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 (Fogarthy). 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 declotting. 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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8. Diagnosis and treatment of central venous obstruction

Guideline 8.1. If symptomatic central venous obstruction is suspected, angiography of the access and complete venous outflow tract should be performed (Evidence level III).

Guideline 8.2. Treatment should be performed by percutaneous intervention (Evidence level III).

Diagnosis of central venous obstruction

Chronic swelling of the access arm is the most important clinical sign of central venous obstruction [1]. The superficial veins may become prominent (collaterals). Pain and paraesthesia may occur. Central venous lesions have to be treated when they are severe and disabling such as those resulting in arm swelling, troublesome pain or inadequate haemodialysis [2]. In obvious central venous obstruction, angiography of the access and complete venous outflow tract must be performed, since the central veins cannot be examined with ultrasonography. Thus, to completely visualize all mediastinal veins, venography using digital subtraction technique is needed [3]. This can be done preferably with direct antegrade puncture of the access [4]. In the majority of patients central vein obstruction is due to previous inserted central vein catheters. In 40% of the patients with subclavian vein catheters central venous obstruction develops, compared with 10% of patients with jugular vein catheters. In patients without a history of central venous catheterization, other causes, such as extrinsic compression of mediastinal veins (e.g. lymphoma, goitre, thoracic aortic aneurysm, mediastinal fibrosis, pacemakers), hypercoagulopathy, thoracic outlet syndrome or pacemaker wires should be considered. In these cases plain X-rays, computed tomography or MR imaging may be helpful for the differential diagnosis. If treatment of the underlying disease is not possible or fails to resolve arm swelling, PTA with stent insertion is indicated [5].

Management of central venous obstruction

Interventional treatment

In the last decade, several studies of patients treated with PTA alone have been published. Primary patency rates of $\leq$10% at 1 year and numerous restenoses were reported [6–8]. Stent implantation has clearly been shown to improve primary 1-year patency rates to 56% and more [1,4,6,9]. Regular follow-up and reinterventions are required to maintain patency and achieve long-term clinical success [10]. These figures do not differ significantly from those of surgical intervention [1,6,9]. Nevertheless, due to the invasiveness of surgery for central venous obstructions and the less invasive interventional therapy, PTA with or without stent implantation is recommended as primary option for treatment [4]. Reports show, that symptomatic central venous obstruction in dialysis patients can be treated with a high success rate through radiological intervention [11,12]. Stent placement should avoid overlapping the ostium of a patent internal jugular vein to achieve a safe and sufficient result, since this latter vein is essential for future placement of central venous catheters. Similarly, a stent placed in the innominate vein should not overlap the ostium of the contralateral vein, otherwise contralateral stenosis may occur and preclude future use of the contralateral limb for access creation [4]. Little data are available on the use of thrombolytic agents in central venous thrombosis. It is, therefore, not recommended as a primary treatment regimen.

Surgical treatment

When interventional treatment of central venous obstruction is impossible or fails, assessment of the patient is necessary to define the most effective surgical method and to guarantee long-term vascular access. Surgical evaluation focuses on the general risk (see ASA Physical Status Classification System [13]) and life expectancy as well as on the vascular pathology. If surgery is an option, all angiograms have to be re-evaluated. If an ipsilateral surgical bypass to the jugular vein is impossible due to innominate vein obstruction, additional venography of the contralateral arm should be performed to assess whether a new access can be constructed in that arm or a subclavian–subclavian or subclavian–jugular cross-over bypass should be performed [14–16]. In case of bilateral obstruction of the mediastinal veins, including the superior caval vein, ultrasonography of ilio-caval veins is indicated in the planning of arterio-venous thigh access. Alternative surgical options for upper extremity vascular accesses with compromised venous outflow, are axillo-saphenous/iliac or right atrial bypasses [17,18].

As ultimate treatment access ligation can be considered, which will relief local symptoms.

Recommendations for further research

Improvement of central venous catheter design, may probably prevent vessel wall damage and the development of central venous stenoses. Stent improvement and newer guidewires may enhance central venous obstruction intervention and outcome.

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9. Diagnosis and treatment of access-induced ischaemia

Guideline 9.1. Access-induced ischaemia should be detected by clinical investigation and the cause should be identified by both non-invasive imaging methods and angiography (Evidence level III).

Guideline 9.2. Enhancement of arterial inflow, access flow reduction and/or distal revascularization procedures are the therapeutic options. When the above methods fail, access ligation should be considered (Evidence level II).

Rationale

Access-induced upper extremity ischaemia is a serious complication that, when not treated in time may lead to major amputation [1] From published series, it can be estimated that the incidence of symptomatic ischaemia varies from 2% to 8% of the haemodialysis population [2,3]. Elderly patients, diabetics and patients with peripheral and/or coronary arterial obstructive disease are more prone for the development of access-induced ischaemia. In addition, previous ipsilateral vascular access increases the risk. Access-induced ischaemia occurs more often in proximally located fistulas [4]. These high-flow AVFs induce a steal phenomenon with lowering of distal perfusion pressures and, when collateral circulation is inadequate, symptoms may occur [5–8]. A grade 1–4 classification for access-induced ischaemia (grade 1: pale/blue and/or cold hand without pain, grade 2: pain during exercise and/or HD, grade 3: ischaemic pain at rest and grade 4: ulceration, necrosis and gangrene) can be used to outline the severity of the disease and this ranges from minor symptoms to finger necrosis. A number of these patients have increasing pain during dialysis treatment. For grade 1 and 2 ischaemia a conservative treatment is indicated, while with grade 3 and 4, interventional treatment is indicated [9].

Diagnosis of access-induced ischaemia

Physical examination, including observation and palpation of peripheral vessels, may be inadequate and misleading for the diagnosis of symptomatic ischaemia. Additional non-invasive testing with measurement of digital pressures and calculation of the digit-to-brachial index (DBI), transcutaneous oxygen determination, ultrasonography of forearm arteries and access blood flow measurement are important steps in the diagnosis and decision-making process [10,11]. Finally, angiography with visualization of the upper extremity arterial tree from the proximal subclavian artery to the distal palmar arches with and without AVF compression to enhance distal flow, is obligatory to outline the strategy for treatment and to determine whether interventional or surgical options are preferred [12].

Management of access-induced ischaemia

The options for treatment depend on the aetiology of the ischaemia: inflow arterial obstruction and/or distal arterial lesions are recanalized with small-calibre balloons and stent implantation [13–15], high-flow AVFs, as mainly observed in patients following successful renal transplantation are eligible to flow-reducing procedures like banding and distal arterial extension [16–18]. Steal in itself may be cured by ligation of the artery distal of the arteriovenous anastomosis [19]. In most patients it is necessary to add a saphenous vein graft bypass to the forearm arteries (DRIL = distal revascularization + interval ligation). The results of these procedures are usually good with relief of symptoms and preservation of the access site (Table 1) [20–27]. A simple alternative of the DRIL procedure is the PAVA (proximal arteriovenous anastomosis) technique, in which the AV anastomosis at the elbow is disconnected and moved to the axilla by means of a graft interposition [28,29]. Intra-operative digital pressure measurement or transcutaneous oxymetry ($TcPO_2$) is mandatory to guarantee an adequate surgical intervention with acceptable outcome. A digital-brachial pressure index >0.60 or $TcPO_2$ of >40 mm Hg is indicative of a sufficient distal hand perfusion [30–32]. The same DBI threshold may be also predictive for the development of ischaemia in predialysis patients receiving new vascular access [33–35]. In some patients, AVF ligation and change in renal replacement modality (to continuous ambulatory peritoneal dialysis = CAPD) or transition to central venous catheter access, may be the only solution.

Prevention of access-induced ischaemia

An adequate preoperative evaluation and surgical technique are the keystones to avoid ischaemia. Physical examination of peripheral pulses, bruits, and measurement of bilateral arm blood pressures are essential for the work-up before AVF creation. Duplex ultrasonography is very useful in the assessment of not only superficial veins but also arteries. Preoperative measurement of digital pressures may be helpful to indicate patients at risk for ischemia. Patients with preoperative digit-to-brachial indices (DBI) <1.0 are more likely to develop steal, but there is no DBI threshold below which steal is inevitable. If there is any doubt concerning the status of the peripheral circulation, angiography or MRA is advised. Steal is more likely in patients undergoing brachial-based arteriovenous fistulae than in those receiving prosthetic grafts.

Table 1. Results of distal revascularization and interval ligation (DRIL) procedure for the treatment of access-induced ischemia

Author	No. of patients	Success in %	AVF patency (%)
Schanzer *et al.* [20]	14	93	82
Haimov *et al.* [21]	23	96	73
Katz *et al.* [22]	6	83	100
Berman *et al.* [23]	21	100	94
Lazarides *et al.* [3]	7	94	–
Stierli *et al.* [24]	6	100	100
Knox *et al.* [25]	52	90	83
Diehl *et al.* [26]	12	100	100
Sessa *et al.* [27]	18	73	94

Therefore, a limited length of the arteriovenous anastomosis of 10 mm in radial-cephalic and 5–7 mm in graft and/or brachial-cephalic/basilic AVFs, may contribute to the prevention of large volumes of blood shunting through the AVF. Either a "non-smooth" anastomosis (90° or 180° angle) adds to a greater anastomotic resistance and thus limitation of flow.

Recommendations for further research

Further search for pre-operative indicators that outline the risk on post-operative ischaemia may help to take adequate measures for prevention.

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10. Central venous access

Guideline 10.1. Central venous catheters should be inserted as a last resort in patients without a permanent access and the need for acute haemodialysis (Evidence level III).

Guideline 10.2. The percutaneous route should be used for both acute and chronic catheter insertion. Insertion should be guided by ultrasound. A plain X-Ray (chest or abdomen) should be performed before use to locate catheter and detect any complication (Evidence level II).

Guideline 10.3. The right internal jugular vein is the preferred location for insertion (Evidence level II).

Guideline 10.4. Non-tunnelled catheters should only be used in emergency situations and should be exchanged as soon as possible for tunnelled catheters (Evidence level III).

Indications for catheter insertion

Central venous catheter insertion is required in incident CKD-stage 5 patients who need to start dialysis in an acute or emergency situation, and are not equipped with a permanent vascular access [1,2]. Catheter insertion is also indicated in prevalent CKD-stage 5 patients on renal replacement therapy presenting with vascular access failure [3], and waiting for interventional or surgical access salvage or the creation of a new access. In some patients, all surgically created arteriovenous vascular access options may have been exhausted. A central venous catheter may then represent the only access option. Some patients have a contraindication for the creation of an arteriovenous fistula (severe cardiac failure, chronic respiratory insufficiency) [4], because of the risk of heart failure.

Patients with severe pain in the hand due steal syndrome, causing peripheral ischaemia, or with major difficulties in needling [5], may also benefit from a permanent central venous catheter. Catheters offer immediate vascular access for haemodialysis and may be used over several months or years. Long-term catheters also have positive properties: they are easy to use and do not need maturation.

Technique of catheter insertion

Catheter insertion is considered a high-risk intervention which deserves careful attention, must be performed under strict aseptic conditions and should ideally be performed by trained and senior physicians. Percutaneous catheter insertion is the preferred method for catheter insertion. The right internal jugular vein is the first option for insertion, followed by the left internal jugular vein. The femoral route is preferred for short-term catheters (<1 week) since there is no risk for central vein stenosis. Ultrasound-guided insertion technique is mandatory to prevent accidental carotid artery puncture and to ensure successful cannulation [6,7]. In addition, fluoroscopy to follow and locate the position of the guide wire is advisable. In a recent study 60 patients were randomized for ultrasound guided *vs* 'blind' catheter insertion. First attempt venous cannulation success rate was 56.7% compared with 86.7% in non-guided *vs* guided insertion technique. The risk of adverse outcome was significantly greater in the blind procedure ($P = 0.020$). The ultrasound-guided procedure for internal jugular vein catheter insertion using an ordinary ultrasound machine was significantly safer and more successful as compared with the blind technique [8]. For patients presenting with acute and/or life-threatening conditions requiring immediate dialysis (pulmonary oedema, hyperkalaemia, respiratory distress) the femoral vein is the most favourable insertion site. Because of the high risk on central venous stenosis (see Guideline 8), the subclavian vein route has been abandoned [9].

Catheter performance and care

Catheter performance (maximum flow rate, blood resistance and recirculation) should comply with delivery of adequate dialysis dose without altering treatment schedule (frequency, dialysis duration) [10,11]. Tunnelled catheter morbidity (dysfunction, thrombosis, infection) is significantly reduced compared with non-tunnelled catheters and tunnelled catheters should be preferred in all patients [12]. Port-catheter devices (Dialock, LifeSite) offer comparable flow performances to long-term catheters while improving patients' aesthetic satisfaction and improving patients comfort [13,14]. Unfortunately, the risk on infection is high with these devices. Catheter care and handling conditions under aseptic manipulation are essential to prevent infection in catheter and venous port devices.

Recommendations for further research

Improvement of catheter design and locking solutions are major subjects for further research.

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11. Management of central venous access complications

Guideline 11.1. Catheter dysfunction should be corrected by local fibrinolysis designed to restore flow patency. Repetitive catheter dysfunction requires local fibrinolysis with additional catheter imaging, microbiological assessment and systemic coagulation evaluation (Evidence level III).

Rationale

Catheter dysfunction is a relatively common event for haemodialysis patients. It reduces the effective blood flow rate and reduces dialysis dose. Catheter dysfunction may be minimized by using the appropriate material, a perfect insertion technique [1] and strict protocols for catheter care [2]. Catheter design and material are essential for achieving high blood flow and adequate performance [3,4]. Tunnelled catheters provide usually higher flows (up to 400 ml/min) at low resistance and reduced recirculation compared with non-tunnelled catheters. Dual catheters with independent lines and side holes at the tip provide higher flows than dual-lumen catheters [5,6]. Catheters inserted in the right internal jugular vein offer the best flow compared with other central venous sites [7]. Catheter dysfunction must be detected and corrected early in order to restore blood flow and dialysis dose. Catheter dysfunction may occur in different ways:

(i) Complete obstruction, making dialysis impossible.
(ii) Incomplete obstruction (endoluminal fibrin deposits restricting catheter lumen or obstructing catheter side holes at the tip, external fibrin sleeves surrounding catheters) resulting in inadequate flow and/or excessive extracorporeal blood pressure alarms during the dialysis session. Depending on the location of the fibrin clot (arterial and/or venous line), there may be high negative arterial pressure (obstruction at the arterial catheter line) or high positive venous pressure (obstruction at the venous catheter line).

Catheter care and handling are very important to prevent catheter dysfunction. Prevention of catheter clot formation in the catheter tip during the interdialytic period is crucial. This may be achieved by installing an antithrombotic lock solution (standard heparin, low molecular weight heparin, sodium citrate) [8,9]. A certain amount of the antithrombotic lock solution may leak into the circulation via side- and central catheter holes. Loss of antithrombotic locking solution facilitates catheter clot formation while it increases the haemorrhagic risk. Regular use of low dose of antithrombotic drugs such as coumarin derivatives or antiplatelet agents in dialysis patients have failed to improve catheter outcomes [10–12].

Catheter performance monitoring is required to detect catheter dysfunction. Such monitoring is an integral part of the quality assurance process to ensure dialysis efficacy and to reduce catheter-related morbidity [13]. It relies on markers evaluating catheter flow performances such as estimations of effective blood flow rate, venous and arterial pressure values at constant flow, recirculation and dialysis dose delivery as measured by Kt/V [14].

Catheter maintenance is important to achieve the prescribed blood flow during dialysis sessions. To prevent and/or to correct catheter dysfunction it is recommended to clean the catheter lumen periodically by applying fibrinolytic agents (urokinase, tPA) either as lock solution or continuous infusion on both arterial and venous lines [15]. Occluded catheters are reopened either by means of a mechanical method (brush) or pharmacological method (urokinase, tPA) [16–18]. Removal of the fibrin sleeve may be achieved either by lasso wire stripping or by infusing a fibrinolytic solution (urokinase, tPA), during 3–6 h [19]. Alternatively, the catheter may be exchanged over a guidewire [20].

Recommendations for further research

Investigation into better thrombolytic agents and mechanical tools to declot thrombosed catheters are of importance.

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12. Management of the infected vascular access

Guideline 12.1. Infection of autogenous AV fistulae without fever or bacteraemia should be treated by appropriate antibiotics for at least 2 weeks (Evidence level III).

Guideline 12.2. Infection of autogenous AV fistulae with fever and/or bacteraemia should be treated by appropriate antibiotics given intravenously for 2 weeks. Excision of the fistula is required in case of infected thrombi and/or septic emboli (Evidence level IV).

Guideline 12.3. Infected graft AVFs should be treated by appropriate antibiotics given intravenously for 2 weeks and continued orally for 4 weeks. Depending on the presence of bacteraemia and/or infected thrombi segmental explantation of the graft with bypass needs to be considered (Evidence level III).

Guideline 12.4. Anastomotic infection is an indication for total graft explantation (Evidence level II).

Guideline 12.5. Catheter removal must be considered when catheter infection is suspected. Immediate removal should be performed in non-tunnelled catheters when infection is diagnosed (Evidence level III).

Guideline 12.6. In tunnelled catheters with a short febrile and/or bacteraemic reaction, a delayed removal may be considered (Evidence level III). In septicaemia, immediate removal should be performed in tunnelled catheters as well.

AVF and prosthetic graft infection

Infection of autogenous AVF usually responds well to appropriate antibiotics given either orally or intravenously according to the presence of fever and/or bacteraemia. Surgical revision or excision of the fistula is required when infected thrombi, aneurysms and/or septic emboli are detected. Infection of graft AVFs is two to three times more frequent than autogenous AVFs [1]. Infection of the graft bears a worse prognosis and requires usually a surgical revision and/or explantation in addition to the antibiotic therapy. Salvaging prosthetic grafts may be attempted in certain circumstances. Several surgical techniques have been described in combination with antibiotic therapy. For localized abscesses, incision and drainage with graft preservation is needed. For more severe infection, such as infected thrombi, false aneurysms, cellulitis, explantation of the infected graft segment and segmental bypass with a new graft is indicated. However, these salvaging techniques may be complicated because of local or generalized infection and sepsis. Therefore, in severe cases a complete explantation of all graft material with drainage is usually necessary.

Central venous catheter infection

Catheter-related infection is the major cause of morbidity in HD patients with central venous catheters [2–4]. Catheter infection is a potentially severe event that requires early diagnosis and appropriate management to prevent further complication. Diagnosis of catheter infection is relatively easy in symptomatic patients presenting with fever, pain, skin exit and/or track infection and bacteraemic episodes. It is much more difficult in silent catheter endoluminal contamination or low grade infection. In these cases, only specific blood and catheter clot culture will help to make the diagnosis [5]. Recently, it was shown that catheter clot culture after endoluminal brushing was more sensitive than blood culture to identify asymptomatic catheter infection (catheter contamination) [6,7]. Symptoms of infection includes chronic fever, bacteraemic episodes, catheter pain, inflammation of the exit site or tunnel. Infection of the catheter exit site or tunnel tract is usually observed by the dialysis nurse while clinical examination is performed at the time of dialysis connection. Silent contamination is suspected when recurrent febrile reactions during haemodialysis occur and bacterial pathogens (*Staphylococcus aureus, S. epidermidis* or other bacteria such as Gram-negatives) are identified in blood cultures. Catheter-related septicaemia is usually associated with symptoms of endocarditis, arthritis, spondylarthritis or osteomyelitis.

Specific blood markers (leucocyte count and differentiation), C-reactive protein (CRP) and procalcitonin (PCT), help to diagnose early bacterial catheter infection. Catheter-related infection should be considered as a severe and potentially lethal complication. Prevention of infection should be a permanent preoccupation for care providers, that relies on hygienic measures [8] and strict protocols for handling catheters based on aseptic manipulation [9] and using specific dressings [10]. The regular and pre-emptive use of locking solutions (Citrate) with both antithrombotic and/or antiseptic properties has been confirmed to be effective in preventing catheter infection [11–14]. The topical application of antibiotic ointment on the skin exit site has proved to be efficient in reducing the incidence of bacteraemia at the expense of selecting antibiotic-resistant strains of bacteria [15–17]. The use of antibiotic-coated catheters or silver-treated catheters has been proposed to reduce the risk of infection, but conflicting results has been reported [18–20]. Identification of patients at risk of infection is particularly important in diabetic patients and nasal

carriers of methicillin-resistant *S. aureus* (MRSA). In the latter patients, eradication of bacteria by means of topical antibiotic ointment has been associated with a significant reduction of bacteriaemias [21,22].

Catheter removal should be considered as the first line of treatment. Catheter withdrawal must be immediate when infection occurs in non-tunnelled catheters. Removal may be postponed for several days in tunnelled catheters. When this last option is applied the risk of septic complications of delayed catheter removal should be balanced with the benefits of keeping it *in situ*. This conservative option implies that the patient is regularly and carefully observed. In addition, the catheters should be disinfected by means of antimicrobial lock solutions and dissemination of the infection must be prevented by adequate systemic antibiotic therapy. When the catheter is left in place and in the absence of precise microbial information, antimicrobial therapy should include systemic antibiotic therapy effective against Staphylococcus species plus an adjunctive antimicrobial catheter lock. Antibiotic therapy is given for 2 weeks in order to sterilize all potential bacterial foci. Topical antibiotic therapy (catheter exit site) is initiated when there is associated local infection. Imaging techniques may help to diagnose catheter-related infection. Ultrasound doppler methods can detect tunnel infection and/or subcutaneous abscesses along the catheter track. Phlebography and catheterography are indicated to diagnose infected thrombi located in the vein or fibrin sleeves surrounding the catheter tip. Isotopic imaging techniques using positron emission tomography (PET) may help to identify infected venous catheters and port devices [23].

Recommendations for further research

Improvement of needle design and education on strict aseptic cannulation techniques may possibly lower the incidence of infection in fistulae and grafts. Antibiotic-bonded grafts may possibly lower the incidence of graft infection. Newer catheter designs and locking solutions are important issues for further investigation of the prevention of central venous catheter-related infections.

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