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CryoPlasty Therapy

Treatment for Infrainguinal Disease and Critical Limb Ischemia

Experts discuss the current state of the art in lower-extremity therapy, today's data and devices, and effective treatment strategies.

Robert S. Dieter, MD, RVT

John R. Laird, Jr, MD

Zvonimir Krajcer, MD

Alan B. Lumsden, MD

Ali Morshedi-Meibodi, MD

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Anatomy and Physiology of the Superficial Femoral Artery

Should these factors dictate the type of intervention?

BY ALAN B. LUMSDEN, MD

The superficial femoral artery (SFA) is one of the most common vessels in the lower extremity to be affected by atherosclerosis.[1-3] It is also one of the most heavily diseased arteries in the body; diffuse disease and heavy calcification are common. However, clinically significant stenoses are traditionally described as developing in the "the adductor canal," specifically in the region of the adductor hiatus. Little is known about the anatomy of this canal.[4] Almost nothing is known regarding how this canal functionally interacts with the SFA as it transits from the adductor compartment into the popliteal fossa. Functional imaging may be more important in this location than perhaps at any other vascular location[5] because of the complexity of the flow patterns and the mechanical forces exerted by walking, kneeling, and exercise.

To understand the localization of plaque and the poor performance of stents, several issues must first be considered:

- What is the adductor canal and the adductor hiatus?
- How does it impinge on the SFA?
- What happens when the knee is bent passively and actively?
- How does the development of atherosclerosis affect these interactions?

WHAT IS THE ADDUCTOR CANAL?

A mentor of ours once asked: Is anatomy an elective subject in this medical school? Thus, he emphasized the lack of basic anatomic knowledge in finishing medical students. Understanding the anatomy of the femoral artery is an important starting point. Anatomically, there is no SFA. Many clinical specialists call the femoral artery the artery below the profunda femoris origin. This is to distinguish the femoral artery segments above (common femoral artery) from that distally (SFA) to the profunda or deep femoral artery. These terms, historically, have not

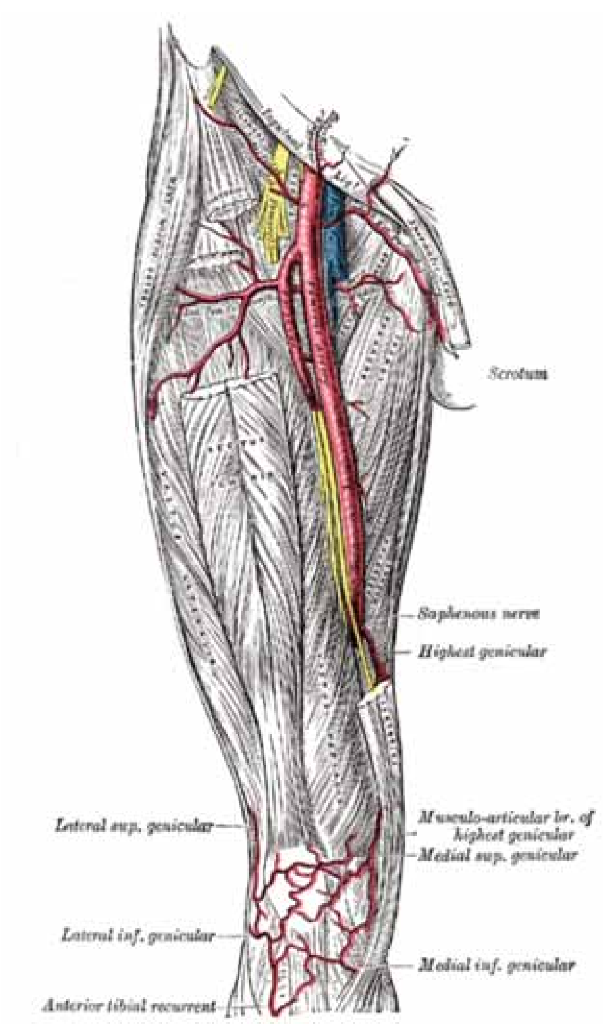

Figure 1. Anatomy of the femoral artery showing the transit of what clinicians refer to as the SFA through the adductor canal to the adductor hiatus.

been used by anatomists, but are so entrenched in the clinical vernacular that change is unlikely. It does, however, lead to confusion when reading anatomical texts. The SFA courses through the adductor canal, terminating where it passes through the adductor hiatus, becoming the popliteal artery (Figure 1). The adductor canal (Hunter's canal) is an aponeurotic tunnel in the middle third of the thigh, extending from the apex of the femoral triangle to the opening in the adductor magnus. It is bound, in front and laterally, by the vastus medialis; behind by the adductor longus and magnus; and is covered by a strong aponeurosis that extends from the vastus medialis, across the femoral vessels to the adductor longus and magnus; lying on the aponeurosis is the sartorius muscle. The canal contains the femoral artery and vein, the saphenous nerve, and the nerve to the vastus medialis.

The adductor hiatus is an opening in the tendon of insertion of the adductor magnus muscle, whose boundaries are medial (portion of the tendon of adductor magnus that attaches to the adductor tubercle of the femur); lateral (insertion of the adductor magnus into the linea aspera); and inferior (femur). Through this hiatus at the end of the adductor canal passes the SFA and vein, becoming the popliteal artery and vein.

HOW DOES THE ADDUCTOR HIATUS IMPINGE THE SFA?

Numerous studies have confirmed that the distal SFA is the site of 60% to 70%[2,4] of all occlusive lesions in the femoropopliteal region, and that the disease is typically symmetrical in both legs. Scholten,[5] in a duplex ultrasound study, noted that 72% of all occlusions occurred at the region of the adductor hiatus.

Exactly which factors localize atheroma in this region is not understood. The likely importance of local anatomic factors in atherogenesis is widely assumed but poorly defined. The recent finding of a high incidence of stent fractures in this area has further focused attention on local mechanical forces. Important regional factors may include surrounding structures, branches, and local morphologic characteristics of the vessel. The physical properties of the surrounding tissues are dissimilar.[6] In the adductor canal, the femoral artery is surrounded by the firm muscles of the thigh. When it leaves the canal, the artery crosses the sharp edge of the aponeurosis of the adductor magnus and enters the soft, fatty tissue of the popliteal fossa.

The mechanisms of atherogenesis at the adductor hiatus are (1) pulsatility of the femoral artery as it lies in contact with the fibrous, inelastic aponeurosis of the adductor magnus is speculated to result in repeat trauma to the artery;[3] (2) perivascular movement or deformation could be an initiating event in atherogenesis caused by hypoxia of the vessel wall due to obstruction of the vasa vasorum; and (3) spiral anatomy leading to spiral flow patterns that create low shear and can initiate atherogenesis.[1]

In a recent cadaver study by Wensing,[6] however, no relationship was found between surrounding structures of the femoral artery and location of atherosclerotic lesions. Indeed, no lesion localization to the adductor hiatus could be determined. Three-dimensional (3D) reconstructions showed that atherosclerotic lesions were spiraling throughout the artery in 18 of 23 cases. Consequently, the investigators concluded that hemodynamics may play a dominant role in localization of atherosclerotic lesions.

Despite the widespread clinical impression that the adductor hiatus impinges on the femoral artery, the authors have never seen such compression demonstrated radiographically. This could be a consequence of how most imaging is performed with the patient in the supine position and the leg extended. Clearly, this is an easy area for dynamic imaging.

WHAT HAPPENS WHEN THE KNEE IS BENT PASSIVELY AND ACTIVELY?

That there is a paucity of information regarding the effect of exercise on the SFA became apparent with the identification of stent fractures in the SIROCCO trial.[7] Since then, there has been increasing interest in dynamic imaging to improve understanding of flow patterns and the effect of exercise. Magnetic resonance imaging (MRI) is a useful tool for studying arterial geometry noninvasively and, after 3D reconstruction, MRI data can be used in computational fluid dynamics (CFD) modeling.[8] The SFA spirals across the medial aspect of the thigh as it passes through the adductor canal. Geometric risk factors may have a role in the distal SFA, the important relevant feature being curvature or tortuosity of the vessel. This may result in low shear stress, prominent secondary flows, or increased variation in shear stress, which can predispose to atherosclerosis. Flow curvature is known to promote secondary motions with helical streamline patterns, resulting in variations in wall shear, with incipient or actual regions of flow separation, and the observations of helical patterns of atheroma in the femoral artery suggest a relationship of flow patterns with subsequent atherosclerotic disease.

A variety of mechanical forces act on the SFA (Figure 2). It is repetitively deformed in multiple directions by leg movement. With crossing of flexion points and interaction with the surrounding musculature, the SFA is exposed to compression, torsion, and elongation. Deformation is also exacerbated by an intrinsic stiffness of

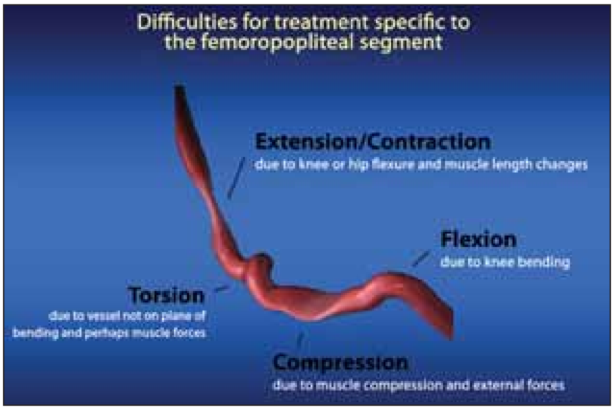

Figure 2. Diagram of the forces acting on the SFA within the thigh. (Courtesy of Joel Morrisett, MD.)

the vessel, which can often be calcified and thus less elastic. The SFA, furthermore, is fixed at the groin and knee and is somewhat immobile in between.

MRI-derived data have been used to measure the 3D tortuosity and curvature of the SFA above, within, and below the adductor canal examining tortuosity and curvature, and to test the hypothesis, via CFD, that these result in hemodynamic differences in the areas of the SFA prone to atherosclerosis (Figures 3 and 4). However, these studies have largely been limited to healthy, normal volunteers, and it is unknown how flow patterns are affected by development of atherosclerosis.

Tortuosity and curvature are significantly greater for men than women, related to increased body surface area, body mass index, or weight in men. In both sexes, tortuosity increased from the mid-thigh to the popliteal fossa. The greatest curvature is found within the distal SFA. CFD modeling was undertaken on MRI-based reconstructions of the SFA. Wall shear stresses were extracted from the computations. Wall shear stresses showed greater spatial variation in the men than in the women, and men exhibited lower mean wall shear stresses. These data indicate that gender differences related to body size and anatomic course of the femoral artery may contribute to the enhanced risk of focal atherosclerosis in the adductor canal.[8]

In a postangioplasty study using intravascular ultrasound (IVUS), The et al[9] reported that extrinsic compression could be identified in 33% of patients at the adductor hiatus. This was apparent in only one patient (7%) before angioplasty. IVUS, however, was useful in distinguishing residual plaque from extrinsic compression. IVUS, therefore, could be an important tool in evaluation of device performance at the adductor hiatus.

Although such studies are beginning to shed light on the mechanisms surrounding the development of distal SFA atherosclerosis, there remain few data on the effects of exercise. That there is clearly repetitive mechanical trauma, not only at the adductor hiatus, but transmitted along the length of the SFA, is clearly demonstrated by the increasing number of reports of stent fractures (Figure 5). In a recent study of SFA stent placement, a

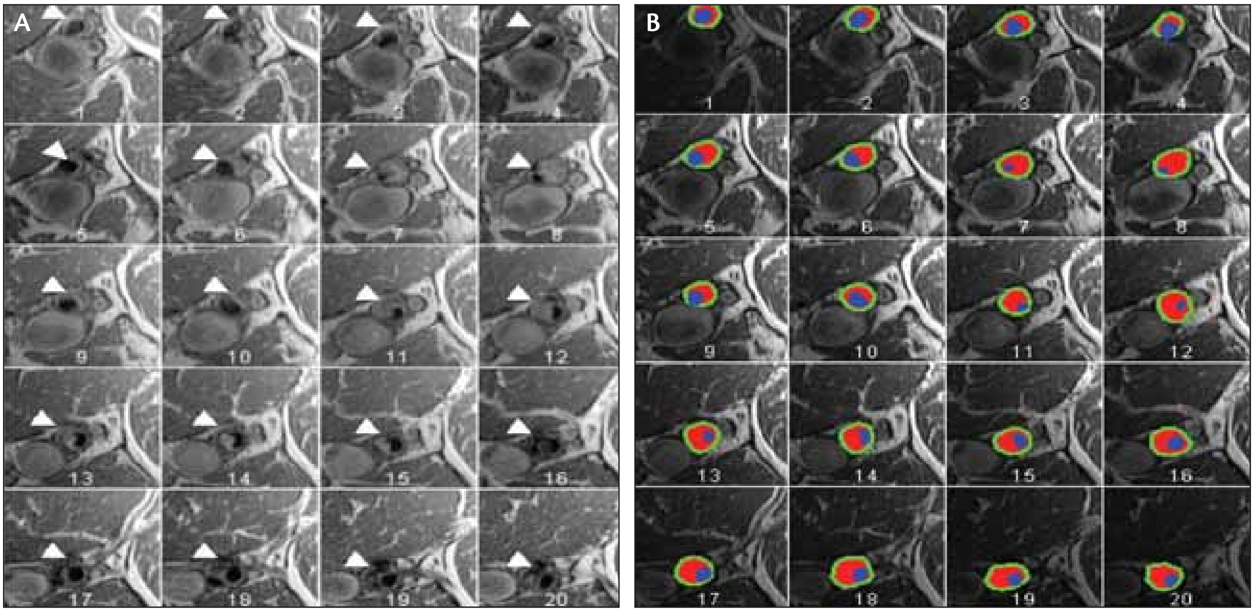

Figure 3. A series of MRI slices of the SFA. Using a semiautomated, edge-tracking algorithm, the lumen of the artery is depicted in blue, the wall in green, and the atherosclerosis in red. This clearly depicts how the atheroma spirals around the vessel wall as it descends through the thigh. (Courtesy of Joel Morrisett, MD.)

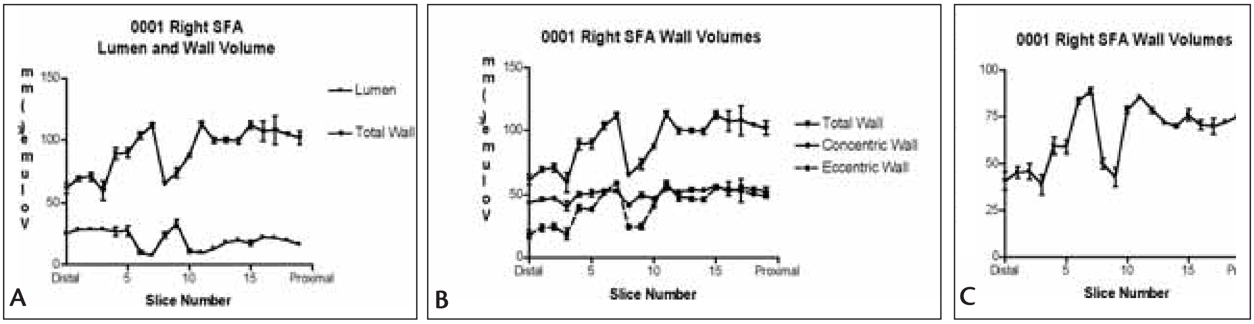

Figure 4. Plots of slice thickness as a function of their linear location along the length of the artery as measured from the profunda bifurcation. Volumes of eccentric wall plaque (A), concentric wall (normal vessel) (B), and lumen (C) are demonstrated. (Courtesy of Joel Morrisett, MD.)

28% fracture rate was noted, and the single most predictive factor was exercise. Walking more than 5,000 steps per day was the strongest independent determinant associated with stent fracture by discriminant analysis (*P*=.0027). Vigorous exercise increases the stent fracture rate in patients with a nitinol stent in the SFA.[10]

HOW DOES THE DEVELOPMENT OF ATHEROSCLEROSIS AFFECT THESE INTERACTIONS?

The answer to this question is very simple: there is almost no evidence on how the development of atherosclerosis affects flow patterns in the SFA, nor how exercise may influence the development of atherosclerosis at the adductor canal. The relative stiffness of the atherosclerotic vessel may result in stress points at the edges of these less compliant regions.

Selecting the Appropriate Intervention in the Distal SFA

The treatment of SFA occlusive disease is undergoing rapid evolution and is currently the focus of much ongoing research. The incidence of lower-extremity occlusive disease is significantly underdiagnosed and, because of limitations in therapy, interventions have been strongly advocated only for those patients with limb-threatening ischemia, or severe short-distance claudication. However, traditional vascular surgical teaching has largely generated negativism regarding SFA interventions: first, do no harm.

This minimalist approach has been based largely on the failure mode of femoropopliteal bypass grafting (ie, when the graft fails, the patient is usually worse than his or her previous baseline, and claudicants could be converted into limb-threatening ischemia). We now know that this is rarely the case with endovascular interventions. When an endovascular intervention fails, the patient is most likely to perceive no continued benefit. Importantly, in most cases, they are not made worse. This understanding clearly changes the dialogue with our patients and lowers, in my opinion, the therapeutic threshold. The role of the physician is to explain to the patient the pros and cons of a procedure, not to deny care.

Once the decision has been made to intervene, the next dilemma is the choice of procedures to perform. This remains under constant evolution. A few widely held

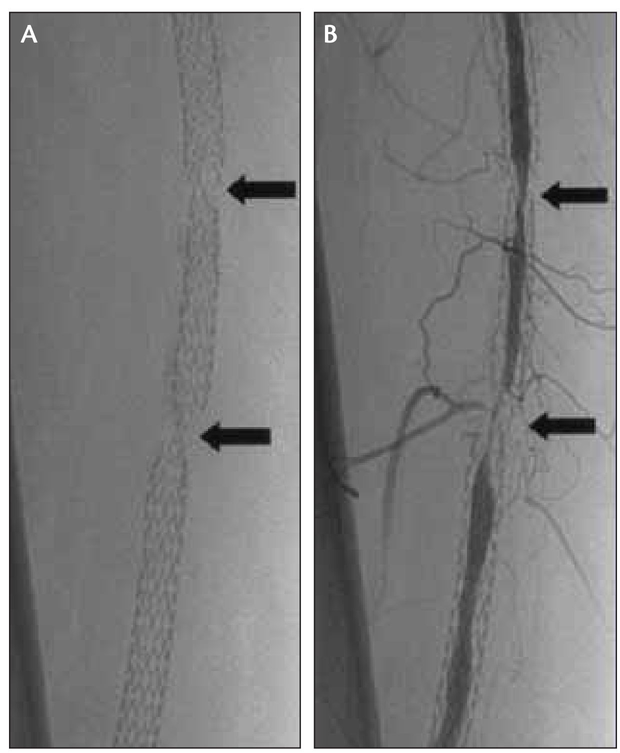

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Figure 5. Angiography of a self-expanding nitinol stent 9 months after implantation showing a severe stent fracture in the distal and a moderate stent fracture in the proximal part of the stent (A). Angiographically, both lesions were associated with a restenosis >50% diameter reduction and an apparent direct relationship with development of neointimal hyperplasia is demonstrated (arrows, B).

beliefs, which are used to guide procedure choices are:

- Short-segment lesions do much better than long-segment lesions.
- There are no durable stents or stent grafts for the SFA.
- Stent fractures have markedly diminished enthusiasm for stenting, which is now largely used for salvage of failed angioplasty.
- The most common reason for failed angioplasty is arterial dissection and resultant flow limitation.
- Controlled-crossing devices will play an important and expanding role to limit loss of normal artery.
- No magic bullet has yet been identified to control SFA neointimal hyperplasia. This remains the single largest limitation of SFA endovascular interventions.

The Case Against Stents

That placement of stents across joints could result in a higher incidence of restenosis was reported in animal studies in which Wallstents (Boston Scientific Corporation, Natick, MA) were placed across the hip joint of pigs. Although in this study, no stent fractures were identified, and follow-up was only 3 months.[11] In most human stent studies, routine stent surveillance with plain x-rays was not performed, so it is likely that fractures were underestimated. The SIROCCO trial was the first major report to identify the prevalence of stent fractures.[7,12] Although initially described as being of no clinical significance, this is now known to be false (Figure 6).[13-15] Indeed, a classification system for the severity of stent fractures has been described, and the risk of restenosis or pseudoaneurysm formation is directly related to the severity of the fracture.[14,15] Consequently, clinical practice has evolved from liberal use of stenting to its use primarily for salvage of failed angioplasty.

Subintimal Angioplasty

Subintimal angioplasty has rapidly gained popularity and, with the development of controlled re-entry systems, now makes a great deal of sense. However, crossing the lesion has not been the principal limitation of lower-extremity intervention. The major technical advances that have been achieved consist of consistent crossing of long-segment lesions, with re-entry at the most proximal suitable lumen. The issues that remain to be resolved are (1) providing an angioplasty result that minimizes the need for stenting, and (2) controlling restenosis. Controlling restenosis remains a remote goal; however, CryoPlasty is one of the few interventions that may reduce the incidence of dissection, which is the main reason for stenting.

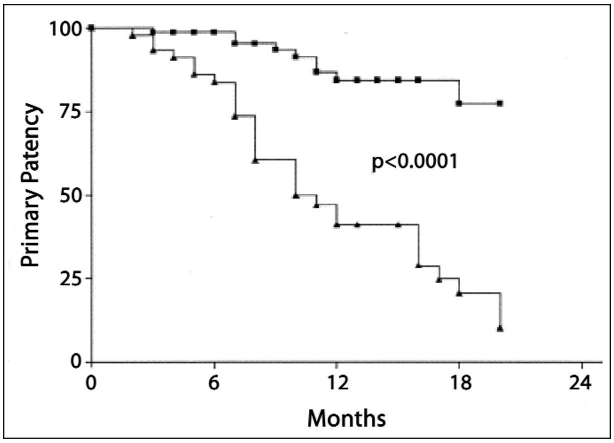

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Figure 6. Primary patency rate at 12 months was significantly lower for patients with stent fractures (41.1% vs 84.3%; *P*<.0001).

The Rationale for CryoPlasty

CryoPlasty uses liquid nitrous oxide as the inflation media, which expands into gas, inflating the balloon to 8 atm.[16-19] The phase change from liquid to gas draws energy, driving balloon surface temperature to -10°C. The theory of the effect of CryoPlasty on a vessel involves:

- *Altered plaque response.* This may occur due to the effect of cooling, which causes the interstitial saline to freeze; as ice forms and expands, microfractures are created that weaken the plaque. This action contributes to more uniform dilation of the vessel and less medial injury.
- *Reduced elastic recoil.* Cooling also induces an alteration of the collagen and elastin fibers, reducing vessel elasticity and protecting against recoil.
- *Induction of apoptosis.* Cooling causes smooth muscle cells to eject water. It is speculated that dehydration and subsequent rehydration may trigger an apoptotic response. This programmed cell death is a noninflammatory response and theoretically may reduce neointimal formation. It has also been demonstrated in cell culture that the physical conditions of the cell and surrounding matrix affects the cellular response to cryotherapy.[20]

Support for the concept that CryoPlasty causes a fundamental change in the arterial wall properties can be extrapolated from bench studies on the freezing of pig femoral arteries. Results suggest that freezing does have an effect on stress-strain properties, particularly in the low-stress region corresponding to physiological conditions. The mechanisms of this change in mechanical properties may include the loss of smooth muscle cell viability, alteration in extracellular matrix, bulk redistribution of water, or changes in alignment caused by ice crystal growth.[21]

CryoPlasty combines the time-tested mechanics of percutaneous transluminal angioplasty with the biologically favorable effects of cryotherapy. In addition, the combined mechanical and freezing effects on the architecture of the vessel wall and the plaque itself have been found to reduce the likelihood of significant dissection after CryoPlasty as compared with those expected with percutaneous transluminal angioplasty. In an era in which there remains considerable concern in placing nitinol stents in the SFA, CryoPlasty may reduce the need for stenting, potentially reducing costs and prolonging patency.

Despite suboptimal results, angioplasty of femoropopliteal arterial lesions has been a mainstay of endovascular therapy for many years. Laird[18] reported the results of a prospective, multicenter trial that evaluated the efficacy of CryoPlasty for femoropopliteal disease. One hundred two patients with claudication and lesions of the superficial femoral and popliteal arteries ≤10 cm were studied. All patients were treated with CryoPlasty alone using the PolarCath system (Boston Scientific). The primary endpoints of the study were acute technical success and clinical patency at 9 months. Technical success was defined as the ability to achieve residual angiographic stenosis no greater than 30%, and residual stenosis <50% by duplex ultrasound (US) imaging. Clinical patency was defined as freedom from target lesion revascularization within 9 months. Primary patency was defined by a duplex US systolic velocity ratio no greater than 2.0.

A total of 102 patients were enrolled at 16 centers. The majority of the lesions were confined to the SFA (84.3%) and 14.7% presented with total occlusions. The technical success rate was 85.3%, with a mean residual stenosis after CryoPlasty of 11.2% ± 11.2% ($P<.05$ vs baseline). Clinical patency was 82.2%. Primary patency determined by duplex US was 70.1%.

Extended clinical follow-up (ranging from 11 to 41 months; mean, 31 months) was subsequently reported for 70 of these initial patients.[16] The clinical patency rate (freedom from target lesion revascularization) calculated by the Kaplan-Meier method was 83.2% after the original follow-up period of 300 days. After >3 years (1,253 days), the clinical patency rate was well maintained at 75%. These limited but encouraging long-term data indicate that CryoPlasty is a durable therapy, with relatively low long-term restenosis rates compared to other endovascular treatment approaches. Other investigators have reported additional experience with the PolarCath system. Fava[22] reported 0% restenosis at 6 months after femoropopliteal angioplasty and noted "CryoPlasty was able to achieve substantial dilation of femoropopliteal lesions with well-preserved late angiographic patency." Patel[23] used CryoPlasty for treatment of a variety of arterial restenotic lesions and found no advantage over other therapies. However, once a lesion has declared itself as "restenotic," I believe that the patient and the lesion are highly prone to recurrence, regardless of intervention. There have been reports of promising results from the use of CryoPlasty to treat restenotic lesions, namely the venous anastomosis of arteriovenous grafts.[24] ■

Alan B. Lumsden, MD, is from the Methodist DeBakey Heart Center, Baylor College of Medicine, The Methodist Hospital, Houston, Texas. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Lumsden may be reached at (713) 441-6201; ablumsden@tmh.tmc.edu.

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Endovascular Treatment of Infrapopliteal Lesions

An overview of the promising approaches to treating CLI and the role of today's endovascular devices.

BY ALI MORSHEDI-MEIBODI, MD; JOHN R. LAIRD, JR, MD; AND ROBERT S. DIETER, MD, RVT

Chronic critical limb ischemia (CLI) generally results from advanced, multilevel atherosclerotic disease of the lower-extremity vasculature. Involvement of the tibioperoneal vessels is normally encountered (Figure 1). CLI presents with ischemic rest pain and progressive tissue loss. Historically, the gold standard for treatment of CLI has been surgical revascularization; however, this approach is limited to subjects with good distal target vessels and lack of severe comorbid condition.

Several case series and the recent randomized BASIL trial[1] have questioned the superiority of surgical revascularization and have confirmed the therapeutic role of tibioperoneal vessel interventions (TPVI) in this patient population. This is due to technical and technological advancements, which have resulted in improved safety and higher success rates of endovascular therapy for TPVI (Table 1).

INDICATIONS

CLI

The recently published BASIL trial demonstrated equivalent 6-month amputation-free survival in patients with CLI treated percutaneously.[1] Furthermore, the endovascular approach was associated with lower 30-day morbidity and mortality. The initial strategy of attempting percutaneous revascularization was not found to hinder future surgical revascularization strategies. Arguably, TPVI should be the initial treatment of choice for CLI caused by infrapopliteal occlusive disease, especially if the life expectancy is less than 1 to 2 years and in patients with significant comorbidities.

It is important to remember that the clinical success of this modality has been superior to its angiographic success because after wound healing was achieved, further restenosis may not be associated with recurrence of ulcers due to the presence of sufficient flow.[2]

Claudication

Classically, infrapopliteal occlusive disease is not associated with lifestyle-limiting claudication. However, some patients will indeed have claudication with infrapopliteal occlusive disease, particularly those with concomitant pedal arch vessel disease. Traditionally, angioplasty for the treatment of intermittent claudication has been avoided due to the concern over major complications. However, recent studies are beginning to question this long-standing dogma. A prospective study of TPVI in subjects with Rutherford class 2 to 3 demonstrated 89.4% procedural success (defined as <30% residual stenosis) after balloon angioplasty and stent placement (if deemed necessary). TPVI resulted in increased walking distance and ankle-brachial index. The complication rate was 5.8%, with no instances of amputation or surgery. The primary patency (on color-flow duplex or angiography) after 12 months was 66.3%.[3]

TECHNICAL CONSIDERATIONS

Access

Appropriately planned access is an important part of the procedure (Table 2). Currently, the most common access methods are contralateral, antegrade ipsilateral, and rarely,

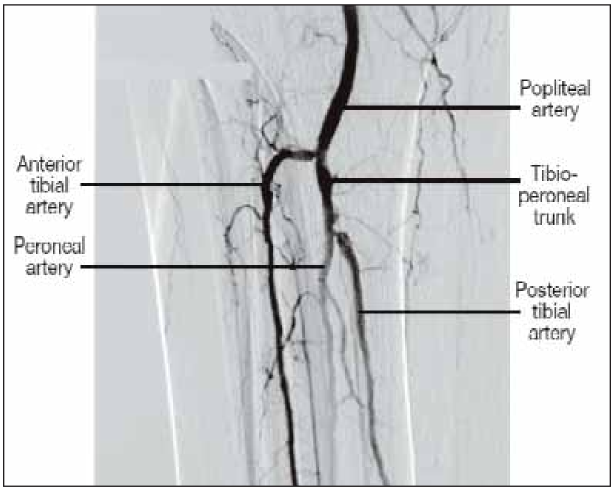

Figure 1. Popliteal artery and bifurcation into a diseased anterior tibial artery and a diseased tibioperoneal trunk. The tibioperoneal trunk divides into a diseased peroneal and posterior tibial artery.

retrograde tibial access.[4] The contralateral approach is the most commonly used method. This approach allows for sufficient pushability in the majority of cases and is ergonomically less challenging. When additional backup is necessary, a guide catheter can be advanced to the level of the popliteal artery. This method also allows contrast injection closer to the region of interest, potentially allowing for less utilization of dye and improved imaging. The other advantage of the contralateral access approach is that during hemostasis, antegrade flow down the treated vessel is not impaired while the contralateral femoral artery is compressed.[5]

In some cases, the contralateral approach is not possible. In patients with an occluded iliac artery or previous aortoiliac reconstruction (either through surgery or stent placement), it may not be technically possible to gain access to the tibial vessels from the contralateral approach. In such cases, an antegrade approach may be preferred. Test angiograms or "road-mapping" through the introducer needle may aid in confirming that the guidewire is directed down the superficial femoral artery prior to sheath insertion. Comparatively, the antegrade access approach is associated with an increased complication rate.

The retrograde tibial access is an alternative approach used when usual antegrade or contralateral retrograde has been unsuccessful. This type of access requires sufficient caliber of anterior or posterior tibial artery below the ankle to allow a percutaneous approach. Access is obtained by using a 21-gauge micropuncture kit (Cook Incorporated, Bloomington, IN). The vessel is wired using a .014-inch or .018-inch guidewire, and the inner component of a 4-F dilator is used to maintain the access. The micropuncture wire is then exchanged for a .018-inch angled Glidewire (Terumo Medical Corporation, Somerset, NJ), and the lesion is crossed in a retrograde fashion. This wire is advanced to the popliteal artery where it can be snared and exteriorized at the femoral artery.[4]

Adequate anticoagulation in below-the-knee intervention is crucial to minimize thromboembolic complications. An activated clotting time of 250 to 300 seconds should be maintained throughout the procedure. Some interventionists have advocated the use of bivalirudin. Allie et al studied the combination of bivalirudin and tirofiban as an anticoagulation method in peripheral intervention. This combination was a safe alternative to unfractionated heparin.[6] Furthermore, infrapopliteal arteries are sensitive to manipulation, and intra-arterial use of vasodilators is recommended during the procedure.[2] "No-reflow" is known to occur and can respond to nitroprusside, adenosine, and verapamil.

TABLE 1. TECHNOLOGY AVAILABLE FOR TPVI
• Balloon angioplasty
• CryoPlasty
• Stenting
• Laser
• Excisional atherectomy
• Cutting balloon
• Rotational atherectomy
• Drug-eluting stents

TABLE 2. VASCULAR ACCESS FOR TPVI
• Contralateral
• Ipsilateral antegrade
• Retrograde tibial
• Retrograde tibial/pedal

WIRES AND TECHNIQUES TO CROSS THE LESION

The leading cause of a technical failure is the inability to cross the lesion with the guidewire. Generally, in the tibial arteries, the .014-inch or .018-inch wire systems are utilized. Although a standard wire may be useful for stenotic lesions, occlusions—particularly long occlusions—require a more aggressive approach. If initially unsuccessful with the standard wire, additional wire support can be provided by either a balloon or a catheter (eg, Quickcross, Spectranetics Corporation, Colorado Springs, CO). In a stepwise approach, hydrophilic wires of advancing stiffness are used. Hydrophilic wires have higher risk for perforation and should be used with caution and, once through the occlusion, exchange for a standard wire may be helpful. Ideally, an intraluminal approach is maintained; however, for longer occlusions, subintimal recanalization could be attempted.[2]

Balloon Angioplasty

Primary balloon angioplasty has been used as the main revascularization modality in CLI.[7] Generally, long balloons in 2.5-mm to 4-mm diameters are used, with prolonged (5-minute) inflations. Suitable lesions include short and discrete lesions, diffuse stenotic lesions, and occlusion <10 cm in tibioperoneal vessels. In a study by Dorros et al, this method was associated with 94% revascularization success, and limb salvage was achieved in 91% of subjects during 5-year follow-up. Compared to a surgical approach, this technique resulted in better outflow revascularization with a significant improvement in distal extremity perfusion, immediate relief of rest pain, and augmentation of ulcer healing. The use of the cutting balloon in the infrapopliteal arteries was associated with a 20% rate of intimal dissection and inadequate hemodynamic result, necessitating use of adjunctive stenting.[8] Cutting balloons, however, may have niche uses, such as treating ostial lesions or lesions from neointimal hyperplasia.

CryoPlasty

The PolarCath System (Boston Scientific Corporation, Natick, MA) consists of an inflation unit, catheter, nitrous oxide cartridge, and a power module. The PolarCath System is indicated to dilate stenosis in the peripheral vasculature (the iliac, femoral, popliteal, infrapopliteal, renal, and subclavian arteries) as well as to treat obstructive lesions of polytetrafluoroethylene (PTFE) access grafts or native arteriovenous dialysis fistulae. In addition, it is indicated for postdeployed stent expansion of self-expanding peripheral vascular stents.

As the CryoPlasty balloon is placed in position, the CryoPlasty inflation unit is activated by a touch of a button after the nitrous oxide is placed in the system. Nitrous oxide is released and delivered to the balloon and turns into a gas, generating the pressure that is required for luminal expansion. Using nitrous oxide, the balloon is cooled to -10ºC. The PolarCath inflation is achieved in an automated fashion at 2-atm increments to 8 atm. The .035-inch catheters are available in 80-cm to 120-cm lengths, while the balloon diameters range from 4 mm through 8 mm, and balloon lengths range from 20 mm to 100 mm. The .014-inch catheters are available in 20-mm to 100-mm balloon lengths and 135-cm and 150-cm working lengths. The balloon diameters include 2 mm, 2.5 mm, 3 mm, 3.5 mm, 4 mm, 5 mm, and 6 mm.

Stenting

Data regarding the use of stents in the infrapopliteal arteries have been limited. The most common reasons to use stents in this area are flow-limiting dissection[9] and vascular recoil. Small-diameter (4-mm) self-expanding stents are available (eg, Xpert, Abbott Vascular, Santa Clara, CA). The use of drug-eluting stents (sirolimus-eluting stents) in the infrapopliteal artery has been compared to bare-metal stent placement in a prospective study of 58 patients. The use of sirolimus-eluting stents was associated with improved 6-month patency rates (92% vs 68%, respectively, *P*<.002). This was associated with statistically significant reduction of target vessel revascularization at 6 months.[10] Future technology may address the problem of in-stent restenosis in the lower extremity by using bioabsorbable stents with drug-eluting capability.[11]

Laser

The excimer laser light (at 308 nm) removes plaque by photoacoustic ablation. The excimer laser delivers, via a flexible fiberoptic catheter, ultraviolet energy in short pulses. This technology allows for plaque ablation and reduces the potential for embolic complications. Recently, the results of the LACI trial were published, which showed 86% procedural success with the use of an excimer laser (Spectranetics Corporation) in subjects with high risk for surgical intervention due to the presence of prohibitive comorbidities or inadequate target vessels or saphenous veins.[12] This study

CHALLENGING BELOW-THE-KNEE CASE

Presentation

A 68-year-old diabetic man with a history of left femoral to anterior tibial bypass in May 2006 presented in September 2006 with a painful, cold left foot with an ischemic toe ulcer. The patient was referred for angiography, which revealed occlusion of the bypass graft with severe popliteal and tibial artery disease. Figure A shows severe, eccentric popliteal artery stenosis (arrow), and Figure B shows severe stenosis of the proximal anterior tibial artery and total occlusion of the midanterior tibial artery (arrows). The peroneal and posterior tibial arteries were occluded.

CryoPlasty Procedure

Based on the promising data from the CryoPlasty Below the Knee Study (BTK CHILL), CryoPlasty using the PolarCath device (Boston Scientific Corporation) was decided upon as the treatment strategy. A 4-mm X 6-cm PolarCath balloon was inflated in the popliteal artery (Figure C). A 3-mm X 4-cm PolarCath balloon was then used in the proximal anterior tibial artery (Figure D), and finally, a 2.5-mm X 6-cm PolarCath balloon was inflated in the midanterior tibial artery (Figure E).

Results

The CryoPlasty procedures were technically successful, with no complications. Figure F shows the final result after treatment of the popliteal and proximal anterior tibial artery stenoses. Figure G shows the successfully treated midanterior tibial artery occlusion. Straight-line flow to the foot was reestablished. ■

demonstrated a 6-month limb salvage rate of 93% of the limbs intervened upon. The advantage of the laser is that it may facilitate recanalization of difficult-to-cross chronic occlusions. Furthermore, in complete occlusions, one can use a "step-wise" approach of lasing, and then probing with the wire for the true lumen, until successful recanalization. Adjunctive balloon angioplasty is commonly required after lasing due to the catheter size.[13]

Excisional Atherectomy

The SilverHawk (FoxHollow Technologies, Redwood City, CA) is an excisional atherectomy device that has been used for TPVI. In a study by Kandzari et al, use of the device in the setting of CLI resulted in a 99% procedural success and prevented an amputation in 82% of patients at 6 months.[14] In this study, 40% of the lesions were in the tibioperoneal vessels or dorsalis pedis artery. Adjunctive therapy was needed in less than one-fifth of the patients. Compared to balloon angioplasty, excisional atherectomy theoretically results in a higher luminal gain, without plaque displacement and vessel injury. It is promoted that excisional atherectomy can be used as a stand-alone therapy. Restenosis after atherectomy using the FoxHollow device is approximately 22% at 6-month follow-up, with cumulative event-free survival at 76.9% across several below-the-knee vessel beds,[15] although there have been no large prospective trials with routine angiographic follow-up. Distal embolization has been reported with the device.

COMPLICATIONS

In the study by Soder et al,[16] the mortality rate after percutaneous transluminal angioplasty was 1.7%, which compares favorably to a perioperative mortality rate of 1.8% to 6% for distal bypass surgery.[2] The 30-day mortality rate was 2.9% after endovascular therapy and 5.6% after surgery in the BASIL trial.[1] The arterial perforation rate can occur up to 3.7% and is more common in diabetic and elderly subjects. This complication can be addressed by balloon tamponade with or without reversal of the anticoagulation.

Other major complications occur at 2% to 6% and include access-site hematoma, acute arterial occlusion from embolization or "no-reflow," and amputation. Iatrogenic arterial occlusion can be secondary to spasm, dissection, or distal embolization. Antispasmodic agents, stents, and thrombolysis with or without aspiration thrombectomy could be used to help resolve the condition.[2]

CONCLUSION

The endovascular approach to infrapopliteal arterial disease can be a safe and effective method to address this problem. The interventions should be thought out in a deliberate manner, and the lesions should be approached systematically, in a stepwise fashion. Several devices are capable of restoring patency of the tibial vessels. Ultimately, the acute procedural success usually drives the clinical result of ulcer healing. Studies have shown decreased incidence of dissection[17,18] and recoil[19,20] with CryoPlasty using the PolarCath System. As illustrated in the below-the-knee case study on the previous page, our experience has shown that CryoPlasty is a safe and effective treatment option. ■

Ali Morshedi-Meibodi, MD, is a vascular medicine, endovascular, and cardiology fellow at Loyola University in Maywood, Illinois. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Morshedi-Meibodi may be reached at (708) 216-4466.

John R. Laird, Jr, MD, is Director of the Clinical Vascular Program at the University of California Davis Medical Center in Sacramento, California. He has disclosed that he is a Scientific Advisory Board Member for and receives honorarium from Boston Scientific. Dr. Laird may be reached at (916) 734-1546; john.laird@ucdmc.ucdavis.edu.

Robert S. Dieter, MD, RVT, is Assistant Professor of Vascular, Endovascular, and Interventional Cardiology at Loyola University in Maywood, Illinois. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Dieter may be reached at (708) 216-4466; rdieter@lumc.edu.

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CryoPlasty for Treatment of Popliteal and Tibioperoneal Disease

Preliminary results suggest that CryoPlasty with the PolarCath System is a promising new treatment for patients with complex tibioperoneal arterial disease.

BY ZVONIMIR KRAJCER, MD

Because surgical treatment is frequently associated with severe comorbid conditions, a high incidence of complications, and disappointing long-term results, many interventionists have determined incapacitating claudication, rest pain, nonhealing ulcers, and gangrene as standard indications for tibioperoneal revascularization.[1,2]

Recent reports have indicated that there has been a shift in treating infrapopliteal disease, from surgical revascularization and amputation to an endovascular approach.[3] Some investigators have achieved primary technical success rates of 85% for PTA in the infrapopliteal arteries.[3-5] Mean patency rates ranging from 9 months to 1 year are reported to be approximately 70%, but vary significantly. These reports identify the causes of restenosis as dissection, vascular recoil, and fibrointimal hyperplasia, among others.[3-5] It is generally agreed that better long-term results are needed and that currently available technology has not achieved the desired outcome; bare-metal and drug-eluting stents have not demonstrated significant reduction of restenosis,[6] whereas other technologies initially showed promise but failed to live up to expectations (ie, atherectomy, excimer laser ablation, and brachytherapy).[7,8] It was these limitations in treatment options that brought about the development of CryoPlasty as a treatment option.[9-11]

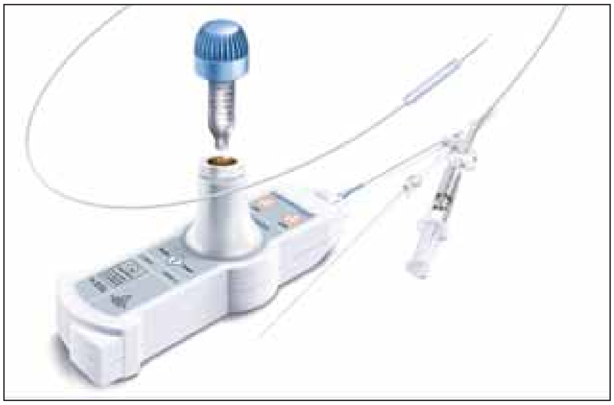

Figure 1. The PolarCath System consists of an inflation unit, a nitrous oxide cylinder, a reusable battery pack, and a PolarCath disposable catheter.

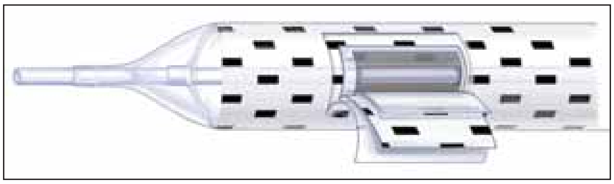

Figure 2. The PolarCath balloon consists of double-layer balloons with radiopaque markers.

POLARCATH SYSTEM

The PolarCath Peripheral Dilatation System (Boston Scientific Corporation, Natick, MA) consists of three sterile one-time use components: catheter, inflation unit, nitrous oxide cylinder, as well as one nonsterile reusable battery pack (Figure 1). The PolarCath is compatible with a .035-inch guidewire and also with a .014-inch guidewire. The .035-inch catheters are available in 80-cm and 120-cm lengths. The balloon diameters range from 4 mm through 8 mm, and balloon lengths range from 20 mm to 100 mm. The .014-inch catheters are available in 20-mm to 100-mm balloon lengths and 135-cm and 150-cm working lengths. The balloon diameters are as follows: 2 mm, 2.5 mm, 3 mm, 3.5 mm, 4 mm, 5 mm, and 6 mm.

The PolarCath System is indicated for use in the peripheral vasculature (iliac, femoral, popliteal, infrapopliteal, and renal arteries). This device is also approved for treating stenotic lesions of polytetrafluoroethylene access grafts and arteriovenous dialysis shunts. The PolarCath System is a unique device in which nitrous oxide is delivered as a pressurized liquid into the angioplasty balloon (Figure 2). During this process, nitrous oxide changes into gas, which causes

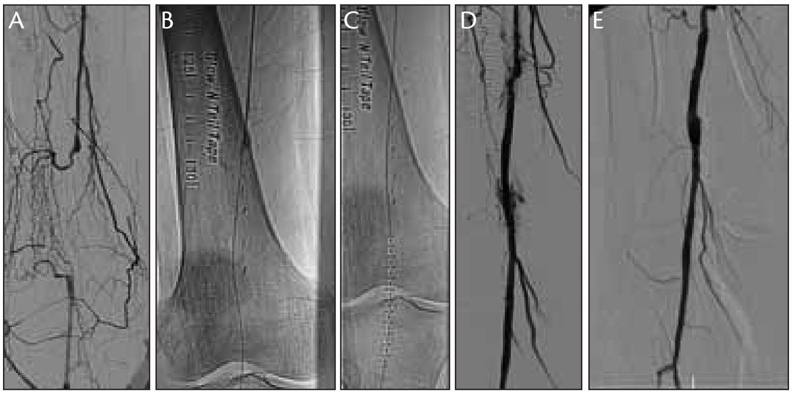

Figure 3. Angiographic image depicting the 10-cm–long occlusion of the right popliteal artery (A). An angiogram reveals successful recanalization of the right popliteal artery with an .018-inch hydrophilic wire (B). An angiogram depicts the PolarCath, which measures 5 mm in diameter and 60 mm in length, during CryoPlasty of the right popliteal artery (C). The final angiogram reveals a patent right popliteal artery after successful CryoPlasty (D). At 6-month follow-up, a femoral arteriogram reveals a patent right popliteal artery, without evidence of restenosis (E).

expansion of the balloon. At the same time, the process delivers cold thermal energy to the artery, which causes crystallization of fluid in the interstitium. This process enables more uniform expansion of the lesion than the standard balloon angioplasty, which in effect reduces the incidence of intimal dissection.[9] It has been shown in previous studies that rapid freezing of smooth muscle cells leads to programmed cell death, or apoptosis.[10] Fava and Tatsutani, in their initial studies, have shown that there was a 50% reduction in smooth muscle cells when exposed to the temperatures generated during CryoPlasty (-10°C).[9,10] The reduction in the number of smooth muscle cells and the lack of inflammation after the intervention may lead to a lower incidence of restenosis. The impact of cold energy also disrupts the elastin fibers while retaining the collagen fiber architecture, which may produce less recoil of the vessel after angioplasty while maintaining the integrity of the vessel wall.[9,10]

CASE STUDIES

Case 1

A 67-year-old woman presented with pain at rest in her right lower leg of 1 week's duration. Ten years previously, she had undergone right femoropopliteal bypass. Her physical examination revealed normal right femoral artery pulse, and absent right popliteal, dorsalis pedis, and posterior tibial pulses. Her right leg ankle-brachial index was 0.42/0.41, and the angiogram revealed an occluded right popliteal artery and the right femoropopliteal bypass (Figure 3A). She underwent endoluminal treatment of the occluded right popliteal artery via contralateral percutaneous approach. The popliteal artery occlusion was successfully crossed with a .014-inch hydrophilic wire (Figure 3B). She then underwent successful CryoPlasty with a 5-mm X 60-mm PolarCath catheter, with restoration of blood flow and limb salvage (Figure 3C, D). At 6-month follow-up, she was free of intermittent claudication and her ankle-brachial indices improved to 0.78/0.81. Femoral arteriography at 6-month follow-up revealed a patent right popliteal artery, without evidence of restenosis (Figure 3E).

Case 2

An 83-year-old woman presented with critical limb ischemia and rest pain of 1-month duration in her right lower leg. She had multiple comorbid conditions, including severe chronic obstructive pulmonary disease and insulin-dependent diabetes mellitus. Her physical exam revealed absent right popliteal and tibioperoneal artery pulses. Her right leg ankle-brachial index was 0.46. Her right femoral artery angiogram revealed severe stenosis of her SFA and occlusion of her distal popliteal artery (Figure 4A, B).

She underwent endoluminal treatment of her SFA and of the occluded right popliteal artery via contralateral percutaneous approach. The popliteal artery occlusion was successfully crossed with a .014-inch hydrophilic wire. She underwent successful CryoPlasty of her SFA and popliteal stenosis with a 5-mm X 60-mm PolarCath catheter (Figure 4C, D). The distal popliteal artery and the tibioperoneal trunk were successfully treated with a 3-mm X 20-mm PolarCath catheter, with restoration of blood flow and limb salvage (Figure 4E, F). At 6-month follow-up, the patient was free of intermittent claudication, and her ankle-brachial indices improved to 0.76.

RESULTS

A retrospective analysis of data from 28 patients treated at our institution, who underwent infrapopliteal CryoPlasty for treatment of limb-threatening ischemia, showed a high degree of technical success (100%). The incidence of dissection was low, occurring in one patient (3.6%). The need for bailout stenting was also low (3.6%). Clinical patency at 3 and 6 months was 92.9% and 90%, respectively. Limb salvage at 6-month follow-up was accomplished in 27 patients (96.4%).

INDICATIONS FOR CRYOPLASTY TREATMENT OF POPLITEAL AND TIBIOPERONEAL DISEASE

In our practice, based on our preliminary encouraging results, CryoPlasty is one of the most commonly used initial

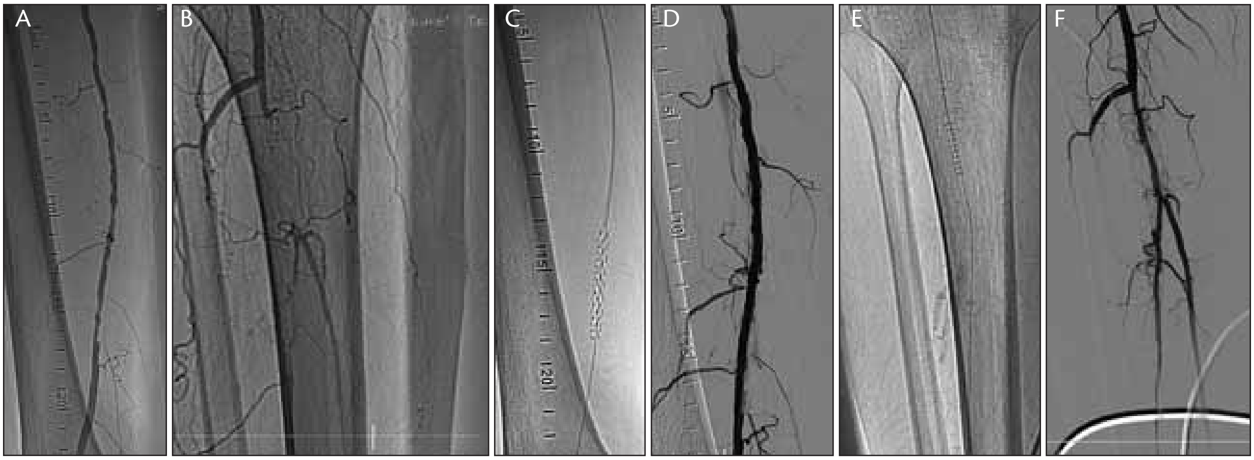

Figure 4. Angiographic image of a patient with critical right leg ischemia revealing multiple right superficial femoral (SFA) and popliteal artery lesions (A). Angiographic image revealing occluded distal popliteal and tibioperoneal arteries (B). CryoPlasty of the patient's right SFA and popliteal artery with 5-mm X 60-mm PolarCath balloon (C). Final result of the right SFA and popliteal artery CryoPlasty with 5-mm X 60-mm PolarCath balloon revealing satisfactory result (D). CryoPlasty of the right popliteal and the right tibioperoneal trunk arteries with 3-mm X 20-mm PolarCath balloon (E). Final result of the right popliteal and tibioperoneal trunk arteries (F).

treatment modalities for severely symptomatic patients with popliteal and tibioperoneal disease. CryoPlasty has also been effective in patients with severe tibioperoneal disease, when other technologies have failed. One of the contraindications of CryoPlasty is in severe, circumferential vessel calcification and severe fibrosis. In this scenario, we have been successful in first using the Cutting Balloon (Boston Scientific Corporation), followed by CryoPlasty. Excimer laser angioplasty has been shown to be an effective debulking tool in severely fibrotic and calcified vessels. We have also combined excimer laser with CryoPlasty when recanalizing and debulking long femoral, popliteal, and tibioperoneal occlusions. In our experience, this approach has offered us a lower incidence of dissection and restenosis.

CryoPlasty has been effective in preventing dissections and avoiding the need for stenting after suboptimal results of lesion debulking with excimer laser angioplasty and atherectomy devices.

CONCLUSIONS

Our preliminary experience with CryoPlasty for treating complex popliteal and tibioperoneal arterial disease reveals excellent procedural success. The incidence of dissection and the need for stenting was very low. This preliminary study in a limited number of patients also indicates that CryoPlasty offers encouraging midterm results when other technologies have failed.

The initial data from the US registries have shown promising results of treating femoropopliteal and tibioperoneal disease with CryoPlasty.[11] However, longer follow-up will be necessary to determine the long-term benefits of CryoPlasty for treating popliteal and tibioperoneal disease.

FUTURE IMPLICATIONS

We hope that our encouraging preliminary results, as well as the BTK CHILL registry data, will open the avenue for further studies in patients with severe infrapopliteal arterial disease. If long-term results are promising, there may be a future shift in the paradigm of treating patients with infrapopliteal disease before the development of severe ischemic changes. ■

Zvonimir Krajcer, MD, is a Co-Director of Peripheral Vascular Intervention at St. Luke's Episcopal Hospital and Texas Heart Institute; and Clinical Professor of Medicine at Baylor College of Medicine and at the University of Texas Health Science Center in Houston, Texas. Dr. Krajcer is in a speakers bureau for Boston Scientific and receives educational grants from Boston Scientific. Dr. Krajcer may be reached at (713) 790-9401; zvonkomd@aol.com.

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