2) Acute Arterial Disorders – Dr. Amanj

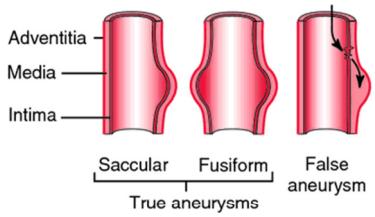
Abdominal Aortic Aneurysm

Types of Aneurysms/ Layers

- **True**: involves all 3 layers of the arterial wall
- **False (pseudo aneurysm)**: presence of blood flow outside of normal layers of arterial wall. Wall of false aneurysm is composed of the compressed, surrounding tissues.
- **Dissecting**: tear in the intima, blood goes to the space between intima and media.

Types of Aneurysms/Etiology

- **Degenerative**- complex process that involves some degree of calcification, atherosclerosis.
- **Inflammatory**- thick inflammatory wall with fibrotic process; Takayasu's, Giant cell arteritis, Polyarteritis nodosa, Behcet's.
- **Traumatic**- false aneurysms
- **Developmental Anomalies**. aberrant right subclavian artery.
- **Infectious**- Can be primary or secondary infections.
- **Congenital**- Tuberous sclerosis, aortic coarctation, Marfan's.



Types of Aneurysms/Shapes

- Fusiform
- Sacular

Background

- Aneurysm: Swelling/dilation
- 1 - 5 % of general population affected
- Incidence is increasing
- Smoking increases risk
- 100,000 – 250,000 new cases discovered each year in the U.S.
- Natural history is to enlarge & rupture unless death occurs from other cause
- Rupture carries a 90% mortality
- Prevalence
    - 7.5 % of men older than 65
    - 1.3 % of women older than 65

Aortic Aneurysms

- Thoracic (19%)
- Thoracoabdominal (2%)
- Abdominal (AAA) (78%)
    - Infrarenal (95% of all AAA)
    - Juxtarenal
    - Pararenal
- False aneurysms (pseudoaneurysms)
    - Traumatic
    - Anastomotic

Pathophysiology

- Degredation of tuncia media
- Destroyed elastin making aortic wall more susceptible to change in BP
- Abdominal aorta contains less elastin compared with the thoractic aorta; higher chance in abdomen for aneurysm
- Age causes decline in elastin

Risk Factors

- Smoking history
- Family history
- Older age (per 7 years interval)
- CAD
- High cholesterol
- COPD
- Height (per 7 cm interval)
- DVT history
- Diabetes
- Black race
- Female gender
- Hypertension

Symptoms

- Often asymptomatic
- Can manifest as back, abdomen, groin pain
- Palpable abdominal mass upon examination

Rupture Risk

- Diameter (> 6 cm)
- Expansion (> 0.6 cm/year)
- Smoking/COPD
- Family history
- Hypertension
- Gender (Female)
- Shape (Fusiform < Saccular)

Size Predicts Rupture

Size (cm)	Annual rupture risk (%)
< 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

Diagnosis

- Physical examination
- Ultrasound (size, follow up)
- CT (size, thrombus inside)
- MRI
- Angiography used less often

Treatment

- Conservative:
    - Surgery more risky
    - Lifestyle changes: cholesterol meds, stop smoking, etc.
- Surgical:
    - Endovascular stent insertion EVAR
    - Open surgery

Medical Management

- **Smoking Cessation**- Single most important modifiable risk factor
- **Exercise Therapy**- Evidence suggests may benefit small aneurysms
- **Beta Blockers**- May decrease the rate of expansion.
- **ACE inhibitors**- Implicated in less aneurysm rupture.
- **Doxycycline**- Against chlamydia species
- **Statins**- associated with reduced aneurysm expansion rates.

Prognosis

- Mortality 40% for ruptured AAA
- Pre-rupture surgery: 1-6%

Open Repair-Complications

- Cardiovascular (MI, Stroke)
- Pulmonary (pneumonia)
- Renal (impairment)
- Lower Extremity Ischemia
- Spinal Cord Ischemia
- Incisional Hernia (14.2%)
- Graft Infection

Vascular Trauma

Basic Principles

- Anatomy
- Types of Injury
- Mechanisms of Injury
- Clinical Manifestations
- Clinical Evaluation
- Investigations
- Management

Anatomy

- Know the named vessels – arterial and venous – in the vicinity of injury
- Know anatomic principles of proximal and distal control
- Appreciate the adjacent structures (nerves, organs etc)

Types of Injury

- Laceration
- Transection; With or without defect
- Dissection
- Crush
- Thrombosis / Embolus
- Spasm

Mechanisms of Injury

- Penetrating
    - Knife
    - GSW/ Shrapnel (low/high velocity)
    - Catheter (Iatrogenic)
- Blunt
    - Direct (Contusion)
    - Traction / Avulsion
    - Deceleration
    - Torsion

Clinical Manifestations

- Early
    - Hemorrhage
    - End-organ ischemia
    - Fistula?
- Late
    - Fistula
    - False Aneurysm

Evaluation

- History and PE
    - Type of weapon
    - Time since injury
    - 5 P's
    - Associated Injuries (Nerve, Bone, …)
    - Climate
    - Age of the patient
- "Hard" Findings
    - Active Bleed
    - Expanding Hematoma
    - End-organ ischemia
    - Loss of pulses
    - A-V fistula
- "Soft" Findings
    - Reduced pulses
    - Neurologic deficits
    - History of bleeding
    - Shock
    - Injury in proximity to major vessel

Investigations

- Plain Films
- Doppler/ Duplex
- Arteriography
- CT
- MRI

Doppler ultrasound

- Determine presence/absence of arterial supply
- Assess adequacy of flow

Angiography

- Locates site of injury
- Characterizes injury
- Defines status of vessels proximal and distal
- May afford therapeutic intervention
- Expensive
- Time-consuming
- Difficult to monitor/treat patient
- Procedural risks
    - Renal burden from dye
    - Possibility of anaphylaxis
    - Injury to proximal vessels

Arteriography

- Recall “hard” vs “soft” findings
- Role
    - Detect occult injury
    - Operative planning
    - Endovascular Repair

Management

- Conservative
- Endovascular
- Operative
    - Local – suture, patch, primary anast
    - Bypass
        - Anatomic, extra-anatomic
        - Autogenous , prosthetic
    - Adjunct
        - Fasciotomies, Fracture fixation

Prognostic factors

- Level and type of vascular injury
- Collateral circulation
- Shock/hypotension
- Tissue damage (crush injury)
- Warm ischemia time
- Patient factors/medical conditions

In general treatment proceeds as follows:

1. Hypovolemia and **shock** should be treated by controlling external blood loss and restoring blood volume.
2. Initiate **antibiotic** and tetanus prophylaxis when indicated, and crossmatched blood.
3. Prepare a wide **operative field** and an uninvolved lower extremity for ready access to the saphenous vein.
4. **Localize the site** of arterial injury by the location of the wound missile track, pulse deficits, or arteriography.
5. The usual **incisions** should be utilized for exposure of blood vessels. **Unnecessary injury** to associated structures is avoided. **Proximal control** is always obtained prior to entering the field of injury.
6. Obtain **distal control** of the artery to minimize the loss of blood by back bleeding. Fogarty or Foley catheters may be inflated to control both proximally and distally.
7. **Inspect or palpate** the site of injury and determine the need for repair.
8. Proximal and distal control should be obtained at least **2 to 3 cm**. from the site of injury so that the intima can be examined.
9. Remove the proximal **clot** by flushing and the distal clot by milking the vessel, squeezing the distal limb, or passing a Fogarty catheter.
10. Systemic **heparinization** is usually not used in multiply injured patient; local heparinization with dilute heparinized saline (100 units per ml.) is enough to discourage local clot formation.
11. Prior to anastomosis, determine the **need for graft** replacement by estimating the amount of difficulty in approximating the severed ends. In general, **1 to 2 cm**. of artery wall may be resected without graft replacement.
12. Repair the injured artery using interrupted or continuous fine **monofilament sutures**.
13. Reversed **saphenous vein** is the graft material of choice. Prosthetic graft material is avoided if possible because of risk of infection.
14. **De-airing** should be done prior to completion of the repair,
15. **Pulses** in the distal extremity should be palpable.
16. The wound should be thoroughly **debrided** and explored and other injuries should be identified and repaired as necessary. Every effort is made to cover the repaired artery with viable muscle and fascia. **Skin closure** is dictated by the nature of the injury and risk of infection.
17. **Drains** are occasionally employed for a period of 24 to 48 hours.