Anomalous dual posterior tibial arteries

Ryan P. Lin, MD,[a] Rachel N. Rohrich, BS,[a] Sami Ferdousian, MS,[a] *and* Cameron Akbari, MD, MBA,[a,b]
Washington, DC

ABSTRACT

Congenital vascular anomalies present significant diagnostic and therapeutic challenges in the scope of vascular surgery. We present a case of anomalous dual posterior tibial (PT) arteries in a diabetic patient with an infected nonhealing foot wound. The clinical, radiologic, and pathological results are presented. We report on a case of a 45-year-old patient with type 2 diabetes mellitus and an infected nonhealing ulceration on the left heel and midfoot. Given concern for ischemic ulceration, an angiogram was performed revealing anomalous dual PT arteries. This rare variant of PT artery anatomy presents challenges in initial diagnosis and limb salvage. (J Vasc Surg Cases Innov Tech 2025;11:101752.)

Keywords: Anomolous; Posterior tibial artery; Embryology

Congenital vascular anomalies present significant diagnostic and therapeutic challenges in the scope of vascular and plastic reconstructive surgery. Unrecognized vascular anomalies can complicate procedures such as bypass, angioplasty, and trauma management, potentially leading to increased morbidity or mortality. Approximately 10% of the United States population has a popliteal branching pattern variation, the most common being a hypoplastic or aplastic posterior tibial (PT) artery, trifurcation of all three tibial vessels at the same level, or high origin of the anterior tibial (AT) artery.[1-3] Rarely, the PT artery may arise from the peroneal artery.[4] We report a case of dual PT arteries providing dominant run-off to the foot. Consent was obtained from this patient to publish their case details and images.

CASE REPORT

A 45-year-old male patient with uncontrolled type 2 diabetes mellitus and hypertension was admitted with concern for an infection of his chronic nonhealing ulceration on the left heel and midfoot. His last hemoglobin A1C level was 9.4 despite compliance with diabetic medication and insulin regimen. The patient never underwent previous lower extremity computed tomography angiography (CTA) or percutaneous angiography. On examination, he had nonpalpable left dorsalis pedis (DP) and PT pulses, a noncompressible ankle-brachial index, and there was concern for acute osteomyelitis of the left calcaneus and fifth metatarsal bone.

After discussion with the multidisciplinary limb salvage team including plastic and reconstructive surgeons, maximal blood flow to the foot needed to be ensured given the significant tissue loss, acute osteomyelitis, and need for planned foot and heel procedures. The patient underwent a left lower extremity arteriogram, revealing a rare vascular anomaly: dual PT arteries providing dominant tibial runoff to the foot. The AT artery was occluded from just beyond its origin to the distal leg, with reconstitution at the ankle continuing as the DP and lateral tarsal arteries (Fig 1). Both PT arteries were patent and independently supplied the plantar vessels. The peroneal artery was also patent to the ankle. Initial attempts to traverse the long segment AT occlusion were unsuccessful and further intervention was aborted. A joint decision was made with the multidisciplinary team to attempt angioplasty of the AT artery for future limb salvage purposes. Additionally, the wound located on the lateral aspect of the foot was in the angiosomal distribution of the lateral tarsal artery. The patient subsequently underwent successful left AT percutaneous transluminal angioplasty via retrograde left DP access. Retrograde access was performed given inability to traverse the AT artery occlusion from an anterograde approach. Final angiography demonstrated patent AT artery, dual PT arteries, and peroneal artery with flow to the foot (Figs 2 and 3). Dual antiplatelet therapy was started. A postangioplasty examination revealed palpable DP, nonpalpable PT pulses, and biphasic DP and PT signals.

The patient underwent a left partial calcanectomy and fifth metatarsal head resection. Plantar vessels were preserved during calcanectomy. He underwent additional debridements but, owing to repeated positive wound cultures and osteomyelitis, the wound was left open. No local ischemia was noted. The patient was discharged home with long-term intravenous antibiotics, nonweightbearing instructions, daily dressing changes, and close follow-up in our tertiary, multidisciplinary wound healing clinic. Although he was compliant with weight bearing and wound care, failure of wound healing was likely caused by calcaneal location healing difficulty, poor diabetes control, microvascular disease, and infection. He later presented with significant soft tissue infection and required an urgent below-the-knee amputation. At his most recent postamputation visit, the surgical site was healing without issue.

DISCUSSION

Within a human embryo, vasculogenesis of the lower limb begins with modification of splanchnic mesodermal cells into angioblasts. Driven by vascular endothelial growth factor, these angioblasts then develop

From the Department of Plastic and Reconstructive Surgery,[a] and the Department of Vascular Surgery,[b] MedStar Georgetown University Hospital.

Correspondence: Ryan P. Lin, MD, Georgetown University Hospital, 3800 Reservoir Rd, NW, Washington, DC 20007 (e-mail: ryan.lin@medstar.net).

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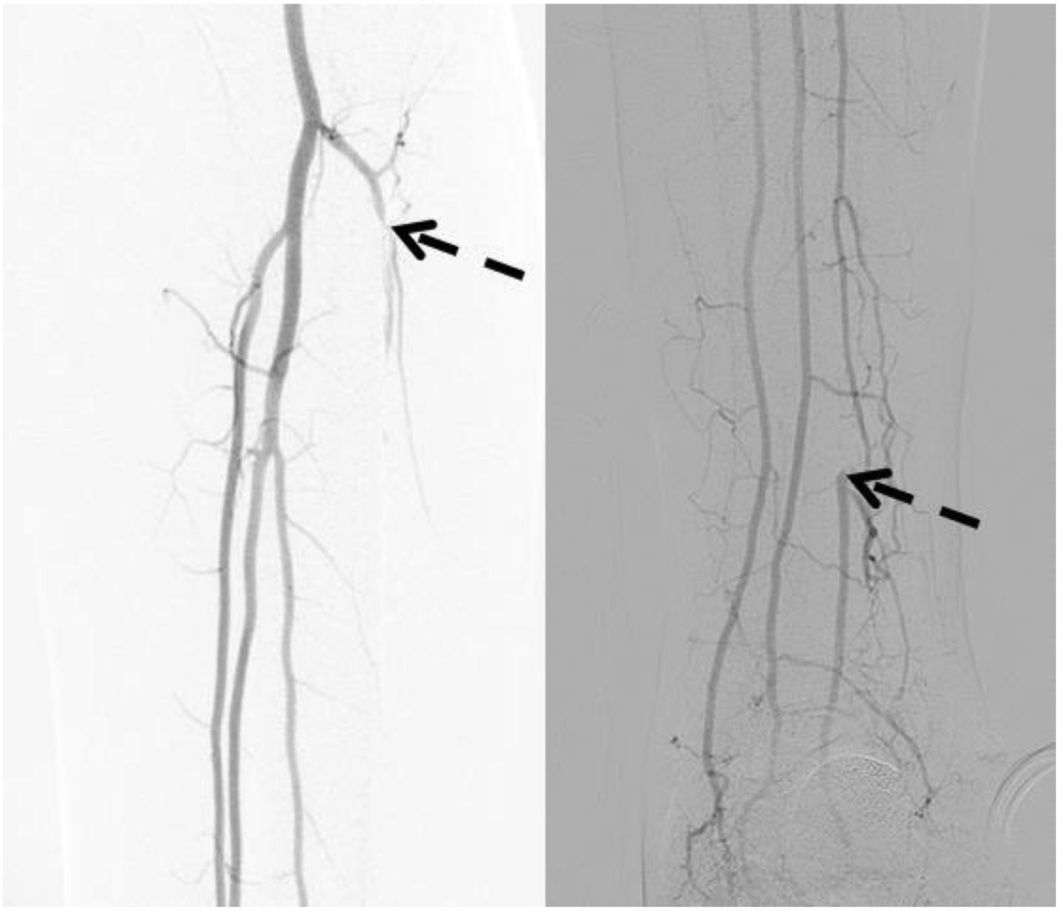

Fig 1. Left tibial arteriogram showing long-segment occlusion of the anterior tibial (AT) artery with distal reconstitution pre-angioplasty (interrupted *black arrow*).

into flattened endothelial cells and form small vessel cords that then form a primitive capillary plexus.[5] Now as vascular elements and in response to hypoxia-inducible factor 1α and vascular endothelial growth factor, endothelial cells will undergo angiogenesis, forming simple capillary networks, which later becomes a robust vascular network.[6] Variations in these molecular processes via genetic predisposition, environmental factors, and failure of regulation can ultimately lead to vessel hypoplasia, aplasia, or duplication.[6]

In the lower limbs, during embryogenesis, the sciatic (axial) artery arises from the internal iliac artery and provides perfusion to the lower limb. The external iliac artery forms as a second branch of the fifth lumbar intersegmental artery, developing into the iliofemoral artery which penetrates into the lower limb after the axial artery.[6] By the eighth week, the sciatic artery regresses and the external iliac artery develops into the common femoral artery. A portion of the sciatic artery persists as a segment of the popliteal and peroneal artery. Simultaneous to the interosseus artery regression, an anastomosis between the early distal femoral artery and the popliteal artery forms the PT artery.[7]

Normal popliteal branching anatomy involves the popliteal artery dividing inferior to the knee into the AT and tibioperoneal trunk. The PT and peroneal artery then bifurcate from the tibioperoneal trunk. At the level of the ankle, the PT gives off the medial plantar, lateral plantar, and medial calcaneal arteries.[6] Embryonic vascular development determines anatomic variability of these popliteal branches from degenerating, persisting, nonunion, or abnormal fusion of vessels.[7] Double PT anomalies and their implications are not well-represented in the literature. Additionally, dual PT arteries do not fall under any popliteal branching variation category described by Kim et al[3] in a study of 1000

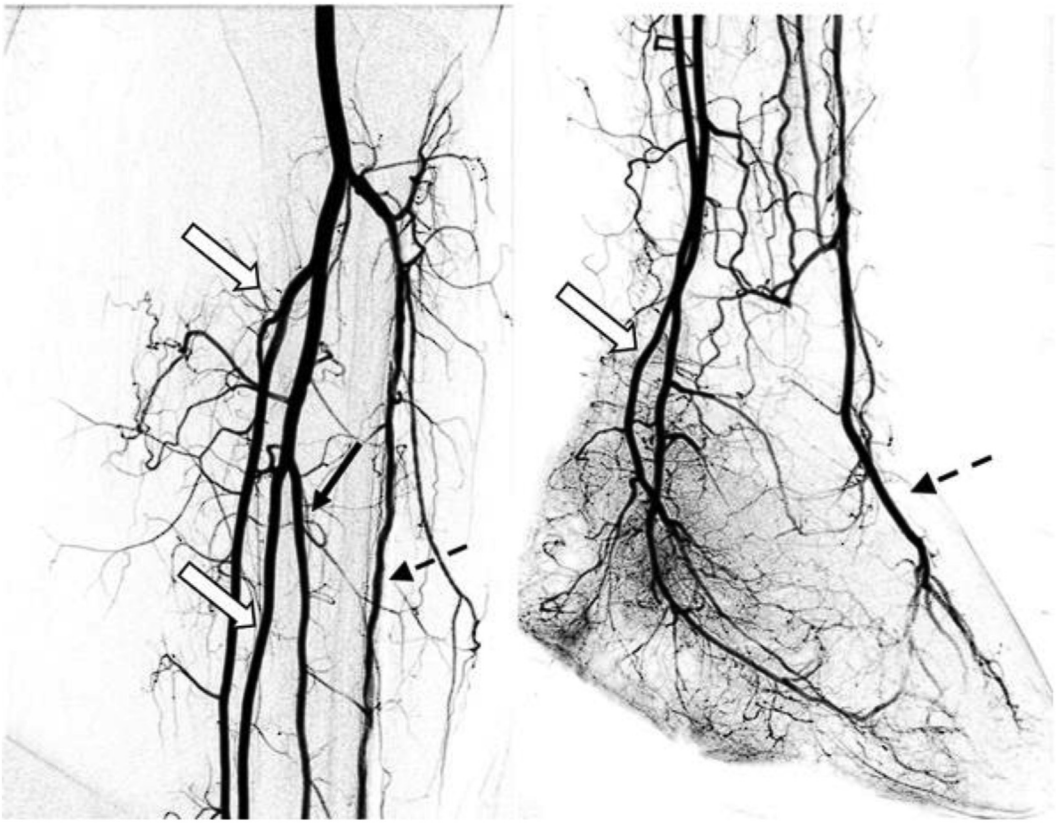

Fig 2. (Left) Left tibial arteriogram following angioplasty of the left anterior tibial (AT) artery. Thick white arrow denotes the dual posterior tibial (PT) arteries (*superior arrow*: arising from the tibioperoneal trunk and inferior arrow: arising from the peroneal artery). Solid *black arrow* denotes the peroneal artery. Interrupted *black arrow* denotes the AT artery. (Right) Lateral view of the left ankle demonstrating in-line flow to the foot from the dual PT arteries (thick white arrow) and the newly patent AT artery (interrupted *black arrow*).

arteriograms. Summary of popliteal branching variants is displayed in the Table.[3]

Anomalous tibial vessels are typically discovered on CTA, arteriograms, or dissection of cadaveric specimens. If one of the PT arteries was to be injured or occluded, then possibly the other PT artery may provide adequate runoff to supply the foot. Conversely, failure to recognize dual PT arteries in a patient with known vasculopathy may lead to limb ischemia or life-threatening hemorrhage should the aberrant artery be injured. In the setting of a free flap or bypass, dual PTs may provide an anastomotic option if recognized. In the case presented, the additional PT may have aided in delaying ischemic necrosis of the plantar wound.

If a patient with a known double PT variant requires a procedure on their contralateral lower extremity, a similar variant in that extremity must be considered. In patients with anatomical variation in one limb, contralateral variation may present in ≤50% of patients.[8] During free flap or arterial bypasses, adequate preparation and knowledge of the patients' vascular anatomy is essential to prevent accidental vascular trauma. Noninvasive imaging such as lower extremity arterial duplex ultrasound examination or CTA can be performed to prevent unanticipated encounter with a double PT anomaly in the contralateral limb of a patient with a known double PT variant.[9] If a patient has a known double PT and has trauma to the contralateral lower leg causing persistent bleeding despite three tibial vessels identified operatively, then a contralateral double PT must be considered.

In summary, we present a unique case of dual PTs on lower extremity arteriogram. Detailed vascular imaging facilitated comprehensive considerations if limb salvage or revascularization is required. A vasculo-plastic approach may help to prevent or manage vascular trauma in the setting of double PT anomaly. Overall, this case highlights the need for vigilance in detecting

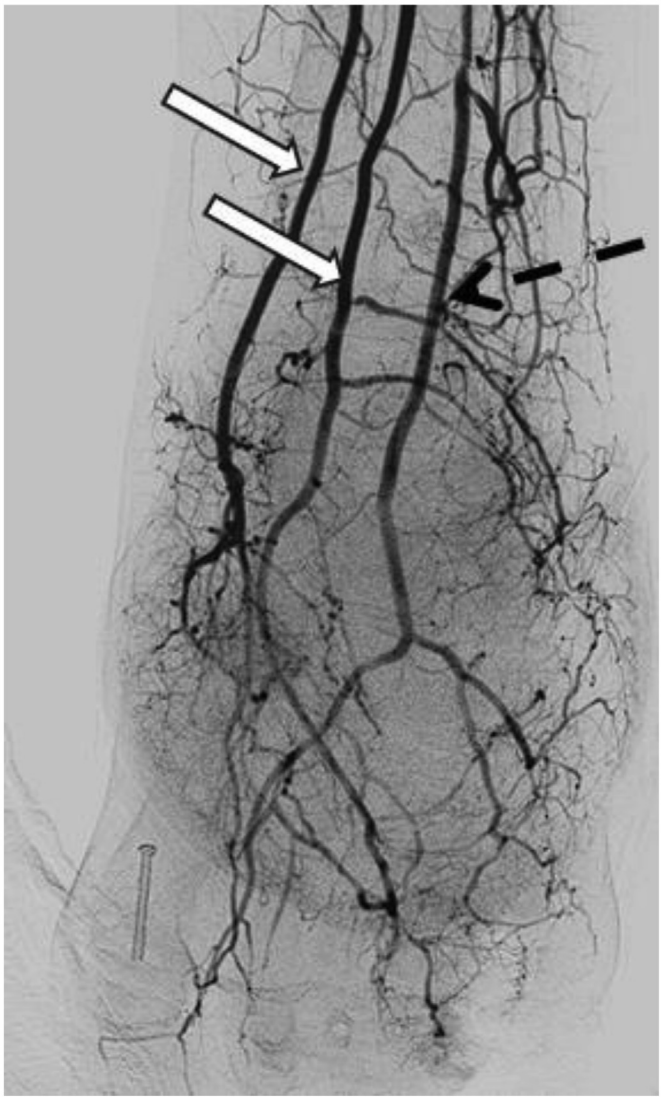

Fig 3. Anteroposterior view of the left ankle with in-line flow from the dual posterior tibial (PT) vessels (*white arrows*) and the anterior tibial (AT) artery (interrupted *black arrow*).

vascular anomalies on imaging and to consider implications of double PT anomalies on the patient's need for future procedures.

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DISCLOSURES

None.

Table. Summary of popliteal artery variants, with an additional category for duplicated tibial arteries

Type	Description
1	Normal level of branching
1A	Usual pattern
1B	Trifurcation
1C	Anterior tibioperoneal trunk
2	High division of popliteal artery
2A	High division of AT artery
2B	High division of PT artery
2C	High division of peroneal artery
3	Hypoplastic or aplastic branching with altered distal supply
3A	Aplastic/hypoplastic PT artery
3B	Aplastic/hypoplastic AT artery
3C	Aplastic/hypoplastic PT artery and AT artery
4 (novel)	Duplicated PT, peroneal, or AT arteries

AT, Anterior tibial; *PT*, posterior tibial.

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