Acute Limb Ischemia

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KEYWORDS

• Acute limb ischemia • Limb thrombus • Limb embolus • Phlegmasia
• Myoglobinuria • Compartment syndrome

KEY POINTS

- Acute limb ischemia is classified according to clinical findings and severity. Accurate classification of the limb ischemia is essential in determining the timing and type of intervention.
- Despite the cause, class II ischemia (threatened limb) encompasses most patients presenting with acute limb ischemia and requires intervention. Familiarity with the different types of limb ischemia cause will assist in the further workup and treatment options.
- Upper-extremity ischemia is relatively uncommon with different disease processes compared with lower-extremity ischemia. Differentiation between small vessel and large vessel disease in the upper extremity leads to a significantly different workup. Open surgical therapy remains the mainstay of therapy for large vessel upper-extremity ischemia.
- Postoperative complications attributed to myoglobinuria and compartment syndrome are crucial to monitor and have specific treatments. Operative technique, as discussed in the article, is based on different anatomic locations for compartment syndrome and should be familiar for all surgeons performing revascularization of acutely ischemic extremities.

INTRODUCTION

Acute limb ischemia is defined as any sudden decrease in limb perfusion causing a potential threat to limb viability.[1] Acute limb ischemia is a critical, potentially end-of-life, clinical condition that presents in patients with multiple medical comorbidities. This critical condition threatens the viability of the extremity and the patient's survival due to systemic acid-base, electrolyte, and other abnormalities. The diagnosis and initial assessment are mainly clinical. Diagnostic errors have severe consequences resulting in amputation or possible death. A variety of treatment modalities are available to the clinician, including anticoagulation, catheter-directed thrombolysis, pharmacomechanical thrombectomy, percutaneous mechanical thrombectomy, and operative intervention. Depending on the patient and underlying limb ischemia cause, the most appropriate intervention is essential to the final limb outcome.

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This article details the classification of limb ischemia, outlines the numerous causes of limb ischemia, highlights the diagnosis with treatment options and describes common postoperative conditions after limb ischemia intervention. The acute limb ischemia causes in the article are divided into sections, including the presentation, diagnosis, and therapy for each cause. The broad limb ischemia causes include the following:

- Embolism
- Thrombosis
- Venous obstruction
- Trauma
- Upper extremity: uncommon causes

Postoperative management is extremely important after revascularization of an acutely ischemic extremity. Reperfusion injury, myoglobinuria, and compartment syndrome are summarized in the postoperative section.

CLASSIFICATION OF ACUTE LIMB ISCHEMIA

The classification system of acute limb ischemia is based on the severity of the ischemia, which determines the therapy and timing of intervention plus implications for outcomes. The Rutherford classification of limb ischemia is accepted as the standard reporting system for limb ischemia (**Table 1**). The three ischemia categories are based on clinical findings and Doppler measurements, which can be performed bedside.[2,3]

Class I: Viable, nonthreatened extremity, no neurologic deficit, audible Doppler signal

Class II: Threatened extremity, manifested by neurologic deficit and sluggish/absent Doppler signals in the affected limb. Class II is divided into 2 subcategories: class IIA has mild sensory deficits, whereas Class IIB is associated with both motor and sensory deficits

Class III: Irreversible ischemic nerve and sensory deficits

Table 1
Rutherford's acute limb ischemia classification

		Findings		Doppler Signals	
Category	Description/ Prognosis	Sensory Loss	Muscle Weakness	Arterial	Venous
I. Viable	Not immediately threatened	None	None	Audible	Audible
II. Threatened					
a. Marginally	Salvageable if promptly treated	Minimal (toes) or none	None	Inaudible	Audible
b. Immediately	Salvageable with immediate revascularization	More than toes, associated with rest pain	Mild, moderate	Inaudible	Audible
III. Irreversible	Major tissue loss or permanent nerve damage inevitable	Profound, anesthetic	Profound, paralysis (rigor)	Inaudible	Inaudible

From Rutherford RB, Baker JD, Ernst C, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg 1997;26:518; with permission.

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Class I limb ischemia may only require medical therapy such as anticoagulation. Any revascularization, endovascular or open therapy, can be scheduled electively.

Class II acute limb ischemia encompasses most patients with acute limb ischemia and requires intervention. There is a distinct difference between class IIA (marginally threatened) and IIB (immediately threatened). Class IIA patients should undergo either endovascular or open intervention on an urgent basis depending on the duration of their symptoms. With less than 2 weeks of symptoms, prospective studies comparing thrombolytic and surgical interventions favor percutaneous endovascular options, such as thrombolytic or pharmacomechanical thrombectomy. Ischemic symptoms presenting duration are better treated with open surgical intervention.[4] Class IIB ischemia, manifested by motor and sensory deficits, requires emergency intervention. Surgical therapy has been the preferred therapy; however, advances in catheter-based thrombolytic therapy and pharmacomechanical thrombectomy have shortened the time to reperfusion. In addition, the hybrid operating room has allowed surgeons to perform diagnostic imaging, endovascular intervention, and open surgical therapy in a single setting.

Class III ischemia presents with profound neurologic deficits (insensate, paretic limb), muscle rigidity, and the absence of arterial or venous Doppler signals in the affected limb region. Revascularization is usually futile and potentially harmful if therapy leads to myoglobinuria. Primary amputation should be considered.

CAUSE

Embolism

Presentation

Embolism as a cause of acute limb ischemia is defined by debris in the vascular system that obstructs a distal artery. The most common source for an embolus is the heart, where mural thrombus dislodges and obstructs smaller peripheral arteries leading to acute disruption of blood flood to the extremity.[5] Proximal atherosclerotic debris is another source of emboli, where debris from the proximal aorta dislodges and obstructs peripheral arteries.[6] Whether from the heart or proximal aorta, the emboli travels through the vascular system, and as the caliber of vessels decreases, the likelihood of obstructing a peripheral vessel increases. The embolus tends to obstruct at bifurcations, where the lumen of the arteries is narrowed. In the lower extremity, this occurs most frequently at the common femoral artery and popliteal artery. In the upper extremity, the embolus obstructs most commonly at the origin of the profunda brachialis or brachial artery bifurcation.

Acute embolic ischemia presentation is dramatic in nature because it likely occurred in a healthy artery without established collaterals. The patient usually presents with an acute white extremity and a neurosensory deficit. As time passes, the occlusion also worsens because of secondary thrombus that forms both proximal and distal to the emboli.[7] Early diagnosis and treatment are paramount because secondary clot can propagate into distal vessels making revascularization difficult if not impossible.

Diagnosis

Patients presenting with acute limb ischemia from an embolic source have an acutely white leg or arm secondary to the lack of time for collaterals to form. Neurosensory deficit is one of the early signs of acute limb ischemia because sensory nerves are the first to be affected. Motor nerve deficit then appears, leading to muscle weakness in the extremity. Extremity musculature is the last to show symptoms exhibited with extremity tenderness in the affected compartment followed by muscle rigidity. Neurosensory and motor deficit with muscle rigidity is considered end-stage signs of acute limb ischemia.

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Duplex ultrasonography is used to evaluate the level of arterial occlusion in acute limb ischemia due to embolic disease. Computed tomographic arteriography (CTA) is widely available in hospitals and has become the imaging modality most frequently used to evaluate acute limb ischemia. Image quality of computed tomographic scanners using intravenous (IV) contrast is comparable to arteriograms. Arteriogram, which was once the mainstay imagining for acute limb ischemia, has been replaced by CTA. Arteriography is the best choice when the problem could be treated endovascularly because it is both diagnostic and therapeutic.

Management

Systemic therapeutic anticoagulation with unfractionated heparin should be started as soon as the diagnosis of acute limb ischemia is made as long as there are no contraindications. An IV bolus of 80 to 100 units/kg should be given and then titrated to maintain partial thromboplastin time between 2.0 and 3.0 times normal values (60–100 seconds). The patient should receive IV analgesia and hydration because they are volume depleted and in tremendous pain. Volume repletion may mitigate the contrast load they will receive and the potential myoglobinuria from reperfusion.

Treatment of acute limb ischemia is based on the Rutherford's classification, extent of clinical ischemia present, and available expertise with endovascular technology.[2] Class I acute limb ischemia, where the limb is not immediately threatened, can be treated with anticoagulation. If revascularization is deemed necessary, both endovascular and open surgical interventions are available.

Acute subcritical ischemia (class IIA) with stable acute ischemia has both endovascular and open surgical options. All obvious emboli should be treated with open embolectomy. With the clear embolus exception, intra-arterial thrombolysis is the primary treatment option for class IIa ischemia. The patient should be interrogated for contraindications to thrombolysis (**Box 1**), and the surgeon should have a low threshold for embolectomy and possible open bypass if a contraindication is present.

Acute critical limb ischemia (class IIB) needs urgent intervention. If institutions are limited in vascular and endovascular resources, transferring a patient with a full range of vascular and endovascular services should be considered. With time to reperfusion as a key factor, most class IIB patients are best treated in the operating room with embolectomy, surgical bypass, or catheter-directed thrombolysis. Hybrid operating rooms now allow surgeons to perform repeated diagnostic imaging, endovascular intervention with catheter-based thrombolysis, or percutaneous mechanical thrombectomy and open surgical revascularization.

In patients presenting with class III acute limb ischemia wherein major tissue loss has occurred along with permanent nerve damage, revascularization is not indicated and amputation should be considered.[8]

Thrombosis

Presentation

Thrombosis as a cause of acute limb ischemia is caused by a blood clot within an artery; this can be caused by atherosclerotic obstruction or hypercoagulability. When thrombosis is due to progressive atherosclerotic narrowing in peripheral arteries, this leads to a platelet thrombus forming once the stenosis becomes critical and leads to acute arterial occlusion. Unlike embolic disease, thrombosis is progressive, and thus rich collaterals have formed over time. Acute critical limb ischemia occurs when this process occurs at multiple levels. In hypercoagulable states, thrombosis can occur within the arterial systems that do not have atherosclerotic disease.

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Box 1
Contraindications to pharmacologic thrombolysis

Absolute contraindications

Active bleeding disorder

Gastrointestinal bleeding within 10 days

Cerebrovascular event within 6 months

Intracranial or spinal surgery within 3 months

Head injury within 3 months

Relative contraindications

Major surgery or trauma within 10 days

Hypertension (systolic >180 mm Hg or diastolic >110 mm Hg)

Cardiopulmonary resuscitation within 10 days

Puncture of noncompressible vessel

Intracranial tumor

Pregnancy

Diabetic hemorrhagic retinopathy

Recent eye surgery

Hepatic failure

Bacterial endocarditis

From Kwolek CJ, Shuja F. Acute ischemia: treatment. In: Cronenwett JL, Johnston KW, editors. Rutherford's vascular surgery. 8th edition. Philadelphia: Elsevier; 2014. p. 2528–43; with permission.

Hypercoagulable thrombosis is usually seen in small arterial vessels and is associated with malignancy, hyperviscosity, and low flow states.[9]

Diagnosis

Patients presenting with acute limb ischemia from thrombosis usually present with worsening claudication symptoms and rest pain. Owing to their rich collaterals that have formed, the leg does not appear acutely white like embolic acute limb ischemia. Similar to embolic disease, neurosensory deficit is one of the early signs of acute limb ischemia because sensory nerves are the first to be affected. Muscle weakness in the extremity from the ischemic motor neurons follows. Finally, extremity tenderness followed by rigidity in the muscle compartment is seen. Unlike embolic disease wherein these changes are seen within hours, thrombosis is more forgiving owing to the collaterals that have formed over time.

Duplex ultrasonography can be used to evaluate the level of arterial occlusion in acute limb ischemia due to thrombosis. CTA has replaced invasive arteriography in the setting of acute ischemia. The exception is when the patient is brought to the hybrid operating room for diagnostic arteriography followed by immediate intervention.

Management

Management of acute limb ischemia from thrombosis is similar to embolic limb ischemia treatment. If no contraindication, therapeutic anticoagulation with unfractionated heparin should be initiated.[10] A bolus of 80 to 100 units/kg should be given and then titrated to maintain partial thromboplastin time between 2.0 and 3.0 times normal values (60–100 seconds). The patient should receive IV analgesia and hydration

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because they are volume depleted and in tremendous pain. Volume repletion may mitigate the contrast load they will receive and the potential myoglobinuria from reperfusion.

Treatment of acute limb ischemia from thrombosis is based on Rutherford's classification, extent of clinical ischemia, and available expertise with endovascular technology.[8] As previously noted in the embolism section, class I acute limb ischemia is treated with anticoagulation alone. If revascularization is deemed necessary, both endovascular and open surgical interventions are available. Class II acute limb ischemia, where the limb is salvageable with prompt intervention, requires revascularization by either endovascular or open surgical technique or hybrid approach.[11] Because of the thrombosis cause, most class II patients will benefit from an endovascular technique involving either low-dose arterial thrombolysis, high-dose pulse spray thrombolysis, or pharmacomechanical thrombectomy due to the acute thrombus present. Additional open techniques might be warranted especially in the class IIB setting. In patients presenting with class III acute limb ischemia where major tissue loss has occurred along with permanent nerve damage, revascularization is not indicated and amputation should be considered.

Venous Obstruction: Phlegmasia Cerulea Dolens

Presentation

Phlegmasia cerulea dolens is a rare venous condition caused by a severe form of venous thrombosis. The venous thrombosis extends into collateral veins resulting in severe venous congestion with massive fluid sequestration and significant edema. Approximately 40% to 60% of phlegmasia cerulean dolens cases progress to venous gangrene when there is retrograde progression of the venous thrombosis to include the capillary bed.[12,13] Phelgmasia cerulea dolens is identified by sudden pain, swelling, purple ecchymosis, and arterial ischemia with loss of distal pulses in the extremity[14] (**Fig. 1**). Risk factors include malignancy, femoral vein catheterization, heparin-induced thrombocytopenia, antiphospholipid syndrome, recent surgery, heart failure, and pregnancy.[15]

Diagnosis

The diagnosis of phlegmasia cerulea dolens is clinical with a high index of suspicion for the severity of the venous disease process. The 4 key diagnostic signs include edema, violaceous discoloration, pain, and severe venous outflow obstruction. Duplex ultrasonography remains the diagnostic test of choice for the detection of deep vein thrombosis. Duplex ultrasound diagnostic criteria for acute deep vein thrombosis require intraluminal echogenicity, increased venous diameter, noncompressibility of the vein with pressure from the transducer, and absence of flow augmentation with distal compression.[16] Computed tomographic venography and magnetic resonance venography are useful additional diagnostic modalities when imaging larger venous segments, especially the inferior vena cava and iliofemoral veins; however, sensitivities diminish for these expensive modalities when smaller diameter veins are evaluated.[17,18]

Management

Treatment of phlegmasia cerulean dolens with systemic anticoagulation should be initiated as soon as the diagnosis is suspected. Heparin administration is started with IV bolus (80–100 U/kg) followed by continuous infusion rate of 15 to 18 U/kg/h. Activated partial thromboplastin time should be monitored with a goal of 2.0 to 3.0 times normal values (60–100 seconds). Newer oral Factor Xa inhibitors (apixiban, rivaroxban) are approved for DVT treatment, however, after all interventions are completed and the patient is clinically improved.

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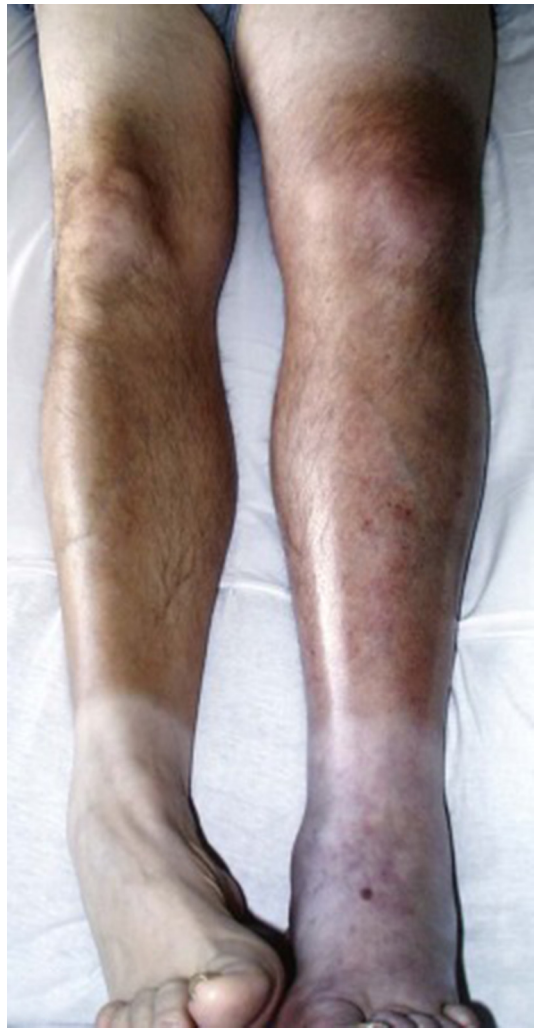

Fig. 1. Phelgmasia cerulea dolens. (*From* Comerota AJ, Aziz F. Acute deep venous thrombosis: surgical and interventional treatment. In: Cronenwett JL, Johnston KW, editors. Rutherford's vascular surgery. 8th edition. Philadelphia: Elsevier; 2014. p. 792–810; with permission.)

Surgical therapy for phlegmasia cerulea dolens centers around relief of proximal large-caliber vein thrombosis to allow for maximal venous outflow. If the patient has no contraindications to thrombolysis, catheter-directed thrombolytic therapy is the primary treatment of extensive DVT associated with phlegmasia. Under ultrasound guidance, the popliteal or tibial vein is accessed. After crossing the segment of thrombus, a multihole infusion catheter is placed across the clot, and thrombolysis infusion is started. For patients with tissue compromise, aggressive maneuvers such as pharmacomechanical thrombolysis, aspiration thrombectomy, or angioplasty can be performed in addition to initiating thrombolysis on the initial venogram.[19,20] With the thrombolysis infusion, patients are monitored in the intensive care unit setting and returned for repeat angiograms every 24 hours for 1 to 3 days. Venous occlusive or stenotic lesions are treated with stenting after the resolution of acute thrombus.

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If the patient has a contraindication to thrombolysis, open surgical thrombectomy is indicated (Fig. 2). Under general anesthesia in the operating room, the patient is placed in the Trendelenburg position. The femoral vein is exposed from a longitudinal incision. After circumferential vessel control, a longitudinal venotomy in the common femoral vein is performed to allow for a Fogarty balloon (no. 8 or 10 balloon catheter) passage proximally into the iliofemoral veins. For infrainguinal thrombus, the leg is elevated and compressed with an esmarch wrap from the foot toward the groin. If thrombus persists, the posterior tibial vein is exposed in the distal lower extremity. To preserve the lower-extremity vein valves, advanced technique calls for passage of Fogarty catheters from both the femoral and the tibial veins by connecting each with silastic stem from an IV catheter (12–14 gauge). A no. 4 femoral Fogarty catheter is pulled down to the tibial venotomy (by the connected tibial catheter) and then inflated and pulled back from the infrainguinal veins to perform the thrombectomy.

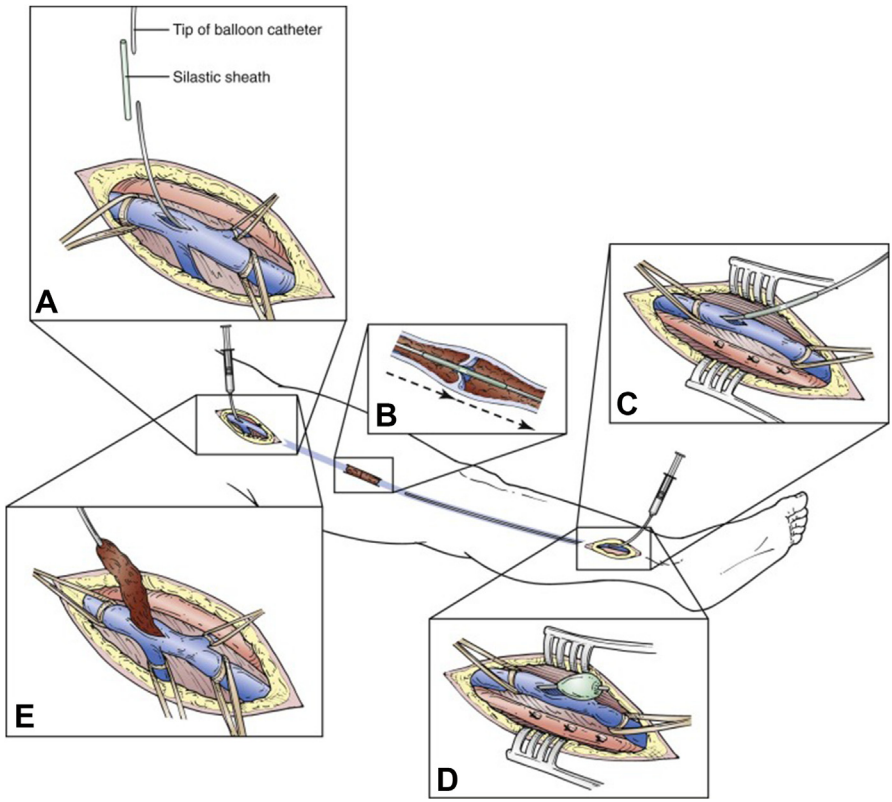

Fig. 2. Open surgical venous thrombectomy. (*A*) Femoral vein exposure and longitudinal venotomy. Passage of Fogarty balloon catheter from exposed posterior tibial vein. (*B,C*) Connection of femoral fogarty catheter to tibial Fogarty catheter with silastic sheath to allow for catheter passage distally and preservation of lower extremity vein valves. (*D*) Inflation of proximal femoral Fogarty catheter and pulled back through the lower extremity veins. (*E*) Thrombus removal after catheter thrombectomy. (*From* Comerota AJ, Aziz F. Acute deep venous thrombosis: surgical and interventional treatment. In: Cronenwett JL, Johnston KW, editors. Rutherford's vascular surgery. 8th edition. Philadelphia: Elsevier; 2014. p. 792–810; with permission.)

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After the venous thrombectomy, the venous system is flushed with a red rubber catheter placed in the tibial venotomy. Completion venogram is carried out with any iliac vein stenosis treated with angioplasty or stenting at that time. After femoral venotomy closure, an end-to-side arteriovenous fistula is created between the femoral artery and vein using the proximal saphenous vein to assure patency of the revascularized venous segment.[21] Frequently, 4-compartment fasciotomy will be required for acute compartment syndrome. If venous revascularization is unsuccessful, amputation is sometimes required.

Trauma

Presentation

Acute limb ischemia caused by traumatic disruption of blood flow to a limb presents with either hard or soft signs of vascular injury. Hard signs of vascular injury are pulsatile bleeding, expanding hematoma, absent distal pulses, cold limb, palpable thrill, and audible bruit. Soft signs are significant hemorrhage on history, nerve deficit, reduced palpable pulse, and injury in proximity to major artery.[22] Patients who present with hard signs of vascular injury need to be taken emergently to the operating room for exploration and definitive repair. Traumatic injuries, whether blunt or penetrating, that are in close proximity to vascular structures need to be thoroughly investigated to rule out vascular injury.

Diagnosis

The diagnosis of acute limb ischemia due to trauma is usually identified on initial physical examination because there is usually trauma near to the vasculature of that extremity. Whether it is a gunshot wound to the leg that is actively hemorrhaging or a fracture without distal pulses, knowing the anatomy of the extremity with certain vascular injuries is expected as is the case with posterior knee dislocations and high incidence of popliteal artery injury. Evaluation of the extremity and evidence of hard or soft vascular signs will dictate whether further imagining is necessary versus operative exploration. Patients presenting with soft signs of vascular injury can be further evaluated with Doppler ultrasound, CTA, or angiography.[23]

Management

In acute limb ischemia caused by trauma, the goal is restoration of flow to extremity as soon as possible. If the patient is actively hemorrhaging, a tourniquet is placed proximally for hemostasis and the patient is taken to operating room for surgical exploration. In the operating room, the following basic principles of vascular surgery should be adhered to:

1. Obtain proximal and distal control of the injured vessel
2. Repair: primary repair, venous angioplasty, or bypass with suitable conduit

After perfusion has been restored, it is important to determine whether the extremity requires a fasciotomy. In most instances, if an extremity has gone 6 hours or more with ischemia, a fasciotomy should be performed.[24] Patients who do not receive a fasciotomy during initial revascularization should be watched closely for the development of compartment syndrome and taken promptly back to the operating room for a fasciotomy. Special consideration should be given to blunt trauma in which patient has a posterior knee dislocation because physical examination may not be revealing. A high index of suspicion is required for posterior knee dislocations with CTA or arteriography needed to rule out vascular injury. Undiagnosed popliteal injuries have as high as a 50% amputation rate.[25]

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Upper Extremity: Uncommon Causes

Acute ischemia of the upper extremity is relatively uncommon, accounting for only one-fifth of all patients presenting with acute limb ischemia.[26] Women are affected twice as often as men, and patients are significantly older than those with acute lower-extremity ischemia.[27] Numerous different disease processes affect the upper-extremity vasculature versus atherosclerosis, accounting for most of the lower-extremity vascular disease. The cause, pathophysiology, and treatment of upper-extremity ischemia can be differentiated between small vessel and large vessel disease. Small vessel arteriopathies in the upper extremity lead to distal extremity and hand ischemia. These small vessel diseases include autoimmune or connective tissue disease, such as scleroderma, rheumatoid arthritis, systemic lupus, Buerger's disease (thromboangiitis obliterans), and Raynaud phenomenon.[28] Large vessel artery disease in the upper extremity is mainly attributed to atherosclerosis. The most common location of occlusive disease in the upper extremity is the left subclavian artery origin. Other specific large vessel pathologic conditions potentially leading to acute ischemia are arterial thoracic outlet syndrome, thromboembolism from subclavian or axillary artery aneurysms, iatrogenic trauma from cardiac catheterization, steal syndrome after dialysis access placement, aortic dissection with great vessel involvement, trauma, or embolic occlusion from atrial fibrillation.

Presentation

Acute ischemia presents with upper extremity symptoms, such as sudden pain, paresthesias, pallor, and paralysis. Physical examination reveals diminished or absent brachial, radial, or ulnar pulses. Other examination findings reveal arm pallor, dependent hand rubor, and reduced extremity temperature. Because of the diffuse arterial collateral network, acute occlusion in the upper extremity rarely results in tissue loss.[27]

Diagnosis

A detailed history and physical examination can help narrow down the ischemia cause. Important questions in the history regard signs and symptoms for connective tissue disorders (dry eyes, dry mouth, arthritis), remote trauma, recent arterial access for peripheral or coronary catheterization, or occupational history with vibrating tools. In addition to the vascular examination, bilateral blood pressures are assessed with greater than 20 mm Hg difference, suggestive of arterial inflow disease. Auscultation of the supraclavicular and infraclavicular fossa for a bruit can signify subclavian artery stenosis. Splinter hemorrhages in the nail beds are seen with chronic emboli and can give a clue as to the acute pathologic condition, such as cardiac valve vegetation with embolic phenomenon.

The initial diagnostic test is noninvasive segmental pressure measurements of the upper extremity and fingers. Arterial pressures are compared with a wrist to brachial pressure index ratio, where a normal index range is 0.85 to 1.0 and an index less than 0.85 is abnormal. CTA and magnetic resonance arteriography (MRA) provide detail of large vessel disease to the level of the wrist. Catheter-based arteriography is reserved for nondiagnostic CTA/MRA or if small vessel disease is suspected. Unilateral arm or hand ischemia in an athlete is suggestive of subclavian or axillary artery aneurysmal disease, and multiple imaging views should focus on this area. Bilateral digital ischemia warrants bilateral upper extremity arteriography as well as blood testing for systemic disease. Studies for unilateral hand or finger ischemia include duplex ultrasonography, echocardiography, bilateral arteriography, and blood test screening. Blood test screening for autoimmune disease entails erythrocyte sedimentation rate, C-reactive protein, antiphospholipid antibodies, antinuclear antibody titer, and rheumatoid factor. There should be a low threshold for rheumatology consultation if blood screening tests are positive in the setting if digital ischemia.

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Management

Surgical As opposed to most lower-extremity revascularizations being completed endovascularly, upper-extremity therapy for acute ischemia has stayed predominately with an open surgical approach, likely because of the infrequency of interventions and the underlying arterial causes. Most of the literature describes treatments of occlusions in the axillary, brachial, radial, and ulnar arteries with surgical bypass or embolectomy.[29,30] Brachial artery embolectomy is the most common upper-extremity embolectomy. Exposure of the distal brachial artery is recommended as well as exposure of both the forearm artery origins to assure embolectomy catheter (2 or 3 French) passage down each artery. Upper-extremity bypasses have excellent patency rates compared with lower extremity. Autogenous vein conduits with greater saphenous vein or upper-extremity vein (basilic, cephalic) are recommended for bypass with anatomic tunneling along the axillary or brachial artery axis.[31] Arterial thoracic outlet syndrome is most commonly treated with a hybrid approach requiring thrombolysis of the affected extremity followed by cervical/first rib resection and possible subclavian artery aneurysm repair. Because of the infrequency of the pathologic condition (least common of all thoracic outlet pathologic condition, 1%), arterial thoracic outlet should be treated at centers with up-to-date endovascular technology and experience in open decompression of the thoracic outlet.

Endovascular The main exceptions to open therapy with large vessel arterial endovascular interventions have been described with proximal subclavian artery and axillary artery angioplasty and stent placement. There are increasing reports of covered stents for subclavian and axillary injury.[32] Numerous other endovascular case reports are reported in the literature with low case numbers and minimal follow-up.

Medical For patients presenting with acute digital ischemia with normal results on physical examination, screening blood tests and noninvasive vascular laboratory tests but a history of vasospasm, Raynaud phenomenon is diagnosed. Raynaud phenomenon diagnosis is one of exclusion after other causes are ruled out. Medical management is the mainstay of therapy. Two medications have proven beneficial in randomized, double-blinded control trials: nifedipine (30 mg daily) and losartan (50 mg twice daily).[33,34]

POSTOPERATIVE COMPLICATIONS

Myoglobinuria

Myoglobinuria is common after treatment of acute limb ischemia. Myoglobinuria exerts its nephrotoxic effects by inducing renal vasoconstriction, tubular cast formation, and direct heme protein–induced cytotoxicity.[35] Preexisting renal insufficiency, large volumes of iodinated contrast (>150 mL) delivery, and hemoglobinuria are all risk factors for myoglobinuria and subsequent acute kidney injury. Hemoglobinuria is commonly seen after percutaneous mechanical thrombectomy, which causes lysis of red blood cells. Treatment mandates maintaining a urine output more than 100 mL/h and alkalinization of the urine with sodium bicarbonate added to IV fluids. Acute renal failure due to myoglobinuria may require temporary dialysis until the kidney function improves.

Compartment Syndrome

Compartment syndrome, regardless of cause or anatomic location, is caused by an increase in intracompartmental pressure (ICP) within an unyielding fascial envelope that impairs tissue perfusion.[36] The most common vascular causes for compartment

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syndrome are ischemia-reperfusion injury associated with acute ischemia, arterial and venous traumatic injuries, crush injuries, phlegmasia cerulea dolens, and hemorrhage within a compartment. Compartment syndrome may complicate up to 21% of case of acute limb ischemia.[24,37] Risk factors for compartment syndrome after acute arterial ischemia include prolonged ischemia time (>6 hours), young age, insufficient arterial collaterals, acute time course for arterial occlusion, hypotension, and poor back-bleeding from the distal arterial tree at embolectomy.[24]

The diagnosis of compartment syndrome relies on a high index of suspicion. Pain disproportionate to the injury and paresthesias in the distal extremity are the key symptoms of compartment syndrome.[38] On examination, the most common finding is a tense, swollen compartment with pain on passive movement of the muscles in that compartment. ICP measurements are not required for a diagnosis but can be useful in equivocal cases, unconscious patients, and pediatric patients. Normal compartment pressures measure less than 10 to 12 mm Hg. Fasciotomy is recommended if the difference between ICP and mean arterial pressure decreases to less than 40 mm Hg or the difference in ICP and diastolic pressure is less than 10 mm Hg.[39]

Compartment syndrome is a surgical emergency, and once clinically diagnosed, fasciotomy of the affected compartment is indicated. Several fasciotomy techniques (lower extremity, thigh, and forearm) will be described below with accompanied operative diagrams.

- Lower-extremity fasciotomy is carried out most commonly through the double-incision technique (**Fig. 3**) (single-incision fasciotomy is an accepted technique but not described here). A longitudinal incision in made on the lateral aspect of the lower leg between the fibula and crest of the tibia (approximately 4 cm lateral to the tibia crest). The intermuscular septum is identified, and the anterior and lateral compartments are decompressed with attention to avoid injury to the common superficial and deep peroneal nerves near the fibula head. The second incision is made on the medial lower leg 2 cm posterior to the tibia to decompress the 2 posterior compartments. Deep posterior compartment decompression requires dividing the attachments of the soleus muscle to the tibia.[40]
- The thigh contains 3 compartments: anterior, posterior, and medial. Thigh decompression is accomplished most commonly through a single lateral incision to decompress the posterior and anterior compartments (**Fig. 4**). Medial compartment decompression is rarely required. A lateral thigh incision is placed just distal to the intertrochanteric line and extends distally to the lateral

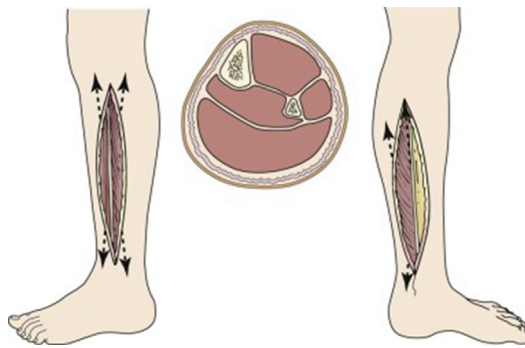

Fig. 3. Fasciotomy technique: lower-extremity double-incision technique. (*From* Janzing H, Broos P, Rommens P. Compartment syndrome as a complication of skin traction in children with femoral fractures. J Trauma 1996;41:156; with permission.)

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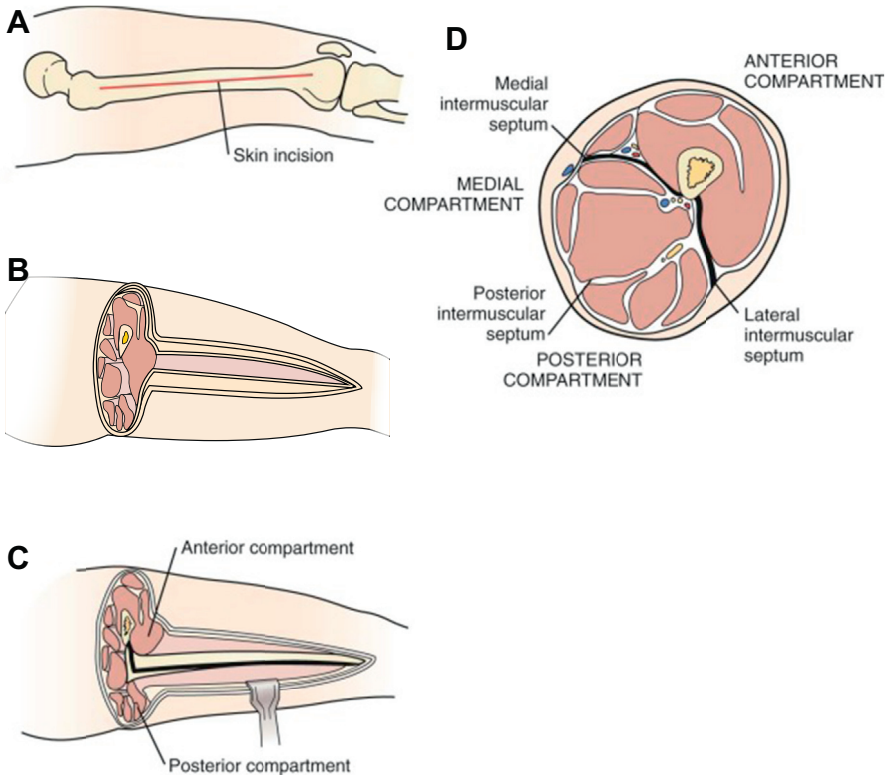

Fig. 4. Fasciotomy of the thigh. (*A*) The incision extends from the intertrochanteric line to the lateral epicondyle. (*B*) Skin incision with anterior and posterior muscle compartments visualized in transverse plane. (*C*) The anterior compartment is opened by incising the fascia lata. The vastus lateralis is retracted medially to expose the lateral intermuscular septum, which is incised to decompress the posterior compartment. (*D*) Thigh compartments and appropriate incision. (*From* Chung J, Modrall JG. Compartment syndrome. In: Cronenwett JL, Johnston KW, eds. Rutherford's vascular surgery. 8th ed. Philadelphia, PA: Elsevier; 2014: 2544–54; with permission and Tarlow SD, Achterman CA, Hayhurst J, et al. Acute compartment syndrome in the thigh complicating fracture of the femur: a report of three cases. J Bone Joint Surg Am 1986;68:1439; with permission.)

epicondyle. The iliotibial band is exposed and incised longitudinally to decompress the anterior compartment. The vastus lateralis is reflected medially to expose the lateral intermuscular septum, which is then incised for posterior compartment decompression. If necessary, a separate incision over the adductor muscle group in the medial thigh will decompress the medial compartment.[41]

- Upper-extremity compartment syndrome is seen most frequently in the forearm. The forearm compartments consist of the volar (flexor, superficial, deep), lateral (mobile wad), and extensor (dorsal, superficial, deep) compartments. The volar approach decompresses the lateral and volar compartments with a single incision (Fig. 5). A curvilinear incision begins proximal to the antecubital fossa and medial to the biceps tendon, crosses the antecubital crease, and extends to the radial side of the forearm, where it extends distally along the medial border of the brachioradialis muscle. From the distal forearm, the incision extends

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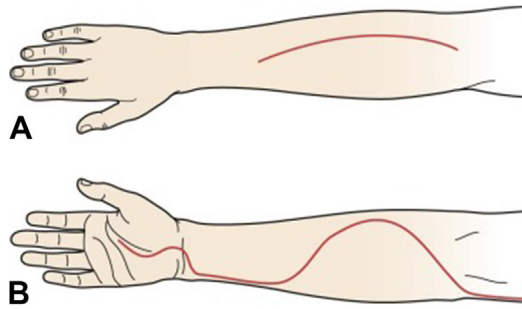

Fig. 5. Fasciotomy of the volar forearm for severe Volkmann's contracture. (*A*) Extensive opening of the fascia of the dorsum of the forearm for dorsal compartment syndromes. (*B*) Incision used for anterior forearm compartment syndromes. The skin and underlying fascia are released completely throughout. (*From* Chung J, Modrall JG. Compartment syndrome. In: Cronenwett JL, Johnston KW, editors. Rutherford's vascular surgery. 8th edition. Philadelphia: Elsevier; 2014. p. 2544–54; with permission.)

across the carpal tunnel along the thenar crease. The fascia overlying the superficial flexor compartment is incised along the entire length of skin incision. The fascia overlying each of the muscles of the deep flexor compartment is incised to complete the volar fasciotomy. The extensor compartment is decompressed through a separate incision from the lateral epicondyle to the wrist between the extensor carpi radialis brevis and the extensor digitorum communis.[42]

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