

Peripheral artery aneurysms.

Extra cranial carotid aneurysms.

Defined as 1.5 times the diameter of the normal common carotid artery (Attigh EJVES 2009). These are rare, accounting for 8 out of 1500 carotid procedures. They most often involve the common carotid artery, followed by middle and distal internal carotid artery (ICA). Aneurysms at the bifurcation are usually fusiform, whereas those in the ICA are usually saccular. The main risk is thromboembolism as rupture is rare. Thrombosis can occur.

Causes:

- Atherosclerosis – 50%. Usually fusiform aneurysm at the bifurcation
- Trauma – usually saccular
- Intimal dissection
- Congenital/familial
- Mycotic (0 – 2.8%) e.g. Tuberculous, usually causing pseudoaneurysms. Most common in IV drug addicts but also tooth and jaw infections.
- Secondary to previous surgery
- Cystic medial necrosis – associated with other peripheral aneurysms
- Fibromuscular dysplasia
- Marfans syndrome
- Polyarteritis nodosa
- Syphilis – now rare.
- Irradiation
- Neurofibromatosis
- Bechets syndrome
- Takayasu's – commonest cause in young patients

Usually present with a mass in the neck, tonsillar fossa or oropharynx (dysphagia). Distal ICA aneurysm may cause facial pain, 5th or 6th nerve palsy, deafness or Horner's syndrome. Some present with a TIA or stroke.

Raeders paratrigeminal syndrome – intermittent facial pain and oculosympathetic paresis – aneurysm situated at base of skull. Hoarseness is due to vagal or recurrent laryngeal palsy. Hypoglossal nerve may be involved.

Differential diagnosis:

- Lymph nodes
- Branchial cyst
- Carotid body tumour
- Tonsillar abscess

CT or MRI needed for high lesions.

Treatment.

Ligation had a 50% mortality in early reports but recently the results have been better (25% risk of stroke and 20% mortality). Ligation is considered when the aneurysm is very high. Can occlude the artery slowly over several days during which the patient is anticoagulated to prevent embolisation (Selverstone clamp). Can do test ligation with balloon.

Interposition grafting (vein or prosthetic) is possible in most. Stent grafting may be possible.

Operative mortality 1%. Permanent neurological deficits – 5%, cranial nerve injury 11 – 22%.

Subclavian/axillary artery aneurysms.

Subclavian aneurysms are usually due to thoracic outlet syndrome. Axillary aneurysms are usually due to crutch use. Usually present with embolisation. Can cause neurological symptoms due to stretching of the brachial plexus.

Treatment.

Conventional surgical treatment. Proximal left subclavian aneurysm requires left thoracotomy for proximal exposure.

Endovascular stent grafts may have a roll.

Brachial artery aneurysm.



Figure 1. A brachial artery aneurysm in a patient with polycystic kidney disease.



Figure 2. Brachial artery aneurysm repair with vein graft to ulnar and radial artery

Femoral artery aneurysms.

Causes:

- Atherosclerosis, in which case 70% have bilateral disease and 85% will have an aneurysm, at another site.
- False aneurysms at vascular anastomosis or vascular access
- Trauma
- Infected or mycotic aneurysms.

Treatment.

Profunda femoris aneurysms.

Rare, maybe because of the muscle coverage of the artery and the arterial wall being rich in muscle fibres and poor of elastic fibres. Can rupture, thrombose. May involve the common femoral artery.

Popliteal artery aneurysms.

The commonest peripheral artery aneurysm. Prevalence of approximately 1% of men aged 65 – 80. Found almost exclusively in men. Bilateral in 50%, 75% have an aneurysm at another site. 10% of patients with an AAA have a popliteal artery aneurysm.

Percivall Pott (1714 – 1788) advocated amputation as being the best treatment for symptomatic popliteal aneurysms.

The normal popliteal artery diameter is up to 9mm. Some have described 10mm or more as being aneurysmal in the popliteal artery (Wolf 2006 JVS) but conventionally, 2cm is the diameter above which most would describe a popliteal artery as being aneurysmal.

Causes:

- Atherosclerosis.
- Trauma – e.g. knee dislocation, knee replacement
- Inflammation – Behcet's, Kawasaki disease
- Infection – staph, salmonella
- Popliteal entrapment syndrome
- Marfans

Presentation:

70% are symptomatic at presentation.

- Thrombosis, causing an acutely ischaemic leg (40% amputation rate)
- Embolization
- Popliteal nerve compression – sciatic nerve
- Popliteal vein compression
- Rupture – very rare. Can present as acutely ischaemic leg due to arterial compression or cause AV fistula.

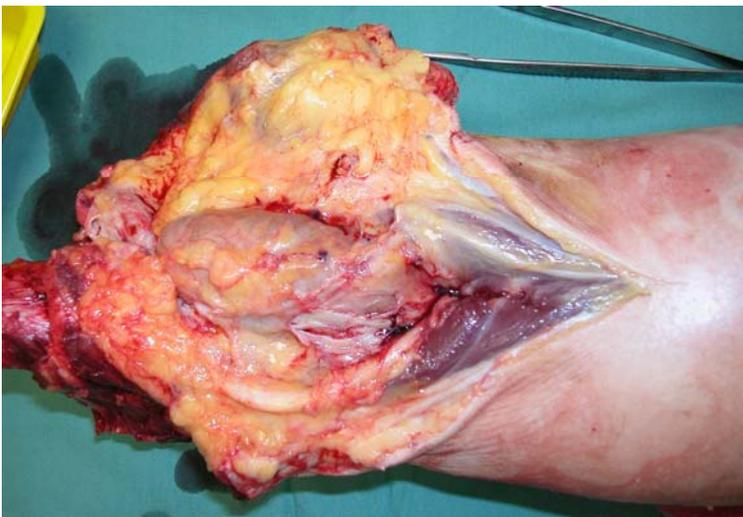


Figure 1. A popliteal artery aneurysm which resulted in an amputation.

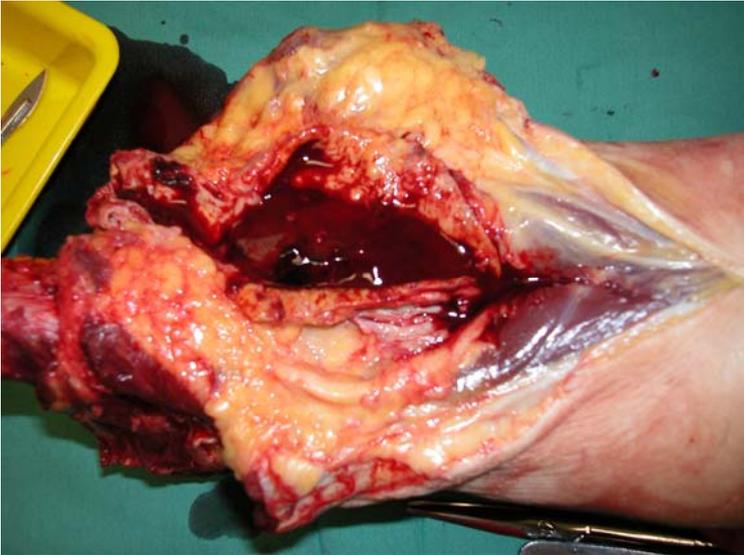


Figure 2. The aneurysm was full of fresh clot.

Treatment

Indications for intervention:

- Acute ischaemia
- Embolisation
- Size greater than 2cm diameter
- Intraluminal thrombus
- rupture

Thrombolysis is useful for acute thrombosis, but risks distal embolisation. Mainly used to treat run off prior to ligation and bypass.

Bypass with reversed vein. Approach via medial or posterior incision. Early primary patency is better with the medial approach, possibly because of limited access in posterior approach. However, due to the risk of continued aneurysm expansion with the medial approach (22%), the posterior approach may have long term advantages (Kropman JVS 2007). The posterior approach is better when there are compressive symptoms. Embolisation may require bypass to a distal tibial vessel. 5 year primary patency is 60 – 80%. Bypass grafts placed urgently have a 10% early failure rate vs. 1.2% if placed electively. 35% - 43% amputation rate for PAA presenting with acute thrombosis and 15% amputation rate due to early graft thrombosis. Operative mortality of 5%. Results are better for asymptomatic PAA's. 5 year primary patency of 80 – 90% can be expected for elective treatment as opposed to 40 – 70% for acute presentations.

Proximal and distal ligation may not prevent genicular vessels from continuing to casue aneurysm expansion in 15%.

EVR may be possible. Vice president Richard Cheney had his incidental popliteal artery aneurysm repaired endovascularly.



Figure 4. Popliteal artery aneurysm repaired endovascularly with 4 stent grafts

Splenic artery aneurysms.

The most common visceral artery aneurysm account for 60% of all splanchnic artery aneurysms. Incidence – 0.8% but may be up to 10.4% of elderly patients when specifically sought at post mortum. Female:male 4:1. In younger patients female dominance is more obvious. Most arise in the distal third of the splenic artery, close to the splenic hilum. Predisposing conditions:

- Arterial fibrodysplasia - usually with hypertension secondary to renal artery medial fibroplasia. This association is present only in women. 2% of those with renal artery fibrodysplasia have splenic artery aneurysm.
- Pregnancy – due to increased splenic artery blood flow and alterations in vessel wall elastin. 40% are grand multiparas. May also be related to portal hypertension and splenic arteriovenous fistula seen in pregnancy.

● Portal hypertension and splenomegaly – 10% develop splenic artery aneurysm. Seen more commonly after liver transplants (greater risk of rupture), cirrhosis (7 – 17% of patients with cirrhosis), porto-systemic shunts.

Less common causes – pancreatitis (usually false aneurysm), trauma (US President James Garfield died secondary to rupture of traumatic SAA produced by a bullet wound from his attempted assassination), Mycotic, polyarteritis nodosa, Ehlers Danlos syndrome, etc.

Splenic artery aneurysms exhibit typical calcific arteriosclerotic changes (curvilinear aneurysmal calcification or signet ring calcification) – more likely a secondary event. Most are saccular. Multiple in 20%.

Presentation.

Most are asymptomatic. Can present with a double rupture, first into the lesser sac then into peritoneum. Pancreatitis aneurysms usually rupture into the stomach or the pancreatic duct (haemosuccus pancreatitis). May rupture into splenic vein causing AV fistula.

2 – 10% incidence of rupture. Rupture in pregnancy is associated with a 70% maternal mortality and a fetal mortality greater than 75%. The mortality rate for surgery for ruptured splenic artery aneurysm is 25%, higher when associated with portal hypertension. Operation for SAA due to pancreatitis has a 30% mortality due to persistent intra abdominal sepsis and in the presence of rupture, a mortality of 50%.

Indications for elective intervention:

- If patient is or may become pregnant
- Aneurysm 2cm or larger
- Symptomatic – abdominal pain.
- Patients undergoing liver transplant

Treatment Options:

- Embolisation using coils, sponge, cyanoacrylate or balloons – collateral circulation usually preserves splenic function.
- Simple ligation and or excision – open or laparoscopic. Open – upper midline or left subcostal incision. Divide gastrocolic ligament to enter lesser sac. May require splenectomy.
- Inflammatory aneurysm in tail of pancreas – distal pancreatectomy
- Endovascular stent graft
- Percutaneous thrombin injection for pseudo aneurysm

Hepatic artery aneurysms.

Account for 20% of splanchnic aneurysms. Males:female 2:1. Atherosclerotic aneurysms mainly in 60's. Those due to trauma and infection associated with IV drug abuse and endocarditis occur in the 3rd and 4th decade. Caused by medial degeneration in 25%. Polyarteritis nodosa causes microaneurysms. Usually solitary, 80% extrahepatic.

Most are asymptomatic prior to rupture. Large aneurysms can cause obstructive jaundice.

AXR – eggshell calcification.

Mortality rate for ruptured aneurysms is 35%. Classic triad – haemobilia, pain and jaundice. Bleed into biliary tract (haemobilia) or peritoneum.

Treatment

Simple ligation and bypass. For aneurysms proximal to gastroduodenal artery, ligation is sufficient. In 40% of patients the hepatic artery can have an anomalous origin – from left gastric or SMA.

Thus pre op angiogram mandatory.

Embolisation is useful for intrahepatic aneurysms.

Endovascular stent graft.

Percutaneous thrombin injection.

Superior mesenteric artery aneurysm.

6% of splanchnic aneurysms. M = F. Most commonly mycotic (60%) from bacterial endocarditis (*streptococcus, staphylococcus*) in IV drug addicts. Other causes – medial degeneration (can also cause SMA dissection), arteritis, atherosclerosis (rare), trauma and neurofibromatosis.

Most have symptoms of intestinal ischaemia prior to rupture. Rupture is unusual, thrombosis is more common. Mortality from rupture – 50%.

May require medial visceral rotation for access to the proximal vessel. Simple ligation is possible in some. If bowel viability is compromised, used saphenous vein graft.

Coeliac artery aneurysm.

4% of splanchnic aneurysms. M = F. Usually saccular. 38% have other splanchnic artery aneurysms, 18% have AAA. 60% present with abdominal symptoms, 30% present with rupture. Rupture mortality = 50%.

Surgical exposure requires thoracoabdominal incision. 30% tolerate coeliac ligation without reconstruction.

Gastric and gastroepiploic artery aneurysms.

Gastric artery:gastroepiploic 10:1. male:female 3:1. Usually solitary. Usually due to arteritis or medial degeneration. Most rupture into GI tract. Rupture – mortality rate 70%.

Jejunal, ileal and colic artery aneurysms.

M = f. Solitary in 90%. Rupture mortality 20%. Usually rupture into GI tract.

Pancreaticoduodenal, pancreatic and gastroduodenal artery aneurysms.

Pancreaticoduodenal artery aneurysms can be associated with coeliac artery stenosis or occlusion in 63% of cases. Male:female 4:1. Associated with pancreatitis. Segmental arterial mediolysis – diffuse cytoplasmic vacuolar transformation of medial smooth muscle. Pathogenesis remains obscure. Mortality from rupture = 50%. Treatment recommended regardless of size. Options:

- Ligation
- Pancreaticoduodenectomy
- Percutaneous embolisation.

Renal artery aneurysms.

True aneurysms– 0.1% of general population. F>M due to the association with fibromuscular dysplasia. Right more than left. Dysplastic disease is more common and sever on right. Mostly saccular. Can be associated with polyarteritis nodosa.

Rupture mortality – 10%. Rupture during pregnancy – 85% fetal mortality and 45% maternal mortality.

Dissecting renal artery aneurysms – usually due to blunt trauma or iatrogenic. M:F 10:1. r>l. 30% are bilateral.

Iliac artery aneurysms.

More often associated with AAA than found in isolation. Incidence 0.03%. Anastomotic IAA can occur following previous aneurysm or bypass surgery. Isolated IAA more than 3cm in diameter are recommended for repair.

The close anatomic relation of veins and ureters results in risk of damage to these structures.

Endovascular repair is a good option in those suitable. This may require internal iliac embolisation.

This can result in complications – gluteal claudication, The iliac extension piece of the various aortic bifurcated stent grafts provide good stent graft for iliac aneurysm. One advantage of these is their tapered design.

Internal iliac artery aneurysms.

Usually present with abdominal aortic or common iliac aneurysms. In isolation they are rare. Prevalence lower than 0.03%. Males:female 6:1. Most are due to atherosclerosis. Other causes include mycotic (salmonella, staphylococcus aureus, E. coli, pseudomonas, Klebsiella). In neonates can be due to umbilical artery catheterisation. Other causes – iatrogenic following hip replacement, lumbar disc surgery, traumatic delivery, connective tissue disorders (Marfans, Ehlers danlos, FMD, Takayasu's, Kawasaki disease, Bechets, cystic medial necrosis).

Most would recommend surgery for aneurysms greater than 3cm in size. But 40% present with rupture, of whom 52% die. Urological symptoms are common and reported in 54% of cases.

Current treatment options:

- Ligation – unilateral ligation is well tolerated if the contralateral artery is free of disease. Historically, only proximal ligation was performed due to difficult distal access.
- Excision – high risk of bleeding and venous injury.
- Endoaneurysmorrhaphy-
- Embolisation – coils should be placed as proximal as possible prevent interference with pelvic collateral circulation.
- Endovascular stenting. – usually by means of covering the internal iliac origin with a stent graft in the common to external iliac artery following coil embolisation of the distal internal iliac.

There is a 7 – 11% operative mortality for elective cases.

Carotid artery aneurysms.

Causes:

Atherosclerosis – 26 - 50%

Post traumatic, - 16%

Fibromuscular dysplasia

Post carotid endarterectomy

Treatment.

Open surgery – resection and interposition grafts, resection and patch angioplasty, resection and end to end anastomosis and carotid ligation (up to 50% mortality).

Endovascular options – stent graft exclusions, coil exclusion and endovascular balloon exclusion.

Radial artery aneurysms.

These are usually false aneurysms secondary to radial artery cannulation. Most true aneurysms are idiopathic but can be due to repetitive trauma (Tailors Thumb) (Behar EJVES 2007).

Treatment.

Provided the ulnar artery is patent, arterial ligation is usually enough.