Core Modality 2: Peripheral Arterial Duplex

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Protocol Document

Introduction and scope:

Duplex scanning can identify site, severity and extent of lower limb arterial disease. It is valuable in identifying lesions which can be treated with percutaneous transluminal angioplasty. The scan is also used for planning surgical intervention.

Referral criteria:

Referrals can be accepted from vascular surgeons and the diabetic foot team for patients with clinically appropriate symptoms of lower limb arterial disease. Referrals can also be accepted from other departments if a poor ABPI result indicates arterial disease and it is likely that the patient will need arterial intervention.

Responsibilities:

Test staff: scientific or technical staff trained in vascular duplex scanning.

Equipment:

Duplex scanner with 2, 3.5, 5 and 7 MHz transducer.

Method:

<u>Examination protocol:</u>

Complete ABPI assessment as per VAS-PRO-002.

The examination can cover the arterial supply to the lower limb from the abdominal aorta to the pedal arteries, or just a specific region of interest.
Perform the examination in a longitudinal plane with colour Doppler, identifying any regions of disease.

If the patient is a new referral the scan should commence at the level of the abdominal aorta (in order to check for aneurysmal disease). If the patient has had previous imaging of the abdominal vessels the examination can begin by taking a waveform in the common femoral artery, if this is triphasic there is no need to scan the iliac arteries.

Take representative waveforms and images in the common femoral, superficial femoral and the profunda femoral arteries. Move distally down the leg to the distal popliteal artery, noting any disease. If a significant lesion has been found proximally, take ankle waveforms in the PTA, peroneal and ATA arteries. If no significant lesion has been found the crural vessels (TPT, PTA, ATA and peroneal artery) may be scanned in full if clinically indicated.

If a haemodynamically significant (≥2x PSV increase) lesion is suspected, examine the area in detail with spectral Doppler. Where possible use an angle ≤60°.

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If a haemodynamically significant (≥2x PSV increase) lesion is suspected, examine the area in detail with spectral Doppler. Where possible use an angle ≤60°.

Record velocities proximal to and at the site of the stenosis and document the anatomical location of any haemodynamically significant lesion identified.

If an occlusion is identified record the site of the occlusion and level of reconstitution of the vessel. Document presence of collateral vessels where seen.

<u>Pedal arteries:</u>
The pedal arteries can be examined for the presence of disease, also the consultant may request the pedal arteries be examined for planning surgical intervention (distal bypass). In instances where the patient presents with a foot ulcer and normal flow to the crural vessels the pedal arteries should also be assessed. A high frequency linear array transducer should be used. The scan should include the distal ATA, the DPA, distal PTA, and medial and lateral plantar arteries. Take representative images of each vessel and waveform, and also measure the diameter of the vessel where patent.

<u>Popliteal entrapment:</u>
Where popliteal artery entrapment is suspected the popliteal artery should be interrogated as follows: With the patient in a normal standing position examine the popliteal artery and assess waveforms. The vessel should then be examined in active plantar flexion, this is achieved with the patient pushing up onto ‘tip-toe’ position. Assess the popliteal artery

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It is important to note that compression of the popliteal artery is also seen in normal subjects, therefore the presence of this is not confirmation of popliteal entrapment however the absence of compression could exclude the condition.

Reporting
Any areas of stenosis should be reported along with their velocity increase and anatomical location. The site of any occlusive disease and level of reconstitution of the vessel. Representative waveforms from all arteries scanned should also be reported.
The site and diameter of any aneurysmal disease should be reported.
Any anatomical variations should also be described.

Where pedal arteries have been assessed the patency, waveform and diameter of the vessels should be reported.

When the patient has been examined for popliteal artery entrapment the appearance of the vessel and waveforms when relaxed and during plantar flexion should be commented on.

All reports are completed on the CRIS system. Where it aids clarity and understanding, the written report should be augmented with a diagram completed on the lower limb arterial template (VAS-FRM-

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Where it aids clarity and understanding, the written report should be augmented with a diagram completed on the lower limb arterial template (VAS-FRM-7); diagrams are scanned into the PACS system, and this noted in the written report.

In the event of acute onset, critical limb ischaemia and/or aneurysmal disease the vascular team should be informed immediately.

Table 1. Criteria for lower limb artery duplex assessment.

PSV ratio	% Stenosis
< 2	< 50
2	50
4	75
Numerous lesions without any alteration in PSV ratio or with PSV ratio < 2	Diffuse disease
Absence of flow	Occluded

Images:

- Abdominal aorta with maximum diameter (if part of scan).
- Representative waveforms (if patent) from
  - CFA with velocity.
  - SFA (proximal, mid and distal) with velocity.
  - PFA origin
  - Popliteal (proximal, mid and distal) with velocity.
  - PTA, ATA and Peroneal arteries, with velocity.
  - Pedal vessels (where required), with waveform and diameter.
- Areas of stenosis and/or occlusion described in report.

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  - PTA, ATA and Peroneal arteries, with velocity.
  - Pedal vessels (where required), with waveform and diameter.
- Areas of stenosis and/or occlusion described in report.
- Areas of aneurysm with diameter measurement.

Inspection criteria:
Complete CRIS database patient tested / DNA / re-booked.

References:
SVT guidelines:
https://www.svtgbi.org.uk/media/resources/Arterial_PPG_-_new_format.pdf

NICE Guideline CG147 (December 2020) Lower limb peripheral arterial disease: diagnosis and management

Vascular Ultrasound, How, Why and When; 3rd Edition, A Thrush and T Hartshorne pg 117-140

End of document

Evidence: 25x Peripheral arterial duplex reports

Right:
CFA, Prof.A origin and SFA are patent with triphasic pulsatile flow.

At proximal Pop.A, flow becomes low velocity and resistive.
From mid Pop.A, there is occlusive acute looking thrombus; some small, short channels of cannulised flow identified proximally. Occlusive thrombus appears to extend throughout the mid-distal Pop.A and below the level of the TPT.

The ATA is origin appears occluded, with arterial flow fed into the proximal ATA via anterior collaterals. The remaining ATA is patent to ankle level. The DPA at ankle level is patent, albeit, with low velocity mono/biphasic pulsatile flow, PSV 0.3 m/s.

Unable to ascertain flow at proximal PTA, ?occluded.
Flow appears to reconstitute at prox-mid calf; the remaining PTA is patent to ankle level, with low velocity pulsatile monophasic flow, PSV 0.3 m/s.

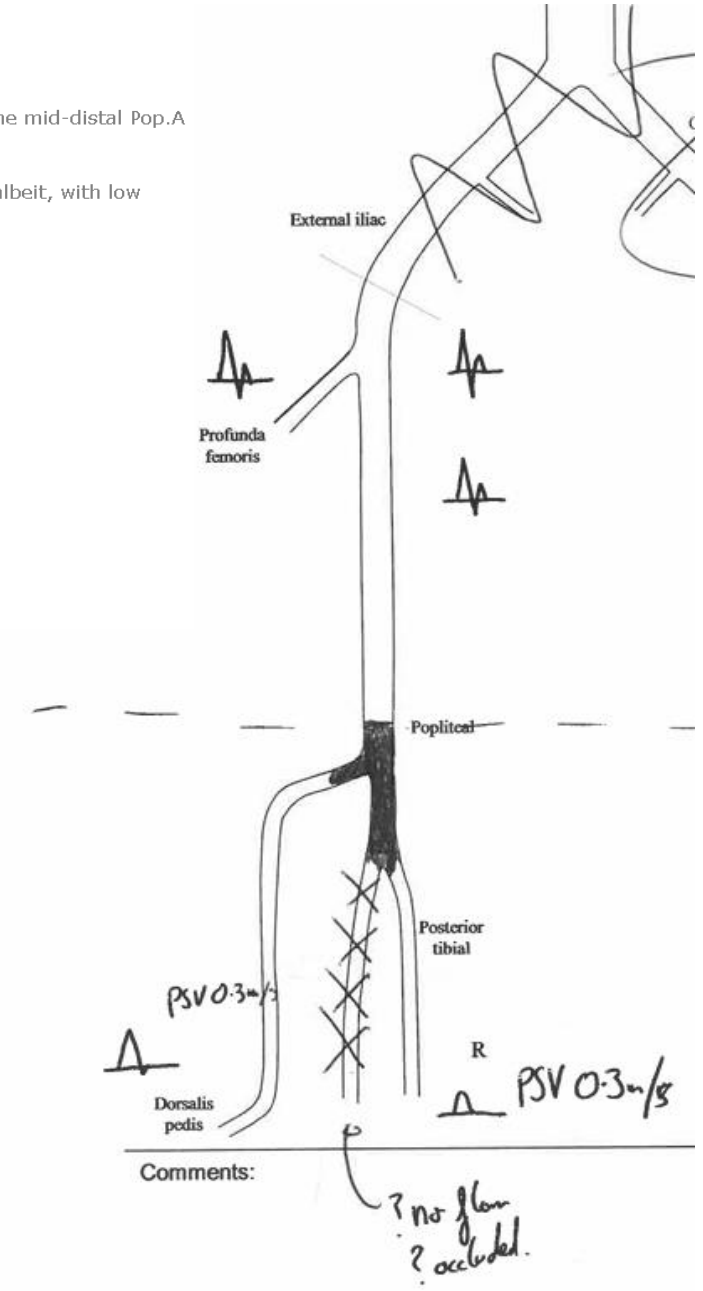
Unable to ascertain flow in the Pero.A, ?occluded, ?calcified.

Conclusion:
Pop.A/TPT occluded, ?acute thrombus embolus

Low velocity mono/biphasic pulsatile flow sampled at ankle level
Pero.A ?occluded.

Unable to obtains accurate ABPI - dense crural vessel calcification artificially raising ankle pressures.

Discussed with ward nurse over the phone.



Left:
Consistent strong triphasic pulsatile flow sampled in the CIA, EIA, CFA, Pop.A and distal PTA and ATA/DPA.

With pt standing, the Pop.A remained patent during neutral, dorsiflexion and plantar flexion positions - only significant change observed was a 2-3x velocity increase (PSV 1.1 m/s) in the Pop.A with plantar flexion, however, positioning was exaggerated and Pop.A remained widely patent - highly unlikely to be significant enough to be attributed with symptoms.

Conclusion:
Consistent triphasic pulsatile flow sampled throughout.
No obvious Pop.A entrapment observed with dorsiflexion and plantar flexion.

Left:
As per dedicated plantar artery examination.

Distal PTA is patent with triphasic pulsatile flow, PSV 0.4-0.6 m/s.

Where seen the proximal lateral plantar artery is patent with triphasic pulsatile flow, PSV 0.6 m/s.

The medial plantar artery is patent with triphasic pulsatile flow; flow was followed in continuity to mid foot, typical PSV 0.2-0.5 m/s.

Left:
Abdominal aorta is patent and of normal calibre/borderline ectatic (not aneurysmal), ~2.6 cm, AP.

Poor views of the CIA and EIA - however, where seen they are patent with mono/triphasic pulsatile flow - no obvious significant stenosis identified in this region.

The CFA is patent, albeit, with a >4x focal velocity increase (PSV 4.2 m/s); suggestive of a >75% stenosis.
Raised turbulent velocities continue into the Prof.A origin - low-resistant pulsatile monophasic flow sampled proximally.

There is a second >4x focal velocity increase (PSV 3.5 m/s) sampled at SFA origin; suggestive of a >75% stenosis.
Shortly below this point the SFA becomes occluded; low-velocity severely dampened monophasic flow reconstitutes at mid-distal SFA (at the level of the adductor canal).

Borderline ~2x focal velocity increase also sampled at distal SFA (PSV 0.4 m/s); suggestive of a ~50% stenosis.

Pop.A/TPT are patent with low-velocity severely dampened monophasic flow.

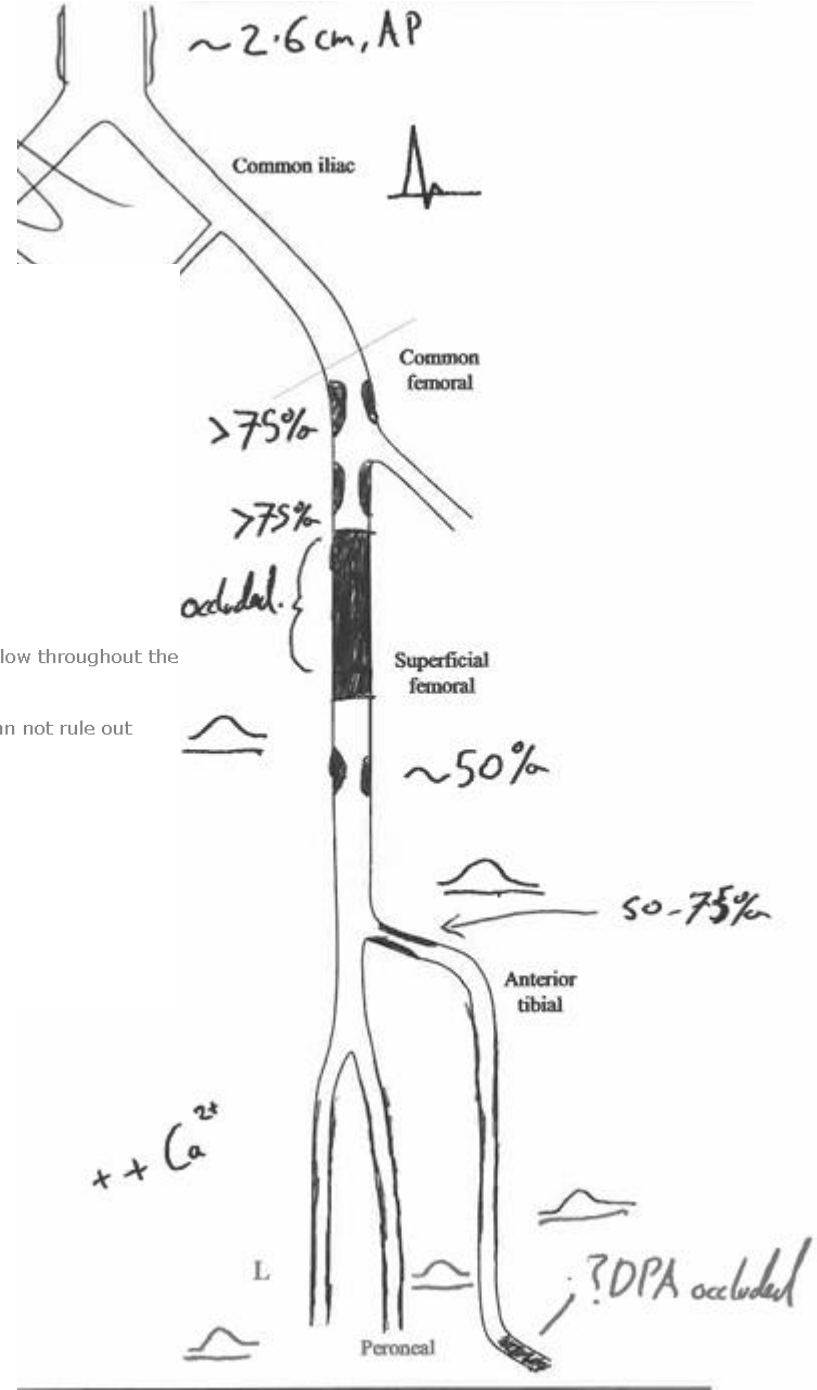
At ATA origin, there appears to be a 50-75% stenosis.
The remaining ATA is patent with severely dampened monophasic flow - just above ankle level, the ATA appears to feed collaterals, with no filling into the DPA - unable to ascertain flow throughout the DPA, ?DPA occluded.

The Pero.A and PTA appear patent throughout with severely dampened monophasic flow, with no obvious evidence of significant stenosis/occlusion (poor views due to calcification, can not rule out small focal disease), distal PSV 0.3-0.4 m/s and 0.1-0.2 m/s, respectively.

Conclusion:
Separate CFA and SFA origin >75% stenoses
Extensive SFA occlusion
~50% distal SFA stenosis

ATA origin 50-75% stenosis
?Occluded DPA

Severely dampened monophasic flow sampled at ankle level (Pero.A and PTA).



Right:
Dense calcified disease seen throughout.

Abdominal aorta is patent and of normal calibre, ~1.7 cm, AP.

CIA, IIA, and EIA are patent with bi/triphasic pulsatile flow.

The CFA is patent, albeit, with a >4x focal velocity increase (PSV 6-7 m/s); suggestive of a >75% stenosis - visually appears very tight (?>90%).

Turbulent high velocity triphasic/monophasic pulsatile flow continues into the Prof.A and SFA, respectively.

The SFA is patent throughout with pulsatile monophasic flow - at distal SFA, there is a ~2x focal velocity increase (PSV 1.4 m/s); suggestive of a ~50% stenosis.

There appears to be a short segmental occlusion of the mid Pop.A, with severely dampened monophasic flow reconstituting at distal Pop.A.

The distal Pero.A, PTA and ATA/DPA are patent with low velocity dampened/severely dampened monophasic flow, PSV 0.3 m/s, 0.1 m/s and 0.1 m/s, respectively.

Conclusion:
>75% stenosis at distal CFA.
~50% stenosis at distal SFA
Short occlusion at mid Pop.A

Low velocity, dampened monophasic flow sampled at ankle level.

Left:
Dense calcified disease seen throughout.

Abdominal aorta is patent and of normal calibre, ~1.7 cm, AP.

CIA, IIA, EIA, CFA and Prof.A are patent with bi/triphasic pulsatile flow.

The SFA is patent with triphasic pulsatile flow sampled proximally, transitioning to monophasic pulsatile distally.
At the level of the adductor canal, there is a 2-3x focal velocity increase (PSV 2.2 m/s); suggestive of 50-75% stenosis.

The Pop.A/TPT is patent with pulsatile monophasic flow.
At proximal Pop.A there is a 2-3x focal velocity increase (2.4 m/s); suggestive of a 50-75% stenosis.

The distal Pero.A, PTA and ATA/DPA are patent with pulsatile monophasic flow, PSV 0.3 m/s, 0.6 m/s and 0.7 m/s, respectively.

Conclusion:
Separate 50-75% stenoses identified at mid-distal SFA, and proximal Pop.A.

Pulsatile monophasic flow sampled at ankle level.

Right:
Dense calcification seen throughout.

Abdominal aorta is patent and of normal calibre ~1.3 cm, AP.
The CIA, IIA, EIA, CFA and proximal prof.A are patent with mono/triphasic pulsatile flow.
The SFA origin is patent with bidirectional pulsatile flow - then immediately below this the SFA and Pop.A are occluded.

The crural vessels are densely calcified with very poor views.

Unable to ascertain colour flow throughout most of the ATA; only able to obtain a low velocity severely dampened monophasic spectral trace in the distal ATA, ~5 cm above the medial malleolus - no colour filling or spectral trace could be achieved above this point, ?prox-to-mid ATA occlusion. DPA appears patent with severely dampened monophasic flow; flow continues to mid/distal foot, PSV ~0.1 m/s.

The distal PTA is patent with low velocity, dampened, monophasic, hyperaemic flow. Below medial malleolus the anatomy becomes unclear due to dense calcification, however, it appears the lateral plantar artery is patent with dampened monophasic retrograde flow, PSV ~0.2 m/s. Unable to ascertain flow in the medial plantar artery, ?calcified, ?occluded.

Right:
Diffuse calcified disease seen throughout.

CFA is patent with triphasic pulsatile flow, albeit, there is a borderline ~2x focal velocity increase (PSV 1.9 m/s); suggestive of a ~50% stenosis.

Prof.A origin is patent with biphasic pulsatile flow.

The SFA is patent throughout with biphasic pulsatile flow.

There appears to be short segmental occlusion at mid Pop.A with a cluster of surrounding collaterals; pulsatile monophasic flow reconstitutes at distal Pop.A.

The distal Pero.A, PTA and ATA/DPA are patent with pulsatile monophasic flow, PSV 0.3 m/s, 0.4 m/s and 0.4 m/s, respectively.

Conclusion:
Borderline ~50% CFA stenosis.
?short mid-Pop.A occlusion.

Pulsatile monophasic flow sampled at ankle level - ABPI 0.5-0.6

Left:
CFA and Prof.A origin are patent with triphasic pulsatile flow.

SFA and Pop.A/TPT are patent with monophasic pulsatile flow; mild-moderate calcified plaque seen throughout.
At very distal SFA there is a ~2x focal velocity increase, PSV 2.1 m/s; suggestive of a ~50% stenosis.
At mid Pop.A there is a 2-3x focal velocity increase, PSV 1.6 m/s; suggestive of a 50-75% stenosis.
At distal Pop.A there is a >4x focal velocity increase, PSV 3-4 m/s; suggestive of >75% stenosis.

The distal Pero.A, PTA and ATA/DPA are patent with pulsatile monophasic hyperaemic flow, PSV 0.4 m/s, 0.3 m/s and 0.8 m/s, respectively.

Conclusion:
Distal SFA and Pop.A are stenotic with at least two separate 50-75% and one >75% stenoses identified.
Pulsatile monophasic hyperaemic flow sampled at ankle level - ABPI 0.7-0.8.

Right:
Abdominal aorta, right CIA, EIA, and CFA are patent throughout with triphasic pulsatile flow.
AA 1.6 cm AP. Generalised right EIA disease however no velocity increase > 2 fold.

Slightly elevated velocities in the iliac segment attributable to presence of cross over graft. Proximal X-over graft anastomosis is patent with no evidence of stenosis.

Prominent PFA is patent proximally with monophasic pulsatile flow.

Right SFA occluded from origin. There are short recanalised segments throughout the proximal and mid-thigh SFA. Monophasic pulsatile flow reforms in mid/distal SFA just above the adductor canal level. PopA patent with monophasic pulsatile flow.

At ankle, all three tibial vessels are patent with monophasic dampened/pulsatile flow.

Distal PSVs:
ATA/DPA 0.3 m/s
PTA 0.4 m/s
PeroA 0.2 m/s.

Right pre-/post exercise ABPI: 0.7-0.8 / 0.3-0.4

Conclusion:
Long right SFA occlusion.

Left:
Pop.A remains widely patent with triphasic pulsatile flow during neutral, dorsiflexion and plantar flexion positions.
Moderate velocity increases seen with forced positions:
Neutral - PSV 0.3 m/s
Dorsiflexion - 0.6 m/s
Plantar flexion - 0.9 m/s

Conclusion:
Pop.A remains patent with forced positions (dorsiflexion and plantar flexion), albeit, with raised velocities suggestive of haemodynamically significant compression. Unable to comment weather this is significant enough to be contributing towards pt symptoms.

Right:
CFA and Prof.A are patent with triphasic pulsatile flow - Prof.A is well-developed with high velocity flow, ?compensatory flow.

The SFA is occluded from origin, with a short segment of in-stent patency at mid-thigh, before becoming occluded again.
Monophasic pulsatile flow reconstitutes at distal SFA - dampened monophasic flow continues into the Pop.A/TPT.

Low velocity, monophasic severely damped flow sampled in the distal Pero.A, PTA and ATA/DPA, PSV 0.2 m/s, 0.2 m/s and 0.1 m/s, respectively.

Conclusion:
Long SFA occlusion.
Monophasic severely damped flow sampled at ankle level - ABPI <0.3

Sent pt back to diabetic foot clinic - discussed results with vascular reg over the phone.

AA patent and uniform in calibre, 2.2cm diameter AP.

On the left:
The CIA and EIA are patent with triphasic pulsatile flow.
There is a ~50% stenosis at the CIA origin.
The CFA and proximal PFA are patent with triphasic pulsatile flow and no stenosis.
The SFA is occluded throughout.
The Popliteal artery is patent with calcified, non stenotic disease. The distal PopA flow is monophasic pulsatile flow.

In thr calf:
ATA:
Patent at origin.
? occluded in the proximal-mid calf.
Patent from mid-lower calf with monophasic pulsatile flow.
DPA patent with monophasic pulsatile flow, PSV 0.17m/s.

PeA and PTA are calcified however appear patent throughout with monophasic pulsatile flow.
Unable to fully exclude stenosis.
Distal PSV in the PTA = 0.48m/s and PeA = 0.19m/s.

Please see diagram on PACs.

Right:
Abdominal aorta is patent and of normal calibre, ~1.6 cm, AP.

The CIA appears chronically occluded.
The IIA is patent with retrograde pulsatile monophasic flow.
The EIA, CFA, Prof.A, SFA and Pop.A/TPT are patent with monophasic pulsatile flow - only mild generalised plaque detected (<50%). Large collaterals seen filling into the distal EIA and CFA.

The distal Pero.A, PTA and ATA/DPA are patent with pulsatile monophasic flow, PSV 0.3 m/s, 0.5 m/s and 0.4 m/s, respectively.

Conclusion:
Long chronic CIA occlusion; retrograde flow sampled in the IIA.
Monophasic pulsatile flow sampled at ankle level - ABPI 0.5-0.6

Left:
Abdominal aorta is patent and of normal calibre, ~1.6 cm, AP.

CIA is patent, albeit, with focal raised velocities sampled at origin (PSV 3-4 m/s), suggestive of a 50-75% stenosis - appears very tight.

The IIA, EIA, CFA, Prof.A, SFA and Pop.A/TPT are patent with bi/triphasic pulsatile flow - only mild generalised plaque detected (<50%).

The distal Pero.A, PTA and ATA/DPA are patent with pulsatile monophasic flow, PSV 0.4 m/s, 0.7 m/s and 0.9 m/s, respectively.

Conclusion:
50-75% stenosis at CIA origin.
Monophasic pulsatile flow sampled at ankle level - ABPI ~0.8

Abdominal aorta + Visceral arteries:
Abdominal aorta is a patent and of normal calibre, 1.5 cm, AP.
Mild calcified plaque/mural thrombus seen throughout (~10-19%)

Incidental note is made of increased velocities at the IMA origin suggestive of haemodynamically significant (>50%) stenosis however the SMA and coeliac arteries are seen widely patent with no evidence of stenosis.

The aorta-SMA angle was noted to be relatively shallow/acute, maximum angle recorded ~14 degrees, which may be indicative of SMA syndrome - pt complained of postprandial pains/nausea after eating, however clinical assessment/correlation is advised.

Left lower limb:
The CIA, IIA, EIA, CFA, and prof.A origin are patent with pulsatile monophasic flow - no evidence of EIA in-stent stenosis.

At SFA origin, there is a heavy burden of predominately hypoechoic (?atheroma/thrombus) with a small channel of flow; no significant velocity change could be ascertained at this point, however, appears to be causing at least a >75% stenosis.

Occlusive (?acute-on-chronic) plaque/atheroma extends throughout the proximal (~10 cm below origin) to mid-distal section of the SFA; damped monophasic flow reconstitutes at distally thigh. The remaining distal SFA appears stenotic, with at least one >75% stenosis.

Damped monophasic flow sampled in the Pop.A/TPT.

The Pero.A, PTA and ATA/DPA are patent throughout, with damped/pulsatile monophasic flow sampled distally, PSV 0.2 m/s, 0.5 m/s and 0.5 m/s, respectively - ABPI 0.3-0.4.

At proximal PTA there is a 3-4x focal velocity increase, PSV 1.5 m/s, suggestive of a 50-75% stenosis.

Conclusion:
Occlusive (acute-on-chronic) and stenotic SFA disease.
Proximal PTA 50-75% stenosis

Damped/pulsatile monophasic flow sampled at ankle level - ABPI 0.3-0.4.

Abdominal aorta + Visceral arteries:
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Right:
The CIA, IIA, EIA, CFA, and prof.A origin are patent with pulsatile monophasic flow.

There is a heavy burden of predominately hypoechoic plaque/atheroma at SFA origin, appears to be causing a 2-3x focal velocity increase, PSV 4.2 m/s, suggestive of a 50-75% stenosis.

The remaining SFA, Pop.A and TPT are patent with monophasic pulsatile flow.

The Pero.A, PTA and ATA/DPA are patent with pulsatile monophasic flow sampled at ankle level, PSV 0.3 m/s, 0.2 m/s and 0.8 m/s.

There appears to be a ~2x focal velocity increase at the ATA and Pero.A origins, PSV 2.3 m/s and 1.1 m/s, respectively; both suggestive of borderline ~50% stenoses.

Conclusion:
50-75% stenosis at SFA origin.
ATA and Pero.A origin ~50% stenosis.

Right:
Triphasic pulsatile flow sampled in the CFA, Prof.A origin and proximal SFA.

At mid-distal SFA (puncture site), there appears to be an AVF, with high-velocity, pulsatile, low-resistive flow sampled in the SFV at this level.
Unable to rule out ?pseudoaneurysm or dissection, due to surrounding haematoma and pt pain tolerance creating poor views.

The proximal SFV and CFV has high velocity, pulsatile venous flow - further supporting evidence for AVF.
Below the level of the puncture site there is an extensive acute DVT that extends distally (see separate right lower limb DVT scan report)

Distal SFA and Pop.A are patent with normal arterial flow sampled.

Triphasic pulsatile flow sampled in the PTA and ATA/DPA at ankle level, PSV 0.9 m/s and 0.7 m/s, respectively - ABPI ~1.0.

Conclusion:
?AVF at mid-distal SFA/SFV
Extensive proximal DVT.

Right:
Triphasic pulsatile flow sampled at CFA and proximal Prof.A.

Highly resistive, low-velocity pulsatile flow sampled at proximal SFA.
At prox-mid SFA there is a >4x focal velocity increase (PSV ~2.0 m/s); suggestive of a >75% stenosis - appears very tight.
Below this point, flow becomes low velocity damped monophasic.
At mid-distal SFA, there is a further ~5 cm stenotic segment, with at least one focal ~50% stenosis (PSV 0.6 m/s).

Pop.A and TPT are patent with dampened monophasic flow.

ATA is patent throughout, with borderline damped/pulsatile monophasic flow.
At distal ATA (~5 cm above ankle level) there is a 2-3x focal velocity increase (PSV ~0.4-0.5 m/s); suggestive of a 50-75% stenosis.

The DPA is patent with borderline damped/pulsatile monophasic flow - followed in continuity to mid-foot, PSV 0.1-0.2 m/s.

The Pero.A is now patent throughout, with low-velocity pulsatile monophasic flow, distal PSV 0.1 m/s.
At Pero.A origin, there appears to be a ~2x focal velocity increase (PSV 0.6 m/s); suggestive of a 50% stenosis.

No flow could be ascertained from the PTA, ?occluded throughout.

Conclusion:
SFA is patent, albeit, with severe chronic disease - at least two significant stenoses detected (>75% and 50-75%)

Pero.A and ATA/DPA patent throughout - two vessel run-off
- 50% stenosis at Pero.A origin
- 50-75% stenosis at distal ATA

PTA ?chronically occluded throughout

Left:
Abdominal aorta patent, and of normal calibre (~1.7 cm) - mild degree of mural thrombus detected (10-19%).

The CIA, IIA, EIA and CFA are patent with triphasic pulsatile flow.

The Prof.A origin is patent, albeit, with 2x focally raised velocities (PSV 2.3 m/s); suggestive of a 50% stenosis - raised velocities appears to be caused by the proximal SFA stent terminus which extends juxta to Prof.A origin, opposed to intraluminal disease.

The SFA stent is patent throughout, however, at 1-2 cm below stent origin there is a 3-5 cm segment of homogenous hypoechoic narrowing/canalised flow causing at least a 2-3x focal velocity increase (PSV 4.4 m/s); suggestive of a 50-75% stenosis - visually it appears to be very tight, more likely to be nearer to ~75%.

Below this point the remaining flow in the SFA and Pop.A becomes pulsatile monophasic.

The TPT is patent, with a >4x focal velocity increase (PSV 2.0 m/s), suggestive of a >75% stenosis.

There remains >75% stenoses at the origins of the Pero.A (PSV 2.2 m/s), PTA (PSV 2.7 m/s) and ATA (PSV 2.0 m/s), respectively.

The remaining crural vessels are patent, with pulsatile hyperaemic flow sampled at ankle level, typical PSV 0.6 m/s.

Conclusion:
No significant change compared with previous scan (06/04/23).

50-75% stenosis just below stented SFA origin.
~75% stenosis distal TPT.
>75% stenoses at PTA, PeroA and ATA origins.

ABPI ~0.6

US Graft Crossover or Extra-Anatomical

Bypass graft follow-up

F/U interval:	>12M	**Location:**	R-L X-OVER	**Type:**	PTFE
Best Resting Ankle Pressure	94	mmHg			
Brachial systolic Pressure:	148	mmHg			
ABPI:	0.64	mmHg			
ABPI Not Measured	-				

Comments:

Inflow:

Distal aorta is patent and of normal calibre (~1.5 m/s).

Right CIA, IIA, and CFA are patent with biphasic pulsatile flow.
The right CIA stent is patent, albeit, at the distal stent terminus there is a ~2x velocity increase (PSV 2.4 m/s); suggestive of a ~50% stenosis.

At right EIA origin (just below junction of IIA), there is a 2-3x focal velocity increase (PSV 4.1 m/s); suggestive of a 50-75% stenosis.

Proximal Anastomosis:

Patent - no evidence of significant stenosis.

Graft:

Patent - no evidence of significant stenosis, typical PSV 0.6-0.9 m/s.

Distal Anastomosis:

Patent, albeit, with a 2x focal velocity increase (PSV 1.3 m/s); suggestive of a 50% stenosis, however, appears widely patent with B-mode and colour imaging.

Run-Off:

Left CFA and Prof.A run-off are patent, with biphasic pulsatile flow.
Know SFA occlusion from origin.

Damped monophasic flow reconstitutes are Pop.A

The left distal Pero.A and PTA are patent dampened/pulsatile monophasic flow, PSV 0.3 m/s and 0.3 m/s, respectively.
No flow ascertained from distal ATA/DPA, ?occluded.

Date of next scan

Please advise.

Comments:

Right CIA and EIA stenoses.
No significant change to ABPI or in graft velocities compared with previous scan

Right:
Limited views of aorta-iliac region due to stoma in situ; however, where seen:
The EIA and proximal CFA are patent with biphasic pulsatile flow.

Heavy calcified plaque burden identified at mid-to-distal CFA (2-3 cm in length); unable to trace colour flow in continuity in the this region, ?acoustic shadowing/short occlusion (~1-2 cm length). In addition, there is at least a focal >75% stenosis at very distal CFA/Fem.A origin.

Prof.A origin is patent with turbulent monophasic flow - flow quickly transitions into dampened monophasic - further suggestive of significant disease at CFA.

The SFA and Pop.A are patent with dampened monophasic flow - diffuse calcified disease seen throughout this region (<50%).

Proximal ATA is patent; albeit, becomes occluded ~5 cm below knee level, flow does not appear to reconstitute distally.

The prox-to-mid PTA is patent with dampened monophasic flow; with an additional 50-75% stenosis at mid-calf.
The PTA is occluded from mid-calf to ankle level. The distal PTA reconstitutes with dampened monophasic flow, via a large collateral that originates from the distal Pero.A, PSV 0.3 m/s - ABPI 0.4-0.5

The Pero.A is patent throughout, with dampened monophasic flow.
Raised velocities sampled at TPT/pero.A origin (<50%); likely attributed with compensatory flow.

Conclusion:
Heavy calcified plaque burden identified at mid-distal CFA, >75%/?short occlusion.
Relatively poor flow sampled in remaining patent vessels.

Pero.A main arterial source to foot/single vessel run-off.

PTA occluded mid-to-distal - reconstitutes distally via Pero.A collateral.

ATA/DPA occluded.

Right:
Suboptimal views of abdominal aorta due to bowel gas and large over-arching liver. However, where seen:
Diffuse calcified plaque seen throughout the aorta and proximal leg arteries.
Abdominal aorta is patent and of normal calibre (~1.5 cm).

Triphasic pulsatile flow sampled in the CIA, IIA, EIA, CFA, Prof.A, Fem.A, Pop.A and TPT - no evidence of significant stenosis.

Bi/triphasic pulsatile flow sampled in the Pero.A, PTA and ATA/DPA at ankle level, PSV 0.4 m/s, 0.7 m/s and 0.4 m/s, respectively.

Of note, large heterogenous non-vascularised collection identified in the popliteal fossa, extending down the medial calf, ?bakers cyst. Does not appears to be causing any significant compression to popliteal artery or vein - beyond our area of expertise, alternative imaging is advised.

Right:
Poor views of aorto-iliac region - where seen:
The distal abdominal aorta is patent and of normal calibre (~1.5 cm, AP).

Diffuse calcified plaque seen throughout.
The CIA, EIA, CFA, Prof.A, and Pop.A are patent with biphasic pulsatile - generalised calcified plaque seen throughout (<50%).

Raised velocities sampled at ATA origin (PSV 1.2 m/s), suggestive of at least a ~50% stenosis; however, raised velocities also likely attributed with compensatory flow.

At prox-mid ATA (~8 cm below knee) there is a >4x focal velocity increase (PSV 2.7 m/s); suggestive of a >75% stenosis.

At mid-distal ATA (~10 cm above ankle), there is a 3-5 cm stenotic lesion, with at least four separate ~3-4x focal velocities increases; equating to two 50-75% stenoses and two >75% stenoses.

Borderline pulsatile/dampened monophasic flow continues into the DPA, PSV 0.3 m/s - ABPI 0.5-0.6

The PTA and Pero.A appear occluded from TPT - no flow reconstitutes distally.

Conclusion:
Generalised diffused calcified disease.
ATA patent, single vessel run-off/main arterial source to foot.
Several ATA stenoses - damped/pulsatile monophasic flow sampled distal - ABPI 0.5-0.6

TPT, PTA and Pero.A occluded - no flow detected distally.

Left:
Diffuse calcified disease seen throughout.

CFA is patent with triphasic pulsatile flow.

Prof.A origin is patent, albeit, tightly stenosed - a >4x focal velocity increase (PSV 4.0 m/s) is suggestive of >75% stenosis.
Turbulent triphasic pulsatile flow continues into the proximal Prof.A.

The SFA origin (above stent) is patent, albeit, stenosed with at least a ~50% stenosis (PSV ~2.5 m/s).
The proximal SFA stent is patent throughout; no evidence of in stent stenosis.

At proximal SFA flow is triphasic pulsatile, and progressively becomes high resistive low velocity monophasic pulsatile flow towards mid thigh.
At mid thigh there is a ~10 cm SFA occlusion; damped low velocity monophasic flow reconstitutes at distal SFA.

At very distal SFA there is a >4x focal velocity increase (PSV 0.5 m/s); suggestive of a >75% stenosis - however, this could be also attributed with a junction of a collateral.

The Pop.A/TPT is patent throughout, with damped monophasic flow.

At Pero.A origin, there is a ~50% stenosis. Damped monophasic flow sampled in the Pero.A at proximal calf, however, appears occluded throughout the remaining calf.

ATA and DPA is occluded throughout - no flow reconstitutes distally in the foot.
Distal ATA to prox-mid DPA are incompressible, with mixed echogenic material within lumen.

PTA is patent throughout - single vessel run-off.
At PTA origin there is a >75% stenosis.
Damped monophasic/anaphasic flow continues throughout the PTA, PSV at ankle level ~0.3 m/s - ABPI 0.2-0.3.
Medial and lateral plantar arteries are patent with damped monophasic flow, PSV 0.1 m/s and 0.1 m/s, respectively.

Conclusion:
SFA origin and Prof.A origin significant stenoses.

~10 cm occlusive segment in the Mid-to-distal SFA.
?>75% stenosis at very distal SFA

Pero.A origin ~50% stenosis - patent to mid calf and occluded distally.

ATA/DPA occluded throughout - no flow reconstitutes distally.

PTA and plantar arteries patent albeit with severely dampened flow (single vessel run-off/main arterial source to foot).
>75% stenosis at PTA origin.

Discussed results with vasc DR over the phone.
Sent pt to diabetic foot clinic.