Acute arterial embolism
Definition
Thrombus come from heart or blood vessel or other embolus such as tumor, air gas or fat flow with blood stream and occlude distal limb or visceral arteries which causes associated limb or viscera ischemia or even necrosis.
Etiology
Approximately 80% of arterial emboli are cardiac in origin.

In the past, the majority of these emboli were due to complications of rheumatic heart disease.

Recently, atherosclerotic cardiovascular disease has become the major contributor.
Approximately 70% of patients with cardiogenic emboli have atrial fibrillation, with the emboli arising in atrial mural thrombus.

Acute myocardial infarction is the second most common cause of cardiogenic emboli, preceding approximately one third of peripheral embolic events.

Ventricular mural thrombus, which occurs after acute myocardial injury, is another source of emboli.
Other sources of emboli

- Blood vessel
  - Aneurysm or atherosclerotic plaque
  - Artificial valves
  - Artificial blood vessels and catheters
- Tumor
- Air gas
- Fat
- A large thrombus often occludes the abdominal aorta and arteries of lower extremities, especially at the site of bifurcation.
- Small thrombus flows into small arteries of the brain, viscera, and upper limb.
## Site of Peripheral Embolization

<table>
<thead>
<tr>
<th>Site</th>
<th>Percentage</th>
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</thead>
<tbody>
<tr>
<td>Aortic bifurcation</td>
<td>10-15</td>
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<tr>
<td>Iliac bifurcation</td>
<td>15</td>
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<tr>
<td>Femoral bifurcation</td>
<td>40</td>
</tr>
<tr>
<td>Popliteal</td>
<td>10</td>
</tr>
<tr>
<td>Upper extremities</td>
<td>10</td>
</tr>
<tr>
<td>Cerebral</td>
<td>10-15</td>
</tr>
<tr>
<td>Mesenteric/visceral</td>
<td>5</td>
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</tbody>
</table>
The lower extremity may be salvaged after up to 5 to 6 hours of profound ischemia.

- The various tissues that comprise the extremity have different susceptibilities to ischemic injury and they manifest this injury in different fashions
  - Skin and bone are relatively resistant to the effects of ischemia
  - Nerve tissue is generally the most sensitive
  - Skeletal muscle plays a pivotal role in the numerous local and systemic manifestations of extremity ischemia-reperfusion injury
Presentation
classic presentation (5P sign)

- Pain
- Pallor
- Pulselessness
- Paresthesia
- paralysis
Pain

- The most common complaint in alert patients.
  - The degree of pain depends on the severity of ischemia.
  - The sudden onset of severe ischemic pain in a previously asymptomatic patient is most suggestive of an embolic occlusion.
Pallor

- is a common but relative finding that depends on the degree of ischemia and the underlying skin color.
Pulselessness

- Patients with acute arterial embolism generally have normal palpable pulses above the occlusion with a complete absence below.
Paresthesia

- The peripheral nerve is the tissue that is most sensitive to ischemia.

  - With severe ischemia, profound sensory loss may lead to complete anesthesia of the foot, indicative of impending tissue loss without early revascularization.
Paralysis

- Weakness of the extremity is another important sign of neurologic ischemia of the extremity.

- Mild ischemia results in a weakness or subjective "stiffness" of the toes and foot.

- As ischemia becomes more progressively severe, the weakness may progress to frank paralysis of the effected extremity.
Systemic responses

- Blood pressure decrease, shock, heart failure and even death
  - Patients’ heart can not compensate for the hemodynamic changes

- metabolic disorder such as hyperpotassemia, metabolic acidosis and even renal failure.
  - a lot of tissues necrosis
Diagnosis
- History of atrial fibrillation
- 5 P sign
- Skin temperature detection
- Duplex examination
- arteriogram
Management
Nonoperative treatment

- **Anticoagulation therapy**
  - Heparin therapy: repeatedly inject into proximal artery or continuously infused into veins

- **Antiaggregation therapy**
  - Low MW dextrans: 500ml iv qd

- **Antispasmodic, nacotic and artery dilated drugs**
  - Morphine and dolantin, or procaine
  - Sympathetic nerve blocker such as phentolamine and/or PGE1

- **Thrombolytic therapy**
  - urokinase and streptokinase
Operative treatment

- **Thrombectomy**
  - Within 6-8 hours of onset
  - Before gangrene of the affected limb occurs
    - Forgarty balloon catheter

- **Amputation**
  - Gangrene occurs and the margin between died and vital tissues is clear
Thrombectomy

- Patients with a saddle embolus to the aortic bifurcation with bilateral lower extremity ischemia are approached through simultaneous bilateral femoral artery cutdowns.
Patients with upper extremity emboli are approached in a similar fashion.

- Arterial closure and assessment are similar to that performed for the lower extremity.
Angiography

- useful in the evaluation of completeness of the thrombectomy
Postoperative therapy

- Patients should be given anticoagulant therapy to minimize the risk of recurrent embolization
  - Heparin
Reperfusion Injury

- As ischemic skeletal muscles reperfuse, a variety of intracellular ions, structural proteins, enzymes, and other components are released through the damaged sarcolemma into the circulation.

- The resulting "myonephropathic syndrome," with its associated hemodynamic instability, lactic acidosis, and hyperkalemia, is well recognized by surgeons.
Reperfusion Injury

- Myoglobin released from injured muscle cells into the circulation is cleared through the kidneys, resulting in dark urine (without red blood cells).
- Acute renal failure may ensue from myoglobin casts developing in the renal tubules as well as direct toxic effects of the myoglobin on the tubules.
- Treatment: hormone, dialysis, diuresis