Compartment Syndrome Following External Iliac Artery Stenting and Lower Extremity Thrombolysis

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Compartment syndrome is an infrequent yet very serious complication of endovascular revascularization of ischemic extremities. Believed to be a consequence of ischemic-reperfusion injury, postischemic compartment syndrome can lead to devastating consequences such as amputation, renal failure, or death. Therefore, when performing endovascular procedures, it is essential to have a thorough knowledge of the clinical signs and symptoms of developing compartment syndrome.

CASE REPORT

A 62-year-old woman with a history of peripheral vascular disease, cerebrovascular accident, and bilateral femoral-popliteal bypasses 7 years earlier was referred to our Vascular and Interventional Radiology Section for evaluation and management of acute, severe left lower extremity pain with absent pulses below the left femoral artery. The presumptive diagnosis was acute left bypass graft occlusion and thrombolysis was planned.

After informed consent, conscious sedation was achieved with midazolam and fentanyl. Using a micro-puncture catheter system, a guide wire and 5F sheath were inserted in the right common femoral artery. A pigtail catheter was placed at the iliac bifurcation and bilateral pelvic angiograms were performed, showing near occlusive stenosis of the left external iliac artery, high-grade stenosis of the right external iliac artery, and occlusion of the left femoral-popliteal bypass graft (Fig. 1).

After administering 5000 IU of heparin, angioplasty of the right external iliac artery was performed

using a 6 × 20 mm angioplasty balloon catheter and stenting of the left external iliac artery was performed using a 7 × 40 mm balloon and self-expanding nitinol stent. Postintervention angiogram confirmed resolution of the stenosis and improved flow through the iliac and femoral artery systems bilaterally (Fig. 2).

The left femoral-popliteal bypass graft was selected and a 10-cm infusion catheter was positioned proximally in the thrombosed bypass graft (Fig. 3). Tissue plasminogen activator (Alteplase[®]; Genentech, San Francisco, CA) infusion was initiated through the infusion catheter at 0.5 mg per hour and heparin was instilled at 200 units per hour via the sidearm of the vascular sheath. The patient tolerated the procedure without immediate complications and was transferred to the intensive care unit for observation while receiving overnight thrombolysis.

On postprocedure day 1, a routine follow-up physical examination showed significant pain on passive range of motion of the left calf. The patient was taken emergently to the operating room for 4-compartment fasciotomy of the left calf. Upon entry to the posterior compartment of the calf, the muscles were pale and swollen, but did regain limited color and slow twitch. A thrombectomy of the bypass graft was also performed as it continued to be occluded distally. Unfortunately, reestablishing blood flow (Fig. 4) did not avert progress of symptoms and the patient underwent guillotine below-knee amputation of her left leg 3 days after fasciotomy. She was ultimately discharged to an acute rehabilitation facility after completion of an above-knee amputation.

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Figure 1 62-year-old woman with threatened limb. (A) Initial digital subtraction aortogram shows high-grade lesions of the both external iliac arteries. (B) Conventional angiogram of the thighs reveals occlusion of the left bypass graft.

DISCUSSION

Acute lower extremity compartment syndrome is a limb and life-threatening condition caused by bleeding or edema in a closed muscle compartment. Because skeletal muscles are enclosed in a deep fascial covering (Fig. 5), raised intracompartmental pressure may reduce capillary perfusion below a level necessary for tissue viability, ultimately leading to muscle and nerve ischemia. Compartment syndrome is most often caused by traumatic insults such as fractures, crush injuries, or prolonged limb compression; however, ischemic reperfusion injuries following thrombolysis or embolectomy have been reported to be potential causes as well. The anterior compartment of the calf is most frequently affected by compartment syndrome due its less compliant nature.

Known as *postischemic compartment syndrome*, revascularization of a limb following a prolonged period of ischemia can lead to tissue swelling and compartmental hypertension. Pathophysiologically, postischemic compartment syndrome is believed to be a consequence of ischemic-reperfusion injury, in which tissue damage is worsened by the reintroduction of oxygenated blood flow to the ischemic limb. New damage may occur by increased generation of oxygen-derived free radicals from parenchymal and endothelial cells, and from infiltrating leukocytes. Superoxide anions produced in reperfused tissues result from incomplete reduction of oxygen by damaged mitochondria or because of the normal action of oxidases from invading inflammatory cells. These free radicals peroxidize the lipid contained within cell membranes, causing destabilization and increased permeability. Ultimately, loss of integrity of the microcirculation with fluid exudation into the interstitial space results in edema formation, muscle swelling, and raised intracom-

partmental pressure that eventually leads to compression of blood vessels and nerves.

Risk factors identified for development of compartment syndrome following revascularization are prolonged ischemic time, acute onset of ischemia, increasing age, and decreased arterial pressure. In addition, a combination of revascularization and deep vein thrombosis may be a further risk factor for the syndrome. Qvarfordt et al have shown that intracompartmental pressure and swelling after revascularization of an ischemic leg rises with a maximal increase on the sixth or seventh postoperative day.

The mainstay of diagnosis in compartment syndrome is to suspect it early to prevent irreversible damage to threatened muscle groups and nerves. The 6 Ps (pain, pressure, paresthesia, paralysis, pulselessness, and pallor) are used as a mnemonic for clinical diagnosis; however, all symptoms are rarely present. Because pain secondary to ischemia prior to revascularization can be difficult to discern from pain derived from compartment syndrome, pain alone is a nonspecific symptom. Pain from developing compartment syndrome is characteristically out of proportion to that expected in the clinical situation and is aggravated during passive stretching or compression of the muscle groups involved in a certain compartment. An additional complicating factor is the common finding of increasing pain and clinical signs of ischemia during the early stages of thrombolysis, sometimes referred to as the "storm before the calm." The localization of the pain to the calf rather than the foot and the presence of calf tenderness may aid in diagnosis of compartment syndrome.

Pressure refers to the tension felt during palpation of the involved limb, as the compartment is difficult to

Figure 2 Iliac endovascular interventions. (A) Fluoroscopic image shows balloon angioplasty of right external iliac artery. (B) Digital subtraction angiogram after angioplasty shows complete resolution of stenosis. (C) Fluoroscopic image shows balloon angioplasty of newly deployed stent in left external iliac artery. (D) Digital subtraction angiogram after dilation shows resolution of stenosis.

compress. Paresthesia is a very important early symptom and is an indicator of elevated intracompartmental pressure and impending nerve ischemia. The loss of two-point discrimination is a sensitive sign in differentiating compartment syndrome from raised intracom-

partmental pressure alone. The end point of paresthesia is paralysis, and is caused by prolonged nerve compression or irreversible muscle damage. Pulselessness is present if the diagnosis is delayed and represents a very late stage of the syndrome; however, pulses distal to a

Figure 3 Insertion of thrombolytic infusion catheter. Fluoroscopic image of thrombolytic infusion catheter into bypass graft.

compartment experiencing increased pressures will not necessarily be affected by the development of compartment syndrome. Pallor confirms lack of blood supply, but is an unreliable sign. In terms of laboratory tests,

Figure 4 Digital subtraction angiogram obtained in the operating room after thrombectomy at time of fasciotomy.

Figure 5 Illustration showing compartments in the calf.

increased levels of creatinine phosphokinase (CPK) above 1000 U or markers of myoglobinuria may indicate severe muscle damage and can be used as an additional tool to make a diagnosis; however, they are not appropriate for the early detection of compartment syndrome.

The measurement of intracompartmental pressure may be helpful when the clinical exam signs and symptoms of compartment syndrome are unclear, such as in a young child, or in an unconscious patient. However, it should be noted that it is not necessary if the diagnosis is clinically evident. It is important to measure all of the compartments included in the extremity of concern. The normal pressure in the muscle compartments is below 10 to 12 mm Hg, and pain and paresthesia first appear at pressures between 20 and 30 mm Hg. Mubarak et al used an intracompartmental pressure of 30 mm Hg as a basis for performing fasciotomy, identifying that the critical pressure of 30 mm Hg can be present only for a period of 6 to 8 hours before irreversible changes occur. It is theorized that the capillary pressure is insufficient to maintain muscle blood flow when the tissue fluid pressure is greater than 30 mm Hg.

Considered a surgical emergency, treatment of acute compartment syndrome requires emergent decompression by fasciotomy to prevent muscle infarction, nerve damage, and potential renal failure. Elevation of the limb is sometimes used by clinicians as a temporary measure in an attempt to reduce intracompartmental pressure, but has not been proven to be beneficial and may even increase the pressure in the limb. Because there is no definitive test to determine muscle viability, fasciotomy is advised whenever clinical signs of compartment syndrome are apparent. As a rule, most vascular surgeons have a very low threshold for performing fasciotomy if compartment syndrome is

suspected. If fasciotomy is performed within 12 hours of symptoms onset, it can prevent most muscle and nerve deficits and most patients regain excellent limb function. Finkelstein et al reported that fasciotomies performed after 36 hours from injury were often associated with severe infection, markedly increased amputation rates, and even death. In the leg, one should consider decompression of all four compartments: anterior, lateral, deep posterior, and superficial posterior. The fasciotomy can be performed using a single lateral incision or by a double incision technique, with medial and lateral incisions.

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