Prosthetic vascular access

The prosthetic vascular accesses will always be carried out after having exhausted all the possibilities of executing autologous accesses. They have the advantage of having a relatively simple technique, not requiring a prolonged maturation period and being easy to puncture as the most convenient area to tunnel them can be freely chosen. However, they also have the highest complication rate and require a greater number of revisions, with the subsequent economic cost and impairment of the patient's quality of life.

Different prosthetic materials have been used for vascular accesses. The most commonly used material is ePTFE (expanded polytetrafluoroethylene), although other materials such as polyurethane are also used. Xenografts derived from bovine mesenteric vein or alografts with femoral vein or internal saphenous vein have also been used. The ideal calibre for these channels is not clear; but what nobody questions is that with a lumen of less than 6 mm, there are considerable probabilities of occlusion, and if it is greater than 6 mm, the possibilities increase of developing an arterial steal syndrome or cardiac volume overload. To avoid these situations, tapered prostheses with a greater calibre at entry and smaller calibre at exit have been used. The length of the prosthesis varies between 20 and 40 cm in order to provide a minimum of 15 cm trajectory to carry out the punctures. They can be implanted in a straight or U-shaped position. Whenever a prosthesis is implanted, carrying out an antibiotic prophylaxis is inexcusable given that the prosthetic materials are especially susceptible to infection during implantation and they do not possess the defence mechanisms that protect the autologous fistulae.

Alternative vascular access

This group includes those vascular accesses that are not carried out on a routine basis, and those that must be resorted to when the normal autologous or prosthetic vascular access possibilities have been exhausted. This type of access can be carried out on the anterior side of the thorax taking the axillary or subclavian artery as the donor artery and the axillary or jugular vein as the receiving vein. The position of the prosthesis may be U-shaped, tunnelling the prosthesis subcutaneously on the pectoralis major muscle or crossing over the anterior side of the sternum from one axillary vessel to another contralateral one.

The abdominal vessels can also be resorted to, where the external iliac artery or the common femoral artery is the donor artery and the iliac vein or inferior cava is the receiving vein. In both cases an attempt must be made to carry out the anastomoses as distal as possible to minimise the risk of steal syndrome. The prosthesis can be tunnelled subcutaneously in the iliac fossa or on the antero-external side of the thigh.

Dysfunction due to vascular access stenosis

When the dysfunction of a vascular access is detected early on, the stenosis can be identified and corrected, reducing the risk of thrombosis and increasing the survival of the vascular access.

The routine physical examination before and after each haemodialysis session may be useful in detecting access stenosis. The existence of stenosis can be suspected by observation, palpation (thrust) and auscultation (murmur) of the trajectory of an arteriovenous access. In an autologous fistula, stenosis can be appreciated as a depressed anastomosis and the stenotic area will have a greater pulse, whilst the venous trajectory after the stenosis will have less pressure and will even have lost the thrill or will have collapsed. A discontinuous and sharp murmur may be auscultated in the stenosis area, which may indicate critical stenosis. The venous segment proximal to the stenosis may develop a venous aneurysm. These stenoses in the venous trajectories of autologous fistulae may be due to fibrosis of the venous wall, caused by repeated punctures at the same point during the haemodialysis sessions. The existence of a severe and progressive oedema, with cutaneous cyanosis and collateral circulation, in an extremity that bears an arteriovenous access, may mean the existence of high venous pressure due to stenosis or venous occlusion proximal to the access. Furthermore, depending on the location of the oedema, it will be possible to infer which access drainage vein contains the obstruction.

Likewise, during the haemodialysis sessions, attention must be paid to certain situations that may be indicative of access dysfunction. Difficulty in the cannulation of the access may indicate a lack of maturation of the access. Changes in pre-pump blood pressure with respect to previous sessions, as well as the impossibility of maintaining normal blood flows in three consecutive sessions, may indicate a reduction of the access flow. An increase in venous pressure during the haemodialysis at normal flows may be indicative of venous stenosis at proximal level. Increased bleeding time after removing the needles, and after ruling out coagulation alterations, may be caused by an increase
in the intraluminal pressure, secondary to stenosis after the puncture area.

Studying dynamic venous pressure (DVP) to detect stenosis is more useful in prostheses than in autologous fistulae. This is due to the fact that, in autologous fistulae, the majority of stenoses are located in the anastomosis, causing a reduction in flow whilst stenoses in prostatic accesses are primarily located in the venous anastomosis, generating a resistance that translates into an increase of venous pressure. Moreover, autologous fistulae, unlike the prostatic ones, have the ability to develop collateral circulation that may compensate the venous return without a retrograde increase in pressure. Three consecutive determinations of over 150 mmHg and/or an increase of more than 25% the basal value is considered suggestive of stenosis, although this may vary depending on the conditions of the haemodialysis (needle calibre, pump flow, machines and lines used).

The static or intra-access venous pressure (IAVP), unlike the DVP does not depend on variations of the pump flow or on the calibre or position of the needles. It is only influenced by the systemic blood pressure. It is considered normal when the IAVP is less than 50% the average blood pressure determined simultaneously. As mentioned above, it is more useful in prostatic accesses.

Vascular access recirculation is a dysfunction marker of the vascular access in venous return obstructions; depending on the measurement method, recirculation values of more than 5-10% are considered altered.

According to the aforementioned, if a dysfunction of the vascular access is suspected, and having an idea of what type of access stenosis exists, this can be confirmed by means of the most adequate imaging technique in each case: Eco-Doppler, fistulography or magnetic resonance.

Treatment of arteriovenous fistula stenoses is considered adequate when the diameter has been reduced by more than 50% and it is associated with alterations in the surveillance and monitoring parameters of the access.

The most frequent location of stenoses in autologous fistulae is near to the arteriovenous anastomosis, above all in radiocephalic ones due to intimal hyperplasia or residual technical effect. In all other cases, it affects the venous trajectory or the points of union between the cephalic and the subclavian, in humeral-cephalic fistulae, or between the basilic and axillary in humeral-basilic fistulae. Stenoses in the arterial segment are rare. Surgical treatment in stenoses proximal to the anastomosis consists in the reanastomosis of the healthy venous trajectory in the artery, proximally to the previous anastomosis. When the stenosis affects the venous trajectory and if the stenotic area is short, sometimes the exeresis of this segment and an end-to-end direct reanastomosis of the venous trajectory can be carried out. If the stenosis is longer, an angioplasty with patch can be offered or else a bypass graft that replaces the stenotic segment. This graft can be carried out with a vein from the actual patient, a cryo-preserved vein or PTFE.

Stenoses in arteriovenous prostheses are usually located in the venous anastomosis, due to intimal hyperplasia or in the central veins, in connection with the prior use of central catheters. Stenoses in the trajectory of the prostheses are not very frequent, and are usually related to the deterioration of the PTFE after multiple punctures, so a change of prosthesis is preferable at times. In stenoses due to intimal hyperplasia of venous anastomosis, the surgical treatment consists in angioplasty with patch or in the extension of the prosthesis to the proximal area of the vein, which is healthy. In central venous stenoses a bypass can be offered to the subclavian or jugular vein.

### Vascular access thrombosis

When, despite the aforementioned, vascular access thrombosis occurs, this means that it will be impossible for it to be used for haemodialysis. This thrombosis may not be preceded by the dysfunction of the access, but rather be related to phenomena of low blood pressure, infection, previous formation of aneurysms and pseudoaneurysms, or hypercoagulability of the patient. All of this will have to be taken into account to carry out a correct surgical intervention.

Thrombosis of the vascular access must be evaluated as an emergency and the action time available is going to depend on two circumstances: firstly, it must be taken into account that the fixation of the thrombus to the wall and the secondary thrombosis increases with the passing of time; and secondly, and perhaps most importantly, the delay permitted by the dialytic requirements of the actual patient must be assessed. Any delay in the surgical treatment of the access thrombosis must be prevented from entailing the need to place a temporary catheter, as these are not free from acute iatrogenic type complications. They also favour the subsequent appearance of central stenoses which may hinder the future execution of accesses in this extremity. In the majority of the cases, if the access was already useful for the dialysis, immediate puncture will be possible. Likewise, if the access has still not been punctured and the patient is dialysed by catheter, the intervention can be deferred.

The surgical treatment after a thrombosis is the Fogarty balloon thrombectomy. In the case of autologous fistulae, this thrombectomy is only efficient in the immediate postoperative moment, when the thrombosis is associated with technical defects in the anastomosis. In arteriovenous prostheses, the thrombectomy may be efficient even several days after the thrombosis. If the thrombosis of the prosthesis has been related to an episode of high blood pressure or of hypercoagulability, a simple thrombectomy may be sufficient. However if the thrombosis is associated with a previous dysfunction, with a venous stenosis, it must be solved in the same surgical act, to avoid early re-thrombosis. After the prosthesis thrombectomy, it is recommendable to carry out an intraoperative phlebography to evaluate the existence of an intimal hyperplasia in the venous anastomosis. In this case it is compulsory to carry out an angioplasty with patch or prolong the prosthesis to a more proximal area of the vein, via a new prosthesis segment. If the prosthesis thrombosis has been related to a central venous stenosis, a balloon angioplasty can be carried out in the same surgical act.
**Aneurysms and pseudoaneurysms**

Approximately 5 to 8% of arteriovenous fistulae develop aneurysms.

Apart from producing discomfort and aesthetic impairment of the extremity they may be a potential cause of access thrombosis, distal embolisation or erosion of the subjacent skin with infection and/or bleeding.

A genuine aneurysm is a dilation of a territory of the fistula due to degeneration of the collagen matrix of the vascular wall, but keeping the venous or arterial wall intact, that is, there is no loss of continuity of the wall. It is more frequent to see them in venous trajectories of long-term autologous fistulae. We can also find venous aneurysms in the arteriovenous anastomosis as a result of the blood hyper-afflux. Genuine arterial aneurysms have sporadically been described, mainly of the axillary or humeral artery, following the ligature of an elbow fistula. Genuine aneurysms are not very frequent in prosthetic accesses.

Pseudoaneurysms are pulsatile or expandable dilations caused by persistent subcutaneous bleeding through a loss of continuity of the fistula wall or of the prosthesis. They can be found both in autologous and prosthetic fistulae, and they can also affect anastomoses.

The etiopathogeny of the aneurysms and pseudoaneurysms can be caused by careless handling of the vein, or by a bad anastomosis technique during its surgical construction. But, the appearance of aneurysms and pseudoaneurysms may be due to an incorrect cannulation during the haemodialysis sessions. Repeated punctures at one same point can cause the weakening and loss of continuity of the venous or prosthetic wall, giving rise to a pseudoaneurysm, or else they can cause a stenosis area that will favour the appearance of a proximal venous aneurysm.

The diagnosis is mainly clinical, as a progressively growing, pulsatile and expandable mass is seen in the vascular access trajectory, and at times, the patient refers to local swelling or pain. To support the clinical diagnosis, the EcoDoppler, fistulography or magnetic resonance can be used.

The indications of treatment of aneurysms and pseudoaneurysms in vascular accesses are: the existence of symptoms due to local compression or thrombosis, the impairment of the skin on the fistula with cutaneous necrosis and risk of bleeding and/or infection, the points available for puncture are limited, when the aneurysm encompasses the arterial anastomosis and the rapid growth of a pseudoaneurysm.

The treatment must preferably be carried out and it usually consists in the exclusion of the aneurysm and interposition of a new graft (autologous or prosthetic). In general, the complete exeresis of the aneurysmatic trajectory is not necessary, and at times it is not advisable, as it is usually closely adhered to the skin. The new graft can be implanted adjacent to the old segment. The isolated plicature of the aneurysmatic trajectory usually occurs early on, so, in general, the exclusion and interposition of the graft are preferred.

The dilation of the venous trajectory in long-evolution fistulae are nothing more than a mere aesthetic problem and surgical correction is not advised, unless associated with stenosis, necrosis or cutaneous disorders with a risk of breakage. In all other cases, only the venipuncture must be avoided in these dilations.

**Haemorrhage**

Postoperative bleeding of vascular accesses can appear early or later on. Early bleeding can occur through the suture line of the arteriovenous or prosthetic anastomosis in relation to a bad surgical technique, and sometimes favoured by the effect of anticoagulant agents administered during the dialysis. More often, prolonged late bleeding appears in the puncture areas during dialysis; in these cases, after ruling out alterations of the coagulation parameters, the most frequent cause is retrograde high blood pressure generated by stenosis in the fistula drainage trajectory.

The treatment will depend on the etiology of the haemorrhage. The coagulation of the patients must be improved whenever possible. Likewise, a meticulous surgical technique and a thorough intraoperative haemostasia must be carried out. In the case of early haemorrhage through the suture line of the anastomosis, an early revision may be necessary to control the haemorrhage and avoid the access thrombosis due to extrinsic compression. And finally, the stenosis in the fistula drainage trajectory must be identified early on, via the adequate monitoring of the flows and pressures during the haemodialysis sessions, to prevent secondary bleeding to a retrograde high blood pressure.

**Access breakage**

The spontaneous breakage of the access is an infrequent complication. It is more than likely that it is associated with the previous formation of pseudoaneurysms and degeneration of the subjacent skin, whose breakage can cause an acute haemorrhage. Another possible cause of acute bleeding can be an infection in the fistula trajectory or for it to affect the anastomosis, with the subsequent risk of dehiscence of the suture. More rarely, a direct trauma to the fistula can cause acute bleeding.

It requires urgent surgical action, at times with ligature of the access or with exclusion of the pseudoaneurysm, if any, and reconstruction of the access when possible.

**Access infection**

Infection is the second cause of vascular access loss. Furthermore, infectious complications are responsible for 15 to 30% of all deaths in patients in haemodialysis. In this sense, the presence of a vascular access is a risk factor of local infection and a triggering agent of bacteraemia, contributing to the mortality of these patients.

Catheters are the elements that become infected more often, followed by prosthetic accesses and finally autologous accesses. Thus, it is recommended to give priority to autologous accesses rather than to prosthetic accesses.
The germs that are most frequently involved are gram-positive cocci, especially staphylococcus, both aureus and negative coagulase. Enterococci have been found in 10 to 20% of the infections and gram-negative in 33%.

The infection of an autologous fistula is not very frequent and may be secondary to an infection of the surgical wound or to a lack of asepsis during the dialysis.

This can occur as diffuse cellulite; in this case antibiotic treatment for 2 to 4 weeks is usually sufficient. If it is associated with an abscess, this may have to be drained, prolonging the antibiotic treatment. Recurrent infections or those that affect the anastomosis may require ligature of the access. And finally, if a serious infection of the vascular wall occurs with subsequent regeneration, the exclusion of the area with bypass with autologous tissue through healthy tissue or the ligature of the access would be necessary.

Prosthetic accesses are, after the central catheters, the ones that present greater incidence of infectious complications.

Prosthetic infection may occur due to contamination during the surgical act, due to repeated punctures during haemodialysis or transient bacteraemia due to remote infections. The location that is infected most frequently is the femoral loop, and there are certain factors of the patient, such as obesity, diabetes, hypoproteinaemia, immunodeficiency or bad personal hygiene, which are considered risk factors. Surgical revisions to prolong the secondary permeability of the access also increase the possibilities of infection.

Complications of the infection of a prosthetic access are not limited to systemic implications of fever, sepsis and remote affection (endocarditis, arthritis, osteomyelitis...), but rather, they can also derive in access thrombosis, anastomotic dehiscence and haemorrhage, or the formation of pseudoaneurysms.

As a general rule, the infection of a prosthetic access entails the need to remove all the prosthetic material, with arterial closure with autologous material.

**Distal ischemia**

Although an inversion of the flow is observed from the distal artery to the anastomosis in 75% of the autologous accesses and in 90% of the prosthetic accesses, the symptomatic steal syndrome is much less common.

10% of the patients experience slight coldness as well as pins and needles in the fingers; however, the majority of the symptoms are spontaneously resolved in the first few weeks. Distal ischemia only affects 3 to 5% of patients with AV accesses, with decreasing frequency the more distal the access, and less in autologous accesses than in prosthetic accesses (<1% in distal autologous accesses and 9% in prosthetic grafts).

There are certain risks or favouring factors, such as peripheral arteriopathy, diabetes, advanced age, previous ipsilateral fistulae, creation of high-flow fistulae, the use of the brachial artery as a donor artery instead of more distal arteries, and the use of prosthetic grafts rather than autologous fistulae.

Between half and two thirds of the patients who develop a vascular steal syndrome do so within the first 30 days. The symptoms may appear during the dialysis or worsen with it. The symptoms are numbness and progressive pain, coldness, pallor, decrease of sensitivity, ischemic ulcers, gangrene and muscular atrophy. We can distinguish four stages that go from less to greater intensity. In stage I, there is pallor and/or coldness of the hand, without obvious pain. In Stage II the pain appears during exercise and/or HD. In Stage III there is pain at rest and in Stage IV ulcers, necrosis or gangrene appear. Ischemias in stage I and the majority of those of stage II regress or improve spontaneously and do not require treatment. In stages III and IV, the ischemia is serious and requires treatment.

The diagnosis is carried out clinically and via physical examination. The distal pulses are usually absent whilst the fistula remains permeable and they are usually recuperated during manual compression. A more complicated evaluation of the ischemia is carried out by non-invasive diagnostic methods such as digital pletismography, measurement of the digital pressure and of the finger-arm index, the eco-Doppler and the transcutaneous measurement of O2. All these tests must be carried out with the functioning access and during its compression. Ischemia is considered to be serious when the digital pressure is 50 mmHg or less or with a finger-arm index of < 0.6. If arterial lesions are suspected, an arteriography may be carried out with and without compression of the fistula.

No reliable preoperative test has been evidenced to predict who will develop steal: the most important factor to prevent it is a correct preoperative evaluation of the risk factors, an adequate physical examination and an adequate surgical technique.

Within the treatment options, the ligature of the access is the safest method to eliminate the phenomena of steal, but obviously this means eliminating a functioning access and leads to the necessity to create a new one and the temporary placement of a catheter.

Other treatment options are to limit the flow through the fistula or the distal re-vascularisation with ligature at intervals: the DRIL procedure.

In Banding, an attempt is made to limit the flow to the fistula, increasing its resistance by narrowing the efferent lumen. This narrowing can be carried out by partial ligature or by a circumferential patch, generally of PTFE, around the vein close to the anastomosis. This second option could be more adequate, as a short stenosis, like the one achieved with simple ligature, produces a slight reduction of the flow until a critical stenosis is reached: at this point, the resistance would exponentially increase and the turbulence generated would favour thrombosis. The broader band could generate less turbulence. However, the long-term results of banding are disappointing, as either sufficient reduction of the flow is not achieved for the distal ischemia to disappear or the fistula ends up being thrombosed.

The DRIL technique consists in creating a bypass graft from the native artery proximal to the arteriovenous anastomosis to an artery that is distal to this anastomosis, and attaching this same artery distal to the arteriovenous anastomosis, but proximal to the distal anastomosis of the bypass graft. This bypass acts as a low resistance collateral in parallel, reducing the resistance of the peripheral circulation; on reducing the resistance proportion between the periph-
eral circulation and the access, the blood fraction towards the fistula decreases and the peripheral one increases.

Before carrying out a DRIL, it is advisable to rule out both input and output arterial stenoses. Certain technical factors must be taken into account for the DRIL technique to be successful. In the immediately proximal arterial segment to the arteriovenous anastomosis, there is a low pressure area (pressure background) due to the great capacitance of the venous output flow. Locating the origin of the bypass graft 3 to 5 cm above the AV anastomosis is sufficient to avoid this low-pressure background. Although it is preferable to use autologous material, the procedure has been successfully carried out with prosthetics. The output anastomosis must be carried out in the dominant distal artery.

High venous pressure

High venous pressure may cause an important oedema of the extremity. This is usually caused by stenosis or obstruction of the veins proximal to the fistula, often related to the previous use of central catheters.

The treatment possibilities include: ligature of the access, simple angioplasty or with stent in the central stenosis or the execution of an axillary jugular bypass.

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Venous catheters: problems and measures in nursing care
Pilar Urzainqui-Laborda
Nephrology, Navarre Hospital Complex, Virgen del Camino Hospital, Pamplona, Spain

The quality of vascular access (VA) is a determining factor in clinical results of patients receiving periodic haemodialysis treatment (HD). The complications arising from VA dysfunction constitute one of the main causes of morbidity and mortality amongst patients and substantially increase health care costs.

The need for vascular access is as old as HD itself, given that access to the bloodstream is necessary to eliminate toxic substances.

Ideal VA should bring together at least three requirements: i) a safe, continuous approach to the vascular system; ii) provision of sufficient flow to supply an adequate HD dosage, and iii) present no complications.

Risk factors

Intravascular catheters are plastic devices that provide access to the intravascular compartment at a central level, they vary in design and structure according to if they are temporary (days) or permanent (weeks, months) as well as in the material they are made of and the reason why they are implanted.

These devices have been of great clinical use as they enable fast, safe access to the bloodstream to administer medications, treatments fluids and parenteral nutrition. However, they are not exempt from risks such as infectious or mechanical complications arising from their use.

1. Infection from use of central vascular catheters (CVC) constitutes one of the main complications and the main cause of primary nosocomial bloodstream infection. The incidence of infection attributable to catheter use varies amongst hospitals and is about 4-5 events per 1000 days of catheterisation, and an average mortality of 3% is linked to this sizeable prevalence.1,2

More is starting to be known about the mechanism through which CVC contamination is produced thanks to the development of experimental models with animals. Studies using electron microscopes show that the immense majority of catheters, even those where the quantitative culture gives negative results, are colonised by microorganisms. The germs are usually immersed in a biofilm sticking to the interior and exterior of the catheter formed by the interaction of the catheter wall with host proteins.

Appearance of the film is very early, even in the first 24 hours after insertion, but is not necessarily a determining factor in the appearance of infection. When the density of microorganisms reaches a certain level, the probability of catheter related sepsis increases considerably.1,2

2. Vascular access dysfunction causes the highest consumption of resources in the population with chronic kidney disease (CKD). Experts consider that current standards for pump flow (Qb), venous pressure (Pv), molecular clearance and dialysis time could be improved and reductions in clinical complications can be reduced by enhancements in the process, thereby increasing the quality of life for patients and reducing the health care costs caused by this problem.

Reed et al, has described all the variables that play a part in CVC complications in a microbiological and ultrastructural study.2,3

a) Characteristics of patient
- Underlying disease
- Immune system compromise
b) Personnel training
- Aseptic measures
- Choice of catheter
c) Insertion of catheter
- Preparation of implant site
- Choice of site
- Insertion technique
- Tunnelling
d) Handling
- Management
- Rescue
e) Care of insertion site
- Skin antiseptic
- Type of dressing
- Local anti-bacterial application
f) Clinical monitoring
g) Connections and perfusions
- Periodic washing and lumen sealing
- Impregnation of catheter with antiseptics