Traumatic Arteriovenous Fistula of the Leg

An Easily Missed Diagnosis

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An arteriovenous fistula is an abnormal communication between the arterial and venous systems. It may be an incidental finding in an asymptomatic patient or it may manifest clinically with pain, edema, varicosities, and even heart failure. It is important for clinicians to be aware of this disorder because early diagnosis and treatment can prevent its long-term sequelae. This report presents a patient with a posttraumatic arteriovenous fistula.

KEY WORDS. Arteriovenous fistula; wounds and injuries; leg. *(J Fam Pract 1998; 46:247-250)*

An arteriovenous (AV) fistula is defined as an abnormal communication between the arterial and venous system. It may be congenital, or acquired secondary to trauma, tumors, or as a result of surgery. Although the physiological effects of traumatic fistulas have been well characterized, their clinical manifestations are quite variable due to differences in location, size, and duration.[1] As a consequence, the diagnosis may be delayed. Although physical findings are suggestive, an angiography will confirm the diagnosis and is necessary when planning surgical treatment.

We present the case of a 43-year-old woman with ongoing leg pain who was eventually discovered to have a traumatic AV fistula. This uncommon condition may be overlooked earlier in its course when characteristic clinical features may be absent. This patient was initially evaluated for neurological disease and, when that was excluded, referred for psychiatric care.

Case History

A 43-year-old woman presented to the office with a chief complaint of left leg pain that had been present on an intermittent basis during the past 3 years. The pain, most prominent over the calf and lower leg, was sharp, nonradiating, and was exacerbated by prolonged standing. There was associated numbness, heaviness, and nocturnal leg movements. She complained of fatigue, but was able to care for her family and work part time as a housecleaner. She denied back, neck, or chest pain, palpitations, peripheral edema, recent trauma, claudication, or Raynaud's phenomenon.

In October 1994, 3 years before this office visit, she presented to a neurology clinic with similar leg pain and was hospitalized for a possible herniated lumbar disc. An extensive battery of blood chemistries and radiologic tests were performed, including lumbar puncture, syphilis serology, thyroid function, antinuclear antibody, B_{12} and folate levels, serum protein electrophoresis, urine for heavy metals, pelvic ultrasound, computed tomography of the abdomen and pelvis, magnetic resonance imaging of lumbar spine, and electromyography of leg and spine. All results were within normal levels. However, Doppler studies of arterial and venous flow in the lower extremities revealed some mild abnormal signals in the left inguinal region. Despite this finding, she was referred to a psychiatrist. Her condition was subsequently diagnosed as a somatoform disorder with psychogenic pain secondary to dysthymia, and she was given a prescription of fluoxetine 20 mg daily and clonazepam 1 mg at bedtime for sleep.

A second neurological opinion was sought in January 1995, because of continued leg discomfort. The diagnosis of leg pain of unknown etiology was made and treated with amitriptyline. Three months of therapy provided no improvement. A third neurological consultation in April 1995 resulted in the treatment of restless leg syndrome with carbidopa and levodopa, which did not ease her pain. In May 1995, she was referred for psychological evaluation and treated for major depression. After 2 months of antidepressant therapy, she returned with worsening pain, visible swelling, and a pulsatile mass on the lateral aspect of her left leg. A venous Doppler study performed at that time was reported as normal. Her complaints were believed to be psychosomatic and she was referred to a pain clinic in July 1995.

Submitted, revised, August 12, 1997.
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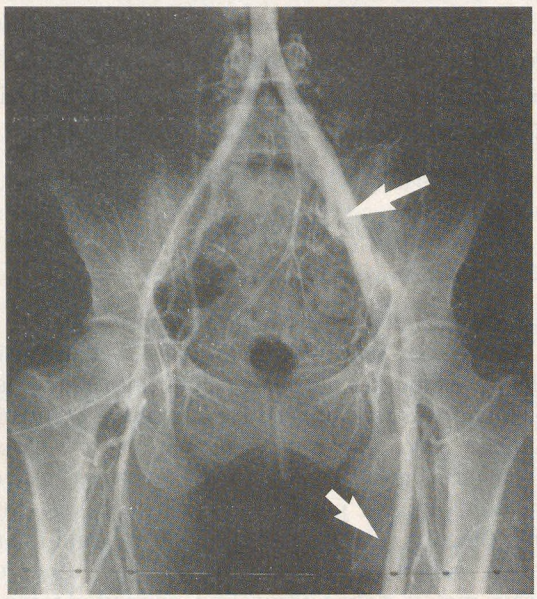
FIGURE 1

An arteriogram showing enlarged iliac artery (large arrow) and femoral artery (small arrow) on the left.



One month later she presented as a new patient to our clinic with the history of leg pain as previously described. Her physical examination was normal at that time. Four months later, in December 1995, she returned to our office after 2 days of severe, throbbing, left leg pain associated with edema and numbness. She complained that the pain was worse than ever before. She recalled that 15 years ago, a nail was thrown from a lawn mower and penetrated her left calf.

Physical examination was significant for edema, a palpable thrill and pulsatile mass over the lateral aspect of the leg, and a bounding popliteal pulse. Auscultation over this area revealed a continuous systolic-diastolic murmur. An arterial and venous Doppler study demonstrated the absence of a triphasic wave form in the popliteal artery, common femoral and superficial femoral arteries on the left, as well as increased flow and triphasic wave forms in their associated veins. An arteriogram was subsequently performed and it demonstrated an enlargement of the external iliac, common femoral, and superficial femoral arteries on the left (Figure 1), and an AV fistula from the peroneal artery to the deep calf veins (Figures 2 and 3). Also noted was delayed flow into the left foot, primarily through the posterior tibial artery with some collateral formation noted at the left ankle.

The patient was referred to a vascular surgeon. Her treatment consisted of ligating the peroneal artery and associated vein proximal and distal to the fistula. The result was ablation of the thrill. At the postoperative follow-up 2 weeks later, the patient's principal complaints were of moderate swelling in the left leg and diminished sensation in an area lateral to the incision site. She has since stopped taking antidepressants and is otherwise well.

Discussion

The most common causes of traumatic AV fistulas are penetrating injuries into adjacent arteries and veins.[2] Today, these lesions are largely sequelae of medical procedures.[1] Iatrogenic fistulas have been reported to follow phlebotomy,[1] percutaneous biopsy of the kidney, cardiac catheterization, nephrectomy,[3] and orthopedic procedures.[4] In contrast, penetrating trauma from knife stab wounds, gunshot injuries, and shrapnel fragments account for the largest percentage of cases in times of war. Occasionally, AV fistulas may follow a blunt trauma that causes a fracture whereby bone fragments lacerate adjacent vessels.[5]

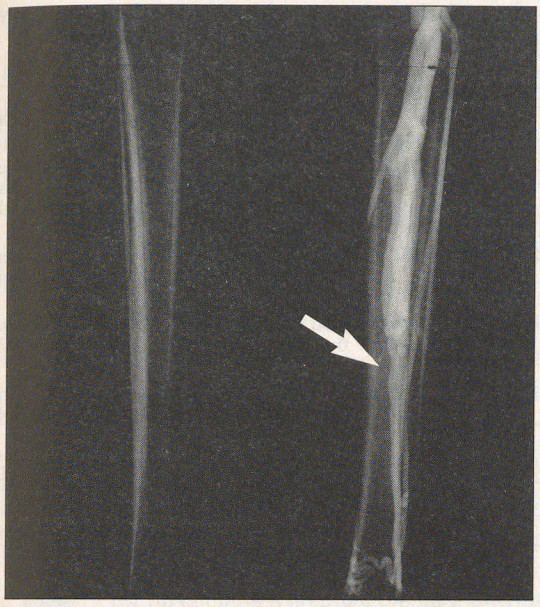
The incidence of AV fistula formation in vascular injuries ranges from 2.3% to 3.9%.[2] In one published series,[3] the most frequently reported locations of AV fistulas were in the extremities (43%), of which 17% involved the femoral vessels. The second most prevalent site involved the head and neck region (33%), of which carotid-jugular (13%) and carotid-cavernous (11%) fistulas were most common. This was followed by visceral fistulas (24%), most often involving the renal vessels (10%).

Clinically significant physiologic changes occur as a consequence of the shunting of arterial blood to the venous system. The first is the formation of collateral vessels around the fistula in response to diminished distal arterial pressure.[2] If severe, symptoms and signs of ischemia may occur.[2] Second, increased blood flow through this direct communication increases cardiac workload and, if sufficiently large, may ultimately lead to heart failure.[1]

The diagnosis of an AV fistula can frequently be established clinically. The patient will usually have a history of trauma, particularly penetrating injury.[1] Although occasionally asymptomatic,[7] an AV fistula will often cause a patient to complain of a pulsatile mass, pain, edema, and a vibratory sensation over

FIGURE 2

An arteriogram showing an arteriovenous fistual (arrow) from the peroneal artery to deep calf veins on the left.



the fistula.[1] Rarely, signs and symptoms of heart failure may be the initial dramatic presentation.[5] Auscultation over the fistula reveals a "machinery" murmur, so called for its continuous to-and-fro sound.[1] An associated thrill and pulsatile mass is found in more than half of patients with long-standing AV fistulas.[5] Edema, superficial varicosities, and an elevated skin temperature overlying the fistula may be apparent on examination.[6] In chronic AV fistulas, signs of venous stasis including increased pigmentation, ulcerations, and induration may be present.[2] The patient may also exhibit Branham's sign. This is a decrease in heart rate that occurs in response to the manual compression of the fistula or the artery proximal to the fistula. This is an arterial baroreceptor reflex triggered by the temporary increase in systolic blood pressure that occurs with closure of the fistula by this maneuver.[1]

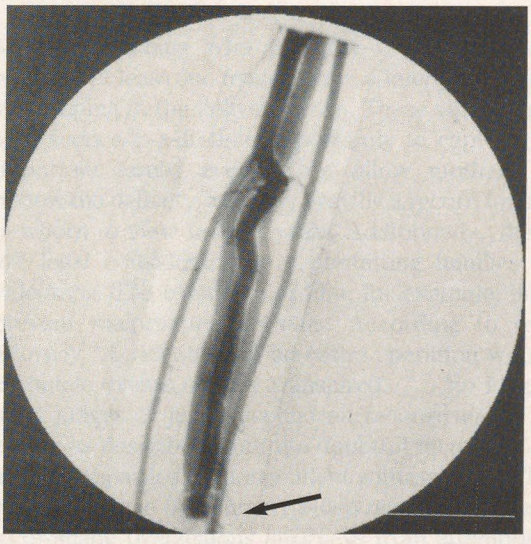
The workup of an AV fistula may include angiography, Doppler examination, duplex scanning, computed tomography, and magnetic resonance imaging. Angiography is the most definitive diagnostic technique.[2] In addition to localization, it delineates the size, anatomy, and hemodynamic effects of a fistula.[2] Although invasive, an angiogram is suggested when the diagnosis is clinically evident[1] and when surgical treatment is being considered.[2] Angiography is also recommended in the setting of acute penetrating trauma adjacent to major blood vessels because a physical examination alone may miss nearly half of traumatic AV fistulas.[2]

Initial evaluation by noninvasive methods may be preferred when an AV fistula is suspected but the clinical presentation is not typical. A Doppler study may reveal an increased velocity in the proximal artery or a pulsatile flow pattern in the proximal veins, substantial evidence that a lesion is an AV fistula.[2] Duplex scanning, which is felt to be the best noninvasive study to diagnose a fistula,[2] provides the same basic information but has the added advantage of combining real-time, B-mode ultrasound images of blood vessels with a color-coded analysis of Doppler signals.[1] Computed tomography with intravenous contrast and magnetic resonance imaging have also been used in the study of AV fistulas. They provide anatomical information regarding size, location, and involvement of adjacent structures. Magnetic resonance imaging has the added advantage of not requiring intravenous contrast and of being able to image in coronal and sagittal planes.[1]

The preferred treatment is surgical correction. Ideally, the surgeon will close the fistula and restore continuity to the involved artery and vein.[2] When the fistula arises from a nonessential artery, proximal

FIGURE 3

An arteriogram providing a magnified view of the arteriovenous fistula. Note the aneurysmal dilatation at the site of the fistula (arrow).



and distal ligation of the involved artery is sufficient for obliteration.[1] In some cases, catheter embolization or balloon occlusion has allowed treatment of fistulas previously considered inoperable.[2]

There are many factors that may have accounted for the delayed diagnosis in this patient. On initial encounter, she may have been perceived as a chronic complainer. Her long history of leg pain that appeared to have been extensively evaluated and treated unsuccessfully by many competent physicians could lead many physicians to this conclusion. Additionally, the relative rarity of this condition and the physicians' unfamiliarity with it may have been a factor. This would seem to be plausible given that many of the findings, when evaluated retrospectively, pointed clearly to the presence of an AV fistula, the exception being the report of a normal Doppler study. As the films were not available for review, the accuracy of their interpretation cannot be substantiated. A final reason for missing the diagnosis may be related to the natural history of an AV fistula. What may have begun as a very small arteriovenous communication with little or no signs and symptoms, progressively enlarges with time to become more symptomatic and more easily recognized.

REFERENCES

1. Rutherford RB, ed. Vascular surgery. 4th ed. Philadelphia, Pa: WB Saunders Co, 1995.
2. Gloviczki P, Hollier LH. Arteriovenous fistulas. In: Haimovici H, Ascer E, Hollier LH, Strandness Jr DE, Towne JB, eds. Haimovici's vascular surgery, principles and techniques. 4th ed. Cambridge, Mass: Blackwell Science, 1996:870-92.
3. Kollmeyer KR, Hunt JL, Ellman BA, Fry WJ. Acute and chronic traumatic arteriovenous fistulae in civilians. Arch Surg 1981; 116:697-702.
4. Quigley TM, Stoney RJ. Arteriovenous fistulas following lumbar laminectomy: the anatomy defined. J Vasc Surg 1985; 2:828-833.
5. Robbs JV, Carrim AA, Kadwa AM, Mars M. Traumatic arteriovenous fistula: experience with 202 patients. Br J Surg 1994; 81:1296-9.
6. Kontos HA. Vascular diseases of the limbs. In: Bennett JC, Plum F, eds. Cecil textbook of medicine. 20th ed. Philadelphia, Pa: WB Saunders Co, 1996: 346-57.
7. Coy JD. Delayed diagnosis of post-traumatic arteriovenous fistula from trip wire-detonated booby trap in Vietnam. Mil Med 1991; 156:636-7.