm is one of the abdominal aorta (AAA) with an incidence of 7-8% in men over 65 in the MASS Trial(2) and 1.19% in UK screened male 65 year olds last year (3). Within the abdomen there are many other arteries that may develop aneurysmal disease. These are collectively termed visceral artery aneurysms (4).

Causes of aneurysmal disease include atherosclerosis, trauma, infection, genetic predisposition, inflammatory diseases and haemodynamic changes such as distal to a stenosis or anastomosis (1). A true aneurysm (case study 1) is one that involves all three layers of the wall of an artery (intima, media and adventitia).

A false aneurysm, or pseudoaneurysm, is a collection of blood leaking completely out of an artery or vein. This may follow injury to the vessel as in case study 2 but remains confined adjacent to the vessel by the surrounding tissue. Options for repair include both open surgical repair or endovascular treatment depending on the anatomy of the aneurysm.

Splenic Artery Aneurysm

The splenic artery originates at the coeliac trunk and passes behind the stomach and along the superior aspect of the pancreas in the retroperitoneum giving branches to the pancreas and stomach before terminating at the spleen. These are the most common of the visceral aneurysms accounting for about 60% (4) (figure 3a).

When measuring over 50mm, it is classed as a giant aneurysm. These aneurysms can be found following acute or chronic pancreatitis, in patients with atherosclerotic disease or hypertension or in patients with liver disease such as cirrhosis (5) and seem to present more often in pregnant females (6).

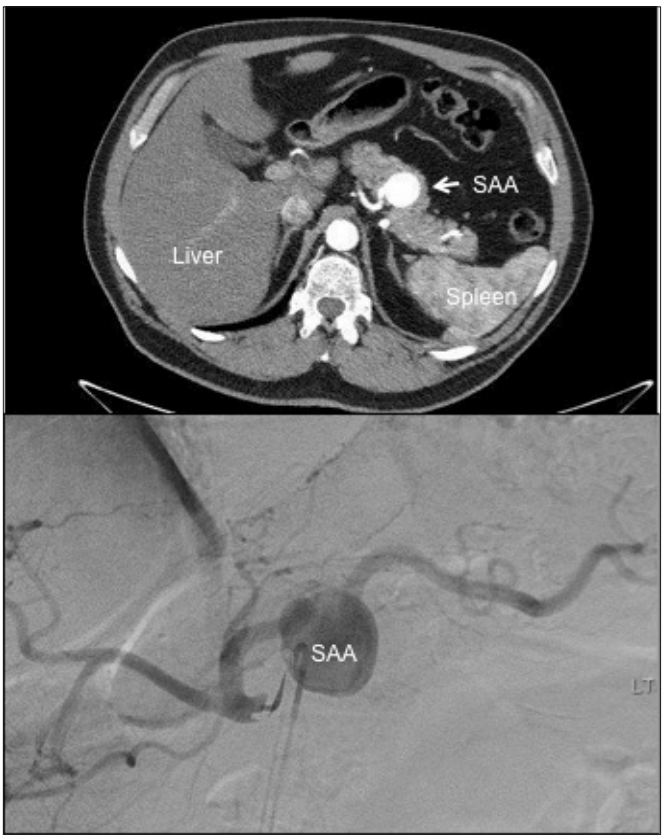

Figure 3: Contrast CT scan (3A) demonstrating splenic artery aneurysm (SAA). Selective ceoliac axisangiogram demonstrating splenic artery aneurysm prior to embolization (3B).

THE PRINCIPLES & MANAGEMENT OF ANEURYSMS OF THE VISCERAL & RENAL ARTERIES

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Splenic artery aneurysms can either fistulate or rupture. Rates of rupture of giant ones increase with size and fistula formation has been reported with communication into the stomach (gastrosplenic), the colon (colosplenic) or the portal venous system (portosplenic). Both rupture and fistula into hollow viscera carry high mortality rates.

Treatment should be offered if the aneurysm is symptomatic, rapidly expanding, above 20mm or in patients who are female and of child bearing age or awaiting a liver transplant (6). First line treatment should be endovascular (figure 3b) as this has been shown to be cost effective and has less morbidity than an open repair (7). It is possible to either place a covered stent or occlude the aneurysm, both of which are safe options and can allow splenic preservation. Embolisation is the favoured technique in some centres (8).

Renal Artery Aneurysm

The renal arteries originate from the aorta below the level of the superior mesenteric artery. The right renal artery passes behind the vena cava and both arteries give off branches to the ipsilateral adrenal gland before dividing at the renal hilum. They may appear as extensions of aneurysmal disease of the aorta or following renal artery biopsy but causes of isolated renal artery aneurysms remains unknown(9) (figure 4) and the incidence is very low in the general population.

Like aneurysms of the splenic artery, those of the renal artery are more at risk of rupture than thrombosis, but reports have also been made of fistula formation with the adjacent renal vein(10). Treatment is indicated above 20mm, in females of child bearing age, if ruptured, if dissecting, showing signs of thromboembolism or if there are symptoms of pain or haematuria(9). Most are associated with hypertension.

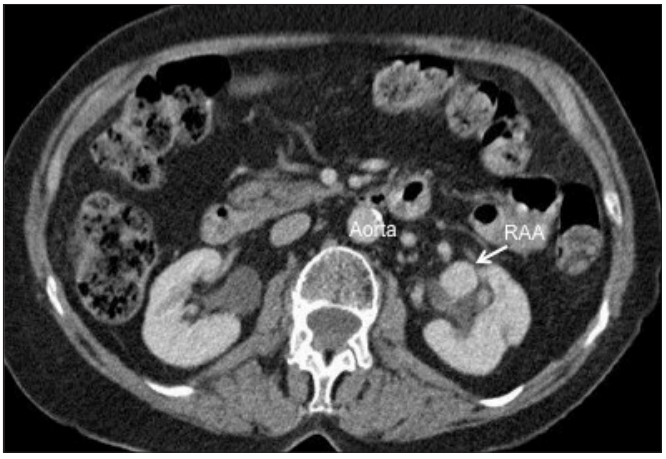

Figure 4: Renal artery aneurysm (RAA) hilum of left kidney.

Treatment options for renal artery aneurysms include surgical excision and reconstruction or endovascular stenting. If there is adequate renal function from the unaffected side then excision of the aneurysm and ipsilateral nephrectomy can be performed rather than performing a more complicated vascular reconstruction . Embolisation of distal aneurysms and stenting of more proximal ones can be performed. A recent review article has not reported any benefit of endovascular approaches over open surgical ones(9).

Hepatic Artery Aneurysm

The hepatic artery originates at the coeliac axis and passes along the lower border of the foramen Winslow giving branches to the stomach, duodenum and pancreas. It divides into the left and right hepatic arteries, which then subdivide to perfuse the different segments of the liver. Most reports of hepatic artery aneurysms are following blunt trauma or pancreatitis and tend to be pseudoaneurysms rather than true aneurysms(11,12).

Treatment by endovascular embolisation is safe as long as there remains collateral perfusion of the liver.

Gastroduodenal Artery Aneurysm and Pancreaticoduodenal Aneurysm

The gastroduodenal artery originates from the hepatic artery and passes behind the duodenum supplying the distal stomach and duodenum. It gives off branches to the pancreas and the duodenum by terminating as it divides into the superior pancreaticoduodenal artery and gastroepiploic artery. True aneurysms of either of these arteries are extremely rare and seem to be associated with atherosclerotic disease or haemodynamic changes from stenosis. Pseudoaneurysms, are more common than true aneurysms (case 2) and have been reported after pancreatitis and trauma(13,14).

Presentation is usually incidental or with symptoms of upper abdominal pain. In cases of rupture or duodenal fistulation, patients can present in extremis with haematemesis. Treatment depends on size and symptoms. Pseudoaneurysms should be treated, as should ruptures and symptomatic aneurysms(4).

Summary and Management principles

1. Aneurysms of branches of the intra-abdominal aorta are rare

2. If discovered, the case should be discussed with the specialist team responsible for managing surgical disease relating to the organ that the artery supplies. This will often involve a multidisciplinary approach. In the case of a renal artery aneurysm, the urology MDT should be involved as an occlusive option or complication would fall to them to manage and may require the nephrologists to provide renal replacement therapy.

3. Options for treatment may include surgery or interventional radiology depending on the site, anatomy and end organ of the arterial aneurysm.

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THE PRINCIPLES & MANAGEMENT OF ANEURYSMS OF THE VISCERAL & RENAL ARTERIES

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These remain rare aneurysms in the UK population and complications of rupture or fistula formation are even more rare. An awareness of the significance of the pathology is important for all clinicians.

Questions

1. A 75 year old male is admitted under the gastroenterology team with hematemesis. At OGD he is found to have evidence of recent bleeding and a clot overlying a pulsation on the posterior wall of the first part of the duodenum. Which artery is most commonly found at this point?

a) Splenic Artery

b) Pancreaticoduodental Artery

c) Left Gastric Artery

d) Gastroduodenal Artery

e) none of the above

2. A 17mm splenic artery aneurysm (SAA) is reported as an incidental finding in a 70 year old male being investigated for an abdominal aortic aneurysm (AAA) which measures 6cm. How should this patient be managed?

a) Ignore both findings and discharge the patient

b) Discuss the patient at the vascular MDT, treat the AAA if appropriate and keep the SAA under surveillance

c) Perform a combined procedure stenting the AAA and embolising the SAA

d) Treat both conservatively for now and rescan in 6 months

e) Perform open AAA repair with excision of SAA and splenectomy

3. On examination of a 75 year old patient complaining of back pain and recent collapse you note a large pulsatile mass in the epigastrium which is tender. The patient is currently haemodynamically stable and has no history of renal failure or allergies. What single investigation would best confirm your diagnosis and allow the surgeon to plan treatment?

a) MR Angiogram

b) CT abdomen and pelvis with oral and iv contrast

c) FAST scan

d) Plain abdominal XRay

e) CT Aortogram

4. Pregnant women should be offered treatment for which of the following aneurysms if present?

a) Splenic artery aneurysm of 5cm

b) Renal artery aneurysm of 2.4 cm

c) Gastroduodenal artery aneurysm of 1.5cm

d) all of the above

e) a and b only

5. Which if the following is not a possible treatment options for a VAA which requires intervention because of the risk of rupture?

a) Foam sclerotherapy

b) Endovascular coil placement

c) Endovascular covered stent placement

d) Laparotomy and ligation of VAA

e) Laparotomy, excision and reconstruction

Answers

1. Answer 'd'

Gastroduodenal artery. This artery is a branch of the common hepatic artery and passes behind the first part of the duodenum. None of the other options pass behind the first part of the duodenum.

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2. Answer 'b'

The splenic artery aneurysm is not of a size requiring treatment and can be monitored, unlike the AAA. Threshold for AAA intervention in the UK is 5.5cm. Patients with threshold AAA's need formal assessment in a vascular clinic followed by a CT aortogram and assessment of fitness for surgery. The results of these should be discussed at the MDT before proceeding to intervention if indicated.

3. Answer 'e'

A CT scan is required to diagnose the presence of an aneurysm and the diagnosis of rupture. It also allows for assessment of suitability for endovascular repair of AAA or planning for occlusion or stenting of VAAs. The other modalities do not allow diagnosis and treatment planning to that level. If the patient in case 1 had been stable enough for a CT scan, the surgeon would have known it was a SAA rather than assuming it was an AAA and may have considered other options.

4. Answer 'e'

a and b are over the threshold of 20mm. c is not above the threshold that is generally considered appropriate and would be best managed by surveillance.

5. Answer 'a'

Foam sclerotherapy is used for superficial venous disease and would not be appropriate in aneurysmal disease. Coil placement and ligation will both render the end organ ischaemic if there is not good collateral circulation. Endovascular stents would need adequate lengths of normal artery proximal and distal to the diseased segment to secure them. Laparotomy and reconstruction may be a very complex procedure depending on the location of the VAA.

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THE DIABETIC FOOT

K Stenson, W Edrees

The Diabetic Foot
Teaching & Training

Abstract

Background

Foot problems are one of the most common complications of diabetes mellitus. 15% of diabetic patients will develop a foot problem in their lifetime. Diabetic foot problems cause considerable morbidity and mortality and are the leading non-traumatic cause of limb amputation. 70% of diabetic patients who undergo amputation will be dead within 5 years. 85% of these amputations are preventable with early diagnosis and treatment.

Aetiology

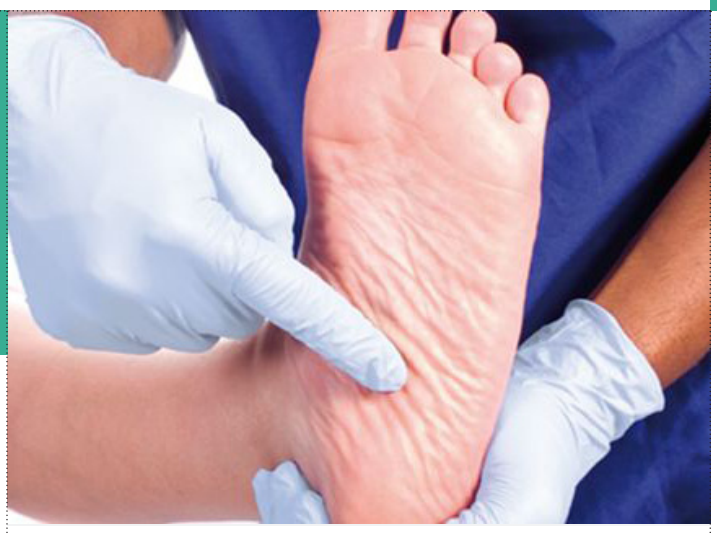
The term, "diabetic foot", encompasses a number of conditions, including peripheral vascular disease, peripheral neuropathy, neuropathic pain, infection, ulceration and Charcot neuroarthropathy. All of these may lead to lower limb amputation. The main aetiological components of diabetic foot disease are peripheral neuropathy, peripheral vascular disease and biomechanical abnormalities in the foot. 45-60% of diabetic foot ulcers are neuropathic in origin, 10% are ischaemic and the remaining 25-45% are of mixed neuroischaemic origin.

Diagnosis and management

The primary goal of treating diabetic foot disease is to promote and maintain mobilisation. A multidisciplinary team approach to looking after patients with this condition is imperative. The mainstay of treatment is prevention by regular screening to identify those at risk of developing complications, and maintaining tight glycaemic control. Treatment of established disease involves treating the causes, specifically treating infection and revascularising the foot where appropriate.

Summary

Every hospital should have a dedicated multidisciplinary team and care pathway for the diabetic foot. Prompt, aggressive treatment has excellent results in terms of patient quality of life and reduction in the rates of amputation and re-ulceration.



The Diabetic Foot

In 2010, it was estimated that 3.1 million people in the United Kingdom were living with diabetes. Diabetes UK predicts that upwards of 4.6 million people will have diabetes by 2030 (1). As people are living longer, it stands to reason that the incidence of diabetes-related complications will increase. Among the many complications of diabetes, foot problems are one the most common; it has been estimated that 15% of diabetic patients will develop a foot ulcer in their lifetime (2).

Diabetic foot problems are responsible for considerable morbidity and mortality, and lead to more hospital admission than do any other complications of diabetes (3). Moreover, diabetic foot disease is the most common cause of non-traumatic limb amputation (4); the International Diabetes Federation stated in 2005 that every 30 seconds a leg is lost to diabetes somewhere in the world (5).

Diabetic foot disease has significant impacts both on the patient and the health service. The individual's quality of life may be severely impaired due to decreased mobility as result of damage to, or loss of limbs; which in turn can lead to loss of employment and independence and depression. The health service is impacted from a financial point of view due to outpatient costs, increased bed occupancy and prolonged stays in hospital.

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Any delay in diagnosing and treating diabetic foot disease can lead to increased rates of morbidity and mortality, as well as higher amputation rates. It has been estimated that up to 70% of patients who undergo amputation due to diabetes will be dead within 5 years (6). Therefore, urgent attention is required wherever the condition is suspected. The primary goal of any treatment is to promote or maintain mobilisation. In order to adequately treat the medical, surgical and rehabilitative aspects of the condition, a multidisciplinary approach is essential.

Such a team will involve vascular surgeons, diabetologists, ophthalmologists, nephrologists, podiatrists, physiotherapists, occupational therapists, as well as tissue viability, vascular and diabetes specialist nurses. Diabetic patients are up to 24 times more likely to require lower limb amputation than non-diabetics (7) and possibly 85% of these amputations are avoidable with early diagnosis and subsequent involvement of a multidisciplinary foot team (8).

Aetiology of diabetic foot disease

"The diabetic foot" covers a multitude of conditions, including peripheral vascular disease, peripheral neuropathy, neuropathic pain, infection (including osteomyelitis), ulceration and Charcot neuroarthropathy. All of these conditions may ultimately lead to lower limb amputation. In terms of foot ulcers, 45-60% of these are of neuropathic aetiology, 10% are ischaemic and 25-45% are of mixed neuroischaemic aetiology (9).

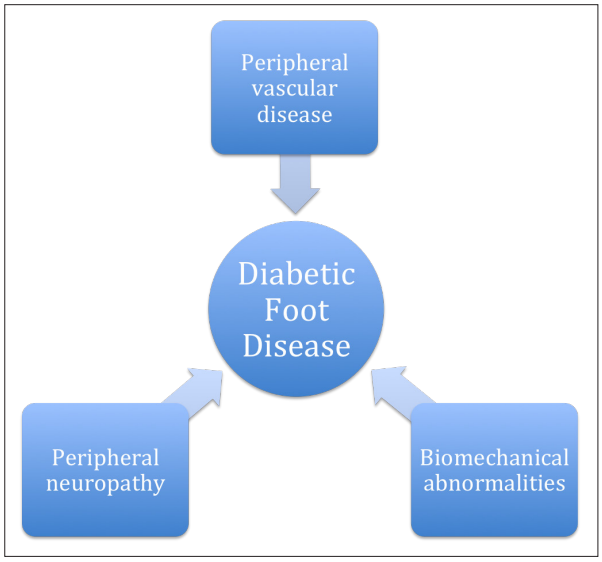

Figure 1: Aetiology of diabetic foot disease.

Peripheral neuropathy

Approximately half of all diabetic patients will have some element of neuropathy; this can range from a mononeuropathy to much more complex polyneuropathies that can involve the autonomic nervous system. Most common in diabetics is a distal polyneuropathy involving sensory nerves, giving rise to the characteristic "glove and stocking" distribution. There is considerable debate as to the exact pathophysiological processes that lead to neuropathy.

It is likely to involve either (i) nerve hypoxia as a result of microvascular disease or (ii) persistent hyperglycaemia acting directly on the metabolism within neurons. It does appear clear however, that there is a correlation between poor glycaemic control over a period of years and the development of neuropathy. Two independent studies (Diabetes Control and Complications Trial and United Kingdom Prospective Diabetes Study) showed that neuropathy could be decreased by up to 60% by tightly controlling blood sugars in diabetic patients (10,11).

Peripheral neuropathy may be subdivided into sensory, motor and autonomic neuropathies. Sensory neuropathy is often manifest by loss of sensation, which is best detected by regular examination of the asymptomatic diabetic foot. In time, patients with sensory neuropathy may develop more unpleasant symptoms, such as altered temperature perception, burning pain, paraesthesia (often described as "pins and needles") and allodynia. This is a very important symptom to identify in diabetic patients, as without the protection of sensation in the feet, unperceived external trauma and repetitive injury can lead to ulceration and infection. This may occur due to something as seemingly trivial as poorly-fitting shoes that rub on pressure areas.

Motor neuropathy is most commonly seen in the most distal muscles of the limb, in particular the intrinsic muscles of the foot. This gives rise to wasting of the intrinsic muscles and as such, alters the shape of the foot profoundly; the metatarsal heads becoming more prominent and the toes taking on a clawed appearance. This can exacerbate the risk of injury to pressure areas brought about by a sensory neuropathy. An early sign of peripheral neuropathy can be callus formation on weight-bearing areas of the foot.

Autonomic neuropathy brings about both vasodilatation in the foot but also microvascular shunting within the cutaneous tissue; this combination renders the skin of the foot very vulnerable to infection and ulceration. Sweating may also be decreased in the presence of autonomic neuropathy. The diabetic neuropathic foot is typically therefore, warm with bounding pulses and dry, cracked skin; it is often hairless.

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Peripheral vascular disease

All types of vascular disease are a major concern in diabetic patients. A World Health Organisation (WHO) study in 2001 found that 44% of deaths in type 1 diabetics, and 52% of deaths in type 2 diabetics were due to vascular disease (12). Diabetic foot disease may develop as a result of both macrovascular and microvascular disease. Macrovascular disease, in the form of atherosclerotic peripheral arterial disease is common in patients who smoke.

Its distribution is often somewhat different to the pattern seen in smokers without diabetes, in that it affects more distal vessels as a general rule. It is often the case that the atherosclerotic disease is diffuse and occurs at multiple levels through the arteries of the lower limb. The profunda femoris artery is affected more commonly than in non-diabetics, so the ability to form a collateral circulation to maintain a blood supply to the leg is reduced.

Microvascular disease in contrast is not related to atherosclerosis; it results from prolonged hyperglycaemia, which causes thickening of the basement membranes of arterioles and capillaries, thus leading to impaired diffusion of oxygen and subsequent tissue ischaemia.

Biomechanical abnormalities

Diabetic patients characteristically develop ulcers at pressure areas. These may be bony prominences that undergo repetitive (painless) trauma, leading to tissue injury. Ulcers may also develop on the plantar surfaces of the feet during standing and walking. High plantar pressures in the presence of sensory neuropathy appear to predispose to ulcer formation (13). Changes that occur in the foot as a result of sensory and motor neuropathy can lead to increases in the plantar pressure. For example, clawing of the toes makes the metatarsal heads more prominent, thus increasing the plantar pressures in these areas, predisposing to ulceration with the repetitive trauma of walking.

The term "Charcot's joint" refers to a chronic, progressive and degenerative arthropathy that occurs secondary to the loss of sensory innervation of a joint and its ligaments. Diabetes mellitus is the cause in the vast majority of cases. The most commonly affected joints are the tarsal, tarsometatarsal and metatarsophalyngeal joints. Such deformities are often associated with joint dislocation and bony deformities, which can also lead to increased foot pressures and ulceration.

Impaired joint mobility is seen in some diabetic patients with chronic hyperglycaemia. Glycosylation of collagen proteins causes the collagen bundles to become thickened and cross-linked; this results in thick, tight, waxy skin that restricts joint movement. When this process occurs at the subtalar joint, the mechanics of walking are significantly affected, which may also be associated with high plantar pressures (14).

Diagnosis and management

Currently, there is no guidance on which investigations are the most accurate or cost-effective in the initial management of diabetic foot disease. Indeed, there is considerable variation in the care of such patients across the country, both at a trust or even departmental level, i.e. whether management is led by vascular or orthopaedic surgeons, or diabetologists.

Of note, there is great discrepancy in the availability of podiatrists who have the required expertise in managing diabetic foot disease. The National Institute for Health and Care Excellence (NICE) have developed a series of guidelines designed to secure consistent, high quality, evidence-based care for patients using the National Health Service; such a set of guidelines for the treatment of Diabetic Foot Problems was published in 2011 (15).

The key priorities set out by the 2011 NICE guidelines are:

- *Multidisciplinary foot care team.*
- *Patient information and support.*
- *Initial examination and assessment.*
- *Care of patients within 24 hours of an admission with diabetic foot disease and the detection of diabetic foot disease in those already in hospital.*
- *Investigation of suspected diabetic foot infection.*
- *Management of diabetic foot infection.*
- *Management of diabetic foot ulcers.*

The NICE guidelines suggest that all care should be patient-centred and take into account patients' needs and preferences. Patients should be given the opportunity to make informed decisions regarding their treatment, in partnership with their healthcare professionals. Good communication is essential. Each patient should have a named contact throughout the care pathway.

Regular foot examination is of paramount importance in this patient population, not only to diagnose existing diabetic foot problems but also to identify those patients who have developed neuropathy. Such patients can therefore be educated and preventative measures be taken to avoid the complications that may arise due to the loss of protective sensation in the feet. Sensory neuropathy can be detected by relatively simple methods such as testing vibration sense with a 128 Hz tuning fork and testing light touch sensation with a 10 g monofilament.

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Clinical examination of the foot should also involve inspection of its shape and the presence of callus; callus formation can represent a valuable early sign of evolving neuropathy and may also identify those patients who would benefit from pressure-relieving footwear. As detailed and accurate measurement of plantar pressures is not widely available, the presence of callus may be a very useful alternative. When there is bleeding into a callus, this suggests impending ulceration and therefore requires urgent attention.

Clinical Features of Diabetic foot disease
Cracked, dry skin
Wasting of intrinsic muscles of foot
Clawed toes
Swollen & deformed ankle joint (Charcot joint)
Swollen & deformed mid- & forefoot with flat foot (due to destruction of midfoot ligaments)
Callus formation on weight-bearing areas
Ulceration/gangrene (dry or wet) at tips of toes
Ulcers over pressure areas, i.e. heel, metatarsal heads, over Achilles tendon and sole of foot (due to altered weight distribution)
Loss of sensation in foot/ insensate foot
Warm foot with bounding pulses, unless peripheral vascular disease present, in which case absent pulses
Signs of abscess & deep, tracking infection

Figure 2: Key features which may be present in the diabetic foot.

There is no proven pharmacological treatment for diabetic neuropathy but there are some drugs that may treat painful neuropathy in diabetic patients. NICE guidelines from 2010 suggest duloxetine or amitriptyline as first line; combination with pregabalin as second line and tramadol or topical lidocaine as third line, whilst referring to a specialist pain service (16).

Ulceration only occurs in the neuropathic foot when there is trauma; this may be repeated trauma due to ill-fitting footwear, or it may be a single episode such as treading on a sharp object whilst barefoot. The additional presence of ischaemia means that the pressure required to bring about tissue breakdown is reduced.

This pathway to ulceration may be halted at various points; (i) preventing neuropathy by maintaining tight glycaemic control, (ii) preventing macrovascular disease by not smoking, (iii) wearing appropriate footwear to prevent trauma leading to ulceration. These facets of prevention are also necessary to allow healing of a foot ulcer.

The most important aspect in the management of neuropathic ulcers is pressure relief and off-loading. This requires bespoke footwear at the very least, and often a period of bed rest. Pressure-relieving boots exist which transfer the load (when walking or standing) from the forefoot to the heel and then directly to the leg via the wall of the boot. In addition, regular debridement of callus and necrotic tissue from the wound is necessary, to allow the migration of epidermal cells from the wound margins and the facilitation of healing.

Neuroischaemic ulcers require very prompt vascular referral, as revascularization is necessary to heal the ulcer and prevent further ulceration. Gangrene may develop in the presence of neuroischaemic ulcers, due to occlusion of digital vessels by septic thrombi produced by microorganisms in infected ulcers.

When gangrene is dry, autoamputation may occur, leaving a healed stump. When gangrene is wet, this indicates infection and local amputation is almost always required; all necrotic and infected tissue must be excised. Closure of the wound should ensure no bone is exposed and that drainage of the wound is possible. Excised tissue, especially bone, should be sent for culture to ensure that the necessary antimicrobial therapy is being administered.

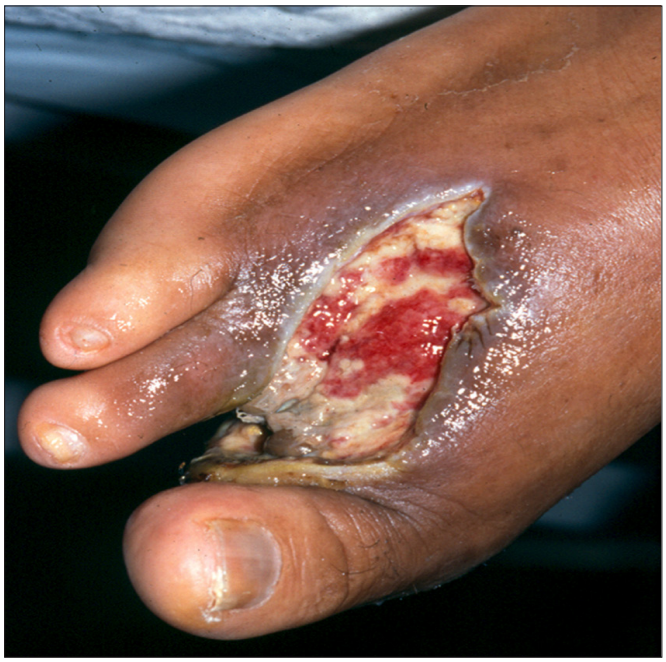

Figure 3: A diabetic foot ulcer showing slough, necrosis and unhealthy granulation tissues. Note the ischaemic looking forefoot with evidence of previous toes amputation.

Infection in the diabetic foot should be treated aggressively as an infected foot ulcer can result in major limb amputation in just a few days. Not all foot ulcers are infected, but most are colonized with bacteria. When there are signs of systemic infection or local inflammation, swelling or abscess, antibiotics must be given. Multiple microbes are often implicated.

NICE guidelines suggest selecting antibiotics with activity again Gram-positive organisms for mild infections, and activity against Gram-positive, -negative and anaerobic organisms in moderate to severe infection. In moderate to severe infection, antibiotics should be administered via the intravenous route. Samples of the ulcer from as close to its base as possible should be sent for culture.

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THE DIABETIC FOOT

K Stenson, W Edrees

Osteomyelitis is a common reason for amputation in the diabetic foot. It may be detected on plain radiography but the classic bony destruction may not be seen for up to two weeks. Magnetic resonance imaging is often more useful as it can reveal evidence of bone marrow oedema before the bony cortex is destroyed. One should have a very low threshold for initiating antibiotic treatment when osteomyelitis is suspected. Any infection must be treated according to local microbiology guidelines to ensure good antibiotic stewardship.

A rather more ancient treatment may be used to treat some necrotic, sloughing or neuroischaemic ulcers: greenbottle larvae (Lucilia sericata) break down and ingest necrotic tissue. Maggots are increasingly being used to successfully treat ulcers in patients with antibiotic-resistant infection (17).

Grade	Denomination	Description
0	Foot at risk	Thick calluses, bone deformities, clawed toes & prominent metatarsal heads
1	Superficial ulcers	Total destruction of the thickness of the skin
2	Deep ulcers	Penetrates through skin, fat & ligaments, bone not affected, infection present
3	Deep ulcers with abscess	Deep tissues always involved, osteomyelitis may be present
4	Limited gangrene	Limited necrosis in the toes or foot
5	Extensive gangrene	Necrosis of the entire foot, with systemic effects

Figure 4: Wagner's classification of diabetic foot disease.

In patients without diabetes, the first presentation of peripheral vascular disease is often intermittent claudication. In patients with diabetes, this may also be the case, but this classic symptom is often absent due to associated peripheral neuropathy. As such, in diabetics, the first presentation of peripheral vascular disease may be distal ulceration in the foot; if the foot is not neuropathic, this ulceration may be painful.

The ischaemic foot is usually cool with absent pulses; if a neuroischaemic foot is warm and swollen, this may well indicate Charcot neuroarthropathy with underlying deep infection. So, it can be difficult to accurately diagnose ischaemia in the presence of neuropathy. Measurement of the ankle brachial pressure index (ABPI) is very helpful in this situation, as it is in non-diabetic patients! ABPI less than 0.9 correlates very well with haemodynamically significant arterial stenosis. In those patients with a history of cramp-like leg pain when walking and without pulses, ABPI < 0.9 predicts the presence of peripheral arterial disease with both a specificity and sensitivity of 95% (18).

It is well known that ABPI readings may be falsely elevated in individuals with diabetes; this is because of the difficulty in compressing the calcified arterial walls with the sphygmomanometer cuff, giving rise to falsely high ankle pressure measurements. In such an eventuality, assessment of the Doppler waveform in the distal vessels is extremely useful, as loss of the normal triphasic signal is indicative of arterial disease.

Arterial duplex scan is commonly used in one stop clinics as non-invasive investigation and it can detect & differentiate between stenotic & occlusive arterial disease especially in the major proximal arteries since arterial calcification reduces it sensitivity in detecting distal arterial disease.

Further imaging with magnetic resonance angiography and less commonly CT angiography may be required in some patients for assessment of the distal arteries before vascular or endovascular intervention. These non-invasive modalities have replaced transfemoral angiography in many vascular centers as the preferred line of investigation.

Revascularisation of ischaemic diabetic foot is an essential aspect of treatment. Endovascular intervention with angioplasty and/or stenting is usually tried as first line of treatment in suitable patients with stenotic, short or moderate occlusive arterial disease. Surgical reconstruction, commonly involves bypass surgery to the distal arteries, may be required in selective group of patients with long occlusive lesions or distal arterial disease where endovascular intervention may be of high risk or unsuccessful. Long-term outcomes of bypass surgery seem to be similar in diabetic and non-diabetic patients (19).

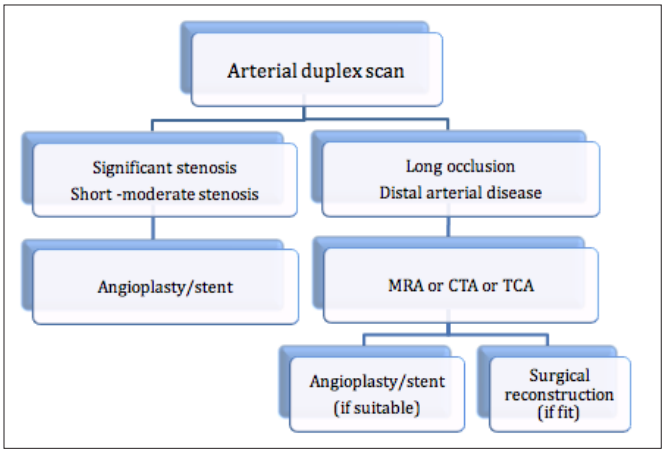

Figure 5: Algorithm for the investigation of arterial disease in diabetic patients. MRA = magnetic resonance angiography, CTA = computer tomographic angiography, TCA = trans-catheter angiography.

Despite significant advances in surgical technique, there is a cohort of patients who are not fit enough to undergo a prolonged distal bypass, despite favourable distal arteries to graft on to. In certain patients, for example those who are very elderly, with ischaemic heart disease and renal failure, a decision to undertake primary amputation may offer a better risk-benefit profile than bypass surgery. Limb salvage requires an aggressive approach to be taken but many diabetic patients will have co-existing conditions that may require optimisation prior to surgery. In the presence of severe foot infection, it is imperative that this is controlled prior to any vascular surgery taking place.

THE DIABETIC FOOT

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Summary

Management of the diabetic foot poses a considerable challenge. Every hospital should have a dedicated multidisciplinary team and care pathway to take care of patients who have, or who are at risk of developing this condition. Regular, thorough screening and patient education is essential, along with tight glycaemic control to prevent foot complications in diabetes. Once ulceration has developed, prompt, aggressive treatment can have excellent results in terms of patient quality of life, and reduction in the rates of amputation and re-ulceration.

Multiple Choice Questions

1: What is the most common cause of diabetic foot ulceration?

A. Infection
B. Ischaemia
C. Neuropathy
D. Trauma
E. Hyperglycaemia

2: Which of the following statements regarding diabetic foot disease is false?

A. Diabetic patients are more likely to require lower limb amputation than non-diabetics.
B. Over half of those patients undergoing lower limb amputation for diabetic foot disease will die within 5 years.
C. 5% of patients with diabetes will develop foot ulceration in their lifetime.
D. Every 30 seconds, a leg is lost to diabetes somewhere in the world.
E. Early diagnosis and involvement of a multidisciplinary foot team can prevent most lower limb amputations.

3: Which of the following is of greatest importance in preventing diabetic neuropathy?

A. Regular podiatry assessment.
B. Good glycaemic control.
C. Regular administration of duloxetine 120 mg daily.
D. Regular assessment by diabetologist.
E. Wearing correct footwear.

4: Regarding diabetic foot infection, which of the following statements is false?

A. Clinical signs of infection may be diminished in the diabetic patient.
B. Untreated infection may rapidly lead to necrosis and major amputation.
C. The distinction between colonisation and infection may be difficult and not always helped by microbiological investigation.
D. Antibiotic therapy should be commenced only once culture and sensitivity results are available.
E. Infections are usually polymicrobial.

5: Which of the following complications of diabetes accounts for the most hospital admissions in the United Kingdom?

A. Diabetic ketoacidosis.
B. Hyperglycaemic hyperosmolar non-ketotic coma.
C. Diabetic nephropathy.
D. Diabetic retinopathy.
E. Diabetic foot infection.

Answers

1. Answer: C. Neuropathy

45-60% of diabetic ulcers are of neuropathic aetiology. 10% are ischaemic and 25-45% are neuroischaemic. Trauma may be implicated in the development of many diabetic foot ulcers, often in the form of repeated painless trauma, but in the absence of neuropathy, this does not lead to ulceration. There is a strong correlation between poor glycaemic control and the development of neuropathy, possibly as a result of hyperglycaemia affecting neuronal metabolism. Infection is a common complication of foot ulceration.

2. Answer: C. 5% of patients with diabetes will develop foot ulceration in their lifetime.

In fact, 15% of diabetic patients will develop foot ulceration. Diabetic patients are up to 24 times more likely to require lower limb amputation than non-diabetics. Diabetes is the most common non-traumatic cause of lower limb amputation worldwide. Up to 70% of diabetic patients who undergo leg amputation will be dead within 5 years. In 2005, the International Diabetes Federation stated that every 30 seconds, somewhere in the world, a leg is lost to diabetes.

Avoiding delays in diagnosing diabetic foot problems can reduce the associated morbidity and mortality Early involvement of a multidisciplinary foot team (including vascular surgeons, diabetologists, neurologists, nephrologists, podiatrists, physiotherapists and specialist nurses) can help to identify those at risk and prevent the development of foot problems in the first place, but can also prevent their deterioration to the extent that amputation is required.

3. Answer: B. Good glycaemic control.

Both the Diabetes Control and Complications Trial and the United Kingdom Prospective Diabetes Study suggested that good glycaemic control was fundamental in the prevention of neuropathy. These studies showed that meticulous glycaemic control reduced clinical neuropathy by 60% and reduced abnormal nerve conduction by 44%.

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4. Answer: D. Antibiotic therapy should be commenced only once culture and sensitivity results are available.

An infected foot ulcer can lead to limb loss in a matter of days, so it is imperative to treat infection aggressively. Empirical antibiotic therapy should be started immediately and then tailored to the individual situation once information from microbiological investigation is available.

Ulcer swabs should be taken from a close as possible to the base of the ulcer, as superficial swabs are most likely to grow colonising rather than pathogenic bacteria. Although clinical signs are sometimes diminished, they are the most reliable indicators of infection; in the presence of systemic upset, e.g. local inflammation, swelling, pus, antibiotic treatment must be initiated.

5. Answer: E. Diabetic foot infection.

Foot complications account for more hospital admissions than any other complication of diabetes. They are also one of the most common complications of diabetes: 15% of diabetics will develop a foot ulcer in their lifetime. Foot disease is a leading cause of morbidity and mortality.

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THE USE OF VIDEO IN IMPROVING QUALITY OF CLINICAL INDUCTION AT ST MARYS HOSPITAL VASCULAR UNIT

S Nahas, C Bicknell

Introduction

Junior doctor changeover in August is associated with a significantly increased mortality of 6% (1). As a result, The Academy of Medical Royal Colleges has instituted recommendations in the 'safe trainee changeover' publication (2), which discusses the need for "high quality clinical induction". Currently, our doctors have a generic combined corporate induction and online training, which meets almost none of the recommendations. We sought to create innovative, cheap, and easily distributable videos in order to dramatically improve the quality of our clinical induction and meet the recommendations.

Methods

A digital video camera was used to make induction videos on several key aspects of the job from current trainees. Trainees from the new August rotation 2014 were given a questionnaire after their corporate induction. They were then shown the videos and then questioned again.

Results

100% of trainees felt that the corporate induction was not sufficient in areas of service processes, personal and comfort requirements, orientation to the new environment, and essential practical skills within the vascular unit. 100% of trainees agreed that the new videos were a useful addition and added value to the induction. 66% agreed that they improved patient safety and that they should be continued as a part of the induction process.

Conclusion

Induction of new trainees is paramount in patient safety, trainee satisfaction, and their effective use in a new job. We have shown that using a simple a cheap and simple tool has the potential to improve induction dramatically.

The Use Of Video In Improving Quality Of Clinical Induction At St Marys Hospital Vascular Unit
Teaching & Training

Introduction

Junior doctor changeover in August is colloquially referred to as 'Black Wednesday', and is well known to be associated with a significantly increased morbidity and mortality (by an average of 6%), as well as an increased the length of stay (1). Notably this data published in 2009 is from a study conducted in a hospital in our Trust.

These figures were unacceptable and as such official recommendations have since been put in place in order to prevent issues associated with junior Doctor changeover. The UK Medical Education Scrutiny group endorsed the Academy of Medical Royal Colleges to investigate the current system. Part of the many recommendations produced by the Medical Colleges are in the 'safe trainee changeover' (2) publication, and include a 'high quality clinical induction'.

Nationally foundation year 1 (FY1) trainees have a minimum of 4 days of salaried shadowing, which occurs prior to employment (2), a minimum of 2 days of which need to be clinically orientated. This clinical orientation is open to interpretation, and may not even be directly relevant to a doctor's new job. It can be argued that 2 days of orientation in a potentially completely new, large and multi-faceted environment is not enough. Notably Foundation Year 2 (FY2) doctors inevitably have no pre-induction in any form.

With this in mind, there is a need for more inventive ideas in order to voluntarily show our trainees the new clinical environment before they attend the workplace, especially for the FY2 doctors. John Lewis department store has been ranked number 10 in The Times Top graduate employers list 2012-2013(5). It offers all employees an induction DVD video (3). The use of video induction has been recommended for use in the NHS in this way.

A survey of 2521 respondents (6) showed that individuals up to the age of 28 learn best when it is 'fun' or through 'mentoring or coaching'(3), which would indicate that if induction is to be improved, then new innovative ideas need to be formulated in order to catch the imagination and interest of our trainees. Notably, the computer based and classroom style teaching (as in our Trust induction) has been shown to be an unpopular way of learning in the under 28 age group (6).

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THE USE OF VIDEO IN IMPROVING QUALITY OF CLINICAL INDUCTION AT ST MARYS HOSPITAL VASCULAR UNIT

S Nahas, C Bicknell

We have chosen to provisionally make a fun, interesting, innovative video series in which FY2s can enjoy watching important topics voluntarily online before they start work. This will not only motivate trainees to watch the videos, but will also make them more memorable. The use of video also allows trainees to refer back to the material.

Choosing which topics to include in a supplementary induction is difficult, and certainly needs to be evidence based. There is guidance in previously published articles (7) (8), as well as Health Education England (9) and the Academy of Royal Medical Colleges (2). A summary of key induction topics found from our literature search can be found in table 1.

Key Topic	Examples and details
Staff	Important for team bonding and introductions of key staff
Tour	Essential in familiarisation with the new environment including key locations specific for the job
IT	Booking job specific investigations, viewing specific investigations, and how patient lists are maintained
Equipment	How to use effectively use equipment that may be specific to that speciality e.g. Doppler ultrasound in vascular
Previous batch of doctors 'job tips'	Key in any job in transfer of information onto the new batch of doctors

Table 1

Previously our induction for junior doctors usually consists of a single day of formal didactic corporate presentations. Our work aims to radically modernize induction, with cheap, effective and innovative ideas. We aim to make induction a fun, inspiring, and motivational process whereby ultimately our doctors become a welcome part of the team quicker, and patient safety and clinical effectiveness is improved all round.

Methodology

Videos were produced using a digital video camera. See figure 1 and figure 2 for example screenshots of these videos. These included a video tour of key areas in the hospital pertinent to a junior doctor undertaking vascular surgery. The existing group of the junior doctors were surveyed regarding their 'top tips' they would impart to new foundation doctors joining the department.

Figure 1: A screenshot of the induction video led by one of the foundation trainees.

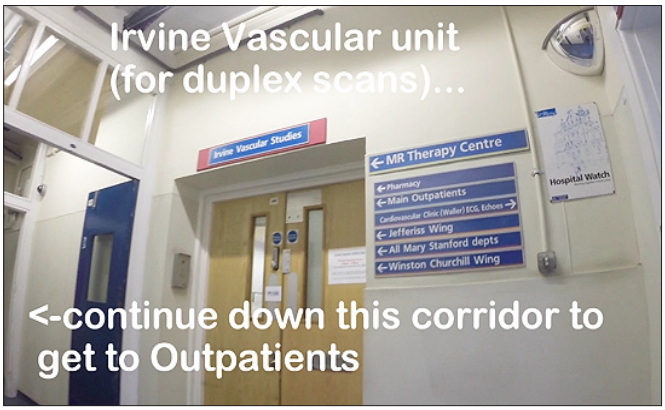

Figure 2: A screenshot from the video Tour of the hospital (with an example of video annotations)

These responses were collated, presented and recorded with two of the foundation year 2 doctors. These responses included key topics, such as how oncall works, daily tasks, how IT systems work, and advice specific to the job.

Based on the aforementioned induction guidance, we submitted our new trainees in august 2014 to the following questionnaire day 1 after finishing their induction:

1. *Were you given appropriate induction on Service processes and procedures within the vascular unit? (Ordering specific vascular investigations, hospital forms and notes relevant to vascular service, admitting and transferring procedures, whom to contact in clinical situation)*
2. *Were you given appropriate induction on understanding vascular services? (Bleeps, switchboard, support staff)*
3. *Were you given appropriate induction on personal and comfort requirements within the vascular unit? (Accommodation, housekeeping, catering facilities)*
4. *Were you given appropriate induction on Orientation to the new environment within the vascular unit? (Mess and hospital facilities, timetables, rota)*
5. *Were you given appropriate induction on essential practical skills within the vascular unit? (CPR and hospital computer)*
6. *Were you given appropriate induction on Professional concerns within the vascular unit?*

After the above questionnaire induction we gave them the induction videos and submit them to the following questionnaire based on a likert scale:

1. *The videos were a useful and informative*
2. *The videos added value to the induction*
3. *The videos were interesting*
4. *The videos added to improving patient safety*
5. *The videos should be continued as part of the induction process*
6. *Are there any other topics you would like covered in the videos?*
7. *I feel like a welcome part of the vascular team*

THE USE OF VIDEO IN IMPROVING QUALITY OF CLINICAL INDUCTION AT ST MARYS HOSPITAL VASCULAR UNIT

S Nahas, C Bicknell

Results

In total three new foundation year 2 doctors joined the vascular unit during the new rotation in August 2014. These doctors attended the Trust induction on day 1, which included the generic corporate induction and generic surgical induction. They had no other official induction process or introduction to clinical areas. The trainees were at this time given the 'pre video' questionnaire, evaluating the trust induction. The same day, trainees were given the video induction, and then given a 'post induction' questionnaire of evaluation.

All trainees felt that the day 1 induction did not cover:

- *Service processes, or procedures within the vascular unit*
- *Vascular services*
- *Personal and comfort requirements*
- *Essential practical skills*

66% of the trainees thought the induction did not adequately cover:

- *Orientation to the new environment.*
- *Professional concerns including educational and clinical supervisors.*

After the induction videos were shown, 100% of trainees agreed that these were useful, informative, and added value to the induction process. 66% agreed that the videos added to improving patient safety.

Discussion

A good induction is paramount in the smooth transition of junior doctors into new jobs. Its success if strongly linked with patient safety. There are now official recommendations for topics that should be included for induction, which cover an array of different areas. We have shown that as things stood, the Trust corporate induction did not cover many important aspects within these areas.

Through reproducible, and easily changeable use of video, we have provisionally shown that our doctors valued this as an addition to the corporate induction process. All of the doctors thought the videos added value to the induction process, and the majority thought that as several key issues were only brought up when the videos were shown and for this reason they improved patient safety.

This project can be built on by adding more of the recommended topics- for example one trainee suggested incorporating theatre systems (such as how to book a patient on to the 'emergency list'). Other key areas in the recommendations were not included in our videos due to time constraints, but will be in future. The sample size was very small, however in due course all of the foundation doctors over several rotations will be sampled, building on our data.

The use of trainee directed video for induction an innovative, easy, and cheap idea, which has captured the imagination of our department. Both trainees and consultants alike are very fond of the idea and look forward to this being taken forward.

We encourage other departments around the country to expand on this in order to lighten, energise and transform induction so that our new doctors can give our patients the best care possible.

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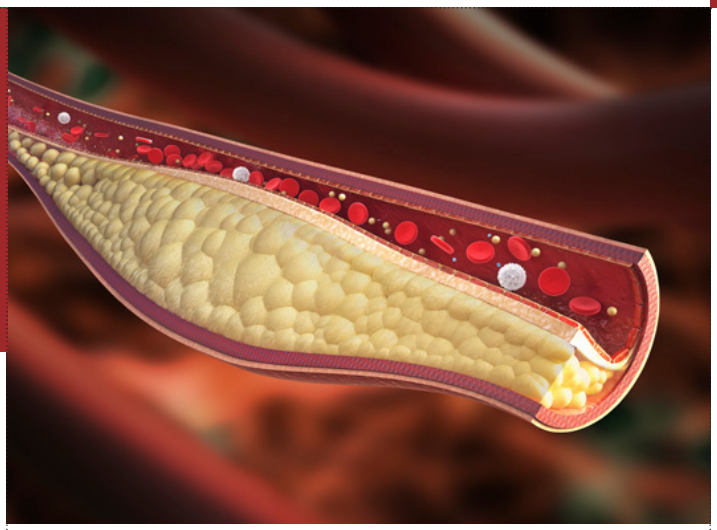
VASCULAR DISEASE OF UPPER LIMB REVISION

S Soulat Raza, AD Fox

Vascular Disease Of Upper Limb Revision
Patient Management



Abstract

Pathology of the arterial and venous systems within the upper limb is rare when compared to the lower limb but the principles of management remain the same. Although rarer, rapid assessment and management is still required for limb salvage and to avoid long term disability with persistent upper limb symptoms such as claudication. Two case reports are presented which demonstrate the variety of clinical signs that can result. The clinical features are discussed and the different aetiologies classified to provide a knowledge base for further reading. The article is aimed at doctors in foundation training and aims to provide the tools and knowledge to safely assess patients presenting with upper limb symptoms, to formulate a differential diagnosis and to understand the different investigations and pathways of care.

Case history 1

A 55-year-old female hypertensive non-smoker presented to her GP with a 2 month history of pain in her right hand and a one month history of colour change within her right index and ring fingers. She had no prescribed medication but on taking her history she described longer standing discomfort within her right arm particularly when raising her arm to hang out her washing. Her hand became stiff and pale and only eased when her arm was returned to the neutral position.

Examination revealed a normal pulse and blood pressure. Her chest was clear. Inspection of her right arm and hand revealed punctate areas of dry ulceration and necrosis of the dorsal aspects of her index and ring fingers with wasting of her finger pulps giving the appearance of spindle shaped fingers (Figure 1a and b). Her radial and ulnar pulses were present. Her left arm was normal in comparison.

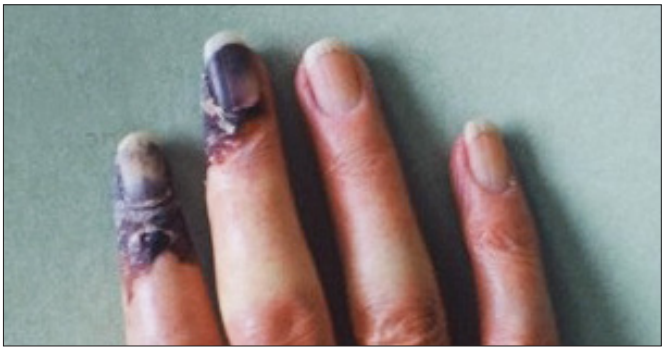


Figure 1a: Necrosis of the index and middle fingers with some associated proximal pallor.

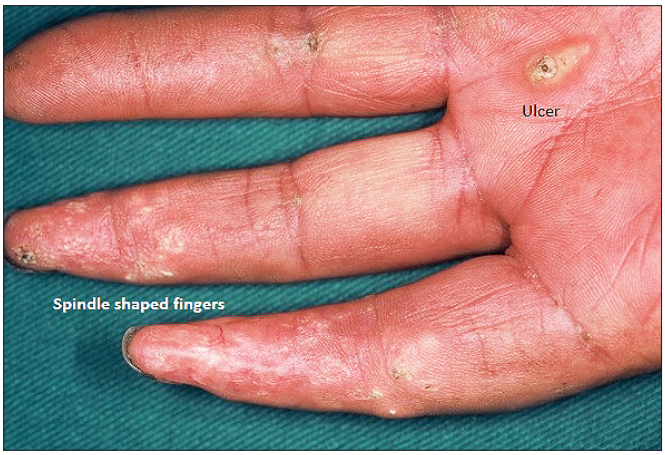


Figure 1b: Palmar aspect demonstrates characteristic punctate dry ulcers consistent with skin infarction secondary to embolisation

What would your initial management be?

- *Complete a target history and informative examination*
- *Bloods -FBC, U & E, Lipids, glucose*
- *Analgesia as required*
- *ECG*
- *Referral to secondary care for further investigation and management*

The patients blood results were mildly deranged:, WCC 15 x10^9/l, CRP 235, Hb 10.4 g/dl, Potassium 5.4 mmol/l, urea 113 mmol/l, creatinine 150 µmol/l, glucose 6 mmol/l. She was in sinus rhythm on her ECG raising doubts as to whether your patient had embolised secondary to atrial fibrillation. You administer pain relief and decide to contact the vascular consultant for advice and guidance. You are concerned that the patient has features consistent with an ischaemic upper limb but remain unsure as to the exact aetiology. The consultant advises immediate referral to the surgical admissions unit.

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Hospital assessment and investigation confirm your findings. An X-ray of her chest and neck reveal bilateral cervical ribs (figure 2a). A CT angiogram is performed followed by a selective upper limb angiogram to clarify the anatomy of the subclavian and axillary arteries (figure 2b). This demonstrates an aneurysm of the subclavian artery. No other pathology is identified. Your patient is discussed in the Vascular MDT and a conclusion made that the post-stenotic aneurysm, containing thrombus, is acting as the embolic source to her hand and fingers. She subsequently underwent surgery to excise the cervical rib and perform an interposition graft to repair the aneurysm and prevent further embolisation. Her medical risk factors were reviewed and medication optimised. She made an uneventful recovery.

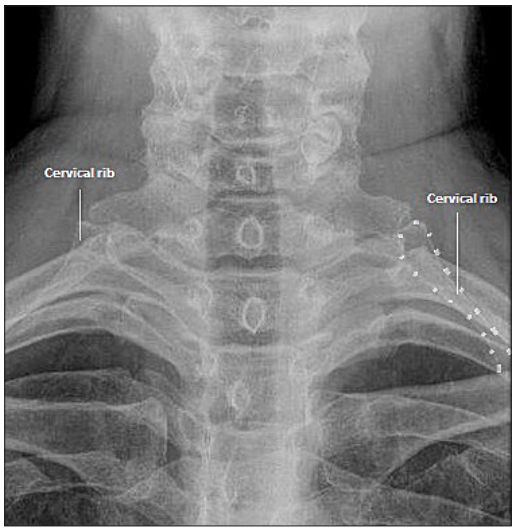

Figure 2a: Bilateral cervical ribs on plain Xray. Left cervical rib highlighted.

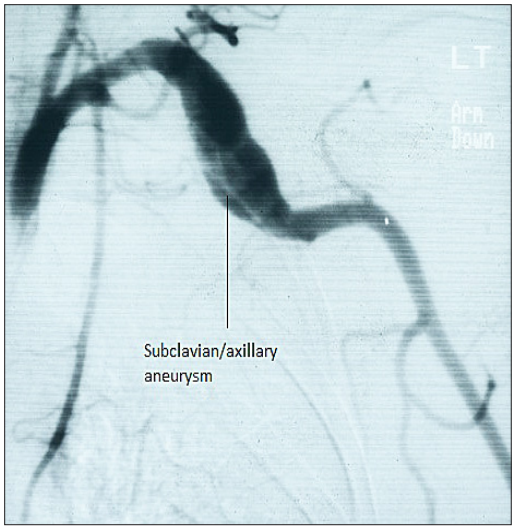

Figure 2b: Subclavian artery aneurysm.

Case history 2

A 28 year old man presented to the accident and emergency department with a 2 day history of pain and swelling in his left arm. He had no past medical history of note, smoked 15 cigarettes per day and worked on a building site. As well as being physically active at work he enjoyed bodybuilding at his local gym.

You examine him and find a healthy patient. His blood pressure was normal. Inspection of his left arm revealed that is was swollen to elbow level. It was redder in appearance compared with the other side. There was no pitting oedema. Both radial pulses were present and symmetrical. You notice some congested veins around the anterior aspect of his left shoulder (figure 3).

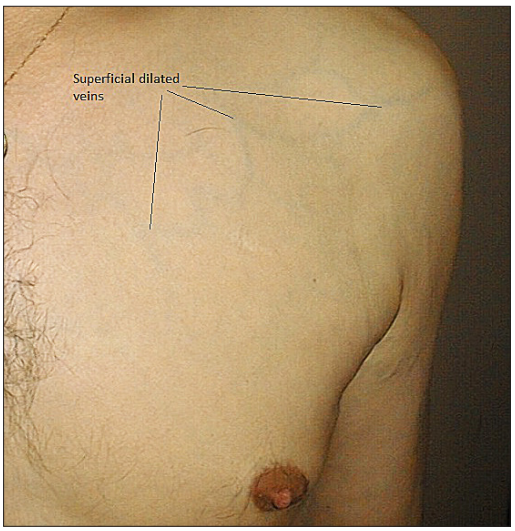

Figure 3: Prominent veins on chest wall signifying collateral venous outflow from arm due to thrombosed axillary/subclavian vein.

What would your initial management be?

- *Complete a target history and informative examination*
- *Bloods -FBC, U & E, Lipids, glucose and D-dimer*
- *Analgesia as required*
- *ECG*
- *Referral to specialists for further investigation and management*

The patient's results revealed an elevated D-dimer suggesting a possible thrombotic event. No other abnormal results were identified. You refer him to the medical team who advise on anticoagulation and ask you to contact the vascular surgeons as they have an interest in upper limb deep vein thromboses in the younger population. The vascular surgeons review the patient and discuss their differential diagnosis of Paget Schroetter syndrome (1).

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Your patient is admitted to the vascular ward for planned imaging and further management. You follow your patient's progress. A duplex confirms thrombosis of the axillary vein . The patient is thrombolysed successfully, the vein stricture is dilated using an angioplasty balloon although no stent is deployed (figure 4) and the patient is then anticoagulated. You understand that he will undergo a first rib resection in the near future to decompress the thoracic outlet and to help prevent the risk of further thrombosis.

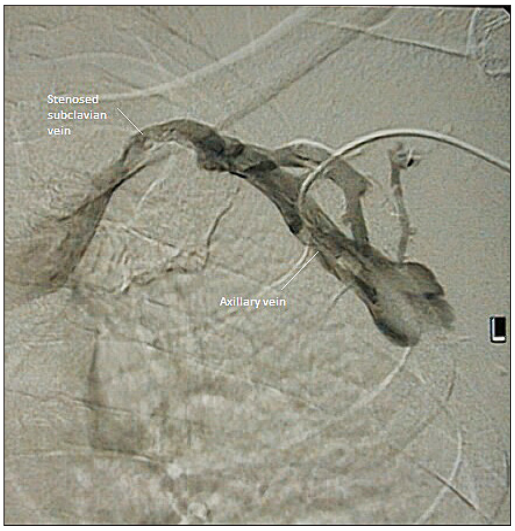

Figure 4: Narrowed and irregular subclavian vein despite subclavian vein angioplasty. Post thrombolysis.

Assessment of Upper Limb Vascular Disease

Diagnosis

Patients can present with acute and chronic symptoms depending on the site and extent of the vascular insult. Differentiation between arterial and venous pathology can be difficult but exactly the same principles of diagnosis are used in the upper limb as in the lower limb (2,3).

The commonly described features of acute ischaemia include pain, pallor, paraesthesia, pulselessness, perishing cold and paralysis. These features reflect the immediate cessation of blood flow to the peripheries and the resultant physiological tissue response. An acutely ischaemic limb needs rapid diagnosis, investigation and management to avoid limb loss and to reduce the cardiovascular risk of death. The most common cause is due to embolisation usually from a proximal source of blood clot – often the heart in those patients with atrial fibrillation. Thrombosis in situ can occur in those with subclavian and axillary artery pathology and occasionally in those patients who have undergone radiotherapy for breast cancer.

It is essential to grade of the level of ischaemia and ascertain if the limb is viable at presentation. Symptoms of arterial disease can be non-critical including weakness of the arm and claudication, or critical (impending limb loss) including rest pain, gangrene or ulceration of digits due to micro emboli, vasospasm or occlusion. The skin of the arm will become mottled but the limb can be saved if the mottling blanches with local pressure implying reversibility. After 3-6 hours of severe ischaemia muscle and nerve undergo irreversible changes and the mottling will start to become fixed.

A careful history may reveal symptoms of dizziness or syncope during arm exertion. This results from cerebral hypoperfusion due to retrograde flow of blood down the ipsilateral vertebral artery due to an occlusion or stenosis of the subclavian artery proximal to the origin of the vertebral artery. This is known as subclavian steal syndrome. Thoracic outlet syndrome (TOS) describes a spectrum of symptoms that may be both vascular and neurological. It is not the purpose of the article to describe TOS in details but case 1 would be classified as a secondary complication of vascular TOS. The signs may also be present in normal individuals and a combination of clinical findings together with other investigations will help aid diagnosis.

Small vessel arterial disease can result in skin colour changes and are typically seen in cold weather (Raynaud's phenomena) or by repetitive movements (vibrating tools). The history must therefore include an occupational history. Co-existing medical conditions like connective tissue disorders, myeloproliferative disease, recent history of radiotherapy and cancer can often help in forming differential diagnosis of the vascular pathology. The risk of in situ thrombosis is higher in those patients with active malignancy.

Venous occlusive disease of upper limb usually presents with painful swelling of the arm worse on exercise. The veins on the back of the hand may be distended and there may be evidence of collateral venous flow where dilated veins are present over the upper chest wall. Some patients will have discolouration and cyanosis of the arm. The degree of swelling will vary according to the level of venous occlusion.

Clinical evaluation of upper limb vascular problems

Physical examination includes a detailed cardiovascular examination, examination of the thoracic outlet along with a targeted peripheral vascular examination including inspection for signs of arterial or venous insufficiency like pallor or cyanosis, atrophy of muscles and the presence of collateral veins on the chest wall. Palpation and auscultation of carotid, axillary, brachial, radial and ulnar pulses is essential and comparison with the contra lateral arm is mandatory.

The pulses should be palpated in neutral position and with arm abducted and externally rotated (surrender position) to detect arterial thoracic outlet compression. The loss or reduction in radial pulse during Adson's manoeuver (abduction and external rotation of shoulder whilst taking a deep breath then extending and turning the head to the side being tested) indicates thoracic outlet compression. The test is not very reliable as it can be positive in up to 53% of healthy volunteers (4).

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To assess the palmar arch Allen's test is performed, the test involves occlusion of the ulnar and radial arteries with the fist clenched, the hand is then opened, releasing one of the arterial occlusions (radial or ulnar); prompt capillary refilling under 10 seconds should result. The same maneuver should then be performed with the release of the other artery, if the palmar arch is not intact; the release of the affected artery produces a sluggish capillary refill. Blood pressure should be measured in each arm using hand-held Doppler, a difference of more than 15 % is considered abnormal. Future blood pressures should always be performed using the arm with the higher pressure although this assumes that the pressure in this arm is normal.

Some signs will point to a specific diagnosis such as a pulsatile mass in the supra clavicular fossa indicating a possible subclavian artery aneurysm.

Assimilation of the above facts at the conclusion of examination should allow you to distinguish between venous insufficiency and arterial disease and allow you to establish a differential diagnosis that can be confirmed or excluded by further investigations.

Doppler assessment is a useful investigation to establish the presence or absence of arterial flow. Local pathology can be investigated using plain X-rays (evidence of cervical ribs, previous trauma case history 1) or non-invasive ultrasound imaging (thrombosis, embolisation, dissection, false aneurysm - case history 2). Ultrasound should be considered the initial investigation of choice and helps establish which other investigations should follow for both arterial and venous assessment.

Cross sectional imaging (CT angiogram), MR angiography or venography are useful modalities that provide rapid information to aid diagnosis and operative planning. Invasive angiography or venography will provide additional information as well as providing an opportunity to treat some of the underlying pathologies. Electrophysiological studies may also be requested to assess nerve involvement – these my be abnormal in some patients following trauma to the upper limb and also in those with a cervical rib or band causing compression of the lower roots of the brachial plexus.

Overview of Management

Timely assessment and intervention improves outcomes and prognosis. The principles in acute ischaemia in the upper limb are the same as those in the lower limb. They include patient resuscitation (oxygen, iv fluids, bloods, heparinization. catheterisation) and assessment of limb viability. Acute on chronic presentations require exactly the same management but may vary in the speed with which the problem is investigated and treated. Outcome is dependant upon treating the patient in a holistic way. All risks factors should be investigated and addressed to establish a successful short and long term treatment plan.

A number of interventional and operative treatments may be considered for limb salvage (heparinisation, anticoagulation, thrombolysis, embolectomy, thrombectomy, endoluminal stents, bypass grafts). This may be a simple procedure under local anaesthetic (brachial embolectomy) but may need a multidisciplinary approach to treatment and possible complex bypass grafting (carotid to brachial bypass in patients with radiotherapy induced arterial thrombosis). A collateral circulation can develop quickly in more elderly patients who are not using their arms significantly. They may find that they remain asymptomatic even without surgery.

Administration of heparin prevents further propagation of thrombus within the occluded artery and vein. The prescription of longer-term anticoagulation should be discussed early with the senior members of the team.

Some patients presenting with acute ischaemia are at risk of developing compartment syndrome. Pain out of proportion to what is expected and the reappearance of the signs of ischaemia should raise concerns. Immediate senior support is required. Fasciotomies may however be performed at the time of surgery to protect against this scenario. This may require both vascular and upper limb orthopaedic input depending on the experience of those involved.

Causes of upper limb vascular disease

Arterial	Venous
1. Occlusive	**1. Occlusive**
Large artery Thoracic outlet syndrome Atherosclerosis Radiotherapy Arteritis (giant cell and Takayasu's) Trauma **Medium and small artery** Connective tissue disorders (CREST , scleroderma & Lupus Atherosclerosis Buerger's Vibration syndrome Trauma	**Sub clavain axillary vein thrombosis** **Primary** Thoracic outlet syndrome (Paget- Schroetter syndrome) **Secondary** Central venous catheters Hypercoaguable states (Malignancy, Anti thrombin III and protein C&S deficiency, Anti phospholipid Syndrome, Cryoglobulinaemia)
2. Vasospasm	
Large artery Ergot containing medications **Small artery** Raynaud's disease Cocaine use Vibrating tools	
3. Embolic	
From proximal sources Heart Ulcerated arterial plaques from major vessels of thorax Thoracic outlet syndrome **Distal embolic disease** Vibrating tools Arterial line insertions	
4. Aneurysmal Brachiocephalic, Subclavian ,axillary brachial, radial and ulnar arteries. Post stenotic	
5. Iatrogenic **Procedure related, arterial lines**	

The table above illustrates a classification of both arterial and venous disease.

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Arterial disease

Arterial conditions are considered more important than venous pathologies primarily because any delay in diagnosis and management can cause significant long-term disability and loss of function.

A detailed discussion of all arterial pathologies is beyond the scope of this article but we will discuss a few common pathologies starting from a more proximal upper limb position.

Brachiocephalic artery stenosis is asymptomatic in 13- 22 % of patients (5,6), however the majority of patients have varying symptoms including ischemia of upper limb, carotid territory symptoms or vertebrobasilar symptoms. Signs include cervical bruit, absent axillary pulse and can be confirmed by duplex scanning, MRA/ CT or conventional angiography. Aortic arch lesions can be present in 61-84% of patients with brachiocephalic artery occlusion (7). The management includes open surgery, often extra-anatomic in nature (Aorto –brachiocephalic bypass) with endarterectomy reserved for distal segment disease only. Less invasive endovascular treatment with angioplasty or stent placement can also be performed.

Subclavian artery occlusion can present with similar symptoms and signs and in the presence of healthy ipsilateral carotid artery and can be managed with carotid- sub clavain bypass or transposition as the method of choice.

Thrombo embolism of large vessels is the main cause of upper limb acute ischaemia and accounts for 20-32 % (8) of all the peripheral cases. Embolism from atrial fibrillation is the source in at least 50% of cases whereas in 30 % recent myocardial infarction is the source of embolism. It usually affects the larger vessels brachial, axillary, sub clavian and forearm arteries are involved in 55, 25, 18 and 2 % cases respectively. The brachial bifurcation is the most common site for the embolus to lodge.

Management of acute ischaemia includes immediate systemic heparinisation to limit the propagation of thrombus and to prevent recurrent embolism followed by emergency surgical thrombo-embolectomy. Imaging is usually not required except if a cardiac source is not evident or if the Subclavian artery pulse is either absent (dissection or occlusion) or unduly prominent (aneurysm of Subclavian artery). Intravenous drug abuse, ergot poisoning, dopamine overdose and cytotoxic drug administration can also cause acute upper limb ischaemia so these causes should be excluded by careful history taking and examination.

True aneurysms of the upper limb are uncommon the two most common arteries effected include the subclavian artery due to TOS and the axillary artery due to blunt or penetrating trauma. Radial artery aneurysms are seldom seen and mostly are caused by infection or inadequate compression following intra arterial catheter insertion. If Allen's test is normal then the management is simply to ligate the artery above and below the aneurysm. Ulnar artery aneurysms even though rare are far more important to recognize because it may lead to digital necrosis.

The two other conditions that are worth mentioning including Buerger's disease which is characterized by segmental thrombotic occlusion in small and middle sized arteries in male smokers under 45 years old and Raynaud's disease where patients have vasospastic disorders without underlying systemic conditions or occlusion. The main stay of treatment for both is conservative management, avoidance of smoking for the former and avoidance of cold conditions for the latter.

Venous disease

The most commonly seen venous pathology of the upper limb is deep venous thrombosis of the Subclavian –axillary vein thrombosis. Thrombosis of subclavian- axillary vein can be divided into primary and secondary. Primary is termed when there is no underlying pathology to account for the thrombosis. Primary Sub clavian –axillary vein thrombosis has male preponderance with a male to female ratio of 2:1 is usually seen in young patients with the right upper limb three times more likely to be effected than the left. The first choice of investigation is a duplex scan which is non-invasive and has a high sensitivity (94%) and specificity (96%) compared to MRA and venography (). The treatment of choice is anticoagulation and thrombolysis but in patients with thoracic outlet syndrome thrombolysis should be followed by first rib excision to prevent re thrombosis.

Secondary subclavian-axillary vein thrombosis is most commonly caused by central venous catheter insertion and pulmonary embolisation is not uncommon in these patients. Treatment includes anticoagulation until the catheter can be removed.

Summary and Management principles

1. Acute and chronic arterial and venous symptoms in the upper limb require prompt assessment and management to differentiate between conditions that may result in limb loss or long-term disability.

2. A multi disciplinary approach is necessary for most diagnoses in both the short and long term management of risk factors – GP, radiology, medicine, Vascular surgeons, orthopaedics, haematology (prothrombotic syndromes), rehabilitation teams (physiotherapy and occupational therapists)

3. Patient education - Early presentation for an acute event. Simple advice regarding risk reduction (excessive weight lifting – Paget Schroetters). Consider genetic counseling for inherited thrombophilia.

4. To understand that the same principles apply when dealing with either the upper or lower limb.

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MCQs

1. 45 years Asian female presents with light-headedness to A&E. On examination both the radial pulses are absent and her Blood pressure is 85/55. blood tests demonstrate anaemia and high CRP. What is the most likely diagnosis?

A. Ruptured abdominal aortic aneurysm
B. Takayasu's arteritis
C. Thoracic outlet syndrome
D. Aortic dissection
E. Hypothenar hammer syndrome.

2. A 65 year old male smoker in atrial fibrillation complains of pain over his right wrist two weeks after discharge from ITU where he had been admitted with severe pancreatitis. A left subclavian vein catheter and a right radial arterial line had been inserted for inotropic support and fluid resuscitation. A firm, red, tender swelling was identified over the wrist. What is the most likely diagnosis and cause for this condition?

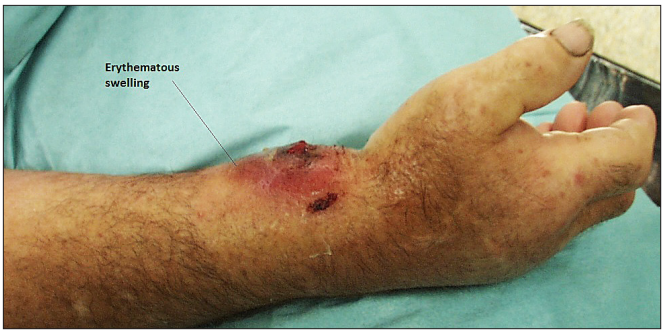

Figure MCQ 2

A. Right Radial artery pseudo aneurysm due to infected arterial line.
B. Subclavian –axillary vein thrombosis due to central line insertion.
C. Right brachial artery embolism due to atrial fibrillation.
D. Haematoma of the right radial artery due to arterial line insertion.
E. Buerger's disease.

3. Which statement is true for subclavian artery anatomy?

A. The subclavian becomes the axillary artery at the border of 2nd rib.
B. The subclavian artery on the right is a branch of brachiocephalic trunk.
C. Subclavian artery on the left is shorter in length as compared to right sub clavian.
D. The common carotid artery is a branch of right subclavian artery.
E. The subclavian artery travels anterior to the scalenus anterior.

4. A 20 year old reports that her hands are constantly moist at work and she finds it difficult to hold objects or chop up meat and vegetables. Her symptoms are better at home. She seldom socialises and finds it difficult to make friends. On examination she appears anxious and refuses to shake hands with the doctor. The examination is normal but the doctor notices shiny and sweaty palms. Which statement describes her diagnosis and initial management in this case?

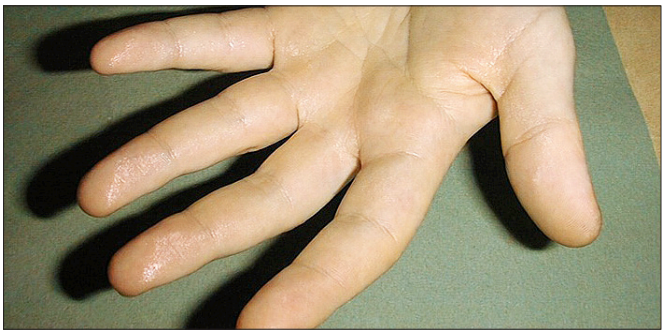

Figure MCQ 4: Glistening appearance to sweaty hand.

A. Thyrotoxicosis, check thyroid functions tests and arrange urgent endocrinology referral.
B. Vibration syndrome, encourage to switch profession to avoid repetitive movements.
C. Generalised anxiety disorder, cognitive behavioural therapy and prescribe a small dose of selective serotonin reuptake inhibitors.
D. Rheumatoid arthritis, arrange blood test and x-rays of hands.
E. Palmar Hyperhidrosis prescribe oxybutynin and arrange review in 4 weeks.

5. A 40 year old lady presents to A&E with a 3 month history of pain in the neck, weakness of her hands and numbness of right 4th and 5th fingers. She was involved in a car accident being hit from behind by another vehicle at 20miles/hour. She had no symptoms following the accident and did not seek medical advice at the time. On examination she is found to have tenderness of right scalene muscle and wasting of the small muscle in the hand along with reduced sensation at ulnar nerve distribution, her symptoms are duplicated when the arm is abducted and externally rotated at 90 degrees (AER position). What is the most likely diagnosis here?

A. Cervical spondylosis
B. whiplash injury
C. Brachial plexus traction injury
D. Neurogenic Thoracic outlet syndrome (NTOS)
E. Ulnar nerve compression due to missed supracondylar fracture

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Answers

1. Answer B

Takayasu's arteritis or pulseless disease is a form of large vessel granulomatous vasculitis affecting young or middle-aged women of Asian descent. Hypertension can occur due to renal artery stenosis and can be associated with anaemia and high inflammatory markers.

2. Answer A

The patient had right radial arterial line insertion and has a pseudo aneurysm of right radial artery due to infected arterial line. He has swelling pain and erythema over the site consistent with infection and the presence of pulsatile mass strongly suggests pseudo aneurysm instead of haematoma.

3. Answer B

The right subclavian artery is a branch of brachiocephalic trunk and the left originates from the arch of aorta directly. It travels posterior to the scalenus anterior and is shorter on the right as compared to the left. The usual branches of the subclavian on both sides of the body are the vertebral artery, the internal thoracic artery, the thyrocervical trunk, the costocervical trunk and the dorsal scapular artery. The subclavian becomes the axillary artery at the lateral border of 1st rib.

4. Answer E

The patient had an otherwise normal examination with excessive sweating of the palms only. Thyrotoxicosis is likely to produce other signs along with generalised sweating. Vibration syndrome is seen in people who work with vibrating tools and tend to have pain and signs of small vessel ischaemia not excessive sweating. The most likely diagnosis is primary hyperhidrosis in young patients who present with excessive sweating either of axilla, feet or palms caused by sympathetic over activity. This can result in occupational, psychological and social issues. Patients become anxious and socially withdrawn, the condition has been termed a silent handicap. Oxybutynin, an anticholinergic drug has shown some promise, other treatments include Botox, chemical or surgical sympathectomy.

5. Answer D.

The signs of scalene muscle tenderness, atrophy of small muscles of hands (T1 nerve root compression and motor loss) and paraesthesia (C8 nerve root compression) along with reproducibility of symptoms with passive abduction and external rotation (AER) are classical for NTOS. Loss of the radial pulse on Adson's test or in AER position is unreliable and may be found in normal people as well.

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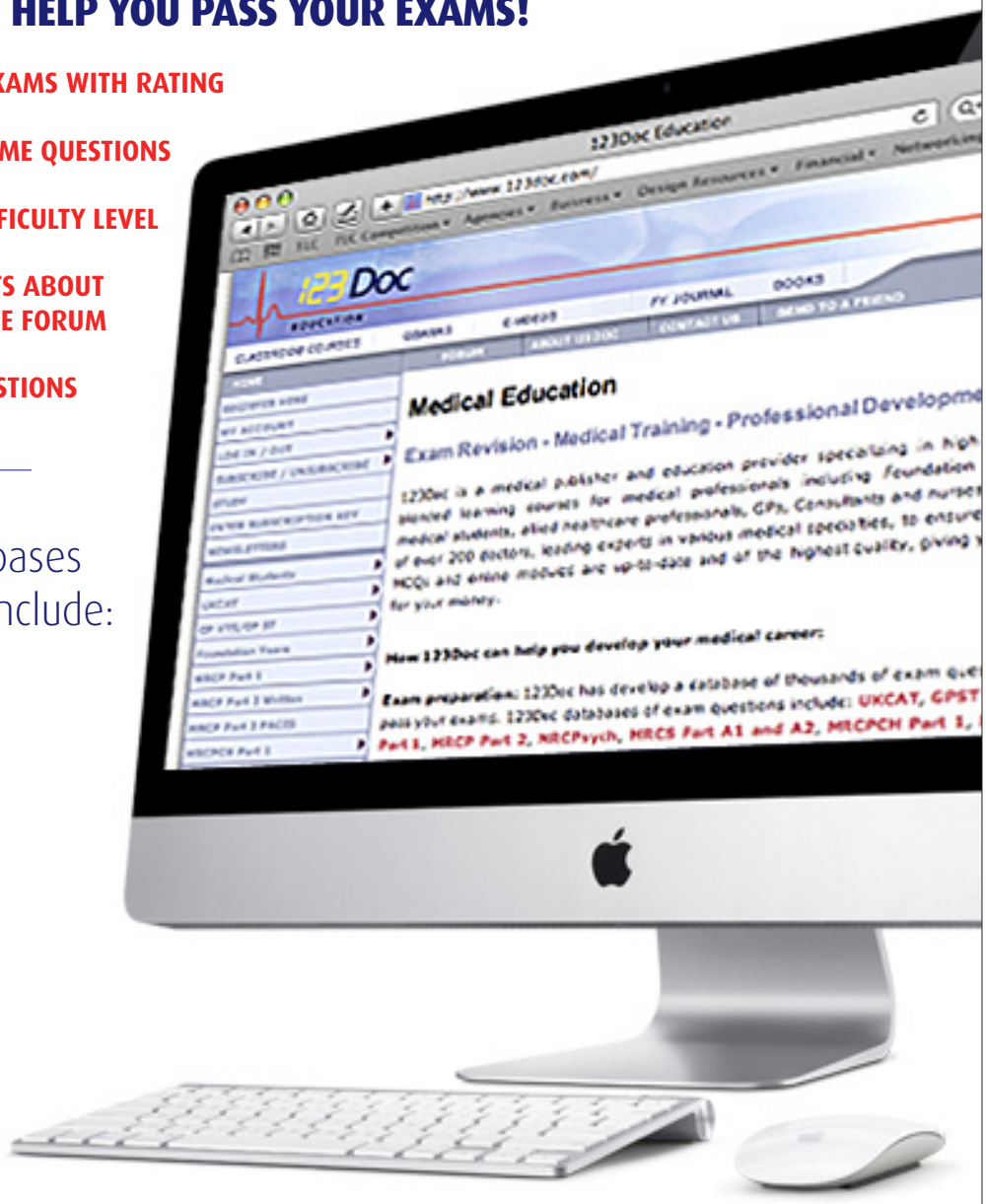

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