Acute Arterial Disease

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ACUTE ARTERIAL OCCLUSION

“The operation was a success but the patient died”

• High Morbidity and Mortality
  – Emergent operations in high risk patients
  – 20% mortality reported (Dale, JVS 1984)
  – Endovascular approaches may lower peri-procedural mortality while preserving outcomes
Etiology of Arterial Occlusion

• Overview
  – Atherosclerosis
  – Thrombotic occlusion
  – Embolic occlusion
  – Trauma
  – Treatment Options
Evolution of Atherosclerosis

- Areas of low wall shear stress
- Increased endothelial permeability
- Sub-endothelial lipid and macrophage accumulation
- Foam cells
- Formation of Fatty Streak
- Fibrin deposition and stabilizing fibrous cap
Evolution of Atherosclerosis

- Necrosis
- Inflammatory environment
- Destabilization of fibrous cap
Evolution of Atherosclerosis

Rupture of Fibrous Cap
- Pro-thrombotic core
- Exposed to lumen
- Acute thrombosis
- Embolization of plaque materials and thrombus
Thromboembolism

• Embolus - Greek “embolos” means *projectile*
• Mortality of 10-25%
• Mean age increasing – 70 years
  – Rheumatic disease to atherosclerotic disease
• Classified by size or content
  – Macroemboli and microemboli
  – Thrombus, fibrinoplatelet clumps, cholesterol
Macroemboli

- **Cardiac Emboli**
  - Heart source 80-90% of thrombus macroemboli
  - MI, A.fib, Mitral valve, Valvular prosthesis
  - Multiple emboli 10% cases
  - TEE
    - Views left atrial appendage, valves, aortic root
    - Not highly sensitive
Thromboembolism

- 75% of emboli involve axial limb vasculature
- Femoral and Popliteal — >50% of emboli
- Branch sites
- Areas of stenosis
Thromboembolism

Non-cardiac sources

• Aneurysmal (popliteal > abdominal)
• Paradoxical
  – Follows PE with PFO
• TOS
• Cryptogenic –5-10%
• Atheroemboli (artery to artery)
Atheromatous Embolization

- Shaggy Aorta
  - Thoracic or abdominal
- Spontaneous
- Iatrogenic
  - 45% of all atheroemboli
- “Blue toe syndrome”
  - Sudden
  - Painful
  - cyanotic
  - palpable pulses
- livedo reticularis
Blue Toe Syndrome
Atheromatous Embolization

- Risk factors: PVD, HTN, elderly, CAD, recent arterial manipulation
- Emboli consist of thrombus, platelet fibrin material or cholesterol crystals
- Lodge in arteries 100–200 micron diameter
Atheromatous Embolization

- Affect variety of end organs
  - extremities, pelvis, GI, kidney, brain
- Work-up:
  - TEE ascending aorta, CT Angio, Angiography
- Laboratory: CRP elevated, eosinophilia
- Warfarin may destabilize fibrin cap and trigger emboli.
Atheromatous Embolization

- Reported incidence of 0.5-1.5% following catheter manipulation
  - Advance/remove catheters over guidewire
  - Brachial access? – controversial
- Limited Sx– Anti-coagulation/ observation
- Temporal delay up to 8 weeks before renal symptoms
Atheromatous Embolization Therapy

• Prevention and supportive care
  – Statins, prostacyclin analogs (iloprost), ASA, Plavix

• Elimination of embolic source and reestablishing blood flow to heal lesions

• Surgical options: endarterectomy or resection and graft placement
  – Abdominal Aorta – Aorta-bi-fem bypass
  – Ligation of external iliac and extra-anatomic bypass if high risk

• Endovascular therapy
  – Angioplasty & stenting - higher rate of recurrence
  – Athrectomy – no data
Acute Thrombosis

- Graft thrombosis (80%)
  - intimal hyperlasia at distal anastamosis (prosthetic)
  - Retained valve cusp
  - Stenosis at previous site of injury

- Native artery
  - Intra-plaque hemorrhage
  - Hypovolemia
  - Cardiac failure
  - hypercoagable state
  - Trauma
  - Arteritis, popliteal entrapment, adventitial cystic disease
Acute Thrombosis

• Heparin Induced Thrombosis
  • White Clot Syndrome
  • Heparin dependent IgG anti-body against platelet factor 4
  • 3-10 days following heparin contact
  • Dx: thrombosis with > 50% decrease in Platelet count
  • Tx: Direct thrombin inhibitors: Agartroban & Hirudin
    – Avoid all heparin products
• Morbity and Mortality: 7.4-61% and 1.1-23%
Other causes of Thrombosis

- Anti-thrombin III Deficiency
- Protein C & S Deficiency
- Factor V Leiden
- Prothrombin 20210 Polymorphism
- Hyper-homocystinemia
- Lupus Anti-coagulant (anti phospho-lipid syndrome)
“The Cold Leg”

• Clinical Diagnosis
  – Avoid Delay
  – Anti-coagulate immediately
  – Pulse exam
  – 6 P’s (pain, pallor, pulselessness, parathesias, paralysis, poiklothermia)

• Acute –vs- Acute on chronic
  – Collateral circulation preserves tissue
  – Traditional 4-6 hr rule may not apply

• The Two P’s-paralysis and paresthesia
Diagnostic Evaluation

SVS/ISCVS Classification
- “Rutherford Criteria”

• Class I: Viable
  - Pain, No paralysis or sensory loss

• Class 2: Threatened but salvageable
  - 2A: some sensory loss, No paralysis > No immediate threat
  - 2B: Sensory and Motor loss > needs immediate treatment

• Class 3: Non-viable
  - Profound neurologic deficit, absent capillary flow, skin marbling, absent arterial & venous signal
Therapeutic Options

– Class 1 or 2A
  • Anti-coagulation, angiography and elective revascularization

– Class 2B
  • Early angiographic evaluation and intervention
  • Exception: suspected common femoral emboli

– Class 3
  • Amputation
Diagnostic Evaluation

• Modalities
  – Non-invasive:
    • Segmental pressure drop of 30mmhg
    • Waveforms
    • CTA / MRA: avoid nephrotoxicity
      – Center dependent
      – Wave of the future?
  – Contrast Angiography
    • Gold Standard
Thrombotic –vs- Embolic

- **Thrombotic**
  - History
    - Claudication, PVD
    - Bypass graft
  - Physical
    - Hair loss, shiny skin
    - Bi-lateral Dz
  - Angiographic
    - Diffuse disease
    - mid vessel occlusion
  - PVD confuses diagnosis

- **Embolic**
  - History
    - Cardiac events
    - Acute onset
    - Hx of emboli
  - Physical
    - Normal contralateral exam
    - A.fib
  - Angiographic
    - meniscus Cut-off in normal vessel
    - Bifurcations affected

Determination of etiology possible in 85% of cases
Treatment Options

• Multiple options available
  – Conventional surgery
    • embolectomy
    • endarterectomy
    • revascularization
  – Thrombolytic therapy
  – Percutaneous mechanical thrombectomy

• Native vessel thrombosis often require more elaborate operations
The Six P’s

- Pain
- Poikylothermia (Polar)
- Pallor
- Pulseless
- Paresthesia*
- Paralysis*
The Important Two P’s

• Indicate **impending tissue loss** and the need to revascularize now; not in six to eight hours

• **Paresthesia**-loss of nerve function; in the foot the peroneal nerve between 1st and 2nd toe

• **Paralysis**-loss of nerve and muscular function
Treatment Fundamentals

- Early recognition and anti-coagulation
  - Minimizes distal propagation and recurrent emboli

- Modality of Tx depends on:
  - Presumed etiology
  - Location/morphology of lesion
  - Viability of extremity
  - Physiologic state of patient
  - Available vein conduit for bypass grafting
Separate graft thrombosis into early and Late groups

**Early thrombosis**
- Technical defect
- Repairable
- Avoid lytic Tx
  - 14 days vein
  - 30 days graft
- Explore both anastamosis
- On-table Angio
  - Twists, kniks, stenosis

**Late thrombosis**
- Duration & degree of ischemia
- Lytic Thearpy (clas1-2a)
  - Good 1st approach
  - Unmasks lesion (valve/stenosis)
    - F/u endo or open repair
- Open surgery (2b)
  - Thrombectomy/patch
  - Re-bypass
Embolectomy

- Fogarty embolectomy catheter
  - Introduced 1961
- Adherent clot catheter
- Graft thrombectomy catheter
- Thru-lumen catheter
  - Selective placement over wire
  - Administer: lytics, contrast
Embolectomy

Surgical Therapy

• Iliac and femoral embolectomy
  – Common femoral approach
  – Transverse arteriotomy proximal profunda origin
  – Collateral circulation may increase backbleeding
  – Examine thrombus
Embolectomy

• Popliteal embolectomy
  – 49% success rate from femoral approach
  – Blind passage selects peroneal 90%
  – may expose tibial-peroneal trunk & guide catheter
  – Idirectly cannulate distal vessels

• Distal embolectomy
  – Retrograde/antegrade via ankle incisions
  – Frequent Rethrombosis
  – Thrombolytic Tx viable alternative
Embolectomy

• Completion angiography
  – 35% incidence of retained thrombus
  – IVUS more sensitive then angio

• Failure requires
  – Thrombolytic therapy
  – revascularization
Thrombolytic Therapy

Advantages

• Opens collaterals & microcirculation
• Avoids sudden reperfusion
• Reveals underlying stenosis
• Prevent endothelial damage from balloons

Risks

• Hemorrhage
• Stroke
• Renal failure
• Distal emboli transiently worsen ischemia
Surgery –vs- Thrombolysis

• STILE Trial
• Surgery vs Thrombolytics for Ischemia of Lower Extremity
  – 393 pts with non-embolic occlusion
  – Surgery vs r-TPA or r-UK
• Thrombolytics: improved amputation free survival and shorter hospital stay (0-14 days)
• Surgery: revascularization more effective for ischemia of > 14 days duration

Surgery –vs- Thrombolysis

TOPAS Trial
• 2 phase
• 544 patients
• r-UK vs Surgery
• Need for surgery
  Reduced 55%
• Similar amputation and mortality rates

NEJM 338, 4/16/98
Indications for Thrombolysis

Category 1-2a limbs should be considered

- Class 2b: Two schools of thought
  1) “Delay in definitive Tx”
  2) “Thrombolytics extend window of opportunity”

• Clots <14 days most responsive
  - But even chronic thrombus can be lysed

• Large clot burden
  - Better response to lytic tx than surgery
  - Requires longer duration of thrombolytics
Technique of Thrombolysis

• Guide Wire Traversal Test (GTT)
  – Ability to traverse lesion best predictor of success
  – Use 0.035 in angled glide wire
  – “knuckling-over” indicates sub-intimal plane
  – Attempt pro-grade, Anti-grade, lytic bolus
Technique of Thrombolysis

- Catheter directed delivery
  1) Lace clot via catheter with side holes
  2) Pulse-Spray technique (mechanical component)
- Urokinase and TPA equally effective
- 4 hr treatment followed by angiogram
  - 4000IU/min x4hr, 2000Iu/M=min x 48h
    - r-UK (TOPAS Trial)
  - no improvement after 4hr >> surgery
  - Continue Heparin gtt
  - Fibrinogen levels
Mechanical Thrombectomy

• Percutaneous aspiration embolectomy
  – Viable alternative in selected patents
  – Variety of devices
  – Combines diagnostic and therapeutic procedure
  – Removes non-lysable debris
  – Effective in distal vessels
  – Risk distal embolization
    • Combine with lytic Tx
Reperfusion Syndrome

- **Ischemic-reperfusion syndrome**
  - **Local**: endothelial damage, capillary permeability, transudative swelling, cellular damage
    - Compartment Syndrome
    - Tx: Fasciotomy
  - **Systemic**: Lactic Acidosis, Hyperkalemia, Myoglobin, Inflammatory Cytokines
    - Cardiopulmonary complications
- **Renal Tubular necrosis**
  - Myoglobin precipitates
  - Tx: Volume, Urinary alklinization
Compartment Syndrome

- Increased Intracompartmental pressure; the Two P’s
- Less than 30 mm Hg difference between ICP and MAP, or 10 mmg difference between ICP and diastolic, or greater than 15mm pressure by Whiteside technique
- Ischemia/reperfusion, trauma, venous outflow obstruction, fracture, crush
- Whiteside catheter
Compartment Pressure

Inject into compartment slowly until meniscus moves and measure pressure on manometer-15 mmHg or less is normal

Struker system
Fasciotomy
Arm Fasciotomy
Reperfusion ARF

- Myoglobinemia, myoglobinuria, hyperkalemia, acidosis
- Bicarbonate on releasing the fascia
- Alkaline urine
- Hydrate
- Mannitol
Summary

• Thrombotic and embolic occlusions are separate processes with different presentations and treatments
• Treatment pathways in AAO are complex and vary depending on clinical situation
• Catheter-based treatments preserve outcomes with less overall morbidity
• Consider fasciotomy on clinical grounds
Vascular Trauma
Cervical Trauma
Zones

• Zone 1-Below the cricoid cartilage
  – Cervical incision plus anterior thoracotomy or median sternotomy

• Zone 2-Cricoid cartilage to angle of jaw
  – Cervical incision

• Zone 3-Above angle of jaw
  – Jaw subluxation
Cervical Injury

- Penetrating versus Blunt
- Treatment with stroke-early revascularization
- Tracheobronchial, esophageal or spinal injury 1-7%
  - Subcutaneous emphysema
  - Hematemesis
# Hard Signs/Soft Signs

## Penetrating Trauma

<table>
<thead>
<tr>
<th>Hard</th>
<th>Soft</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hard</strong></td>
<td><strong>Soft</strong></td>
</tr>
<tr>
<td>Shock</td>
<td>History of bleeding</td>
</tr>
<tr>
<td>Pulsatile bleeding</td>
<td>Proximity</td>
</tr>
<tr>
<td>Loss of pulse with evolving neurologic deficit</td>
<td>Nerve injury</td>
</tr>
<tr>
<td>Expanding hematoma</td>
<td>Stable hematoma</td>
</tr>
<tr>
<td></td>
<td>Unequal blood pressure measurement</td>
</tr>
</tbody>
</table>

**Usually exploration**

**Diagnostic measures**
Penetrating Cervical Trauma

• CTA/CT-penetrating trauma without hard signs
• Occult injury-
  – Flaps-watch
  – Dissections-repair if easy, anticoagulate if not
  – Pseudoaneurysms-repair large ones early
• Anticoagulate only large flaps if can’t operate-not great data!
Blunt Cervical Trauma

• Hyperextension of neck
  – Lateral articular processes of C1-C3
  – dissection

• Direct blow

• Laceration by bone
Screening for Carotid Injury

Denver Criteria
- Hemorrhage, hematoma
- Bruit
- Neuro exam inconsistent with head findings
- Stroke on CT
- Focal deficit
- LeFort II or III
- Basilar skull fx involving carotid canal
- GCS<6
- C-spine fx
- Hanging with anoxic injury

18% of screened had injury

Memphis Criteria
- Nero exam not explained by brain injury
- Horner’s syndrome
- Neck soft tissue injury
- Le Fort II or III
- Basilar skull fracture
- C spine fracture

29% of screened had injury
Cervical Arterial Injury

- Intimal flap
- True channel
- False channel
- Dissection
- Aneurysm
- Vertebal Artery Aneurysm
Classic Stretch Injury

- History of hyperextension (i.e. swimming)
- Severe neck to head ache near ear
- Horner’s syndrome
- Hypoglossal n. injury
- Stroke
Carotid Dissection

A. The tunica intima layer of the arterial wall tears.

B. The arterial wall is dissected. Blood clot (thrombus) forms along edge of intima tear.

C. Emboli and/or thrombus break away and are swept away by blood flow.

D. Emboli course through internal carotid artery and become lodged in the middle cerebral artery. This blocks arterial blood supply to the surrounding brain tissue.
Carotid Dissection
Carotid Dissection
Carotid Dissection

• Thrombosis-After 1-3hrs, no treatment
• Massive stroke-no Rx, anticoagulate if not contraindicated
• No or mild stroke and accessible-repair or stent
• Mild or no stroke and inaccessible-anticoagulate if not contraindicated
Blunt Thoracic Injury
Blunt Aortic Injury

- Below the subclavian artery
- Shearing stress-fixed vs non-fixed aorta, rib cage compression, ligamentum arteriosum
- Mechanism of injury
  - Seat belt strap
  - Fractures of clavicle, ribs, sternum
  - Reversed Toyota sign
- X-ray
  - Apical Cap
  - Widening of mediastinum
  - Indistinct aortic nob
  - Trachea deviation, bronchus depressed
- CT
Xray

- Obscuration
- Aortic Knob
- Tracheal and NG Tube deviation
- Mediastinal widening
- Downward Left Mainstem
- Left Apical Cap
Deviated Trachea

deviated left
depressed downward
Widened Mediastinum/Aortic Knob
Apical Cap
Xray

Chest x-ray screen

Positive predictive value 10%
Negative predictive value 98%

Too many false positives
Treatment

• Emergency treatment-hemodynamically unstable because of aortic injury

• Expectant
  – Hemodynamically unstable because of other injuries
  – Hemodynamically stable

• No treatment-missed, lost to followup
Treatment

- Initial blood pressure control
- Surgery
  - Bypass
  - Clamp and sew
- Endovascular surgery
Surgery
Endovascular Surgery
## Results

### Table 1. Comparison of Operative Approaches to Blunt Aortic Injury.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Clamp and Sew</th>
<th>Shunt-Bypass</th>
<th>Endovascular Repair</th>
</tr>
</thead>
<tbody>
<tr>
<td>Operative stress</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Blood loss</td>
<td>Medium</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Operative time</td>
<td>Medium</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Paraplegia</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Clinical scenario</td>
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<tr>
<td>Patient with high surgical risk</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Patient with severe lung injury</td>
<td>High</td>
<td>Medium</td>
<td>Low</td>
</tr>
<tr>
<td>Patient with severe head injury</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Patient with challenging anatomy</td>
<td>Medium</td>
<td>Low</td>
<td>High</td>
</tr>
</tbody>
</table>

*Relative degree of risk refers to a general comparison among the three operative procedures.*
Peripheral Injury

- Hard signs—explore—especially the 2 p’s
- Soft signs—diagnostic test to rule out or watch
- Mandatory diagnostic test—posterior dislocation of knee or elbow
- Blunt trauma associated with injury to vessels
  - Supracondylar fracture of arm or leg
  - Dislocation fracture at ankle
Posterior Dislocation

Stretch injury
Intimal Flap

- May occur in penetrating or blunt trauma without hard signs
- Issue is when to assess invasively and when to intervene
- Risks are thrombosis, embolism and pseudoaneurysm
- Consensus that most may be observed
- Anticoagulation unnecessary—possible ASA
Observation

• Most have no sequelae
• May thrombose
• Long term
  – Fistula
  – Pseudoaneurysm
• Embolism
Intimal Flap

At Injury

No treatment-6 weeks
A-V Fistula
Posttraumatic Pseudoaneurysm