Management of Acute Lower Extremity Ischemia

Deepak G. Nair MD, MS, MHA, RVT FACS
Sarasota, FL
Financial Disclosures

- No Financial Support from Industry
- No Stocks in Medical Devices/Pharma
- No Speakers Bureau
The ‘Cold Leg Call’

• Not often well received
• One of the most common and potentially devastating problems in Vascular
The ‘Cold Leg Call’

• Don’t get angry…after all…you are the one on-call

• Listen carefully
  – Timely recognition of acute limb ischemia is difficult
  – Presentation can range from subtle to dramatic
The ‘Cold Leg Call’

- Do not delay
- Consequences are dependent on the speed and accuracy of diagnosis and treatment
The ‘Cold Leg Call’

• Questions:
  – What?...chief complaint
  – When?...did it happen
  – Which?...leg
  – Who?...medical history
  – Where?...do you feel pulses...hear doppler signals
  – Why?...haven’t you called the fellow
Pathophysiology

• Lack of oxygen delivery to tissues leads to progressive depletion of high-energy substrate
• Result is anaerobic metabolism
Pathophysiology

• Tissues differ in ability to tolerate ischemia
  – Skin and subcutaneous tissue are relatively resistant
  – Peripheral nerves are sensitive
    • Prolonged functional deficits are seen after 3 hours
  – Skeletal muscle is relatively tolerant
    • Slow resting metabolic rate
    • Stores of glycogen
    • Ability to function anaerobically
Pathophysiology

• ‘Safe Period of Ischemia’ beyond which the viability of the tissue is unlikely, cannot be substantiated
  – TIME is not a reliable predictor of ischemic injury
• Depends on
  – Location of vascular occlusion
  – Rapidity with which it developed
  – Presence of collateral circulation before the occlusion
Pathophysiology

• Ischemia leads to anoxia
  – Cells are unable to sustain cellular functions
  – Transmembrane gradients cannot be maintained
  – Cell membrane becomes compromised
  – A net cellular calcium influx
Pathophysiology

• Two Distinct Phases of Injury
  – Ischemia
  – Reperfusion
    • Ischemia leads to massive catabolism of nucleotides
    • Adenosine $\rightarrow$ Inosine $\rightarrow$ Hypoxanthine
    • An abundance of Xanthine oxidase and its substrate hypoxanthine await the introduction of its other substrate: oxygen
    • During reperfusion a burst of superoxide is produced
    • Xanthine oxidase independent pathways
      – Also involved in oxygen radical injury
      – Likely from ischemic changes to mitochondria
Pathophysiology

• **Reperfusion Injury**
  – “No-reflow” phenomenon
  • **Etiology**
    – Leukocyte-capillary plugging
    – Leukocyte adhesion to venules
    – Endothelial swelling
  • In spite of flow restoration, ischemia continues
  • Muscles subjected to brief ischemia (in vitro) do not exhibit this phenomenon
Etiology

- Embolism
- Thrombosis (native artery or graft)
- Trauma
- Dissection
- Outflow Venous Occlusion
- Popliteal entrapment or cyst
Etiology

• Embolism
  – Heart
    • CAD
      – Acute MI
      – Arrhythmia
  • Valvular Heart Disease
    – Rheumatic
    – Degenerative
    – Congenital
    – Bacterial
    – Prosthetic
  – Artery-to-artery
    • Aneurysm
    • Atherosclerotic plaque
  – Idiopathic
  – Paradoxical Embolus
• Thrombosis
  – Atherosclerosis
  – Low Flow States
    • CHF
    • Hypovolemia
    • Hypotension
  – Hypercoagulable States
  – Vascular grafts
    • Progression of disease
    • Intimal hyperplasia
    • Mechanical
Etiology

• Trauma
  – Penetrating
    • Direct Vessel Injury
    • Indirect injury
      – Missile emboli
      – Proximity
  – Blunt
    • Intimal Flap
    • Spasm
  – Iatrogenic
    • Intimal Flap
    • Dissection
    • Closure devices
    • External compression
  – Drug abuse
    • Cocaine
    • Intra-arterial administration
Etiology

- Outflow Venous Occlusion
  - Compartment syndrome
  - Phlegmasia
Differential Diagnosis

• **Mimics**
  – Low Flow States
    • In the presence of chronic occlusive disease
  – Venous Thrombosis
    • Especially in early stages
  – Acute Compressive Neuropathy
    • Peroneal Nerve
    • Tibial Nerve
    • Saphenous Nerve
Initial Evaluation

• Symptoms
  – Assess severity of limb ischemia
    • Suddenness
    • Time of onset of pain
    • Weakness
    • Numbness
    – Location, intensity, and change over time
  – Determine functional status of extremity
Initial Evaluation

• Past Medical History
  – Claudication
  – Coronary artery disease
  – Arrhythmias
  – Atherosclerotic risk factors
  – Clotting problems
  – Recent percutaneous interventions
  – AGE
  – LONGEIVITY OUTLOOK
  – ANESTHETIC RISK
Initial Evaluation

• Physical Examination
  – Pulses
  – Skin color and temperature
  – Focus on sensory and motor deficits
  – Compare with normal opposite extremity
Initial Evaluation

- **Doppler Interrogation**
  - Check pedal vessels for signals
- **If doppler signals are clearly audible:**
  - Can allow delay for transfer or referral, arteriography, or identification / treatment of causative factors and co-morbidities.

- **Ankle/Brachial Index**
  - Normal >0.95
  - Claudication 0.40-0.80
  - Rest Pain 0.20-0.40
  - Ulceration/Gangrene <0.10
Staging

• **Rutherford Criteria**
  – SVS standardized criteria

  – **Class I**
    • Limb is viable and will remain so without intervention
      – Life style limiting claudication

  – **Class IIa**
    • Limbs are threatened and require revascularization for salvage, albeit not always on an emergency basis
      – Parasthesias and numbness w/o motor deficit

  – **Class IIb**
    • Limbs require very urgent revascularization to prevent limb loss
      – Sensory and motor deficits

  – **Class III**
    • Irreversible ischemia
      – Permanent paralysis and sensory loss
Treatment

• **Heparin**
  – Prevent clot propagation
  – Obviate further embolism
  – **NO studies have established a role for any antithrombotic agent in ALI**
  – Increased wound complications and hematomas perioperatively

  – Patients with ALI should be treated with unfractionated heparin to prevent further clot propagation
    • **Class 1**
      – Conditions for which there is evidence for and/or general agreement that a given procedure or treatment is beneficial, useful, and effective
    • **Level of Evidence C**
      – Only consensus opinion of experts, case studies, or standard-of-care
• Thrombolysis
  – Many randomized trials
  – No clear cut answer (Surgery vs. Thrombolysis)
    • Selected heterogenous patient populations
    • Studied complicated endpoints
  – Intracranial bleeding is the major burden for thrombolytic treatment in ALI
    • 3 American prospective, randomized trials
      – STILE - 1.2%
      – TOPAS I - 2.1%
      – TOPAS II - 1.6%
Treatment

- **Thrombolysis**
  - Consensus
    - Immediate surgical revascularization is preferred if thrombolysis would lead to an unacceptable delay in effective reperfusion.
    - In patients with irreversible ischemia, primary amputation is indicated.
    - In native artery occlusion, thrombolysis followed by correction of the causative lesion in patients with ischemia of < 14 days in duration.
Treatment

• Thrombolysis
  – Consensus
  • For occluded bypass grafts
    – surgical revision and thrombectomy
    – catheter-directed thrombolysis
    – insertion of a new graft
  • Factors to consider in therapeutic decision making
    – age and nature of the graft
    – the duration and degree of ischemia
    – availability of vein for a new distal bypass
  • Recent occlusion of a well-established graft
    – thrombolytic therapy as a primary treatment modality
    – May clear the thrombosed outflow vessels as well
Embolectomy
Compartment Syndrome

• **Definition:**
  – > 40mm Hg
  – > 30 mm Hg for 4 hours
  – Pressure within 30 mm Hg of MAP
  – Pressure within 20 mm Hg of the diastolic pressure
  
  – > 25 mm Hg consistent with diagnosis

• **Incidence:** 8% in Acute Leg Ischemia
  – 30% if associated with fracture

• **Predictors of need for fasciotomy**
  – ‘tight swelling’ pre-op or intra-op
  – Combination of arterial and venous injury
  – Soft tissue crush injury

• **May be seen after thrombolysis as well**
Compartment Syndrome

- Lower extremity
  - Anterior compartment is the most sensitive
  - Lateral > Deep Posterior > Superficial Posterior
Spasm

- Montefiore Cocktail (Dr. Frank Veith)
Success is not final, failure is not fatal: it is the courage to continue that counts.

Winston Churchill