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Přehledový článek | Review article

Contemporary management of arteriovenous hemodialysis fistula aneurysms

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SOUHRN

Úvod: Aneurysma arteriovenózní fistuly vzniká až u 60 % hemodialyzovaných pacientů. Často jde o asymptomatickou komplikaci, avšak s možným rizikem rozvoje závažných komplikací. Existuje řada chirurgických a endovaskulárních technik léčby, ale jasná klinická doporučení, kdy a jak intervenovat, chybějí. Autoři popisují klinický obraz, indikace k léčbě a současné možnosti léčby aneurysmatu arteriovenózní fistuly.
Metodika: Nesystematický přehled publikované literatury uvedených databází: MEDLINE, ScienceDirect, Scopus a Cochrane Database of Systematic Reviews. Do hodnocení byly zahrnuty publikace týkající se aneurysmatu arteriovenózní fistuly a terapeutických možností od ledna 1973 do června 2016. Z hodnocení byly vyřazeny práce týkající se aneurysmatu a pseudoaneurysmatu protetických graftů.
Výsledky: Aneurysma arteriovenózní fistuly je definováno jako dilatace všech tří vrstev cévní stěny a průměrem větším než 18 mm. Indikace k léčbě jsou: bolestivost, riziko krvácení a neadekvátní průtok (nízký/vysoký). Samotný průměr aneurysmatu, stejně jako kosmetické důvody, by neměly být indikací k léčbě. Nejčastěji využívané terapeutické modality léčby aneurysma arteriovenózní fistuly jsou: resekce s interpozicí, remodelační techniky a implantace stentgraftu. I když bylo publikováno několik léčebných metod, v současné době neexistuje publikace srovnávající jednotlivé léčebné techniky.
Závěr: Asymptomatické aneurysma arteriovenózní fistuly má být léčeno konzervativně s pravidelným sledováním. Vzhledem k absenci dostatečných důkazů neexistuje jasná strategie léčby pro aneurysma arteriovenózní fistuly vyžadující intervenci. U symptomatického aneurysmatu, zejména s vysokým rizikem krvácení, by měla být co nejdříve indikována terapie.

ABSTRACT

Introduction: Aneurysms develop in up to 60% of patients with an arteriovenous fistula. Frequently arteriovenous fistula aneurysms are asymptomatic with the presence of symptoms potentially heralding the development a significant complication. A range of surgical and endovascular techniques are available to manage arteriovenous fistula aneurysms but clinical guidelines regarding the appropriate application of each approach are lacking. This review will examine the presentation, indications for treatment and management options for arteriovenous fistula aneurysms.
Methods: A non-systematic review of published literature in the following databases was performed: MEDLINE, ScienceDirect, Scopus and the Cochrane Database of Systematic Reviews. Publications relating to arteriovenous fistula aneurysms and treatment options between January 1973 and June 2016 were considered for inclusion. Articles pertaining to aneurysms and pseudoaneurysms of prosthetic arteriovenous access sites were excluded. The literature search was supplemented by a review of the author's experience.
Results: Arteriovenous fistula aneurysms are defined by an expansion of the intimal, medial and adventitial layers of the vessel wall to a diameter of more than 18 mm. Treatment of arteriovenous fistula aneurysm is

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indicated if there is pain, risk of haemorrhage and flow disturbance (either low or high flow). When deciding on whether to actively treat or observe, the diameter of the arteriovenous fistula aneurysm and cosmetic concerns should not be considered in isolation. Commonly applied approaches for treating arteriovenous fistula aneurysm are resection with interposition, remodelling and insertion of an endovascular stent graft. Although various surgical and endovascular options have been reported, there are no prospective studies directly comparing techniques

Conclusions: Asymptomatic aneurysms can be safely observed. Due to a lack of sufficient evidence base, no individual management strategy can currently be recommended for aneurysms requiring treatment. Finally, symptomatic aneurysms, mainly which are in the high risk of bleeding, should be indicated for the treatment as soon as possible.

Keywords:
Aneurysm
Arteriovenous access
Arteriovenous fistula
Haemodialysis
Treatment

Introduction

Renal replacement therapy for patients with end stage renal failure comprises haemodialysis, peritoneal dialysis and renal transplantation. For haemodialysis to commence a permanent arteriovenous access (AVA) must be obtained. AVA can be achieved with either an arteriovenous fistula (AVF) or an arteriovenous graft (AVG). Where feasible an AVF is favoured because of a reduced risk of infection and higher patency rates [1].

An AVF can be complicated by thrombosis, stenosis, steal syndrome, low or high flow and arteriovenous fistula aneurysm (AVFA). In comparison to other AVF complications, aneurysm formation is not uncommon but it has a poorly described evidence base.

Definition

A true aneurysm has been defined by The Society for Vascular Surgery as a focal dilatation of intimal, medial and adventitial layers of the vessel wall [2]. The most recent Kidney Disease Outcomes Quality Initiative (K/DOQI) guidelines defined a true aneurysm as an abnormal blood-filled dilation of the blood vessel wall secondary to disease of the vessel wall [1].

In contrast, a pseudoaneurysm is defined by The Society for Vascular Surgery as a focal dilatation of the vessel wall by neointimal and fibrous tissue [2]. The description of a pseudoaneurysm by K/DOQI is a vascular abnormality that appears like an aneurysm but is lined by external fibrous tissue as opposed to a true vessel wall [1]. Guidelines from the Vascular Access Society regarding the definitions of aneurysms and pseudoaneurysms have not yet been formulated [3].

In terms of aneurysm size, current guidelines offer no strict criteria to define and classify AVFA. The suggested diameter of a usable AVF is 6 mm in the K/DOQI guidelines [1], which is three times greater than the diameter of a typical autologous vein [4,5]. In the published literature, the reported sizes of AVFA range between 19.5 and 80 mm, which encompass a more than threefold expansion of the advocated diameter of an AVF vein. As such, Valenti et al. [6] defined AVFA as any segment of vein with a diameter greater than 18 mm and Balaz et Bjorck [7] proposed a definition of AVFA as a dilatation of all three vein layers to at least a diameter of 18 mm. This denotes an increase of the diameter of a vein in a maturated AVF by three times (3 × 6 mm = 18 mm).

Classification

To our knowledge, there are just a couple of classification systems for AVFA that have been reported to date: one by Valenti et al. [6] constructed from their own clinical findings and one by Balaz et Bjorck [7] based on a review of published evidence.

Depending on the shape of the aneurysm Valenti et al. [6] categorised AVFAs into four different groups:

Type 1: without a "camel hump"

1a: dilatation lengthways along the vein; the vein is uniformly dilated from the site of the arterial anastomosis along the majority or all of its length. The configuration is akin to a hosepipe.

1b: An aneurysm after the anastomosis; the vein is dilated proximally. This type of aneurysm is usually within 5 cm of the arterial anastomosis.

Type 2: with a "camel hump"

2a: The classic "camel hump"; there is at least one localised venous dilatation. However, more frequently there are two. Hence, the description of a classic "camel hump". These dilatations emerge at cannulation sites from previous dialysis sessions. The vein is normal calibre between the locations of the aneurysms but can be stenosed.

2b: A mixture of types 2a and 1b; in other words, a post-anastomotic aneurysm with localised dilations.

Type 3: This class signifies aneurysms that do not have a typical configuration compatible with the description of either type 1 or type 2 AVFAs.

Type 4: These AVFAs clinically appear to be true localised aneurysms but a diagnosis of false aneurysm is confirmed on subsequent duplex imaging.

Balaz et Bjorck [7] described a classification AVA aneurysms on the basis of the type of aneurysm. Depending on the presence of stenosis or thrombosis on ultrasound or fistulography, Balaz et Bjorck [7] classified the type of AVA into four groups:

Type I – No evidence of stenosis or thrombosis.

Type II – Significant (≥ 50%) stenosis in the arterial inflow vessel (A), at the site of the arterial anastomosis (B), along the area used for cannulation (C), or in the central venous system (D).

Type III – Incomplete thrombosis with at least 50% occlusion of the lumen.

Type IV – A complete thrombosis.

The others parameter as: (a) aneurysm diameter, (b) whether there was a arteriovenous graft or AVF, (c) the type of vein that is aneurysmal, (d) the amount of aneurysms are recommended for detailed description of aneurysm degeneration of AVA according to the suggested nomenclature in the clinical practice recommendation [8].

Both classification systems are important, even though Valenti's is focused on morphology and should be assessed in outpatient clinic without ultrasound. However, this system does not include intravenous pathology. On the other hand, Balaz's system does not describe shape of aneurysm but describes presence of stenosis or/and thrombosis, which is important for treatment strategy. For detailed description of AVFA, the combination of both systems seems to be logical and interesting solution.

Prevalence and aetiology

In the published literature, the rate of aneurysm development in the haemodialysis population varies widely from 6% up to 60% [6,9–11]. However, these figures are not truly representative, as inconsistencies in the definition of true aneurysms, false aneurysms and pseudoaneurysms in different studies mean that these figures represent the total occurrence of all types aneurysms.

The underlying pathogenesis of AVFA development has not been conclusively proven but several theories exist. In the immediate period following AVF surgery there is geometric and haemodynamic maturation that represents the origin of AVFA development. Flow volumes escalate through the AVF due to the pressure differences between the arterial and venous circulation [12]. Resistance in the venous outflow and capacity of the vein to distend facilitates the arterialised vein to cope with high flow rates under reduced pressure gradients [13]. As a result of arterial pressure, the venous component of the AVF distends both laterally and distally but crucially not in the axial direction, which leads to a convoluted conformation [14].

Furthermore, increased venous pressure may also result from stenosis within the central venous system and hasten aneurysm formation. Central venous stenosis typically results from extended central venous catheterisation and previously published studies suggest a broad range of incidence between 13% and 100% [15–20]. Imaging of the central venous system by fistulography or ultrasound is not routine however, and potentially explains the wide incidence rates reported. Finally, repeated venepuncture during regular dialysis sessions can lead to localised tissue damage, trauma, inflammation and infection resulting in injury of the vessel wall and subsequent aneurysmal defects [21].

Clinical presentation and management of arteriovenous aneurysm

Many patients consider AVFA a distressing cosmetic issue but in the absence of symptoms a line of conservative management can be safely adopted. This is an approach advocated by K/DOQI, who recommend conservative management consisting of avoidance of cannulation of aneurysmal areas in patients with an asymptomatic AVFA [1]. To facilitate use of fistulae with aneurysmal dilatations for dialysis a modified buttonhole cannulation technique has also been described [22].

However, additional specific guidance on when and how to treat AVFA in symptomatic patients is currently unavailable [1–3]. Symptomatic AVFA can present with discomfort, protracted bleeding after dialysis, low flow and inadequate dialysis or high flow with risk of steal syndrome or high output cardiac failure. Bleeding can also occur from rupture of the aneurysm when skin over the aneurysm becomes progressively thin, necrotic or gets infected.

A review of the literature reveals that important considerations in the management of AVFA include the condition of overlying skin, presence or absence of symptoms, difficulty with cannulation, and performance of the AVA. To facilitate clinical decision-making, measurements of access flow, cardiac output and cardiac index by Doppler ultrasound and echocardiography should be obtained. When considered in isolation, the diameter of the aneurysm and cosmetic considerations of the patient are not indications for intervention. The four main groups of clinical presentations of AVFA are discussed further below.

Group A – patient discomfort or cosmetic concerns

Pain and cosmetic problems can arise in all types AVFA described in the classification system of Balaz et Bjorck [7]. Discomfort around the AVFA is an infrequent symptom and alternative sources of pain should first be excluded before attributing symptoms of pain to the aneurysm. Pain can result from peripheral nerve compression by the aneurysm but this diagnosis is made more complex by the frequency of associated uremic or diabetic polyneuropathy in the haemodialysis population [23]. Issues related to cosmetics from the AVFA are subjective in nature and are generally not considered an indication for intervention. If operative intervention is considered for an otherwise asymptomatic aneurysm on the basis of cosmetics, this decision must be weighed carefully against potential post-operative complications. The risks of intervention including bleeding, thrombosis and loss of access should be explicitly explained to the patient.

Group B – risk of bleeding

With the exception of type IV AVFA, bleeding may occur in all types of AVFA in the classification system of Balaz et Bjorck [7]. Aneurysmal bleeding may result from rupture or trauma but most often it occurs more indolently at the conclusion of a dialysis session following removal of the haemodialysis needle. Factors that increase the risk of bleeding include degradation of the skin, brisk expansion of the aneurysm, high blood pressure, intra-access pressure and use of anticoagulants. If these warning signs exist or the patient is actively bleeding, immediate intervention is required.

Group C – low flow issues

Low flow occurs in Balaz et Bjorck type II and type III AVFA [7]. Low flow resulting from impaired arterial inflow or venous outflow stenosis leads to inadequate dialysis. A haemodynamically significant venous outflow stenosis is usually defined on angiography or ultrasonography as greater than 50% reduction in vessel diameter [1]. Treatment of AVFA with low flow aims to address the stenosis, usually by means of angioplasty of the stenosis in the first instance. If concomitant stenoses with the aforementioned high-risk features for bleeding are present then an open surgical approach is recommended.

Group D – high flow issues with steal syndrome or risk of high output cardiac failure

Distal perfusion may become compromised by a high flow AVFA or high-output cardiac failure may result, particularly in patients with coronary artery disease [24]. Ischemia of the hand, known as high flow steal syndrome, can also be caused by stenosis proximal or distal to the access anastomosis. However, this article focuses on steal syndrome secondary to high flow. Although it should be noted that steal syndrome is not always accompanied by aneurysm formation.

Steal syndrome

Steal syndrome can be graded from 0 to 3 [8]:
Grade 0: No steal present.
Grade 1: Mild – peripheral coolness, minimal symptoms, augmented flow with occlusion of access.
Grade 2: Moderate – intermittent ischemia only during dialysis sessions or claudication.
Grade 3: Severe – ischemic rest pain or loss of tissue.

Steal syndrome is a potentially limb threatening complication and if it is clinically significant it should be promptly diagnosed with a view to rapid treatment. The objectives of managing steal syndrome are re-establishment of antegrade flow to maintain distal perfusion and safeguard use of the AVA for dialysis. A cut-off value for when the high flow AVF presents a significant risk for the development of steal syndrome has not been clearly defined. Intervention is occasionally needed for grade 2 steal syndrome and obligatory for grade 3 steal syndrome.

High output cardiac failure

An AVFA with high flow may result in substantial increase in cardiac index, heart re-modelling secondary to volume overload and congestive cardiac failure [25,26]. Clinical signs consistent with systemic or pulmonary congestion accompanied by a cardiac output > 8 L/min or cardiac index > 3,9 L/min/m^2 is an accepted definition of high output cardiac failure [27].

The majority of AVA have an upper limit of flow of 2.5 L/min, which surpasses the flow required to produce reversible symptoms [28]. In a study of stable patients on long-term haemodialysis, Pandeya et al. presented the notion of cardiopulmonary recirculation using the ratio of access flow rate/cardiac output [29]. They reported a mean access flow of 1.6 L/min and a mean cardiac output of 7.2 L/min, hence designating a mean cardiopulmonary recirculation of 22%. MacRae et al. [30] proposed that harmful effects of high access flow can occur when the access flow rate is higher than 3 L/min or when access flow rate/cardiac output is 30% or higher. Wijnen et al. [31] reported that access flow rate was significantly and positively associated to cardiac output and cardiac index, and inversely correlated to peripheral vascular resistance. However, when a high flow AVFA is at risk for the development of high output cardiac failure has not been specifically defined as yet and treatment guidelines are required.

Previous studies have demonstrated that AVF closure reduces left ventricular diameter and mass [32–34]. However, it is unclear what effect AVF reduction has on heart remodelling. In a published series of thirty patients with AVF flow ≥ 1.5 L/min treated by aneurysmorrhaphy with external mesh prosthesis, Wohlfahrt et al. [35] evaluated the effect of reduction of AVF flow on cardiac remodelling. Reverse cardiac remodelling (decreased LV end-diastolic diameter and mass, left atrial and right ventricular diameter and pulmonary pressure) was observed only in patients with elevated cardiac index (≥ 3.9 L/min/m^2). The study concluded that the effect of AVF reduction on heart remodelling is dependent on the pre-operative cardiac index and not on the access flow rate. This study therefore intimates that increased cardiac index may be the most significant factor to consider when contemplating AVF reduction techniques in patients with a high flow AVF. As such, in the majority of patients, AVF reduction/closure may not be required and intervention can be reserved for patients who develop adverse cardiac remodelling or congestive cardiac failure.

It is thus our recommendation that treatment should be offered to all patients with access flow rate >2.5 L/min who have either stage C or stage D heart failure as described by the American College of Cardiology/American Heart Association (ACC/AHA) [36]. In terms of pre-emptive treatment in asymptomatic patients, our recommendation is to intervene in patients with a high flow AVFA only when either there is an access flow rate >3.0 L/min or cardiopulmonary recirculation ≥30% or cardiac index >3.9 L/min/m^2. Conversely, no intervention is required in asymptomatic patients with high flow AVFA with normal cardiac index and no LV dilation.

Techniques for the treatment of arteriovenous aneurysm

Various techniques for treating symptomatic AVFA have been described in the literature. The principle methods of surgical intervention are resection with substitution, remodelling techniques and ligation. Stent grafting is the endovascular technique of choice. However, despite the array of options available, the majority of these techniques have only been reported as individual case studies or small case series. Furthermore, to date none of the described techniques have been scrutinised in a head to head fashion, either in a randomized control trial or prospective non-randomised study.

Resection and/or substitution techniques

Resecting the aneurysmal component of the AVF and forming a fresh anastomosis more proximally can deal with post-anastomotic aneurysms situated within a short venous segment. All types of AVFA can be managed in this fashion, even those with thrombosis. However, the proximal and distal venous segments must be free from thrombosis to allow construction of an anastomosis either in an end-to-end or end-to-side fashion. The principle benefit of this approach is that a healthy section of vein is available straightaway for use in dialysis. If a substitution is required, either prosthetic material or autologous vein (e.g. great saphenous vein) can be utilised. Prosthetic conduits have the advantage that they can be cannulated earlier than autogenous grafts but they car-

ry a higher risk of infection and thrombosis. When using substitution techniques, either with prosthetic or venous conduits, 12-month patency rates of 47–100% have been reported [20,37,38].

Remodelling techniques

There are a variety of remodelling techniques (aneurysmorrhaphy, reinforced aneurysmorrhaphy, plication) that utilise native vein to maintain the character of the fistula. One method is aneurysmorrhaphy, which involves resection of part of the aneurysm sac. Another method is reinforced aneurysmorrhaphy, which involves resection of the aneurysm and supporting parts of the vein followed by mesh external prosthesis. The rationale for implantation of external mesh prosthesis on the surface of the vein to be repaired is that it reportedly reduces shear stress on the wall of the vein. This could result in less turbulent blood flow, endothelial injury, and thrombus formation [39,40]. Balaz et al. [41] first reported the reinforced aneurysmorrhaphy with polyester mesh tube. This novel approach is feasible to treat multiple extensive AVFAs but since cannulation of the repaired fistulae has to be delayed for 4–6 weeks of recovery a temporary tunnelled dialysis catheter is needed. The 1-year primary patency results of remodelling techniques with and without support appear similar (86% [42] vs. 80–93% [19,43]). Primary patency rates of 88%, 84%, and 69% at 1, 2 and 3 years, respectively have also been reported using stapled aneurysmorrhaphy [18]. Going forward there is a need for comparative studies to assess the efficacy of different aneurysmorrhaphy techniques as well as the need for external support.

Endovascular techniques

In keeping with the upsurge in endovascular techniques in vascular surgery in general there has been an emergence of endovascular approaches to vascular access problems. First reported in 2002 [44], proponents of covered stent graft insertion to treat AVFAs, cite the fact that the procedure can be performed on an outpatient basis and does not require interruption of dialysis since early venepuncture is still possible [45,46]. In a patient with active bleeding, treatment with an endovascular stent graft can also secure haemostasis. Although the reported one year patency rates of 87% [16] are comparable to the previously described remodelling techniques, criticism of stent graft insertion centre on the increased costs of the procedure and lack of utility in specific subgroups of patients. For instance, situations where the endovascular approach is not suitable include patients with aneurysms close to the anastomosis, steal syndrome and large aneurysms lacking an adequate landing zone for sealing the stent graft.

Surgical techniques for high flow AVFA

Treatment options are similar for steal syndrome and high output cardiac failure. These include flow-limiting procedures (e.g. plication, banding and minimally invasive limited ligation endoluminal-assisted revision [MILLER]), distal revascularization with interval ligation (DRIL), proximalization of arterial inflow (PAI), revascularization using distal inflow (RUDI), transposition of radial artery, proximal radial artery ligation (PRAL) and ligation of the AVF.

While several studies have reported on the treatment of high flow vascular access, only a few of them assess high flow vascular access in the presence of an aneurysm. Aneurysmorrhaphy with external mesh can significantly reduce flow rates [35,43] and precipitate reverse cardiac remodelling with flow rates ≥ 1.5 L/min in patients with elevated cardiac index (≥ 3.9 L/min/m^2) [35]. However, aneurysmorrhaphy has proven unsuccessful with flow rates >2.5 L/min [19]. Therefore our recommendation is that for high flow AVFA sites in the upper arm, aneurysmorrhaphy and anastomosis relocation to forearm arteries is performed, with aneurysmorrhaphy and reduction of the anastomosis used for those sited in the forearm.

Ligation

Ligation is technically straightforward, and can successfully treat steal syndrome or high output cardiac failure but unlike surgical revision techniques, it does not preserve vascular access. When other revision techniques have failed, ligation should be considered as a last option. Haemorrhage from an AVFA is a potentially fatal complication [47], and if acute bleeding is associated with haemorrhagic shock then immediate ligation of the AVF should be performed, with reconstruction deferred until after the patient has recovered. A further indication for ligation is a high flow AVFA in patients who have received a kidney transplant with expected good long-term graft function.

Summary

An AVFA is defined by an enlargement of all three vessel layers to a diameter greater than 18 mm. In asymptomatic patients conservative management is appropriate. The principle indications for intervention in symptomatic patients are pain, bleeding prevention and low or high flow. The diameter of AVFA and issues purely related to cosmetics are not indications for intervention. Despite a lack of strong evidence base concerning which surgical treatment option is optimal, in order to preserve the nature of the AVF we recommend techniques utilizing the native vein as first-line. According to the literature, the best long-term results are achieved by using aneurysmorrhaphy with or without a prosthetic support. Insertion of a stent graft or ligation of AVFA is recommended for emergent treatment in actively bleeding patients.

Conflict of interest
None declared.

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Authors state that the research was conducted according to ethical standards.

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