Interventional Nephrology: Catheter Dysfunction—Prevention and Troubleshooting

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Summary
Despite recommendations by various national guidelines advocating arteriovenous fistulae as the access of choice in patients undergoing hemodialysis (HD), the use of central venous catheters (CVCs) remains widespread among both incident and prevalent HD patients. Unfortunately, long-term use of CVCs is fraught with complications, which are a major cause of morbidity and death in this patient population. Complications include a high rate of infections, as well as thrombus and sheath-related mechanical dysfunction. This review addresses prevention and management of noninfectious catheter-related dysfunction.


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Introduction
Vascular access dysfunction is a major cause of morbidity in patients undergoing hemodialysis (HD), and the need to provide a suitable vascular access is an ongoing challenge. Although HD is the predominant modality for renal replacement therapy in patients with ESRD in the United States (1), multiple comorbid conditions affecting the vasculature, combined with an aging population, have led to a dearth of suitable options for vascular access (2). National guidelines recommend the use of autogenous arteriovenous fistulae followed by prosthetic arteriovenous grafts (3,4) for long-term access. Despite this, there is considerable dependence on central venous catheters (CVCs)—not just for bridge therapy, as was originally intended, but also for long-term access (1). Unfortunately, CVC use is associated with a high rate of infection (5), thrombus-related dysfunction (6,7), and the potential for central venous stenosis (8).

Catheter Dysfunction
The National Kidney Foundation’s Dialysis Outcome and Quality Initiative (KDOQI) guidelines define catheter dysfunction as failure to attain or maintain an extracorporeal blood flow \( \geq 300 \) ml/min at a prepump arterial pressure more negative than \(-250 \) mm Hg (3). This may be translated into failure to maintain an extracorporeal blood flow sufficient to perform adequate HD without significantly lengthening the HD treatment. This failure can be due to many reasons, including mechanical causes, such as kinking or improper positioning of the catheter tip; patient positioning; drug precipitation; thrombotic occlusion; or development of fibrin sheath. One caveat to keep in mind is that this minimally accepted dialyzer blood flow rate is a rather conservative estimate and can easily be accomplished by using newer catheters with larger luminal diameters that are capable of achieving rates of \( \geq 400 \) ml/min when properly placed (9). A delay until blood flow decreases to 300 ml/min may lead to an avoidable loss of the catheter or the access site.

Early Catheter Dysfunction
The catheter may be dysfunctional early in the HD course, usually as a result of such mechanical issues as incorrect tip location (which may be further affected by patient position), a disruption in catheter integrity, or flow obstruction, when the catheter tip lies against the vessel wall. The catheter invariably needs to be exchanged and repositioned to allow for optimal function. Once the catheter is placed, the test for the three Ts is essential: tip, top, and tug (10). Through use of fluoroscopy, the placement of the tip of the catheter should be confirmed, making sure that it does not abut the vessel wall, and the top of the catheter should be evaluated to ensure a smooth curve without any kinks (Figure 1). The tug test refers to the rapid flow of blood when a 10-ml syringe is attached to both venous and arterial ports and vigorously flushed.

Late Catheter Dysfunction
If a catheter has been used successfully and later becomes dysfunctional, the dysfunction is most commonly due to progressive occlusion of the catheter tip by fibrous connective tissue sheath or thrombus (Figure 2, A and B). The thrombus may be intraluminal or at the tip of the catheter; the entire catheter may be encased in a fibrous connective tissue sheath, causing partial or complete occlusion; or right atrial or mural thrombus may be causing extrinsic compression.

Catheter Thrombosis
The phenomenon of thrombus formation was first described in an in vivo study from Belgium. The investigators placed silicone catheters in the anterior caval veins of 123 rats. At scheduled intervals, the pathologic changes were studied under light and electron microscopy (11). In 36 rats, the catheter was
withdrawn immediately; in 72 rats, it was left for 6 months; and in 15 rats, the study was performed up to 10 months after withdrawal of a catheter that had been in place for 6 months. Endothelial damage to the cell wall resulted in a pericatheter thrombus as early as 24 hours after insertion. In the group in which the catheter remained in situ, the pericatheter thrombus was invaded by smooth muscle cells and collagen within 7 days. Over time, the number of cells decreased and the relative amount of collagen increased, transforming the thrombus into an organized sheath that disrupted flow through the catheter.

The pathophysiology of thrombus formation in CVCs used for HD can be further elucidated by application of the Virchow triad: (1) disruption in vessel walls (initial insertion of catheter leading to endothelial damage of the vessel wall), (2) coagulability (initiation of the coagulation and inflammatory cascade), and (3) changes in blood flow (intraluminal stasis of blood in the interdialytic period), which lead to the continuum of thrombus and fibrin sheath formation (12) (Figure 3). Attempts have been made to address each of these factors in order to minimize catheter dysfunction, as detailed below.

**Prevention of Catheter Dysfunction**

**Minimize Disruption in Vessel Walls: Advances in Catheter Design**

Tunneled central venous catheters were first introduced for dialysis access in the 1980s (13,14). Since then, catheter design has evolved (15), with constant modifications in an attempt to provide maximal blood flow while minimizing infections and intimal trauma that lead to thrombosis (10). These changes in catheter design include individual catheters placed side by side, dual-lumen catheters, catheters with varying tip designs (step-tip, split-tip, and symmetric tipped catheters), and the self-centering superior vena cava catheter (15-17).

A prospective, randomized trial in 132 patients comparing the step-tip and split-tip catheters showed higher mean flow rates with the step-tip catheter, but these rates were offset by higher recirculation. The split-tip catheters, on the other hand, had a significantly longer half-life (78% versus 64% at 120 days), as well as fewer late complications (17).

Another group of researchers evaluated the effect of adding side holes to catheters. During a period of 16 months, patients were arbitrarily assigned to dual lumen cuffed tunnel catheters with or without side holes. The authors retrospectively analyzed catheter flow rates, patency, catheter survival, and catheter-related infections. They reported an increase in adherent clots and catheter-related bloodstream infections (2.54 versus 0.254/1000 catheter-days) in catheters with side holes and a slightly improved catheter survival in catheters with no side holes (16).

In the self-centering central venous catheter, the distal tip of the catheter has a unique curved configuration, which keeps the ports of the catheter centered in the superior vena cava. Theoretically, this may reduce fibrin sheath formation and may lead to a decrease in thrombosis. Preliminary clinical studies demonstrated high blood flow rates and no evidence of sheath or clots.

**Initiation of Coagulability Cascade: The Use of Surface-Coated Catheters**

Attempts have been made to decrease the initiation of the coagulability cascade by coating the surface of catheters with antithrombotics, including heparin. Heparin is a polysaccharide anticoagulant that inactivates thrombin, inhibiting its ability to convert fibrinogen into fibrin. Heparin is covalently bonded to the surface of the catheter and may reduce thrombin-activated factors, proliferation of smooth muscle cells, and fibrin sheath formation. In unpublished animal studies, thrombus weight and fibrin sheath formation were both decreased, although the results are yet to be validated in humans. A retrospective case-control study evaluated catheter function and patency in two groups: 38 uncoated split-tip catheters and 50 heparin-coated catheters, all placed in internal jugular veins during a 13-month
The authors found no differences in catheter function or long-term patency in the two groups (18). Other researchers retrospectively queried a prospective database to analyze outcomes of 175 tunneled HD catheters placed in internal jugular veins: 89 heparin-coated and 86 uncoated catheters. The two groups did not significantly differ in cumulative catheter survival or the need for thrombolytic therapy (19). At present, it is difficult to justify the increased cost of surface-treated catheters for long-term HD in the absence of robust clinical data demonstrating that they reduce catheter-related complications in this patient population.

Changes in Blood Flow: The Use of Catheter-Locking Solutions

Another approach to mitigate catheter dysfunction has been the use of varying concentrations of an anticoagulant solution, such as heparin or citrate, in the interdialytic period as prophylaxis against intraluminal thrombus formation. The underlying hypothesis is that “locking” the catheter with an anticoagulant will inhibit thrombus formation and prolong catheter patency. However, despite

Figure 2. | Fibrin sheath. (A) Contrast injection reveals long segment fibrin sheath (arrowhead) after retracting left internal jugular tunneled dialysis catheter (tip of catheter at arrow). (B) Fibrin sheath occluding tip of hemodialysis catheter.

Figure 3. | Pathophysiology of thrombus formation. Virchow triad: endothelial damage (initial insertion of catheter leading to disruption in vessel walls), coagulability (initiation of the coagulation and inflammatory cascade), and stasis (intraluminal stasis of blood in the interdialytic period), which leads to the continuum of thrombus and fibrin sheath formation.
the widespread use of catheter-locking solutions, most CVCs still become dysfunctional. This may be partly explained by the leakage of the anticoagulant into the systemic circulation (20) due to the parabolic flow through the catheter. This lost solution is replaced by blood, and prolonged stasis in the interdialytic period accelerates the process of thrombosis and leads to catheter dysfunction.

A multitude of antithrombotic locking solutions have been used, including heparin, citrate, and recombinant tissue plasminogen activator (tPA), but none of these have been shown to be optimal. Heparin, as mentioned earlier, produces its major anticoagulant effect by inactivating thrombin and activated factor X through an antithrombin-dependent mechanism and inhibiting the conversion of fibrinogen into fibrin (21). The fill volume of heparin used varies with the length and intraluminal diameter of the catheter. However, because of parabolic flow within the catheter, 20% of the lock solution leaks into the systemic circulation (22). This is reflected in elevated activated partial thromboplastin time greater than twice the normal value in patients receiving high-dose heparin locks (23). Further, this increase is proportional to the strength of heparin used (5000–10,000 units/ml > 1000 units/ml).

Not surprisingly, these findings translate into clinically significant issues, including inadvertent systemic anticoagulation (23), an increased potential for bleeding (24) (especially in uremic patients who are already predisposed to it), and heparin-induced thrombocytopenia.

These effects may be alleviated by the use of low-dose heparin (1000 units/ml). In a retrospective analysis comparing bleeding rates in 52 patients who received a concentrated heparin-lock solution (5000 units/ml) with rates in 91 patients who received citrate or low-dose heparin-lock solution (1000 units/ml), the likelihood of a composite bleeding event in the high-dose heparin group was 11.9 times higher than that in the control group (24). In various other protocols, investigators compared the effectiveness of different heparin concentrations (5000 or 10,000 units/ml versus 1000 units/ml) in maintaining catheter patency. Although the incidence of catheter malfunction did not change with the lower dose of heparin as a prophylactic locking solution, the use of tPA to maintain patency increased significantly (25–27).

These observations, along with recent issues with contamination (28) in the manufacturing process for heparin, have prompted efforts to identify alternatives to heparin for locking solutions. A widely used and effective anticoagulant is sodium citrate, which prevents activation of calcium-dependent coagulation pathways by chelating ionized calcium. It was first used as an anticoagulant to preserve blood and for many years since then has been used in continuous renal replacement therapy. A randomized clinical trial comparing a higher concentration (30%) of trisodium citrate with heparin, 5000 units/ml, found no difference in catheter thrombosis or flow-related problems (29). Of note, the study had to be stopped prematurely because of a significantly higher incidence of catheter-related bacteremia in the heparin group (4.1 per 1000 catheter-days compared with 1.1 per 1000 catheter-days in the citrate group). However, complications have been reported with high concentrations of citrate, including perioral or peripheral paresthesia or a metallic taste immediately after locking with a high (30%) concentration of trisodium citrate (29). These disappeared within 1 minute after instillation and did not return after reduction of the volume used for locking. A case report of a fatal cardiac arrest after the use of trisodium citrate 46.7% led to the withdrawal of TriCitrasol, a commercially available product, by the U.S. Food and Drug Administration in 2000 (30). However, lower concentrations of trisodium citrate 4% are extensively used, and multiple investigators have shown that it is at least as effective in maintaining catheter patency as heparin (31–34). This evidence led to the American Society of Diagnostic and Interventional Nephrology recommendations for the use of heparin, 1000 units/ml, or 4% sodium citrate as suitable choices for catheter-lock solutions (35).

The search for the optimal prophylactic solution continues, and many other locking solutions, as well as oral anticoagulants, have been investigated, although in small, uncontrolled studies. A prospective, randomized, crossover study compared 2 ml of recombinant tPA with 2000 units of heparin per ml (36). The recombinant tPA group had better flow and fewer complications, but the sample size was small and follow-up was limited. Thirty patients with ESRD using temporary dialysis catheters were randomly assigned to 5000 units of heparin per ml, 4% citrate, or 3.5% polygeline. Catheter use time or clot volume did not significantly differ among the three groups (34). In another trial, 85 patients who were randomly assigned to placebo or low-dose warfarin (1 mg daily) were followed for the first year of the tunneled catheter or until catheter removal. Although the authors found no improvement in thrombosis-free survival, they observed that an international normalized ratio < 1 was significantly associated with a higher rate of tunneled catheter malfunction (37).

The TROPICS (Tenecteplase for the Restoration of Functional HD Catheters) study evaluated tenecteplase, a newer thrombolytic, in dysfunctional catheters in initially 1-hour, and subsequently extended (72-hour), dwells (38). Use of this agent was associated with improved catheter patency and function.

In a novel protocol, the Pre-CLOT (Prevention of Catheter Lumen Occlusion with rt-PA versus Heparin) study randomly assigned 225 patients undergoing maintenance HD, in whom a CVC had been newly inserted, to a catheter-locking regimen of heparin (5000 units/ml) three times per week or recombinant tPA (1 mg in each lumen) substituted for heparin at the midweek session, with heparin used in the other two sessions (39). Catheter patency was the primary outcome, and catheter-related bacteremia was the secondary outcome. The researchers found an increased risk of catheter malfunction by a factor of almost 2 and an increased risk of bacteremia from any cause by a factor of 3 among patients treated with heparin only compared with those receiving recombinant tPA once weekly, with no increase in the risk of adverse events, including bleeding, in the two groups. The authors concluded that the use of once-weekly recombinant tPA significantly reduced the incidence of catheter malfunction and catheter-related bacteremia.

The AZEPTEIC (Assessing Zuragen Efficacy and Safety in the Prevention and Treatment of Infection in Catheters) trial (40) randomly assigned 407 patients (49,565 catheter-days):
201 patients in the treatment group, who received 7.0% sodium citrate, 0.15% methylene blue, 0.15% methylparaben, and 0.015% propylparaben, and 206 in the heparin group. The incidence of catheter-related bloodstream infections was significantly lower in the treatment group (0.24 versus 0.82 per 1000 catheter-days; relative risk, 0.29 [95% confidence interval, 0.12 to 0.70]; \(P=0.005\)). Catheter loss due to patency failure was also less likely in the treatment group (0 versus 4 catheters lost; \(\log\) rank, \(P=0.04\)). Table 1 summarizes the randomized trials that evaluate prevention of catheter dysfunction.

**Evaluation of Catheter Dysfunction**

Early detection of catheter dysfunction before it becomes nonfunctional is essential, and all members of the vascular access team should be involved. The goal is to provide safe, efficient, cost-effective, and sustained catheter function. This minimizes inadequate dialysis caused by recirculation, prevents extension of treatment times, and leads to decreased morbidity.

A dysfunctional catheter may be easily assessed at the bedside. Clues to diagnosis include blood pump flow rates >300 ml/min, arterial pressures more negative than −250 mmHg, or venous pressures >250 mmHg at blood flow rates of 400 ml/min. Routine monthly laboratory values may show decreased clearance: urea reduction ratio rates of 400 ml/min. Routine monthly laboratory values mmHg, or venous pressures.

**Bedside Management of Catheter Dysfunction**

Management of catheter dysfunction involves bedside maneuvers initially. If these are unsuccessful, the patient should be referred for an endovascular procedure. Bedside management includes repositioning the patients (Trendelenburg position) or the use of rapid saline flushes to dislodge a possible thrombus. Reversal of lumens may provide temporary respite and allow for completion of the HD treatment.

Endoluminal brushing is a technique initially developed to sample the endoluminal surface of the catheter and confirm a diagnosis of catheter-related bacteremia. Its use was later expanded in a small sample to determine the effect on patency (46) by passing through the entire length of the catheter from hub to tip and removing the fibrous connective tissue from the intraluminal surface. The authors reported a >60% success rate in restoring patency with this minimally invasive procedure. However, concerns were raised about the possibility of showering micro-organisms into the venous circulation (47), as well as causing arrhythmias, especially if the biofilm-laden tip was passed beyond the distal port of the catheter.

Another potential option in management of catheter dysfunction is the use of thrombolytic agents, which may be left to dwell as catheter-lock solutions (48–50) or may be

<table>
<thead>
<tr>
<th>Study (Reference)/Design</th>
<th>Intervention</th>
<th>Results</th>
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<tbody>
<tr>
<td>Buturovic et al., 1998 (34) Randomized ((n=30))</td>
<td>Heparin (1666 U/ml) versus polygeline 3.5% versus citrate 4%</td>
<td>No difference in catheter use time or clot volume</td>
</tr>
<tr>
<td>Hendrickx et al., 2001 (41) Randomized ((n=19))</td>
<td>Heparin (5000 U/ml) versus citrate 5%</td>
<td>Significantly more dialysis sessions with clot formation in citrate group but no difference in use of thrombolytic infusion</td>
</tr>
<tr>
<td>Weijmer et al., 2005 (29) Randomized ((n=291))</td>
<td>Heparin (5000 U/ml) versus citrate 30%</td>
<td>Similar patency as measured by thrombolytic therapy or catheter removal; citrate associated with lower costs and decreased CRB</td>
</tr>
<tr>
<td>Macrae et al., 2008 (31) Randomized ((n=61))</td>
<td>Heparin (5000 U/ml) versus citrate 4%</td>
<td>Similar patency as measured by thrombolytic therapy</td>
</tr>
<tr>
<td>Power et al., 2009 (42) Randomized ((n=232))</td>
<td>Heparin (5000 U/ml) versus citrate 46.7%</td>
<td>Thrombolytic use more common in citrate group, but higher adverse events with high-concentration citrate</td>
</tr>
<tr>
<td>Hemmelgarn et al., 2011 (39) Randomized ((n=225))</td>
<td>Heparin (5000 U/ml) versus rtPA (1 mg/ml)</td>
<td>Catheter malfunction significantly less in rtPA group</td>
</tr>
<tr>
<td>Maki et al., 2011 (40) Randomized ((n=225))</td>
<td>Heparin (5000 U/ml) 0.24 M of (7.0% sodium citrate, 0.05% methylene blue, 0.15% methylparaben, and 0.015% propylparaben)</td>
<td>Treatment group had significantly lower incidence of CRBSI; catheter loss due to patency failure in treatment group less likely</td>
</tr>
</tbody>
</table>

CRB, catheter-related bacteremia; rtPA, recombinant tissue plasminogen activator; CRBSI, catheter-related bloodstream infection.
Infused (51,52). Thrombolytics are advantageous because if they are successful, catheter patency is restored noninvasively, missed HD treatments are minimized, and the primary access site is preserved. Early thrombolytics included urokinase and streptokinase, but these are no longer used because of the potential risks for anaphylaxis with repeated streptokinase infusions and contamination concerns with urokinase (48). Reteplase safely restores flow through occluded and poorly functioning catheters. In a retrospective analysis, reteplase (0.4 unit) was used in 50 instances to restore or improve blood flow rates in 23 catheters among 19 patients and established adequate blood flow rates during the current and next dialysis session in 44 of 50 (88%) cases (49). In another open-label observational trial in dysfunctional catheters, varying doses of reteplase were instilled into the catheter lumens and allowed to dwell there until the next HD session (53). Reteplase installation restored catheter function in 87% of episodes, and a dose of 1 unit was as effective as 4- and 6-unit doses.

Low-dose recombinant tPA (2.5 mg in 50 ml of normal saline at 17 ml/h infused over 3 hours) was evaluated for management of radiographically documented fibrin sheaths in 124 episodes of catheter dysfunction. The immediate technical success rate was 91%, but the primary patency rates were low. However, with re-treatment of failed catheters, secondary patency rates improved (52). The use of alteplase in short (1 hour) or long (>48 hours) dwells led to a 78% overall catheter patency rate immediately after treatment; patency rate decreased to 48% at 2 weeks. Although restoration of patency was feasible with either regimen, the effect was temporary and not sustained (50). The authors concluded that tPA was ineffective in the long-term management of catheter dysfunction and provided a median gain of catheter function of only 14 days. Their results were confirmed by a longitudinal study of a cohort of 570 catheters over a 2.5-year period. The investigators demonstrated that treatment of recurrent catheter malfunction with alteplase allowed for a median of only five to seven additional dialysis sessions before the treatment needed to be repeated or the catheter exchanged (54). Table 2 summarizes the results of these studies.

### Endovascular Management of Catheter Dysfunction

If catheter patency is not restored successfully in the dialysis unit, the patient may be referred for endovascular management. The most common cause for referral is the development of a fibrous connective tissue sheath, which may be removed by a variety of techniques, including fibrin sheath stripping, internal snare, or over-the-wire catheter exchange with or without balloon disruption.

Fibrin sheath stripping is a percutaneous procedure that involves mechanical stripping of the fibrin sheath with a snare introduced via the common femoral vein. Although fibrin sheath stripping has excellent technical success rates, the primary patency is limited (57–59). Further, with the increased morbidity from a second venipuncture, this technique has been abandoned in favor of newer, less invasive procedures. A small series reported the use of an internal snare that bypasses the need for a second cannulation, with excellent initial restoration of patency (60). The authors used a nitinol wire looped in the middle and passed it through the luminal surface of the catheter. As it exits the port, the loop was tightened to form a snare that disrupted the fibrous sheath. This promising, effective, inexpensive, and minimally invasive approach needs to be evaluated in larger studies.

The method of choice advocated by KDOQI is disruption of the fibrin sheath with a balloon (Figure 4) and placement of a new catheter (catheter exchange). However, the comparisons of sheath disruption by stripping or catheter exchange with and without an angioplasty balloon do not show any one technique to be more efficacious than the other (Table 3). The advantage of this technique is that it may be performed through the existing venotomy and exit sites, thus minimizing the morbidity associated with additional cannulations. Further, it does not cause an increase in infectious complications and provides patency rates similar to those of de novo catheter placements (61,65). Figure 5 depicts a proposed algorithm to the endovascular management of dysfunctional catheters.

In cases of catheter-associated right atrial thrombus (CRAT; Figure 6), superior vena cava/inferior vena cava thrombus, or catheter tip thrombus, there are no consensus guidelines for treatment. In our institutions, if a catheter

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**Table 2. Catheter-locking solutions in the management of catheter dysfunction**

<table>
<thead>
<tr>
<th>Study (Reference)/Design</th>
<th>Intervention</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haire et al., 1994 (55)</td>
<td>Urokinase (10,000 IU/ml) versus tPA (2 mg)</td>
<td>tPA significantly better in restoring catheter function</td>
</tr>
<tr>
<td>Savader et al., 2001 (52)</td>
<td>tPA infusion (2.5 mg over 3 h)</td>
<td>91% technical success rate; primary patency low but improved secondary patency</td>
</tr>
<tr>
<td>Little and Walshe, 2002 (54)</td>
<td>tPA (1 mg/ml); dwell time 2–8 h</td>
<td>Patency declined with successive use; median patency only 5–7 treatments</td>
</tr>
<tr>
<td>Haire et al., 2004 (56)</td>
<td>Urokinase (5000 IU/ml) versus placebo</td>
<td>Urokinase significantly better in restoring catheter function</td>
</tr>
<tr>
<td>Falk et al., 2004 (49)</td>
<td>Reteplase (0.4 units); dwell time 30 min–1 h</td>
<td>Initial patency 88%; no adverse events</td>
</tr>
<tr>
<td>Macrae et al., 2005 (50)</td>
<td>tPA (1 mg/ml); dwell time 1 h versus 48–96 h</td>
<td>Initial patency 78% but short-lived; median patency only 6 treatments</td>
</tr>
</tbody>
</table>

* tPA, tissue plasminogen activator; rtPA, recombinant tPA.
Tip thrombus is suspected, we will perform a guidewire catheter exchange. This is usually a cause of late catheter dysfunction, as presented in Figure 5. In the case of large vein thrombus, such as in the superior or inferior vena cava, documented by ultrasonography or angiography, we would usually provide anticoagulation if no contraindications are present and then exchange the catheter if it is dysfunctional. We understand that these patients usually have no other option for access and that their catheter is their “lifeline.” As recently reported in a meta-analysis on CRAT (66), overall mortality is very high in this cohort of patients, and nonremoval of the catheter is a significant risk factor. According to the algorithm presented in that review, an alternative access should be placed. If there is no alternative, then the catheter should be exchanged over a wire. Anticoagulation should be instituted if it is not contraindicated; if the CRAT is ≥6 cm, the thrombus should be followed to see whether it diminishes in size. If it does not, then surgical or percutaneous thrombectomy should be considered.

**Conclusion**

It is a common misconception that catheter thrombosis and dysfunction are a “way of life” with catheters. Poor flow must be evaluated and addressed immediately. CVC dysfunction has become a central issue in the field of nephrology because of the steady increase in the number of patients undergoing renal replacement therapy and the increasing number of autogenous arteriovenous fistula placements. Despite a variety of prophylactic methods and novel approaches to the treatment of catheter dysfunction, results have been less than stellar. The best prophylaxis still remains avoidance of catheters. If a CVC does need to be placed, there should be a documented plan to eventually replace it with another form of access as soon as possible.

**Table 3. Endovascular procedures in the management of catheter dysfunction**

<table>
<thead>
<tr>
<th>Study (Reference)/Design</th>
<th>Intervention</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duszak et al., 1998 (61)</td>
<td>De novo catheter placements versus catheter exchange</td>
<td>No significant difference in patency or infections</td>
</tr>
<tr>
<td>Merport et al., 2000 (62)</td>
<td>Fibrin sheath stripping versus catheter exchange</td>
<td>Immediate technical success 97%, but primary patency significantly better in the exchange group</td>
</tr>
<tr>
<td>Janne d’Othée et al., 2006 (63)</td>
<td>Fibrin sheath stripping versus catheter exchange versus angioplasty disruption and exchange</td>
<td>All 3 equivalent in terms of technical success, patency, and complications</td>
</tr>
<tr>
<td>Oliver et al., 2007 (64)</td>
<td>Catheter exchange versus angioplasty disruption and exchange</td>
<td>Improved patency and statistically significant increased blood flow and urea replacement ratio in the exchange group</td>
</tr>
</tbody>
</table>
Figure 5. Algorithm for the endovascular management of dysfunctional catheters in the interventional suite.

Figure 6. Right atrial thrombus associated with hemodialysis catheter.
Disclosures

None.

References


40. Maki DG, Ash SR, Winger RK, Lavin P, for the AZEPTIC Trial Investigators: A novel antimicrobial and antithrombotic lock...


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