Repeated Failure of Arterio-Venous Haemodialysis Access: (Causes and possible solutions)

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How to Cite This Article

ORIGINAL ARTICLE

Repeated Failure of Arteriovenous Hemodialysis Access: Causes and Possible Solutions

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Abstract

Background: In 1960, Quinton and Scribner developed an external shunt that allowed repeated accesses to the circulation, making chronic hemodialysis a viable option for treating end-stage renal disease. Patients may now be kept on dialysis for decades owing to the improvement of vascular access methods and equipment.

Aim: The aim was to evaluate the most possible causes behind repeated arteriovenous access failures in patients with end-stage renal disease.

Patients and methods: This retrospective, nonrandomized study was carried out at the Vascular Surgery Department of Al-Azhar University Hospitals (Al-Hussein and Sayed Glaal Hospitals).

Results: Access thrombosis comes first as a cause of access failure, as it is responsible for 41% of all failed accesses. Among the cases of access thrombosis, 25.5% were associated with access stenosis, 10.5% with hypotension, and 2.5% with external compression. The commonest cause of access stenosis was hypotension (14%) followed by hypotension together with central venous stenosis (4.5%) and then central venous stenosis alone (2.5%). Failure of maturation comes next to access thrombosis, and it is responsible for 35% of all access failures. Among the cases of failure of maturation, 16% were due to small vein diameter (<2.5 mm), 9% associated with hypotension, and 7% with hypotension together with central venous stenosis.

Conclusion: Management of repeated arteriovenous access failure needs changing the attitude of vascular surgeons to be more conservative and based on access salvage protocols (access preservation), directed to correction of early access failure and the predisposing factors behind it.

Keywords: Arteriovenous, End-stage kidney disease, Hemodialysis access

1. Introduction

A rising number of diabetics and an aging population are contributing to an increase in the incidence of end-stage kidney disease (ESKD).[1] More than 2 million patients worldwide use hemodialysis, making it the most prevalent kind of kidney replacement treatment.[2,3] Functional vascular access is necessary for effective hemodialysis to continue.[4] There are three types of vascular accesses for hemodialysis: central venous catheter (CVC), arteriovenous graft (AVG), and arteriovenous fistula (AVF). An end-or-side vein-to-artery anastomosis is used to link a native artery and vein to form the AVF. A prosthetic graft [often made of polytetrafluorethylene (PTFE)] is placed between an artery and a vein to form an AVG.[5] An adequate blood flow rate, minimal flow resistance, a low incidence of problems, and, for AVF and AVG, simplicity of cannulation are essential components of such an access.

As a mature native AVF has better long-term results than a synthetic AVG or CVC, such as lower rates of thrombosis, infection, and interventions to preserve patency, it is seen as being preferable.[6] On the contrary, between 20 and 60% of AVFs fail to

mature to an adequate caliber to permit repeat cannulation and provide adequate blood flow for hemodialysis, preventing timely usability of the AVF for hemodialysis. This is due to early thrombosis, neointimal hyperplasia formation, and insufficient vasodilation (outward remodeling).[6] AVGs may be utilized immediately after access formation, but over the long term, they have a greater chance of developing thrombosis, infection, and venous stenosis than a working AVF.[7] In comparison with a working AVF, more than 50% of AVGs thrombose after a year of formation and need much more procedures to maintain patency.[7,8] CVCs may be used right away after insertion, but prolonged usage is not recommended owing to the elevated risks of thrombosis, catheter-associated bacteremia, and insufficient solute clearance.[9]

A significant contributor to morbidity, death, and excessive health care expenses is vascular access failure.[6] Vascular access function is, in fact, regarded by medical experts, patients, and caregivers as a key goal for hemodialysis research and clinical practice.[10] Inadequate outward remodeling and neointimal hyperplasia leading to venous stenosis have been recognized as the two main causes of dialysis vascular access failure in light of recent developments in our knowledge of the biology of vascular access and its dysfunction.[11] Owing to this understanding, various strategies to enhance and maintain vascular patency have been developed, including the identification of prospective therapeutic targets.[9]

Given the lack of a technical advancement that would considerably lower the morbidity and expense associated with vascular access, current efforts have concentrated on creating algorithms to better define patient selection criteria for each access technique.[12]

This study's primary objective was to evaluate the most possible causes behind repeated AV access failure in patients with ESRD and finding the relationship between those causes and the timing of access failure, and putting the possible solutions in the form of interventions and guidelines for those patients.

2. Patients and methods

A retrospective nonrandomized trial was performed on 100 patients with ESRD of both sexes of different ages on regular hemodialysis in Al-Hussien and Sayed Glal Hospitals and with repeated AV access failure, that is, two or more failed accesses, either autogenous or synthetic, in the upper limb. The study aimed for the following: (1) detect the most possible causes of repeated AV access failure of upper limb, both autogenous and synthetic, in patients with ESRD and their classification into predisposing and precipitating factors; (2) classify failure into early (within first 3 months) and late (more than 3 months); (3) find the relationship between timing of access failure and its cause; (4) evaluate the results of specific interventions applied on the patients to correct access failure such as surgical and percutaneous thrombectomy and balloon angioplasty when present; and (5) try to find the best algorism or guidelines for creation and saving the AV access in patients with ESRD and the last resorts for those with upper extremity access, precluded and completely failed access.

A total of 245 failed access cases were recorded, and according to the timing of failure, the failed accesses were categorized into two main groups.

(1) Accesses with early failure: 104 access (43%).
(2) Accesses with late failure: 141 access (57%).

Inclusion criteria were patients with ESRD on regular hemodialysis with two or more failed AV accesses in upper limb, either autogenous or synthetic.

2.1. Data collection and analysis

The failed access is usually identified by loss of thrill and machinery murmur over the access and inability to use it for sufficient and regular hemodialysis. Early access failure occurs within the first 3 months after access creation, and late access failure occurs after that. Failure of maturation is identified when the access fails to maintain considerable blood flow (either immediately or early postoperative during the first 3 months) with gradual decline in the thrill sufficient to incorporation of the access into the surrounding connective tissue and thickening (arterilization) of the venous wall. Failing access is a term used to identify early access dysfunction before complete failure.

The patients are evaluated through applying specific questionnaire, clinical examination, and reviewing their medical records regarding operative data of previous AV access operations and any interventions to correct access failure, laboratory investigations, and duplex ultrasonography of venous (and sometimes of arterial) tree and AV accesses of upper limb.

The questionnaire was designed to collect data about the presence of relevant systemic diseases, mainly, diabetes mellitus, hypertension (HTN), and

obesity; BMI more than 30 kg/m^2; systemic collagen diseases such as systemic lupus erythromatosis; hypercoagulable diseases; and the original cause of renal failure, especially when related to vascular diseases. Moreover, a detailed history was recorded about the distribution of the previously failed AV accesses, both autogenous and synthetic; the timing of their failure; and occurrence of postoperative AV access complications such as infection, aneurysmal dilatation, and hematoma formation. The patients was asked for the occurrence of any access dysfunction before complete failure such as elevated venous pressure and decreased arterial pressure (suction and inability of the access to bring the amount of blood adjusted on the dialysis machine), and inadequate dialysis session, which is a strong indicator of access stenosis. This pattern of access failure was differentiated from another pattern which occurs acutely and not preceded by dysfunction. History of access trauma and external compression also was excluded.

The patients were asked also for history of post-operative anticoagulant treatment and history of specific interventions applied to correct access failure such as surgical and percutaneous thrombectomy and their results in regaining the patency and functionality of the access and for how long, that is, primary patency rates.

The patients were evaluated clinically regarding arterial pulsations; blood pressure; the condition and accessibility of superficial veins, dilated or collapsed, compressible or cord like, near the surface or deep; the distribution of the superficial veins and failed accesses and their relation to the course of cephalic and basilic veins; signs of deep venous stenosis of upper limbs; unrelieved upper limb edema and congested veins in arm, upper chest wall, and neck; history of repeated insertion of CVCs in lower neck; and signs of upper limb ischemia such as brittle nails, brick red discoloration, coldness and pain, and thin unhealthy skin of upper extremity.

All the relevant available medical records and investigations of the patients were evaluated such as color duplex of the venous system of the upper limbs, both superficial and deep, including the failed AV accesses. It gave information about the actual distribution of superficial veins, their diameters and depth, and sites of stenotic segments if present. It also revealed the stenosis and occlusion related to the AV accesses and hemodynamic changes; slowing or absence of blood flow, within the access, and helps to exclude deep venous stenosis and thrombosis. Color duplex of arterial system of upper limbs is evaluated for one diabetic patient with arterial occlusive disease in upper limb. Coagulation profile, that is, prothrombin time and concentration; international normalized ratio; partial thromboplastin time; and platelets count were evaluated to exclude hypercoagulability. Moreover, the available operative data were evaluated.

The data collected about the patients and their failed accesses were arranged under two main groups: early and late failure. Each subsequent cause of access failure was analyzed separately.

2.2. Statistical analysis

Statistical Package for the Social Sciences (SPSS) version 22 for Windows was used to code, process, and analyze the obtained data (IBM SPSS Inc., Chicago, Illinois, USA). Using the Shapiro–Wilk test, the distribution of the data was examined for normality. Frequencies and relative percentages were employed to depict qualitative data. To determine differences between two or more sets of qualitative variables, the χ^2-test was used. Quantitative information was presented as mean ± SD. Two independent groups of normally distributed variables were compared utilizing the independent samples t-test (parametric data). P value less than 0.05 was regarded as significant.

3. Results

A retrospective nonrandomized study was carried on 100 patients with ESRD on regular hemodialysis (in Al-Hussien, Sayed Glal Hospitals) and with repeated AV access failure, with at least two failed accesses for each patient. The total number of failed accesses was 245, where 230 accesses were autogenous AV fistulae and 15 were synthetic bridge grafts in upper limb.

3.1. Demographic characteristic of studied patients

A total of 55 patients (55%) were females and 45 patients (45%) were males. Their age ranged from 20 to 80 years, with median age of 50 years and mean ± SD age of 50 ± 10 years. The causes of chronic Renal Failure (CRF) among studied patients are shown in Table 1.

A total of 230 accesses (94%) were autogenous and 15 accesses (6%) were synthetic (PTFE bridge graft). All the 15 failed synthetic accesses were brachial artery to ipsilateral axillary vein straight bridge grafts. Of the 230 failed autogenous fistulae, 124 (54%) were radiocephalic around wrist, 83 (36.5%) were brachiocephalic, 20 (8%) were brachiobasilic around elbow, two (1%) were natural vein transfer

Table 1. Age distribution and cause of CRF among studied patients.

	N (%)
Age group	
30—	10 (10)
40—	14 (14)
50—	25 (25)
60—	29 (29)
70—	19 (19)
70 and above	3 (3)
Cause of CRF	
Unknown cause	28 (28)
HTN	27 (27)
Obstructive uropathy	15 (15)
Congenital causes	10 (10)
Toxic nephritis	6 (6)
Rheumatic diseases	4 (4)
Prerenal causes	3 (3)
Chronic pyelonephritis	3 (3)
Nephrotic syndrome	2 (2)
Nephritic syndrome	1 (1)
DM	1 (1)
Total	100 (100)

DM, diabetes mellitus; HTN, hypertension.

(saphenous vein graft), and one (0.5%) was radio-basilic in mid forearm. A total of 104 (43%) of access failures occurred within the first 3 months after creation (early failure), and 141 (57%) occurred after 3 months of creation (late failure). Of the 104 early failures, 94 (90%) were autogenous and 10 (10%) were synthetic. Overall, 84% (87) of early failures were due to failure of maturation, 8.5% (nine) due to pseudoaneurysmal formation, 6.5% (seven) due to early thrombosis, and 1% (one case) was due to early infection. Of the 141 late failures, 96.5% (136) were autogenous and 3.5% (five) were synthetic. Moreover, 66% (94) of late failures were due to thrombosis, 16% (22) due to pseudoaneurysmal formation, 13% (18) due to aneurysmal formation, and 5% (seven) due to infection (Table 2).

Access thrombosis was responsible for 101 failures (41% of all access failures), of which 99 cases (40%) were autogenous and two cases (1%) were synthetic, and seven failures (3%) were early and 94 failures (38%) were late. A total of 64 cases (25.5%) of access thrombosis were associated with access stenosis, 26 cases (10.5%) with hypotension alone, six failures (2.5%) with external compression, three failures (1.5%) with wrong puncture, and two failures (1%) with central venous stenosis alone. In addition, 34 cases of access stenosis (14%) were associated with hypotension alone, 12 cases (4.5%) with hypotension and central venous stenosis, seven (2.5%) with central venous stenosis, seven cases (2.5%) with HTN, one case (0.5%) with arterial occlusive disease (in diabetic patient), and three cases (1.5%) with no obvious cause. Failure of maturation was

responsible for 87 access failures (35% of all access failures), of which 85 (34%) were autogenous and two (1%) were synthetic. Overall, 41 failures (16%) were associated with small vein diameter (<2.5 mm) 22 failures (9%) with hypotension alone, 17 failures (7%) with hypotension and central venous stenosis, four failures (1.5%) of unknown cause (bad surgical technique?), two failures (1%) with central venous stenosis alone, and one failure (0.5%) with arterial occlusive disease (in diabetic patient) (Table 3).

Pseudoaneurysmal formation was responsible for 31 access failures (13% of all failures), of which 27 cases (11%) were autogenous and four cases (2%) were synthetic, and 22 (9%) were late and nine (4%) were early. A total of 18 cases (7%) were due to problems in the puncture process of the access (bleeding after needle punctures due to HTN, wrong puncture technique, repeated puncture of the same site of the access, and difficult puncturing due to deeply seated access as in obese patients), six cases (2.5%) due to bad maturation of the access, three cases (1.5%) due to early puncture of the access before maturation, three cases (1.5%) due to disrupted suture line (due to sever HTN or unsecured sutures), and one case (0.5%) due to external trauma (Table 4).

Aneurysmal dilatation was responsible for 18 autogenous, late failures (7% of all failed accesses), of which 13 cases (5%) were associated with HTN and five cases (2%) without HTN. A total of 15 cases (5.5%) were due to superimposed thrombosis and three cases (1.5%) were due leakage of blood, that is, superimposed hematoma formation. Infection was responsible for eight access failures (4% of all access

Table 2. Site distribution of the failed autogenous accesses, causes of early access failure and causes of late access failure.

	Number of fistulae (%)
Site of autogenous AV fistula	
Radiocephalic	125 (54)
Brachiocephalic	84 (36.5)
Brachiobasilic	20 (8)
Radiobasilic	1 (0.5)
Total	230 (100)
Cause of early access failure	
Failure of maturation	87 (84)
Pseudoaneurysm formation	9 (8.5)
Thrombosis	7 (6.5)
Infection	1 (1)
Total	104 (100)
Cause of late failure	
Thrombosis	94 (66)
Pseudoaneurysmal formation	22 (16)
Aneurysmal formation	18 (13)
Infection	7 (5)
Total	141 (100)

AV, arteriovenous.

failures), of which six cases (3%) were synthetic and two cases (1%) were autogenous, and one was early (0.5%) and seven were late (3.5%). A total of 13 accesses with thrombosis and stenosis were subjected to interventions to correct access failure. Overall, two autogenous accesses (one brachiobasilic and one brachiocephalic) underwent percutaneous thrombectomy with balloon dilatation (PTA). The primary patency rates were 100% at 2 months, 50% at 6 months, and 0% at 12 months (one accesses regain patency for 2 months only, and the other regain patency for 6 months). A total of 11 accesses (five autogenous and six synthetic) underwent open surgical thrombectomy using Fogarty balloon catheter. The primary patency rates were 91% at 2 months, 82% at 6 months, and 36% at 12 months (one access failed to regain patency, one access regained patency for 2 months, four accesses regained patency for 6 months, and four accesses regained patency for 12 months) (Table 5).

Overall, four patients received permicath insertion. The primary patency rates were 100% at 6 months, 75% at 12 months, and 25% at 18 months. A total of three patients received four lower limb AV accesses (one synthetic and three autogenous). The patency rates were 50% at 2 months and 0% at 6 months. Overall, three patients received three natural vein transfers from lower limb (saphenous vein

Table 3. Distribution of the causes of access thrombosis, predisposing factors for access stenosis, and causes of failure of maturation.

	Number of accesses (%)
Predisposing factor	
Access stenosis	64 (25.5)
Hypotension	26 (10.5)
External compression	6 (2.5)
Wrong puncture	3 (1.5)
Central venous stenosis	2 (1)
Total	101 (41)
Predisposing factor	
Hypotension	34 (14)
Hypotension and central venous stenosis	12 (4.5)
Central venous stenosis	7 (2.5)
HTN	7 (2.5)
No obvious cause	3 (1.5)
Arterial occlusive disease	1 (0.5)
Total	64 (25.5)
Predisposing factor	
Small vein diameter	41 (16)
Hypotension	22 (9)
Hypotension and central venous stenosis	17 (7)
No obvious cause	4 (1.5)
Central venous stenosis	2 (1)
Arterial occlusive disease	1 (0.5)
Total	87 (35)

HTN, hypertension.

Table 4. Distribution of the causes of pseudoaneurysm.

Cause	Number of accesses (%)
Problems of puncture process	18 (7)
Bad maturation	6 (2.5)
Early puncture	3 (1.5)
Disrupted suture line	3 (1.5%)
External trauma	1 (0.5)
Total	31 (13)

graft). The primary patency rates were 100% at 6 months and 33% at 12 months. One patient subjected to portacath implantation, which was still functioning after 12 months (Table 6).

4. Discussion

The present study showed that 55 patients (55%) were females and 45 patients (45%) were males, with age ranged from 20 to 80 years, with median age of 50 years and mean ± SD age of 50 ± 10 years. Our results suggested that females were more prone to AV failure in the age group 60–70 years.

The present study was supported by Tanaka et al.[13] who compared 382 patients without failure versus 53 patients with AV failure and reported that in terms of sex, there was a statistically significant difference between the analyzed groups; in the failure group, the average age was 70.7 ± 15.6 years, and 49% of the participants were men and 51% of the participants were women.

However, Lin et al.[14] enrolled 3676 patients with AV failure and 14 704 patients without AV failure. The study reported that age and sex were not statistically significantly different between the groups under study; in the failure group, the average age was 61.7 (10.3) years, with 56% men and 44% females.

The present study revealed that 230 access (94%) were autogenous and 15 access (6%) were synthetic (PTFE bridge graft).

A natural (autologous) AVF, a prosthetic artery-vein interposition graft (AVG), or a CVC may all be used to get vascular access. In comparison with CVCs and AVGs, an AVF is seen to be the preferred hemodialysis access for the majority of patients because of its lifespan and reduced risks of thrombosis, infection, interventions to maintain patency, and overall mortality.[15,16]

Native AVFs, however, take longer to form, and 20–50% of them do not sufficiently mature to sustain hemodialysis.[17] AVGs have a lower primary failure rate than a functional AVF, but they are more prone to thrombosis and need more treatments to maintain their patency.[7] A salvage treatment is required for more than 75% of all AVGs within the

Table 5. Results of surgical and percutaneous techniques for correction of access stenosis and thrombosis.

Modality of intervention	Primary patency rate at 2 months (%)	Primary patency rate at 6 months (%)	Primary patency rate at 12 months (%)
Surgical technique	91	82	36
Percutaneous technique	100	50	0

first year after formation, which results in enormous medical expenses.[18]

The present study revealed that all the 15 failed synthetic accesses were brachial artery to ipsilateral axillary vein straight bridge grafts. Of the 230 failed autogenous fistulae, 124 (54%) were radiocephalic around wrist, 83 (36.5%) were brachiocephalic, 20 (8%) were brachiobasilic around elbow, two (1%) were natural vein transfer (saphenous vein graft), and one (0.5%) was radiobasilic in mid forearm.

According to the KDOQI,[19] the preferred placing of fistulae in patients with persistent renal disorder should be a transposed brachial-basilic vein fistula, a forearm (radiocephalic) primary fistula, an elbow (brachiocephalic) primary fistula, and then an AVG made of synthetic or biological material. This is consistent with our findings.

The present study showed that 104 (43%) of access failures occurred within the first 3 months after creation (early failure) and 141 (57%) occurred after 3 months of creation (late failure). Of the 104 early failures, 94 (90%) were autogenous and 10 (10%) were synthetic. Overall, 84% (87) of early failures were due to failure of maturation, 8.5% (nine) due to pseudoaneurysmal formation, 6.5% (seven) due to early thrombosis, and 1% (one case) was due to early infection. Of the 141 late failures, 96.5% (136) were autogenous and 3.5% (five) were synthetic. Overall, 66% (94) of late failures were due to thrombosis, 16% (22) due to pseudoaneurysmal formation, 13% (18) due to aneurysmal formation, and 5% (seven) due to infection.

Our results were supported by Chang et al.,[20] who revealed that progressive neointimal hyperplasia, which causes stenosis and subsequently thrombosis, is the primary factor in AVF failure.

Stracke et al.[21] also reported that outflow venous stenosis, which is brought on by vascular intimal hyperplasia and thrombosis and is in turn brought on by platelet activation, endothelial cell damage, and vascular smooth muscle cell proliferation, is the primary cause of VA failure. It has been proposed that antiplatelet treatment may stop VA failure.[22]

The current study showed that access thrombosis was responsible for 101 failures (41% of all access failures), of which 99 cases (40%) were autogenous and two cases (1%) were synthetic, and seven failures (3%) were early and 94 failures (38%) were late. A total of 64 cases (25.5%) of access thrombosis were associated with access stenosis, 26 cases (10.5%) with hypotension alone, six failures (2.5%) with external compression, three failures (1.5%) with wrong puncture, and two failures (1%) with central venous stenosis alone.

This comes in line with Masud et al.,[23] who revealed that vascular entry in the majority of patients having an AV access, stenosis, a frequent consequence, manifests, and causes access dysfunction. This issue reduces blood flow by limiting luminal diameter and increases the risk of thrombosis for access. Similar to how catheter dysfunction is brought on by catheter-related fibroepithelial sheath, it similarly has a negative impact on blood flow. In other words, they came to the conclusion that stenotic lesions progress to totally obliterate the lumen, obstruct blood flow, and cause thrombosis of vascular access if left untreated. AV access thrombosis is a significant factor in the eventual loss of vascular access, in fact.

The current study showed that 34 cases of access stenosis (14%) were associated with hypotension alone, 12 cases (4.5%) with hypotension and central venous stenosis, seven (2.5%) with central venous stenosis, seven cases (2.5%) with HTN, one case (0.5%) with arterial occlusive disease (in diabetic patient), and three cases (1.5%) with no obvious cause.

Our findings are corroborated by Tanaka et al.,[13] who revealed that compared with the early VA

Table 6. Results of alternative methods for hemodialysis.

Alternative method of hemodialysis	Primary patency rate at 2 months (%)	Primary patency rate at 6 months (%)	Primary patency rate at 12 months (%)
Permicath	100	100	75
Lower limb AV access	50	0	0
Natural vein transfer	100	100	33

AV, arteriovenous.

failure group without early VA failure, the early VA failure group's diastolic blood pressure was considerably lower.

Moreover, Manne et al.[24] reported that because of intradialytic hypotension, AVFs (6.52%) failed. One of the key factors in vascular access failure is hypotension.

In this study, we also found that failure of maturation was responsible for 87 access failures (35% of all access failures), of which 85 (34%) were autogenous and two (1%) were synthetic. A total of 41 failures (16%) were associated with small vein diameter (<2.5 mm), 22 failures (9%) with hypotension alone, 17 failures (7%) with hypotension and central venous stenosis, four failures (1.5%) of unknown cause (bad surgical technique?), two failures (1%) with central venous stenosis alone, and one failure (0.5%) with arterial occlusive disease (in diabetic patient).

According to earlier studies, the patency and maturation of VA are related to the artery's or vein's diameter,[25–27] which support our findings.

Therefore, it was crucial that the artery that was used to create the fistula be capable of expanding enough to provide for the increased blood flow needed to feed the fistula and distant tissues.[28]

Furthermore, Siddiqui et al.[29] reported that early fistula failure is often brought on by thrombosis, which may be brought on by hematoma, low flow rates brought on by low blood pressure, or a hypercoagulable condition. Reduced arterial remodeling and ultimate venous lumen diameter are related to endothelial function impairment.

Our results showed also that pseudoaneurysmal formation was responsible for 31 access failures (13% of all failures), of which 27 cases (11%) were autogenous and four cases (2%) were synthetic, and 22 (9%) were late and nine (4%) were early. A total of 18 cases (7%) were due to problems in the puncture process of the access (bleeding after needles punctures due to HTN, wrong puncture technique, repeated puncture of the same site of the access, and difficult puncturing due to deeply seated access as in obese patients), six cases (2.5%) due to bad maturation of the access, three cases (1.5%) due to early puncture of the access before maturation, three cases (1.5%) due to disrupted suture line (due to sever HTN or unsecured sutures), and one case (0.5%) due to external trauma.

The current study showed also that aneurysmal dilatation was responsible for 18 autogenous, late failures (7% of all failed accesses), of which 13 cases (5%) were associated with HTN and five cases (2%) without HTN. A total of 15 cases (5.5%) were due to superimposed thrombosis and three cases (1.5%) were due leakage of blood, that is, superimposed hematoma formation.

The etiology of aneurysms is likewise not well understood. Pseudoaneurysms may develop from the anastomosis as a consequence of poor surgical technique during the perioperative period or subsequently as an infection-related complication. When an AVF or AVG is punctured during routine dialysis needling or during an intervention, this may lead to prolonged bleeding and the development of pseudoaneurysms.[3] AVGs in particular are at danger from too localized needling.

Cuadros et al.[30] discovered in their series an elevated relative risk (2.5) of main functional patency loss. HTN makes blood vessels more rigid and encourages the development of arteriosclerosis, both of which reduce blood flow through anastomoses. Moreover, Lin et al.[14] reported that HTN was significantly associated with AV access failure.

The current study showed that infection was responsible for eight access failures (4% of all access failures), of which six cases (3%) were synthetic and two cases (1%) were autogenous, and one was early (0.5%) and seven were late (3.5%).

A total of 13 accesses with thrombosis and stenosis were subjected to interventions to correct access failure. Overall, two autogenous accesses (one brachiobasilic and one brachiocephalic) were undergoing percutaneous thrombectomy with balloon dilatation (PTA). The primary patency rates were 100% at 2 months, 50% at 6 months, and 0% at 12 months (one access regained patency for 2 months only, and the other regained patency for 6 months).

A total of 11 accesses (five autogenous and six synthetic) underwent open surgical thrombectomy using Fogarty balloon catheter. The primary patency rates were 91% at 2 months, 82% at 6 months, and 36% at 12 months (one access failed to regain patency, one access regained patency for 2 months, four accesses regained patency for 6 months, and four accesses regained patency for 12 months).

A total of four patients received permicath insertion; the primary patency rates were 100% at 6 months, 75% at 12 months, and 25% at 18 months. Moreover, three patients received four lower limb AV accesses (one synthetic and three autogenous); the patency rates were 50% at 2 months and 0% at 6 months. In addition, three patients received three natural vein transfers from lower limb (saphenous vein graft). The primary patency rates were 100% at 6 months and 33% at 12 months. One patient was subjected to portacath implantation, which was still functioning after 12 months.

To declot a thrombosed access, a number of thrombectomy techniques have been used.

Procedures for mechanical and pharmacomechanical thrombolysis may be carried out effectively.[4,31,32] It is important to note that primary patency rates after declotting of a clogged vascular access are much lower than those following angioplasty of access stenosis.[4,31]

Nassar et al.[32] reported that in 91.9% of the 520 cases examined, a clinically effective percutaneous thrombectomy was accomplished. At 3, 6, and 12 months, the main patency was 60, 40, and 17.7%, respectively.

Uflacker et al.[33] reported a 37-patient prospective randomized experiment comparing mechanical thrombectomy with the Amplatz device versus surgical thrombectomy. Primary patency at 30 days in this trial was 47% in the percutaneous group against 77% in the surgical group. At 30 days, secondary patency in the percutaneous group was 68%.

Chan et al.[34] performed a comprehensive review and meta-analysis that comprised two retrospective cohort studies with 806 (63%) and 466 (37%) patients in the surgical and entirely endovascular therapy arms, respectively, as well as eight randomized, controlled trials. The 30-, 60-, and 90-day main nonpatency rates did not significantly vary between endovascular and surgical treatment. At 30, 60, and 90 days, there were no significant differences between endovascular and surgical treatments in terms of secondary nonpatency rates. In comparison with surgical thrombectomy, endovascular operations had a substantially greater risk of technical failure (RR: 1.58; 95% CI: 1.06−2.37; $P = 0.03$). Regarding mild and serious issues, there was little variation.

Furthermore, Aydõn et al.[35] reported that among the 42 patients in the pharmacomechanical thrombectomy group, 41 (98%) required further treatments, and four (10%) had primary failure. In the surgical group, 15 (28%) patients had primary failure. Primary patency rates in the pharmacomechanical therapy group were considerably greater than those in the surgical group at 6 and 12 months (85 vs. 67% and 78 vs. 55%, respectively; $P < 0.05$).

5. Conclusion

Creation and preservation of a healthy patent autogenous AV access in upper limb as long as possible represents the big challenge for health care providers of patients with ESRD owing to the steady increase in the number of those patients and the presence of many factors that cause AV access failure.

The care of the AV access starts preoperatively by proper selection of the type and site of the access, passing through meticulous intraoperative technique, and finally proper postoperative care.

Four pillars stand behind AV access failure, namely, bad surgical technique, failure of maturation, access stenosis and thrombosis, and postoperative complications. Access thrombosis together with failure of maturation plays the major role for repeated AV access failure. The most frequent reason for access thrombosis is venous stenosis and that for failure of maturation is the small vein diameter (<2.5 mm) followed by hypotension.

Hypotension and central venous stenosis jeopardize the outcome of AV access by being the most important predisposing factors for the occurrence of failure of maturation and access stenosis.

Pseudoaneurysmal formation comes next to failure of maturation and access thrombosis as a cause of repeated AV access failure followed by true aneurysmal dilatation and finally infection.

The main problem in patients with repeated AV access failure is that they may develop upper extremity access precluded, or more seriously complete failed access.

Management of repeated AV access failure needs changing the attitude of vascular surgeons to be more conservative and based on access salvage protocols (access preservation), directed to correction of early access failure and the predisposing factors behind it as much as possible before deciding access abandonment.

Putting proper surveillance programs for early detection of access dysfunction may be helpful for rapidly correcting the causative factor (early infection or stenosis) aiming at preservation of the access.

Finally, there are four main concerns should be considered in every patient with repeated AV access failure before creation a new access:

Correction of hypotension and central venous stenosis.

Conservative management of access stenosis and thrombosis; surgical and percutaneous techniques.

Trying conservative measures for correction of postoperative complications (such as infection, aneurysmal dilatation, pseudoaneurysmal formation) during early stages aiming at access preservation.

Whatever the circumstances, the priority is for autogenous AV access creation and shortening the time of usage of hemodialysis catheters as much as possible because they are the most important cause of central venous stenosis that predisposes to access failure.

Conflict of interest

Authors declare that there is no conflict of interest, no financial issues to be declared.

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