Vascular access flow reduction for arteriovenous fistula salvage in symptomatic patients with central venous occlusion

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ABSTRACT

Purpose: Vascular access patients with central vein (CV) stenosis or occlusion may have significant symptoms. Treatment is generally by balloon angioplasty, with or without stenting. However, CV lesions may not be correctable and when treated, tend to recur. Surgical bypass of CV obstruction is a major procedure and ligation of the access may leave the patient dependent on catheter dialysis. We review a precision inflow banding procedure to limit vascular access flow and pressure for symptomatic patients with CV obstruction while preserving access functionality.

Materials and Methods: All individuals with symptomatic CV occlusive disease who underwent an autogenous vascular access inflow restriction procedure by the two senior authors were identified. All had failed attempts to correct CV lesions by angioplasty and stent placement. A precision banding procedure was used for access inflow reduction with the addition of real-time intravascular flow monitoring.

Results: Twenty-two patients were identified. Ages were 22-72 years (mean=43 years). Nine patients (40.9%) were women, and 8 (36.4%) obese. Mean access flow was 1640 mL/minute before banding decreased to 820 mL/minute after banding (P<.01). All patients had access salvage. Swelling resolved promptly in 20 patients and was markedly improved in two individuals. Three patients underwent aneurysm repair with simultaneous inflow banding and decreased intra-access pressure after flow restriction. Two fistulas failed at eight and 13 months. Mean follow-up was 8 months.

Conclusions: The symptoms of hemodialysis vascular access patients associated with non-correctable central venous lesions resolved successfully and their access was maintained using a precision inflow banding procedure.

Key words: Angioplasty, Arm swelling, Arteriovenous fistula, AVF, Central venous, Hemodialysis, MILLER Banding, Obstruction, Stenosis, Vascular access

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INTRODUCTION

Central vein (CV) stenosis and occlusion are common problems facing patients undergoing renal replacement therapy by hemodialysis (1). These lesions are generally associated with hemodialysis catheters and are more commonly found in patients with multiple catheters, a history of catheter infections, or long-term catheter use (2). Individuals with central venous disease may be asymptomatic and successfully undergo dialysis on a routine basis with venous outflow provided by multiple collateral vessels (3). In cases where the total cross-sectional area of draining collaterals is insufficient to handle arterialized flow, patients may have significant symptoms with swelling and pain in the affected extremity that may extend to the head, neck, and chest wall or breast. These individuals are often treated successfully by CV angioplasty with or without stent placement; however, the lesions tend to

recur (2,4,5). Surgical bypass is less commonly utilized as it is often a major procedure in these patients (6-9). In many symptomatic patients physical findings are consistent with a large, well developed, high flow access with little peripheral access pathology. However, diseased central veins and ensuing symptoms may lead to ligation of an otherwise well functioning access for some patients. Since access sites are limited and contralateral central veins are frequently strictured from prior catheter placement, it is often difficult to establish a new permanent access and patients may become catheter dependent for dialysis access. This study reviews our experience with an arteriovenous fistula (AVF) inflow restriction technique (restoring inflow-outflow balance) that limits access blood flow and pressure while preserving functional fistulas in patients where other methods of treatment were not successful or feasible.

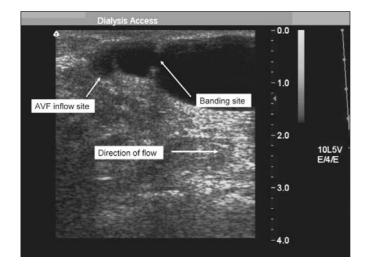


Fig. 1 - Ultrasound image of an arteriovenous fistula (AVF) following inflow restriction (banding).

MATERIALS AND METHODS

We reviewed the separate databases of consecutive vascular access patients of two senior authors (WCJ and GAM), identifying all individuals who underwent an autogenous vascular access inflow restriction procedure because of CV occlusive disease. Each patient had symptoms and physical findings that warranted access abandonment and ligation if no other option was available. Each patient had similar findings leading to the procedures that included multiple attempts to correct CV stenosis or occlusion by angioplasty and stent(s) placement. All patients had 3+ to 4+ pitting edema, significant discomfort related to pain as well as limited mobility of the affected extremity, and lack of surgical opportunities for correction of the CV lesion without thoracotomy or extra-anatomic (extracavitary) bypass.

The inflow-limiting technique (MILLER) reported by Goel, et al. for patients with steal syndrome and by Miller et al. for flow reduction was used with the addition of realtime US flow monitoring, or Transonics intravascular flow monitoring (10,11). Most commonly, a four millimeter angioplasty balloon supplied the restrictive configuration and two adjacent polypropylene sutures were tied over the inflated balloon (banding over the balloon served as a sizing dowel). Ultrasound and physical examination were used to locate a small transverse incision site and establish the restriction point close to the anastomosis, just past the AVF surgical scar, as the vein assumes a superficial location. Figure 1 shows a post-operative ultrasound image with the banding in place. MILLER banding procedures were performed with real time flow volume measurements using ultrasound assessment. Patients with enlarging aneurysms requiring simultaneous repair also had intra-access pressure monitoring during the procedure.

Our aim in these challenging patients was resolution of symptoms with maintenance of a functional vascular access. Patency in this report refers to functional access cannulation with two needles and the prescribed dialysis flow. Access flow data were analyzed by paired t test using Prism 4 software with statistical significance of differences determined at P=.05. This study was approved by our institutional review board.

* GraphPad Software, La Jolla, CA.

RESULTS

Twenty-two patients were identified as meeting the above criteria and represented all individuals undergoing an AVF inflow-limiting procedure for CV stenosis or occlusion treated by the two authors (WCJ n=12, GAM n=10). All patients in this study had an autogenous access with stable function for more than six months. Ages were 22-72 years (mean=43). Nine patients (40.9%) were women and eight (36.4%) were obese. The cause of end-stage renal disease was diabetes in seven (31.8%) patients and hypertension in nine (40.9%) individuals. Each patient's AVF was dramatically pulsatile prior to treatment. The AVFs treated had fusiform enlargement, and three had expanding aneurysm formation requiring surgical correction. Twelve patients underwent the MILLER banding procedure in the angiogram suite using a 2 cm transverse incision just proximal to the AV fistula anastomosis (3) and 10 were performed using two lateral 0.5 cm incisions coupled with a blind, blunt dissection (11). The veins were uniformly mature and easily dissected free with passage of two interrupted 3-0 polypropylene sutures secured over a 4 mm balloon angioplasty balloon (a 3 mm diameter balloon was used in some instances). Individual demographic data with pre and post-procedure ultrasound flow monitoring values are shown in Table I. One patient did not have access flows measured. The mean pre-banding access flow was 1640 mL/minute (range 870-4200 cc/min) and mean post-banding access flow was 820 mL/minute (range 412-2050 cc/min) (P<.01). Twenty of the 22 AVFs treated remain functional with follow-up of 3 to 24 months (mean=eight months).

The three individuals with enlarging aneurysms were treated in the operating room with aneurysm repair and simultaneous inflow banding. Each of these patients had intra-access pressure monitoring before and after banding in addition to ultrasound flow monitoring. Pre-banding mean access pressures were 98, 74, and 38 mmHg for these patients. After banding mean access pressures fell to 63, 41, and 25 mmHg, respectively, while systemic mean arterial pressures remained stable.

Vascular access was initially salvaged in all 22 individuals. Swelling resolved promptly in 20 patients and was markedly improved in two individuals. All AVFs were firm and pulsatile prior to banding and these findings

resolved immediately following the procedure. There were no wound infections, disruptions, hemorrhage, or other complications associated with these procedures. Five patients had swelling prior to banding that involved the neck, face or breast; in all cases these findings resolved post banding.

Four patients later had recurrent swelling with symptoms less severe than pre-treatment levels but warranted repeat fistulagrams and central venous imaging. All had intact banding sites. One of these patients underwent repeat angioplasty of collateral outflow veins with improved symptoms and swelling. Another patient had a closed central venous stent that was eventually recannulated and successfully treated by balloon angioplasty and repeat stenting. Two patients had repeat banding 1 cm proximal to the previous band to create additional resistance and flow reduction. One patient died during the study period from causes unrelated to vascular access with a functional AVF.

One patient had access thrombosis eight months after banding and the AVF could not be salvaged. A second individual had extensive and gradual aneurysm formation with concern over bleeding risk that resulted in access ligation 13 months after banding. Both of these patients later had new AVFs established in the contralateral extremity. No aneurysms developed proximal to the flow restriction sites. No deaths were related to the vascular access or banding procedures. One individual required placement of a temporary catheter following repair of an associated aneurysm. All other patients had uninterrupted cannulation of their access for hemodialysis.

TABLE I - PATIENT DEMOGRAPHIC INFORMATION AND ULTRASOUND MEASURED FLOW VOLUME BEFORE AND AFTER MILLER BANDING.ONE PATIENT DID NOT HAVE ACCESS FLOWS MEASURED. MEAN PRE-BANDING ACCESS FLOW WAS 1640 ML/MINUTE AND MEAN
POST-BANDING ACCESS FLOW WAS 820 ML/MINUTE (P<.01).</td>

Age (years)	Gender	Obese	Cause of Renal Failure	Pre-Banding Flow (cc/min)	Post-Banding Flow (cc/min)	Post-Banding F/U (mos)
33	Female	Yes	Diabetes	1640	830	3
35	Female	Yes	hypertension	N/A	N/A	3
69	Male	No	Hypertension	2690	1360	3
54	Female	Yes	Chemotherapy	930	520	4
32	Male	No	Hypertension	1430	645	5
44	Male	Yes	Hypertension	2600	1000	6
56	Male	No	Diabetes	1240	530	6
24	Female	No	Hypertension	970	560	7
68	Female	Yes	Diabetes	870	420	7
46	Male	No	Polycystic Kidney Disease	4200	1800	8
35	Male	No	Hypertension	2010	820	8
43	Female	Yes	Diabetes	3900	1300	11
70	Male	No	Renal Cancer	2000	900	12
56	Male	No	Hypertension	1420	530	13
58	Male	No	Diabetes	1480	412	13
22	Female	No	Obstuctive uropathy	1640	820	14
26	Male	No	Glomerulonephritis	1920	640	14
72	Male	No	Hypertension	1600	850	17
38	Male	No	Hypertension	3000	2050	17
66	Male	Yes	Diabetes	2500	1100	18
41	Female	Yes	Failed Transplant	2000	1000	21
40	Female	No	Diabetes	3000	800	24

DISCUSSION

The incidence of CV stenosis or occlusion in hemodialysis patients is substantial. MacRae et al. found 55 of 235 individuals had significant central venous stenosis in their patient population (1). Although patients will most often have a history of CV catheters, some individuals may have symptomatic CV obstruction without a prior catheter placement and in these cases a combination between anatomic variants and turbulent high flow are likely causative agents (12,21). Swelling with associated pain may progress quickly or slowly worsen over many weeks or months as access flow increases and/or outflow collaterals develop intimal hyperplasia and occlude, worsening the inflow-outflow mismatch. Physical findings include arm, neck, head, or breast swelling often associated with vascular access dysfunction in the form of recirculation and unsuitable dialysis, prolonged cannulation site bleeding, etc (2,5,13). Severe edema may increase the risk of lymphatic congestion and cellulitis. Patients may have asymptomatic CV stenosis with venous outflow provided by multiple collateral veins. Levit, et al. found better outcomes in these individuals followed by observation than those treated by intervention with angioplasty (3).

Treatment options are summarized in Table II and include observation for asymptomatic individuals or those with only moderate edema and no pain, inflammation, or threatened tissue ischemia. The most common method of treatment for symptomatic CV stenosis or occlusion is by interventional technique; advancing a guidewire through the stenosis or penetrating a short segment of occlusion followed by balloon dilatation. A retrograde or antegrade approach may be utilized, and in difficult lesions, both techniques may be necessary (14). A stent may be placed primarily during the initial treatment or reserved for recurrent or difficult lesions. Various stents have been developed for this purpose, including bare metal

TABLE II - OPTIONS FOR TREATMENT OF CENTRAL VENOUS STE-NOSIS OR OCCLUSION IN VASCULAR ACCESS PATIENTS

- Observation for asymptomatic patients with modest or no physical findings or symptoms
- Central venous angioplasty
- Central venous angioplasty with stenting
- Angioplasty of collateral veins.
- Banding (inflow-limiting procedure)
- Surgical bypass to unobstructed venous outflow
 - Surgical bypass to ipsilateral unobstructed central venous outflow such as external or internal jugular, or basilic vein
 - Surgical bypass to contralateral venous outflow such as subclavian or internal jugular vein
 Surgical bypass to lower extremity venous outflow such as femoral or
 - Surgical bypass to lower extremity venous outliow such as remoral of popliteal vein
 - Surgical bypass to intrathoracic or intraabdominal vena cava or right atrium

Sacrifice of the access with ligation of the AV fistula or graft

and covered stents. These therapies have a high initial success rate but recurrence or later failure is common, often requiring retreatment (2,4,5,13,15,16). The NKF-K/DOQI guidelines state "Stent placement combined with angioplasty is indicated in elastic central vein stenosis or if a stenosis recurs within a 3 month period." (17). Yevzlin and Asif et al. reviewed available evidence for stent usage and found no conclusive patency benefit for stent insertion compared with angioplasty alone in central venous stenosis (18). Stent placement is costly. Salman and Asif calculated a cost/benefit ratio for general stent placement for dialysis access and found the total cost per patient benefited amounted to \$47 665.12 when accounting for all stents used in each patient (19).

Oquzkhurt et al. reported a patient where flow reduction was utilized in the treatment of arm swelling caused by central venous obstruction (12). In the patient reported, flow was reduced from 2900 mL/minute to 720 mL/minute with maintenance of the access and resolution of symptoms (12). Tellioglu, et al. measured access flow with ultrasonography to guide the degree of surgical restrictive banding in patients with highflow vascular access for treatment of cardiac failure and ischemic steal syndrome (20). Miller and Friedman also reported success with flow reduction procedures in the treatment of recurrent cephalic arch stenosis (21). Other studies found AVF flow restriction banding to resolve high output cardiac failure (11,22). Such an inflow limiting procedure may become increasingly important with concerns over a potential risk of higher flow access in dialysis patients with heart disease (23).

Brachytherapy has been utilized in CV lesions as an addition to angioplasty and stenting, hoping to extend initial success following stent placement; however, patency was not improved (24). Extrinsic compression may also play a role in symptomatic CV stenosis or occlusions. Itkin et al. felt extrinsic lesions are more likely to require stent placement for success (25). Ahmad reported salvage of arterial venous fistulas with symptomatic swelling because of central venous stenosis by angioplasty of collateral veins (26). In a small subset of patients we experienced success in restoring inflow-outflow balance and achieved symptomatic relief by dilating collateral veins. Surgical treatment has been utilized when feasible with bypass to outflow options such as a patent and non-obstructed basilic, internal jugular or external jugular vein, gaining uninterrupted flow into the central venous system (8,27). More extensive procedures such as bypass to the contralateral jugular, subclavian or axillary vein have been reported (6,7,28). Intrathoracic procedures or femoral extra-anatomic bypass have been reported for access salvage (9,29,30). Among the surgical bypass procedures, Kalra, et al. reported higher success rates

using autogenous vein bypass as opposed to PTFE in their access patients (8). Dammers, et al. reviewed patients treated by surgery versus angioplasty. Seventyfive percent of those treated with surgical bypass had a patent access at 12-month follow-up as opposed to a 63% success rate in those treated with angioplasty (27).

Occasional patients may have moderate flow within a vascular access associated with symptomatic venous hypertension that requires treatment. If significant symptoms are present, such a patient would likely have a marked elevation in AVF venous outflow pressure that may be because of acute collateral outflow thrombosis complicating a chronic CV occlusion. The sudden loss of outflow collateral veins may result in elevated access pressure in spite of a relatively moderate AVF flow volume. For these individuals with moderate or even lower flow AVFs, a restrictive inflow banding procedure may lower pressure in the access outflow, restoring or improving inflow-outflow balance while maintaining adequate blood flow for dialysis. In our opinion, banding an access with flow volume lower than 700-800 mL/ minute may not be successful. The lowest access flow in the patients treated in this study was 870 mL/min. Monitoring access flow volume during flow restriction procedures should predict a successful outcome and minimize the risk of lowering access flow to the point of AVF dysfunction or thrombosis. In general, the minimum post-banding access flow for AVFs should be approximately 500 mL/min.

All of the accesses in this study were mature AVFs. However, one of the authors (GAM) has used the reported banding technique to treat grafts associated with symptomatic steal syndrome, high access flow, and/or significant arm swelling in a limited number of patients (11). The primary patency of banded graft accesses is lower than that of AVFs. However, banding remains an option to consider, particularly in patients where significant collateral outflow is present and graft ligation would be otherwise necessary. Grafts with noncorrectable occluded CV outflow and few collaterals are likely to have recurrent thrombosis and early failure. These accesses are rarely worth attempts at salvage. Banding a graft when the initial access flow is less than 800 mL/min may be problematic. In addition, banding of any access with chronic CV outflow occlusion is not likely to be successful when venous outflow collaterals are notably absent.

For those patients where interventional techniques with balloon angioplasty were not successful and a surgical bypass is not anatomically feasible or carries unwarranted risk, ligation with abandonment of the access remains a final option for resolution of symptoms. The number of fistulas or grafts ligated because of venous outflow disease is unknown but it is likely to be a common problem in the United States. We suggest many of these patients may have access salvage and relief of symptoms using the relatively simple banding procedure utilized in this study.

The symptoms of vascular access patients associated with central venous lesions resolved successfully and their access was maintained using a precision inflow banding procedure by treating the primary pathology of these AVFs which is high inflow in relation to limited and non-correctable access outflow. When flow through the central veins is impeded by stenosis, occlusion, or anatomic variants, this banding technique restored inflow-outflow balance and decrease pressure within the venous outflow tract, successfully mitigating symptoms while maintaining access patency.

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