Acute Limb Ischemia

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Acute limb ischemia (ALI)

- Sudden decrease in limb perfusion
- Due to embolism or thrombosis
- Threatens limb viability
- Requires urgent evaluation and management

• VASCULAR EMERGENCY
Thrombotic causes of ALI

• PAD progression is most thrombotic cause

• Arterial bypass graft thrombosis

• Embolism from heart, aneurysm (i.e. popliteal), plaque or critical stenosis

• Oral anticoagulation in patients with afib decreased ALI d/t cardiac embolization
Embolic causes of ALI

- Aortic dissection or embolization
- Popliteal entrapment, popliteal adventitial cyst
- Trauma, compartment syndrome
- Hypercoagulable states (hyperhomocysteinemia, protein S or protein C deficiency, Factor V Leiden)
Embolic causes of ALI, cont.

• Iatrogenic complications r/t:
  • cardiac cath
  • endovascular procedures
  • intra-aortic balloon pump,
  • ECMO
  • vessel closure devices
  • TAVR

• Embolic ischemia > thrombosis ischemia
Symptoms of ALI

- Symptoms develop within hours or days
- Foot or leg pain at rest
- Paresthesias
- Muscle weakness
- Paralysis of affected limb
- Six P’s: pain, pallor, paralysis, pulse deficit, paresthesia, poikilothermia (level of coldness)
Physical findings in ALI

• Embolic symptoms present after a few hours of onset
• Thrombotic symptoms occurs within two weeks of onset
• Absence of pulses distal to the occlusion
• Cool, pale, mottled skin
• Reduced sensation
• Decreased strength
Patho of embolic ALI

- Intense spasm in distal arterial tree
- Limb appears marble white initially
- Over next few hours: spasm relaxes, skin fills with deoxygenated blood leading to mottling (light blue or purple), fine reticular pattern, blanches on pressure
- Limb salvageable at this stage
- Profound paralysis with complete lack of sensation = irreversible state of ischemia
Embolic vs. Thrombotic presentation

- Embolus lodges in vascular bed, no prior collateral development

- Thrombosis occurs in vessels with prior, gradual atherosclerotic narrowing with stimulated collateral channels

- Collateral channels lessens severity/rapidity of symptoms development
Risk Factors for Embolic ALI

• Acute onset, patient able to time moment of event

• History of embolism

• Known embolic source i.e. cardiac arrhythmias

• No prior history of intermittent claudication

• Normal pulse or doppler examination in unaffected limb
Diagnosis of ALI

- Ankle-brachial index (ABI)- measured by dividing the highest ankle systolic pressure to highest, <.90 used to define a decline in limb perfusion
- Doppler ultrasound
- Angiogram-used to localize the site of occlusion
- CTA or MRA
## Rutherford classification for ALI

<table>
<thead>
<tr>
<th>Class</th>
<th>Category</th>
<th>Prognosis</th>
<th>Sensory loss</th>
<th>Muscle weakness</th>
<th>DUS—arterial and venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Viable</td>
<td>No immediate limb threat</td>
<td>None</td>
<td>None</td>
<td>Audible</td>
</tr>
<tr>
<td>IIa</td>
<td>Threatened: marginal (salvageable if treated promptly)</td>
<td>Salvageable if treated promptly</td>
<td>Minimal–none</td>
<td>None</td>
<td>Often inaudible</td>
</tr>
<tr>
<td>IIb</td>
<td>Threatened: immediate (salvageable with immediate revascularisation)</td>
<td>Salvageable if treated immediately</td>
<td>More than just toes</td>
<td>Mild–moderate</td>
<td>Usually inaudible</td>
</tr>
<tr>
<td>III</td>
<td>Major tissue loss or permanent nerve damage inevitable</td>
<td>Limb loss or permanent damage</td>
<td>Profound, anesthetic</td>
<td>Profound, paralysis</td>
<td>None</td>
</tr>
</tbody>
</table>
Systemic risk factor assessment

• Routine blood studies/coagulation tests - before heparin administered

• Plain chest x-ray and EKG

• Echocardiogram as soon as time allows
Revascularization of ALI

- Early heparinization-immediate anticoagulation prevents proximal and/or distal thrombus propagation/preserves microcirculation
- Catheter-directed thrombolysis-work by converting plasminogen to plasmin, which then degrades fibrin
- Thrombectomy (aspiration or mechanical)
- Surgery bypass if unable to remove thrombus
- Amputation – for irreversible damage
Acute Limb Ischemia

Initial treatment with intravenous heparin

Detailed examination and imaging

Stage I
  Viable Limb

Stage IIa
  Threatened marginally

Stage IIb
  Threatened immediately

Stage III
  Irreversible

Treatment according to severity

Stage I, II: Endovascular Revascularization or hybrid therapy
  - Thrombectomy (Aspiration or Mechanical)
  - Thrombolysis (Catheter-directed)
  - Surgery (especially if event >14 days)

Stage III: Amputation
  (For irreversible damage)
Contraindications to thrombolysis

- **Absolute:**
  - active bleeding
  - intracranial hemorrhage
  - presence/development of compartment syndrome
  - severe limb ischemia (requires immediate operative intervention)
Outcomes with ALI

- ALI associated with prolonged hospital stay
- Limb loss rates range from 12% to 50% - majority of amputation are above knee
- 15% mortality rate
Post-operative care

• Neurovascular checks q 15 min for first hour, then q 4 for 4 hours, then q 4 hours
• Assessment of peripheral pulses
• Assessment of motor function: dorsiflexion of foot
• Assessment of sensory function: dorsum of foot and first web space
• Reinstitution of heparin
• Vasodilation (nitro or papaverine) if evidence of vasospasm
Reperfusion injury

- Profound limb swelling with dramatic increases in compartmental pressures
- Symptoms: severe pain, hypesthesia and weakness of the affected limb
- Myoglobinuria, hyperkalemia, and acidosis can occur
- If compartment syndrome occurs—surgical fasciotomy to prevent irreversible neurologic and soft-tissue damage
Follow-up care

• Anticoagulation-warfarin, apixaban, dabigatran, rivaroxaban for 3-6 months or longer (thromboembolism is life long)
• Statins-improves graft patency
• Antiplatelet-aspirin, clopidogrel
• Beta blockers-decreases perioperative CV complications
• Surgical intervention with bypass-requires long term follow-up with ABI and arterial duplex imaging
  • Post-op visit, 3 months, 6 months then annual if no evidence of stenosis within the lumen
Case study

• 27 year old female with history of anxiety presents to OSH ER with worsening numbness and pain to left leg. Patient reports muscle cramps to RLE for past 6 months. She reports left leg poikilothermia and discoloration for past 2 days. Now has decreased sensation and pulse deficit. Heparin IV was started and CTA c/a/p showed thrombotic occlusion of the left CFA as well as right internal iliac artery. Reports OCP and smokes 2 packs per week.
Case study

• Left femoral popliteal thrombectomy was attempted day #2
• TEE showed 1.5 x 1.7cm clot in left ventricle
• Transferred to TMC for higher level of care
• No CAD noted on LHC
• Hypercoagulable work-up negative
• BLE angio obtained that showed collateralization to RLE but thromboembolectomy was unsuccessful
Case study

- Patient considered hypercoagulable due to OCP and smoking history
- Left leg deemed unsalvageable and the patient underwent left BKA with delayed healing over the next 2 months
- Lifelong AC
- 2 years later doing well with left leg prosthetic
THANK YOU

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https://med.uth.edu/cvs/
References

• Acar, R. D., Sahin, M., & Kırma, C. (2013). One of the most urgent vascular circumstances: Acute limb ischemia. SAGE open medicine, 1, 2050312113516110. doi:10.1177/2050312113516110


