Fibrin Sheath Angioplasty: A Technique to Prevent Superior Vena Cava Stenosis Secondary to Dialysis Catheters

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Abstract

Fibrin sheaths are a heterogeneous matrix of cells and debris that form around catheters and are a known cause of central venous stenosis and catheter failure. A total of 50 cases of central venous catheter fibrin sheath angioplasty (FSA) after catheter removal or exchange are presented. A retrospective review of an outpatient office database identified 70 eligible patients over a 19-month period. After informed consent was obtained, the dialysis catheter exiting the skin was clamped, amputated, and a wire was inserted. The catheter was then removed and a 9-French sheath was inserted into the superior vena cava, a venogram was performed. If a fibrin sheath was present, angioplasty was performed using an 8 × 4 or 10 × 4 balloon along the entire length of the fibrin sheath. A completion venogram was performed to document obliteration of the sheath. During the study, 50 patients were diagnosed with a fibrin sheath, and 43 had no pre-existing central venous stenosis. After FSA, 39 of the 43 patient's (91%) central systems remained patent without the need for subsequent interventions; 3 patients (7%) developed subclavian stenoses requiring repeat angioplasty and stenting; 1 patient (2.3%) developed an occlusion requiring a reintervention. Seven patients with prior central stenosis required multiple angioplasties; five required stenting of their central lesions. Every patient had follow-up fistulograms to document long-term patency. We propose that FSA is a prudent and safe procedure that may help reduce the risk of central venous stenosis from fibrin sheaths due to central venous catheters.

Keywords

- inferior vena cava thrombosis
- percutaneous
- proximal
- renal failure
- subclavian stenosis
- vascular access
- deep vein thrombosis

In North America between 40 and 60% of new hemodialysis vascular access is achieved with a centrally placed venous catheter. Care for these catheters and catheter-related complications are estimated to cost over \$1 billion dollars a year.^{1–3}

Despite the strong evidence that an arteriovenous fistula (AVF) is superior to a hemodialysis catheter for long-term dialysis, there are certain situations where dialysis catheter use is the preferred method of hemodialysis. These include need for immediate dialysis, a bridge to permanent hemodialysis access, comorbid conditions limiting life expectancy, those with systolic hypotension in whom attempts to create/maintain a permanent access site have failed and those in whom all available sites for fistula are no longer feasible.^{4,5}

Central venous catheters are associated with a range of complications, which include the following: malfunction, thrombosis, central venous stenosis, extremity edema, infection, and the development of fibrin sheaths.^{6,7} When compared with a fistula, central venous catheters carry a relative risk of death 1.6 times greater and a fivefold increase of infection.^{1,8–17} As a result of the multiple complications associated with dialysis catheters, catheters need to be removed or replaced frequently.⁵

Central venous stenosis has a reported incidence of 40 and 70%.^{5,11,18–24} Central venous stenosis may lead to superior vena cava syndrome (SVC syndrome); this represents the end stage of the stenosing process.²⁵ SVC syndrome is associated

with a litany of management complications including fistula failure and neurological derangement.

The formation of central venous stenosis and its relationship to central venous catheters is not well understood. A current hypothesis begins with the placement of a central venous catheter. Repeated vein wall trauma from the catheter and blood flow turbulence causes endothelial damage resulting in hyperplasia. The repeated vein wall trauma leads to central venous stenosis.^{26,27} The addition of an AVF accelerates and accentuates this flow abnormality. Identification of central stenosis is important, even when not clinically significant, because it may lead to increased venous pressures and increased risk of access failure, including dialysis catheters as well as AV grafts and fistulas.

Currently, strategies to prevent central stenosis do not exist. There are a few treatment options for existing central stenosis. Balloon angioplasty, stent placement, clot lysis, and surgical reconstruction are all possible options. Long-term patency without reintervention is only 25% after 3 years. Unfortunately, these procedures cannot be performed in all patients, and long-term outcomes are variable and generally poor, even with multiple interventions. Therefore, identification and prevention of central venous stenosis before it symptomatic is paramount.

Fibrin sheaths occur in 80 to 100% of all hemodialysis catheters within 1 week of insertion. When the sheath progresses to cover the end holes of the catheter, the catheter may malfunction. Fibrin sheath occlusion should be considered when a well-positioned catheter fails to function properly.²⁸⁻³¹

Standard strategies for management of fibrin sheaths have demonstrated limited success. Current therapies include percutaneous fibrin sheath stripping, thrombolysis infusion, and catheter exchange.³¹ While each generally demonstrates good initial results, there appears to be poor patency of catheters beyond 1 month and thus represent a poor long-term solution to management of fibrin sheaths.³¹⁻³⁴ Medical management with antiplatelet agents and vitamin K antagonists such as warfarin are associated with an increased rate of bleeding complications.³⁵

The exact relationship between central venous stenosis and fibrin sheaths has not been well defined. This study attempts to identify and analyze the relationship between fibrin sheaths and central venous stenosis. Additionally, we present the benefits of fibrin sheath angioplasty (FSA) in the potential prevention of central stenosis in patients with long-term hemodialysis catheters.

Materials and Methods

A retrospective review of 70 patients was performed on patients who underwent hemodialysis catheter removal from July 2008 to February 2010. Patient demographics can be found in ►Table 1. Patient information and images were obtained from an office database ►Figs. 1 and 2. All patients at the time of initial evaluation signed consent for study and image acquisition, and the study was reviewed and approved by a review board.

Table 1 Demographics

Total patients	70
Male	38
Female	32
Race	
Caucasian	35 (50%)
African American	19 (27.1%)
Other	10 (14.3%)
Hispanic	3 (4.3%)
Asian	3 (4.3%)

All patients, 70 (100%), were diagnosed with end stage renal disease, 52 (74.3%) were hypertensive, 17 (24.3%) were insulin-dependent diabetics, 17 (24.3%) were noninsulin-dependent diabetics, and 10 (14.3%) had an alternative diagnosis.

All patients had 15.5-French, dual lumen, low lying tunneled, cuffed internal jugular (IJ) catheters in place. All catheters were being removed because patients had functioning AVF or AV grafts, and thus were no longer needed. A total of 59 (84.3%) of the patients had right-sided catheters and 11 (15.7%) had left-sided catheters with an average catheter insertion time of 29 (range 3 to 58) weeks.

Patients were taken to the operating room and positioned supine on the operating room table. The catheter was clamped 1 cm from the level of the skin, and the remaining catheter was amputated. Both the patient and catheter were then prepped and draped in a sterile fashion; precutting allowed for a more sterile field. The skin was injected with 3 to 5 mL of local anesthetic, and the catheter cuff was freed and delivered to the level of the skin. A 0.035-inch 150-cm Bentson wire (Cook Medical, Bloomington, IN) was introduced through the transected catheter into the central venous system. The remaining catheter was removed leaving the wire in the SVC. A 9-French 25-cm introducer (Terumo, Somerset, NJ) was placed over the wire to obtain access to the inferior portion of the IJ vein. Dilute Visipaque (four parts Visipaque to six parts injectable saline) (GE Healthcare, Milwaukee, WI) was used as the contrast agent. Venograms were obtained and analyzed for central venous stenosis and a fibrin sheath.

If a fibrin sheath was identified, an 8 × 40 mm ($n = 36$) or 10 × 40 mm ($n = 12$) angioplasty balloon was introduced over the wire. Selection of balloon size was at discretion of the individual performing the procedure. Working from the distal aspect of the sheath proximally (central to peripheral), sequential disruption of the fibrin sheath was performed. The balloon was removed and a completion venogram was performed to document resolution of the fibrin sheath. Angioplasty was repeated if the fibrin sheath was not completely disrupted.

Upon termination of the procedure, the introducer was removed and hemostasis achieved by placing a 2-0 proline suture around the tract above the puncture site; it was removed after 24 hours. Patients were recovered and

Fig. 1 Venogram findings and group characteristics. Above right IJ with a fibrin sheath pre- and postfibrin sheath angioplasty. Below subclavian stenosis with central stenosis pre- and postangioplasty.

discharged home the same day. ► **Fig. 3** shows the various steps of FSA.

Routine venograms were performed on all patients during follow-up. The venograms were performed as a planned evaluation of the fistula. Re-evaluations were performed on average 27.4 (range 2 to 40) weeks postcatheter removal, and 4 any identified central stenosis underwent balloon angioplasty.

Fig. 2 Venogram findings and group characteristics.

Catheter removal and follow-up images were studied for every patient by the senior author and an independent reviewer. Discrepancies of interpretation were presented to all study members blindly for a final consensus. Chi-squared analysis of the three groups was performed to determine statistically significant differences between the groups with a $p < 0.05$.

Results

Over a 19-month study period, a total of 70 patients underwent a catheter removal/exchange and venogram. In 20 (28.6%) patients, no fibrin sheath was identified and venograms demonstrated a normal IJ vein and SVC (Group 1). A total of 50 (71.4%) patients were identified as having a fibrin sheath; 43 (68.3%) of the 50 patients had fibrin sheaths and normal underlying vein (Group 2) while the remaining 7 (10%) patients had a fibrin sheath and an underlying stenosis or occlusion of their central vein (Group 3). In total, 63 (90%) patients had no central venous stenosis or occlusion. Total 48 (96%) of the 50 patients identified as having a fibrin sheath underwent balloon angioplasty. Angioplasty was not performed in two patients; one had a large thrombus with risk of embolization, and the other had a previously placed central venous stent.

Fig. 3 (A) Left fibrin sheath, (B) post-FSA, (C) balloon angioplasty, and (D) 1-year follow-up.

Follow-up results are depicted in ►**Fig. 2**. In Group 1 (no sheath, normal vein) only 1/20 (5%) developed subsequent central venous stenosis in the subclavian vein. No patient in this group developed a SVC or IJ stenosis or occlusion. In Group 2 (fibrin sheath, normal vein) 4/43 (9.3%) developed central venous lesions; 3 stenoses (7%) and 1 (2.3%) occlusion. All seven patients in Group 3 developed central venous disease on follow-up; two SVC stenoses and five SVC occlusions.

Comparing Group 1 with Group 3, there was a statistically higher incidence of central venous stenosis/thrombosis in patients with a fibrin sheath and pre-existing central venous disease even after FSA ($p < 0.05$). Patients in Group 2 had a lower incidence of central venous stenosis compared with Group 3, but did not reach statistical significance in this study.

No reports of postprocedure bleeding, shortness of breath, cardiac arrhythmias, hospital admission, or pulmonary embolism were reported. Patient complaints were limited to having slight discomfort during the placement of the introducer. There were no mortalities.

Discussion

Central venous stenosis and SVC syndrome are real and devastating consequences of central venous catheter usage. Early identification and prompt treatment is of paramount importance to minimize both morbidity and mortality; preventions serving as the primary goal.

Fibrin sheaths are a complex of fibrin, collagen, and thrombus around a foreign body, which over the course of several weeks organizes into a fibrin sheath. They are re-

ported to occur in to 50 to 100% of patients with central venous catheters. In the first 24 hours, a newly placed catheter activates the coagulation cascade thrombus and fibrin adhere to it.^{2,8,10,36,37} As the catheter continuously rubs against the vein wall, it perpetuates the initial inflammatory process; endothelial cells are denuded from the vein wall exposing smooth muscles cells and fibroblasts below. This inflammatory process results in thrombus development, intimal hyperplasia, and smooth muscle cell migration onto the adjacent catheter. Originating where the catheter enters the vein, cellular migration progresses distally along the catheter toward its tip. After several weeks, thrombus and fibrin are gone and a well-organized sheath composed primarily of collagen, fibroblasts, epithelial cells, and smooth muscle cells encompass the catheter. Cellular bridging between the vein wall and the newly formed fibrin sheath occurs, stabilizing it and making it a permanent structure vessel. It has been reported that 50% of catheter malfunctions are the result of a collagen sheath; thrombus, infection, and unknown reasons representing other causes.^{28–31} Bacteria can embed themselves into the sheath increasing the risk of line-related infections and sepsis. Left alone, a fibrin sheath may persist months to years after catheter removal, and it is this longevity that may contribute to the increased risk of developing central venous stenosis after central venous catheter usage.

It is documented that central venous catheters and central venous stenosis are related; a relationship between fibrin sheaths and catheter malfunction has also been reported.^{28–30} The association between fibrin sheaths, central stenosis, and SVC syndrome remains unexplored.

Venogram results suggest that narrowing alters venous return and laminar flow that is present in a normal vein creating a turbulent flow pattern. This turbulent flow is damaging to the fragile venous intima and causes inflammation within in the vessel wall.³⁸ Inflammation encourages intimal hyperplasia and continued stenosis; unchecked this process may lead to a critical stenosis and ultimately SVC syndrome. Disruption of the fibrin sheath may restore laminar or near laminar flow halting the inflammatory cycle and the progression of central venous stenosis. Of the seven patients who redeveloped stenosis or occlusion post-FSA it is hypothesized that a critical point of no return in their inflammatory process was passed. Once this occurs, hyperplasia and inflammation are irreversible and thus angioplasty had no long-term benefits. This may also explains why stenting serves only as a temporary fix. A stent's radial force may be irritating to the vein wall promoting the inflammatory cycle leading to intimal hyperplasia and stenosis.

In this study, 70 patients were involved, more patients than most previously published articles about fibrin sheaths. Sheaths were identified in 60% of the patients which correlates with other published studies. Central venous stenosis postangioplasty was only 7%, well below the reported average of 40 to 70%. Comparing Groups 1 and 3 a *p* value of <0.05 was obtained. Significance was not achieved when comparing Groups 1 and 2 or Groups 2 and 3. Additionally, the length of catheter insertion time and follow-up period is longer than other studies with, most averaging less than 90 days for either group. No patients suffered any adverse event, neither had signs or symptoms of PE nor required hospitalization.³⁹

There are several weaknesses of the study, it is a small study, retrospective and while our results give indirect evidence that fibrin sheaths are associated with subsequent central stenosis and that FSA may reduce development of central stenosis, a prospective, randomized study is required to confirm our suspicions. Additionally, this study did not evaluate types, locations, or length of physical catheter insertion or data about a patient's previous history of catheter or central line usage. Finally, there is no specific control arm and results are compared with the historical published record.

Conclusion

Fibrin sheaths are an easily diagnosed entity that may be treated with FSA at the time of a central catheter exchange or removal. Study results suggest a correlation between fibrin sheaths and central venous stenosis, noting that obliteration of a fibrin sheath may reduce the risk of both short- and long-term central venous catheter complications.

Patients with central venous stenosis and fibrin sheaths present at the time of catheter removal or exchange did worse after FSA with a 100% recurrence of their stenosis as compared with their counterparts. This population may not benefit from FSA where as those with a sheath and no stenosis in large remained complication free.

FSA may be a beneficial procedure but a larger prospective study with a control group would be necessary to make any final conclusions.

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