Definitions and Historical Development of Treatment of Chronic Limb-Threatening Ischemia

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Abstract

In 1954, chronic limb-threatening ischemia (CLTI) was identified as a separate category of peripheral arterial disease (PAD). The definition and treatment of CLTI have been evolving, recognizing the importance of early recognition and aggressive treatment required for those patients. In this article, we discuss the historical definitions of CLTI and provide an overview of how the management of patients with CLTI has developed, including surgical and endovascular interventions.

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Chronic limb-threatening ischemia (CLTI), previously known as critical limb ischemia (CLI), was identified as a separate category of peripheral arterial disease (PAD) in 1954. The definition and treatment of CLTI have been evolving, recognizing the importance of early recognition and aggressive treatment required for those patients. In this article, we discuss the historical definitions of CLTI and provide an overview of how the management of patients with CLTI has developed, including surgical and endovascular interventions.

Historical Development of the Definition of CLTI

CLTI refers to rest pain or tissue loss (nonhealing ulcer or gangrene) lasting for more than 2 weeks and related to decreased perfusion of the lower extremity.[1] The definition of CLTI has evolved throughout the last few decades without a complete consensus on the vascular hemodynamic parameters required to make the diagnosis. CLTI was first identified as a separate category of PAD by Fontaine et al in 1954 when they categorized patients with rest pain as stage III PAD and patients with tissue loss as stage IV PAD.[2] It was not until 1982 when CLI was initially defined as rest pain with an absolute ankle systolic Doppler pressure less than 40 mm Hg or tissue loss, including ulcers or gangrene, with an ankle systolic Doppler pressure less than 60 mm Hg in patients without diabetes. In that publication, the authors advised that patients with diabetes should be excluded and studied separately because of the possible confounding effects of neuropathy and susceptibility to infection.[3] In 1986, the Society for Vascular Surgery and the North American Chapter of the International Society of Cardiovascular Surgery steering committee adopted the same resting ankle pressure (AP) limit (<40 mm Hg for rest pain and <60 mm Hg for tissue loss) but added toe pressure (TP) as a diagnostic tool, with TP less than 30 mm Hg for rest pain and less than 40 mm Hg for tissue loss.[4]

In 1992, the European consensus document adopted an AP limit of less than 50 mm Hg and a TP limit of less than 30 mm Hg for patients with either rest pain or tissue loss.[5]

In 2007, the TASC II working group recommended similar hemodynamic parameters for the diagnosis of CLTI in patients with rest pain (AP <50 mm Hg or TP <30 mm Hg) but recommended higher values for patients with tissue loss (AP <70 mm Hg and TP <50 mm Hg), as these patients usually require more blood supply for wound healing.[6]

The more recent Global Vascular Guidelines recommend using any of the following hemodynamic parameters to assess decreased perfusion in CLTI patients: an ankle-brachial index (ABI) less than 0.4 (using the higher of the dorsalis pedis and posterior tibial arteries); an AP less than 50 mm Hg, as it involves most of the patients with rest pain or tissue loss who require revascularization for improvement of symptoms or healing of the ulcers; a TP of less than 30 mm Hg in cases of noncompressible arteries caused by significant calcifications; a transcutaneous partial pressure of oxygen less than 30 mm Hg; and flat or minimally pulsatile pulse volume recording waveforms.[7]

The proposed criteria to define CLTI varied based on data correlating the impaired peripheral hemodynamics and the increased risk of cardiovascular events and amputation. In clinical practice, these criteria were used mainly to have comparable patient populations among different studies, and sole dependence on the above criteria to diagnose CLTI can be misleading. It can cause under-recognition of patients with CLTI who are at high risk for amputation.[8,9]

Historical Development of Surgical Treatment of CLTI

PAD was first described by the ancient Egyptians in a papyrus in 1700 BCE.[10] Later, during ancient Greek times, the concept of ischemia was first described as the lack of perfusion.

In the Roman era, Claudius Galen proposed that the only treatment for the interruption of blood flow in a limb was amputation.[11] After the fall of the Roman empire, the center of medical research was moved to the Arab-Muslim civilization recovering the important findings of Greek-Roman medical science. Abu Ali Sina, also known as Avicenna, was the first to use the term saphenous. The saphenous vein graft (SVG) would become the mainstay of arterial replacement in ischemic disease.[12]

In that early era, no differences were reported between infected and ischemic gangrene, and both were treated with amputations. In the 1600s, however, Andrea Cesalpino and Girolamo Fabrizi described the small and big circulation and the condition of ischemia as a possible pathophysiologic mechanism leading to gangrene. Fabrizi's student, Dr. William Harvey, later illustrated the whole circulation in detail.[13]

During the same period, with many wars being fought, war surgeon Ambroise Paré was the first to use ligation of vessels instead of cauterization to stop hemorrhage after amputations.[14]

In the 17th and 18th centuries, physicians realized the importance of understanding pathological anatomy for appropriate treatment. Laennec described acute arterial thrombosis as the interruption of blood caused by an obstacle opposing flow sufficient to form an organized fibrinous clot. In that writing, he clearly described the aortic atheroma and its ulceration.[15]

The ischemic condition of the lower limb starts its history when Luigi Porta (in his "Opera Magna") described the capacity of the vasculature to produce collateral circulations in the presence of vessel occlusion, not understanding its implications in the clinical setting, which would keep the appearance of treatments yet very far in the future. The intuition of the causal connection between pathologic lesions and clinical evidence was discovered by a veterinary surgeon, Jean François Bouley, who first described the condition of claudication intermittens in a horse. The first observation of human claudication was made in 1858 by Jean-Martin Charcot, who described serious claudication in a 54-year-old man. The term claudication intermittent, coined by Charcot, was the first step toward modern vascular pathology.[16]

The term atherosclerosis appeared in 1833 by Jean Lobstein, identifying vascular injury due to the pathological process defined in that period with various names: "ossificant lesions, lithiasic lesions, cartilaginous focus, ulcerous focus, steatosis focus, atheromatosis focus."[17]

The history of CLTI treatment is anchored to the legend of the Arab twin Saints Cosmas and Damian, who were the first to save a limb affected by severe gangrene. The cause of the gangrene was possibly associated with ergotism due to fungal infection. From this "miracle" and other events, the twins became "protectors of surgeons."[18]

During the 20th century, the French school was one of the leading groups that allowed innovation in this field. In 1923, René Leriche reported his observation of occlusion of the terminal aorta and proposed a surgical treatment with resection and replacement of the diseased segment with a graft.[19] Thirty years later, Jacques Oudot published the first case of homograft replacement of the terminal aorta with an end-to-end anastomosis.[20] In 1946, João Cid dos Santos performed the first "disobliteration" of an occluded left femoral artery, a procedure that later became known as endarterectomy.[21] With the development of the endarterectomy, vascular surgeries became more advanced.

In 1948, Kunlin, from France, first described the use of a long-reversed SVG to bypass occlusive disease of the superficial femoral artery in a patient with a nonhealing foot ulcer despite the recommended treatment, at this time, of sympathectomy and atherectomy.[22] The procedure was not widely adopted until 1962, when Linton and Darling performed a similar procedure in the United States.[23] In 1959, Dr. Charles Rob from London performed the first in-situ SVG to do a femoropopliteal bypass after destroying the venous valves using an internal vein stripper.[24] In

1963, Connolly and Harris first performed Dr. Rob's procedure in the United States with in-situ SVG to perform a femoropopliteal bypass surgery.[25]

In the 1950s through the 1960s, a new terminology, extra-anatomic bypass (EAB), emerged. It referred to vascular bypass surgeries in unusual locations. The first EAB was performed by Oudot and Beaconsfield in 1953, who performed a crossover bypass between the external iliac arteries for a thrombosed leg of an aortoiliac graft.[26] The EAB concept rapidly evolved to include other anatomical variations, including thoracic aorto-femoral bypass in 1956, a procedure used to treat patients with CLTI and aortic occlusion for many years.[27] Other EAB surgeries included a femoral-femoral bypass in 1960,[28] an obturator femoral bypass in 1962,[29] an axillofemoral bypass in 1963,[30] and many other EAB variations.[31]

Earlier, patients with compromised arterial flow beyond the popliteal trifurcation with extensive stenosis or occlusion of the tibial and peroneal arteries were deemed unsuitable for vascular surgery. They were left with major amputation as their only choice. In the late 1960s, distal tibial artery bypass surgery using autogenous vein grafts was introduced as a revascularization method for patients with CLTI. In 1968, Garrett et al published their distal tibial artery bypass analysis using autogenous vein grafts in 56 patients.[32] A few years later, multiple reports of SVG bypass to the ankle and beyond were reported with acceptable success rates. This approach developed more in the 1970s and 1980s with better outcomes and reasonable graft patency.

Although outcomes of earlier arterial homograft bypass procedures were reasonable, the limited availability and the challenge of appropriate preservation of grafts were a major concern. This led to extensive research using artificial grafts made of nylon, Ivalon, Orlon, Teflon, and Dacron as alternatives.[33] Both Teflon and Dacron had better characteristics and were more commonly used. Multiple research projects on patients showed favorable outcomes and good patency for extended years.[34,35] In 1978, Veith et al published their usage of polytetrafluoroethylene (PTFE) for treatment of patients with CLTI. They performed extra-anatomical complex long vascular bypasses, including axillopopliteal, crossover axillopopliteal, crossover femoropopliteal, and femorotibial bypass. In that publication, they reported using PTFE to bypass the anterior tibial, dorsalis pedis, and posterior tibial arteries, with some patients requiring a secondary extension of the above-the-knee bypass to a distal below-the-knee artery with reasonable patency at 12 months.[36] In 1985, the same group reported a new surgical approach using short (8 cm–33 cm) segments of the reversed autologous vein to perform tibial-tibial bypass in fourteen patients with CLTI. Eleven out of the 14 patients (79%) had a patent graft and a functional limb 6 to 50 months post procedure.[37]

Despite the development in surgical bypass surgeries and artificial conduits, a single great SVG remains the best conduit option if available. The recent BEST-CLI trial reported excellent outcomes in these patient populations with a low rate of major adverse limb events and death.[38]

Revascularization remains the cornerstone of limb preservation in patients with CLTI. The advanced endovascular and open surgical techniques over the last few decades favor better outcomes with decreased amputation rates.[39] Unfortunately, not all patients are candidates for revascularization, and minor or major amputations remain an important treatment option in managing CLTI patients with severe arterial disease beyond salvage. Although often considered a failure of treatment, major amputations provide definitive therapy for unsalvageable disease.[40]

Historical Development of Endovascular Treatment of CLTI

A frequently encountered, yet not highly publicized, fact about medicine and its history is that it takes decades for innovations to reach the stage of broad acceptance. Only then can we generate new questions that launch new avenues of research to deepen our understanding, knowledge, and ability to help our patients better. In 1963, Dr. Charles Dotter inadvertently passed a guidewire and then a catheter through a complete total arterial occlusion of a patient with an abdominal aortogram performed via retrograde catheterization of the right iliac artery. He thought that by using this technique, he could dilate obstructed arteries.[41] A year later, he passed a guidewire through a tight stenosis in the femoral artery of a female patient with gangrenous toes in what appears to be the first documented endovascular treatment of a patient with CLTI. This term would take 18 years to be born. He then passed a catheter followed by a larger catheter, achieving a step-by-step dilatation of the artery.[42] Later attempts were made to modify the technique by placing a balloon catheter across the stenotic area and then inflating it. The available balloons had insufficient strength and became deformed, acquiring an hourglass shape.[41] In 1974, Andreas Grüntzig, a Swiss radiologist, revolutionized balloon angioplasty when he developed a double-lumen catheter with a balloon made of polyvinyl chloride near its tip, which had the strength to dilate stenotic arteries without deforming.[43] It took 15 years from Dotter's original publication before the medical world accepted this technique. In 1979, dilatation of iliac stenoses had an initial success rate of 92%, with 2-year patency rates of 87%.[44] In 1981, Grüntzig reported an 84% initial success rate for occlusions of less than 10 cm with a 3-year patency rate of 70% for femoropopliteal lesions.[45] This number has not improved significantly after 40 years, despite the availability of innovative technologies. Twenty-four years elapsed until the publication of the BASIL trial (comparison of surgical bypass vs endovascular angioplasty for CLTI), which showed similar rates of amputation-free survival between groups.[46] Given the significant morbidity, high surgical risk, and complication rates that most CLTI patients have, BASIL led and fed the revolution of the endovascular-first approach, fueling the development of novel

strategies encompassing the spectrum from exotic arterial access, the study of lesion morphologies, best approaches to complex lesions, lesion preparation, treatment algorithms, creation of new risk-predictive models, and new techniques designed to treat so-called "no option" patients. The use of ultrasound guidance (USG) to obtain arterial access has increased in the last decade as it represents an essential part of any endovascular intervention (EVI). Access to the infrapopliteal (IP) vessels is arguably one of the most important steps in achieving adequate revascularization in patients with CLTI. A study of 86 patients with CLTI sought to determine the safety and efficacy of using USG to obtain antegrade/retrograde common femoral artery (CFA) and tibiopedal retrograde access; it showed success in 95.3% of patients, concluding it was safe and efficacious.[47] This finding was later validated in a larger cohort of patients.[48,49]

Traditionally, operators have used the contralateral retrograde CFA access with an "up and over" approach as the preferred strategy to perform IP EVIs. However, this strategy has 20% to 40% failure rates in patients with CLTI, leading operators to use retrograde and combined (ante-retro) access to treat the chronic total occlusions (CTOs) from both ends.[50,51] This approach has been increasingly used daily and supported by clinical studies.[52]

The lesion preparation and treatment of IP vessels represent a conundrum that suffers from a drought of generalizable scientific evidence to support percutaneous revascularization; hence, the optimal treatment modality remains controversial. Percutaneous transluminal angioplasty (PTA) continues to represent the standard of care worldwide, even though outcomes remain suboptimal. Twenty-seven years after Grüntzig's work, a meta-analysis reported the 3-year outcomes of PTA as the primary treatment modality with limb salvage rates (LSR) of 82.4%.[53] A more recent meta-analysis of 6769 patients treated between 2005 and 2015 concluded that PTA, as primary treatment for IP disease, leads to suboptimal procedural and 1-year outcomes.[54] A plethora of newer technologies, including patency-enhancing drug coatings for balloons and drug-eluting technologies for stents, adjunctive endovascular devices (orbital, rotational, and ablative atherectomy, cryoplasty, focal force/cutting balloons, laser, tack implants, bioabsorbable stents, and intravascular lithotripsy) are feasible and safe in IP vessels but have failed to provide comparative data and to show superior efficacy when compared with conventional, less expensive therapies. As these devices add cost, their added expense must be justified, and most available data emanates from single-center retrospective reports or uncontrolled registries, subject to selection bias.[55]

Following BASIL, the preferred endovascular approach was PTA with self-expanding nitinol bare metal stents (SENBMS) used as a "bailout" technique. This strategy was compared with primary stenting with a SENBMS in the EXPAND study of 92 patients with IP PAD and severe claudication or CLTI, showing no difference in 1-year outcomes.[56]

As drug-eluting stents (DES) became established in the coronaries, it did not take long for this technology to be transplanted to the PAD territory. The DESTINY[57] and YUKON-BTK[58] study randomized 301 patients with CLTI to DES vs BMS. At 1 year, there was no difference in functional outcomes. DES had statistically superior patency and freedom from target lesion revascularization (TLR). At 3 years, event-free survival, amputation, and TLR rates were superior for the DES group. The ACHILLES study[59] randomized 200 patients with IP disease to DES or PTA and found DES to be statistically superior in patency rates, quality of life score, and restenosis rates (for lesions <120 mm length, especially among patients with diabetes) at 1 year, without significant difference in complete wound closure, death, amputation, or improved functional status.

These data,[57-59] together with several meta-analyses,[60-64] generated a class 1, level of evidence B, favoring IP DES over PTA and BMS for (1) improved patency, (2) reduced re-interventions, (3) reduced amputation, and (4) improved event-free survival.

The evidence supporting drug-coated balloon (DCB) use for IP lesions is less robust. The DEBATE-BTK[65] trial randomized 158 IP lesions in patients with diabetes and CLTI to DCB vs PTA. Restenosis at 1 year was significantly better in the DCB group ($P<.001$). However, there was no difference in the rates of amputation, limb salvage, or mortality between the groups. The IN.PACT Deep CLI study[66] resulted in the removal of the IN.PACT Amphirion DCB from the market worldwide by the sponsor. The trial randomized 358 CLTI patients to DCB vs PTA. At 1 year, there was no difference in efficacy, but there was a higher amputation rate in the DCB group. Unfortunately, these results trumped the use of DCBs in the IP territory; however, novel devices and different platforms are being studied.

In the current era of rapidly evolving technology and techniques, we have now jumped into the inframalleolar territory, looking for answers and furthering our quest for better and more long-lasting treatments for our patients with CLTI. The RENDEZVOUS registry studied 257 patients with IP and inframalleolar disease. All patients underwent IP EVI: 140 had adjunctive pedal artery angioplasty and 117 did not. The authors found that the rate of wound healing was significantly higher and the time to wound healing significantly shorter in patients who received adjunctive inframalleolar PTA, thus suggesting that patients with CLTI and pedal arterial disease would benefit from treating these distal vessels.[67] Lastly, concerning the so-called "no-option" patients with CLTI, there have been a few major advances in the last 5 years. These patients were typically present with a combination of "small artery disease," diffuse arterial wall calcifications, and the absence of patent pedal vessels ("desert foot"), leading to the failure of established "conventional" revascularization attempts. Recently, a simple scoring system using just 2 radiographic views of the foot allows for prediction response to conventional endovascular treatments among these patients[68] and, more importantly, to

identify patients who would benefit from the latest technique in CLTI revascularization by transcatheter deep vein arterialization (DVA) with the creation of an IP arteriovenous fistula, to bring arterial blood through patent venous conduits to the foot. DVA showed safety and feasibility with promising results and creation of new options for this previously termed "no-option" patient with CLTI cohort.[69] The ongoing clinical trials will hopefully help us better treat more patients with CLTI.

Conclusion

CLTI is the most extreme form of PAD, which can be a limb- and/or life-threatening condition. The cornerstone of diagnosis of CLTI depends on presence of rest pain or tissue loss in the setting of decreased limb perfusion. It is critical to use clinical judgment and variable hemodynamics in the diagnosis of CLTI; sole dependance on a certain measurement can be deceiving, leading to underdiagnosis and inappropriate treatment of these patients. Early recognition and proper treatment are essential to improve outcomes. Both surgical and endovascular treatment of PAD have been tremendously evolving over the last few decades. Revascularization option should be tailored according to the patient's comorbidities and candidacy for surgical or endovascular interventions.

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