Aortic disease

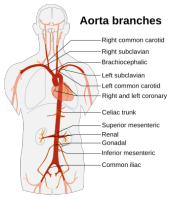
Friday, 20 June 2025 12:07

Types of aortic diseases:
- Stenosis - Narrowing
- Thrombosis - clot
- Embolus - travelling clot
- Dissection - tearing
- Aneurysm - swelling

Aorta anatomy:

Layers:
- Tunica intima
- Tunica media
- Tunica adventitia

Branches:
- Thoracic: ABCS
  - Brachiocephalic
    - Right subclavian
    - Right common carotid
  - Left common carotid
  - Left subclavian
- Abdominal:
  - Inferior phrenic (dorsal)
    - Superior suprarenal
  - Middle suprarenal
  - **Coeliac (ventral)**
  - **SMA (ventral)**
  - Renal
    - Inferior suprarenal
  - Gonadal
  - **IMA (ventral)**
  - Left and right common iliacs



Aortic dissection:
- 2 classification methods:
  - Stanford, or BeBakey

Stanford (A/B):
- Type **A**: **A**scending aorta
- Type **B**: **B**elow/descending aorta

DeBakey: BAD
- Type I: **B**oth (asc + desc)
- Type II: **A**scending
- Type III: **D**escending

Diagnosis:
- Clinical:
  - Chest pain radiating to back (usually between shoulder blades)
  - Different pulse pressures (BP both arms)
    - Different blood flow to the left vs right subclavian arteries
  - Hypotension (bleeding)
- Imaging:
  - CT aortogram (angio the aorta) - only if pt stable to do so
    - Angiogram = contrast to visualise blood vessels

Primary survey:

A - Airway: Is it patent?

B - Breathing: RATES
- R - Rate
- A - Auscultation
- T - Trachea - Midline?
- E - Exertion
- S - Saturation (O2) and symmetry

C - Circulation
- BP, HR
- Radial-Radial delay
- IV access

D - Disability
- GCS, drugs, disability, diabetes

E - Exposure
- Wounds, temp., etc

Chest pain protocol:
- ECG
  - May be normal, or show non-specific changes
  - LVH and strain (d/t long standing HTN)
- CXR
- Troponin (rare elevation in dissection)

Other investigations:
- FBP
- UECs + glucose
- Coags
- D-dimer - elevated in dissection

Predisposing factors:
- HTN
- Age
- Connective tissue disease e.g. Marfans
- Autoimmune vasculitis
- Pregnancy
- Aortic valve disease
- Aneurysms
- Iatrogenic causes e.g. catheterisation
- Cocaine/methaphetamine use

Ddx:
- Acute coronary syndrome
- PE
- Pericarditis/myocarditis
- Oesophageal rupture
- Pancreatitis

Complications:
- Rupture
- Aortic branch occlusion

Management:
- If stable, conservative management
  - Stanford B/DeBakey 3
  - High rate of morality with ascending aorta

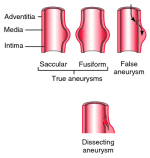
1. ABCs and large bore IV access
2. Monitoring - Pulse ox + ECG continuous, arterial line if possible
3. Hypotension
   - Most pts with AD have HTN. If hypotensive = bad
   - IV fluids
4. Analgesia - IV opioids
5. Beta blockers - ESSENTIAL - started as soon as diagnosis is made
6. Vasodilators - GTN - only after BB's
7. Surgery or EVAR

Aortic aneurysm:

Definitions:
- Arteriomegaly: Diffuse enlargement of an artery
- Ectasia: Diffuse or focal dilation
  - Increased diameter of >50% of normal
- Aneurysm: Focal dilation
  - Increased dimeter of >50% of normal
- Types:
  - Ascending - cardiothoracic surgery
  - Descending - vascular surgery
    - Thoracic
    - Abdominal

True vs false aneurysm:
- True aneurysm: Incorporates all 3 layers of the vessel wall
- False aneurysm AKA pseudoaneurysm: Usually d/t trauma

Types of aneurysms:



Common locations:
- Aorto-iliac: 40%
- Femoro-popliteal: 25%
  - If presence of popliteal aneurysm, then 50% chance of contralateral popliteal artery aneurysm
  - AAAs are associated with popliteal aneurysms

Presentation:
- Asymptomatic - 80%
- Symptoms:
  - Abdo pain
  - Flank pain
  - Back pain
  - Abrupt onset of pain = rupture or acute expansion of aneurysm

Diagnosis:
- Usually incidental finding
- Physical exam
  - Abdominal
    - Pulsatile and expansile mass in epigastrium
    - Assess for tenderness
    - Iliac fossa pulses
  - Lower limb
    - Femoral pulse
    - Popliteal pulses, feel for aneurysm
- Imaging:
- US:
  - Pros:
    - Easy diagnosis
    - Accurate measurement of infra-renal diameter
    - Easy, accessible, cheap
    - No radiation
    - Non-invasive
  - Cons:
    - Cannot visualise thoracic or supra-renal segment
    - Can't establish relationship with renal arteries
    - Operator-dependent
- CT angiogram:
  - Pros:
    - Reliable and reproducible
    - Image entire aorta
    - Detailed anatomy
  - Cons:
    - Contrast use
    - Radiation
    - More expensive

Risk factors:
- Smoker (10x)
- Male (4x)
- HTN - 40% of pts with AAA have HTN
- Carotid artery stenosis - 10% have AAA

Common aetiology:

Degenerative: Most common
- ↓ in elastin and collagen in media
- Mild inflammatory response
- Increase in collagenase and elastase activity = further degradation of strength of artery wall
  - Aging
  - Smoking
  - HTN
  - Hypercholesterolaemia - atherosclerosis
  - Connective tissue disorders - Marfan's, Ehlers-danlos syndrome

Mycotic (infective):
- 3% of aneurysms
- Destruction of media and intima
- S.aureus, E.coli and Klebsiella species

Inflammatory:
- Auto-immune process, vasculitis

Less common aetiology:
- Dissection
- Cystic medial necrosis - Familial thoracic aortic aneurysm - Autosomal dominant
- Ehlers-danlos
- Syphilis

Aneurysm physics:

Laplace's law: T = P X R
- T - tension
- P - pressure
- R - radius

Prognosis:

Complications of AAA:
- Thrombosis
- Distal embolisation
- Rupture
  - Pre-arrival mortality 50%
  - Mortality for open repair of AAA - 43%
  - Mortality for EVAR repair - 36%
  - Elective repair mortality 1-3%

Indications for Tx:
- Infra-renal diameter >5cm - consider for repair
  - >4cm needs referral to avascular surgeon for surveillance
- If yearly enlargement is >10% per year
- Acute symptoms
- Repair threshold dependent on comorbidity

AAA open repair:
- Replaces the diseased aneurysmal aorta with synthetic material
- Tube or trouser graft
  - Pros:
    - Durable repair
    - Less risk of long-term complications
  - Cons:
    - Longer hospital stay (7+ days)
    - Cardiac and renal complications
    - Spinal cord and lower limb ischaemia

AAA endovascular repair:
- Percutaneous access via both groins
- Graft inserted intraluminally below renal arteries
- Sealed with aorta and common iliac arteries - trouser graft
  - Pros:
    - 2 day stay in hospital
    - Less invasive
  - Cons:
    - Higher risk of long term complications
    - 10% of pts require further procedures
    - Life long monitoring with US or CT if EVAR

COPD

Saturday, 20 September 2025 21:36

Obstructive lung diseases:
- COPD
- Asthma
- Bronchiectasis
- Chronic bronchiolitis

Obstructive diseases are airway diseases. Restrictive diseases are parenchymal diseases.

Chronic obstructive pulmonary disease:
Obstructive lung disease (incompletely reversible) that is progressive. Includes emphysema and chronic bronchitis.
Causes:
- Smoking
- Air pollution
- A1-antitrypsin deficiency causing protease mediated damage

Emphysema:
Enlarged air spaces and destruction of walls, loss of elasticity. Leads to airway collapse on exhalation.

Chronic bronchitis:
A COPD characterised by chronic productive cough (>3 months in 2 years). Mucous causes airflow impediment and irritation.
Causes:
- Smoking
- Recurrent infections
- Pollutants

Stasis of mucous leads to recurrent infections

Emphysema = Type A "Pink puffers"	**Chronic bronchitis = Type B "Blue bloaters"**
Cough develops later	Early cough
Less common infections	Frequent infections
Severe and early SOB	Mild, late SOB
Little sputum	Copious sputum
Low elastic recoil	Normal elastic recoil
Hyperinflated chest/barrel chest	Hyperinflated chest
Can maintain their PaO2	CO2 retention
Low body mass	Overweight

COPD Hx:
- Onset of dyspnoea
  - Gradual
  - Episodic exacerbation (usually infective)
- Severity
  - Compared to baseline
  - SOB at metres, ADLs, speaking, rest
- Associated symptoms
  - Persistent cough, usually worse in AM with mucoid sputum (clear/white)
  - Chest tightness
  - Wheezing
  - Fatigue
  - Poor appetite
  - Weight loss
- High risk features
  - Frequent exacerbation
  - Previous hospitalisation
  - ICU admission
  - Oral steroid use
- Exacerbating factors
  - Exercise
  - Altitude
  - Smoking
  - Respiratory infections

Examination findings:
- "Barrel chest"
- Hyper-resonant percussion sounds
- Reduced air movement
- Crackles
  - Over production of mucous
- Reduced chest expansion
- Prolonged expiratory phase

Signs of respiratory distress in severe disease:
- Tachypnoea
- Accessory muscle use
- Pursed-lip breathing
- Tracheal tug

Pathophysiology of COPD:
- Increased cholinergic bronchomotor tone
- Muscarinic cholinergic receptors cause bronchoconstriction and mucous hypersecretion
  - Vagal innervation to larger airways
  - Small airways contain muscarinic receptors

Exacerbation of COPD:
Any change in:
- Dyspnoea
- Cough
- Sputum

Beyond day to day variation and acute in onset

Diagnosis:
- Hx + exam
- Spirometry - FEV1
- Arterial blood gas (ABG) analysis - O2 and CO2 levels
- Alpha-1 antitrypsin testing

Tx:
1. B2 adrenoreceptor agonists (B2 AR):

Directly dilate airway smooth muscle
- Short acting B2 AR (SABA):
  - Salbutamol, Terbutaline
  - Rapid onset: 5-10 mins
  - Short acting: 4-6 hrs
- Long acting B2 AR (LABA):
  - Salmeterol, Formoterol
  - Delayed onset: 15-20 mins
  - Long acting: 12 hours

Side effects of B2 AR agonists:
- Tachycardia (action on heart B1)
- Muscle tremor (Skeletal B2 AR)
- Restlessness
- Hypokalaemia
- Hypoxaemia

2. Muscarinic cholinergic receptor antagonists

Ach causes bronchoconstriction - inhibits this effect - Inhibits vagal mediated airway tone
Greater effect in COPD than asthma due to affect on vagal tone
- Short acting muscarinic antagonists (SAMA):
  - Ipratropium
  - Onset: 20-30 mins
  - Effect: 4-8hrs
- Long acting muscarinic antagonists (LAMA):
  - Tiotropium
  - 1-2x daily dosing

- Start with short acting relivers: SAMA better than SABA
- Add long term bronchodilators: LAMA or LABA

3. Inhaled corticosteroids
   - Reduce airway inflammation, swelling and mucous production (suppress the immune system and inhibit inflammatory cells)
     - Beclomethasone, Fluticasone, Cyclosonide
     - Reduced pro-inflammatory proteins + increases anti-inflammatory proteins

Non-pharmacological:
- Stop smoking
- Influenza vaccine
- Exercise
- Treat comorbidities: CVD, lung cancer, anxiety, osteoporosis
- Pulmonary rehabilitation: Exercise, nutrition
- O2 therapy in severe hypoxaemia
  - Aim for 88-92% sats (treat if <88%)
  - Why don't you always give O2?
    - Giving O2 causes CO2 rises = drowsiness = risk of aspiration
- Positive airway pressure in exacerbations

Non-pharmacological management:
- Physiotherapy
- Encourage sitting up
- Encourage to stop smoking
- Maintain adequate nutrition
- Regular physical activity

Chronic kidney disease (CKD)

Sunday, 12 October 2025 14:34

CKD:
A decline in kidney function - At least 3 months of reduced eGFR (<60 mL/minute/1.73m^2) OR
Evidence of kidney damage with or without reduced eGFR for >3 months with evidence of:
- Albuminuria
- Haematuria (after exclusion of urological causes)
- Structural abnormalities
- Pathological abnormalities (e.g. on renal biopsy)

Presentation = Non-specific:
- Lethargy
- Nocturia
- Nausea
- Pruritus
- Restless legs
- Dyspnoea
- Haematuria (usually microscopic)
- HTN

Signs of ESRF:
- Pruritus
- Electrolyte imbalances
- Metabolic acidosis (loss of HCO3-)
- Severe nausea
- Neurological impairment (renal/uraemic encephalopathy)

Clinical stages of CKD:

eGFR	Description	Symptoms
Normal >90	Stage 1: Below normal to mild loss of kidney function	Often no Sx
Mild 60-89	Stage 2: Mild to moderate loss of kidney function	- HTN - Protein in urine
Moderate 30-59	Stage 3: Moderate to severe loss of kidney function	- Anaemia - Early bone disease
Severe 15-29	Stage 4: Severe loss of kidney function	- Fatigue - Swelling - N+V
ESRF <15	Stage 5: Kidney failure: Dialysis/transplant	- Kidney failure

3 categories of CKD:
- Glomerular (glomerulonephritis)
  - Nephritic syndromes
  - Nephrotic syndromes
- Tubulo-interstitial
  - Acute tubular necrosis
  - Acute and chronic tubulointerstitial nephritis
  - Chronic pyelonephritis
  - Myeloma kidney
- Vascular
  - Benign hypertensive nephrosclerosis
  - Malignant hypertensive nephrosclerosis
  - Renal artery stenosis

Risk factors:
- Obesity (BMI >30)
- Diabetes
- HTN
- CVD (HF, MI, stroke)
- FHx
- Alcohol
- >60yo
- ATSI

Complications of CKD:
- Coronary artery disease
- Metabolic acidosis
- Fluid retention
- Hyperkalaemia
- Increased infection risk
- Anaemia
  - D/t reduced renal output of EPO
- Bone mineral disease (less active Vit D (calcitriol)
- Uraemia

4 top causes of CKD:
- Diabetic nephropathy - most common
- Chronic glomerulonephritis
- Hypertensive nephropathy
- Polycystic kidney disease (autosomal dominant)

How to distinguish between these 4 causes:
Urine + ultrasound. Why these 2 tests?
- Proteinuria occurs before a reduction in kidney function = 1st sign of diabetic nephropathy

	Urine	US
Diabetic nephropathy	Protein	Normal or large kidneys
Glomerulonephritis	Blood + protein	Small shrunken kidneys
Hypertensive nephropathy	Normal	Small, asymmetrical kidneys
ADPKD	Normal	Diagnostic for this condition

Does urine volume help?
- No reduction until kidney function is <1-2%
- Can still make 15L of urine per day with 10% renal function

3 prognostic markers of CKD:
ABC
- A: ACR = Albumin : Creatinine ratio (proteinuria)
- B: BP - HTN
- C: Creatinine/eGFR

How to calculate eGFR:
- Age
- Gender
- Serum creatinine

What the presence of both protein and blood in the urine means:
- Must be a glomerulonephritis
- Unless it's a diabetic (proteinuria) with a UTI

Management - How to prevent progressive renal failure:
1. Reduce proteinuria - ACE-I
2. Good BP control
3. Good glucose control
4. SGLT-2 inhibitors
5. GLP-1 agonists (if diabetic nephropathy)