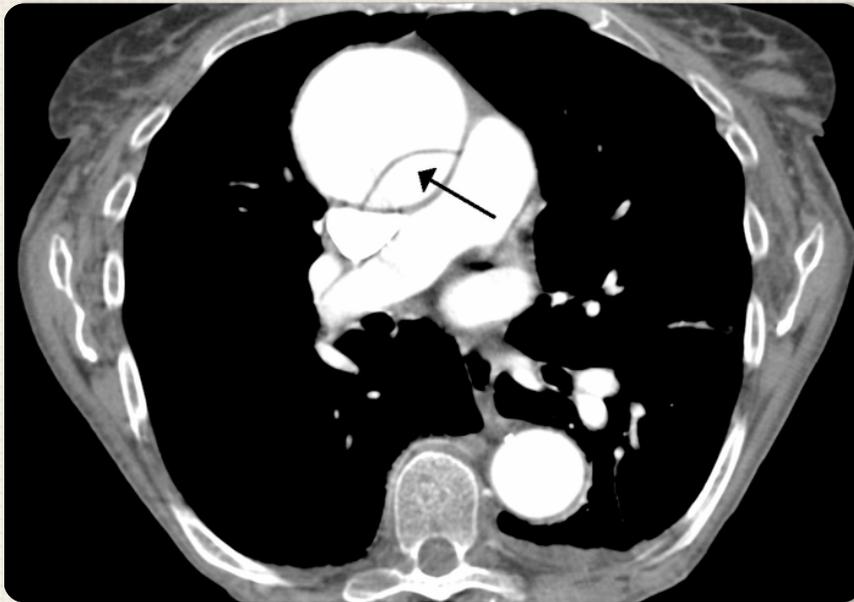


# EMERGENCY MEDICINE CASES



EPISODE 28: VASCULAR  
EMERGENCIES WITH DR. ANIL  
CHOPRA  
& DR. DAVID CARR

## Clues to diagnosis of Aortic Dissection:

**Aortic dissection** is an uncommon diagnosis with high mortality, and often difficult to identify.

**The Classic presentation:** acute, “tearing” or “ripping” chest pain reaching maximal intensity at onset, radiating to back and/or between shoulder blades. Only 25% of patients have the triad of this pain, widened mediastinum, and pulse deficit. The most common descriptor of the pain is “sharp”, ~5% presented painlessly (1), and 10% with syncope (IRAD(2)).

### Risk factors for Dissection:

Hypertension, age, male gender, family history, recent deceleration injury (i.e. MVC with airbag), prior cardiac surgery, known preexisting aortic aneurysm, recent cardiac cath.

In younger patients (<40): cocaine or amphetamine use, pregnancy, connective tissue diseases (e.g. Marfan Syndrome), congenital heart disease, bicuspid aortic valve (9x risk), and weight lifting.

### 3 Important Questions:

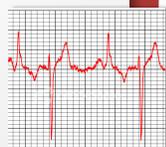
- *quality* of pain (most commonly “sharp” but highest LR for “tearing”)
- pain intensity *at onset*
- radiation of pain (back and/or belly)

90% of the ED docs suspected dissection even before investigations were done if all 3 of these questions were asked (1).

**“Chest pain Plus...”** CP + focal neuro deficit or pain below diaphragm or limb ischemia: **think dissection!**

### WHAT TO LOOK FOR ON THE ECG

First: exclude other causes of chest pain such as MI! ECG abnormalities are common (~70% in IRAD database) (2), but abnormalities include non-specific ST changes, LVH, infarction (i.e. inferior territory from dissection of right coronary artery), or other ischemic changes. Ischemic ECG changes are either due to chronic coronary disease or extension of dissection into RCA.



### Pitfalls of ECGs, troponin, and D-dimer:

An ECG showing ischemic changes or a slightly elevated troponin may lead to assumption that pain is due to cardiac ischemia, and a positive D-dimer may lead to assumption that the patient has a PE. Treating PE or ACS equates to “a clean kill” for a patient who has a dissection!

Although D-dimer will usually be positive in nearly all patients with dissection, it cannot be used as a rule-out test as low levels have been found in younger patients, or patients with a thrombosed “false lumen”. Both D-dimer and troponin could be normal in patients presenting early with dissection.

# Aortic Dissection continued:

## Physical examination:

Carefully check for diastolic murmur of aortic regurgitation (retrograde dissection), signs of Marfan syndrome (pectus excavatum, 'gangly' appearance), and pulse deficit in radials and femoral arteries.

**What about BP?** Many patients have a difference in blood pressure between arms normally, so a BP difference does not rule in dissection; nor does a lack thereof rule out dissection. However, a BP difference may heighten your suspicion of dissection in the right clinical context.

## Chest X-Ray Findings:

Although 1/3 of patients have a normal chest x-ray, the 2 most important abnormalities are a widened mediastinum and the "calcium sign".



*Calcium Sign: separation of the outermost portion of the aorta from the calcified intima by >5mm.*

## Other possible findings on

**CXR:** loss of aortic knob, trachea displaced to right, left mainstream bronchus displaced downward, disparity of ascending and descending aorta caliber, apical capping, pleural effusion (usually left), localized bulge in the aorta (3).

Remember to compare with previous CXR if available!

## What about on bedside

**ultrasound:** Check for a pericardial effusion, which occurs from dissection into the pericardial space. If an effusion is seen, management is similar to other scenarios for tamponade (drain only if unstable). Although sensitivity is poor for dissection, check if the abdominal aorta appears normal.

## What imaging test to get?

Stable Patient: CT scan with arterial contrast.

Unstable (or unable to receive IV contrast): transesophageal echo (TEE) which is equally sensitive to CT with arterial contrast (4)

## ED Treatment of Dissection

**Aggressive BP & HR control to decrease stress on the aortic wall!! Goal SBP 110-120, HR 60**

1st Line: short acting B-blocker such as esmolol or labetolol

2nd line: nitroprusside (0.25-1.0mcg/kg/min) but only in addition to B-blocker (to avoid reflex tachycardia!)

**Consult a surgeon!** All type A dissections require urgent surgery! While most type B dissections can be treated medically, *certain* type B dissections require urgent surgery (for ongoing pain, expanding diameter, evidence of aortic rupture) => consult surgery early *in all cases*.

**Time is key:** type A (aortic arch involved) kill 1-2% of patients per hour. Type B (arch not involved) fare better.



## ACUTE LIMB ISCHEMIA

### Major causes include:

- acute thrombosis (i.e. arterial thrombus, or thrombosed graft)
- embolism (Afib, prosthetic valve)
- aortic dissection to limb
- DVT causing limb ischemia by arterial vasospasm (*phlegmasia cerulea dolens*)
- compartment syndrome

Other rare causes: inflammatory arteritis, vasculopathies, HIV arteritis.

### Clinical Signs (6Ps):

pain,  
pallor,  
parasthesias,  
pulselessness,  
"poikilothermia," and  
paralysis.

*However, these signs are not always present! Pain is the earliest, and may be the only sign. Paresthesias are often the earliest and only physical exam finding. Test 2 point discrimination (more sensitive than light touch). Ask about pain association with activity or position, and whether the limb feels "different" to the patient. Pulse absence cannot distinguish acute from chronic ischemia, so look for unilateral ischemic skin changes (skin necrosis, blistering, mottling) which would raise clinical suspicion for an chronic ischemia.*

# Acute Limb Ischemia continued:

**Blue Toe Syndrome:** Painful cyanotic discoloration of portions of the foot, caused by micro-embolic showers from proximal source. Although pulses are preserved, this is managed as an acute occlusive condition.

## Who goes to the OR?

True acute limb ischemia should get angiography *in the OR* and definitive management. Arterial dopplers may be done for stable patients with a history of claudication.

A rough rule of thumb for surgical indication: loss of light touch.

## Treatment of limb ischemia:

Time is limb! Salvage time depends on collaterals, and patient-specific factors. If sensory loss to light touch is minimal, viability may be excellent, but ischemia can occur within hours.

**Acute Treatment:** ASA, UFH 80U/kg bolus, then 18U/kg/h\*, ample pain meds, and an **urgent surgical consult** for endovascular or surgical revascularization.

*\*UFH may inhibit clot propagation and further distal thrombosis, but no established benefit in literature.*

## What about thrombolysis?

While not superior to surgery, it may be the treatment of choice in patients with occluded grafts, collaterals and chronic insufficiency, or for occlusions of small, inaccessible arteries.

Sending patients home with non-critical ischemia? Remember to help optimize comorbidities, and review anti-platelet agents: ASA (plus clopidogrel in refractory patients) to improve outcomes.



## COMPARTMENT SYNDROME

**Definition:** Increased pressure within a limited space which compromises tissue function.

Most commonly from fractures (*including open fractures*), but also soft tissue injury, reperfusion injury, minor trauma, major burns, and limb compression in pts “found down”. Compartment syndrome can occur in leg, arm, hand, or abdomen.

### Key clinical features

**Increasing Pain out of Proportion** (intractable severe pain, usually “aching”)

**Altered Sensation**

check loss of light touch / 2pt

**Pain on Passive Stretch**

**Muscle weakness** (late finding)

**Tenderness and swelling in the compartment** (can feel woody, tense)

*\*\*pulses are usually still present, and the limb oxygen saturation is often preserved.\*\**

### How to Examine and Stretch Compartments of Lower Leg:

Anterior: pain with passive ankle plantar-flexion, +/- weak ankle dorsiflexion, and loss of sensation between first two toes

Lateral: pain with passive ankle inversion, +/- weak ankle eversion, and similar findings to anterior compartment  
Superficial posterior: pain with passive ankle dorsiflexion

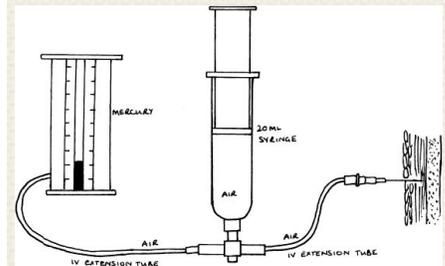
Deep posterior (difficult to palpate): pain with passive toe extension, weak toe flexion

**Do serial physical exams and call a surgeon if you are suspicious!**

Consider measuring a compartment pressure if unsure of diagnosis (see left).

## MEASURING COMPARTMENT PRESSURES

Compartment pressure measurement can help confirm Compartment Syndrome, but the Dx can often be made clinically alone. The needle/tubing/manometer technique can be done with readily available ED



equipment. **\*\*Measure pressures as close to the fracture or injury site as possible. If unsure, call the surgeon to assist in the diagnosis!**

**While awaiting for definitive management by fasciotomy:** remove constrictive dressings, elevate limb to the *level of heart*, keep blood pressure adequate (fluids for hypotensive patients!), give oxygen, and relieve pain.

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- 3) Marx et al. Rosen's Emergency Medicine: Concepts and Clinical Practice, 6th ed. 2006.
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