

Acute limb ischemia from gunshot wound secondary to arterial vasospasm

Corbin E. Goerlich, MD,^a Apurva B. Challa, MBBS,^a and Mahmoud M. Malas, MD, MHS, RPVI, FACS,^b
Batimore, Md

ABSTRACT

Gunshot wounds are rising in incidence, morbidity, and mortality. It is thought that about half of nonfatal injuries occur in an extremity. Although the incidence is not known, arterial vasospasm can result in acute limb ischemia. We present the case of a 33-year-old man who suffered a gunshot wound to the left lower extremity resulting in arterial vasospasm of the superficial femoral artery. He quickly regained arterial flow, and we were able to manage his acute limb ischemia nonoperatively and to document restoration of flow through serial examinations and Doppler imaging. He was subsequently discharged the next day and is experiencing a full recovery. (*J Vasc Surg Cases and Innovative Techniques* 2019;5:99-103.)

Keywords: Vasospasm; Acute limb ischemia; Gunshot wound; Trauma; Management

Gunshot injuries are rising in incidence, morbidity, and mortality.^{1,2} There are currently almost 70,000 instances of firearm-related injuries yearly in the United States in addition to >30,000 deaths annually. Of the nonfatal injuries, about 50% occur in an extremity.³ The exact incidence of traumatic vasospasm resulting in acute limb ischemia in adults from ballistic injuries is not known. At one level I trauma center, 82% of patients presenting with vasospasm resulting from trauma were younger than 10 years, and these cases are largely related to long bone fractures.⁴

Here we present a case of a 33-year-old man who presented to a level I trauma center within 1 hour after suffering a gunshot wound to the left lower extremity. This patient presented with acute limb ischemia, presumably from vasospasm of the superficial femoral artery (SFA) that resolved spontaneously. Whereas some would argue that regardless of etiology, all signs of acute limb ischemia should be explored surgically or endovascularly, we present a case in which conservative management was appropriate. We also review the literature for trauma-induced vasospasms and make a case for this mechanism as a true cause of acute limb

ischemia, albeit solely as a diagnosis of exclusion. We received the patient's written permission to publish this case.

CASE REPORT

A 33-year-old man was brought to the emergency department at approximately 10 PM with an isolated gunshot wound that traversed just above the left knee, with no apparent exit wound. He was hemodynamically stable and did not show signs of significant bleeding. The patient presented with stigmata of acute limb ischemia—a cold left extremity, no pulse, sensory loss to the knee without discrete dermatomal distribution, and paralysis. The patient did not know the caliber or type of gun he was assaulted with, but plain films of the left femur suggest it is a moderate-caliber bullet with a metallic fragment within the medial aspect of the distal left thigh with additional small metallic fragments overlying the distal femoral diaphysis without any acute fractures. Computed tomography angiography (CTA) of the lower extremity after discovery of the examination findings demonstrated no flow through the mid-SFA downward. Immediately after CTA, the patient was re-examined; the dorsalis pedis and posterior tibial arteries were still not palpable, and there were no audible Doppler signals (Figs 1 and 2). The patient was immediately posted to the operating room (OR) for emergent exploration of the left lower extremity for a presumed SFA lesion. However, before transport, the patient was again examined, and biphasic Doppler signals were heard. This was about 30 minutes after arrival and about 1.5 hours since the injury. It was decided instead of going to the OR to obtain further vascular studies to characterize the lesion as a vascular imaging laboratory was readily available adjacent to the OR.

The patient was taken for arterial duplex ultrasound and ankle-brachial index (ABI) studies for further characterization of the left lower extremity. Examination showed a biphasic signal, normal ABIs, and a toe pressure difference between extremities of 40 mm Hg. The right lower extremity was unremarkable (Fig 3). Findings on vascular ultrasound examination of the left

From the Department of Surgery, Johns Hopkins Hospital^a; and the Johns Hopkins Bayview Vascular and Endovascular Research Center.^b

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Correspondence: Mahmoud M. Malas, MD, MHS, RPVI, FACS, Vice Chair of Surgery for Clinical Research, Chief Vascular and Endovascular Surgery, University of California San Diego Health System, 9300 Campus Point Dr, MC 7403, La Jolla, CA 92037 (e-mail: mmalas@ucsd.edu).

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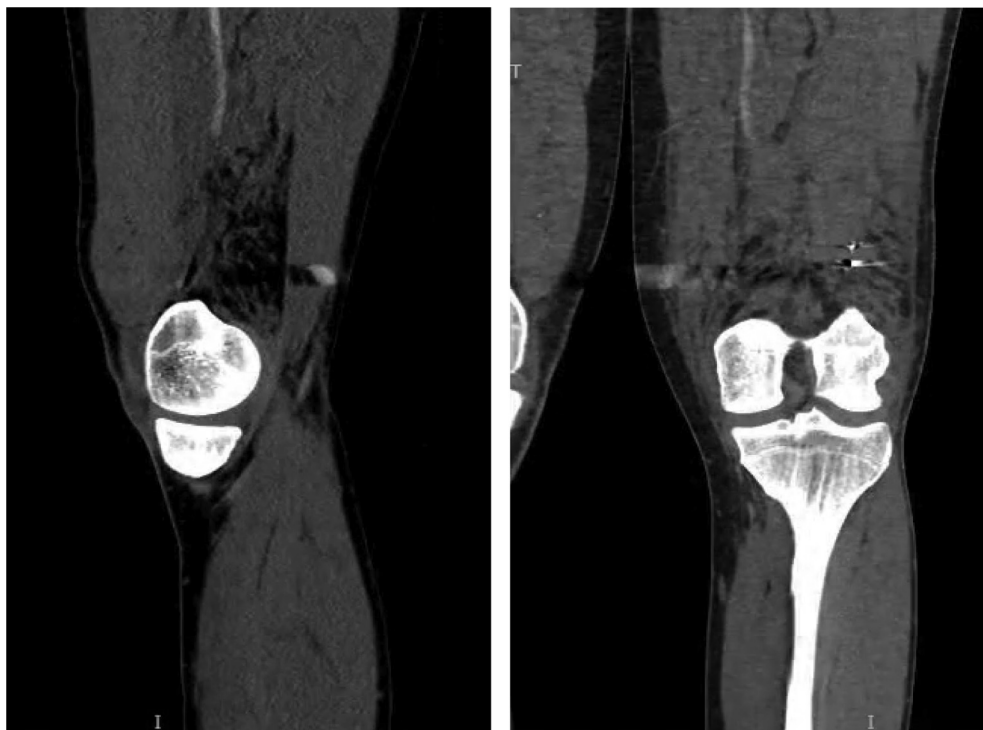


Fig 1. Sagittal and coronal views of abrupt narrowing of superficial femoral artery (SFA) at the level of soft tissue defect from gunshot wound.

lower extremity were also normal (Fig 4). This was approximately 1.5 hours from arrival. The affected leg showed no intimal flap, dissection, stenosis, aneurysm, or area of thrombus. The patient's vascular examination findings also continued to improve. Sensation of the foot returned around the time of the duplex ultrasound examination, and a palpable 1+ dorsalis pedis pulse was appreciated. It was decided, given the patient's reassuring examination and duplex ultrasound findings, that he could be observed overnight and monitored with serial neurovascular examinations and a repeated duplex ultrasound examination the following day. He was started on single antiplatelet therapy, and his examination findings continued to improve. By the morning, the patient exhibited palpable dorsalis pedis and posterior tibial pulses, 5/5 strength, and full sensation in the left lower extremity. Repeated Doppler ultrasound examination showed normal waveforms bilaterally and equal toe pressures and again exhibited no abnormal lesions to explain the vascular deficits in this patient. The patient was discharged and seen in outpatient follow-up and is making a full recovery.

DISCUSSION

Here we describe the case of a 33-year-old man who presented in a hemodynamically stable condition with an isolated gunshot wound to the left lower extremity. Whereas he initially exhibited signs of acute limb ischemia, they began resolving spontaneously. Most causes of acute limb ischemia in this setting result from an intimal flap, thrombosis or embolus, or

complete transection of a vessel; we have evidence to believe that this patient's acute limb ischemia resulted from vasospasm.

Vascular injuries of the extremity occur in about 30% to 45% of all traumatic vascular injury patients. Most of them occur in men, with an average age in the 30s, with a trend in the 20s in penetrating vascular injuries. About 15% to 45% of penetrating injuries are from firearms, 55% to 65% from stab wounds, and the remainder from other mechanisms, depending on the study. Mortality rate from penetrating vascular injuries is around 2.8%, largely from more proximal extremity or central vascular injuries.⁵⁻⁸

In terms of arterial spasms, there are many clinical scenarios in which this is a known and well-documented occurrence. Cerebral artery spasm secondary to subarachnoid hemorrhage results in delayed cerebral ischemia and is caused by irritation of cerebral vessels by surrounding blood products.⁹ Coronary artery spasms, known as variant angina or Prinzmetal angina, occurs in healthy coronary arteries or minimal coronary artery disease and can result in significant myocardial ischemia in otherwise healthy patients with minimal coronary risk factors.^{10,11} There are a variety of nonoperative modalities to treat these types of vasospasms.

There are case reports of vasospasm secondary to both penetrating and blunt trauma in the setting of adjacent fractures, and it would not be unreasonable to believe that large arteries of the extremities can also exhibit

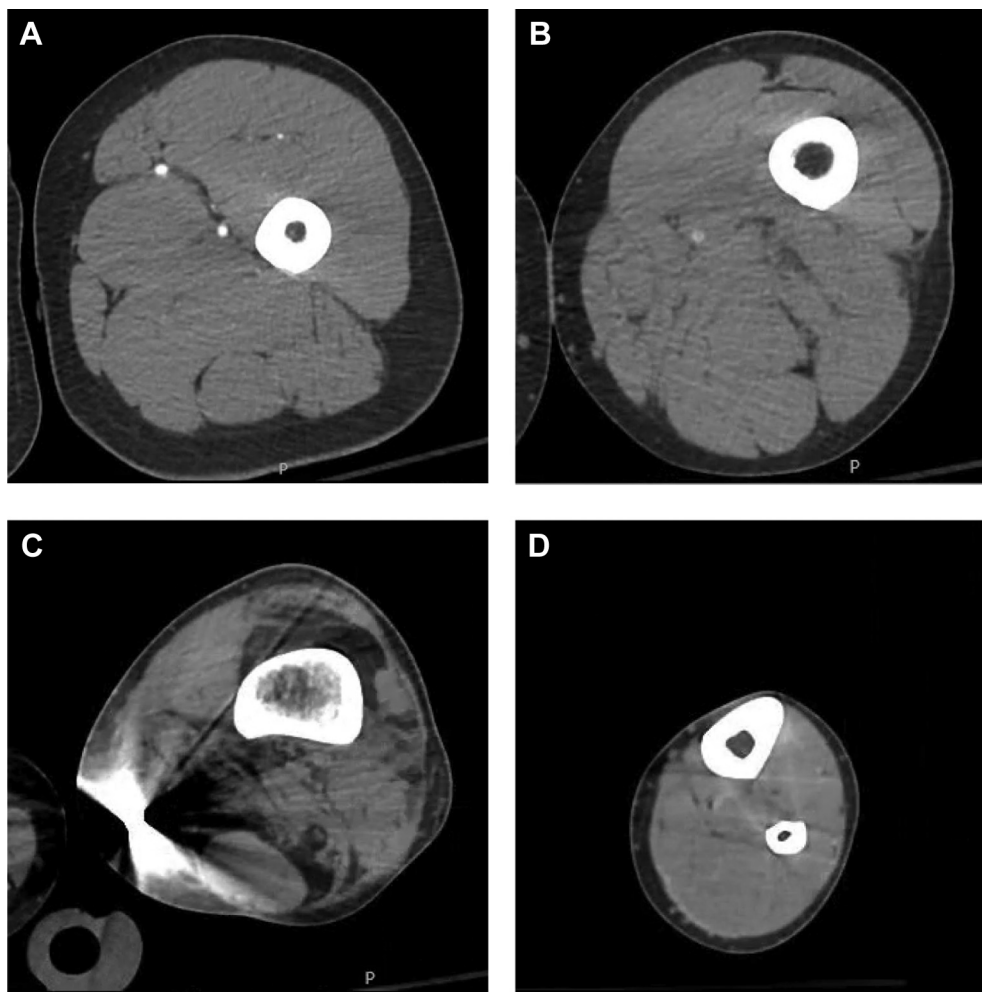


Fig 2. Axial view of superficial femoral artery (SFA) and deep femoral artery. **A**, Upper thigh with patent SFA and deep femoral artery. **B**, Cutoff of SFA just proximal to soft tissue defect from gunshot wound. **C**, Bullet visualized at distal thigh; no SFA visualized. **D**, Distal extremity without tibioperoneal trunk reconstitution.

these properties within proximity of ballistic injuries.¹²⁻¹⁴ Moreover, in a study of 93 patients who presented with blunt or penetrating vascular injuries, 41% of asymptomatic patients who exhibited signs of arterial spasm were managed nonoperatively. No patients were found to have delayed vascular injuries.¹⁵ Notably, the rate of ballistic injury-associated spasms resulting in acute limb ischemia is unknown.

The media layer of blood vessels, largely made up of smooth muscle, is known to respond to external stimuli. These smooth muscle cells can contract because of external compression or stretch (as seen during dissection of a vessel during surgery) or endothelial injury (like an arterial puncture) or dilate with mechanical vessel dilation. The extent by which a vessel actually contracts is determined by its burden of atherosclerosis and intrinsic response to internal and external stimuli. Thus, young, healthy vessels are more likely to spasm.

Hard signs of vascular injury should warrant surgical exploration. Hard signs include a pulse deficit, pulsatile bleeding, bruit, thrill, and expanding hematoma. Patients without hard signs of vascular injury or an ABI of <0.9 should be further evaluated. CTA can be used as an adjunct to further elucidate vascular injury patterns in a patient who is hemodynamically stable, and obtaining CTA does not extend ischemia time significantly.¹⁶ Although we used Doppler imaging in our vascular laboratory because it was readily available, this should not be used if it would cause delay in timely decision-making for the patient.

In this case, the patient had an improvement on vascular examination from hard signs of vascular injury on arrival to return of a Doppler signal in the affected extremity by the time he was being transported to the OR. This afforded an opportunity to obtain further imaging to elucidate the mechanism of injury, with a low threshold of going to the OR if there was further

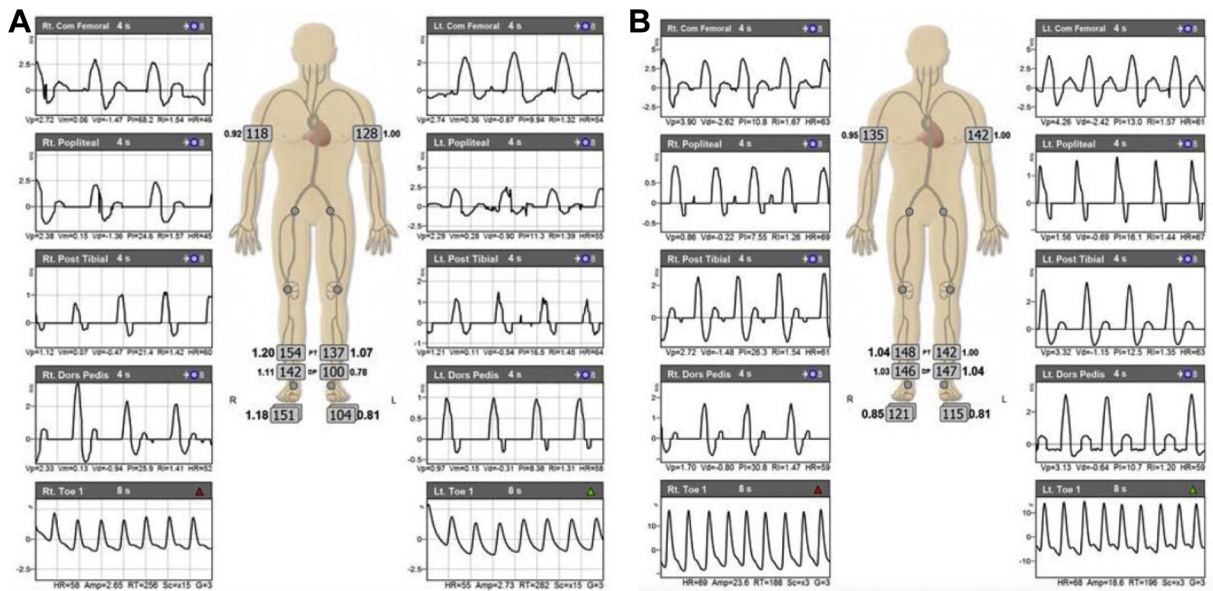


Fig 3. Doppler waveforms, ankle-brachial indices (ABIs), and toe pressures, initially (A) and the following day, 15 hours later (B).

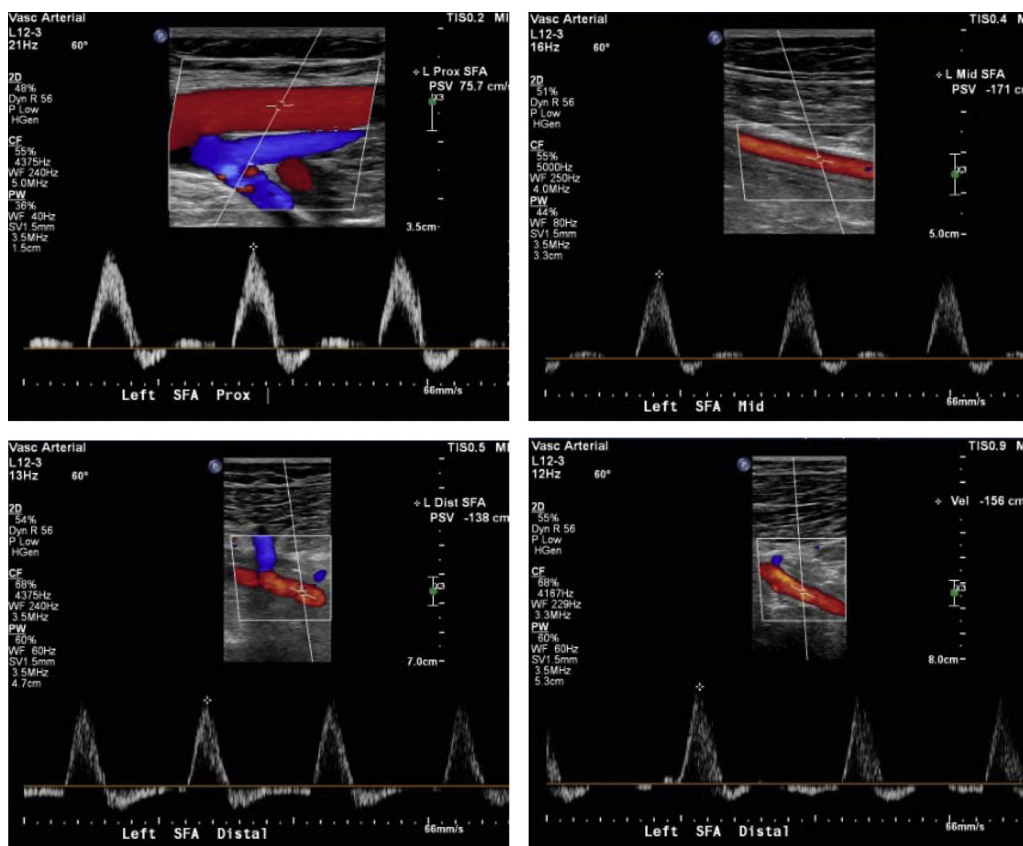


Fig 4. Doppler ultrasound of left lower extremity superficial femoral artery (SFA) showing no dissection, intimal flap, or other signs of vascular injury.

suggestion of a vascular injury. Whereas it is certainly not standard of care to perform nonoperative management for a patient with hard signs of vascular injury, this patient presented with a unique set of circumstances

that proved his vascular injury (spasm) was improving and lacked any other injuries, such as a neurologic injury, nerve compression, expanding hematoma, or compartment syndrome. Moreover, his limb ischemia

time was about 1.5 hours from arrival and a little more than 2 hours from the onset of injury. We were able to further characterize his lesion solely because he rapidly regained signs of perfusion in his extremity and his acute limb ischemia reversed spontaneously within a timely fashion. This also afforded us the ability to safely treat this patient with nonoperative management.

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