

## 7. Treatment of stenosis and thrombosis in AV fistulae and AV grafts

Guideline 7.1. For venous outflow stenosis percutaneous transluminal angioplasty (PTA) is the first treatment option (Evidence level III).

Guideline 7.2. Thrombosed autogenous and graft fistulae should be treated either by interventional radiology or surgery. Individual centres should review their results and select the modality that produces the best results for that centre (Evidence level III).

### Management of autogenous AV fistula stenosis

#### *Relevant stenosis*

Stenoses should be treated if the diameter is reduced by >50% and is accompanied with a reduction in access flow or in measured dialysis dose. Other indications for stenosis treatment are difficulties in cannulation, painful arm oedema, prolonged bleeding time after cannulation or after removal of the cannulae (due to high venous pressure) and handischaemia due to arterial inflow or distal stenoses. A stenotic lesion, due to intimal hyperplasia, is the most common cause for low access flow. In RCAV fistulae, 55–75% of these stenoses are located close to the AV anastomosis and 25% in the venous outflow tract [1,2]. In brachial-cephalic and/or basilic AV fistulae, the typical location (55%) is at the junction of the cephalic with the subclavian vein and the basilic with the axillary vein, respectively [1]. An arterial inflow stenosis >2 cm from the anastomosis is uncommon, but may endanger the flow in the AV fistula.

#### *Stenosis of the anastomotic area*

Surgical treatment is indicated in stenoses of the anastomotic area located in the lower forearm. Alternatively, PTA is possible although its results are likely to be less long-lasting. Primary interventional treatment is indicated in stenoses of the anastomotic area located in the upper forearm and in the upper arm. Surgery should be considered in cases of early or repeated recurrences of the lesions. Dilatation or surgical revision of anastomotic stenoses in upper arm fistulae can cause steal syndrome and access-induced hand ischaemia. Careful dilatation up to 5 or 6 mm initially is recommended. Dilatation to >6 mm is rarely indicated.

#### *Venous outflow stenosis*

PTA is the first treatment option in the outflow veins (cephalic/basilic) [3]. Junctional stenoses, of the superficial veins with the deep venous system, can also be

treated by PTA. If a stent is placed in the final arch of the cephalic vein, it must not protrude into the subclavian vein where it could induce stenosis and preclude future use of the distal (basilic, brachial and axillary) veins [4].

#### *Balloon angioplasty*

In order to visualize the stenoses, angiography is performed by retrograde puncture of the brachial artery, in case of anastomotic problems, or by direct antegrade puncture of the vein above the anastomosis if an outflow problem is suspected [5]. It is controversial, whether long-segment stenoses should be treated radiologically or surgically. While some authors recommend surgical intervention [6], either by graft interposition [7] or vein transposition, others recommend radiological intervention [8]. Studies proving the superiority of one of the two treatment options for the treatment of long-segment obstruction are not available. However, PTA of short-segment stenoses (<2 cm) has a better outcome compared with long-segment stenoses (>2 cm) [9].

#### *Persistent stenosis*

Some stenoses cannot be dilated by conventional balloon angioplasty. These ‘hard’ stenoses can be treated with cutting balloons or ultrahigh pressure balloons (up to 32 atm) [10,11].

#### *Recurring stenosis*

Recurring stenosis can be treated radiologically, with or without stent placement, or surgically [5]. The strategy for treatment should be made considering the individual condition of the patient in relation to the invasiveness of the surgical treatment. In spite of complete opening of the PTA balloon of sufficient diameter, the dilated vessel wall may collapse immediately after removal of the balloon. This elastic recoil can be prevented by stent implantation, especially in central veins [12]. Stent placement in the needling areas of forearm fistulae should be avoided except for PTA-induced ruptures not controllable by protracted balloon inflation.

#### *Management of autogenous AV fistula thrombosis*

Fistula thrombosis should be treated as soon as possible or within 48 h. The duration and site of AV fistula thrombosis as well as the type of access are important determinants of treatment outcome. Timely declotting allows immediate use without the need for a central venous catheter. Thrombi become progressively fixed to the vein wall, which makes surgical removal more difficult. Thrombosis may affect the

post-anastomotic vein segment as result of anastomotic stenosis or may begin at the needle site. When the clot is localized at the anastomosis in radial-cephalic and brachial-cephalic fistulae, the outflow vein may remain patent due to the natural side branches that continue to carry venous blood flow. In these accesses it is possible to create a new proximal anastomosis [7,13]. Thrombosis in transposed basilic vein fistulae usually leads to clot propagation of the entire vein. Although comparative studies are missing, the available literature [4,5,14–22] suggests that thrombosed autogenous AV fistulae should, preferably, be treated by interventional radiology. The single exception may be forearm AV fistulae, thrombosed due to anastomotic stenosis. It is likely that in such cases, proximal re-anastomosis will provide good results.

#### *Interventional thrombolysis*

Thrombolysis can be performed mechanically or pharmacomechanically [23–25]. While the immediate success rate is higher in grafts than in autogenous AV fistulae (99 vs 93% in forearm fistulae), the primary patency rate of the forearm AV fistula at 1 year is much higher (49 vs 14%). One year secondary patency rates are 80% in forearm and 50% in upper arm AV fistulae, respectively [14]. In AV fistulae, the combination of a thrombolytic agent (urokinase or tissue plasminogen activator = tPA) with balloon angioplasty resulted in an immediate success rate of 94%. Liang et al. [21] reported a success rate of 93% and a primary patency rate at one year of 70%. Haage et al. [4] performed 81 percutaneous treatments of thrombosed AV fistulae. Flow restoration was achieved in 88.9% of the AV fistulae. The primary 1-year patency rate was 26% and the secondary 1-year patency rate 51%.

#### *Surgical thrombectomy*

Surgical thrombectomy is performed with a thrombectomy catheter (Fogarty). Manual retrograde thrombus expression can be helpful. On-table venous outflow angiography of the recanalized vein as well as the central veins should be performed whenever possible to find/exclude additional stenoses or residual thrombus. Identification and concurrent correction of the underlying cause(s) of thrombosis are essential parts of any surgical or interventional de clotting. The best results of surgery probably will be encountered after proximal re-anastomosis for anastomotic stenosis of forearm AV fistulae, which is the most frequent location of stenosis in this type of access. Primary patency of the new proximal anastomosis has been reported to be as high as 80% at 1 year and 67% at 2 years [13]. If access failure recurs frequently in a short time period, a new fistula may need to be created.

## **Management of AV graft stenosis**

A diameter reduction of >50% of the lumen together with a significant flow decline is considered as an indication for treatment [26].

#### *Stenosis at the arterial anastomosis*

As in autogenous fistulae, most arterial inflow stenoses in grafts can successfully be treated by PTA [27]. Stenosis of the arterial anastomosis itself can be dilated, if only the afferent artery and the graft at the anastomosis are affected and there is no stenosis in the efferent artery. If there is an additional stenosis of the efferent artery, angioplasty of the anastomosis alone will enhance graft flow with the risk of peripheral ischaemia due to reduced peripheral arterial perfusion. In these patients, either dilatation of the efferent artery by interventional radiology or through surgical revision of the anastomosis may resolve the dilemma.

#### *Intra-graft stenosis*

Intra-graft (or mid-graft) stenoses are found in the cannulation segment of grafts. They result from excessive ingrowth of fibrous tissue through puncture holes. These stenoses can be treated by PTA [28], graft curettage [29], or segmental graft replacement. When only a part of the cannulation segment is replaced, the access can be used for haemodialysis without the need of a central venous catheter. When re-stenosis occurs in a non-exchanged part of the graft, this can be replaced after healing of the new segment.

#### *Stenosis at the venous anastomosis*

The most common cause for graft dysfunction and thrombosis is venous anastomotic stenosis [28,30,31]. Since grafts should be implanted only in patients with exhausted peripheral veins, vein-saving procedures like PTA or patch angioplasty should be favoured to graft extensions to more central venous segments, even though the latter may have superior patency rates. When PTA repeatedly fails, additional stent implantation should be considered [2,32,33].

When a stent or a patch fail, graft extension is still possible. This staged therapy improves cumulative graft function. In 20–30% of the grafts, PTA does not increase blood flow to >600 ml/min, indicating insufficient dilatation with an undersized balloon, immediate recurrence of stenosis, or the existence of an unidentified and not corrected stenosis either more centrally or at the arterial inflow.

## **Management of AV graft thrombosis**

Graft thrombosis should be treated without unnecessary delay and within 48 h, at least before the next

dialysis session. Early declotting allows for immediate use of the access without the need for a central venous catheter [34–53]. There is always a compact “arterial plug” present. Old thrombi (> 5 days) are often fixed to the vessel wall beyond the venous anastomosis, making surgical extraction more difficult. This is less of a problem for the interventional radiological treatment.

### *Surgical thrombectomy*

Surgical thrombectomy is performed with a thrombectomy catheter. On-table angiography should be performed after completion of the arterial and venous limbs of the graft. This should visualize the central venous outflow as well as the graft. It is required to exclude residual thrombi and define the cause of thrombosis. Identification and simultaneous correction of the underlying stenosis are integral parts of any surgical or interventional declotting procedure [30,31].

### *Interventional thrombolysis*

Prosthetic graft thrombosis can be treated with various percutaneous techniques and tools, including combinations of thromboaspiration, use of thrombolytic agents such as tissue plasminogen activator (tPA), mechanical thrombectomy and mechanical thrombectomy devices. An initial success rate of 73%, with primary patency rates of only 32 and 26% at 1 and 3 months, respectively, are reported [36–54]. Smits et al. [55] have compared different mechanical devices for percutaneous thrombolysis and concluded, that the treatment of the underlying stenoses was the only predictive value for graft patency. Each centre should, therefore, choose the technique according to their expertise. Independent of the applied technique it is important to perform thrombolysis as soon as possible to avoid the need for a central venous catheter and as an outpatient procedure to decrease costs, whenever possible. Post-procedural angiography to detect and correct inflow, intra-access or venous outflow stenosis is mandatory.

### **Recommendations for further research**

Development of better catheter and balloon designs and (drug-eluting) stents may improve the outcome of interventional access treatment.

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