CASE REPORT

Percutaneous Balloon Cryoplasty: A New Therapy for Rapidly Recurrent Anastomotic Venous Stenoses of Hemodialysis Grafts?

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• **Vascular access dysfunction is a major source of morbidity for end-stage renal disease patients on hemodialysis. The arteriovenous graft is a common access type for many of these patients. Frequent stenosis formation and thrombosis complicate this form of access. Patients may have a rapidly forming and recurrent venous stenosis at the graft–vein anastomosis that has been seen in both animal models and end-stage renal disease patients to be the result of neointimal hyperplasia. This venous lesion is particularly resistant and sometimes intractable to conventional angioplasty. As a result, new therapies have been developed to reduce the formation and/or recurrence of neointimal hyperplasia. These include special cutting balloons, drug-eluting stents, and endovascular brachytherapy. The authors present the cases of 5 patients with rapidly recurrent venous lesions at the graft–vein anastomosis that derived benefit from angioplasty with the cryoballoon. The time to stenosis or thrombosis in the arteriovenous grafts was increased from a mean of 3 weeks to more than 16 weeks with this technology. Cryotherapy with the cryoballoon (cryoplasty) may represent a useful therapy for patients with intractable stenoses at or near the venous anastomosis of arteriovenous grafts. *Am J Kidney Dis* xx:xxx.**

INDEX WORDS: Vascular access; arteriovenous graft; angioplasty; cryotherapy; thrombosis; stenosis.

THE CURRENT KIDNEY Disease Outcomes Quality Initiative guidelines encourage the creation of an arteriovenous fistula as first choice over either an arteriovenous graft (AVG) or dual lumen catheter for long-term hemodialysis therapy.[1] Unfortunately, although more arteriovenous fistulas are being placed, AVGs remain a common form of vascular access in end-stage renal disease (ESRD) patients. Arteriovenous grafts are particularly prone to the formation of stenosis at the graft–vein anastomosis and ultimately thrombosis with loss of vascular access sites.[2-5] The underlying histology of AVG anastomotic stenosis is neointimal hyperplasia, a lesion that can be difficult to disrupt and often impossible to eradicate.[5,6] To avoid thrombosis, regular vascular access surveillance to identify stenotic lesions and therapies that can adequately treat AVG stenosis formation are necessary.[7-13]

Surgical revision with a patch angioplasty or bypass graft is one solution, but this approach causes vein loss.[14] The current percutaneous intervention used is balloon angioplasty with or without intravascular stent placement.[15] Balloon angioplasty has improved the treatment of AVG venous stenosis, but lesions often recur because the therapy does not prevent neointimal growth and subsequent restenosis.[6,15] Some patients form intractable stenoses that resist initial angioplasty and/or recur rapidly.[6] Promising new therapies to maintain AVG patency and reduce such rapid restenosis include endovascular brachytherapy, angioplasty with cutting and high-pressure balloons, and angioplasty with placement of drug-eluting stents.[6,15,16] These therapies currently are under evaluation. A potentially new technology to treat the rapidly recurrent venous stenosis associated with AVGs is cryotherapy with a "cryoballoon." We describe a series of 5 cases in which cryoplasty improved the time to restenosis compared with conventional balloon angioplasty in patients with rapidly recurrent anastomotic stenosis in their AVG.

PATIENTS AND METHODS

Five patients were identified as having rapidly recurrent venous stenosis at the AVG anastomosis. This was defined as (1) greater than 50% venous stenosis on fistulogram, (2)

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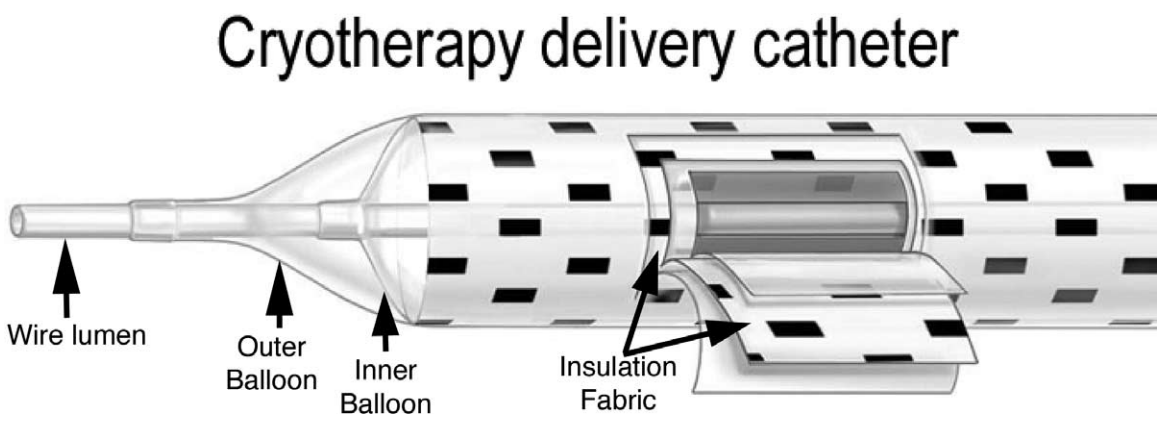

Fig 1. The cryoplasty balloon is composed of components that include a wire lumen, inner balloon, insulation fabric, and outer balloon. Nitrous oxide travels from a high-pressure storage cylinder through the lumen of the catheter into the inner balloon. As it enters the balloon, the liquid changes into gas, expanding the balloon and reducing the temperature. Internal balloon pressure is highly regulated throughout the cycle. At completion of the cycle, gas is evacuated and the balloon deflates.

rapid development of stenosis (in less than 4 weeks after conventional balloon angioplasty), and (3) recurrence at least 3 separate times (documented on fistulogram). Recurrent venous stenosis was identified by vascular access surveillance or an episode of AVG thrombosis. Surveillance criteria utilized to identify clinically significant venous stenosis were (1) access blood flow (ABF) less than 600 mL/min (or 25% reduction) on ultrasound dilution and/or (2) dynamic venous pressure (Vp) greater than 120 mm Hg on at least 50% of measurements using criteria developed by Cayco et al.[8] The AVGs were at least 3 months old.

To assure hemodynamic significance of the stenotic lesion, conventional balloon angioplasty (inflate time of 20 seconds using 8 atmospheres of pressure) was performed based on the following criteria; the diameter of the venous stenosis had to be greater than 50%, and a pressure ratio across the stenosis greater than 0.5 was required. The pressure ratio was calculated by dividing the systolic pressure measured at the apex (midpoint) of the graft by the cuff systolic pressure measured on the opposite arm. Balloon angioplasty was considered successful at the time of intervention based on residual stenosis less than 30%[17] and normalization of the pressure ratio within the graft (<0.4). Angioplasty with the cryoballoon (PolarCath; CryoVascular Systems, Inc, Los Gatos, CA) was pursued when the patient did not respond to conventional balloon angioplasty and met the above-noted criteria for rapidly recurrent venous stenosis. Details of the cryoballoon catheter are noted in Fig 1. It is available in several balloon diameters (4 mm to 8 mm) and lengths (20 mm, 40 mm, 60 mm) and is expanded with nitrous oxide. The catheter is tracked to the stenotic lesion over a 0.035 guidewire using fluoroscopy. Angioplasty with the cryoballoon was preformed using a 6- or 8-mm balloon (determined by the vein size) with an inflate time of 20 seconds using 8 atmospheres of pressure, reaching an estimated temperature of −10°C. As discussed later, the angioplasty details and balloon temperature target for cryoplasty are based on data generated in animal studies. In particular, −10°C is used because that temperature is optimal to induce apoptosis, whereas colder temperatures promote cell necrosis.

RESULTS

Five patients met the criteria of rapidly recurrent venous stenosis in the AVG. All patients had been treated with aggrenox (aspirin 50 mg/dipyridimole 400 mg) daily at the time of initial AVG creation. None were on warfarin. Table 1 summarizes the patient characteristics. Two patients had right forearm loop AVGs, whereas 3 patients had upper arm AVGs (left, 1; right, 2). All patients had greater than 70% stenosis of the venous lesion at the graft–vein anastomosis on fistulogram (range, 70% to 80%). Four patients had 3 episodes of rapid stenosis recurrence, whereas 1 patient (patient 5) had 4 episodes of recurrent lesion formation. At least 1 episode of AVG thrombosis complicated the course of all patients despite successful conventional balloon angioplasty 2 to 4 weeks before the thrombotic event. These 5 patients were the first to undergo cryoplasty for an AV graft stenosis at Yale–New Haven Hospital.

After angioplasty with the cryoballoon, all patients had no restenosis at the venous anastomosis for at least 3 months with a mean of 16.8 weeks (Table 2). This was based on absence of positive surveillance criteria and lack of thrombosis. One patient met surveillance criteria at 13 weeks and was found to have a 70% stenosis at the venous anastomosis. This was successfully re-angioplastied with the cryoballoon. The other

Table 1. Patient Characteristics

Patient	Age, y	Race	Sex	Years on Dialysis	Access Type	Comorbidities
1	73	W	F	6	Right forearm AVG	CAD, HTN, PVD, Hyperlipidemia
2	56	AA	M	4	Right upper arm AVG	HIV+, HTN, PAF, Amyloidosis
3	74	AA	M	5	Left upper arm AVG	CAD, HTN, COPD, Hyperlipidemia, LVH
4	75	W	M	7	Right forearm AVG	CAD, HTN, CMP, Hyperlipidemia
5	50	AA	M	8	Right upper arm AVG	CAD, HTN, LVH, Hyperlipidemia

Abbreviations: W, white; AA, African American; AVG, arteriovenous graft; CAD, coronary artery disease; PAF, paroxysmal atrial fibrillation; PVD, peripheral vascular disease; HTN, hypertension; LVH, left ventricular hypertrophy; COPD, chronic obstructive pulmonary disease.

4 patients underwent fistulogram at times ranging from 16 weeks to 21 weeks (Table 2). Three patients were identified by surveillance criteria, whereas 1 suffered a thrombotic episode. Two patients had recurrent lesions at the venous anastomosis, whereas 2 patients had new venous stenosis several centimeters from the previous stenosis treated with cryoplasty. No patient suffered from access infection or other complication. No stents were deployed in any patients.

DISCUSSION

Over the last 10 years, conventional balloon angioplasty of stenoses has helped to preserve the vascular access of ESRD patients on hemodialysis.[6,7,15] However, despite this, many patients manifest rapidly recurrent lesions from neointimal hyperplasia at the graft–vein anastomosis that are resistant to conventional balloon angioplasty.[5,6] As a result, these patients do not benefit from a durable response and suffer from AVG thrombosis and eventual access loss. New technologies have targeted this resistant venous lesion with the hope of reducing recurrence of neointimal hyperplasia. External beam radiation therapy reduces neointimal hyperplasia in a pig model of AVG stenosis that closely mimics this problem in ESRD patients.[18] After promising preliminary use of endovascular brachytherapy in the treatment of AVG venous stenosis, an intervention trial (BRAVO II) is currently underway to assess the effect of this therapy in ESRD patients.[16,19] Another approach to reducing neointimal hyperplasia is cryotherapy.

Cryotherapy is an important, minimally invasive surgical technique. Cryotherapy or cryoablation is used to minimize restenosis by triggering apoptosis in treated tissues.[20,21] Cryotherapy with liquid oxygen, nitrogen, and air for the treatment of dermatologic and gynecologic disorders dates back to the mid-19th century.[21] Modern cryosurgery began through the collaborative work of physician Irving Cooper and engineer Arnold Lee. They built a cryogenic probe capable of delivering liquid nitrogen to cryogenic lesions with exacting control over the site of cold energy delivery.[21] Cryotherapy expanded to the treatment of cancers of the liver, prostate, and bladder. Unfortunately, the amount of tissue affected by the probe is much greater than that of direct contact, because the freezing domain propagates outward.[22] This made controlling the affected area imprecise. Lasers replaced cryosurgery in the 1980s, and cryotherapy once again was relegated primarily to dermatologic and gynecologic disorders. More recently, interest has resurfaced for the use of cryotherapy in the prevention of vascular restenosis.

To design cryotherapy that will effectively prevent restenosis, it is essential to understand the specific relationship between thermal conditions and the mechanisms of vascular freezing injury.[23] Cooper in 1964 determined that tissue exposed to $-20^{\circ}C$ for 1 minute was sufficient to induce necrosis.[21] The mechanism of cryotherapy-associated tissue damage is complex and highly variable depending on the specific tissue targeted. Cellular survival during cryoablation

Table 2. Access Data in Patients

Pt	Access Dysfunction	Intervention	Outcome
1	(a) ABF = 410, Vp > 120 (initial stenosis identification)	(a) Angioplasty* of VA stenosis	(a) Successful angioplasty† ABF = 810, Vp < 120
	(b) ABF = 400, Vp > 120 (3 wk after angioplasty)	(b) Angioplasty* of VA stenosis	(b) Successful angioplasty† ABF = 780, Vp < 120
	(c) AVG thrombosis (4 wk after thrombolysis)	(c) Thrombolysis with angioplasty	(c) Successful angioplasty† no ABF, Vp < 120
	(d) AVG thrombosis (2 wk after thrombolysis)	(d) Thrombolysis with cryoplasty of VA	(d) Successful angioplasty† ABF = 765, Vp < 120
	(e) ABF = 445, Vp > 120 (13 wk after cryoplasty)	(e) Cryoplasty* of VA stenosis	(e) Successful angioplasty† ABF = 745, Vp < 120
2	(a) ABF = 540, Vp > 120 (initial stenosis identification)	(a) Angioplasty* of VA stenosis	(a) Successful angioplasty† ABF = 965, Vp < 120
	(b) AVG thrombosis (3 wk after angioplasty)	(b) Thrombolysis with angioplasty of VA	(b) Successful angioplasty† ABF = 875, Vp < 120
	(c) ABF = 515, Vp > 120 (4 wk after thrombolysis)	(c) Angioplasty* of VA stenosis	(c) Successful angioplasty† no ABF, Vp < 120
	(d) ABF = 520, Vp > 120 (2 wk after angioplasty)	(d) Cryoplasty* of VA stenosis	(d) Successful angioplasty† ABF = 890, Vp < 120
	(e) ABF = 495, Vp > 120 (16 wk after cryoplasty)	(e) Cryoplasty* of *new* venous stenosis	(e) Successful angioplasty† ABF = 845, Vp < 120
3	(a) ABF = 490, Vp > 120 (initial stenosis identification)	(a) Angioplasty* of VA stenosis	(a) Successful angioplasty† ABF = 885, Vp < 120
	(b) ABF = 510, Vp > 120 (2 wk after angioplasty)	(b) Angioplasty* of VA stenosis	(b) Successful angioplasty† no ABF, Vp < 120
	(c) AVG thrombosis (4 wk after angioplasty)	(c) Thrombolysis with angioplasty of VA	(c) Successful angioplasty† ABF = 835, Vp < 120
	(d) ABF = 465, Vp > 120 (4 wk after thrombolysis)	(d) Cryoplasty* of VA stenosis	(d) Successful angioplasty† ABF = 890, Vp < 120
	(e) ABF = 500, Vp > 120 (18 wk after cryoplasty)	(e) Cryoplasty* of *new* venous stenosis	(e) Successful angioplasty† ABF = 855, Vp < 120
4	(a) ABF = 350, Vp > 120 (initial stenosis identification)	(a) Angioplasty* of VA stenosis	(a) Successful angioplasty† ABF = 720, Vp < 120
	(b) ABF = 385, Vp > 120 (2 wk after angioplasty)	(b) Angioplasty* of VA stenosis	(b) Successful angioplasty† no ABF, Vp < 120
	(c) ABF = 325, Vp > 120 (3 wk after angioplasty)	(c) Angioplasty* of VA stenosis	(c) Successful angioplasty† ABF = 750, Vp < 120
	(d) AVG thrombosis (2 wk after angioplasty)	(d) Thrombolysis with cryoplasty of VA	(d) Successful angioplasty† no ABF, Vp < 120
	(e) ABF = 345, Vp > 120 (16 wk after cryoplasty)	(e) Surgical patch angioplasty of VA stenosis	(e) Successful surgical patch angioplasty, ABF = 725, Vp < 120
5	(a) ABF = 570, Vp > 120 (initial stenosis identification)	(a) Angioplasty* of VA stenosis	(a) Successful angioplasty† ABF = 1,050, Vp < 120
	(b) AVG thrombosis (4 wk after angioplasty)	(b) Thrombolysis with angioplasty of VA	(b) Successful angioplasty† no ABF, Vp < 120
	(c) ABF = 595 Vp > 120 (2 wk after thrombolysis)	(c) Angioplasty* of VA stenosis	(c) Successful angioplasty† ABF = 975, Vp < 120
	(d) ABF = 620, Vp > 120 (4 wk after angioplasty)	(d) Angioplasty* of VA stenosis	(d) Successful angioplasty† ABF = 950, Vp < 120
	(e) ABF = 495, Vp > 120 (3 wk after angioplasty)	(e) Cryoplasty* of VA stenosis	(e) Successful angioplasty† ABF = 985, Vp < 120
	(f) ABF = 550, Vp > 120 (21 wk after cryoplasty)	(f) Cryoplasty* of VA stenosis	(f) Successful angioplasty† ABF = 955, Vp < 120

Abbreviations: Pt, patient; VA, venous anastomosis; ABF, access blood flow (mL/min); Vp, dynamic venous pressure (mm Hg).

*Angioplasty performed for venous lesions associated with greater than 50% stenosis and a pressure ratio across the stenosis > 0.5.

†Successful angioplasty ≤ 30% stenosis, pressure ratio across the stenosis < 0.4, ABF > 600 mL/min, and/or Vp < 120 mm Hg postprocedure.

depends on the following factors: (1) the freezing and thawing rates of cells, (2) the lowest temperature reached, and (3) the duration of time the tissue is exposed to subzero temperatures.[20]

Most processes within the cell are temperature dependent. Lowering the microenvironment temperature reduces the ability of membrane proteins to control intracellular ion content.[21] It also inhibits the ability of cytoskeleton proteins to maintain chemical bonds. Cellular damage also results from ice formation. Ice is unable to contain any solutes, and when an aqueous solution freezes, the solutes accumulate in front of the change of phase interface.[22] Through this effect, cells are exposed to hypertonic solutions between ice crystals, causing the cells to crenate from osmotic dehydration, damaging membrane and cellular machinery.[20,22] As cells warm and ice melts, the cells are briefly exposed to hypotonic solutions, causing them to swell and rupture. Cellular damage is cumulative over time and most evident upon rewarming, leading to necrosis or apoptosis.[21] Most cells can tolerate cooling above freezing for a brief time, as occurs with most procedures. Some cells, however, are highly resistant to freezing. For example, cells in the renal collecting system are not damaged by cryosurgery even when exposed to very low temperatures.[21] Platelets, in contrast, are particularly sensitive to cooling below their lipid phase transition temperature. Cooling triggers an influx of calcium, which then activates platelet aggregation and obstruction of blood vessels in the region around frozen lesions.[20] This may lead to ischemic damage of adjacent tissues. Other tissues, including arteries, are very dependent on their ionic content for function and may experience tissue damage beyond the frozen lesion.[21]

Cryotherapy causes selective damage to cellular components while preserving matrix structure.[22] This effect results in less fibrosis in tissues such as liver and skin. Cryotherapy triggers apoptosis at temperatures between −15°C and 0°C but induces necrosis at colder temperatures.[21] Apoptotic bodies sequester cellular components and prevent inflammation. Because inflammation is a trigger for neointimal hyperplasia and luminal stenosis, induction of apoptosis with cryotherapy could be a beneficial therapy for AVG venous stenosis. In vitro studies have examined the effect of cryoablation on the 2 main components of vascular tissue, endothelial and smooth muscle cells.[24] Favorable results for the prevention of smooth muscle hyperplasia were described.

The effects of cryotherapy on vessel wall repair after angioplasty in vivo are still unknown. Studies in rabbit iliac arteries evaluated the early (72 hours) and late (10 weeks) effects of 3 consecutive applications of angioplasty with cryotherapy compared with angioplasty alone.[25] Extensive cell loss throughout the media and adventitia in cryoplasty-treated animals occurred primarily through induction of necrosis. There was no significant difference in rates of apoptosis in the 2 groups. Intravascular cryotherapy induced early arterial wall cell loss; however, no benefit on luminal area was observed compared with balloon angioplasty alone at 10 weeks. The absence of benefit in this study might be related to excessive freezing of tissue because the average vessel wall temperature was −26°C. This temperature might be expected to induce necrosis rather than apoptosis, negating any potential benefit. In contrast, a porcine coronary plaque model utilized cryoplasty (−10°C for 30 seconds) to assess the effect on arterial stenosis. Cryoplasty was compared with placebo (sham) 4 weeks after induction of arterial stenosis in the animals. On angiography, cryoplasty was associated with a relative reduction in percent stenosis of 25.4% compared with sham therapy.[26] Further refinements of cold energy protocols (temperature and application time) will require evaluation in similar trials before the efficacy of cryotherapy in human vasculature can be determined. The risks of cryoplasty are similar to those encountered with conventional balloon angioplasty. Because the balloon inflates with nitrous oxide, rupture with release of this gas causes no untoward effects.

In this case series, 5 patients with rapidly recurrent venous stenosis at the graft-vein anastomosis after conventional balloon angioplasty garnered benefit from cryoplasty. It is notable that the venous stenoses that developed in these patients were hemodynamically significant as evidenced by degree of stenosis (>50% stenosis and elevated pressure ratio across the stenosis), surveillance criteria (ABF and Vp), and thrombotic events. Cryotherapy increased the time to restenosis and/or thrombosis from a mean of 3

weeks to a mean of 16.8 weeks (range, 13 to 21 weeks). Importantly, 2 patients did not have recurrence of the initial lesion but formed stenoses at sites several centimeters from the initial lesion. One could argue that surgical revision might be a better option to promote increased AVG patency. However, this approach requires an operative procedure and sacrifices vein in patients who have limited-access sites. The positive results obtained in these 5 cases are promising but clearly preliminary. Whereas cryoplasty significantly increased time to restenosis of venous lesions, it did not eliminate them completely as seen by recurrence in 3 of the 5 lesions (patients 1, 4, and 5). In addition, cryoballoon technology is extremely costly. This report should prompt study of cryotherapy in the pig model of AVG venous stenosis.[5] If positive results are noted, a prospective study in ESRD patients with rapidly recurrent AVG stenosis would be in order. An even more provocative approach would be to use cryoplasty at the graft–vein anastomosis at the time of initial AVG creation to prevent early development of neointimal hyperplasia.

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