ANEURYSM DISEASE

Definition – abnormal permanent dilatation of any part of the circulatory system (arterial: >50% adjacent seg)

True: all layers of arterial involved in dilatation
SACCULAR: dilatation of part of circumference communicates with arterial lumen via narrow neck (focal wall weakness eg infection)
FUSIFORM: generalized circumferential dilatation (commonest eg atherosclerosis)

False: blood leaks extraluminally and flows in a separate channel
DISSECTION
PSEUDOANEURYSM
AV FISTULA

Epidemiology
AAA: increasing incidence with age; 6x higher in men; prevalence 4% of men 65-74
Popliteal:
Femoral:

25% of AAA patients have co-existing femoral/popliteal aneurysm (implies common aetiology)

Aetiology
Congenital: CoW berries/E-D, Marfan's, Loey-Dietz etc (dissections and sacculars)
Degenerative: atherosclerosis (fusiforms)/HTN eg Charcot-Bouchard's brain aneurysm
Trauma: blunt or penetrating, iatrogenic or otherwise
Inflammatory: connective tissue disease (eg AS, Reiter's, RA, SLE, Takayusu's, GCA)
Infective: mycotic from endocarditis emboli/syphilitic/microbial aneurysmal arteritis
Ischaemic: post-MI ventricular aneurysm

Pathology
Abnormal local MMP production (especially elastolytic subtypes MMP-9 and MMP-2)
Histology: chronic inflammatory infiltrate (T cells, B cells, macrophages, plasma cells)
No genetic anomaly identified in all AAA patients but genetic risk identified in first order relatives

Pathogenesis
Ailawadi's unifying theory:
Initial AAA trigger is combo of factors (fragmented medial proteins, haemodynamic stress etc)
Causes inflammatory infiltrate rich in chemokines/cytokines/ reactive oxygen species
Expression of proteases (esp from MMPs)
Unregulated connective tissue turnover → medial degeneration → aneurysmal dilatation
As AAA wall expands, further stress exacerbates proteolysis → eventual aortic rupture

Inflammatory aneurysms
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Infected aneurysm
Mycotic: septic emboli of cardiac source lodge in vasa vasorum or arterial lumen of healthy vessels
Middle-aged, multiple aneurysms at differing sites (aorta, femoral, visceral, intracranial)
Usually G+ cocci (*Strep spp. and Staph aureus)*

Microbial aneurysmal arteritis
Blood-borne bacteria seed into already diseased arterial intima → suppuration, perforation, pseudoaneurysm
Healthy vessels not affected (unlike mycotic aneurysms); aorta affected much more than peripheral vessels
Usually *Salmonella spp* but can be *Staph/kleb/E. coli*
Management: abx and surgical management (resection, wide debridement, irrigation, drainage, microbiological diagnosis, arterial reconstruction (autologous vein graft)

ABDOMINAL AORTIC ANEURYSM

Normal aortic calibre 2cm, aneurysmal at 3cm

90% infrarenal (>1cm below ostia), 10% juxtarenal (within 1cm of ostia) or pararenal (involve ostia)

Risk factors (ADAM study): *(male sex and smoking most important):*

F: Age (elderly)/Male(6x risk)/Height/Ethnicity/Family history (double-risk if FDR had AAA; Swedish survey)

R: Smoking (7x risk) duration rather than number smoked/Hypercholesterolaemia/Hypertension

Lower risk: women, Africans/Asians, diabetics

Symptoms

Pain: dull central/epigastric, bloating/stretching

Compression: duodenum/IVC(DVT, syncope, leg oedema)/pelvic nerves (iliac and femorals)/sciatica and vertebral erosion

Extraluminal flow: renal, visceral, spinal arteries

Thrombosis/Embolism: limb ischaemia/digital ischaemia (moreso with peripherals)

Rupture: retroperitoneal or into peritoneal cavity

Fistula: aortocaval/aortoduodenal (herald bleed)

*most asymptomatic until rupture (75% symptom-free at diagnosis)

*(a)rapid size increase (>1cm/yr) (b)pain (c)saccular) → surgical/endovascular intervention regardless of size

Examination findings

1.Expansile, pulsatile mass

*if non-expansile then (i) abdo mass transmitting aortic pulsation (ii) dilated, tortuous artery

2.Tenderness: (i) inflammation (ii) imminent rupture

Rupture: massive haemorrhage and circulatory collapse; may rupture into retroperitoneum with tamponade

Aortocaval fistula: high output cardiac failure (machinery murmur)

Aortoenteric fistula:pain, pulsatile mass, GI bleed; CT=contrast in duo, air in aorta; extra-anatomic repair + close intestinal defect (SVS)

Inflammatory AAA:

Triad: thickened aneurysmal wall, retroperit fibrosis, dense neighbouring visceral adhesions (50% involve ureter)

Another triad: Pain, weight loss, raised ESR = IAAA until proven otherwise

IMAGING

USS: detection and surveillance; imprecise diameter measurement and limited by bowel gas/obesity

CT: good for extent and morphology; overestimate size (not always truly axial diameter)

Population screening

Rationale: 75% asymptomatic/elective surgery effective/75% of ruptures die pre-hospital

Ultrasound: accurate and inexpensive (single scan of men at 65 yrs detects 90% of AAA at risk of rupture

MASS (Multicentre Aneurysm Screening Study):aneurysm-related mortality reduced 53% in screened 65-74 male population

NAASP (NHS AAA Screening Programme): US screening of men at 65

PRINCIPLES OF AAA MANAGEMENT (ie surveillance and when to intervene)

1. Prevent rupture: risk-factor modification (BP/cholesterol reduction/anti-platelet therapy/smoking cessation)
2. Intervene when risk of rupture outweighs risk of intervention: (a)size (b)pain (c)rapid growth 1cm/yr (d)saccular

   Size is most important factor in predicting risk of rupture
   Intervene at 5cm in women

UK Small Aneurysm Trial and US ADAM (Aneurysm Detection and Management) Trial:
-randomised 1090 4.0-5.5cm to US surveillance or surgery
-surgery group: 30-day mortality 5.8%
-no difference between groups so intervention below 5.5cm not indicated
-found that women had higher rupture rate in small aneurysms so suggested earlier intervention here

AAA size (cm)	Risk of rupture per year (%)
<3	0
3-3.9	0.4
4-4.9	1.1
5-5.9	3.3
6-6.9	9.4
7-7.9	24

Thus:
Asymptomatic: <5.5cm (men)/<5.0cm (women) managed conservatively with regular interval ultrasound
(i)Annual for 3.5-4.4 (ii)3 months for 4.5-5.4 (iii)2 week urgent vascular OPD at 5.5cm *(NICE)*
Offer intervention at 5.5 men/5.0 women if fit, rapid growth 1cm/yr, pain

Expansion risk:
Rupture risk (UKSAT): female(4x risk)/large initial diameter/low FEV1/current smoker/high MBP/transplant patient

Investigations
Risk of rupture: size and growth, morphology (US for detection and surveillance, CTA when intervening)
Pre-op assessment: vasculopath workup (FBC, U&E, CS, GS)(ECG, ECHO)(CXR, PFTs) + optimise
Assess anatomical suitability for EVAR: CTA
Discuss at MDT
Reiscuss options with patient in clinic and proceed

DVLA: cars must notify at 6cm and suspended at 6.5cm; HGVs etc notify and suspended at 5.5cm

ELECTIVE OPEN AAA REPAIR

Indicated if unsuitable for EVAR (prox length <15mm, renal ostia involved or within 1cm, neck >3cm, angulated >60deg, unhealthy iliacs <7mm or ectatic

GA/Epidural/Antibiotics vs graft infection/Heparin just before cross-clamping (JVRGGBI showed heparin has no effect on bleeding or clotting events but reduces peri-operative MI by 75%)
Cell-saver: backbleeding from lumbar arteries or from anastomotic suture lines
Approach: midline transperitoneal (can do oblique left extraperitoneal)
Incise posterior peritoneum and mobilise duodenum to right (right renal vein marks dissection limit)
Dissect both common iliacs (avoid hypogastric plexuses)
Fabric graft (Dacron or PTFE): anastomosed to infrarenal neck and bifurcation (tube or tube + bifurcation graft)

*suprarenal clamping in juxtarenal aneurysms – approach aorta via lesser sac and hiatus to easier visualise renal arteries and reduce risk of renal athero-embolisation; thoraco-abdominal approach in obese patients considered

Outcomes: **mortality 5.6% (UKSAT)**

Complications:
1. As per open surgery
2. Graft infection (presents ~3 yrs later; higher if graft into femorals; excise, debride, extra-anatomic recon)
3. Limb occlusion (EVAR>OSR, women, grafts into femorals;

RUPTURED AAA

Presentation
Investigations: CTA to diagnose (impending= calcification breach, mural haematoma)
(rupture= retroperitoneal haematoma, peri-aortic stranding, contrast leak)
Pre-operative workup (FBC, U&E, CS, G&C)(ECG, ECHO)(CXR)
Management: CTA to determine if OAAAR or EVAAAR (50% mortality)

EVAR

Aim: exclude aneurysm from systemic circulation with preoperatively-sized stent graft to prevent further expansion/rupture

SUITABILTY (based on aneurysm morphology)
Site: 1cm+ below renals (true infrarenals)
Neck: limited angulation (less than 60deg), at least 15mm long, no more than 30mm diameter, no mural thrombus
Iliacs: (i)7mm calibre to deploy delivery apparatus and device; (ii)non-ectatic (need good distal seal) (iii)non-tortuous

DEVICES
All form proximal seal in infrarenal aortic segment with different distal landing site; tight seal at all points is crucial
Single vs modular (need both iliacs patent)
1. Straight aorto-aortic tube endograft
2. Bifurcated systems
3. Aorti-mono-iliac systems (require fem-fem crossover to maintain contralateral femoral supply)
4. Combined bifurcated and iliac branched stent grafts (if aneurysmal iliacs)

Fusiform disease can expand into distal neck and into aorto-iliac segments, leading to graft failure (esp aorto-aortic) and this is where bifurcated systems are useful
Not such an issue in saccular/ulcerative/pseudoaneurysm

COMPLICATIONS
(1)Graft complications:
Endoleak: presence of blood flow outside lumen of an endovascular graft but within aneurysm sac → enlargement and rupture
Primary (at time of EVAR) or Secondary (not seen at completion angiography ie seen later)
Classified by source of aberrant blood flow
Type I and III (communicate directly with lumen, need intervention before completing procedure)
Type II (filling from collaterals ie lumbar, IMA; image 3-6mthly) *commonest*
Type III (between devices; need intervention)
Type IV (stent porosity; seal spontaneously)
Type V (endotension: no leak seen by sac expands)

Graft migration: failure at any attachment site allows migration and Type I endoleak or even Type III
Graft dislocation: aortic cuff to repair proximal seal
Graft fracture:
Kinking/occlusion

(2)Operative complications:
Microembolisation due to manipulation of stent in disease segment
Rupture, dissection and pseudoaneurysm due to instrumentation
Renal failure: contrast nephropathy, renal artery trauma, stent induced stenosis and aortic neck thrombosis
Abdominal compartment syndrome
Exclusion of hypogastric artery to prevent TIIEL→ buttock claudication/erectile dysfunction (improves if unilateral)
MORTALITY 1.7% (cf 5.6% in open repair)

SURVEILLANCE AFTER EVAR
CT at 1 and 12 months after (SVS)
US can detect Type I leaks reliably, less so for Type 2 but if the sac isn't enlarging, this is not an issue to worry about

OUTCOMES AFTER EVAR
UK-EVAR-1: 1082 patients fit enough for elective open AAA randomised to surgery or EVAR
EVAR: lower mortality (1.8%) but higher complication, reintervention and later rupture rates than open
OPEN: higher mortality (5.6%), LOS but less reintervention and secondary complications

THORACIC AORTIC ANEURYSMS

EPIDEMIOLOGY

Age: increases with age ||Sex: 75% male

AETIOLOGY

TRUE:	FALSE:
Degenerative (most)	Trauma
Congenital: Marfan's, Ehler's Danlos	
Infective: Mycotic	
Inflammatory: Connective tissue disoders	

MORPHOLOGY

True: **Fusiform** (majority) **Saccular** (less common; infection/trauma)
False: dissections/pseudoaneurysms

Commonest site: descending aorta
Classification by dividing chest at 6th IC space:
A Upper half (ICS 2-6) B Lower half(ICS6-10) C Entire length

CLINICAL PRESENTATION

Pain retrosternal, back, shoulder)
Compressive symptoms: SVC syndrome/dysphagia/dyspnoea/stridor/hoarseness
Rupture (risk increases with size)

MANAGEMENT

Indications for intervention: 1. SIZE: 5.5-6cm in asymptomatics 2. SYMPTOMS
diameter thresholds should be reduced in connective tissue disorders

Surgical repair:
Left thoracotomy is mainstay (allows aortic clamping, inlay grafting and intercostal re-implantation)
Need to maintain VISCERAL and SPINAL perfusion

Endovascular repair:
Need landing zone above and below (at least 15mm on lesser curve, 20mm optimal) for effective seal
Bypass to debranch aortic arch or abdominal aorta if landing zone too short (hybrid procedure)
Oversizing by 15-20%; need about 5cm overlap if multiple grafts for long lesion
Need adequate calibre for deployment

Management of spinal cord perfusion:
Paraplegia is feared complication (coverage of T6-L1 intercostals → spinal cord ischaemia)
Increase SpC perfusion by:
1. reducing CSF pressure (to 10-12mmHg) with spinal drain placement (48-72 hrs post-op)
2. Increasing MAP (to 85-100mmHg) with inotropes

Marfan's: tend to be younger and fitter with excellent results from open surgery and little evidence necessitating EVR

OUTCOMES

Untreated: 13-39% 5 year survival
Open repair: 30- day mortality 12-20%, paraplegia 4%
TEVAR: EUROSTAR= 249 patients with 30 day mortality 5.3% in electives and paraplegia 4%
Mortality predictors: age, renal insufficiency, emergency presentation (main cause of operative mortality: stroke)

THORACO-ABDOMINAL AORTIC ANEURYSMS

Def: TAA involving visceral segment of abdominal aorta
Classification: Crawford (extent of aneurysmal disease)

Aetiology
-medial degeneration
-chronic dissection

CLINICAL FEATURES

Most are symptomatic (unlike infrarenal AAA, 75% of those asymptomatic)
Back pain

MANAGEMENT

Rationale: Crawford and DeNatale: 94 patients unsuitable for surgery, 24% survived 2 yrs but 59% of those operated on survived this long → recommended repair unless significantly co-morbid

SURGICAL (still the mainstay as TEVR emerges)
Detailed imaging and risk assessment (surgical risk vs rupture risk from diameter)
Left thoracotomy usual with left medial visceral rotation
Diaphragm cut circumferentially (preserves innervation)
Clamp either side and make lateral arteriotomy away from visceral origins
Proximal anastomosis on transected aorta (prevents aorta-oesophageal fistula) and try to include intercostal and visceral arteries
Any other branches re-implanted or jump-grafted
Distally, anastomose to aortic bifurcation or iliac arteries

Adjuncts: Partial left heart bypass to reperfuse distal aorta/femorals (maintain renal and visceral perfusion); selective catheterisation of visceral branches

Paraplegia (20%) increased in proximal aneurysms with lengthy clamp time, renal impairment, age, emergency (caused by: division of arteries to cord, prolonged ischaemia, reperfusion injury, post-op hypotension) and maintenance of perfusion by reimplantation of spinal cord arteries, distal aortic perfusion, CSF drainage

Hybrid visceral revascularization and endovascular repair:
Transperitoneal retrograde visceral revascularisation (maintains visceral and renal perfusion) creates adequate distal landing zone for endograft exclusion; may combine with great vessel transposition or bypass to create adequate proximal landing site

TOTAL ENDOVASCULAR REPAIR
Fenestrated endografts allow target vessel cannulation with additional covered stents → antegrade visceral and renal perfusion; also have branched endografts to achieve this
Anatomical constraints and tortuosity limit usefulness of TEVR

THORACIC DISSECTION

Acute aortic syndrome= group of 3 pathological conditions of thoracic aorta causing severe thoracic pain

1. Aortic dissection 2. Intramural haematoma 3. Penetrating aortic ulcers

Aortic dissection:

Tear in intima allows blood to penetrate and propagate cleavage plane within media

Two channels of flow: true and false lumens; pressure in false lumen is higher so true lumen compressed

90% have secondary tear allowing re-entry into true lumen

(a)True lumen compression →malperfusion (dynamic)

(b)Rupture of false lumen

(c)Malperfusion: static (dissection excludes vessel from flow) and dynamic (compression of true lumen)

Aetiology: HTN, trauma, connective tissue disease, inflammatory diseases atherosclerosis, other (cocaine, pregnancy etc)

Features: Pain (chest/interscapular), syncope, ischaemia (visceral, renal , limb, spinal cord), pulse deficits

Complications: aneurysm, rupture, coronary dissection, aortic valve involvement, tamponade, malperfusion, pseudoaneurysm

Intramural haematoma:

Blood clot in intramural space without obvious intimal tear (?rupture of medial vasa vasora)

Precursor to dissection (40% dissect, account for 20% of dissections)

Similar clinical features, classification and management to aortic dissection

Penetrating aortic ulcer:

Ulceration of atherosclerotic plaque

Classifying dissections

Site: Stanford and DeBakey systems

Chronicity: (i)acute <2 weeks after onset of symptoms (ii)subacute 2wks to 3mths (iii)chronic >3 mths

Presentation: (i)complicated (ii)uncomplicated

Stanford A=ascending and descending (70%) and B=descending only; starts distal to left subclavian ostium (30%)

DeBakey: I= ascending and descending (10%), II=ascending (60%), III=descending= 30%

MANAGEMENT

Rapid control of BP and d*P*/d*t* (100-120mmHg systolic and HR <60) to reduce left ventricular ejection force and limit wall stress

B-blockers (metoprolol/esmolol) + GTN if necessary

Type A: Emergency surgical graft repair +/- aortic valve replacement

1% mortality per hour from rupture. aortic regurg, pericardial tamponade, coronary ischaemia

Type B:Medical with intervention for complications

Mortality 10%

EVAR

(a)Cover entry tear to depressurise FL → TL expands, relieves dynamic malperfusion + eliminate rupture

(b)Stents for static malperfusion

Mortality 10% with paraplegia 3% so now first-line for Type B in vascular centres

OPEN REPAIR

High mortality 25-50% with high complication rate

CHRONIC DISSECTIONS

Treat at size 5.5-6.0cm or symptom development

OUTCOMES

Open repair: mortality 5-16%; paraplegia 4-11%

PERIPHERAL ANEURYSMS: ILIAC

Usually in association with AAA
Large (4-8cm) CIA/IIA(EIA extremely rare)
Intervention: (i)asymptomatics 3-4cm (ii)symptoms

PERIPHERAL ANEURYSMS: POPLITEAL

Commonest peripheral aneurysm (80% of all) 50% have AAA
50% bilateral, two thirds symptomatics
Complications: (1) ischaemia (2)rupture (3) thrombosis (4)embolism(trash foot due to flex/ext disrupting thrombus) (5)compression
Indications for intervention: (i)2cm (ii)complications (iii)mural thrombus

PERIPHERAL ANSUERYSMS: COMMON FEMORAL

TRUE
Second commonest peripheral after popliteal; 85% of AAA
Embolic phenomena/ischaemia (thrombosis rare)
Intervene: (i)symptoms (ii)size 3cm+

FALSE (pseudoaneurysms)
Trauma (femoral access/IVDU)
Commonest infected aneursyms seen in practice
Features: pulsatile groin mass (tender if infected), compressive symptoms (nerve, vein), ischaemia
Management (i)Small/asymptomatics= conservative(usually thrombose in 2-4 weeks)
(ii)Size/symptoms: compression/thrombin/resection/graft recon if non-IVDU only

UPPER LIMB ANEURYSMS

Subclavian: thoracic outlet compression (ischaemia and steal, embolism, acute thrombosis) *resembles unilateral Raynaud's*
Aberrant right subclavian artery from descending thoracic aorta - can compress oesophagus against trachea (dysphagia lusoria)
-aneurysmal degeneration (Kommerell's diverticulum)

UPPER ARM

Axillary artery: trauma (anterior shoulder dislocation, humeral head fracture)
Pseudoaneurysm compresses brachial plexus
Management: resected with rLSV graft (deltipectoral incision); stents compromised by compression between R1 and clavicle

LOWER ARM AND HAND

Ulnar artery aneurysm: "hypothenar hammer hand" due to repetitive hand trauma in labourers
Ulnar artery vulnerable to trauma between distal margin of Guyon's canal and palmar aponeurosis, lying anterior to hook of hamate with only palmaris brevis as cover
Thrombosis with embolisation to fingers 4 and 5 → cold, cyanotic digits (thumb spared)
Management: calcium channel blockers and avoiding hand trauma; arterial recon +/- thrombolysis

VISCERAL ANEURYSMS

True: atherosclerosis, HTN, infective || False: trauma, inflammation (eg pancreatitis)

1. SPLENIC: portal hypertension/pregnancy; treat at 2cm/symptoms/pregnant/women of CBA (ligate/embolise)
2. COMMON HEPATIC: Quicke's triad= ; treat at 2cm (stent/resect)
3. SMA mycotic/degenerative; most symptomatic; treat all sizes (stent/recon)
4. COELIAC: syphilitic; most symptomatic; treat at 1.5cm (stent)
5. OTHERS

CAROTID ANEURYSM

Diameter >150% of CCA or twice diameter of distal ICA
Unknown aetiology
Features: pulsatile swelling with or without pain, Horner's syndrome, thrombosis, dissection, rupture, embolization (TIA/stroke)
Treatment: resect/bypass in proximal ICA; stent distal ICA