The Role of Percutaneous Angioplasty in the Management of Chronic Hemodialysis Fistulas

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One hundred forty-one dilatations of stenotic lesions in dialysis access fistulas were performed. The initial success rate was 82%. The one-year patency rate was 45%, with a 2-year patency rate of 24%. Best results were obtained with a discrete stenosis at a graft-to-vein anastomosis. The procedure can be done on an outpatient basis and, although long-term results are poor, in appropriate patients multiple dilatations can be performed to keep a fistula functioning for many years.

THE INCREASING NUMBER of patients with end-stage renal disease in the United States has created the need for reliable vascular access. The initial and favored procedure for the creation of an access fistula is the direct arteriovenous (A-V) fistula of the Brescia-Cimino type.[1,2] If this fistula fails or is not feasible, a graft made of polytetrafluoroethylene (PTFE) or bovine carotid artery can be placed subcutaneously between an artery and vein to provide access.[3–6]

The most common problems encountered with A-V fistulas and grafts are inadequate dialysis and graft thrombosis.[2,7] These usually result from a venous outflow stenosis or, less frequently, from inadequate inflow from an arterial lesion. Treatment has consisted of surgical revision of the stenosis or graft replacement at a different site. An alternative to surgical revision is treatment *via* percutaneous angioplasty. In this article we present our experience with dilatation of 141 stenotic lesions in dialysis access fistulas over a 7-year period.

Materials and Methods

Over the past 7 years we have performed 141 dilatations in 95 patients. Indications for angiography included repeated graft thrombosis (31 patients), inadequate dialysis with high recirculation (65 patients), high venous pressure during dialysis (75 patients), arm edema (15 patients), and enlarging venous aneurysms or graft pseudoaneurysms (13 patients).

One hundred twelve dilatations were performed in patients with grafts. Of these, three stenoses were at the arterial anastomosis, and 93 at the venous anastomosis. Sixteen venous stenoses were proximal to the anastomosis in the axillary or subclavian vein. Twenty-nine lesions were dilated in patients with direct A-V fistulas. Of these, 15 were in the outflow vein within 5–6 cm of the A-V connection and 14 in a vein distant from the A-V connections (five at the elbow, five in the axillary vein, and four in the subclavian vein).

Direct A-V fistulas were studied either by direct brachial artery puncture or by a venous puncture with inflation of a pressure cuff proximally to allow reflux of contrast into the arterial system.[8,9] Grafts were studied *via* a 5-F catheter placed directly in the venous end of the graft. If the arterial anastomosis needed to be visualized, a pressure cuff was inflated proximally to allow reflux of contrast into the arterial system. No study results were considered negative until the entire venous system to the level of the subclavian vein was visualized. Spurious narrowing of the axillary vein may be created by compression of chest wall muscles when the arm is in an adducted position, so this area is always studied with the arm abducted.

After delineation of the lesion, the stenosis was crossed with a 5-F catheter and a pressure pullback obtained. An appropriate size dilatation catheter was then chosen, depending on the size of the graft and normal vein on either side of the stenosis. Lesions longer than 4 cm were referred for surgery because these were univer-

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TABLE 1. *Patency Rates of Percutaneous Angioplasty*

Site	Initial	6 Months	1 Year	2 Years	3 Years	5 Years
Arterial anastomotic stenosis	2/3 (67%)	2/2 (100%)	1/2 (50%)	0/2	0/2	0/2
Direct arteriovenous fistula						
Anastomotic venostenosis	9/15 (60%)	4/9 (44%)	3/8 (38%)	1/7 (14%)	0/7	0/4
Graft anastomotic venostenosis	80/93 (86%)	42/71 (59%)	32/64 (50%)	16/50 (32%)	6/38 (16%)	0/20 (0%)
Far proximal venous stenosis (graft and direct arteriovenous fistula)	25/30 (83%)	13/25 (52%)	8/23 (35%)	2/20 (10%)	1/18 (6%)	0/14
Total	116/141 (82%)	61/107 (57%)	44/98 (45%)	19/79 (24%)	7/63 (11%)	0/40 (0%)

sally unsuccessful in our early experience. The dilatation catheter was introduced through the graft. There have been no problems introducing large balloon catheters through grafts that are older than 1 month old, and no pseudoaneurysms have developed at the puncture site after dilatation. Before dilatation the patient is given 3000 units of heparin intravenously (I.V.). Dilatation of veins may be painful. Infiltration of lidocaine around the stenotic lesion before balloon inflation may alleviate some of the pain. The first 50 cases were dilated with standard balloons that generated pressure of 3–4 atmospheres. The last 91 dilatations were done with high-pressure balloons that generate 10 atmospheres pressure. The dilatation pressure is monitored and the balloon left inflated for 1–2 minutes. Most patients received treatment on an outpatient basis and were observed for 30 minutes in the angiography suite after the procedure before being sent home.

Immediate results after the procedure were evaluated by angiography and pressure pullback. Long-term results were evaluated *via* clinical symptoms and efficacy of dialysis.

Results

Three patients died within 2 months of the procedure (causes of death unrelated to the dilatation), and 18 patients were lost to follow-up. Initial and long-term results are summarized in Table 1.

There were nine complications (6%): six graft thromboses within 48 hours of the procedure and three venous ruptures. All three ruptured veins were controlled by manual occlusion of the fistula and pressure dressings. None required transfusion or emergency surgery. All three were operated on electively within 1–7 days after procedure.

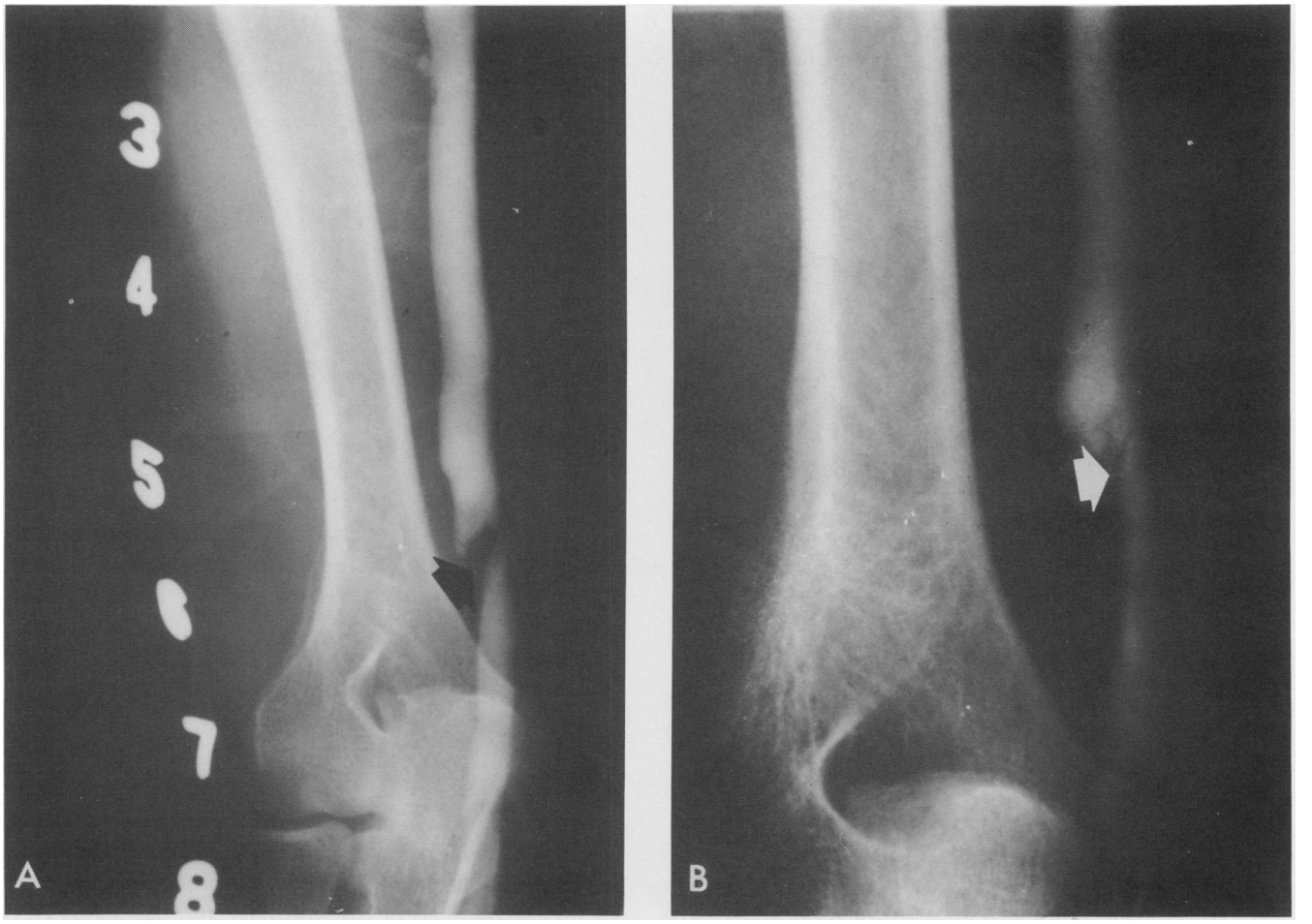
Discussion

Clinical problems necessitating fistula study include repeated thrombosis and inadequate dialysis as manifested by arterial sucking, high venous pressure, and recirculation of dialyzed blood.[2,9] These problems usually indicate poor arterial inflow or inadequate venous outflow. Other problems include arm edema, enlarging venous or graft aneurysms and pseudoaneurysms, and venous varicosities. These usually result from venous outflow obstruction.

Over the past 2 years, 800 graft and direct A-V fistulas have been surgically revised at Downstate Medical Center. Causes of fistula failure included a venous outflow stenosis in 70%, an arterial stenosis in 10%, and a combination of the two in 15%. Both the venous and arterial lesions invariably resulted from intimal hyperplasia, even in those fistulas corrected as early as 2 months after creation.

The most common operation performed was a thrombectomy and venous patch angioplasty. Only 20% of patients with a vascular access problem have diagnostic angiography. The remainder generally have complete fistula thrombosis and have surgical correction. Of those patients studied angiographically, approximately 90% have a venous outflow problem, either a total occlusion or significant stenosis. Approximately 40% of all patients studied have a lesion amenable to angioplasty.

Venous stenoses in direct A-V fistulas generally occur within 5–6 cm of the A-V communication. Graft venous stenoses usually occur at the graft-to-vein anastomosis or within 2–3 cm of the anastomosis.[9–13] Anastomotic venous lesions result from the combination of intimal hyperplasia and perivenous fibrosis. These stenoses are often resistant to dilatation and necessitate the use of high-pressure balloons.[14] Our last 91 angioplasties have been performed with high-pressure catheters (10 atmospheres), resulting in improved initial results (88% *vs.* 67% with standard balloons generating 3–5 atmospheres). Long-term results have been the same with both types of balloons. The turbulence and abnormal shear stresses thought to play a role in the deposition of platelets and thrombi that eventually lead to endovenous fibrosis and stricture are still present after dilatation and inevitably lead to restricture.

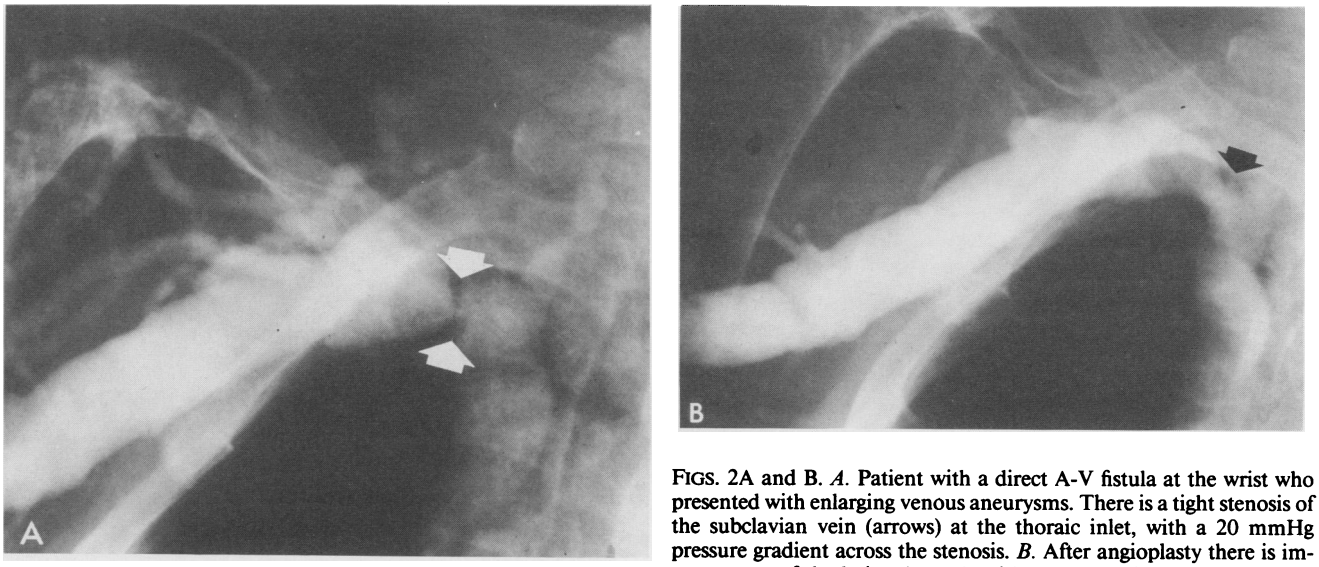


FIGS. 1A and B. *A*. There is a tight stenosis (arrow) at the anastomosis of a GORE-TEX (W. L. Gore and Associates, Inc.) graft to the basilic vein. *B*. After angioplasty there is significant widening of the stenosis (arrow), and the pressure gradient has been reduced from 60 mmHg to 0 mmHg.

Venous stenoses near the anastomosis of a direct A-V fistula are generally longer than the more discrete graft anastomotic venous lesions. This probably accounted for the better results obtained with graft dilatations (Fig. 1). Our results with long stenoses were so poor that we no longer perform dilatation in patients with lesions longer than 4 cm. Two total occlusions were treated. One was a short segment occlusion at the anastomosis of a graft to the basillic vein and the other was at the anastomosis of a graft to the axillary vein. A straight wire passed through the occluded segment easily. One dilatation was unsuccessful, and the other was patent for only 2 weeks. We now refer patients with total occlusions for surgical correction. New anastomotic sites are not dilated for a minimum of 4 weeks after operation, to preclude dehiscence of the anastomosis.

Stenosis may also occur within a graft because of progressive pseudointimal hyperplasia or trauma from repeated puncture. We have not dilated intragraft lesions because of the lack of muscular structure and elasticity in a graft and the subsequent risk of graft rupture. Intragraft lesions are usually long because of the widespread nature of pseudointimal hyperplasia.

Thirty proximal venous strictures were dilated. These tend to occur at sites of physiologic narrowing, the most common being in the cephalic vein at the elbow; the axillary vein, which is compressed by chest wall muscles when the arm is adducted; and the subclavian vein as it crosses between the clavicle and first rib (Fig. 2). Hypertrophied valves may also cause venous obstruction. Large valves most commonly occur in the axillary vein at the lower border of the subscapularis muscle and in the subclavian vein 2–3 cm proximal to its junction with the internal jugular vein (Fig. 3). Sites of previous central venous lines may also stricture when subjected to abnormal flow patterns. Three of the subclavian vein stenoses were at sites of a previous Shiley catheter. The turbulence created by high flow in a venous system with a physiologic or iatrogenic narrowing leads to eventual intimal hyperplasia and stenosis. Because of the fre-



FIGS. 2A and B. *A.* Patient with a direct A-V fistula at the wrist who presented with enlarging venous aneurysms. There is a tight stenosis of the subclavian vein (arrows) at the thoraic inlet, with a 20 mmHg pressure gradient across the stenosis. *B.* After angioplasty there is improvement of the lesion (arrow), with a decrease in the pressure gradient to 3 mmHg. There is still a residual narrowing caused by compression of the vein as it passes between the clavicle and first rib.

quency of proximal venous lesions (21%), all patients should be evaluated to the level of the subclavian vein before study results are considered negative. If angiograms are equivocal, pressure pullback from the superior vena cava to the anastomosis should be performed. Although there is normally a gradual venous pressure increase as one pulls back from the superior vena cava to the axillary vein, a discrete gradient of more than 5–10 mmHg should be considered significant. Because the anatomic problem that precipitated these stenoses is still present after dilatation, proximal venous lesions tend to recur more quickly than anastomotic stenoses.

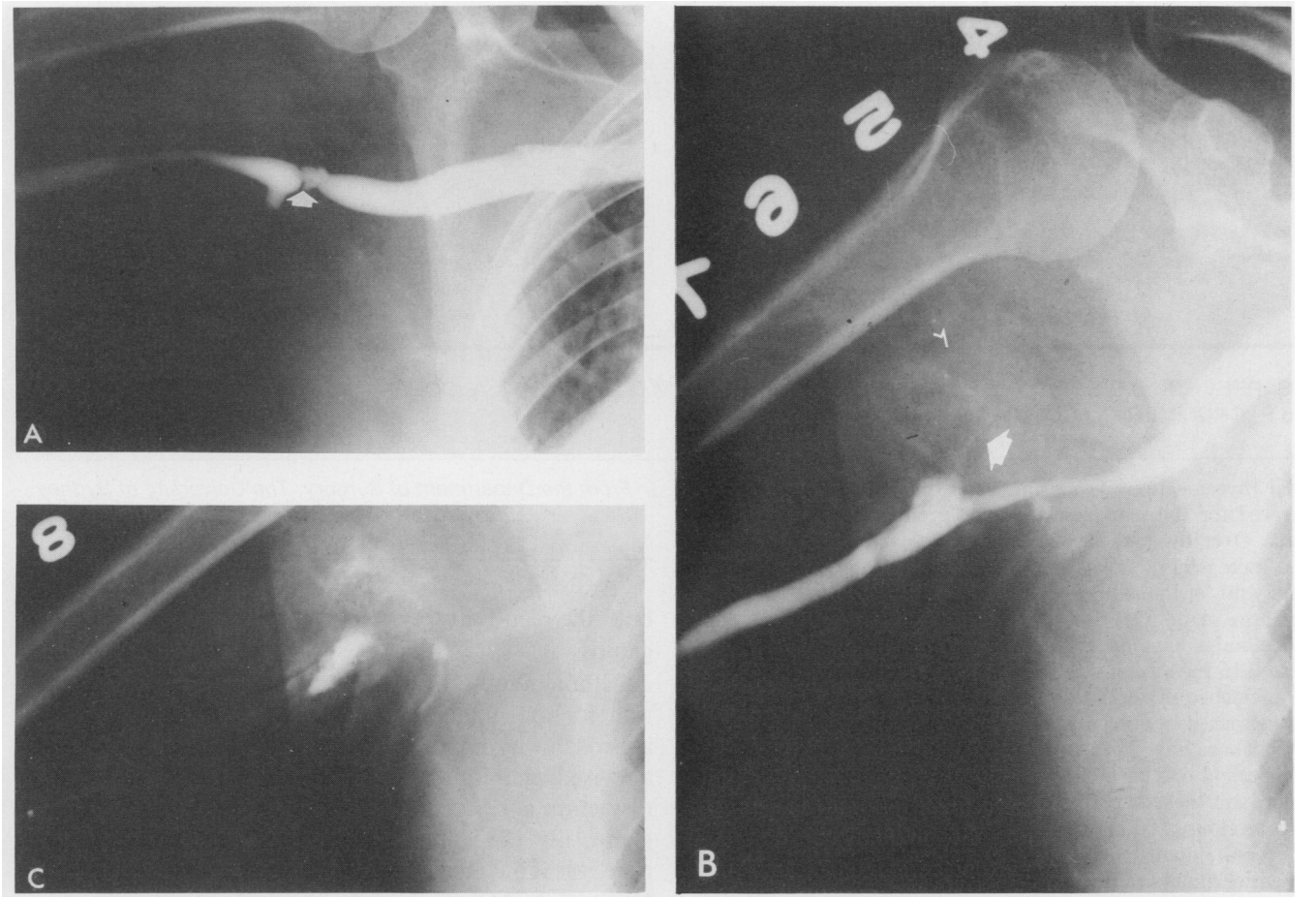
Venous stenosis and hypertension may cause other problems such as arm edema, enlarging venous aneurysms and varicosities, and enlarging graft pseudoaneurysm.[9,15] These clinical problems should alert the physician to a possible venous outflow obstruction and study should include the entire venous system to the subclavian vein. These problems can often be alleviated by angioplasty (Fig. 2).

Most angioplasties were technically not difficult and were performed on an outpatient basis. Once a graft has been incorporated into the subcutaneous tissues, puncture of the graft and introduction of a balloon catheter can be performed without difficulty. No significant problems have been encountered in removal of the catheter from the graft, and no pseudoaneurysms have occurred at the puncture site.

Complications included graft thrombosis (4%) and extravasation at the dilatation site (2%) (Fig. 3). Extravasation was controlled by compression of the fistula and a pressure dressing at the site of venous rupture. No patients required emergency surgery or blood transfusion.

It is clear that angioplasty, although initially effective, does not solve the long-term problem of recurrent venous outflow obstruction. However, angioplasty is safe and relatively simple and can be performed on an outpatient basis. A failed angioplasty or fistula thrombosis means the patient can still have the surgical correction that would have been done before angioplasty. Dilatation can be repeated numerous times to keep a fistula functioning for many years, often for the rest of the patient's life. Several patients have had multiple dilatations to keep a fistula patent for 4–5 years. We feel that any patient with a stenotic lesion less than 4 cm in length at or near a graft-to-vein anastomosis should have angioplasty before surgical repair. Expected patency rates at this location are 50% at 1 year and 32% at 2 years. Patients with proximal lesions in the axillary or subclavian vein, areas difficult to approach surgically, should also be treated primarily with angioplasty. Patients with a Brescia-Cimino type direct A-V fistula at the wrist who have a good radial artery and cephalic vein proximal to the stenosis may do better with a more proximal side-to-side direct fistula because of the high long-term patency of this type of vascular access.

Patients on chronic hemodialysis for more than several years are likely to have multiple surgical corrections of their vascular access. More aggressive angiography at the earliest sign of any problem coupled with angioplasty can help preserve many of these fistulas and decrease the hospitalization time and the number of corrections in this group of patients who have already had



FIGS. 3A–C. *A.* Patient with a GORE-TEX graft to basilic vein anastomosis who presented with high venous pressure during dialysis. There is a stenosis in the axillary vein caused by a hypertrophied valve (arrow). *B.* After angioplasty there is still narrowing of the axillary vein and the beginning of extravasation (arrow). *C.* A late film demonstrates contrast extravasation. The patient was treated by compression of the graft and a pressure dressing. The fistula was electively corrected.

and are still likely to have numerous surgical procedures during their lives.

References

1. Brescia MJ, Cimino JE, Appel K, Hurwich BJ. Chronic hemodialysis using venipuncture and a surgically created arteriovenous fistula. N Engl J Med 1966; 275:1089–1092.
2. Butt KMH, Friedman EA, Kountz SL. Angioaccess. Curr Probl Surg 1976; 9:5–67.
3. Jacobson JH, Haimer M. The modified bovine carotid artery as a vascular substitute in man. Personal experience and review of the literature. Isr J Med Sci 1975; 11:287–293.
4. Johnson JM, Kenoyer MR, Johson KE, et al. The modified bovine heterograft in vascular access for chronic hemodialysis. Ann Surg 1976; 183:62–66.
5. Butler HG, Baker LO Jr, Johnson JM. Vascular access for chronic hemodialysis: polytetrafluroethylene (PTFE) versus bovine heterograft. Am J Surg 1977; 134:791–793.
6. Baker LO Jr, Johnson JM, Goldfarb O. Expanded polytetrafluroethylene (PTFE) subcutaneous arteriovenous conduit: an improved vascular access for chronic hemodialysis. Trans Am Soc Artif Intern Organs 1976; 22:382–387.
7. Butt KMH. Blood access. Clin Nephrol 1978; 9:138–143.
8. Gilula LA, Staple TW, Anderson GN, Anderson LS. Venous angiography of hemodialysis fistulas. Experience with 52 studies. Radiology 1975; 115:555–562.
9. Glanz S, Bashist B, Gordon DH, et al. Angiography of upper extremity access fistulas for dialysis. Radiology 1982; 143:45–52.
10. Glanz S, Gordon DH, Butt KMH, et al. Dialysis access fistulas: treatment of stenoses by transluminal angioplasty. Radiology 1984; 152:637–642.
11. Lawrence PF, Miller FJ, Mineau DE. Balloon catheter dilatation in patients with failing arteriovenous fistulas. Surgery 1981; 89:439–442.
12. Probst P, Mahler F, Krneta A, Descoeurdres C. Percutaneous transluminal dilatation for restoration of angioaccess in chronic hemodialysis patients. Cardiovasc Intervent Radiol 1982; 5:257–259.
13. Tortolani EC, Tan EHS, Butchert S. Percutaneous transluminal angioplasty: an ineffective approach to failing vascular access. Arch Surg 1984; 119:221–223.
14. Glanz S, Gordon DH, Butt KMH, et al. Stenotic lesions in dialysis access fistulas. Treatment by transluminal angioplasty using high pressure balloons. Radiology 1985; 156:236.
15. Hashmonoi M, Schromek A, Szylman P, Auslaender L. Saccular aneurysm of the venous limb of an arteriovenous fistula complicating its use in chronic hemodialysis. Angiologica 1973; 10:294–298.