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### Abstract

The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI) has published guidelines for optimal clinical practices aimed at improving dialysis outcome and patient survival. The reporting standards recognize eight categories of complications related to hemodialysis access: thrombosis and failure to mature, bleeding, infection, aneurysm and pseudoaneurysm, seroma, ischemia or steal syndrome, venous hypertension and neuropathy. Venous outflow and anastomotic stenosis from neointimal hyperplasia are the main causes of thrombosis in mature AVF. Early thrombosis occurs more frequently with autogenous AVF and the majority of these occur within the first 5 days. Early thrombosis is usually attributed to technical fault. The commonest cause of late dysfunction in either fistula or grafts is venous stenosis, though the site of occurrence differs. Patients with ESRD have an increased risk of bleeding due to defects in hemostatic mechanisms secondary to uremia or acquired or inherited coagulation abnormalities. Patients on hemodialysis also have periodic heparin exposure. Cardiopulmonary complications: It is the leading cause of the death inpatients receiving chronic renal replacement therapy

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### Introduction

The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI) has published guidelines for optimal clinical practices aimed at improving dialysis outcome and patient survival, [1].

The reporting standards recognize eight categories of complications related to hemodialysis access: thrombosis and failure to mature, bleeding, infection, aneurysm and pseudoaneurysm, seroma, ischemia or steal syndrome, venous hypertension and neuropathy, [2].

Complications can be classified as follows:

A- Thrombotic complications.

B- Non-thrombotic complications.

C- Cardiopulmonary complications.

A. Thrombotic complications

Venous outflow and anastomotic stenosis from neointimal hyperplasia are the main causes of thrombosis in mature AVF [3].

Early thrombosis occurs more frequently with autogenous AVF and the majority of these occur within the first 5 days. Early thrombosis is usually attributed to technical fault. The commonest cause of late dysfunction in either fistula or grafts is venous stenosis, though the site of occurrence differs. [4].

B. Non-thrombotic complications.

i. Bleeding:

Patients with ESRD have an increased risk of bleeding due to defects in hemostatic mechanisms secondary to uremia or acquired or inherited coagulation abnormalities. Patients on hemodialysis also have periodic heparin exposure. [5].

ii. Infection:

Infection is the second leading cause of access loss, after thrombosis.

Impaired humoral and cellular immunity, nutritional deficiencies, and type of vascular access are among the major determinants [6].

iii. Pseudoaneurysm.

Pseudoaneurysms can occur at autogenous or prosthetic AVF anastomosis or puncture sites. They are associated with an increased risk of thrombosis, pain, cosmetic problems, infection, bleeding, and difficulty with access, [7].

iv. Autogenous access aneurysms:

Aneurysm formation may occur because of repeating punctures that can weaken the vein wall in some patients. Sometimes, proximal stenosis accelerates the process by raising the pressure into the AVF. The incidence of aneurysm formation is approximately 5% to 6 % [8].

These patients may have a large, painful aneurysm sac that may lead to necrosis on dermal tissue. The sac can rupture resulting in possible fatal hemorrhage. [9].

v. Access related hand ischemia or steal syndrome:

It has been reported in up to 8% of patients following surgical creation of AVF. Redirection of flow from the distal artery in the direction of AVF can result in ischemic steal, [10].

Historically, standard of care was ligation of the AVF, which would result in loss of dialysis access. Surgical banding, plication, and placement of a tapered graft can reduce flow through

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AVF but have varied success in maintaining sufficient flow for dialysis and may result in complete loss of access, [11].

vi. Neuropathy:

Neuropathy is a common finding among hemodialysis patients. At the onset of dialysis, about two thirds of patients have already developed peripheral neuropathy. Causes can be systemic, such as uremia or diabetes, as well as

mechanical, such as entrapment or compartment syndromes. [12].

C. Cardiopulmonary complications

It is the leading cause of the death inpatients receiving chronic renal replacement therapy. [13].

## LATE VASCULAR ACCESS COMPLICATIONS

Stenosis and recurrent stenosis

Stenosis can occur at any level from the arterial inflow to the venous outflow, often in the juxta-anastomotic areas or even within the graft. [14]

Pre-emptive treatment of all stenoses has not been shown to be of benefit.

Therefore, only stenosis that have a hemodynamic effect (>70% decrease in lumen area) and are associated with decreased flow, elevated venous pressures, or an abnormal physical examination (reduced thrill or pulsatile flow) should be treated. The main benefit of pre-emptive treatment of hemodynamically significant stenoses is decreased thrombosis, avoidance of sub-optimal HD and CVCs, and not necessarily prolonged life of the VA. [15]

Inflow arterial stenosis

Stenoses in the subclavian, brachial, radial or ulnar artery are more frequent in the

elderly, in diabetics and in hypertension. In addition, stenoses often develop at the arteriovenous anastomosis of AVFs or the arterial anastomosis of AVGs. A prospective multicentre study has demonstrated that about 30% of referrals for stenosis intervention were due either to stenosis in the native artery or at the anastomotic site. [16]

In another study 12.5% of dysfunctional AVFs and AVGs were due to inflow stenosis and in 77% endovascular treatment was successful. PTA is a safe and effective technique with a low rate of re-intervention. For elastic recoil, rapidly recurrent stenosis, or residual stenosis >30% after PTA, the implantation of a stent is recommended. [17]

Open options for treatment of stenoses in the native arteries include bypass grafting

and endarterectomy but are seldom performed. No randomized studies have been performed between open and endovascular surgery.[16]

Juxta-anastomotic stenosis

For hemodynamic reasons, stenosis often develops in the juxta-anastomotic area around either the arteriovenous anastomosis of AVFs or the arterial anastomosis of AVGs and the first few centimeters (2-5 cm) into the vein/graft. [15]

Traditionally open surgery with creation of a new proximal anastomosis or graft interposition of a short ePTFE graft, has been the preferred method in forearm AVFs, although PTA can be an alternative. [18]

It has been demonstrated that PTA can be used as the primary approach for juxta-anastomotic stenosis. However, the recurrent stenosis rate is higher than after surgery, and in those patients where early recurrence occurs, surgical revision is indicated. If surgical revision is expected to shorten the usable length of AVF for cannulation PTA is justified as the primary tool. [19]

#### Venous outflow stenosis

Reduced VA flow, prolonged bleeding times and elevated venous pressure suggests

the presence of a venous outflow stenosis often where the peripheral vein enters the deeper system. PTA is the first treatment option in the outflow veins (cephalic/basilic), especially when the lesion is short (<2 cm). For long segment stenoses (>2 cm), treatment is controversial, including PTA or surgery either by bypass grafting or vein transposition. Grafts should be reserved for patients with exhausted peripheral veins whilst fistula preserving procedures such as PTA or patch angioplasty should be favoured over graft extensions to central venous segments. Venous outflow stenoses may be resistant to PTA and require high pressure balloons or cutting balloons. [20]

Stents or open surgery should be considered if repeated PTA fails. Random clinical trials (RCTs) comparing stenting with PTA did not show statistically significant differences in patency. Stents used in previous RCTs may have been inferior to more recently used devices especially when nitinol stents were used. The use of stent grafts to treat VA stenosis has recently gained consensus since they may decrease the incidence of restenosis by interposing an inert layer to separate the thrombogenic vascular wall from the blood flow and impede the migration of smooth muscle cells. [21]

Stent grafts mimic open surgical revision of a graft, preventing elastic recoil and avoiding transient growth of neointimal tissue. A multicentre RCT showed better patency rates for stent grafts vs. simple PTA for the treatment of AVG anastomotic stenosis with a sustained, greater than 2-fold advantage over PTA in the treatment area for primary patency and overall VA patency. Similar favorable results for stent grafts were found in another RCT when treating in stent restenosis in patients with AVFs and AVGs. [22]

Concerns remain about costs, and on the real value in preventing graft thrombosis.

Thus, the use of stent grafts to treat AVG venous anastomosis stenosis is reserved for complicated cases. The consensus is that for stenting the venous anastomosis and venous stenoses, stent grafts may be superior to bare stents. [23]

#### Cephalic arch stenosis

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The cephalic vein forms part of the outflow for RC AVF and is the sole outflow for BC AVF. The cephalic arch is prone to the development of hemodynamically significant stenosis, related to its perpendicular junction with the deeper veins. Stenosis in this region is common and is usually treated by PTA. [14]

The cephalic arch is the most frequent location for stenosis of upper arm dysfunctional AVFs, comprising 30-55% of all upper arm VA stenosis sites. It responds poorly to PTA, with 6 month primary patency rate of 42%, which is below the 50% unassisted patency rate recommended for intervention for VA stenosis. In small RCT, stent grafts were shown to be superior to PTA in treating cephalic arch stenosis. [24]

When the result of PTA is poor or if associated with vein rupture, or if there was early restenosis (<3 months), stent grafts can be used. Because restenosis after stenting in the cephalic arch is an issue, stent grafts have been suggested as an alternative in early recurrent cephalic arch stenosis after PTA. [24]

RCT study on the outcome of 25 consecutive patients with recurrent cephalic arch stenosis has shown the following: DSA at 3 months demonstrated restenosis rates of 70% in the bare stent group and 18% in the stent graft group. Life table analysis at 3 and 6 months showed that primary patency was 82% in the stent graft group and 39% in the bare stent group. One-year primary patency was 32% in the stent graft group and 0% in the bare stent group. It was concluded that the use of stent grafts for recurrent cephalic arch stenosis significantly improved short-term restenosis rates and long-term patency compared with the use of bare stents. The major drawback of stent grafts in the cephalic arch is possible occlusion of the axillary or subclavian vein that may prevent further VA

in the ipsilateral arm, but the rate of this complication is unknown. Therefore, until long-term results are published the use of stent grafts can only be recommended when it is considered unavoidable by an endovascular specialist. The role of drug eluting balloons (DEB) is currently being examined and may offer an alternative to stents in VA. [25]

A small RCT showed that DEB angioplasty may be a cost-effective option that significantly improves patency after angioplasty of venous stenoses of failing VA.

Since the outflow anastomosis can be considered as an experimental model for NIH, future research direction may clarify whether DEBs may offer an alternative to stents in VA. [25]

As an alternative to endovascular therapy, open surgical revision for cephalic arch stenosis has been described and involves diverting the blood flow to other patent veins for example the axillary vein with a primary patency of 60% at 1 year. However, such procedures might jeopardise the creation of a future basilic vein fistula. Furthermore, it has been shown that previous endovascular treatment of the cephalic arch decreases the patency of open surgical revision. [26]

Central venous occlusive disease ( CVOD)

It is a common finding with an incidence of 2-40%. It may be asymptomatic but can cause upper extremity, facial or breast swelling, increased venous outflow resistance, post-cannulation

bleeding, AVF aneurysms, and may lead to VA loss, and preclude future VA creation in the ipsilateral limb. These lesions are associated with prior central vein catheter (CVC) use, increased blood flow and extrinsic compression. [27]

Twelve to thirteen percent of patients with VA have symptomatic CVOD that may require some form of intervention and 25-50% of all subclavian CVCs are associated with subsequent CVOD, whereas lower rates have been reported for jugular vein catheters. [28]

Clinical suspicion of the diagnosis should be confirmed by either fistulography or CTA. DUS is generally less useful since visualization of central venous outflow may be difficult but can be of help using defined criteria. [28]

There is no ideal treatment for this problem. Withholding treatment in patients with no or minor symptoms can even show significantly better short and long-term central vein patency than treatment of symptomatic cases without detrimental effects on overall dialysis circulation. [29]

Since surgery requires sufficient expertise and is associated with increased morbidity, PTA with its low morbidity and good short-term patency has become the accepted treatment for symptomatic CVOD. Poor long-term patency rates after PTA are due to elastic recoil or recurrent NIH and repeated interventions are often necessary. [30]

According to most studies bare metal stents (BMSs) have not demonstrated an advantage in long-term patency over PTA and are not recommended in mobile axillary vein segments or subclavian segments beneath the clavicle. [29]

New self-expandable dedicated venous stents may be more promising. In view of the reported superiority of stent grafts compared with BMSs for recurrent cephalic arch stenosis these have been used for cases of symptomatic CVOD, however, the possible disadvantage of covering major venous confluences must be considered. [31]

Despite a significant morbidity, surgical revision should be considered in patients with CVOD and failed endovascular attempts. [29]

Various procedures include bypassing the central occlusion (axillary or brachial vein to jugular vein; axillary vein to saphenous or iliac veins), intrathoracic central venous reconstructions, extra-anatomical venous reconstruction, and non-venous VA (axillary or brachial artery to right atrium bypass). Hybrid procedures combining surgical bypass with endovascular recanalisation with stent grafts may also be an option. In addition, high flow AVFs with CVOD may also be treated by flow reducing procedures such as fistula vein banding. [32]

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