Understanding the Tibial-Pedal Arterial Anatomy: Practical Points for Current Clinical Presentations

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Abstract: The lower extremity arterial vasculature encompasses a balanced, tapering distribution closely related to the muscular compartments of the leg and foot. From the main ilio-femoral inflow to further remote capillary vessels, a harmonious pyramid of gradually tapering limb perfusion is created. This impressive vascular system is structured into several ranks of tapering vessels that correspond with angiosomes in the lower extremities. Each of these levels expresses unique anatomic features when threatened by atherosclerotic ischemic disease. As atherosclerosis progressively worsens, the vascular anatomy, including the tibio-pedal trunks, foot arches, and collateral distributions, continuously provides coordinated support and dynamic adaptations in regional perfusion of the foot, in accordance with various endogenous and exogenous factors. Critical limb ischemia (CLI) involves characteristic infragenicular patterns of arterial disease and related compensatory flow paths that are useful for interventionists to acknowledge and understand. The present article briefly reviews the main anatomical features describing infra-popliteal arterial vasculature with, and without, ischemic impairment, in accordance with practical clinical issues encountered in daily practice.

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The lower limb arterio-venous vasculature has a gradually tapering distribution, with around 91% of cases showing typical patterns of vasculature and 9% with anatomical variations, and is closely related to the muscular components of the leg.[1,2] Arterial vasculature of the calf and foot gathers three main vascular bundles: the anterior, the posterior, and the peroneal arteries. These arteries correlate with four distinct anatomical compartments in the calf, and nine others in the foot, and are associated with roughly sixteen corresponding inframalleolar bundles.[1-3]

In addition to this balanced compartmental distribution, the lower limb arterial tree follows specific areas of tissue framed in characteristic vascular modules, known as "angiosomes."[4] Similar to genuine muscular compartmental orientation, the angiosome partition expresses topographic reproducibility in humans.[4-6] The angiosomal branches are not indivisible, or "terminal" ramifications of the entire arterial tree. [4,5,7] They are millimetric branches that further divide in smaller divisions ("final ramifications"), [4,5] before reaching the arteriolar level with specific, topographically oriented zones of tissues. From the main ilio-femoral flow sources, throughout the angiosome branches, and down to the capillaries, a harmonious "pyramid of gradual limb flow distribution" is created. [4,5]This vascular system is structured in several levels of tapering vessels (Levels I to VI)[7] toward specific angiosomes.[7,8] Each of these levels continuously provides coordinated and dynamic adaptations in regional perfusion, in accordance with various endogenous and exogenous factors.[7-10]

Every bifurcation becomes progressively thinner than its parent trunk.[9,10] Each arterial path progressively branches into inferior degrees of segmentation that ultimately creates a wider cross-sectional area toward peripheral tissues and increases the amount of perfusion to the tissue.[9,10]

It is important to note that even in the presence of sparse arterial anatomical variants (9%–12%),[1,2] the limb maintains steady vascular distribution among all compartments, angiosomes, and their collateral networks.[1,5-7] No random flow is observed among the calf perfusion sectors, or between the dorsal and the plantar territories of the foot.[2,6,7] Appropriate knowledge anatomical features of the lower limb is beneficial for the interventionist. Such knowledge facilitates diagnostic solutions in various presentations of ischemic limbs, as well as a better perspective of outcomes when planning revascularization for optimal tissue regeneration.[6-8]

MAIN TIBIAL TRUNKS

The anterior tibial artery (AT) originates at the interosseous membrane of the calf as the first principal infragenicular arterial branch. At this level, it reveals a constant angulation (of changing degrees in individuals), "the hook." Calcifications may commonly be encountered at this anterior crossing point[6-8] between distinct leg compartments. This calcification is thought to be due additional stiffness and turbulences that are induced by the surrounding fibro-

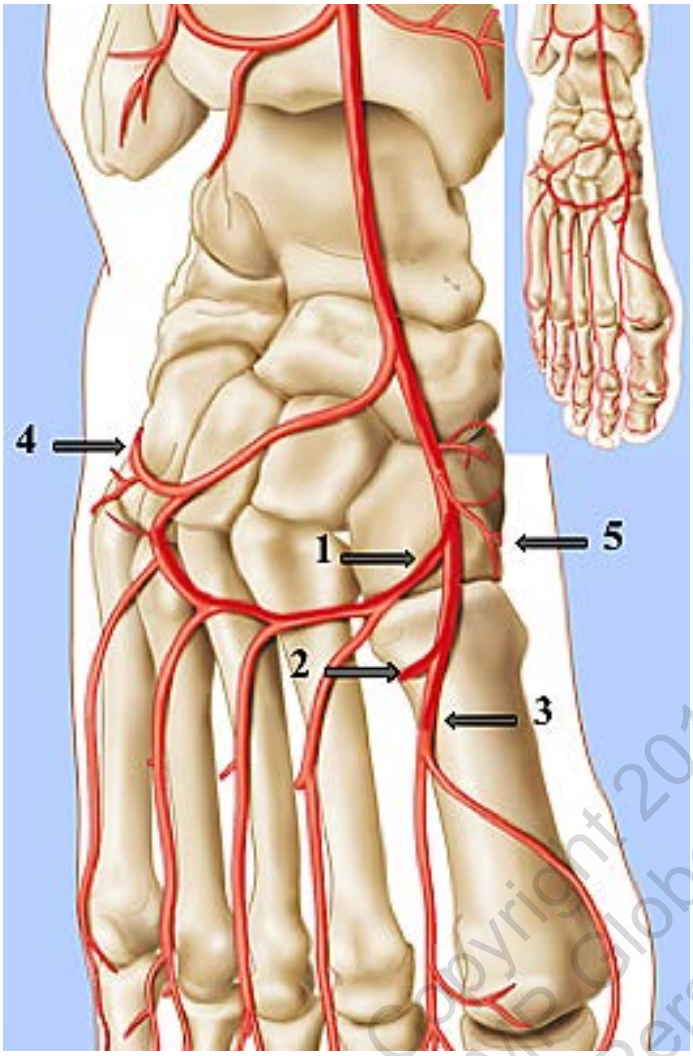

Figure 1. Schematic representation of the dorsalis pedis artery and its main branches:
1. Arcuate artery.
2. Deep plantar artery.
3. First dorsalis metatarsal artery.
4. Lateral tarsal (diagonal) arteries.
5. Medial tarsal arteries.

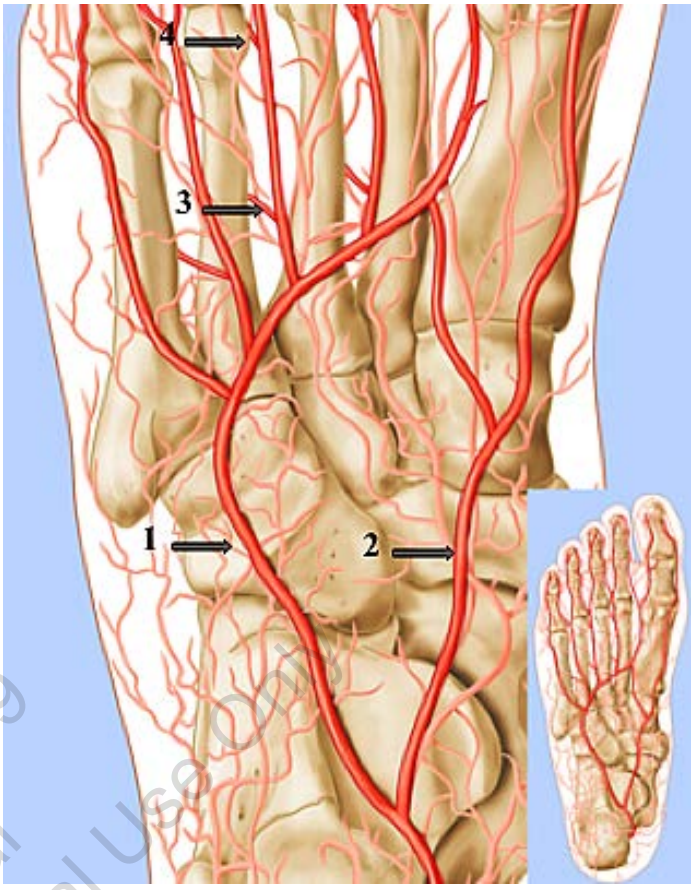

Figure 2. Schematic representation of the main branches of the plantar foot arteries:
1. Lateral plantar artery and plantar arch.
2. Medial plantar artery.
3. Proximal perforating arteries.
4. Distal perforating arteries.

tendinous structures.[7] The AT artery courses within the anterior compartment of the lower leg and foot and is associated with relatively uncomplicated interventional and surgical access for revascularization.[5,11-13]

Interestingly, according to the remarkable anatomical description by Taylor, muscles in the anterior compartment of the lower limb, and also in the dorsal foot are only supplied by one specific AT angiosome.[5] This high-value information can assist in better understanding of certain ischemic wound presentations in the presence of stenotic AT flow and related loss of collaterals.[5,7] It also can facilitate better planning for regional revascularization.

At the ankle level and underneath the extensor retinaculum of the foot, the AT transitions into the dorsalis pedis (DP) branch. This zone of flow towards the pedal circulation represents a second area of increased flow turbulences and a higher risk of local atherosclerotic occlusive disease along the course of the vessels.[7,12]

Both the AT and DP provide flow to the superficial and deep structures (DP angiosome) of the dorsal aspect of the foot, up to the toes.[4-6] The AT also supplies the anterior peri-malleolar ankle perfusion.[4-6] The AT terminates at the first dorsal metatarsal space by dividing the arcuate artery, an influential compensatory vessel of the dorsal angiosome that also affects the entire forefoot and distal limb preservation.[6-8,14,15] At the same level, the DP creates the first dorsal metatarsal artery and the deep plantar artery. These three DP-dependent branches are large collaterals (around 1 mm diameter) and provide a weighty local compensatory flow of > 80 mL/min.[10,12,16]

Anatomical variations. According to a recent meta-analysis by Kropman and colleagues that included 7671 cases, atypical calf and foot arteries were observed in approximately 7.9% to 10% of individuals.[1] High origins (at the popliteal level) of AT, or atypical

tibial trifurcations, were reported in 5.6% to 6.2% of individuals, while abnormal DP origins were found in 4.3% to 6% of cases.[1,2,7] An anomalous first dorsal metatarsal artery origin, associated with atypical first toe collateral perfusion, was described in 8.1% of individuals, concomitant abnormalities of the arcuate artery in 5%, and variants of plantar arches and plantar arteries in 5%.[1,2,7,12] The presence of one atypical tibial or pedal presentation on one leg should alert the interventionalist to a 21% risk of encountering similar abnormalities on the contralateral extremity.[1,2,7] Although it is useful to acknowledge these abnormalities, these anatomical variants may prompt a more detailed local angiosomal flow evaluation, yet only lead to small changes in wound-targeted revascularization.[6-8] This strategy follows and adapts to every available local collateral network, with or without uncharacteristic anatomical features.[7]

Practical issues. Large DP collaterals (+/- 1 mm diameter) on the lateral side of the foot (the "lateral tarsal" or "diagonal arteries") connect the AT territory to the lateral plantar branches that belong to the posterior tibial artery (PT), in an effective regulatory system.[5-8,17] In cases of DP thrombosis in patients with unaffected diagonal vessels, healing of dorsal foot and anterolateral ischemic wounds can be observed as a result of these collateral branches.[6,8,17] Conversely, with thinner (< 1 mm) and less available collaterals on the medial aspect of the foot (medial tarsal arteries), the same DP dysfunction seldom allows recovery of dorsomedial CLI ulcers, and wounds improve only via indirect, medial plantar collateral support.[6,7,17]

The posterior tibial artery (PT) bifurcates the tibio-peroneal trunk (TPT), 2-3 cm distally from the AT emergence. The PT courses along the deep posterior compartment of the calf where current surgical[15,18] or endovascular approaches[11,19] for revascularization procedures can be initiated. A higher frequency of long (>15 cm) calcific obstructions in the segment of the PT appears to be more prevalent in diabetic and renal patients.[13,17] At the ankle level, in the retro-malleolar zone, the PT crosses the retinaculum of the flexor muscles of the foot, a transition zone towards the fixed plantar circulation.[10,17] This high shear-stress zone (similar to the adductor ring for the superficial femoral artery, or the extensor retinaculum for the AT),[16,17] equally inflicts local turbulence of flow and chronic endothelial injuries that may lead to a higher prevalence of atherosclerotic disease.[10,13,17] After releasing its medial calcaneal branch, the PT bifurcates at the plantar aspect of the foot, into the medial and the lateral plantar arteries. The lateral plantar vessel represents an important, large caliber (1-1.5 mm) terminal PT bifurcation that further creates the deep plantar arch. Both foot arches share vital compensatory flow via the deep plantar artery, an important trifurcation branch from the DP.[4-6] The PT, via its medial calcaneal branch, and through the medial and lateral plantar source arteries, provides angiosomal topographic flow for the plantar portion of the foot and toes, in addition to providing 70% of perfusion in the heel.[5-7,16]

Anatomical variations. According to the meta-analysis by Kropman and colleagues, PT native variants can be found in about 6.8% of individuals.[1] Among these variations, PT artery hypoplastic, aplastic, or high emergences were observed in 3.3% of cases. TP dominance (absence of the AT artery) was documented in 1.5% of cases,[1,2] whereas atypical plantar arch and plantar arteries were seen in 5% of cases.[1] In atypical cases, the vast majority of the time, the plantar vessels have a peroneal origin.[1]

Practical issues. As mentioned for the dorsal foot and the arcuate artery (DP/AT), the lateral plantar artery (PT) holds a parallel and key role for the plantar side of the foot. Probably among the most difficult ischemic foot lesions to treat by purely hemodynamic means are those located at the hallux level.[6-8] The hallux and the first interdigital space territories are an important collateral hub of the forefoot.[7,10,17] This zone is a watershed area from at least two or three neighboring angiosomal "source arteries." These watershed arteries are the first dorsal metatarsal artery (DP/AT), and the media and lateral plantar arteries (PT).[1,6,17] Critical ischemic wounds/necrosis confined to this level are often expressions of a wider and multilevel occlusive disease, located upstream of the pedal vessels.[6,13] Necrotic lesions detected in this foot territory frequently indicate severe disease of the plantar and forefoot collateral web, and critical injury of more than half of all native compensatory hallux interdigital collaterals.[7,14,17]

In the anterior and posterior tibial-pedal arterial vasculature, specific "high shear-stress" flow zones have been described. These zones seem preferentially exposed to severe atherosclerosis, chronic occlusions, and heavy calcifications.[8,13] Therefore, the "flexor retinaculum" passage (concerning the PT), the interosseous membrane transition point (the AT), and also the "extensor retinaculum" (the AT), all represent constant challenging zones for endovascular techniques,[8,13] via either antegrade or retrograde passages and approaches.[11-13]

The peroneal artery (PA) supplies the lateral compartment of the leg. The PA is often seen as a "rescue" revascularization trunk, as it shows less significant atherosclerotic occlusive disease in the common CLI context. Accordingly, it can support current surgical[15,18] or more demanding endovascular transcutaneous approaches[11,19] for reperfusion. Despite traveling in the deep posterior compartment of the calf, the PA ends superficially by its lateral calcaneal branch, a "terminal-type" branch that provides 30% of the heel perfusion.[10,16]

From a clinical perspective, the peroneal artery provides two important collateral branches at the ankle level: the anterior and the posterior communicants that join anterior and PT arteries, respectively, in a high-value collateral rescue network.[6-9,15-17]

As an angiosomal "source arteries" provider, the peroneal trunk lends flow to a more narrowed zone of the lateral heel via its lateral calcaneal artery, and also to the anterolateral ankle via its anterior perforating branch and source artery.[4-6]

Anatomical variations. The peroneal artery shares fewer independent abnormal distributions than those described among all tibial trunks. Most cited variants are associated with a high peroneal origin from a dominant calf peroneal trunk in hypoplastic or aplastic PT presentations (+/- 3%).[1,2]

Practical issues. In the CLI context, the peroneal trunk currently has fewer calcifications than the AT or PT, with higher technical

accessibility for surgical or endovascular techniques for limb salvage.[13,17,22] Large-caliber anterior and posterior communicants may provide good filling in the foot arches, although only in isolated collateral patterns.[13,16,17] Accordingly, some authors have labeled the PA as "the best artery to treat," particularly in the multifaceted diabetic foot context.[20-22] Although the PA can provide an effective rescue supply for most CLI Rutherford 4 presentations,[20-22] its usefulness in healing Rutherford 5-6 forefoot or hindfoot complex tissue lesions by unspecific indirect revascularization remains questionable.[14,17,23-25] Meticulous preoperative angiographic assessment may enable us to identify and utilize every individual peroneal flow distribution and collateral partition when planning wound-targeted revascularization.[7,13,17]

PEDAL ARCHES, OR TRUE REDEEMERS OF THE ISCHEMIC FOOT

The deep plantar arch originates from the PT via the lateral plantar artery, while the dorsal foot arch arises from the DP. Following a transverse plane of examination, the plantar arterial arch has a few millimeters more distal foot location than the dorsal arch regarding the location of the toes.[10] Arches are located in deep tissues of the dorsal or plantar aspects of the foot. Occasional direct access for revascularization at this level assumes experienced surgical or interventional teams and equipment.[11,12-15, 26]

Functional foot arches in single or tandem afford "wound-directed revascularization" (WDR) via either antegrade or retrograde distal limb perfusion. Both foot arches are closely interconnected by a large number of collaterals at three main levels: tarsal, metatarsal, and at the toe position. Among these connections, four proximal and four distal (<1 mm diameter) perforating arteries are located at the digital level.[6,10] Their compensatory role is completed by the deep plantar artery (+/- 1 mm diameter) from the DP towards the plantar vessels. These perforating arteries augment arterial supply from the medial and lateral tarsal arteries (+/- 1 mm diameter) in normal conditions.[4-7,17]

Anatomical variations. Foot arches share main anatomical variations that were also described in the dorsal foot or plantar vessels.[2] Atypical plantar arches and plantar arteries were observed in 5% of cases, whereas arcuate artery and DP variants were found in 6% to 8% of daily cases.[1,2]

Practical issues. The integrity of dorsal and plantar arches is of paramount importance for forefoot and midfoot ischemic tissue recovery.[12-15] Yet, appropriately functioning arches do not preclude the topographic importance of foot angiosomes in revascularization.[6,7,17, 24]

An efficient limb reperfusion strategy should achieve in-line flow from the iliac up to the level of the foot arches. Even if Lisfranc or Chopart forefoot amputations are necessary, the maintenance of functional arches is important for correct post-ischemic stump recovery and rehabilitation.

Patent arches were described by some authors as more significant than angiosomal orientation for CLI wound healing and limb salvage.[15] For common forefoot multi-angiosomal ischemic lesions, the presence of one (occasionally two) patent arches implies that at least one patent angiosomal "source arteries"[6,14] (from the dorsal or the plantar sides) perfuses to the foot and toes. Pedal arch reperfusion demonstrates the important role they play in CLI treatment, particularly for topographic foot revascularization.[6,17,24, 25]

ANGIOSOMES OF THE LOWER LEG

Specific source arteries. Taylor and Palmer pioneered a vascular model in humans that is visualized as a "continuous 3D network" of vessels nourishing specific, topographically assigned tissues.[4,5] These networks came to be termed "angiosomes." These 3D structures of skin and underlying deep structures are perfused by specific source arteries that are found in most individuals.[4-6] Each category of source arteries varies in length, density, and caliber, according to different territories of the human body.[4-6] Interestingly, regarding the inferior limb perfusion, beyond the specific source arteries, most adjacent muscles receive secondary arterial branches from two or three neighboring angiosomes.[4,5] These native features, as well as the presence of a vast, compensatory collateral system attached to main flow bundles, inspired a series of controversies in contemporary CLI angiosome-dedicated literature.[7,17,25]

The territories of the lower leg share the flow belonging to six angiosomes (source arteries and appended collaterals), that have been described as follows:[4-6] the AT artery and appended DP angiosome supplies the anterior facet of the ankle, the dorsal aspect of the foot and toes. The PT artery nourishes, via the medial calcaneal angiosome branch, the medial ankle and heel, and through the medial and lateral plantar arteries and angiosomes, the entire plantar portion of the foot and toes. Lastly, the peroneal artery, with its anterior communicant branch angiosome, provides specific perfusion to the anterolateral and upper aspects of the ankle, as well as to the lateral heel compartment via its lateral calcaneal angiosome branch.[4-6]

At the upper ankle, additional source bundles (and related angiosomes) have been described, such as the anterolateral malleolar artery, its related anteromedial malleolar branch (both arising from the AT artery), and the neighboring posteromedial malleolar angiosome that derives from the PT artery.[4-6]

Choke vessels, cutaneous perforators, and arterial-arterial connections. A remarkable compensatory collateral framework that interconnects neighboring lower-leg angiosomes was documented in previous publications.[4-8,14,17] Adjacent foot angiosomes incorporate countless collateral "choke vessels"[4-6] that include small-to large-sized collateral branches beyond true arterial-arterial communicants.[4-6] In addition to these interconnections, cutaneous areas of each angiosomal territory receive either direct collaterals from main source arteries, or indirect (terminal) branches organized in specific clusters, called "cutaneous perforators" or "perforasomes."[4,5,27] Beyond the foot arches, other large diameter (+/- 1 mm) collaterals and communicants play a key role in compensating adjacent angiosomes in CLI conditions.[6,7,14,17] These collaterals play an essential role in intentional wound-targeted revascularization leading to appropriate tissue regeneration.[7,14,17]

Despite the approximately 9% of cases that show occasional individual variations,[1,2] as well as the previously mentioned communications between foot arches, several ranks of collaterals

have been cited as influential for topographic foot flow compensation.[5-7,17] The connections between the PT and PA (via medial and lateral calcaneal branches, and through the posterior peroneal branch) play an important role in healing ischemic heel ulcers.[5-8,23] Communications between the AT, DP, PT, and the plantar arteries directly, via the diagonal vessels, or indirectly via metatarsal perforators, provide hemodynamic support between the dorsal and plantar foot regions.[5-8] Metatarsal twin anterior and posterior interdigital collaterals provide substantial collaterals for the perfusion of the forefoot and toes, and also for tarsal/metatarsal wound healing.[6-8,14] Specific collateral support around peri-malleolar CLI wounds is provided by lateral and medial peri-malleolar anastomoses.[6-8]

Daily clinical experience reveals that not all foot territories may express similar ischemic burden during CLI.[7-9,14,17]

The literature shows that diabetic and renal CLI patients have significant tissue regeneration impairments, beyond specific infragenicular collateral waste.[17, 21, 22, 28] A critical loss in these patients' collateral reserve has been described; it appearsto be proportional to the type and timing of CLI.[17, 21, 28]

Challenging zones of perfusion in the foot. Current clinical experience suggests concerns about wounds that have more challenging topography in the ischemic foot. Two particular presentations deserve scrutiny:

a) *Ischemic wounds of the forefoot and toes*. These lesions represent about 37% of all Rutherford[5,6] CLI foot presentations.[8,21] The forefoot exhibits a crossroad of three main dorsal and plantar source arteries (ie, the DP, and the lateral and medial plantar bundles).[6-8] As stated previously, good patency of foot arches and digital arteries is crucial in CLI revascularization. Appropriate healing of toes can be achieved when greater than two digital arteries are preserved or revascularized.[12,17]

b) *Characteristic ischemic ulcers of the heel and hindfoot*. The heel perfusion is characterized by a "terminal-type" vasculature.[5-7,23] It depends on two sources of regional perfusion: the PT (+/- 70%) and the PA (+/- 30%) arterial flow. Beyond scarce native collaterals, there are no direct arterial-arterial communicants[4-6] and low-caliber collaterals are dominant.[7,8,23] Local angiosomes bear no compensatory connections with AT or DP arteries (unless there are abnormal anatomical variants). These threatening wounds are at higher risk of evolving into Rutherford 6 categorization or major amputation.[6-8,21-23]

Practical issues. Specific forefoot and hindfoot ischemic wounds or multiple CLI ulcers often reveal severe neighboring collateral deprivation that originates from two or three affected neighboring angiosomes.[6-8] In such cases, routine angiosomal evaluation can be arduous to perform. Advanced macro and microcirculatory CLI conditions perpetuate the destruction of collateral and cutaneous perforators.[7-9] These patterns are frequently encountered in diabetic or renal patients[8,21-23,28] with severely distorted angiosomal landmarks.[7,8,17,28] Clinical representation of the most impressive ischemic ulcer or necrosis zone may not always relate to the lowest perfusion area in CLI feet.[8] Irregular decay of collaterals,[7,8,17,28] the patchy distribution of remnant choke vessels and cutaneous perforators,[5,17,27] local capillary shunting by severe neuropathy,[7,28] sepsis triggering edema, and deep compartment hyper-pressure[7,17] may all lead to substantial variations in "real-life" CLI presentations.

Parallel risk factors for tissue recovery such as chronic inflammation, fibrotic scars, recurrent sepsis, extended necrosis, and regional hyper-pressure syndromes, may lead to acute thrombosis of small collaterals, particularly the highly vulnerable interdigital and cutaneous perforator branches.[7,14,17,28] Understanding these elements may help clinicians to better decode the real ischemic burden of each ulcer presentation and more completely assess eventual wound-directed revascularization.

CONCLUSIONS

From main ilio-femoral vascular sources, throughout the angiosome branches, and up to the arteriolar and the capillary vasculature, there is a harmonious pyramid of gradual arterial limb flow distribution. CLI is associated with specific infragenicular patterns of arterial atherosclerotic decay. Compensatory flow pathways are useful for interventionalists to understand for eventual topographic foot reperfusion. Regardless of irregular collateral availability, efficient limb revascularization must involve direct, in-line arterial reperfusion from the level of the iliac down to the foot arches to achieve limb salvage and adequate wound healing. ■

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