A Case Report of Unusual Angiographic Findings in the Setting of a Chronic Medial Calcaneal Ulceration: A Potential Observation of a Competitive Flow Mechanism in the Foot?

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Statement of Purpose and Literature Review

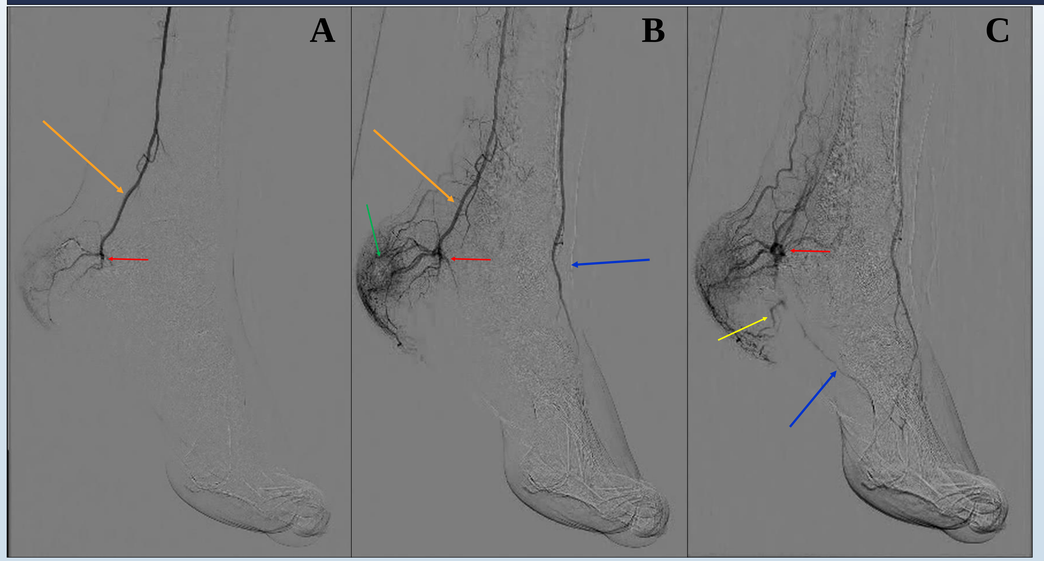
Arterial blood flow is essentially a matter of basic physics. Blood responds to and follows a hemodynamic balance from areas of high pressure to low pressure, in turn leading to distal tissue perfusion. Generally in the diabetic foot, a relatively *static* resistance is encountered in the form of **atherosclerotic occlusion**. This can be explained by Poiseuille's Law [(Pressure Difference)x(Vessel Radius^4) / (8/π) x (Viscosity) x (Length)], where for example, a 20% occlusion increases resistance and reduces the rate of flow by 50%.

Relatively *dynamic* imbalances are also possible potentially leading to distal ischemia. An **AV fistula** is an example where blood flows directly from a high-resistance artery to a low-resistance vein, bypassing distal tissue perfusion and resulting in localized ischemia and the possibility for tissue loss. In diabetic patients, this phenomenon may be exaggerated due to medial arterial calcification and arterial narrowing, resulting in even higher resistances proximal to the fistula site.

Another similar *dynamic* situation exists known as **competitive flow** that is seen following cardiac artery bypass grafting. In this situation, the distal perfusion by a low resistance bypass graft may "push out" arterial flow in an area of tissue normally supplied by a native artery with relatively high resistance caused by occlusive disease. Both arteries have the potential to supply the given anatomic location because of the anastamotic patterns of the heart (potentially similar to the infrapopliteal trifucation anastamosis patterns and penisular anatomy in the foot), but one artery typically supplies the tissue of a location (similar to the angiosome theory). A localized area of increased hemodynamic pressure (from the high flow/low resistance bypass graft) pushes out creates an area of localized ischemic tissue normally supplied by another artery (from the high resistance/low flow diseased native cardiac vessel).

We present a case report of unusual angiographic findings of a seemingly dynamic area of localized ischemia manifested as a cold spot on the angiogram and as a chronic wound clinically. We suspect an idiopathic increase in local tissue hemodynamic pressure is causing a small area of ischemia corresponding to the anatomy of the tissue loss.

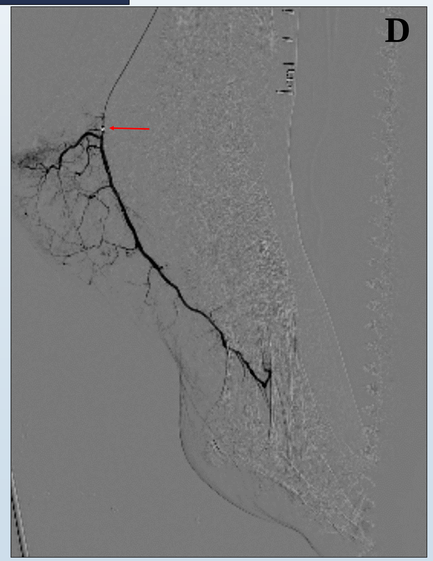
Endovascular Imaging



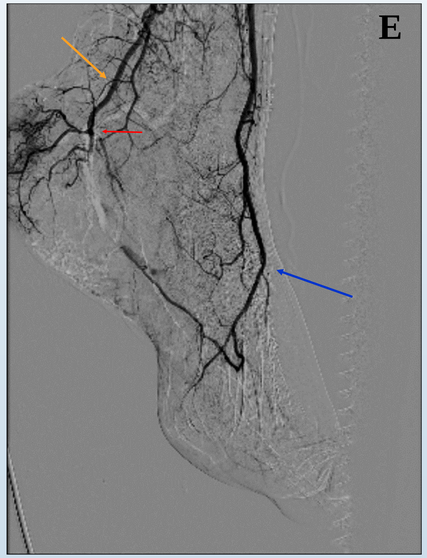
This first series of images is from a diagnostic angiogram. The posterior tibial (PT) artery initially fills (orange arrows) to a specific point near the flexor retinaculum on the medial foot prior to bifurcation into the medial and lateral plantar arteries. This inflow then stops at a specific location without progression into the plantar foot (red arrows).

The PT artery does not progress past this point but does fill the heel where a blush in the area of the wound can be appreciated (green arrow). As the distal run-off continues, the anterior tibial (AT) artery now begins to fill on the dorsum of the foot (blue arrow).

The PT artery continues to fill the posterior foot (even with eventual communication into the peroneal vein [yellow arrow]), but never extends past the one specific location. The AT artery continues to fill the plantar foot in a retrograde manner (blue arrow). A relative "dead zone" of hypoarterial flow exists near the wound location on the medial heel.



A balloon angioplasty is subsequently performed in the PT artery across this location. The wire easily crosses the unusual "stop point" (red arrow) of the PT and normal antegrade filling of the plantar artery is observed if dye is infiltrated just distal to this location.



Following the PT artery angioplasty, the same filling pattern is observed with antegrade flow to the heel through the PT artery to the "stop point", antegrade flow to the dorsum of the foot through the AT artery, retrograde flow to the plantar foot through the AT artery, and a relative "dead zone" of hypovascularity on the medial heel near the clinical area of the ulceration.

Case Report

A 63 y/o non-ambulatory insulin dependent diabetic female with a history of hypertension, dyslipidemia, cirrhosis, emphysema and glaucoma presented to the Temple University Foot and Ankle Institute Center for Advanced Wound Healing with a long history of a right lateral ankle ulcer and an eschar on the medial aspect of the left heel, extending onto the plantar weight-bearing surface. After a recent hospitalization with prolonged immobilization she developed an eschar that eventually broke down to a full thickness pressure ulceration. The dorsalis pedis pulse was palpable, but the posterior tibial pulse was non-palpable. Non-invasive testing demonstrated an absolute pressure of 125mmHg in the posterior tibial with a corresponding index of 1.15.

Figures A, B and C demonstrate the diagnostic angiogram. The PT artery (orange arrows) initially fills on the run-off and stops at a specific spot near the medial heel (red arrow) prior to bifurcation into the plantar arteries. Although the posterior heel fills (green arrow) from the PT and the peroneal vein eventually fills (yellow arrow), the PT artery does not fill the plantar foot in an antegrade manner. The AT artery (blue arrow) eventually fills the dorsum of the foot in an antegrade manner and the plantar foot in a retrograde manner. Interestingly, a crossing wire and balloon angioplasty is easily performed across the PT artery and fills normal plantar artery anatomy distal to the red arrow. (Figure D). The post-angioplasty run-off (Figure E) demonstrates a relatively well-perfused foot given the retrograde filling of the plantar artery from the AT, but a consistent zone of hypoperfusion and local ischemia near the heel distal to the red arrow which corresponds to the clinical location of the ulceration (Figure F).

Discussion

Our intention was to present a case of unusual angiographic findings which corresponded to pathologic clinical anatomy, but without an apparent avenue for endovascular intervention. Although we identified a vascular abnormality with localized hypoperfusion on the angiogram, balloon angioplasty was unable to restore flow to the medial heel despite the plantar arteries demonstrating the potential for normal vascular anatomy. We suspect some form of increased hemodynamic pressure in the area from an alteration produced by the wound itself, an unvisualized fistula, and/or a what we suspect is a competitive flow mechanism. This dynamically contributes to a local ischemia which we suspect to be both the condition leading to the ulceration and the main factor preventing its closure. The wound is responding poorly to standard and advanced wound interventions, it is by definition chronic, and could clearly benefit from increased perfusion. Whatever the etiology of our idiopathic findings, we hope that this case report at least demonstrates the importance of angiogram interpretation with an eye on angiosomes, matching the specific anatomic location of tissue loss with perfusion to a specific source artery or anastamotic pattern.

References

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