Original Article

Endovascular Salvage Procedures in Increasing the Longevity of Hemodialysis Access

Rajendra Prasad Basavanthappa, AR Chandrashekhar, Sanjay C Desai, Aniruddha S Bhuiyan

Abstract

Introduction: The leading cause of failure for an autogenous or prosthetic arteriovenous hemodialysis access is venous anastomotic stenosis & central venous stenosis. Endovascular procedures have come a long way in treating such lesions without significant postoperative morbidity and reliable technical success rates. This study aims at studying the long term results of such central venous and anastomotic site stenosis treated with endovascular approach in our centre.

Materials and Methods: A total of 26 patients were treated, for a duration of 33 months between March 2012 to November 2014 with central venous, anastomotic site angioplasty ± stenting. Patients were followed up for 9 months. We had re-intervention in 3 cases.

Results: Inadequate dialysis, thrombosed graft were the commonest presenting symptoms. Primary patency at the end of 15 months was 34.6% (9 patients), & at the end of 20 months was 11.5% (3 patients).

Conclusion: Endovascular treatment with angioplasty or stenting for central venous stenosis & access salvage is safe, with low rates of technical failure. Only angioplasty without stenting seems by far the preferred approach, since it is less invasive, highly repeatable.

Key Words: Arteriovenous access salvage, arteriovenous fistulae, central venous obstruction, proximal outflow obstruction

Introduction

The leading cause of failure for an autogenous or prosthetic arteriovenous (AV) hemodialysis (HD) access is venous anastomotic stenosis and central venous stenosis. The optimal therapy for symptomatic central venous occlusive disease and venous anastomotic stenosis is not clearly defined. Although both endovascular and open procedures are in vogue for the treatment of stenosis and occlusions, neither are accepted as the standard of care.

The disease has great clinical importance, not only because of the appearance of the debilitating signs and symptoms of chronic venous hypertension but also because of the risk of losing venous access in HD patients. Venous hypertension is commonly associated with chronic pain and edema of the affected extremity.

There are numerous etiologies of symptomatic lesions; however, they most commonly result from prolonged central venous catheterization and ipsilateral AV fistula.

Pathology of disease is a response to vessel injury due to trauma, inflammation, intimal hyperplasia leading to fibrotic response leading to stenosis and occlusions. Causative factors are punctures and cannulations, hyperdynamic circulation accompanied by turbulence, size and compliance mismatch, central venous catheters.[1]

Balloon angioplasty, the first-line therapy, has a tendency to lead to subsequent recoil and restenosis; however, no other therapies have yet proved to be more effective, so endovascular interventions have become the treatment of choice; options being percutaneous angioplasties, with or without the use of stents.

This study aims at studying the long-term results of such central venous and anastomotic site stenosis treated with endovascular approach in our center.

Materials and Methods

This is a prospective observational single center study in a tertiary care center. Patients suffering from end-stage renal disease, undergoing HD either with native AV access or prosthetic graft were included.

The presenting symptoms varied from inadequate dialysis, extended bleeding time postdialysis, dilated veins over the chest and upper limb, upper limb swelling with hyperpigmentation, thrombosed AV grafts within 14 days of inadequate or inability to perform HD.
A total of 26 patients were treated, for the duration of 33 months between March 2012 and November 2014. Eighteen patients were male and eight were female aged between 49 and 68 years. Ten patients were suffering from both hypertension and diabetes mellitus, ten with hypertension, diabetes mellitus, dyslipidemia and two only with hypertension.

We treated 16 central veins [Figure 1] in patients among which 8 were brachiocephalic, 6 were axillary, and 2 were subclavian lesions. Eight venous end anastomosis [Figure 2] of the prosthetic grafts were treated among which two were lower limb thigh loop grafts. We also treated two puncture site stenosis.

Patients were followed up for 9 months. We had re-intervention in three cases among which one was in brachiocephalic and another two being venous end anastomosis stenosis. Ligation of the fistula was done in two of the patients because of persistent venous hypertension. We did not encounter any procedure related complications like vein rupture or hematoma.

The criteria for considering a case to be a clinical success were a significant improvement in the signs and symptoms of chronic venous hypertension and adequate dialysis from the AV access. However, arm circumference measurements were not taken during that period. Recurrence of the symptoms was considered clinical failure, as were attempts to conduct additional angioplasty, whether successful or not.

**Procedure**

Conventional angiograms were done either through radial artery, brachiocephalic fistula or through graft, visualizing both peripheral, central veins for diagnostic assessment and treatment planning for an AV fistula or thrombectomised prosthetic graft in the limb with venous hypertension or failing access.

After confirmation of stenosis or occlusion, with a 6F sheath (7/11 cm), a hydrophilic guide wire supported by a catheter was advanced in an attempt to cross the lesion. After adequate anticoagulation with intravenous unfractionated heparin and placement of the guide wire, lesions were crossed. A percutaneous transluminal angioplasty (PTA) balloon was positioned and expanded. If postplasty, check venogram showed more than 30% residual stenosis, a second angioplasty was performed, and a self-expanding stent was deployed.

All patients were discharged the same day or the next day of the procedure and were given anticoagulants during the postoperative period. All patients were discharged with antiplatelet medication.

Lower limb graft failures were also treated in a similar manner.

**Results**

Total number of patients involved in the study was 26. Majority belonged to age group between 51 and 60 years (42.3%), with mean age 57.38 ± 6.85. Predominantly male (69.2%) population was involved. Hypertension as the only comorbidity was seen in only 7.7% of patients. The main access type treated was AV fistula (57.7%).

Inadequate dialysis (30.8%), thrombosed graft were the commonest presenting symptoms [Table 1]. Others like extended puncture site bleeding (15.4%), limb swelling (11.5%), dilated veins over chest and limbs (11.5%) were almost equal in incidence [Graph 1]. 88.5% of our patients had previous HD catheter insertion before the creation of fistula, which is one of the important causes of central vein stenosis. In patients who underwent access salvage, 61.5% of them had failing access, i.e., reduced flows, compared to no flow or thrombosed access being 26.9% [Table 1]. The most common sites stenosis was brachiocephalic vein (30.8%), venous end of anastomosis (30.8%), and axillary vein (23.1%), and puncture site stenosis (7.7%), subclavian vein (7.7%).

We treated 16 central veins among which 8 were brachiocephalic (30.8%), 6 were axillary (23.1%), and 2 were subclavian veins (7.7%) in which two bare metal stents were put in the innominate vein. Among the 16 central veins, 12 were stenosis and 4 were total occlusions. Among eight venous end anastomosis of the prosthetic grafts treated two were lower limb thigh loop grafts [Figure 3].

With the prosthetic grafts, thrombectomy was done before angiogram and angioplasty. All thrombectomies were done within 14 days of the graft thrombosis.
Discussion

Central venous stenosis and obstruction, along with venous end anastomotic stenosis in prosthetic grafts and puncture site stenosis are an important problem and highly prevalent in patients on long-term HD. They cause venous hypertension with/without debilitating symptoms. The end result being loss of the access due to access dysfunction or ligation for symptom relief. The incidence of central venous stenosis has been reported in the range of 30% in the literature.\textsuperscript{1}

It has been proven beyond doubt that, with the previous placement of central venous catheters will cause central venous stenosis or occlusion, with incidence of 27%.\textsuperscript{2}

Patients with a history of subclavian catheters has 42%–50% incidence of central venous stenosis compared with internal jugular vein catheters.

The primary pathology of central venous occlusion is usually catheter-induced trauma to the venous endothelium and secondary inflammatory damage within the vessel wall, the presence of a foreign body in the vein, along with increased flow and turbulence from the creation of an AV access. Turbulent blood flow has been shown to incite an inflammatory response and stimulate intimal hyperplasia.\textsuperscript{3-7}

Symptoms of central venous occlusion/stenosis vary depending on progression and the anatomic position of the disease.\textsuperscript{5} Upper limb edema, cyanosis, varicose veins, hyperpigmentation, and even ulcers are seen in stenosis and occlusions in subclavian veins.

Edema of face and chest wall with dilated superficial venous network is seen in, brachiocephalic vein, occlusion/stenosis.\textsuperscript{15-7}

Table 1: Symptoms in relation to flow rates

| Symptoms                                | No flow | Normal flow | Reduced flow | Total (%) |
|-----------------------------------------|---------|-------------|--------------|-----------|
| Inadequate dialysis                     | 0       | 0           | 8 (50)       | 8 (30.8)  |
| Thromosed arteriovenous graft           | 7 (100) | 0           | 1 (6.3)      | 8 (30.8)  |
| Dilated veins over the chest and upper limb | 0       | 2 (66.7)    | 1 (6.3)      | 3 (11.5)  |
| Upper limb swelling with hyper pigmentation | 0       | 1 (33.3)    | 2 (12.5)     | 3 (11.5)  |
| Extended bleeding time post dialysis    | 0       | 0           | 4 (25)       | 4 (15.4)  |
| Total                                   | 7 (100) | 3 (100)     | 16 (100)     | 26 (100)  |
In patients on HD either graft or native access, output may be reduced, and venous pressure increased, leading to inefficient dialysis.

Clinically patients on HD, output may be reduced and venous pressure increased, leading to inefficient dialysis. Diagnostic confirmation is by a venous ultrasonography study with loss of cardiac and respiratory phasicity, which is a finding with a high positive predictive value.[6-8]

Venous phlebography with digital subtraction is still considered the gold standard.[9] However, computed tomography and magnetic resonance imaging could be alternatives to conventional angiography, with the risk of nephrogenic systemic fibrosis.[10]

The mainstay of treatment in central venous occlusion is endovascular. Options being PTA, with or without stents and more recently placement of covered stents. The K/DOQI guidelines recommend PTA, with or without stent placement as the preferred treatment approach.[11]

**Conclusion**

Endovascular treatment with angioplasty or stenting for central venous stenosis and access salvage is safe, with low rates of technical failure. Only angioplasty without stenting seems by far the preferred approach since it is less invasive, highly repeatable.

The preventable cause of central venous stenosis is catheter placement. Central venous catheter use should be avoided, particularly in the subclavian vein, in patients at risk or with existing renal dysfunction.

Satisfactory results in venous endovascular procedures are difficult to achieve, even with stenting, also needs multiple interventions. Hence, prevention is more important than treatment by avoiding central venous catheter insertions in patients who need dialysis in the near future, also proper planning in the placement of permanent dialysis accesses should be done.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

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