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Controversies in CLI

In this issue, Dr. Michael R. Jaff, Vice President of the CLI Global Society, seeks input from Drs. Gary Ansel, Sean Lyden, Bruce Gray, Robert Lookstein, and Larry Diaz-Sandoval about how they approach the treatment of patients with multi-segmental disease.

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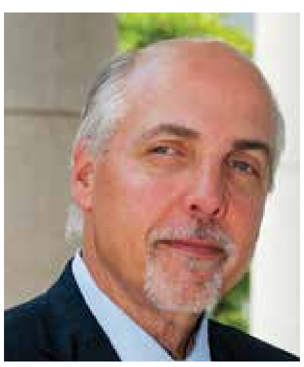
Michael R. Jaff, DO


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Larry Diaz-Sandoval, MD


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Jaff: Do you treat multi-segmental disease in one setting or do you stage it and why?

Ansel: It depends. There are several variables that may affect this. Though we try to do all in one setting, there are occasions when there is a long complex CTO of the SFA as well as a complex CTO of the tibial where time and contrast dose may have us stage the procedure. Also, if there is an iliac CTO that requires retrograde repair, that may be done first and the patient staged for the infrainguinal revascularization. Some patients also tolerate lying on the table for variable amounts of time, which may lead to staging.

Lyden: For patients with CLI, I treat all segments at once. For patients with claudication, I will treat iliac and SFA/popliteal but not tibial at the same stage. If a patient has CKD 4 or greater and is not on dialysis, I will stage it depending on how much dye I need. I will use CO_2 to reduce dye load.

Lookstein: I almost exclusively treat multi-segmental disease in one setting, unless the patient cannot tolerate the procedure.

Diaz-Sandoval: The multi-segmental and multivessel distribution of the disease process characteristic of CLI poses a strategic challenge. It is well-known that 20.2% of patients with PAD have associated CKD stage >2,[1] and as the disease severity increases, so does the severity of the renal involvement. Nowadays, CO_2 can be used in the United States as a strategy to limit the amount of contrast utilized (in an effort to avoid contrast-induced nephropathy [CIN]); however, only injections via hand-held syringes filled with the gas are available. In Europe, data are being generated with the use of an automated system (Angiodroid, Bologna, Italy) that generates high-quality images in CLI patients undergoing endovascular interventions,[2] hence potentially eliminating the concern of CIN — in which case, it could be argued that multiple vessels and/or segments could be treated in only one setting, as long as the amount of radiation would also be kept within safe limits. At our institution, we

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D.R.E.A.M.: Docs Revascularizing Extremities Against Major Amputation


Paul Michael, MD, FSCAI

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On January 16, 1964, the "father of intervention" Charles T. Dotter, MD, and his trainee Melvin P. Judkins, MD, performed the first intentional percutaneous transluminal angioplasty on patient Laura Shaw. She was admitted to the University of Oregon Hospital with a non-healing left foot ulcer and gangrenous toes. Amputation was recommended by all of her physicians; however, Ms. Shaw refused surgery. Because of this refusal, one of history's most

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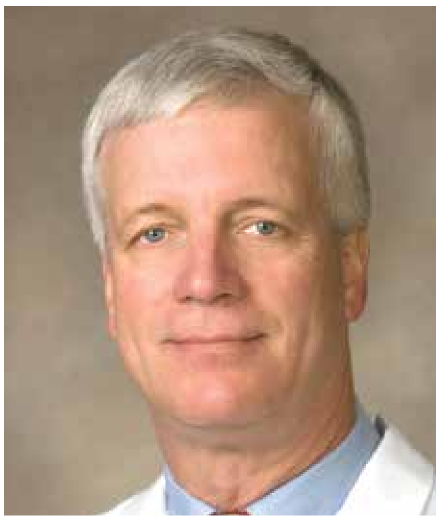
Post-Revascularization Wound Care: Top 10 Priorities

Lee Ruotsi, MD, ABWM, CWS, UHM, and Dot Weir, RN, CWON, CWS



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No discussion of wound care is complete without consideration and analysis of arterial flow to the site in question. It is well accepted that if there is no flow, there is no healing, and a downhill spiral of tissue loss, infection, hospitalization, and amputation often ensues. Early vascular intervention, with close surveillance of ongoing patency, can then set the stage for renewed efforts toward tissue repair and wound healing. However, this is a critical time, as sustained patency is never assured, and efforts toward wound closure must be optimized and expedited during this window of opportunity. What, then, are the wound care evaluation and management priorities in the immediate post-revascularization period?

1) **Ischemia-reperfusion injury:** It is understood that the destructive downstream effects of ischemia-reperfusion injury can be worse than the initial ischemic insult. Mitigation of those deleterious effects should be initiated in the immediate post-op period and may include pharmacologic therapy as well as hyperbaric oxygen therapy.
2) **Debridement:** To debride or not to debride? A most essential part of moving a wound toward a state of readiness to heal is wound bed preparation, and specifically, debridement. There is no greater impediment to wound repair than the presence of devitalized tissue on the surface of the wound. Wound clinicians make a conscious decision to avoid debridement of dry eschar in the poorly vascularized wound. Once the blood flow to the wound has been reestablished, a decision must then be made: to debride the wound of all necrotic tissue and take advantage of adequate flow while it's most robust, or continue to proceed conservatively and allow for demarcation and lifting of a dry eschar.
3) **Infection:** Poorly perfused tissue is at high risk for infection, and wound infection is a common cause of wound healing failure. It is important to consider appropriate clinical and laboratory evaluation in the immediate post-revascularization period so that appropriate treatment can be provided should significant infection be present.
4) **Moisture:** Appropriate moisture balance is another important factor in wound healing. The wound bed should not be dry, nor should it be wet with uncontrolled exudate. Rather, it should be moist. Ischemic tissue often becomes dessicated, and if allowed to stay in this condition, healing cannot progress. The treatment of dry wounds should include moistening agents such as hydrogels and/or moisture retentive dressings such as foams,

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Editor's note: Articles in this supplement to *Cath Lab Digest* did not undergo peer review.

Case Study

Popliteal Artery Entrapment Syndrome in a Young Female

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Michael Sumners, DO

Popliteal artery entrapment syndrome (PAES) is a rare and potentially devastating condition that affects young individuals who are otherwise healthy. PAES was first reported in 1879 by T.P. Anderson Stuart and represents a rare but important cause of lower-extremity ischemia.[1,2] Patients affected by PAES are often young active individuals without a significant medical history. It remains under-recognized and misdiagnosed. The following highlights a typical case from presentation to definitive therapy.

A 28-year-old athletic female was referred for outpatient vascular evaluation due to progressive bilateral lower extremity claudication with ambulation of less than 50 feet. She had no cardiovascular risk factors and no significant contributing family history. Eight years prior, she reported being diagnosed with bilateral exertional compartment syndrome and undergoing bilateral lower-extremity dual compartment fasciotomy. Otherwise, she reported no prior medical history. She denied any significant improvement after the surgery. Due to her recent progressive symptoms, she was referred for a repeat vascular evaluation. A physical exam revealed blunted pedal pulses with plantar flexion. Due to a suspicion for entrapment given the history and physical exam, the patient underwent selective digital subtraction angiography bilaterally with provocative maneuvers, including active plantar flexion and dorsiflexion. This revealed normal angiographic images at rest bilaterally, as shown in Figure 1. With active plantar flexion, angiography revealed complete occlusion at the P1 segment of the popliteal artery consistent with popliteal artery entrapment, as shown in Figure 2. She was referred to a tertiary care center for further evaluation and underwent surgical decompression of the popliteal artery with partial excision of the proximal gastrocnemius and popliteus muscles. At a one-year follow-up, she has made a full recovery with resolution of exertional claudication with a gradual full return to vigorous activity.

DISCUSSION

The described case is typical of the PAES disease course from symptom onset to diagnosis and treatment. This includes under-recognition and delay to diagnosis, as well as treatment for other conditions without improvement. Ultimately, when symptoms become severe and lifestyle-limiting, further evaluation takes place and a correct diagnosis is made with referral for definitive treatment. In this case, the disease process had not progressed to irreversible damage and a full recovery was possible. Others are often not as fortunate.

Patients often present with intermittent unilateral symptoms of lower-extremity claudication in the early disease course or with more acute onset with development of complications such as thrombosis or distal embolism.[3-6] To make the diagnosis, a careful history and physical exam are necessary. Physical exam findings in early disease include decreased posterior tibial and dorsalis pedis pulses upon provocative maneuvers, including leg hyperextension and active plantar flexion. Duplex ultrasonography of the popliteal artery, computerized tomography (CT) scans, and contrast arteriography may also be used to detect active compression and its complications.[3]

The incidence of PAES is only estimated due to lack of awareness and under-recognition. Further compounding the difficulty in diagnosis can be the presence of duplex ultrasound evidence of functional popliteal compression that remains asymptomatic. Possibly only the most severe cases present for evaluation and workup and obtain a true diagnosis. Due to the severe late outcomes of this disease in a young population, awareness and recognition are of paramount importance.

PAES can be divided into groups including anatomical, functional, and a combined subgroup. The underlying pathology involves abnormal congenital

Figure 1. Normal angiographic images bilaterally, at rest.

anatomy surrounding the popliteal artery, functional occlusion due to muscular hypertrophy, or a combination of both. The different types of anatomical PAES are categorized based upon the embryological development of the popliteal fossa. The most commonly used classification system is the Popliteal Vascular Entrapment Forum classification, which describes six types based on anatomical and functional components.[7] The first four classes describe the anatomical anomalies that ultimately cause the entrapment.

The described anatomical types of PAES are illustrated in Figure 3.[8] Type I entrapment occurs when the popliteal artery develops prior to the medial head of the gastrocnemius muscle. This results in medial positioning of the popliteal artery, causing it to become compressed between the gastrocnemius and the femoral condyle.[5,9,10] Type II entrapment involves disruption in development of the gastrocnemius medial head by a prematurely formed popliteal artery. In Type III, portions of muscle or fibrous bands entrap the artery. Type IV is the result of the popliteal artery remaining deep to the popliteus muscle in development, causing compression. Type V entrapment occurs when both the popliteal artery as well as the popliteal vein are involved.[11,12,13] Type VI entrapment is acquired due to gastrocnemius or plantaris muscle tendon hypertrophy causing popliteal artery occlusion during active plantar flexion or leg extension.[1,4,5,11,14] Other acquired forms of entrapment can result from compressive masses, edema, or articular knee disorders.[15] With the underlying anatomical or functional entrapment, movements such as plantar or

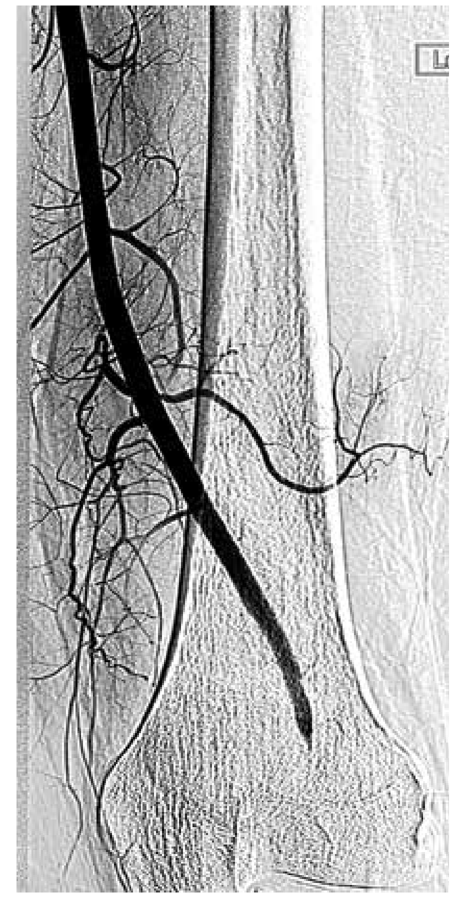

Figure 2. During plantar flexion, angiography reveals complete occlusion at the P1 segment of the popliteal artery consistent with popliteal artery entrapment.

dorsiflexion may exacerbate the degree of compression, eliciting symptoms. This repetitive trauma of the popliteal artery may result in arterial injury, turbulent arterial flow, arterial aneurysm, and thrombus formation.[13,16,17] Diagnosis of PAES early in its course is crucial and relies upon disease awareness, an accurate history and physical, and an evaluation of a differential diagnosis. The differential diagnosis includes atherosclerotic disease, exertional compartment syndrome, thrombosed popliteal artery aneurysm, and cystic adventitial disease. Non-invasive and often invasive tests are needed to demonstrate both the anatomic and functional aspects of the disease. This may include arterial duplex or digital subtraction angiography with provocative maneuvers that can clearly demonstrate the functional compression. CT or magnetic resonance imaging (MRI) may demonstrate the anatomic variant causing the compression and guide treatment. Treatment of PAES is based on the type of entrapment and degree of arterial pathology. Treatment commonly involves a multidisciplinary approach including both endovascular and surgical expertise. This often includes diagnosis and initial stabilization with endovascular options that may include thrombolytic therapy. Surgical treatment is indicated in all symptomatic patients.[2,3] Definitive surgical treatment may range from myotomy of the medial head of the gastrocnemius muscle to vascular repair, including

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Editorial Commentary

Popliteal Artery Entrapment Syndrome

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Popliteal artery entrapment syndrome (PAES) was described more than a hundred years ago in 1879 by T.P. Anderson Stuart as a cause of acute lower-extremity intermittent claudication. However, to this day, it remains underdiagnosed. Since it is primarily seen in young, active individuals with minimal or no cardiovascular risk factors, the diagnosis becomes all the more challenging.[1] Having said that, it becomes imperative to have a high index of suspicion for this pathology in this patient population, as the complications can be devastating and may lead to long-term morbidity, with amputation being one of the most dreaded results.[2] PAES usually affects young, athletic men, presenting as intermittent claudication. It is primarily due to abnormal positioning of the popliteal artery in relation to the musculature of the popliteal fossa, including the popliteus and gastrocnemius muscles, causing compression leading to vascular and neurogenic symptoms. Various classification systems were used to further the understanding of this entity. Currently, the most commonly employed one is the Popliteal Vascular Entrapment Forum classification, which has six different types, each describing a different anatomic variant as a cause for PAES.[3] However, PAES can broadly be divided into two groups, *anatomical* and *functional*. In the anatomical type, there is a clearly defined aberrant anatomical defect or malformation that leads to occlusion of the popliteal artery. In the functional subtype, although there is evidence of transient occlusion of the popliteal artery and subsequent intermittent claudication, no clear anatomic abnormality is noted that can explain the claudication.

In a recent issue of *Vascular Disease Management,* Mustapha et al published a series of four cases describing a varying range of presentations as well as management.[4] They further highlighted the importance of early recognition and timely intervention, which if delayed may lead to grave complications and outcomes. One critical aspect in diagnosing PAES is the presence of dynamic occlusion of popliteal vessels. However, this is also the more challenging aspect of PAES since dynamic occlusion can be seen on duplex ultrasound (US) imaging even in asymptomatic patients, with a prevalence ranging from 25% to 80% based on various studies.[5] Therefore, careful history taking and focused physical examination become paramount. Although a validated clinical test for PAES has not been described, many physicians attempt to provoke symptoms by asking the patient to hop or perform plantar and dorsiflexion while standing on the edge of a step. Dorsalis pedis or posterior tibial pulses are palpated and popliteal fossa are auscultated before and after the maneuver to elicit any drop in the pulses. If patients do not develop any symptoms during or after this maneuver, then the likelihood of PAES may be low. However, development of symptoms or bruit on auscultation does not mean the patient has PAES, as these may be patients with asymptomatic transient occlusion.

PAES can broadly be divided into two groups, *anatomical* and *functional.* In the anatomical type, there is a clearly defined aberrant anatomical defect or malformation that leads to occlusion of the popliteal artery. In the functional subtype, although there is evidence of transient occlusion of the popliteal artery and subsequent intermittent claudication, no clear anatomic abnormality is noted that can explain the claudication.

In these situations, non-invasive imaging modalities such as Doppler ultrasound (US) may provide additive diagnostic value.[6] Given the peripheral location of the popliteal artery, Doppler US makes for a relatively cheap, non-invasive option in this situation. It is the recommended first-line diagnostic modality for PAES. Provocative maneuvers — such as sustained passive dorsiflexion and plantar flexion of the foot, leading to loss of dorsalis pedis pulse or the posterior tibial pulse on Doppler US — may be highly suggestive of PAES in the right clinical scenario. The occlusion can often resolve within 30 seconds of the provocative maneuver, making instantaneous assessment essential for an accurate diagnosis and to avoid false negatives. The position of highest yield involves hyperextension of the knee with simultaneous active plantar flexion.

Traditionally, contrast arteriography has been the definitive test for the diagnosis of PAES. Internal deviation, flatness, narrowing, or post-stenotic dilation are some of the signs on arteriography that raise suspicion for PAES. However, plain-film arteriography often leads to over estimation of the length of the stenosis due to dilution of the contrast material as it passes through the stenosis. This can be minimized with the use of digital subtraction angiography (DSA), since the image acquisition continues until there is no further filling of the artery, thereby enabling assessment of slow flow through any collaterals. Detection of collateral system is an important aspect of preoperative planning of severe PAES.

Other imaging modalities that assist in diagnosing PAES include computed tomography (CT) angiography and magnetic resonance imaging (MRI) angiography. CT trumps Doppler US in its ability to generate three-dimensional reconstructed images, which can be assessed from any angle to best visualize the popliteal artery in relation to its surrounding structures. Additionally, the value of CT imaging is even higher in situations of acute limb ischemia where the exact location of the stenosis is required prior to pursuing any intervention.[7] CT angiography trumps DSA with regard to detection of aberrant muscle in the popliteal fossa, relationship between the artery and surrounding structures, popliteal artery aneurysm, and cystic adventitial disease.

MRI angiography is another non-invasive imaging modality that is increasingly being used to detect PAES, especially in cases with an anatomic aberrancy.[8] It is useful in detecting abnormal insertion of the medial head of the gastrocnemius, medial displacement, level of occlusion of the popliteal artery, and differentiating intrinsic vascular disease from extrinsic compression. However, it falls short in the assessment of functional stenosis since it is challenging for the patient to maintain the provocative position of plantar flexion for the duration of the MRI. Recent studies have proposed the use of intravascular ultrasound (IVUS) to provide information on the exact location of the obstruction in addition to assessing the quality of the affected vessel wall, which may be important in more advanced cases of PAES.[9] Given the limitations with each modality, it is prudent to use these tools in the appropriate clinical setting and use the findings in conjunction with each other, rather than exclusively.

In symptomatic patients, surgical intervention has been the longstanding treatment of choice in PAES in order to reestablish normal anatomy and vascular flow to the distal extremity.[10] In the anatomical variant of this condition, the progression of the occlusive disease is much more rapid, requiring urgent and invasive management. These patients may require exploration, fasciotomy, and myotomy of varying degrees. The stage at which the diagnosis is made becomes important in the surgical approach. Earlier diagnosis may limit the surgical intervention to mainly releasing the popliteal artery by division of the aberrant musculotendinous tissue. If the diagnosis is made at the stage of significant popliteal arterial stenosis or aneurysm, then complete vascular reconstruction of the popliteal artery may be required in addition to division of the aberrant musculotendinous structures. The progression of disease is much slower in function PAES, which allows for longitudinal follow-up in mildly symptomatic patients. Surgery may eventually be required if the symptoms recur more frequently or progress in severity. Unfortunately, surgical intervention has not proven to be as successful in functional PAES as in anatomical PAES, with only around 77% experiencing complete resolution of symptoms after surgery.[5] Recent focus has been on minimally invasive management options for PAES; one that has been gaining ground recently is the use of guided botulinum toxin injection. The proposed mechanism is to paralyze

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Tibio-Pedal Artery Access: Alternative Access Reaches Maturity

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In the not too distant past, alternative access for lower-extremity arterial intervention (LEAI) was making a decision about which femoral or brachial artery to access. Antegrade femoral artery access was perhaps considered "alternative." Fast forward to the current state of endovascular intervention and we find ourselves considering things like radial artery access for LEAI, primary tibial artery treatment from a pedal approach, and even metatarsal artery access. There is no doubt about the advantages we now have as interventionalists with regard to better technology. Improved guidewires, dedicated crossing devices, support catheters, and ultra-low profile balloons have enabled us with the ability to access, cross, and treat obstructive lesions that were previously untreatable. Adjunctive therapies like atherectomy and stents are still finding their way in the treatment of tibial artery disease and critical limb ischemia (CLI).

Tibio-pedal artery access (TPAA) is being used with increasing frequency as alternative access to facilitate procedural success in LEAI. This technique is usually employed in the setting of CLI and tibial artery intervention, but may offer potential practical advantages for popliteal artery and even superficial femoral artery (SFA) intervention in unique situations. While others have described TPAA access for intervention for claudication, we have not found it to be necessary in patients with claudication and primarily femoropopliteal disease. In our practice, TPAA is only used in the setting of CLI and in only 5%-10% of those patients. That said, our threshold for moving to an alternative access site with failed antegrade recanalization in tibial arteries has been lowered.

TECHNICAL CONSIDERATIONS

As our knowledge of pedal anatomy has improved and our understanding of the need for focused or directed revascularization has increased, so too has the importance of case planning. Clinical success in terms of limb salvage is based on procedural success and of course wound care if needed. Procedural success hinges on entry and exit strategies. Basic concepts are discussed below, with our typical preferences.

PATIENT PREP: SET YOURSELF UP FOR SUCCESS

Patient position and comfort are issues that will be important initially during access, since the pedal vessels can be quite small, and incidental patient movement during access can be frustrating. Depending on the access vessel of choice, the leg can be positioned in a neutral position or externally rotated at the hip and/or foot to allow good visualization of the artery. It is important to support the foot in the chosen position so that the patient will be still during access. This can be achieved through use of towels or pillows beneath the sterile drape and also use of straps to secure the limb in the ideal position. Judicious use of conscious sedation to minimize untimely patient movement can be invaluable in anxious patients. Many have described using deep sedation and/or general anesthesia in some patients.

Operator position and comfort is also a key consideration. I am a big believer in maximizing your ability for success by doing the right work up front. There is mounting evidence on the orthopedic and radiation impact we have from being interventionalists. These are potentially long cases and attention to operator position prior to starting the procedure can improve comfort, minimize direct radiation exposure, and still allow adequate visualization of the appropriate monitors. In order to secure stable TPAA, attention should be given to where the operator will stand during access and possible treatment, the position of the image intensifier (II) and monitors, position of ultrasound (if used), and access to any antegrade access that may be needed as well.

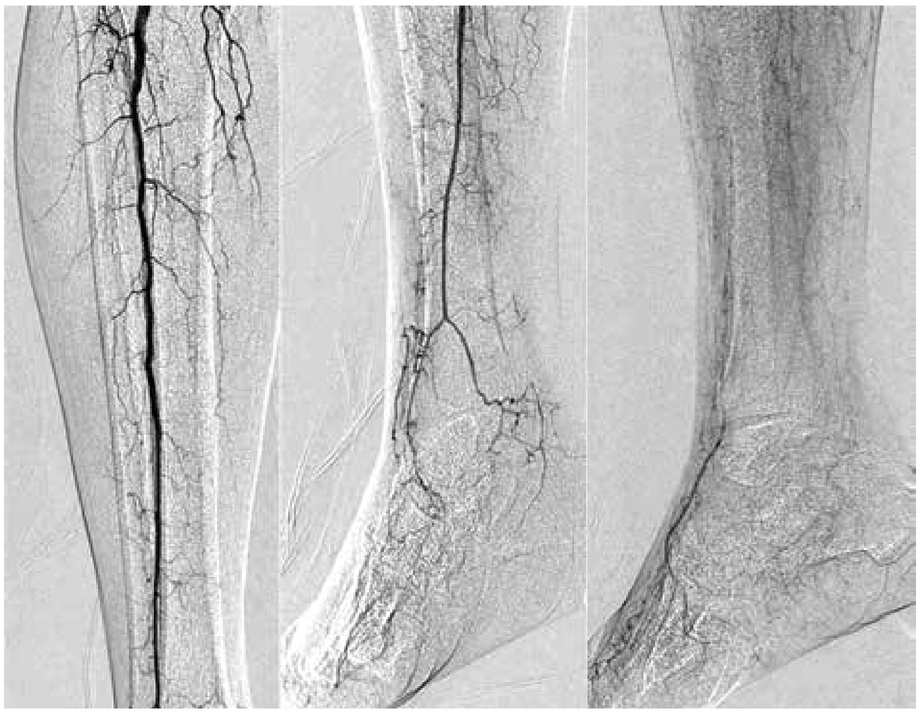

Figure 1. Right-lower extremity angiogram demonstrating patent peroneal artery and flush occlusion of anterior tibial artery and distal reconstitution of the dorsalis pedis artery.

Figure 2. Ideal set-up for simultaneous access for antegrade femoral artery access and retrograde pedal artery access. Note monitors in front for optimal operator comfort.

Sterile preparation of the access site may be considered routine; however, there are a few unique considerations to be taken into account. In the presence of obvious infection, ischemic ulceration, or an open wound, meticulous technique is necessary and should include a sterile prep of the entire foot, even if the access site may be somewhat removed from the ulcer/wound. In our lab, a wide, double prep using chlorhexidine is employed. The toes are covered with a sterile towel if there is active ulceration, and a drape is placed across the intended access site. Prior to the prep, nitroglycerin paste is sometimes applied to the access site to maximize vasodilatation. If this is chosen, it is applied a few minutes prior to application of the chlorhexidine, so that it is not immediately cleared away.

ENTRY STRATEGY: IDEAL ACCESS

Choosing which artery to access with regard to the various philosophies (angiosome-directed therapy, focused

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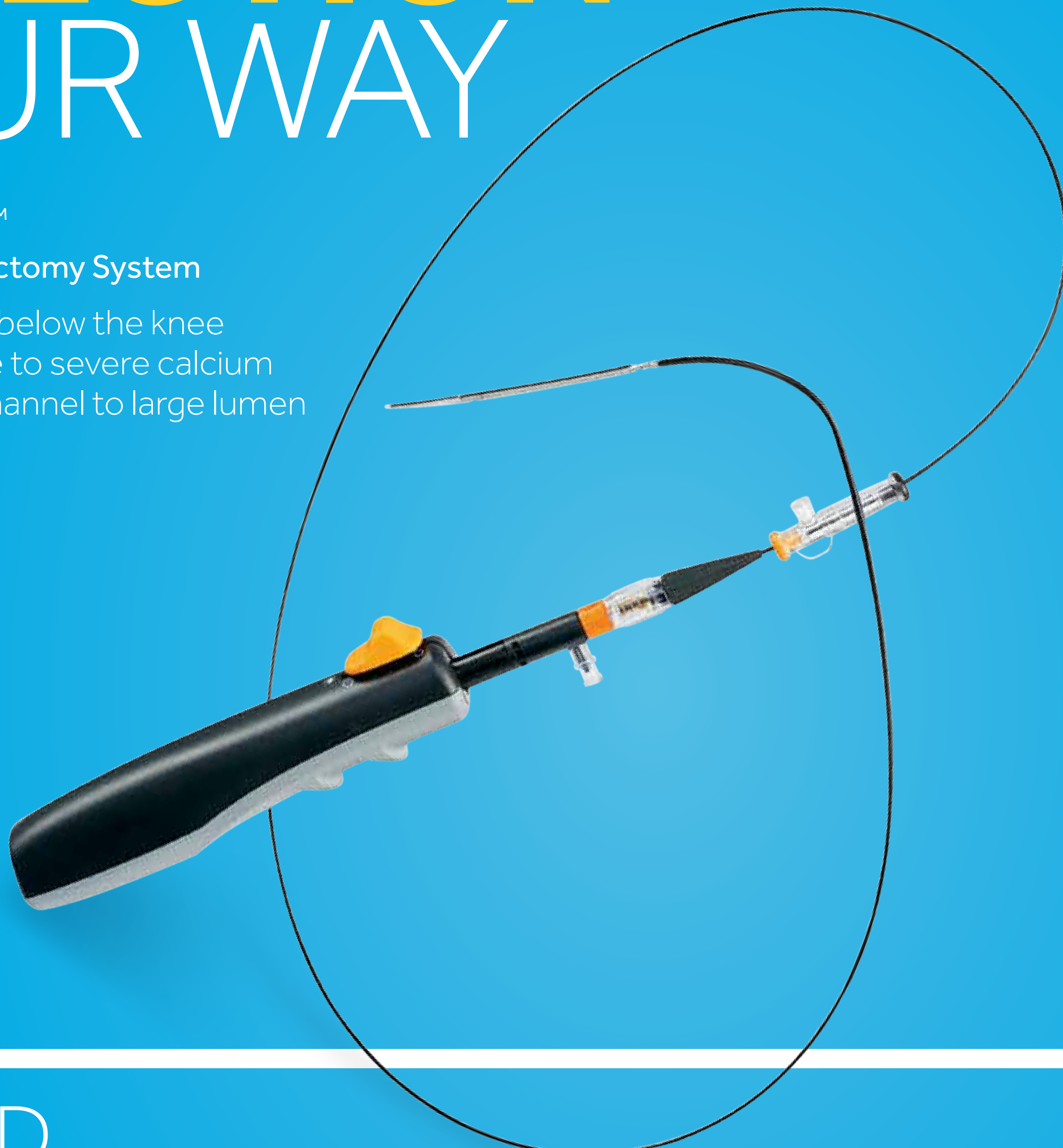

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Figure 3. Retrograde dorsalis pedis access after failed antegrade recanalization.

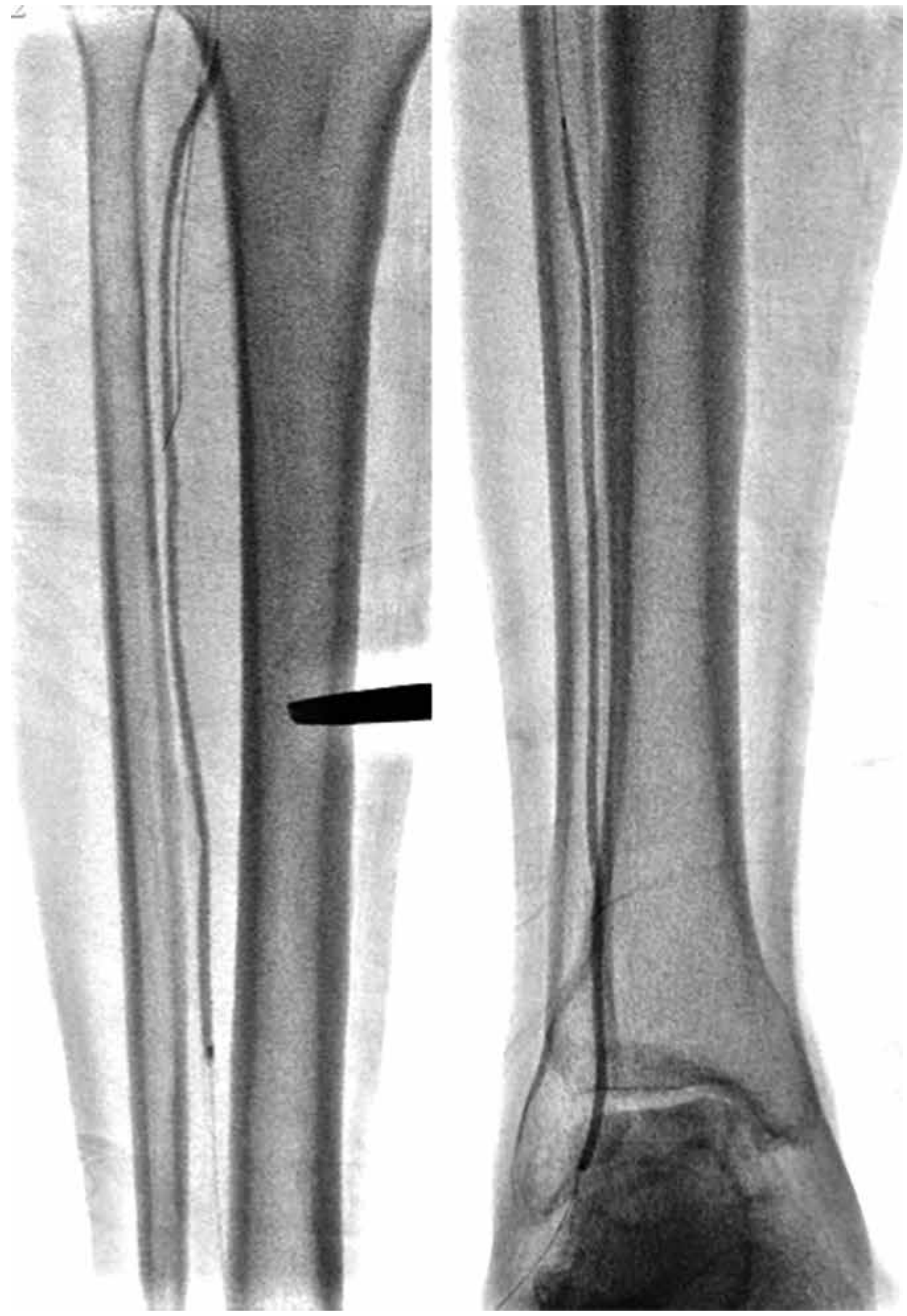

Figure 5. Angioplasty of anterior tibial artery after reversing wire access.

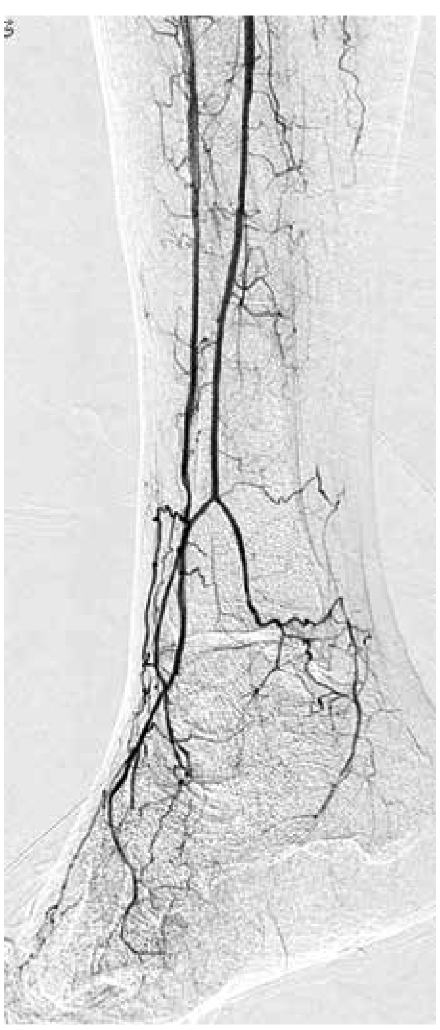

Figure 4. Final distal angiogram demonstrating brisk antegrade flow through the recanalized anterior tibial.

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revascularization, etc.) is beyond the scope of this article and varies from physician to physician. Numerous techniques have been described, and all are equally useful. I will simply describe our technique once the decision has been made regarding the specific artery to be accessed.

Imaging guidance for obtaining access into the target vessel is either by ultrasound or fluoroscopy. The major advantage of ultrasound is real-time imaging without ionizing radiation. Linear, high-frequency transducers (7 or 12 MHz) are used most frequently, depending on target-vessel depth. Choice of using a transverse (axial) or a sagittal (longitudinal, "in-plane") orientation for puncture is a matter of operator choice. Once the artery is punctured, ultrasound can be used to follow the guidewire to the point of obstruction; total recanalization of the lesion by ultrasound guidance has been described. Fluoroscopy provides real-time imaging, but operator radiation exposure can be significant in pedal access cases due to the need to have "working room" while accessing the vessel, which may require the II to be raised, thereby increasing radiation scatter. If present, vessel calcification can provide a target. Roadmapping can be used as well and may even be advantageous if the target vessel is deep (eg, peroneal artery); however, this technique is often compromised by patient motion.

Micropuncture access is universally used due to the size of the native pedal vessels. Several device makers have specific pedal access kits, typically consisting of a 21-gauge needle and a coaxial dilator system. If the primary intervention is planned from below, dedicated sheaths have been designed for this purpose. Our practice is to perform pedal access for lesion crossing only, with rare exception. Our preferred imaging modality is ultrasound, with use of roadmapping for retrograde peroneal artery puncture, due its usual depth.

EXIT STRATEGY: "GETTING OUT OF DODGE"

Equally as important to vessel access is an exit strategy that avoids local complications. Acute occlusion, thrombosis, or dissection can be a devastating outcome after a complex tibial intervention. Manual pressure is most commonly used at the level of the foot; however, prolonged balloon inflation is also very commonly used if any kind of wire-reversal technique has been employed. This is the standard approach in our practice. Use of radial artery bands and blood pressure cuffs are also used, especially if direct manual pressure cannot be reasonably applied to the access site.

CONCLUSION

TPAA provides alternative access for LEAI when routine antegrade access has failed, or in some practices as primary access for below-the-knee intervention. Well-thought-out case planning is imperative to ensure successful entry and exit in this challenging patient population. ISET 2017 offers several sessions to learn more about these techniques in greater detail. ■

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MICHAEL *from page 1*

famous multidisciplinary collaborations was born. Dr. William Krippaehne was a general surgeon involved in the case, and given his knowledge of Dr. Dotter's pioneering catheter technique, asked Dr. Dotter to evaluate her. She was found to have a superficial femoral artery stenosis and was treated by Dr. Dotter with his dilating catheter technique. Her pain was relieved in a week and her foot ulcer healed.[1]

CASE DESCRIPTION

Fast-forward 53 years: A 70-year-old diabetic woman presented to the emergency department with a painful right foot wound sustained after dropping a chef's knife on it four weeks prior. Prompt bedside podiatry evaluation revealed a dry, cold, gangrenous right foot associated with extensive subcutaneous emphysema (Figure 1). Her erythrocyte sedimentation rate was 65 mm/hour and white blood count was 25,000. Bedside decompression and debridement by the podiatry resident revealed extensive liquefactive necrosis with purulent drainage. Her wound was packed and she was scheduled the next day for a second debridement in the operating room with her attending, an experienced podiatrist in foot and ankle trauma, reconstruction, and wound care. Non-invasive arterial studies ordered prior to surgery confirmed occlusions of all three tibial vessels bilaterally on the target limb. Round two of debridement involved incision and drainage of a deep abscess, second ray amputation, and right foot partial amputation. Extensive liquefactive necrosis was present, no healthy bleeding or granular tissue was visualized, and the entire foot was determined to be non-viable (Figure 2). Vascular surgery was consulted for a below-the-knee amputation (BKA). Before a BKA workup was initiated, the patient refused amputation causing a dilemma for the internal medicine service coordinating her care who believed that forgoing treatment for her Rutherford Classification 6 foot would endanger her life.

Two weeks prior, her hospitalist attended a critical limb ischemia (CLI) lecture entitled "A Multidisciplinary Approach to CLI, Amputation Prevention, and Wound Care" given by the author and a wound care podiatrist. She stated, "What do we have to lose by consulting for a limb salvage evaluation?" With the patient refusing amputation, the author was consulted as a second opinion. Evaluation and discussion ensued with the patient, vascular surgery, and podiatry. The group concurred that in-line flow to the foot would be attempted and if established, a trans-metatarsal amputation (TMA) alone would be considered. The next morning, the patient was in the interventional cardiology laboratory for a diagnostic angiogram.

FAST AND FURIOUS PROTOCOL

The author's institution's radial access diagnostic protocol for CLI employs a safe, fast, and economical approach to initiating the limb salvage procedure. If non-invasive studies have not yet been obtained, yet physical exam findings are highly suggestive of ischemia, the pathway to angiography is not delayed, especially as they have been proven to be unreliable in the workup of CLI patients.[2,3] If renal function is not impaired, a 130 cm pigtail catheter is positioned in the terminal aorta for a low contrast DSA angiogram of the terminal aorta and bilateral ileo-femoral vessels to rule out inflow disease. If renal function is impaired, a 150 cm support catheter is used to selectively engage the extremity of interest. A low volume diluted contrast injection is used to take a DSA image of the inflow vessels; if no inflow disease is present, the support catheter is advanced over a wire as distal as possible, and the second DSA angiogram is performed to define the tibiopedal anatomy and plan for wound directed therapy. This protocol allows for decreased bleeding risk, faster recovery and discharge times, and cost savings, all while obtaining detailed DSA images from the wrist. After the angiogram, a CLI revascularization sheet is completed. This quickly details a vascular diagram including CTOP (chronic total occlusion crossing approach based on cap appearance) analysis, timing (inpatient or outpatient) for revascularization, type of anesthesia needed, access site planning, equipment needed, patient/room setup, and WIfI score. The plan is then communicated with the podiatry team to coordinate debridement. The CLI coordinator promptly schedules the intervention. For the limb salvage program, CLI is to peripheral artery disease (PAD) what acute coronary syndrome (ACS) is to coronary artery disease (CAD). It necessitates employment of an early invasive approach to revascularization using whatever strategy necessary to prevent major amputation.

PATIENT ANATOMY

A diagnostic low contrast technique DSA angiogram of the right leg using a 150 cm 0.035-inch Quick-Cross Catheter (Spectranetics) was performed. As expected, the patient had severe infrapopliteal disease with chronic total occlusions (CTOs) of her anterior tibial (AT), peroneal and posterior tibial (PT) arteries, reconstitution via collaterals, and no in-line flow to the wound (Figure 3). The wound-directed therapy plan was to revascularize all three tibials if safe and possible. She was scheduled for intervention the following afternoon.

Figure 1. Rutherford Classification 6 critical limb ischemia (CLI) with extensive subcutaneous emphysema, liquefactive necrosis, and major tissue loss extending trans-metatarsally.

Figure 2. After a second debridement, the foot was deemed to be non-salvageable.

REVASCULARIZATION APPROACH

Ultrasound-guided antegrade micropuncture access was obtained in the right common femoral artery (CFA) and upgraded to a 6 French, 55 cm Flexor sheath (Cook Medical), which was positioned in the P3 segment of the popliteal artery. The peroneal artery was initially chosen to begin angioplasty in order to have access to interventional collaterals to the tibial vessels. A 0.014-inch 300 cm Runthrough wire (Terumo Medical) and 0.018-inch CXI 150 cm support catheter (Cook Medical) were used to cross the multilevel peroneal CTO caps. Orbital atherectomy was performed using a 1.25 solid crown (Cardiovascular Systems, Inc). Sequential balloon angioplasty was performed using a 3.0 mm x 200 mm, 4.0 mm x 200 mm, then 4.0 mm x 80 mm IN.PACT Admiral drug-coated balloon (Medtronic) into the tibioperoneal trunk. The gear was then removed from the peroneal artery and an antegrade approach to the AT was attempted.

When success was not achieved quickly due to distal cap morphology and

Continued on page 14

Figure 3. Diagnostic angiogram with radial access protocol.

Figure 4. "Slip-n-Slide" using the anterior communicating artery as an interventional collateral.

Figure 5. Three-vessel inline flow established.

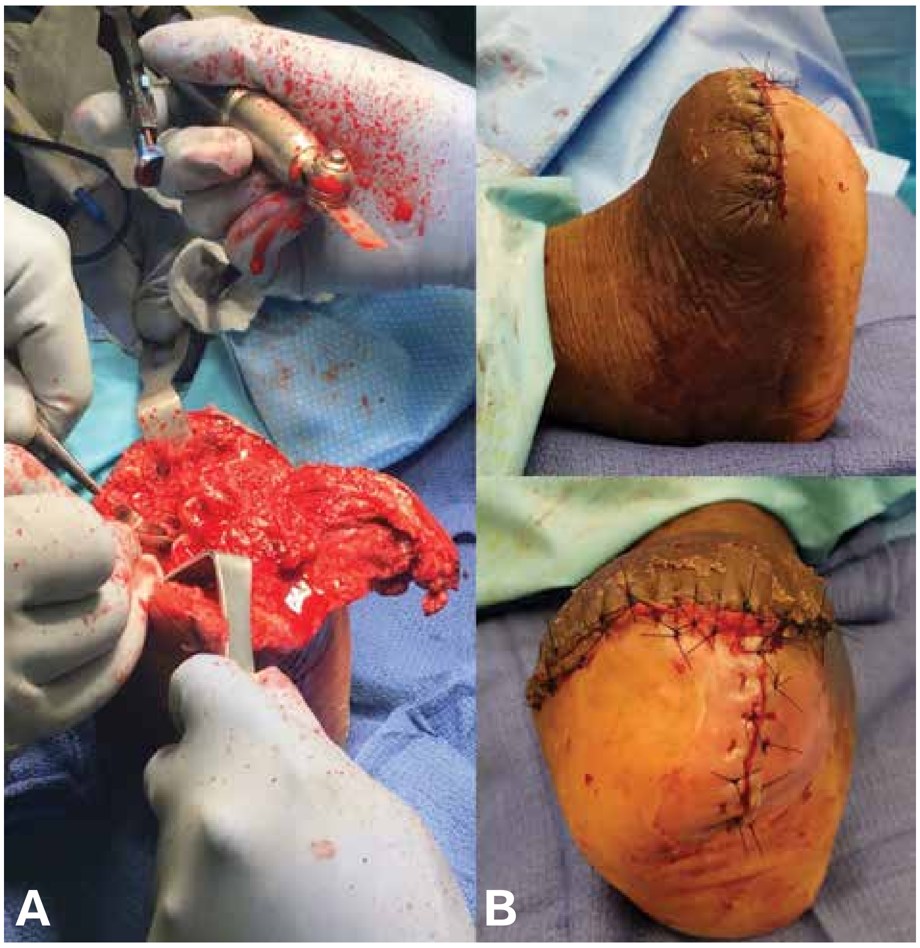

Figure 6. Healthy bleeding (A) and successful trans-metatarsal amputation (B).

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dissection with wire escalation, bail out to a trans-tibial technique to keep the case moving and work against the amputation clock, fueled by radiation and contrast dose. A 0.014-inch CXI 150 cm support catheter was introduced back into the now patent peroneal artery over a 0.014-inch Regalia wire (Asahi). The Regalia was used to gently navigate the tortuous anterior communicating artery. The CXI catheter was advanced gently across the loop, and retrograde trans-tibial crossing of the distal AT CTO cap was achieved. This unit was advanced and "externalized" back into the popliteal artery, forming a complete tibial loop. To save time and cath lab staff energy, a "slip-n-slide" technique was used instead of a traditional floss wire reversal technique to reverse access direction (Figure 4). A 0.014-inch Gladius 300 cm wire (Asahi) was slipped in an antegrade fashion over the Regalia, slid down the AT, and crossed the distal AT CTO cap, delivering the Gladius into the dorsalis pedis artery. A retrograde injection via the peroneal artery was used to confirm in phase with the architecture of the dorsalis pedis (DP) artery. The support catheter was aspirated and diluted contrast was injected to confirm we were in the true lumen of the DP. The Gladius was switched over a 0.014-inch CXI for the 0.014-inch Viper wire (Cardiovascular Systems, Inc). Orbital atherectomy was performed from the popliteal artery down into the DP followed by balloon angioplasty with a 3.0 mm x 200 mm Armada balloon (Abbott Vascular), 4.0 mm x 200 mm Armada, and finishing with the proximal AT into the popliteal artery with a 4.0 mm x 100 mm drug-coated balloon (Medtronic). A low contrast DSA angiogram now confirmed

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JAFF *from page 1*

utilize extravascular ultrasound (EVUS) to help guide our interventions, significantly decreasing the amount of radiation utilized. A combination of contrast and radiation-sparing techniques could be utilized to treat CLI patients with complex, multi-segmental, and multi-vessel disease in the same setting, which is a compelling argument in favor of the wide adoption of these techniques. In the meantime, while these are not widely and frequently utilized techniques, staging is a safe strategy for the most complex anatomies. It should be understood that in patients with Rutherford V and VI, staging should be planned ahead of time, without assuming the old strategy of "lets see what happens" before bringing the patient back to complete the planned revascularization.

Jaff: Do you believe single-vessel runoff provides sufficient blood supply and why?

Ansel: This is an often debated question and should be centered around wounds. Single-vessel runoff appears to be adequate for claudicants with rest pain or whose disease is higher up on the limb. My thinking for Rutherford class V and VI is that if there is a patent plantar arch, then typically one vessel revascularization is sufficient. If the plantar arch is not patent, my personal opinion is that if you can open the vessel to the wound, it is typically adequate. If it is indirect, then only time will tell.

Lyden: Single-vessel runoff can provide sufficient blood supply, based on surgical experience.

Lookstein: I believe that single-vessel runoff provides sufficient blood supply if the single vessel supplies direct circulation to the angiosome in the setting of tissue loss.

Diaz-Sandoval: Ten years ago, there was no scientific evidence upon which to base an answer to this question. Nowadays there is a growing body of literature upon which an attempt can be made to answer this question properly. The problem is that the available data are largely retrospective in nature from single-center experiences and introduce different types of bias. Nonetheless, I believe that the best answer to the question is that it depends. It depends on the wound's 1) location: ulcers in the tips of toes behave differently from those located in the heel; 2) extension: whether it affects only one angiosome or actually spans more than one; 3) size/depth: the bigger and deeper, the most difficult it will be for it to heal, and the concept of "blood volume" supplied to the foot becomes more attractive. In 2008, Davies et al[3] documented that the number of runoff vessels was directly related to the patency and clinical outcomes of interventions performed in the femoropopliteal segment in patients with CLI, seemingly settling the issue. However, in 2010, Iida et al[4] published their experience on 177 CLI patients with Rutherford V and VI, concluding that patients treated with establishment of direct in-line flow to the angiosome where the wound was located had better limb salvage rates at 4 years than those where "indirect flow" to the affected angiosome was obtained. They also concluded that the number of patent runoff vessels did not affect the limb salvage rate in either group. Therefore, based on science, the answer is that we don't yet know whether single runoff or multiple-vessel runoff is the right answer. Furthermore, recently CLI interventions have expanded once more, with the inclusion of infra-malleolar and pedal arch interventions, raising the question of what really matters — tibial runoff or pedal arch integrity? All of these unanswered questions leave the door open for yet another question, based on an attempt at a completely different perspective — would a measurement of end-organ tissue perfusion be more accurate in predicting outcomes of CLI interventions than the number of runoff vessels, runoff quality (direct or indirect), or pedal arch patency?

Jaff: What is the appropriate revascularization strategy for patients with multi-segmental disease?

Ansel: This is often determined by the local expertise. Whichever strategy is chosen, there should be complete revascularization and close follow-up. There are trade-offs between patients and a surgery versus an endo approach or even a high bred approach. Though the BEST CLI trial will attempt to answer this question, there will still be a large group of patients not in that trial with CLI. At our own institution, we certainly are more endo focused but do not hesitate to use whichever therapy will provide complete perfusion at all levels.

Lyden: I believe that inline flow with palpable pulse and perfusion to the area of tissue loss is the appropriate revascularization strategy for patients with multi-segmental disease. When the disease is contiguous, there is no best answer and I hope the BEST trial provides some insight.

Lookstein: When patients present with multi-segmental disease, an attempt to perform complete revascularization as the initial strategy with straight uninterrupted flow to foot should be attempted. If this attempt is not successful at starting wound healing after a few weeks, then plan for a second tibial vessel revascularization, particularly angiosome-directed revascularization.

Table 1: SFA characteristics for endovascular intervention.

Artery Characteristics	Favorable for EVT	Middle Ground for EVT	Less Favorable for EVT
Length of lesion	<10 cm	10-15 cm	>15 cm
Artery diameter	6 or > mm	5-6 mm	4 or < mm
Quality of R/off	All 3 patent	1-2 patent	Zero patent
Plaque dominant	Minimal	Moderate	Extensive
Medical compliance	Yes	Single agent	No

Table 2: Tibial artery characteristics for favorable endovascular intervention.

Artery Characteristics	Favorable for EVT	Middle Ground for EVT	Less Favorable for EVT
Lesion length	<1/3 of vessel	Up to 2/3 of vessel	>2/3 of vessel
Artery diameter	3 or > mm	2-3 mm	2 or < mm
Calcification	None	Mild	Concentric
Location in vessel	Proximal only	Diffuse	Distal only
Direct flow to ulcer	Yes	Indirect	No

Diaz-Sandoval: Yet another complex question without a straightforward answer. This will depend on the patient's anatomy. If there is iliac involvement, with femoro-popliteal and infrapopliteal involvement, it could be argued that a pre-planned strategy could be pursued to either revascularize each segment at a different stage (depending on complexity, renal function, availability of contrast, and radiation-sparing techniques), or to revascularize the iliacs and fem-pop at the same time; then, at a second time, pursue the tibials/pedal vessels. We must also consider the patient's history (number of previous procedures, comorbidities, and availability of suitable conduits) and the available technology. Patients that have involvement of the common femoral and SFA/profunda bifurcation, as well as the SFA and infrapopliteal segment, could be best served by a hybrid approach, which would require (depending on geography) the availability of a team of vascular surgeons and endovascular specialists, or vascular surgeons with endovascular expertise, to potentially address the CFA bifurcation with an endarterectomy and the remainder of the disease with endovascular techniques. Currently, the NIH is funding the BEST CLI trial as an attempt to answer this question in the modern-era. However, enrollment has been slow due to the many real-world limitations that make the treatment of CLI such a convoluted conundrum, which we are passionately trying to untangle. Perhaps the best answer to this question will require breaking the existing walls built by self-interest, in order to generate a new era of team-based approach where the best of each team member is brought forward and the best outcome is achieved.

Jaff: Do you have a guiding principle that helps you make difficult treatment decisions?

Gray: Any treatment starts with knowing the patient. The history and physical examination and non-invasive studies all mean something and impact treatment decisions. The presence of concomitant medical conditions impact risk assessment (ie, renal failure, heart failure, COPD, inactivity, etc.). Secondly, the severity of limb-threatening ischemia as predicted by level of pain, extent tissue necrosis, and location of tissue loss needs to be considered. Thirdly, the anatomic burden of disease (Tables 1 and 2) and the likelihood of a favorable outcome using endovascular intervention should be taken into consideration. Once these pre-procedure facts are known, then by using sound reasoning or judgment, the best treatment option should be provided. For example, for an inactive patient who has a small ulcer on her toe with iliac occlusion and a well-collateralized long SFA occlusion with patent tibial arteries, I would fix the iliac first and see how she does.

Take the same patient with a short SFA stenosis and stenotic anterior and posterior tibial artery involving less than 1/3 of each vessel; I would treat it all in one setting.

When the patient can tolerate the procedure (time, contrast, etc.), in my opinion, the more tibial arteries flowing into the foot, the better. So with favorable anatomy, I treat as much as I can. ■

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the culprit aberrant muscle responsible for the dynamic arterial obstruction in addition to possible smooth muscle relaxation of the popliteal artery, leading to vasodilation. Wang et al recently published an innovative treatment of PAES through an endovascular approach.[11]

A team approach toward management of this condition is essential, since diagnosis and surgical intervention require a high level of coordination between the physicians involved in the invasive and non-invasive diagnostic modalities, as well as the operating surgeons. An increasing number of cardiologists are currently performing peripheral interventional procedures. Sound understanding of PAES becomes imperative to prevent inadvertent percutaneous intervention of this condition with either angioplasty or stent placement. A high degree of suspicion for PAES is necessary on the part of cardiologists and vascular surgeons when faced with this clinical scenario. Despite recent advancement in diagnostic modalities, a surgical approach continues to be the procedure of choice when it comes to treatment of PAES. Although surgery can be curative, it comes with its own set of potential complications, such as postsurgical compartment syndrome. Further research is needed to evaluate other minimally invasive treatment modalities, such as botulinum toxin injection therapy and endovascular treatment. ■

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RUOTSI & WEIR *from page 3*

and exudating wounds should be treated with moisture wicking dressings such as alginates or hydrofibers.

5) **Edge:** Keratinocytes cannot migrate off undermined, callused, or rolled wound edges. Efforts should be directed toward debridement or excision of wound edges so that a clean edge attached to the wound bed is the end result. By the same token, cells will not migrate off the edge unless the wound bed is well prepared and receptive to cellular migration and adhesion.
6) **Periwound skin:** Care to preserve the integrity of the periwound skin must be a consideration in day-to-day wound care. Maceration from excess moisture or trauma from carelessly applied or removed dressings and tape frequently impede the wound healing progress.
7) **Wound treatment:** Modern wound care brings a number of dressing categories that can address the needs of the wound. Gone are the days when wet to dry dressings with cotton gauze will suffice. Moist, or wet to dry dressings, are an occasionally appropriate nonselective wound debridement modality; however, they have no place in the wound healing paradigm. Choosing a dressing material that will match the needs of the wound is essential, whether you're adding moisture to a dry wound, absorbing exudate from a wet wound, filling in space, maintaining temperature, and/or protecting from further injury. To that end, choosing a dressing to meet the anatomic and physiologic needs of the wound is essential.
8) **Advanced modalities:** The use of products that have the ability to supplement wound healing may be a logical choice for a wound that has been stalled due to lack of blood flow. Advanced biologics, cellular- and tissue-based products, negative pressure, and other modalities can enhance wound healing in an otherwise healing-challenged wound. Knowledge of the nuances of obtaining, documenting, and billing these modalities is essential, again reinforcing the need to work with a local wound center.
9) **Underlying comorbidities:** The people we care for are often acutely as well as chronically ill, with multiple comorbidities, laboratory abnormalities, and long lists of medications. A careful analysis of these coexisting disease states with treatment of underlying abnormalities will often improve the healing trajectory. Correction of laboratory abnormalities such as glucose levels and renal parameters can help wounds perform better due to improved metabolic and physiologic status. Last, polypharmacy is an often-overlooked source of wound healing retardation. Many medications, such as systemic corticosteroids, non-steroidal anti-inflammatory drugs, and chemotherapeutic agents can be remarkable impediments to wound healing.
10) **Ongoing perfusion:** A reality of post-revascularization wound care is that arterial patency is not always sustained. Ongoing surveillance of wound and periwound perfusion is essential to assure ongoing progress. Diagnostic modalities, such as laser doppler flowmetry for skin perfusion pressure or transcutaneous oximetry for tcpO_2, are useful adjuncts in this process and can signal when additional revascularization efforts may be indicated.

Polypharmacy is an often-overlooked source of wound healing retardation. Many medications, such as systemic corticosteroids, non-steroidal anti-inflammatory drugs, and chemotherapeutic agents can be remarkable impediments to wound healing.

The above are not necessarily listed in order of priority, but rather as a group of considerations and interventions to assist in moving a previously ischemic and problematic wound toward healing. As always, wound healing requires a multifaceted approach in order to support the anatomic and physiologic factors necessary for wound healing and mitigate those forces that would combine to impede or prevent it. Developing a collaborative relationship with a wound center and specialized wound clinicians should be an essential component of any limb salvage program and an obvious first step after flow has been reestablished. ■

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brisk two-vessel runoff via the peroneal and anterior tibial artery. However, the pedal loop was not yet established to provide true wound-directed therapy. The proximal cap of the posterior tibial artery was engaged with a 0.014-inch Gladius wire in a 0.014-inch 150 cm CXI catheter and the length of the PT was navigated. However, antegrade wire escalation and catheter advancement proved difficult. Therefore, re-entry into the peroneal artery using a trans-tibial interventional collateral was performed through the posterior communicating artery to modify the distal cap in a retrograde fashion. Returning antegrade, a 150 cm Turnpike LP catheter (Vascular Solutions) was rotated with a 300 cm Pilot 200 guidewire (Abbott Vascular) across the distal PT CTO cap. The Pilot wire was exchanged for an atraumatic Regalia wire, which was used to navigate the lateral plantar artery and park the Turnpike in the dorsalis pedis. An exchange was made for the Viper wire and orbital atherectomy was performed again. Successive balloon angioplasty of the PT was performed using a 3.0 mm x 200 mm Armada, 4.0 mm x 200 mm Armada, and finishing proximally with a 4.0 mm x 150 mm drug-coated balloon TPT. Final injection revealed brisk three-vessel runoff to the foot with a completed pedal loop and negligible residual stenosis (Figure 5). An antegrade Perclose (Abbott Vascular) was then used for hemostasis.

Following revascularization, the patient underwent a successful TMA with significant bleeding throughout the surgery (Figure 6). A major amputation was prevented through a true multidisciplinary collaboration.

D.R.E.A.M. (Docs Revascularizing Extremities Against Major Amputation) is a movement consisting of community physicians, training programs, and administrations joining forces against CLI in the Palm Beach County, Florida community. The objective of the collaboration is to promote angiography prior to amputation, achieve wound-directed therapy, ensure close multidisciplinary inpatient and outpatient follow-up, close the gap on disparities in amputation rates and revascularization, and promote community PAD/CLI awareness because saving limbs saves lives. ■

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grafting or endarterectomy. Young athletes are advised to limit or stop contact sports to reduce trauma to the popliteal artery until definitive therapy.[4]

CONCLUSION

Symptomatic PAES is a rare and potentially devastating condition that affects otherwise healthy individuals. There is often a delay after first presentation while other diagnoses are considered or even treated without improvement. Early recognition based on the history and physical exam findings is paramount to initiate treatment and avoid late-stage irreversible damage. Awareness of PAES and an index of suspicion need to be maintained for timely diagnosis and definitive treatment which involves surgical decompression with or without initial endovascular stabilization. ■

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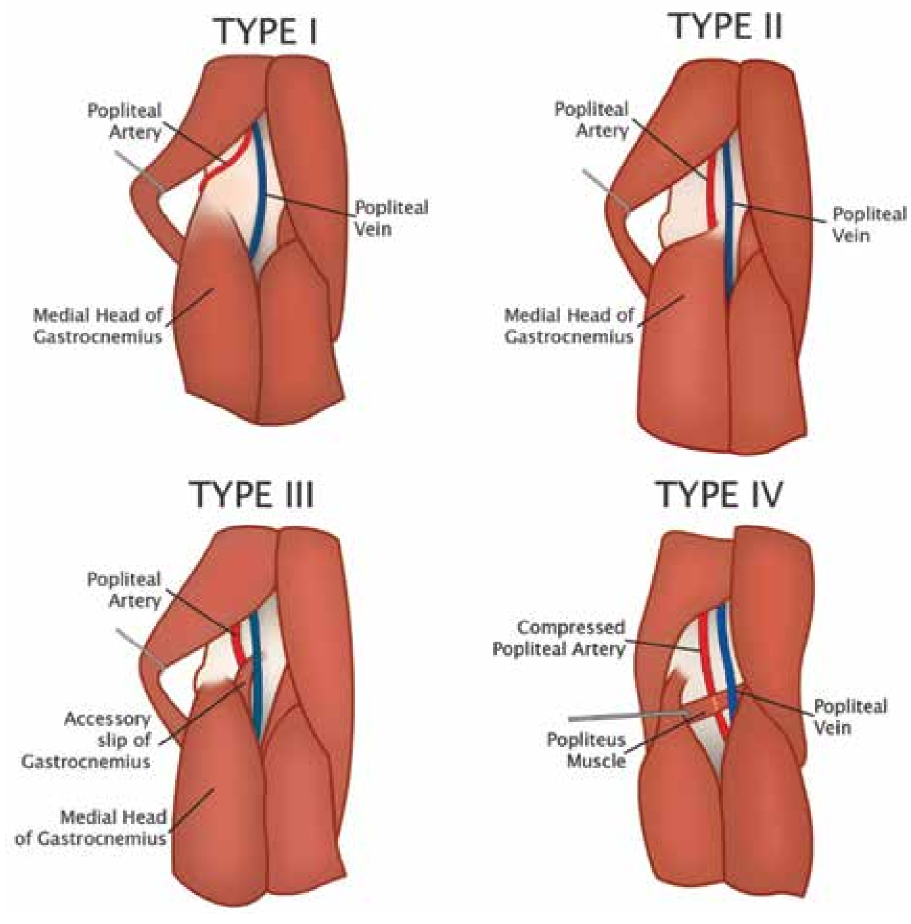

Figure 3. The four most common types of popliteal entrapment.[8]

Upcoming Meetings and Events

January 30–February 2, 2018
Leipzig Interventional Course
Location: Leipzig, Germany
Website: www.leipzig-interventional-course.com

February 3–7, 2018
International Symposium on Endovascular Therapy (ISET)
Location: Hollywood, Florida / Venue: The Diplomat Hotel
Hashtag: #ISET2018
Website: www.iset.org

March 10–12, 2018
American College of Cardiology Scientific Sessions (ACC)
Location: Orlando, Florida
Website: http://acc2018.com

March 17–22, 2018
Society of Interventional Radiology (SIR) Annual Scientific Meeting
Location: Los Angeles, California
Website: www.sirmeeting.org

April 24–27, 2018
Charing Cross International Symposium (CX 2018)
Location: London, UK / Venue: Olympia London
Website: www.cxsymposium.com

April 25–28, 2018
Society of Cardiovascular Angiography & Intervention (SCAI)
Location: San Diego, California
Website: www.SCAI.org

August 8–11, 2018
Amputation Prevention Symposium (AMP)
Location: Chicago, Illinois / Venue: Hilton Chicago
Website: www.amptheclimeeting.com

REVEALING REAL-WORLD GLOBAL DATA

LUTONIX® 035 DCB had the **highest reported freedom from TLR rate** at 2 years among real world patients in two separately-conducted registry studies.[1]

In.Pact Global Study

83.3%

FREEDOM FROM CD-TLR At 24 Months[2]

In.Pact™
Drug Coated Balloon

Lutonix Global SFA Real-World Registry

90.3%

FREEDOM FROM TLR At 24 Months[3]

LUTONIX® 035
Drug Coated Balloon PTA Catheter

[1] Data are not from head-to-head clinical studies and are provided for background and educational purposes only. Different trials have varying patient profiles, protocol structures and other criteria that may affect reported outcomes.
[2] In.Pact™ Global Study, n=1,406. Medtronic reported data, VIVA 17, 2-Year Results from the In.Pact™ Global Study, Thomas Zeller, MD, PhD. Primary endpoint defined as freedom from clinically driven (CD) target lesion revascularization (TLR) within 12 months. Freedom from TLR was 92.6% through 1 year. Freedom from CD-TLR was 83.3% through 2 years.
[3] LUTONIX Global SFA Real World Registry, n=691. Primary efficacy endpoint is defined as freedom from TLR at 12 months. TLR Free rate by subject counts at 12 months was 93.4% (605/648). The Kaplan-Meier TLR-Free survival estimate was 94.1% at 12 months and 90.3% at 24 months. In the LEVANT 2 IDE Clinical Trial, treatment with LUTONIX® 035 DCB resulted in freedom from TLR rate of 87.7% at 12 months (250/285) and a freedom from TLR rate of 82.0% at 24 months. Data on file, Bard Peripheral Vascular, Inc.

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