Stenosis and thrombosis in haemodialysis fistulae and grafts: the surgeon’s point of view

Volker Mickley

Department of Vascular and Endovascular Surgery, Stadtklinik Baden-Baden, Germany

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Introduction

Stenosis and thrombosis caused by stenosis are the most frequent complications of arterio-venous (a-v) access for haemodialysis. The well-known disadvantages and potential dangers of CVC for haemodialysis [1] should be sufficient reason for consequent access surveillance in order to early identify and treat every significant stenosis before thrombosis occurs. Immediate declotting of a thrombosed access with correction of any underlying stenosis in a way that the access can be used again for the next planned haemodialysis session is necessary to further reduce the need for CVC access. Pre-treatment CVC implantation should only be considered in patients with severe electrolyte disturbances or hyperhydration, when immediate haemodialysis is necessary.

Surgeons and interventional radiologists have developed valuable tools to cope with access stenosis and occlusion. However, it is not clear which treatment option should be applied to which clinical problem because comparative studies are scarce. Nevertheless, when there is little evidence it may be allowed to think logically: the basic aim of surgical and radiological intervention on a stenosed or thrombosed access, of course, is to restore its function. This does not only mean to restore sufficient a-v flow but also to preserve or to reconstruct the needleling segment, whenever and as durable as possible. Depending on the individual patient’s access problem, surgical and interventional treatment alternatives may be differently effective in achieving this aim.

Arterio-venous fistula thrombosis

Identification and treatment of the underlying stenosis are integral parts of any access declotting procedure. As only superficial (or superficialized) veins are used for fistula construction, in most of the patients inspection and thorough palpation will reveal a fibrous and stenotic vein segment as the cause of thrombosis. Additional colour-coded duplex-ultrasound may be helpful in adipose patients or otherwise questionable cases to identify the site(s) of stenosis and the extent of thrombosis. Because of their lack of side branches, cephalic or transposed basilic veins will thrombose up to their junctions with the brachial or axillary vein, whereas forearm fistulae often thrombose only up to the next patent side branch.

Treatment of a-v fistula thrombosis must aim at preservation of at least part of the puncture site. Only then can CVC implantation be avoided. Surgical and interventional methods are equally effective as far as clot removal is concerned. Location and extent of the underlying stenosis, however, must be taken into account in every individual patient in order to choose the best treatment modality. The following classification of fistula stenosis [2] is intended to help identify the adequate corrective procedure. It can, of course, also be applied in chronic stenosis without thrombosis.

Type I-stenosis (anastomotic venous stenosis)

About 80% of stenoses leading to occlusion of a-v fistulae are found at or close to the a-v anastomosis. Surgical dissection and mobilization of the vein on the one hand and the extremely turbulent flow within a functioning a-v connection on the other have been incriminated to be the causes.

After interventional decloting, a guide wire can be passed through the stenosis, and a percutaneous transluminal angioplasty (PTA) be attempted. In most cases these fibrotic stenoses necessitate high-pressure balloons and prolonged dilatation times. Long-term results of interventional treatment are so
immediate postoperative haemodialysis access. Distally to the interposition graft may be sufficient for 1 or 2 cm of untouched vein proximally or because larger veins. Comparative studies, however, are lacking. Only the diseased vein segment should be replaced, upstream the stenosis is <5 mm, and ePTFE in veins assisted by digital massage. On-table completion phlebography is mandatory to rule out residual clot and additional stenoses. In peripheral radiocephalic fistulae, distal ligation of the stenotic vein segment and proximal re-anastomosis of the cephalic vein to the radial artery is the easiest and most durable way of reconstruction. Proximal re-anastomosis is often difficult in type I-stenoses of brachiocephalic fistulae, when the vein must be extensively mobilized to bridge the greater distance to the artery. In these cases, a short interposition graft substituting the stenosed segment is the better alternative, because the needling segment of the access vein remains untouched.

Type II-stenosis (stenosis of the needling segment) Short stenoses within the needling segment or between two needling areas of the access vein might be late consequences of venous cannulations before fistula creation. Multiple or long stenoses possibly reflect fibrotic reactions of the vein wall to the repeat cannulations for haemodialysis. PTA is the only means to treat a puncture site stenosis and at the same time completely preserve the site for immediate haemodialysis access. Therefore, it should be attempted first. Sometimes fibrotic stenoses recur immediately after balloon deflation (elastic ‘recoil’). Stent implantation to maintain the result of PTA, however, should not be considered in the needling segment. Therefore, stenosis recoil, early failure after PTA, or repeated failures of PTA within short time intervals are indications for surgery.

In order to preserve as much as possible of the access site, short stenoses should be patched. In tortuous veins, resection of the stenosis and end-to-end anastomosis of the vein can be possible. The skin incision should be as short as possible to leave some untouched access vein for the next haemodialysis sessions. After wound healing, of course, the patched or re-anastomosed segment can be punctured again. Unfortunately, there are no studies comparing the patency rates of autologous and synthetic patches. The latter have at least the theoretical advantage that the venous capital of the ESRF patient is not further damaged by harvesting a segment of a superficial arm or a saphenous vein.

Multiple or long type II-stenoses should be bypassed. The author prefers a greater saphenous vein interposition graft when the calibre of the access vein upstream the stenosis is <5 mm, and ePTFE in larger veins. Comparative studies, however, are lacking. Only the diseased vein segment should be replaced, because 1 or 2 cm of untouched vein proximally or distally to the interposition graft may be sufficient for immediate postoperative haemodialysis access.

Type III-stenosis (junctional stenosis) In brachiocephalic fistulae, a stenosis of the vein at its junction with the axillary vein can be the reason for insufficient haemodialysis or for access thrombosis, because the upper arm cephalic vein in most individuals lacks sufficient collateral side branches. PTA of such a stenosis must be performed very carefully in order to avoid rupture. Once a rupture has occurred, a stent must be implanted reaching into the subclavian vein thus possibly causing axillary vein thrombosis and making later access grafts to the brachial or axillary vein impossible.

The surgical alternative would be dissection of the central part of the cephalic vein distally to its stenosis and transposing it to the central basilic or brachial vein. Of course, late stenosis of this anastomosis can occur. Comparative studies of PTA vs surgery for this rather infrequent problem are lacking.

Arterio-venous graft thrombosis Like in a-v fistulae clinical diagnosis of thrombosis prosthetic a-v access is based on the absence of thrill and murmur. Pre-operative clinical detection of stenosis in a clotted graft, however, is not that easy. Arterial and venous graft anastomoses lie deeply under scar tissue, so that palpation is unlikely to identify stenotic vessel segments. Once the graft is clotted neither colour-coded duplex-ultrasound nor arteriography or phlebography help to detect anastomotic stenoses. In consequence after surgical or interventional declotting, on-table angiography of the graft including both anastomoses together with the feeding artery and the draining vein is mandatory to define and immediately treat the cause of thrombosis.

Per definition patients with prosthetic a-v access for haemodialysis are those with exhausted peripheral veins. Treatment of graft stenosis therefore must not only aim at preservation of the graft as the puncture site but also at preservation of the patient’s already reduced venous capital. In contrast to surgery interventional procedures do not need (venous) graft material or more proximal (venous) anastomotic sites. These advantages make them an intriguing alternative for the treatment of the majority of graft stenoses. Again a simple classification of graft stenoses based on clinical considerations is proposed to help identify the adequate surgical or interventional procedure for every individual finding.

Type I-stenosis (arterial anastomotic stenosis) The arterial anastomosis of a graft almost always is a side-to-end anastomosis. Arterial anastomotic stenosis from intimal hyperplasia therefore will have a complex three-dimensional configuration involving the artery immediately upstream and downstream of the anastomosis, and the anastomotic graft segment. PTA of such a stenosis can be very difficult. Depending on the
access chosen for the intervention and depending on the angle in which the graft was sutured to the artery, one of the three areas of stenosis will be difficult or even impossible to traverse with guide wire and balloon catheter.

Surgical revision with resection of the anastomosis, reconstruction of the artery and re-anastomosis of the a-v graft to the artery is also demanding in small and diseased peripheral arm vessels but allows for complete correction of the stenosis.

Type II-stenosis (midgraft stenosis)
In some individuals with longstanding prosthetic vascular access, midgraft stenosis occurs due to excessive in-growth of fibrous tissue through multiple puncture holes. These stenoses can be dilated or curetted [7], thus leaving in place the cause of the problem, the partially destroyed graft. The more straightforward therapeutic option is to bypass the stenosed graft segment with a new prosthesis. If only a part of the access site must be replaced, the remaining old puncture site can be cannulated for haemodialysis. If the access site is completely stenotic, one-half should be curetted and the rest bypassed, just to avoid CVC implantation. When re-stenosis develops in the curetted graft segment, it can be bypassed at a later date, again without the need for a dialysis catheter.

Type III-stenosis (venous anastomotic stenosis)
The great majority of grafts occlude due to progressive stenosis of the venous anastomosis. The combination of surgical trauma to vein wall and endothelium during graft implantation, compliance mismatch between graft and vein, and flow disturbances in the anastomotic area is believed to be the cause.

Although the graft-to-vein anastomosis frequently is sutured in an end-to-side fashion, haemodynamically it is an end-to-end anastomosis (provided the vein valves distally to the anastomosis are competent). Therefore, PTA of the stenosis is simple and should be attempted first, because it is the most vein-preserving treatment modality, although surgical thrombectomy and graft revision has been shown to be more effective in most controlled trials [8]. In short stenoses the surgical alternative would be patch angioplasty. Stenoses longer than 5 cm or complete occlusions of the draining vein afford graft extension to a more proximal vein segment [9,10]. Graft extension should also be considered in early or repeat re-stenoses.

Conclusions
Despite the significant lack of comparative studies, a rationale of differential indications for surgical and radiological interventions on stenosed and thrombosed a-v access can be presented based on the primary aim of access revision: the optimal restoration of morphology and function. The proposed classification of fistula and graft stenosis is meant to help the nephrologist to refer each patient with a readily identified access stenosis to his respective surgical or radiological partner providing the best treatment option for the individual problem.

Conflict of interest statement. None declared.

References