Handbook for Postgraduate Doctors

Vascular Surgery

09/2023

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Contributions by Vascular Surgery Registrars: Mr Ahmed Hassan, Mr Muhammad Kamel, Mr Goran Majeed, Mr Tawfik Omar, Mr Alex Sergiou

Dear new colleagues,

Welcome to Vascular Surgery!

This guide is to help you navigate your new job. We are all open to feedback and adjustments, so please let us know if it is incomplete.

We will start this guide off with introducing the teams and an outline.

There are two consultant teams with three consultants each. Registrars are not team bound, but will be assigned to cover ward round for one of the teams on a daily basis. For juniors, it is usually helpful to try and stick to the same team to guarantee some continuity for our patients.
You will get some allocated time in theatre and to support on call services, both very helpful for WBAs and to experience the life of a vascular surgeon.

Team 1: Miss S Sayed (4822), Mr A Elsharkawy (1724), Mr F Kahloon

Team 2: Mr A Mahmood (2353, clinical lead), Mr D Srinivasamurthy (4823, educational lead), Mr N Matharu (1786, surgical lead)

Registrars:
Senior trustgrade registrars: Ahmed Hassan (rota coordinator) and Tawfik Omar
Senior HEE registrar: Kamel Muhammad
Junior HEE registrar: Starting in October
Junior trustgrade registrar: Alex Sergiou (allocation rota), Goran Majeed
Also, there will be one General Surgery registrar on their Vascular Surgery rotation

Contents

Day at work for FY1/FY2/SHO

Normal working days on the ward

- FY1s only: Get in at 07:30, open My Computer → G drive (check your emails for your access link) then, go to vascular→Combined lists- vascular and pick the list of today
- Check where all the patients are using vital pac (intranet→ hosportal→vital pac) and add their locations and observations to the list.
- The on call registrar will update you on any new patients to the whatsapp group
- Add patients from yesterday's theatre list to the list as well
- Ward round will start at 08.00, the registrar will find you on ward 22a
- Things to bring with you: multiple dressing packs, roll of tape
- If the registrar is on call/in theatre/clinic: do not hesitate to contact them. If no answer, just wander over. If they are nowhere to be found and you cannot find another registrar either, consultants are more than happy to be contacted if your question is urgent. We all know what it is like to start in your job and will support you!
- Chasing scan dates and times will be a big part of your job. Dragging the registrar to IR after you have put the request on CRRS is the easiest way, otherwise the IR phone number will be on your list. Vascular lab is easier to arrange, again, phone number is on the list
- If any of your patients is due to go to theatre, make sure they have 2x G&S available and that they are valid, ECG, a recent set of bloods (including a clotting) and that they have been marked and consented by the registrar. Check whether their anticoagulation has been stopped
- Putting out bloods for the next day and updating the list at the end of the day will make your job a lot easier (save this under tomorrow's date!)😊

Days in theatre

- Vascular theatre is theatre 20
- Make sure your patients have been consented, marked, their drop form signed (where you confirm the above) and have valid G&S. Patients will either be on one of our surgical ward or ward 10
- Contact your registrar the day before to ask where they want to meet you
- Try and read up on the history of the patient and check the scans. If your registrar is in, you can ask them to go through it with you. Bonus points for studying anatomy!

Days on call for vascular

- The registrars hand over at 8.00 in the ward 22a office. You join them for clerking in SAU and ward reviews. Easy way to get yourself some WBAs!
- This also means you can join them in CEPOD, again, excellent opportunity for DOPS
- Booking a patient for CEPOD goes through the intranet (type in "theatre" in search and it is called Emergency Theatres Booking Form) or use the link below
  Emergency Theatres Booking Form (office.com)
- Call the anaesthetist to let them know about the patient and ask what Hb they want to proceed, this should save a lot of cancellations
- And again, make sure they have been consented, marked and had a drop form signed

On call responsibilities

General Points: When on call over the weekend or bank holidays, surgical FY doctors cross-cover all the acute surgical specialties of urology, vascular and general surgery.

Designated FY1	Roles & Responsibilities
First on FY1 Bleep 2586 – general surgery, vascular and urology admissions on SAU and participate in general surgery ward round (8am to 8:30pm) – long day (8pm – 8:30am) - night	1. General Surgery post take ward rounds (Upper or Lower GI) 2. Assessing Acute General Surgery patients on SAU by day and all specialities – urology, vascular, general surgery at night (clerking, bloods, initial assessment etc.)
Second on FY1 Bleep 2587 – general surgery focus for inpatient care and ward rounds (8am to 8:30pm) Sat & Sun (4:30pm to 8:30pm) Mon to Fri	1. General Surgery ward rounds with priority on the discharge ward round 2. Meet vascular and urology FY1s after their respective rounds to plan ward cover jobs for all surgical inpatients. 3. Provide ward cover to inpatients on surgical wards 4. Support first on FY1 with jobs and acute patient assessment on SAU in particular urology and vascular surgery if other actions complete
Third on FY1 Bleep 5635 – urology focus for ward round, urology for admissions when ward activity done (8am to 4pm) – *weekends*	1. Urology ward rounds with priority on discharge activities 2. Meet vascular and second on FY1s after their respective rounds to plan ward cover jobs for all surgical inpatients. 3. Provide ward cover to inpatients on urology wards 4. Support acute urology admissions and hand over at 4pm to second on call FY1
Discharge FY1 Bleep 5331 – Vascular focus for ward round, vascular for admissions when ward activity done. (8am to 4pm) - *weekends*	1. Meet vascular Reg on ward 22 vascular at 8am for ward round with priority on discharge 2. Meet urology and second on FY1s after their respective rounds to plan ward cover jobs for all surgical inpatients. 3. Provide ward cover for inpatients on vascular wards 4. Support for acute vascular admissions and hand over at 4pm to second on call FY1

Ward cover for General Surgery, Urology and Vascular patients over weekend:

Urology Ward cover:

8am to 4pm- Urology FY1. Bleep 5635

After 4pm: 2nd on FY1. Bleep 2587

Vascular Ward cover:

8am to 4pm- Fourth on FY1 bleep 5331

After 4pm: 2nd on FY1 Bleep 2587

Study leave and annual leave requests

Step 1: Ensure that you give **6-8 weeks advance notice** for any study and annual leave requests.

Step 2: Complete appropriate annual and study leave request forms with details of crossover for on calls. It is your responsibility to ensure your **on calls are covered prospectively** during your annual or study leave periods.

Step 3: Submit these forms to rota co-ordinator Sarah Haynes and Ahmed Hassan- vascular rota reg for approval. This is to ensure there are adequate clinical cover on these dates (minimum staffing levels expected daily are 3 middle grades, 2 FY2 or 1 FY1 and Core trainee/SHO or ACP).

Step 4: After authorisation by rota co-ordinator and rota reg, submit forms to vascular clinical lead (CSL) for final approval (Mr Asif Mahmood).

If there are **extraordinary circumstances for short notice annual leave or study leave** – please **discuss this in person** with Mr Asif Mahmood and/or Mr Devaraj Srinivasamurthy.

Sickness or absence reporting in vascular surgery:

1. If **planned absence**- inform Mr Mahmood- CSL , Sarah Haynes and Ahmed Hassan rota registrar either by email or phone call.
2. **If unplanned absence**- **call** Sarah Haynes rota coordinator as early as possible and call vascular registrar on call to inform.
3. In addition to above- **please email** Sarah Haynes and Mr Asif Mahmood informing them of your absence with reasons for absence and likely date of return to work. Also **inform your educational supervisor** of your absence.
4. For long tern sickness- contact Human Resources and Occupational therapy for advice.

Cheat sheet

Abbreviations

AKA: above knee amputation, ALI: acute limb ischaemia, BKA: below knee amputation, CLI: critical limb ischaemia (CLTI: critical limb threatening ischaemia), DFC: diabetic foot clinic, PAD: peripheral arterial disease

Scans

- CTA: CT angiogram. Easy to get out of hours. Good for anatomy, but will not show flow. These need to be vetted by the on call radiology registrar (#4631) after 5pm. Before 5pm, getting a scan vetted may speed up the process as well. Ask the ward to liaise with CT scan afterwards.
- Angiogram and angioplasty: Done by Interventional Radiology (IR)- contrast injected and xrays taken. Good to see (absence of) flow. Angioplasty is an intervention where they dilate stenosed vessels to improve blood flow. Speak to the IR consultant (in the offices) to get this done.
- Duplex: Vascular lab. USS to assess flow and stenosis. Make sure you remove dressings before sending patients down, otherwise they will not ultrasound whatever area is under there. Phone the vascular lab to get this sorted.
- Vein mapping: Vascular lab will search for usable veins for bypass surgery. Make sure you ask them to mark them as well! Makes life in theatre easier.

Dressings

- Allevyn: padded silicone dressing, works well with pressure sores
- Cosmopore: simple white dressing with sticky borders
- Tegaderm: clear film dressing
- Adaptic/atrauman: non-adherent silicone dressing
- Aquacel: good for packing and for wounds with lot of moisture
- Aquacel silver: see above, but with suspicion of infection, helps to dry it out
- Honey: lifts off necrosis, but needs to stay in situ for 5 days
- Inadine: non-adherent dressing with iodine, helps with dry infected wounds
- Opsite: simple white dressing with clear film borders (often used in theatre)

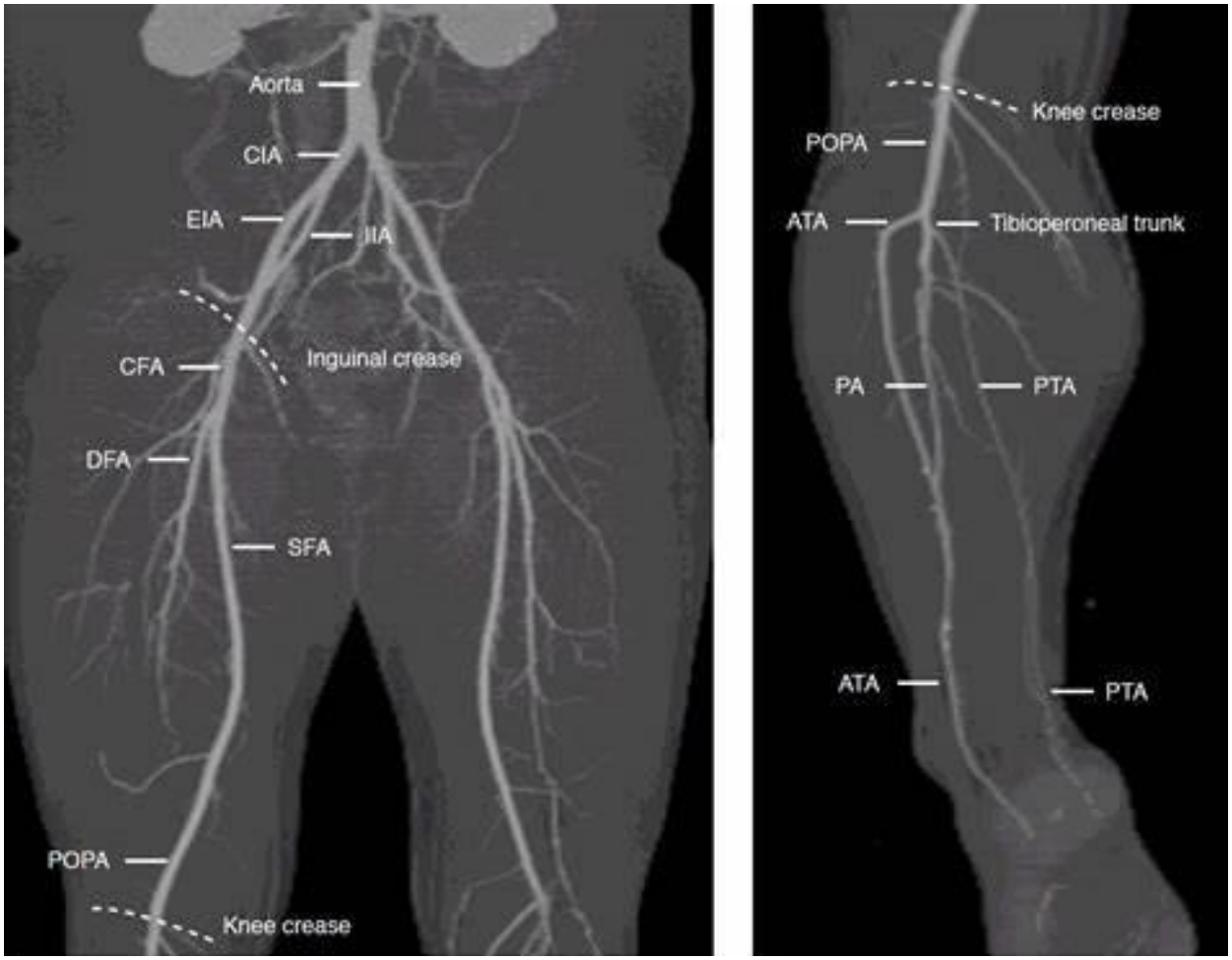
Specialist dressings

- VAC: creates negative pressure environment in wound which should help with healing. Needs review every three days. Not for bleeding wounds. Patients can go home with this.
- VAC veraflo: as above, but with infusion of normal saline, good to lift of slough. Patients need to be in hospital for this. If you turn it off, wait 10 minutes with removing the dressing (so that the saline can be sucked out).
- Maggots: yes, you read that right. The tiny creatures that help clean up a wound. You need the consultant's GMC number to get pharmacy to authorise it.

Anticoagulants

If patients are new to warfarin or a DOAC, they will require a referral to anticoagulation clinic (CRRS->requests/referrals-> inpatient referral->haematology->nurse anticoagulation clinic).

For patients new on DOACs, they will need an area prescribing form (on CRRS-> pharmacy -> Coventry and Warwickshire area prescribing committee-> search for relevant DOAC). There are guidelines on which patient needs what kind of anticoagulation, however this can be consultant specific. If patients are already on warfarin and they are being discharge, they will need to be re-referred to anticoagulation clinic. If they are already on a DOAC and will continue this, they do not require a new referral. Please also council the patient as per checklist.



CIA: Common iliac artery
EIA: External iliac artery
CFA: Common femoral artery
DFA=PFA: Deep femoral artery (AKA profunda)
SFA: Superficial femoral artery
Pop: Popliteal artery
TPT: Tibioperoneal trunk
ATA: Anterior tibial artery
PTA: Posterior artery
PA: Peroneal artery

Management of diabetic foot sepsis

Initial approach in SAU/ED

- As for every other septic patient: A-E approach with adjunct of Sepsis 6 (TAKE blood cultures, lactate, urine output, GIVE antibiotics, oxygen, IV fluid challenge) and escalate to intensive care if necessary.
- Check blood glucose and presence of ketones, diabetic ketoacidosis (DKA) requires immediate treatment (see guidelines on intranet) with involvement of the medical registrar on call.
- Full set of bloods, including group and save (2, if there are no previous samples) with an ECG, MRSA swab and CPE swab if recently been in another hospital. This is all required in case the patient needs surgery.
- If the patient is septic, please inform the on call vascular registrar, even after hours. If they will not review until the morning, instruct the patient to be nil by mouth after 2am and start a sliding scale.
- Insulin-dependent patients need to be prescribed their insulin. If there are no records or pharmacist available, it may be safer to start sliding scale than not prescribing any insulin or the wrong dose.
- Every diabetic drug ending in -flozin (SGLT2 inhibitors), will have to be held during admission (risk of DKA). Prescribe it, then stop it and write: Held during acute illness.
- ALL patients will require a referral to the diabetic team and HbA1c.

Examination

- When examining the patient, look at both feet and also check for signs of peripheral arterial disease.
- Sometimes, the foot ulcer is not the cause of sepsis. Examine the whole patient to check for other sources of infection (such as pneumonia, UTI, etc).
- Assessment of severity of diabetic foot infection:

Mild	Moderate	Severe
- Pus or 2 of: erythema, warmth, pain, induration **- Any cellulitis <2cm around wound**	- Lymphatic streaking, deep tissue infection or abscess - Cellulitis >2cm	- Systemic infection - Limb ischaemia

- Request xray to check for presence of osteomyelitis (cannot be excluded on xray, but if seen, may not need MRI). If bone is visible with the blind eye, you can assume presence of osteomyelitis.

Antibiotic treatment

- As per MicroGuide, which in August 2023 advised the following:

	Standard	Penicillin allergy AND/OR MRSA positive
Mild-moderate	Fluclox 1g QDS PO	Clinda 450mg QDS PO
Severe	Fluxlox 2g QDS IV +Clinda 450mg QDS PO	Vancomycin IV + Clinda 450mg QDS PO

- However, please check the presence of previous wound cultures, sensitivities if applicable and take new wound cultures as well.

Surgical treatment

- Debridement and/or amputation
- Short term predictors of amputation are: moderate-severe infection, previous lower limb amputation, peripheral arterial disease, any walking disability
- “Clean bone”: sample of bone that is deemed not to be infected during surgery. If this is cultured and comes back as non-infectious, antibiotic treatment will only be for 1 week. Otherwise, will need to be treated for 6 weeks, unless surgeon happy that all tissue was macroscopically clean (need further debridement/amputation if infection still not improving)

On the ward

- Chase the diabetic team referral. They do not always prescribe, so check regularly for their instructions.
- Offloading of the foot is usually necessary. Orthotics referral can be done on CRRS. Mr Drew comes every Thursday.
- Chase cultures and discuss with microbiology when they are back. They will ask you how the wound looks, whether the patient had debridement, and if so, about the clean bone sample. Have the drug chart with all prescribed and stopped antibiotics in front of you.
- When the patient goes home, they will need an appointment for diabetic foot clinic the next week.

Ruptured Aneurysm of the Abdominal Aorta (AAA)

Case:

A 77 year old man presented to the emergency department with abdominal pain. He was haemodynamically stable, but due to the nature of his pain, a CTA scan was performed. This showed a 6.8cm aneurysm of the abdominal aorta (AAA), infrarenal, extending into both common iliac arteries. He was transferred to our Coventry Hospital and assessed by the vascular surgeons on call. As there were no signs of rupture, he was discharged and planned for urgent elective endovascular repair within the next week.

Patient, however, represented two days after discharge with hypotension and abdominal pain. Another CTA was performed, which showed contrast extravasation, indicating a rupture of the AAA, with also growth to 7.4 cm. In addition, lesions suspicious for metastases of an unknown cancer were found. The patient was prepared for emergency surgery and underwent an endovascular repair. He recovered well and could leave hospital shortly after.

- ruptured AAA is defined as “acute haemorrhage from the AAA outside the true aortic wall with the presence of retroperitoneal and/or intraperitoneal blood”. If the retroperitoneum seals off the bleed, it is called a contained rupture. If patients do not meet the above criteria, but do have abdominal and/or backpain (without any other cause), pain on palpation of the AAA or emboli, it is called a symptomatic AAA.
- The abdominal aorta has an aneurysm if it is more than 1.5x the normal diameter
- If this is the case in long multiple segments with no discernible aneurysm, it is called arteriomegaly

Risk factors for rupture

Proven	**Suspected**
Large size	Familial inheritance, particularly in females
Hypertension	Eccentric shape
COPD	High expansion rate
Female sex	Absent or minimal thrombus
Smoking	

Presentation

Classic symptoms of rupture are abdominal/back pain, pulsatile abdominal mass and hypotension, but these are only present in 50% of patients.
Less common presentation:

- groin/flank pain, haematuria, groin hernia: All secondary to increased intra-abdominal pressure
- Congestive heart failure with JVD and abdominal bruit if ruptured into inferior vena cava

Management

Treat every man >60 with acute abdominal pain and/or backpain with hypotension/shock or collapse as a ruptured AAA until proven otherwise

- Activate major haemorrhage protocol
- A-E assessment: Make sure you give high flow oxygen, 2 wide bore cannulas, take bloods including G&S and do not forget pain management

- Permissive hypotension is used to prevent worsening of bleeding due to aggressive fluid resuscitation. Fluid resuscitation with blood products and aim for blood pressure high enough to achieve perfusion of end organs, usually assessed with level of consciousness.
- Contact on call vascular registrar, inform theatre, anaesthetist and ITU consultant immediately.
- USS cannot be used to identify a leak and suitability for EVAR cannot be assessed on USS.
- Immediate CTA through the emergency department. Patients who are not stable enough for a CTA, should be sent to theatre straight away.
- EVAR should be considered, as per **IMPROVE** trial, this could even be done under local anaesthetic. However, movement of the patient can lead to difficulties with placing the stent or movement of the stent and not every aneurysm is suitable for this kind of repair.
- Symptomatic AAAs have a higher risk of rupture than asymptomatic AAAs, but emergency repair has a higher risk of complications. Therefore, the recommendation is to schedule for deferred urgent repair, to achieve operating under elective conditions.

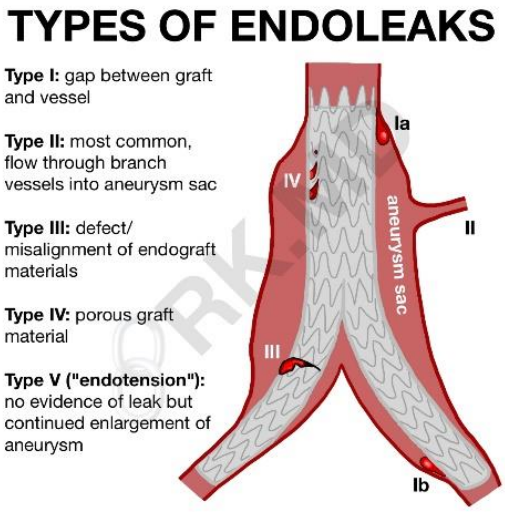
<u>Risk factors of poor outcome</u>

- Systolic blood pressure <90mmHg on admission or intraoperatively
- Pre-operative Cr >200mmol
- Pre-operative Hb <100 or blood loss >7L
- More than 10 units of RBCs
- Urine output <200mL total

<u>Complications aortic surgery</u>

- Haemorrhage
- Death
- Ischaemia (myocardial, colonic, renal, limb, cerebral, spinal)
- Raised intraabdominal pressure and abdominal compartment syndrome (often related to colonic ischaemia)
- Ureteric injury
- Infection (wound, chest, graft)
- Wound related (nerve injury, delayed healing, seroma/lymphocele
- Impotence
- Recurrence (graft dilatation, pseudo-aneurysm, endoleak)

Patients who underwent EVAR, should be followed up as long-term complications due to the graft are more common. Endoleaks can happen for up to 1/3 of the patients and type 2 is currently the most common. Patients have a CT after 30 days to see if there is an endoleak and to look at the fit of the graft, followed by a CT after 6 months and then annually.

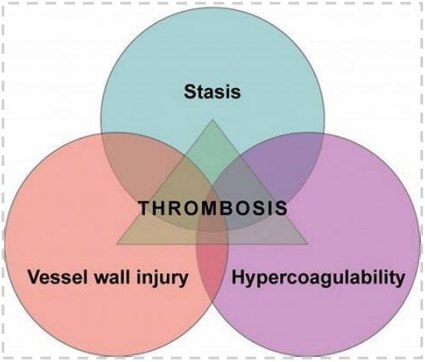


Acute Limb Ischaemia (ALI)

ALI is defined as the sudden decrease in perfusion that threatens the viability of the limb. Complete arterial occlusion will lead to irreversible tissue damage within 6 hours. The primary causes are Embolisation, Thrombosis in situ and Trauma

Common causes

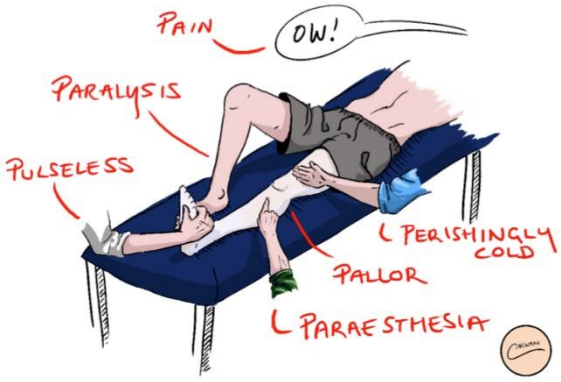
- **Stasis** (embolic) – AF, LV dysfunction/thrombus, proximal arterial aneurysms (aortic, femoral, popliteal)
- **Endothelial damage**– trauma, foreign body, atherosclerosis, PAD, diabetes
- **Hypercoagulability** – malignancy, pregnancy, IBD, nephrotic syndrome, sepsis, thrombophilia disorders



Presentation and Investigations- 6P's

Timely treatment is vital for these patients

- **Urgent escalation** to senior team
- Initial management includes blood tests (FBC, U+E, Clotting screen, CRP, G+S x2)
- **Urgent CT angiogram** to aid diagnosis
- **Urgent anticoagulation** (give loading bolus of heparin)
- Be aware **ACUTE ON CHRONIC ISCHAEMIA** patient will present with **milder symptoms** but are managed the same



Management

Conservative	**Surgical** (immediate presentation)	**Surgical** (late presentation- post 6 weeks)
Medical management with anticoagulation	Endovascular- Arterial thrombolysis	Endovascular- angioplasty
	Open- embolectomy	Open- bypass

Heparin infusion chart can be found on pages 20-21 and the intranet, information regarding thrombolysis on page 22

Critical Limb Ischaemia (CLI)

Definition:

The international consensus on the definition of Critical limb ischaemia (CLI), also known as chronic limb-threatening ischaemia (CLTI) is the following:

any patient with ischemic rest pain, ulcers, or gangrene attributable to objectively proven arterial occlusive disease.

Critical limb ischemia (CLI) occurs after chronic lack of blood supply, setting off a cascade of pathophysiologic events that ultimately lead to rest pain or trophic lesions of the legs, or both. Thus, CLI is considered the "end stage" of peripheral arterial disease (PAD).

CLI is not to be confused with acute Ischemia, but rather a disease process that occurs in a chronic setting of months to years and, if left untreated, ultimately leads to limb loss secondary to lack of adequate blood flow and oxygenation through the distal extremities.

Given that CLI is a severe manifestation of PAD, these patients would be classified in the more severe ends of the Fontaine classification (stage III-IV) or the Rutherford classification (grades 4-6; Table I).

Table I. Classification schemes of peripheral arterial disease

Classification	*Stage*	*Clinical description*
Fontaine	I	Asymptomatic
	IIa	Mild claudication
	IIb	Moderate-to-severe claudication
	III	Rest pain
	IV	Ulceration or gangrene
Rutherford	0	Asymptomatic
	1	Mild claudication
	2	Moderate claudication
	3	Severe claudication
	4	Rest pain
	5	Minor tissue loss
	6	Severe tissue loss or gangrene

Epidemiology and natural history

PAD patients are at an exceptionally high risk for cardiovascular events and most eventually die of a cardiac or cerebrovascular event. Patients with CLI also have a greater risk of sustaining cardiovascular ischemic events than those with PAD alone.

Natural hx of pts with CLI:

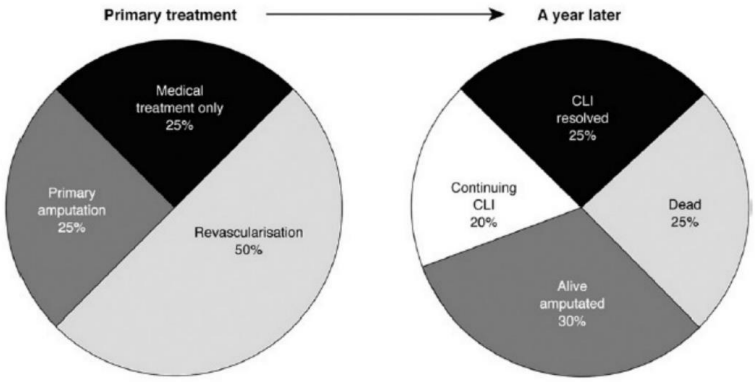


Fig. A5. Fate of the patients presenting with chronic critical leg ischemia. CLI – critical limb ischemia.

- CLI at diagnosis:
  - 50% receive revascularisation
  - 25% are given conservative tx
  - 25% receive primary amputations
- At 1 year:
  - 25% CLI resolved
  - 20% ongoing CLI
  - 30% are ALIVE but amputated
  - 25% dead

Pathophysiology

CLI is usually caused by obstructive atherosclerotic disease; however, CLI can also be caused by atheroembolic or thromboembolic disease, vasculitis, in situ thrombosis related to hypercoagulable states, thromboangiitis obliterans, cystic adventitial disease, popliteal entrapment, or trauma. Regardless of the etiology, the pathophysiology of CLI is a chronic and complex process that affects the macrovascular and microvascular systems, as well as surrounding tissues.

Initially, the body response to ischemia is angiogenesis,or capillary sprouting, as well as arteriogenesis, thereby promoting the enlargement of pre-existing collaterals to aid in the increase of blood flow to the critically ischemic limb. These responses fail to supply the necessary amount of blood flow and oxygen to the limb, causing arterioles in patients with CLI to become maximally vasodilated and insensitive to provasodilatory stimuli. This phenomenon, referred to as vasomotor paralysis, is thought to be the result of chronic exposure to vasorelaxing factors. And that's what's leading to the "Sunset" appearance of the foot.

Diagnosis

Diagnosis of CLI, like any other disease, includes history taking, examination and investigations.

CLI Criteria:

- Severe rest pain requiring opiate analgesics for >4 weeks and/or
    - Ulcer
    - Ankle pressure <40mmHg
    - Toe pressure <30mmHg

History

Usually CLI patients have a history of claudication and/or atherosclerotic risk factors such as smoking, diabetes, ischaemic heart disease , etc.

Also, typical presentation includes rest pain, which is pain at night time that improves by analgesia, or leg dependency, and worsens by leg elevation or coverage of the leg.

Examination

- Tissue loss/gangrene
- Absent pulses
- Sunset appearance of the foot (Buerger's test)

Investigations

- Ankle Brachial Pressure Index <0.3
- Ankle pressure <40mmHg
- Toe pressure <30mmHg

Treatment

The optimal course of action: medical management, revascularization, or amputation.

The prompt diagnosis of CLI cannot be stressed enough, given the high morbidity and mortality associated with the disease process. Although CLI is a clinical diagnosis, it should be confirmed objectively and early in the disease process through ABPI, toe systolic pressures.

Once the diagnosis is confirmed, the goals of treating CLI are:

- Relieve ischaemic pain
- Heal ischaemic ulcers
- Prevent limb loss
- Improve patient function and quality of life and prolong survival

Medical management

Cornerstones of medical management areL

Risk factor control

- Smoking cessation
- Blood pressure control
- Tight diabetic control (in patients with CLI, progression to gangrene occurs in 40% of diabetic patients compared with 9% of nondiabetic patients. Further, limb salvage rates in diabetic patients with CLI have been reported to be lower than nondiabetic patients)

Best Medical Therapy

- Antiplatelet therapy
- Statins

Various reports have demonstrated that cardioprotective medications such as statins, antihypertensive medications, and antiplatelet agents are associated with a decreased cardiovascular event rate in CLI patient.

Surgical management (revascularisation plus wound therapy or primary amputation)

For patients able to tolerate surgical procedures, revascularisation, including bypass surgery, with or without thromboendarterectomy, as well as endovascular techniques offer the best chance for limb salvage.

Essentially, in order to plan for any intervention the patient should first have an arterial duplex done for the affected leg, or ideally CT Angio Aorta and both lower limbs.

The rule in CLI is always that revascularisation should be done first, before any debridement or amputation.

However, in some exceptions, such as when if the infection is extensive, or patient has wet gangrene, a limited debridment/amputation could be done initially to control the infection before revascularisation.

Moreover, Revascularisation should be done promptly, ideally within 5 days of presentation, since complication of CLI can progress very quickly.

Always avoid causing more ulcers or tissue loss for the CLI patient. Avoid tight bandages, always keep an eye on heels, and apply heel protection if any doubt.

Wound inspection is important in CLI patient, since identification of quick worsening of the foot condition would give this patient a priority on the vascular operating list or CEPOD, and could save their limb.

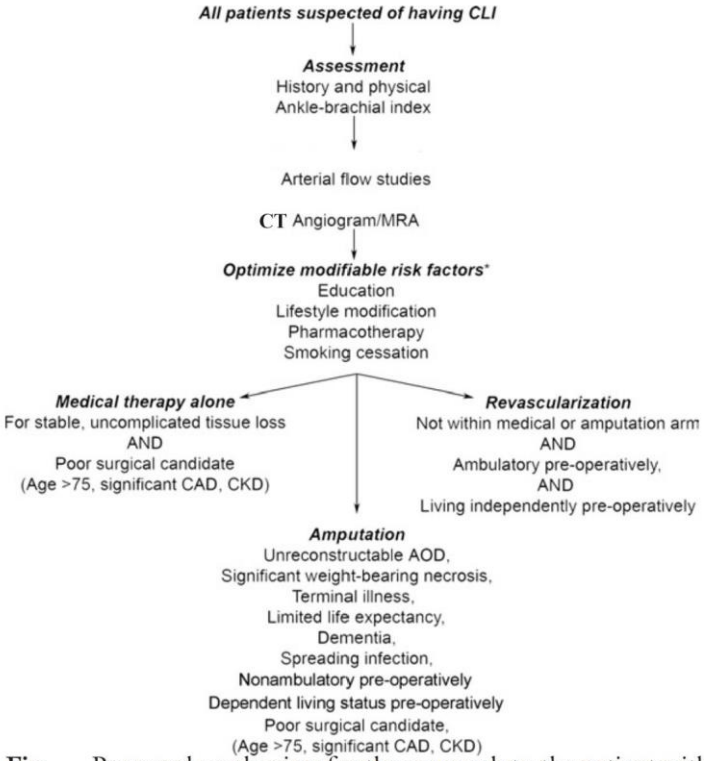
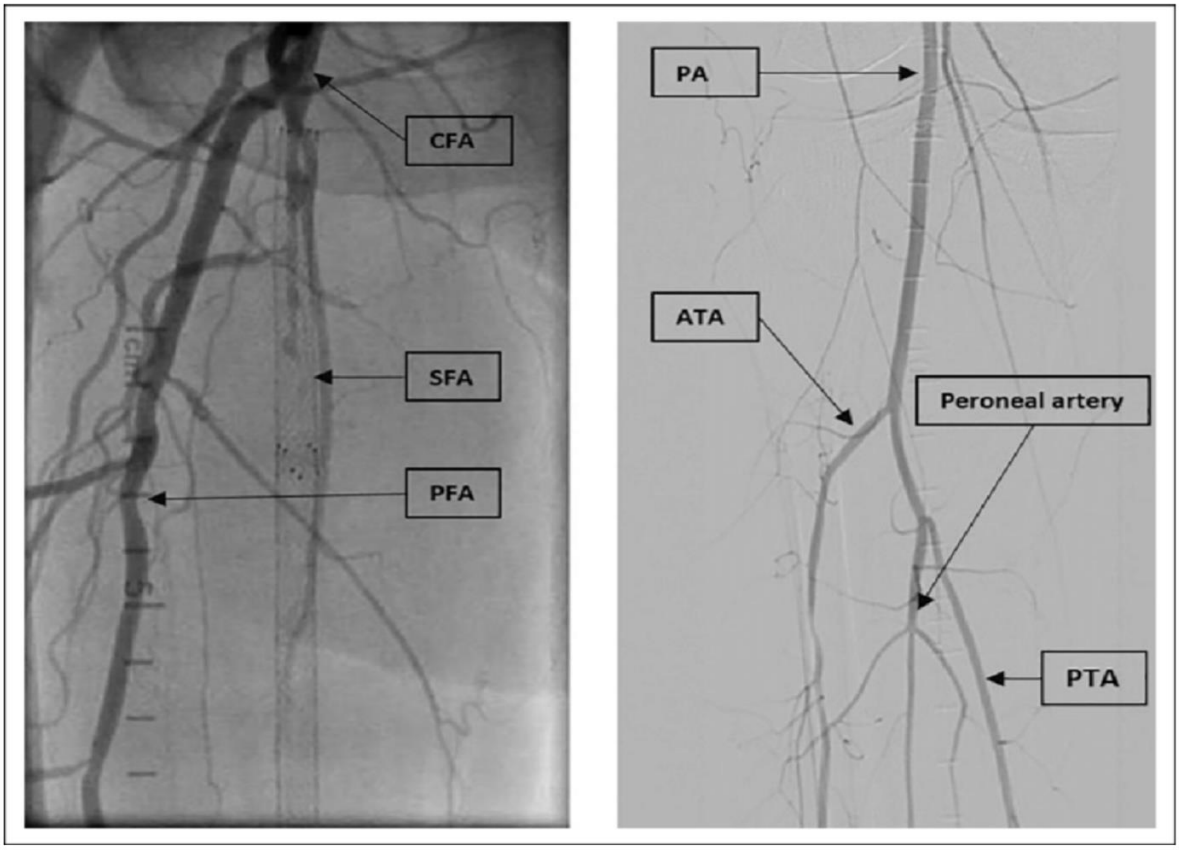


Fig Proposed mechanism for the approach to the patient with critical limb ischemia *(CLI)*. *AOD*, Arterial occlusive disease; *CAD*, coronary artery disease; *CKD*, chronic kidney disease; *MRA*, magnetic resonance angiography; $TcPO_2$, transcutaneous partial pressure of oxygen.

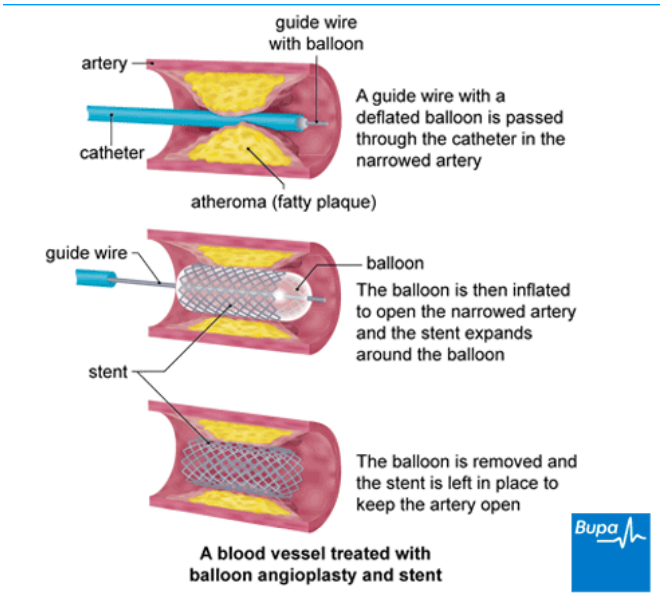
Consent: Angiogram, Angioplasty & Stenting



Peripheral angiogram of the right lower extremity. ATA indicates anterior tibial artery; CFA, common femoral artery; PA, popliteal artery; PFA, profunda femoral artery; PTA, posterior tibial artery.

Overview of Procedure

- An angiogram is performed by percutaneous puncture of the appropriate vessel (typically the common femoral artery) and injection of contrast medium directly in the artery whilst taking X-ray images. This technique is the gold standard for delineating vessel stenosis; however, it is highly invasive compared to ultrasound, CT, or MRI imaging.
- Angioplasty and stenting involve undertaking an angiogram and then expanding the stenosed segment of the vessel. This is achieved through a combination of wires and catheters to navigate to and then through the stenosis, with the subsequent expansion of a balloon or stent to open the vessel.



A blood vessel treated with balloon angioplasty and stent

Intraoperative complications

Complication	**Description**	**Potential ways to reduce risk**
Haemorrhage	This can occur due multiple punctures or insufficient haemostasis.	Apply direct pressure to the puncture site post procedure (the duration of which depends of the size of the sheath used) or via the use of a closure device.
Injury to surrounding structures	The femoral artery lies between the femoral nerve and femoral vein, either of these can be damaged during the puncture if not careful.	Use of ultrasound guidance to identify the relevant neurovascular structures.
Anaesthetic risks	This is a local anaesthetic procedure however some people can react to the local anaesthetic.	

Early postoperative complications

Complication	Description	Potential ways to reduce risk
Pain	Pain during initial insertion or during balloon inflation.	Local anaesthesia will be used in the femoral incisions.
Haemorrhage	There is a small chance of bleeding and bruising in the groin post-surgery.	
Pseudoaneurysm	A pulsating lump may occur in the groin due to ongoing bleeding that can occur around the vessel.	Direct pressure applied to the puncture site as above.
Vessel rupture	There is the potential to rupture the blood vessel, requiring emergency stenting or an open operation.	
Infection	Superficial wound infection is possible; however, this is uncommon in percutaneous approaches.	Perioperative antibiotics will reduce the risk of superficial infections.
Scarring	Although there is minimal visible scar, the site of an angioplasty is typically difficult to dissect at a later time point due to internal scarring.	
Blood clots	DVTs and Pes are a possibility in any operation. The risk is increased in patients with a raised BMI, on the pill, recent flights, previous DVT, pregnancy, smokers, cancer and prolonged bed rest.	The patient will be given anti-embolism stocking and low molecular weight heparin to minimize this risk as deemed appropriate.
Reaction to contrast medium	Skin rashes, vomiting, asthma, hypotension, and cardiac and renal dysfunction can occur as a result of contrast-induced reactions.	
Technical failure	It may not be possible to treat the lesion, requiring either a different interventional approach or surgery.	
Acute limb ischaemia	Disruption of a plaque causing emboli further down the limb or trauma to the vessel causing extravasation of blood into the surrounding tissues can occur. In addition, the equipment can incur a technical fault, whereby a piece of material is left in the vessel and may require surgical removal.	
Stroke, MI, Kidney Failure, Death	Although small, this is always a risk in any major surgery, particularly the injection of contrast medium affecting renal function	

Late postoperative complications

Complication	Description	Potential ways to reduce risk
Reintervention	The stenosis or stent my narrow over time requiring further intervention.	

Heparin infusion chart

Allergy status & nature of allergy:
Signature & stamp: Date:

NHS
University Hospitals Coventry and Warwickshire NHS Trust

FOR SCANNING PURPOSES
AFFIX PATIENT LABEL

WARD:

ADULT: Unfractionated Heparin Infusion Chart

(Not for continuous renal replacement therapy CRRT anticoagulation)

- You must reference IV heparin prescription on the main drug chart, state "see heparin infusion chart".
- See anticoagulation guideline for full information regarding prescribing IV heparin.
- Always refer to haematology before prescribing IV heparin.
- Send blood for baseline aPTT, FBC, coagulation screen, U&Es, LFTs before starting IV heparin.
- **ALWAYS** use 1000units/mL concentration of heparin sodium for IV infusion.

Initial bolus

- Dose = 80 units/kg (rounded to the nearest 100 units) based on actual body weight.
- Use 1000 units/mL concentration only; give via slow intravenous injection over 3 – 5 minutes.

Patient weight (kg)	kg	**INITIAL BOLUS Dose (units)**	units	Pharmacy use
Prescriber, signature, bleep number, date:		Given/Checked	Date/Time Given	

Continuous infusion

- Prepare using readymade heparin sodium for IV infusion (20,000 units in 20mL or 25,000 units in 25mL).
- Contact Pharmacy for advice if the readymade product is not available.
- Discard any unused infusion after 24 hours.

Prescribe the initial infusion rate according to the table below (based on actual body weight – circle below):

Weight (kg)	Infusion rate	Weight (kg)	Infusion rate
40-49	*800 units/hour (0.8mL/hour)*	90-99	*1600 units/hour (1.6mL/hour)*
50-59	*900 units/hour (0.9mL/hour)*	100-109	*1800 units/hour (1.8mL/hour)*
60-69	*1100 units/hour (1.1mL/hour)*	110-119	*2000 units/hour (2mL/hour)*
70-79	*1200 units/hour (1.2mL/hour)*	120-129	*2200 units/hour (2.2mL/hour)*
80-89	*1400 units/hour (1.4mL/hour)*	130-139	*2400 units/hour (2.4mL/hour)*

Dose bands are UHCW agreed and based on 18 units per kg per hour.

Commence infusion at	**mL per hour**	Prescriber, signature, bleep number, date:	Pharmacy use
Given by/Checked by	Date:	Time:	

Dosage adjustment

The first aPTT must be checked at SIX HOURS after starting the infusion.

The dose must be adjusted as shown below by changing the infusion rate. **DO NOT CHANGE THE CONCENTRATION.**

aPTT ratio	**≤ 1.5**	**1.6 – 1.9**	**2.0 – 2.5**	**2.6 – 3.0**	**3.1 – 3.9**	**≥ 4.0**
Give IV bolus	80 units/kg	40 units/kg	**No change required – therapeutic range**	NO	NO	NO
Stop IV infusion?	NO	NO		NO	Stop for one hour	As per Haematology advice
Change continuous infusion dose?	↑ by 4 units per kg per hour	↑ by 2 units per kg per hour		↓ by 2 units per kg per hour	↓ by 3 unit per kg per hour	As per Haematology advice
Next aPTT ratio due	6 hours	6 hours	24 hours	6 hours	6 hours	As per Haematology advice

Approved at UHCW Medicines Management Committee February 2020; to be reviewed February 2022.
UHGEN268

Continuous Heparin Infusion

(Record every rate change and syringe change in this section)

Date & Time prescribed	aPTT ratio result	Syringe concentration	CURRENT infusion rate (mL/hour)	NEW infusion rate (mL/hour)	Prescriber, signature, bleep number	Next aPTT ratio due (date/time)	mL – volume remaining	Date & time given	Given by Checked by	Pharmacy use
		Heparin Sodium 1000units/mL								
		Heparin Sodium 1000units/mL								
		Heparin Sodium 1000units/mL								
		Heparin Sodium 1000units/mL								
		Heparin Sodium 1000units/mL								
		Heparin Sodium 1000units/mL								
		Heparin Sodium 1000units/mL								
		Heparin Sodium 1000units/mL								

Additional bolus doses

(Only if aPTT ratio ≤2.0)

Date & Time prescribed	aPTT ratio result	Bolus dose (units)	Prescriber, signature, bleep number	Date & time given	Given by Checked by		Pharmacy use

Thrombolysis: Catheter directed thrombolysis with alteplase (TPA)

- Decision for this procedure will be made by IR with a consultant Vascular surgeon
- Patient has to be in ECU as process very nurse-intensive
- Alteplase will have to be prescribed by yourselves and given with one of the (trained) nurses. They may well ask you to prepare it
- All other forms of oral and parenteral anticoagulants must be withheld during treatment. Dual antiplatelet therapy must be suspended, but single agent use may still be continued at the discretion of the Vascular Team.
- Contra-indications: Irreversible ischaemia, major trauma/surgery/CPR in last two weeks, CVA in last two months, primary or secondary brain tumour unless cerebral perfusion required, active bleeding with potential for major haemorrhage, pregnancy. Hepatic impairment is a relative contra-indication
- There will be three ports in the intravascular catheter:
    - Drug port: 5mg TPA bolus (to be prescribed as STAT dose, followed by Alteplase IV Infusion 15 mg in 500ml Sodium Chloride 0.9% [0.03 mg/ml] at 33 ml/h over 15 hours (in the regular medicine section).
        - Dissolve 20mg Alteplase in 20mL water for injections (leading to concentration of 1mg/mL). Use 5mL for the bolus, to be given over three minutes. The rest can be injected in 500mL 0.9% NaCl and used for the infusion. If thrombolysis continued after this bag, repeat the above but **discard** the 5mL for the bolus and **solely** give the 15mg in 500mL. Be aware, this replacement has to be done within 10 minutes
    - Heparin port: Heparin Infusion 5000 unit [100 unit/ml] at 5 ml/h (500 unit/h) over 10 hours (in the regular medicine section). No need to monitor APTT
        - Withdraw 1mL of heparin (5000 units/mL) and add 49mL of 0.9% NaCl in a 50mL syringe
    - If EKOS (USS enhanced technique) is being used, coolant port: Sodium Chloride 0.9% 1000 ml Infusion at 55 ml/h over 18 hours (in the regular medicine section)
        - Flush with 5mL (500 units) Heparin (100 unit/mL) and follow this by the infusion
    - All of the infusions will have to be given simultaneously and continuously
    - Stop infusions if the IR or Vascular consultant asks you to or: Systolic BP >180mm Hg or diastolic BP >110mm Hg, signs of stroke, excessive bleeding only if accompanied by signs of shock (bleeding at puncture site is a routine finding). All of these require you to phone your registrar and review immediately
    - Follow up will be by angiography after 24 hours. If Alteplase is no longer required, intravascular catheter will be removed, but introducer sheath will remain in situ with Heparin running for another 4 hours. After this: stop Heparin and leave sheath in for another two hours. Registrar will then have to remove this and apply direct pressure for 20 minutes (sterile gauze). If bleeding persists: call for help

Contrast Induced Acute Kidney Injury (CI-AKI) in vascular surgery patients

Introduction

The use of intravascular iodinated contrast agents has continued to increase over recent years. It is recognised that there are potential risks associated with the intravascular administration of iodinated contrast agents. It is therefore important to have better understanding of the indications for use of iodinated contrast media as well as the potential side effects and their management.Acute kidney injury following receipt of iodinated contrast (CI-AKI) has previously been referred to as contrast induced nephropathy (CIN) defined as a rise in serum creatinine by 25% or 44µmol/L from the baseline value. It is uncommon in the general population, with an incidence of 1-2%, and occurs within 72 hours of receiving contrast media, usually recovering over the following five days. It is important to exclude other causes of AKI as small rises in serum creatinine have been demonstrated to occur in 8-35% of patients admitted to hospital without exposure to contrast media. Its incidence increases significantly in patients with risk factors and is associated with increased mortality.

Definition and staging of CI-AKI

Contrast induced - acute kidney injury is defined when one of the following criteria is met :-

- Serum creatinine rises by ≥ 26µmol/L within 48 hours or
- Serum creatinine rises ≥ 1.5 fold from the baseline value, which is known or presumed to have occurred within one week or
- Urine output is < 0.5ml/kg/hr for >6 consecutive hours

Stage	**Serum Creatinin (Cr) criteria**	**Urine output criteria**
1	increase ≥ 26 µmol/L within 48hrs or increase ≥1.5- to 1.9 X baseline Cr	<0.5 mL/kg/hr for > 6 consecutive hours
2	increase ≥ 2 to 2.9 X baseline Cr	<0.5 mL/kg/ hr for > 12 hours
3	increase ≥ 3 X baseline Cr or increase ≥ 354 µmol/L or commenced on renal replacement therapy (RRT) irrespective of stage	<0.3 mL/kg/ hr for > 24 hours or anuria for 12 hrs

- As the stage of AKI increases, so does risk of mortality

Risk assessment for CI-AKI

The risk of CI-AKI is low in patients with normal kidney function, estimated at 1-2% even in patients with diabetes. However, prior exposure to iodinated contrast media has been identified as the third most common etiological factor for AKI in hospital after renal hypoperfusion and nephrotoxic medication. The risk of CI-AKI has been reported to be as high as 25% in patients with a combination of chronic kidney disease (CKD) and diabetes, cardiac failure, older age and exposure to nephrotoxic drugs. The CI-AKI Consensus Working Panel has recommended that the risk of CI-AKI becomes clinically important with an eGFR < 60 mls/min/1.73m2 . Acutely ill patients with sepsis and/or hypotension are particularly vulnerable to injury following iodinated contrast exposure. There is a general consensus that the risk of CI-AKI is higher after arterial compared to venous administration of iodinated contrast media although this has not been proven convincingly.

Risk factors for patients developing CI-AKI include

- nephrotoxic medication
    - aminoglycosides
    - NSAIDs
    - ACE Inhibitors
- chronic kidney disease (CKD) eGFR < 60 mls/min/1.73m2
- older age (> 75 years old)
- cardiac failure
- hypovolaemia
- sepsis
- volume (dose) of contrast
- Metformin is not nephrotoxic but is exclusively excreted via the kidneys. Therefore patients on metformin who develop AKI following contrast are at risk of developing lactic acidosis due to the accumulation of the drug. The Royal College of Radiologists recommends that there is no need to stop metformin after receiving iodinated contrast if the serum creatinine is within the normal range and/or eGFR > 60 ml/min/1.73m2. If serum creatinine is above the normal reference range or eGFR is < 60 ml/min/1.73m2.

Recommendations for risk assessment

- It's recommended prior to any imaging using iodinated contrast media baseline kidney function and presence of other risk factors for CI-AKI should be identified. The exception to this is when the benefit of very early imaging outweighs the risk of delaying the procedure.
- The estimated glomerular filtration rate (eGFR) should only be used to assess kidney function in stable outpatients .
- On the other hand serum creatinine is used to assess kidney function in acutely ill patients or patients with acute kidney injury. All such patients should be considered as at increased risk of CI-AKI .
- Patients identified to be at high risk of CI-AKI may be discussed with a renal physician to assess whether the potential benefit from the iodinated contrast study outweighs the increased risk of CI- AKI.

5 Point strategy to prevent CI-AKI

1- Unenhanced scanning or alternative imaging techniques should be considered in patients with risk factors for developing CI-AKI.
2- Intravenous volume expansion with 0.9% sodium chloride or isotonic sodium bicarbonate in patients identified as at high risk of CI-AKI .
3- The lowest possible volume of a low or iso-osmolar iodinated contrast medium should be used in patients with risk factors for developing CI-AKI .
4- There is no convincing benefit for prescribing oral or intravenous N-acetylcysteine or any other pharmacological agents to prevent CI-AKI.
5- Further, patients should be counselled about the risk of developing CI-AKI before angiography procedures .

Summary

- Contrast induced - AKI results from a combination of afferent arteriolar vasoconstriction and direct toxicity of the contrast media to the tubular epithelial cells. Prevention is important as there is no specific treatment and involves identification of patients at increased risk of CI-AKI. It should also be considered whether alternative imaging could be utilized such as ultrasound or whether carbon dioxide can be used to reduce the amount of iodinated contrast agent required .
- Magnetic resonance angiography (MRA) may be considered as an alternative but the use of gadolinium (Gd) in MRA is associated with the risk of developing Nephrogenic Systemic

Fibrosis (NSF). Nephrogenic Systemic Fibrosis is a severe fibrosis of the skin resulting in extensive limitation in mobility.

- Intravenous 0.9% sodium chloride at a rate of 1 mL/kg/hour for 12 hours pre- and post-procedure has been shown to be more effective than 0.45% sodium chloride in reducing CI-AKI. More recently it has been demonstrated that intravenous isotonic sodium bicarbonate significantly reduces the risk of CI-AKI
- Most recently a large randomized trial found that acetylcysteine does not reduce the risk of contrast- induced acute kidney injury or other clinically relevant outcomes in at-risk patients undergoing coronary and peripheral vascular angiography. Currently there is no compelling evidence for the routine use of N- acetylcysteine to prevent CI-AKI.

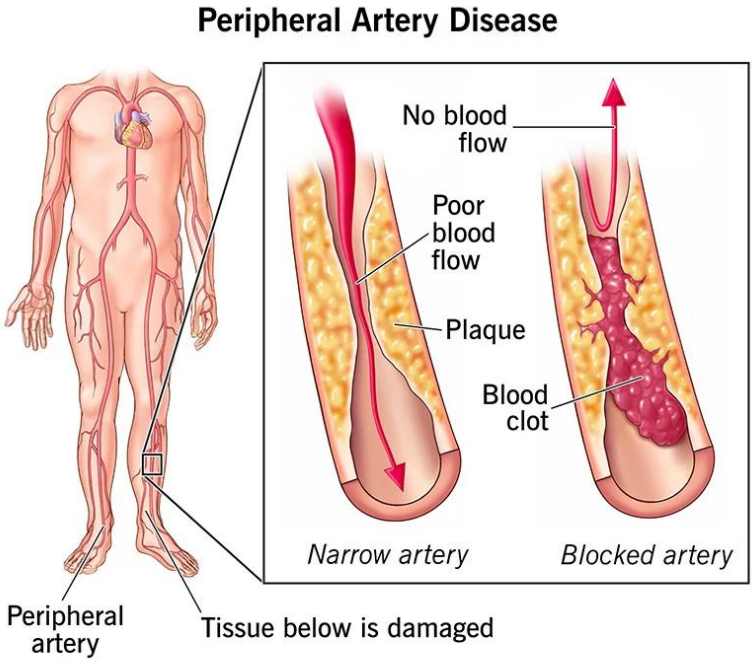
Best Medical Therapy in Peripheral Arterial Disease (PAD)

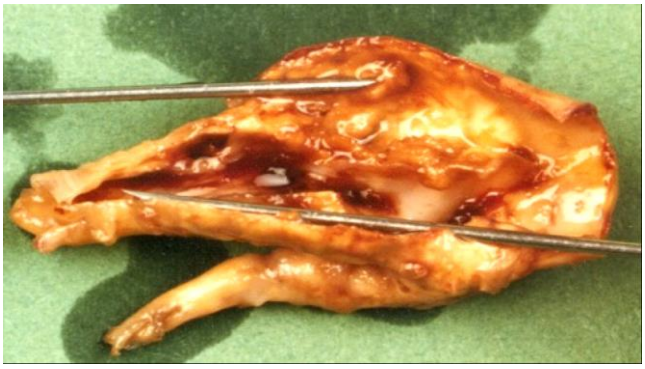
Overview

- **PAD** refers to diseases of blood vessels (arteries) outside the heart and brain. It's often a narrowing of vessels that carry blood to the legs, arms, stomach or kidneys.
- **PAD** is common, affecting 1 in 5 people over 60 in the UK, and carries both the risk of lower limb loss and the increased risk of death from heart attack and stroke.

There are two types of these circulation disorders:

1. **Functional**
   Functional peripheral vascular diseases don't have an organic cause. They don't involve defects in blood vessels' structure. They're usually **short-term** effects of **"spasms"** that may come and go. **Raynaud's disease** is an example. It can be triggered by cold temperatures, emotional stress, working with vibrating machinery or smoking.
2. **Organic**
   Organic peripheral vascular diseases are caused by **structural changes** in the blood vessels, such as inflammation and tissue damage. **Peripheral artery disease** is an example. It's caused by fatty build-ups in arteries that block normal blood flow.





Plaque removed from an occluded artery.

The first line of management for people with PAD is cardiovascular risk factor modifications

Risk Factor	**Description**	**Guideline reference**
Smoking cessation	Reduces the risk of cardiovascular events. Behavioural support in combination with medications such as a combination of **short- and long-acting nicotine replacement therapy** or nicotine-containing e-cigarettes are the most effective smoking cessation strategies.	https://www.nice.org.uk/guidance/ng209
Antiplatelet agents	Patients should receive secondary prevention with **clopidogrel 75mg OD**, unless contraindicated or intolerant. **Second line** is **Aspirin 75mg OD**. Patients on anticoagulation do not benefit from an additional antiplatelet agent. **The COMPASS trial** has more recently shown benefit from **rivaroxaban 2.5mg BD plus Aspirin** in PAD.	https://cks.nice.org.uk/antiplatelet-treatment
Lipid modification	Patients should be offered **secondary prevention** with **high intensity statin treatment** e.g., **Atorvastatin 80mg OD**, if tolerated. Prior to statin initiation, causes of secondary hyperlipidaemia should be identified and treated, including excessive alcohol intake, uncontrolled diabetes, hypothyroidism, liver disease and nephrotic syndrome. Patients should be counselled about the small risk of side effects, including muscle pains. *The **most serious adverse effects of statins** are **myopathy** and **rhabdomyolysis**. The estimated incidence are **5** and **2 cases per 100,000-person years** respectively.* NICE recommend **baseline blood tests** including a **non-fasting lipid profile** (total cholesterol, HDL-C, non-HDL-C, TG and CK, LFTS, renal function, **HbA1c** and **TSH** if dyslipidaemia is present). **LFTs** should be checked at 3 and 12 months, and **HbA1c** at 3 months if at risk of diabetes, with annual check of lipids and review for side effects of statins thereafter.	https://cks.nice.org.uk/topics/lipidmodification-cvdprevention/
Weight management	if **Body Mass Index is >25**, consider referral for dietary advice and provide a goal for weight loss.	https://cks.nice.org.uk/topics/obesity/diagnosis/identificationclassification/
Diabetes	Care should be coordinated with the diabetes team. Aim for **HbA1c of <48mmol** (higher target if elderly). Manage type 1 and type 2 diabetes according to national guidelines.	https://cks.nice.org.uk/diabetes-type-1 https://cks.nice.org.uk/diabetes-type-2
Hypertension	**Blood pressure >140/90 mmHg** in the **outpatient clinic**, or an **average ambulatory blood pressure** recording of **>135/85 mmHg** should prompt further assessment and treatment. In patients aged **>80 years**, target **blood pressure should be <150/90 mmHg**. If blood pressure is elevated, recommend smoking cessation and reduction of alcohol and caffeine intake. Exercise programmes, relaxation therapy and reduced salt intake are effective lifestyle approaches to lowering	https://cks.nice.org.uk/hypertension-not-diabetic

	blood pressure. Consider causes of secondary hypertension and treat as appropriate. Severe or resistant hypertension should prompt referral to specialist hypertension services. **First choice medication** in **patients aged <55 years** who ***are not black African or African- Caribbean*** is an **angiotensin-converting enzyme inhibitor (ACEi)** or **angiotensin II receptor blocker (ARB)** if tolerated. **First line for patients >55 years**, or ***black African or African-Caribbean patients*** is a **calcium channel blocker (dihydropyridine type - e.g., amlodipine)**. If intolerant or in need of second- or third-line agents, it would be appropriate to consider a **thiazide diuretic** such as **indapamide**.	
Nutrition	Diet should broadly be in line with **healthy eating recommendations**, i.e., five portions of fruit and vegetables each day, meals based on starchy foods such as pasta, bread, rice or potatoes, moderate amounts of dairy products and protein-rich foods. Intake of foods high in fat, sugar and salt should be reduced.	https://www.nhs.uk/live-well/eat-well/
Regular activity and exercise	Patients should be advised to break up long periods of sitting with light activity, to aim for at least **150 minutes of moderate aerobic activity every week** and to perform **strength exercises on 2 or more days a week** that work all the major muscles (legs, hips, back, abdomen, chest, shoulders, and arms).	

Iloprost infusion

Indication

- Treatment of severe chronic ischaemia of lower limbs in patients at risk of amputation, in whom surgical revascularisation or angioplasty has failed or is not indicated

- Treatment of severe Raynaud's phenomena in patients with progressive trophic disorders

Contraindications

- Allergy to iloprost
- Pregnancy or breastfeeding
- Other conditions with bleeding risk (think: trauma, intracranial bleeding, peptic ulcer)
- Severe IHD or ACS within last 6 months
- CCF NYHA II-IV, pulmonary oedema
- Severe arrhythmias
- CVA or TIA within last 3 months
- Congenital heart disease with impaired cardiac function

Be more careful with dose increases and monitoring with patients on haemodialysis and monitor blood pressure in patients with hypotension prior to infusion.
Anticoagulants can be continued whilst on iloprost.

Prescription and administration

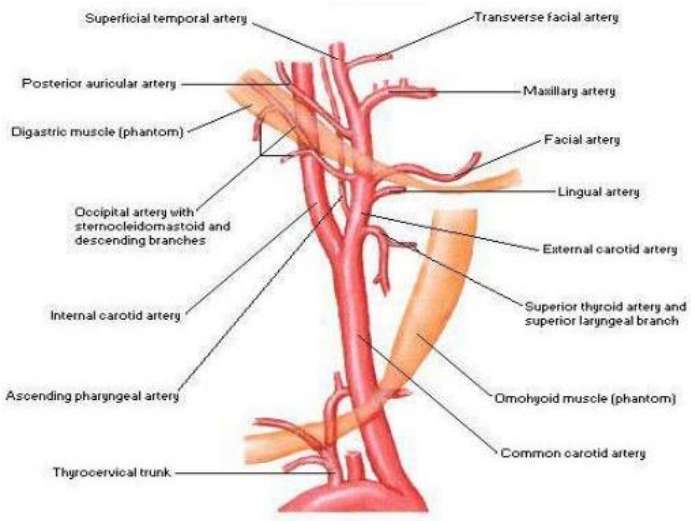
- Use a Syringe Driver with a 50ml Injection Syringe Dilute Iloprost 50 micrograms with 25 ml of 0.9% Sodium Chloride solution or 5% Glucose solution to obtain a final concentration = 2 micrograms/ml
- Dose and Rate: According to Weight (see table below)
- Commence Infusion at Starting dose 0.5 nanograms/kg/min
- Increase rate gradually by 0.5 nanograms/kg/min every 30 minutes until a maximum dose of 2.0 nanograms/kg/min is reached.
- If not tolerated step down to the previous tolerated dose
- Duration: Infusion over 6 hours daily. The treatment course can be continued – with the dose based on the optimal tolerated dose reached in the first 2 to 3 days of treatment.
- Duration of treatment can vary based on indication: - Severe Chronic Lower Limbs Ischemia: up to 4 weeks. Severe Raynaud's phenomenon in patients with progressive trophic disorders: 5 days

	Dose (ng/kg/min)			
	Starting dose: 0.5	1st increase: 1.0	2nd increase: 1.5	3rd increase: 2.0
Weight (KG)	**Infusion rate (mL/hr)**			
40	0.60	1.20	1.80	2.40
50	0.75	1.50	2.25	3.00
60	0.90	1.80	2.70	3.60
70	1.05	2.10	3.15	4.20
80	1.20	2.40	3.60	4.80
90	1.35	2.70	4.05	5.40
100	1.50	3.00	4.50	6.00
110	1.65	3.30	4.95	6.60

Carotid artery disease and carotid endarterectomy

Introduction

Stroke is the third commonest cause of death and the principal cause of neurological disability. It is defined as an acute loss of focal cerebral function with symptoms exceeding 24 hours, with no apparent cause other than that of a vascular origin. **Transient ischaemic attack (TIA)** has the same definition but lasts <24 hours.

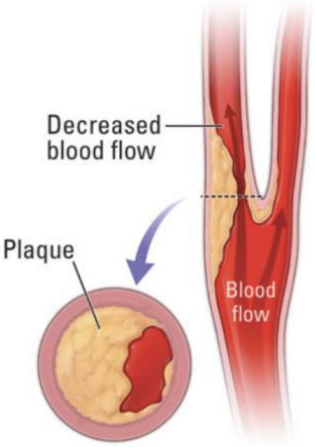


Aetiology and risk factors

- About 80% of strokes are ischaemic, the remainder haemorrhagic (intracerebral/subarachnoid). Approximately 80% of ischaemic strokes affect the carotid territory. Risk factors include increasing age, smoking, hypertension, ischaemic heart disease, cardioemboli, previous TIA, diabetes, peripheral vascular disease, high plasma fibrinogen and hypercholesterolaemia.
- The principal causes of ischaemic, carotid territory stroke are: thromboembolism of the internal carotid artery (ICA) or middle cerebral artery (MCA) (50%); small-vessel intracranial disease (25%); cardiac embolism (15%); haematological disorders (5%); and non-atheromatous disease (5%) such as Fibromuscular dysplasia, Artritis, Aneurysm, Carotid body tumour & Dissection

Large vessel thromboembolism

The commonest cause is thromboembolism of the ICA and/or MCA. Stenoses develop at the ICA origin because of a region of low shear stress, flow stasis and flow separation that predisposes to atherosclerotic plaque formation. Should the plaque undergo acute disruption (rupture, ulceration, intraplaque haemorrhage), the core of subendothelial collagen is exposed, triggering thrombus formation and embolism.



Classical carotid and vertebrobasilar features

Carotid territory

- Hemimotor/hemisensory signs
- Monocular visual loss (amaurosis fugax)
- Higher cortical dysfunction (dysphasia, visuospatial neglect, etc.

Vertebrobasilar

- Bilateral blindness
- Problems with gait and stance
- Hemi- or bilateral motor/sensory signs
- Dysarthria
- Homonymous hemianopia
- Diplopia, vertigo and nystagmus

Investigation of Carotid Artery Disease

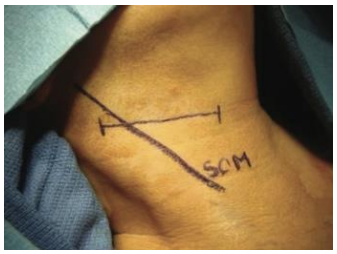
1. **Duplex ultrasound**
   The degree of stenosis is usually first evaluated using duplex ultrasound. In most UK centres, the majority of CEA procedures are planned on the basis of duplex alone. Duplex can only isolate the cervical portion of the extracranial carotid artery and is therefore relatively unreliable at excluding disease elsewhere
2. **Computed tomography angiography (CTA)**
3. **MRA**
4. **Catheter angiography :**In the modern era of high-quality non-invasive imaging there is no role for routine catheter angiography. Arch angiography is employed in some CAS centres as a means of assessing anatomic suitability for CAS and the status of the aortic arch/ arch origins of the great vessels.

Management of cerebrovascular disease

Best medical therapy

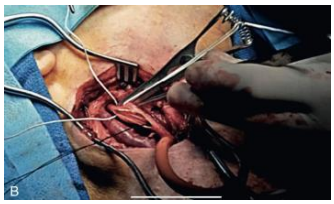
All patients benefit from optimisation of risk factors, antiplatelet/statin therapy and exclusion of important comorbidity. Everyone should undergo an electrocardiogram (ECG) to exclude occult cardiac pathology. Baseline blood tests will exclude diabetes, arteritis, polycythaemia, anaemia, thrombocytosis, sickle-cell disease and hyperlipidaemia.

Carotid Endarterectomy-CEA



- CEA is **not indicated** in symptomatic patients with a 0–50% stenosis. CEA confers benefit in the recently symptomatic 50–99% stenosis to prevent further ischaemic events
- Recommended time from symptom to surgery is 14 days
- Patient need to be on Dual antiplatelet preoperatively
- Goals – remove plaque, repair artery, avoid complications

Operative complications

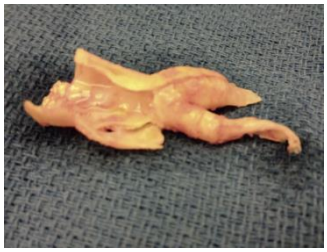


Immediate:

- Neuro: periop stroke, hyperperfusion
- Local : infection, Cranial n. (hypoglossal, marginal) – most reversible
- Systemic: HD instability, MI.

Late:

- Restenosis
- Patch aneurysm



Guidelines on assessing patients for Urgent Carotid Endarterectomy (CEA)

Referral Criteria:

1. Patients with TIA or stroke as a result of symptomatic ICA stenosis of 50-99% confirmed on **carotid Doppler by Vascular Lab at UHCW.**
2. Patients seen and triaged by TIA clinic or Stroke team with confirmation of diagnosis.
3. Patients physiologically and anatomically suitable for CEA (No history of neck operations, radiotherapy to neck or tracheostomy, No tandem distal ICA lesions, clear cervical distal normal ICA visible on Doppler)
4. **Goals:**

To offer CEA to suitable patients with symptomatic ICA stenosis of 50-99% within 2 weeks of onset of first symptom

5. **Process:**

- Urgent Referral received by on call vascular reg (Bleep 2738) or On call Vascular consultant by TIA clinic or stroke team (preferably by stroke consultant or Registrar)
- **Inpatient referrals** : Urgent review by on call vascular team within 24 hrs
- For **out patient referrals**:
    - Contact patient and arrange urgent review in SAU within 48 hrs of referral
    - If Carotid Doppler done outside UHCW- arrange same day carotid Doppler at Vascular lab at UHCW to confirm ICA stenosis grade.
    - History: confirm symptoms suggestive of carotid territory TIA/Stroke- Unilateral arm or leg weakness, speech disturbance/dysphasia (usually Left hemispheric), facial droop, amaurosis fugax and date first symptom occurred.
    - **Any recurrent symptoms suggestive of crescendo TIA?** If yes, arrange immediate admission for IV Heparin and DAPT, TCD and stroke team review. Monitoring microemboli with TCD and response to BMT daily. Consider IV Tirofiban if ongoing microemboli despite BMT.
    - Investigations arranged on SAU: FBC, UE, LFT, clotting screen and 2x group and save and ECG. MRSA screen, COVID swab
    - Discuss with on call vascular consultant regarding suitability for urgent CEA
    - Documentation- leave a note on CRRS under 'Letters' section

6. **Further investigations required in following situations**:
    - Medical comorbidities- AF,MI/CAD- may require anaesthetic review and ECHO pre-op
    - Diagnosis uncertain on clinical grounds: May require DWI MRI Brain and Stroke and vascular MDT discussion
    - Carotid Doppler not conclusive about degree of stenosis
    - Carotid Doppler unable to visualise normal distal cervical ICA
    - Carotid Doppler suggestive of distal ICA disease or tandem lesions/occlusion
    - Carotid Doppler suggestive of significant proximal CCA origin disease
    - Patients with hostile neck- consider referral for carotid artery stenting (CAS)

Above patients are likely to require Urgent CT Angiogram of aortic arch, neck and circle of Willis.

7. Referrals from GEH and Warwick Hospital

- Referrals to be sent to stroke team/TIA clinic at UHCW for urgent triage same day
- Carotid Doppler scan repeated at UHCW vascular lab within 48 hrs to confirm
- Referral from vascular lab to on call Vascular Reg (Bleep 2738)when symptomatic ICA stenosis 50-99%
- Vascular Reg to see patient on SAU same day for above urgent assessment to confirm suitability for CEA and arrange initial blood tests

8. Booking patients to theatre for Urgent CEA

- All patients should be discussed with vascular consultant and agreed for CEA before booking into theatre list
- Email or call vascular secretaries to book. Confirm patient aware and agreeable for CEA, pre-op bloods and G&S and MRSA screen done.
- Ensure all pre-op investigations mentioned above are done
- Leave a note on CRRS under 'letters section' regarding assessment on SAU

Post-operative management following CEA

In Theatre

- Patient extubated in theatre
- Neurological status checked and confirmed to be unchanged compared to pre-op by surgical team
- Blood pressure targets for monitoring set by consultant anaesthetist
- Handover to theatre recovery staff by consultant anaesthetist and theatre staff
- All required medications and IV fluids written up in drug chart
- Post-op TCD monitoring by vascular team arranged

In Theatre recovery/PACU/OIR

- Most patients to stay in theatre recovery for **6 hours post-op unless** specified by anaesthetist
- Monitor BP and neurological status hourly and maintain BP in target range set up consultant anaesthetist and surgeon
- Metaraminol and Labetolol are commonly used and prescribed by consultant theatre anaesthetist
- Monitor BP in A-Line If systolic BP < 105 mmHg and > 180mmHG - before 5PM contact theatre anaesthetist and after 5PM contact duty anaesthetist
- Review by vascular team prior to discharge to ECU or ward

In Theatre recovery, Watch for signs of

- Bleeding- minor bleeding from surgical wound is common and requires change of dressing to pressure dressing by nursing staff
- Significant /large hematoma or expanding hematoma in neck, drain > 75 mls of blood - before 5PM call theatre vascular team and after 5PM call on call vascular Reg on 2738 URGENTLY
- In rare event If patient develops **Airway compromise** (shortness of breath, stridor, difficulty swallowing, drooling of saliva)- **fast bleep duty anaesthetist and on call Vascular Reg on bleep 2738.**
- If patient develops new neurological signs such as persistent severe headache, arm or leg weakness, speech disturbance, visual symptoms/loss-Inform theatre surgical team when possible before 5PM and Contact Vascular Reg on call URGENTLY on bleep 2738

Transfer to ECU after 6 hours

Criteria for transfer:

- If BP within target range without support
- No Significant neck hematoma or drain < 75 mls with no active bleeding
- No new neurological signs
- Transfer agreed with vascular and anaesthetics team- either with review or verbally

On ECU:

- Patient can Eat and drink
- Clexane 40mg SC at 22:00 hrs unless specified otherwise
- Moniter BP and neurological status every 2nd hourly
- Cuff- If BP < 105mmHG or > 180mmHG contact duty anaesthetist and Vascular Reg on call on bleep 2738 for advice
- For bleeding complications- expanding hematoma, drain > 175 mls since transfer, airway compromise- escalate to duty anaesthetist and on call Vascular Reg 2738

Care Pathway for Diagnosis and Management of Patients with Venous Thoracic Outlet syndrome

Diagnosis: *Suspect Venous Thoracic Outlet Compression in following patients*

1. *Age 18-50 yrs*
2. History of Sudden Onset Arm Swelling **usually associated with effort** (upper limb exercises, weights/bench press, yoga, contact sports, gymnastics etc.)
3. Typical presentation is "**Effort Induced Upper Limb Venous Thrombosis"**
4. Clinically present with **Sudden Swollen, Blue Upper Limb** with palpable upper limb pulses
5. **No other precipitating factors such as OCP's, chemo/radiotherapy, cancer, recent central** venous lines in upper limb, recent surgery or road traffic accident affecting upper limb

Investigations

1. **URGENT Venous Duplex/Doppler Scan** of affected upper limb **within 24 hours**.
   This can be done at Local Hospital when feasible or referred to UHCW Vascular Lab via on call Registrar for urgent scan
2. **Plain Chest X-ray** to include first rib and lower C spine to rule out cervical rib or abnormal first rib.
   CT Venogram or MRI of Thoracic Outlet if *of limited value* in acute presentation and is NOT part of standard work up

Management Plan

If patient with history and examination suggestive of Venous TOS (see above):

1. Start Anticoagulation with Treatment Dose of Low Molecular Weight Heparin (LMWH) (e.g. Enoxaparin 1mg/Kg twice daily SC)
2. Arrange Venous Doppler Ultrasound within 24 hours to confirm Upper Limb DVT (Thrombosis of Subclavian and Axillary Veins)
3. Refer patients urgently or same day to on call Vascular Registrar at UHCW Bleep 4823 via switchboard.

Vascular Management

1. Admit patient under on call Vascular Team.
2. **Criteria for admission**: Diagnosis confirmed by Vascular Registrar on call and Venous Doppler scan confirms presence of Upper Limb DVT.
   NO central venous lines, active cancer, recent major bleeding, no chemo/radiotherapy
3. Continue Therapeutic dose of LMWH
4. Consider **Catheter Directed Thrombolysis(CDT)** if history of onset of upper limb swelling **within 2-3 weeks**
5. Discuss these patients with on call Vascular Consultant to confirm patient suitable for CDT
6. Vascular on call team to inform Mr Srinivasamurthy – Consultant Vascular Surgeon through email urgently.
7. If patients suitable for CDT- discuss with Interventional Radiologist
8. After successful CDT, these patients will also **require urgent First Rib Resection and Thoracic Outlet Decompression ideally in same admission** when feasible or within 2-4 weeks of CDT
9. After successful CDT- continue Anticoagulation with Treatment dose of LMWH or Treatment dose of NOAC
10. Post-op Anticoagulation with NOAC for 6 weeks (or Warfarin if NOAC contraindicated)
11. Repeat Venous Duplex Scan 4-6 weeks post-op.
12. Follow-up in Mr Srinivasamurthy in OPD 6-8 weeks post-op