Title
Acute limb ischemia from gunshot wound secondary to arterial vasospasm.

Permalink
https://escholarship.org/uc/item/36c8p66h

Journal
Journal of vascular surgery cases and innovative techniques, 5(2)

ISSN
2468-4287

Authors
Goerlich, Corbin E
Challa, Apurva B
Malas, Mahmoud M

Publication Date
2019-06-01

DOI
10.1016/j.jvscit.2018.10.004

Peer reviewed
Acute limb ischemia from gunshot wound secondary to arterial vasospasm

Corbin E. Goerlich, MD, Apurva B. Challa, MBBS, and Mahmoud M. Malas, MD, MHS, RPVI, FACS

Baltimore, 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 overlaying 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 extremity were normal except for additional metallic fragments in the distal left femur.
lower extremity were also normal (Fig 4). This was approxi-
mately 15 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 fire-
arms, 55% to 65% from stab wounds, and the remainder
from other mechanisms, depending on the study. Mor-
tality 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 sce-
narios in which this is a known and well-documented
occurrence. Cerebral artery spasm secondary to sub-
arachnoid 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
these properties within proximity of ballistic injuries.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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

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

REFERENCES
1. Sakran JV, Mehta A, Fransman R, Nathens AB, Joseph B, Kent A, et al. Nationwide trends in mortality following penetrating trauma: are we up for the challenge? J Trauma Acute Care Surg 2018;85:160-6.
2. Tasigiorgos S, Economopoulos KP, Winfield RD, Sakran JV. Firearm injury in the United States: an overview of an evolving public health problem. J Am Coll Surg 2015;221:1005-14.
3. Fowler KA, Dahlberg LL, Haileyesus T, Annest JL. Firearm injuries in the United States. Prev Med 2015;79:5-14.
4. Shah SR, Wearden PD, Gaines BA. Pediatric peripheral vascular injuries: a review of our experience. J Surg Res 2009;153:162-6.
5. Loh SA, Rockman CB, Chung C, Maldonado TS, Adelman MA, Cayne NS, et al. Existing trauma and critical care scoring systems underestimate mortality among vascular trauma patients. J Vasc Surg 2011;53:359-66.
6. Mattox KL, Feliciano DV, Burch J, Beall AC, Jordan GL, De Bakey ME. Five thousand seven hundred sixty cardiovascular injuries in 4459 patients. Epidemiologic evolution 1958 to 1987. Ann Surg 1989;209:698-705; discussion: 706-7.
7. Perkins ZB, DeAth HD, Aylwin C, Brohi K, Walsh M, Tai NR. Epidemiology and outcome of vascular trauma at a British Major Trauma Centre. Eur J Vasc Endovasc Surg 2012;44:203-9.
8. Shackford SR, Sise MJ. Peripheral vascular injury. In: Moore EE, Feliciano DV, Mattox KL, editors. Trauma. 8th ed. New York: McGraw-Hill Education; 2017. Available at: accesssurgery.mhmedical.com/content.aspx?aid=1141190602. Accessed May 25, 2018.
9. Findlay JM, Nisar J, Darsaut T. Cerebral vasospasm: a review. Can J Neurol Sci 2016;43:15-32.
10. Beller GA. Calcium antagonists in the treatment of Prinzmetal's angina and unstable angina pectoris. Circulation 1989;80(Suppl):IV78-87.
11. De Luna AB, Cygankiewicz I, Baranchuk A, Fiol M, Birnbaum Y, Nikus K, et al. Prinzmetal angina: ECG changes and clinical considerations: a consensus paper. Ann Noninvasive Electrocardiol 2014;19:442-53.
12. Davis CM, Gayle MO. Delayed presentation of traumatic arterial vasospasm. Pediatr Emerg Care 1999;15:22-4.
13. Peach G, Antoniou G, El Sakka K, Hamady M. Traumatic arterial spasm causing transient limb ischaemia: a genuine clinical entity. Ann R Coll Surg Engl 2010;92:e3-4.
14. Gazmuri RR, Muhoz JA, Ilic JP, Urtubia RM, Glucksmann RR. Vasospasm after use of a tourniquet: another cause of postoperative limb ischemia? Anesth Analg 2002;94:1152-4.
15. Gillespie DL, Woodson J, Kaufman J, Parker J, Greenfield A, Menzoian JO. Role of arteriography for blunt or penetrating injuries in proximity to major vascular structures: an evolution in management. Ann Vasc Surg 1993;7:145-9.
16. Fox N, Rajani RR, Bokhari F, Chiu WC, Kerwin A, Seamon MJ, et al. Evaluation and management of penetrating lower extremity arterial trauma: an Eastern Association for the Surgery of Trauma practice management guideline. J Trauma Acute Care Surg 2012;73(Suppl 4):S315-20.