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 thoracic 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

<table>
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<tr>
<th>Size (cm)</th>
<th>Annual rupture risk (%)</th>
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<tr>
<td>&lt; 3</td>
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<tr>
<td>3 – 3.9</td>
<td>0.4</td>
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<tr>
<td>4 – 4.9</td>
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<td>5 – 5.9</td>
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<td>6 – 6.9</td>
<td>9.4</td>
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<td>7 – 7.9</td>
<td>24</td>
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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 debried 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.