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Repeated failure of arterio-venous haemodialysis access (Causes and possible solutions)

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***Abstract*---**Background: The subsequent development of vascular access techniques and devices now permits patients to be maintained on dialysis for decades. Aim of study: To evaluate the most possible causes behind repeated arteriovenous (AV) access failure in ESRD patients 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. Patients and methods: This retrospective non-randomized study was conducted in department of vascular surgery, Al-Azhar University Hospitals – Cairo (Al-Hussein and Sayed Glal Hospitals). This study was conducted on 100 ESRD patients on regular hemodialysis and with repeated AV access failure; 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. Results: Among the cases of access thrombosis 25.5% were associated with access stenosis, 10.5% with hypotension, and 2.5% with external compression. Among the cases of failure of maturation 16% were due to small vein diameter (less than 2.5 mm), 9% associated with hypotension, and 7% with hypotension together with central venous stenosis. Conclusion: 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

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predisposing factors behind it as much as possible before deciding access abandonment.

***Keywords*---**Arteriovenous, Heamodialysis Access, End-stage kidney disease.

Introduction

The prevalence of end-stage kidney disease (ESKD) is increasing in the presence of a growing diabetic and aging population **[1]**. Hemodialysis remains the most common form of kidney replacement therapy [**2**], with over 2 million people on hemodialysis worldwide **[3]**. To maintain successful hemodialysis, functional vascular access is required **[4]**. Hemodialysis vascular access consists of three forms: the arteriovenous fistula (AVF), the arteriovenous graft (AVG), and the central venous catheter (CVC). The AVF is a connection between a native artery and vein that is created via an end-or-side vein-to-artery anastomosis. AVGs are created by interposing a prosthetic graft (classically with polytetrafluorethylene [PTFE]) between an artery and a vein **[5]**. The key requirements of such access are sufficient blood flow rate, low flow resistance, a low rate of complications and, for AVF and AVG, ease of cannulation.

A mature native AVF is considered superior to a synthetic AVG or CVC due to better long-term outcomes, including reduced rates of thrombosis, infection and interventions to maintain patency **[6]**. Balanced against these benefits, as a result of early thrombosis, neointimal hyperplasia formation and inadequate vasodilation (outward remodeling), between 20 and 60% of AVFs fail to mature to an adequate caliber to allow repeat cannulation and provide sufficient blood flow for hemodialysis and thereby prevent timely usability of the AVF for hemodialysis **[6]**. AVGs can be used within days of access creation but long-term, they are at higher risk of developing venous stenosis, thrombosis and infection compared to a functioning AVF **[7]**. More than 50% of AVGs thrombose within 12 months of creation and they require significantly more interventions to maintain patency compared to a functioning AVF **[7, 8]**. CVCs can be used immediately after insertion, but their long-term use is discouraged in light of the significantly higher risks of thrombosis, catheter-associated bacteremia and inadequate solute clearance **[9]**.

Vascular access dysfunction is a major cause of morbidity, mortality and excess healthcare costs **[6]**. Indeed, healthcare professionals, patients and caregivers consider vascular access function a top priority of research in hemodialysis and clinical practice **[10]**. There have been recent advances in the understanding of the biology of vascular access and its dysfunction, with neointimal hyperplasia leading to venous stenosis and inadequate outward remodeling being identified as the two major causes of dialysis vascular access dysfunction **[11]**. This knowledge has led to the identification of potential therapeutic targets and the development of novel interventions to improve and maintain vascular patency **[9]**.

A technological breakthrough that would significantly reduce the morbidity and cost associated with vascular access does not appear to be in the horizon, and

with this realization in mind, recent efforts have focused on developing algorithms to better define patient selection criteria for each access method **[12].** The main aim of this study was to evaluate the most possible causes behind repeated AV access failure in ESRD patients 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.

Patients and Methods

A retrospective non randomized study was carried out on 100 ESRD patients both males and females of different ages on regular hemodialysis in Al-Hussien, Sayed Glal Hospitals and with repeated AV access failure; two or more failed accesses either autogenous or synthetic in upper limb to collect data aiming at: Detection of the most possible causes of repeated AV access failure of upper limb, both autogenous and synthetic, in ESRD patients and their classification into predisposing and precipitating factors. Classification of failure into early; within 1st three months and late; more than three months. Also finding the relationship between timing of access failure and its cause. Evaluation the results of specific interventions applied on the patients to correct access failure such as surgical and percutaneous thrombectomy and balloon angioplasty when present. Trying to find the best algorism or guidelines for creation and saving the AV access in ESRD patients, and the last resorts for those with upper extremity access precluded and completely failed access.

Totally 245 failed access, were collected and according to the timing of failure, the failed accesses were categorized into 2 main groups: Accesses with early failure: 104 access (43%) and Accesses with late failure: 141 access (57%).
Inclusion criteria: ESRD patients on regular hemodialysis with two or more failed AV access in upper limb; either autogenous or synthetic.

Data collection and analysis

The failed access is usually identified by loss of thrill and machinarry murmur over the access and inability to use it for sufficient and regular hemodialysis. Early access failure occurs within the 1st three 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 1st three month) 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 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 as regard 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 DM, hypertension, Obesity; BMI more than 30kg/m2, systemic collagen disease such as systemic lupus erythromatosis, hypercoagulable diseases, and the original

cause of renal failure specially when related to vascular diseases. Also a detailed history about the Distribution of the previously failed AV accesses both autogenous and synthetic, the timing of their failure, occurrence of postoperative AV access complications as infection, aneurysmal dilatation, hematoma formation, was taken. 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 postoperative anticoagulant treatment and History of specific interventions applied to correct access failure as surgical and percutaneous thrombectomy and their results in regaining the patency and functionality of the access and for how long; PRIMARY PATENCY RATES. The patients were evaluated clinically as regard arterial pulsations, blood pressure, the condition and accessabillity 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 central venous catheters in lower neck, signs of upper limb ischemia 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: prothrombin time and concentration, INR, partial thromboplastin time, and platelets count were evaluated to exclude hypercoagulability. Also 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 is analyzed separately.

Statistical analysis

The collected data were coded, processed and analyzed using the SPSS (Statistical Package for Social Sciences) version 22 for Windows® (IBM SPSS Inc, Chicago, IL, USA). Data were tested for normal distribution using the Shapiro Walk test. Qualitative data were represented as frequencies and relative percentages. Chi square test (x2) to calculate difference between two or more groups of qualitative variables. Quantitative data were expressed as mean ± SD (Standard deviation).

Independent samples t-test was used to compare between two independent groups of normally distributed variables (parametric data). P value < 0.05 was considered significant.

Results

A retrospective non randomized study was carried over 100 ESRD patients on regular hemodialysis (in Al-Hussien, Sayed Glal Hospitals) and with repeated AV access failure; 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. 55 patients (55%) were females& 45 patients (45%) were males**,** with age ranging from 20 to 80 years with median equals 50 years, mean equals 50 years, and SD equals 10. The causes of CRF among studied patients (Table 1).

Table (1): Age distribution and cause of CRF among studied patients

	Number of cases	Percentage
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%

230 access (94%) were autogenous and 15 access (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 radio-cephalic around wrist, 83 (36.5%) were brachio-cephalic, 20 (8%) were brachio-basilic around elbow, 2 (1%) were natural vein transfer; saphenous vein graft, and one (0.5%) was radio-basilic in mid forearm. 104 (43%) of access failures occurred within the 1st three months after creation; **early failure** and 141 (57%) occurred after three months of creation**; late failure**. Of the 104 early failures, 94 (90%) were autogenous and 10 (10%) were synthetic. 84% (87) of early failures were due to failure of maturation, 8.5% (9) due to pseudoaneurysmal formation, 6.5% (7) 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% (5) were synthetic. 66% (94) of late failures were due to thrombosis, 16% (22) due to pseudoaneurysmal formation, 13% (18) due to aneurysmal formation, and 5% (7) due to infection (Figures 1, 2 and Table 2).

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

	Number of fistulae	percentage
Site of autogenous AV fistula Radio-cephalic Brachio-cephalic Brachio-basilic Radio-basilic	125 84 20 1	54% 36.5% 8% 0.5%
Total	230	100
Cause of early access failure Failure of maturation Pseudoaneurysm formation Thrombosis infection	 87 9 7 1	 84% 8.5% 6.5% 1%
Total	104	100%
Cause of late failure Thrombosis Pseudoaneurysmal formation Aneurysmal formation Infection	 94 22 18 7	 66% 16% 13% 5%
Total	141	100%

Access thrombosis was responsible for 101 failures (41% of all access failures) of which 99 cases (40%) were autogenous and 2 cases (1%) were synthetic, and 7 failures (3%) were early and 94 failures (38%) were late. 64 case (25.5%) of access thrombosis were associated with **access stenosis**, 26 case (10.5%) with **hypotension alone**, 6 failures (2.5%) with external compression, 3 failures (1.5%) with wrong puncture, and 2 failures (1%) with central venous stenosis alone. 34 case of **access stenosis** (14%) were associated with hypotension alone, 12 case (4.5%) with hypotension and central venous stenosis, 7 (2.5%) with central venous stenosis, 7 case (2.5%) with HTN, one case (0.5%) with arterial occlusive disease (in diabetic patient), and 3 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 2 (1%) were synthetic. 41 failures (16%) were associated with **small vein diameter** (less than 2.5 mm) 22 failures (9%) with **hypotension alone,** 17 failures (7%) with hypotension and central venous stenosis, 4 failures (1.5%) of unknown cause; (bad surgical technique?), 2 failures (1%) with central venous stenosis alone, and one failure (0.5%) with arterial occlusive disease (in diabetic patient) (Table 3).

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

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

Pseudoaneurysmal formation was responsible for 31 access failures (13% of all failures) of which 27 case (11%) were autogenous and 4 cases (2%) were synthetic, and 22 (9%) were late and 9 (4%) were early. 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, difficult puncturing due to deeply seated access as in obese patients), 6 cases (2.5%) due to bad maturation of the access, 3 cases (1.5%) due to early puncture of the access before maturation, 3 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).

Table (4): Distribution of the causes of pseudoaneurysm

Cause	Number of accesses	Percentage
Problems of puncture process Bad maturation Early puncture	18 6 3	7% 2.5% 1.5%

Disrupted suture line External trauma	3 1	1.5% 0.5%
Total	31	13%

Aneurysmal dilatation was responsible for 18 autogenous, late failures (7% of all failed accesses) of which 13 cases (5%) were associated with HTN and 5 cases (2%) without HTN. 15 cases (5.5%) were due to superimposed thrombosis and 3 cases (1.5%) were due leakage of blood; superimposed hematoma formation. **Infection** was responsible for 8 access failures (4% of all access failures) of which 6 cases (3%) were synthetic and two cases (1%) were autogenous, and one was early (0.5%) and 7 were late (3.5%). 13 accesses with thrombosis and stenosis were subjected to interventions to correct access failure. 2 autogenous accesses (one brachiobasilic and one brachiocephalic) were underwent percutaneous thrombectomy with balloon dilatation; PTA. The primary patency rates were 100% at 2months, 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). 11 accesses (5 autogenous and 6 synthetic) were 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 regain patency for 2 months, 4 accesses regain patency for 6 months, and 4 accesses regain patency for 12 months) (Table 5).

Table (5): Results of surgical and pertcutaneous 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%

4 patients received permicath insertion, the primary patency rates were 100% at 6 months, 75% at 12 months, and 25% at 18 months. 3 patients received 4 lower limb AV accesses, (one synthetic and 3 autogenous) the patency rates were 50% at 2 months and 0% at 6 months. 3 patients received 3 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 subjected to portacath implantation which still functioning since 12 months (Table 6).

Table (6): Results of alternative methods for hemodialysis

Alternative method of hemodialysis	1ry patency rate at 2 months	1ry patency rate at 6 months	1ry patency rate at 12 months
Permicath	100%	100%	75%
Lower limb AV access	50%	0%	0%
Natural vein transfer	100%	100%	33%

Discussion

The present study showed that 55 patients (55%) were females& 45 patients (45%) were males, with age ranging from 20 to 80 years with median equals 50 years, mean equals 50 years, and SD equals 10. 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 there was statistically significant difference between the studied groups as regard sex, in the failure group the mean age was 70.7 ± 15.6 years and 49% were males and 51% females. However, **Lin et al. [14]** enrolled 3676 patients with AV failure and 14,704 patients without AV failure. The study reported that there was no statistically significant difference between the studied groups as regard age and sex, in the failure group the mean age was 61.7 (10.3) years and 56% were males and 44% females.

The present study revealed that 230 access (94%) were autogenous and 15 access (6%) were synthetic; PTFE bridge graft. Vascular access can be obtained by use of a native (autologous) arteriovenous fistula (AVF), a prosthetic interposition graft between artery and vein (AVG) or a central venous catheter. For most patients, an AVF is considered the hemodialysis access of choice due to its longevity and lower rates of thrombosis, infection, interventions to maintain patency and overall mortality when compared with central venous catheters and AVGs **[15, 16].**

However, native AVFs take longer to establish and approximately 20%-50% fail to develop adequately to support dialysis **[17].** Compared with a functioning AVF, AVGs have a lower primary failure rate but have a higher risk of thrombosis and require more interventions to maintain patency **[7].** More than half of all AVGs will thrombose within the first year after creation and >75% will require a salvage procedure resulting in significant health costs **[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 radio-cephalic around wrist, 83 (36.5%) were brachio-cephalic, 20 (8%) were brachio-basilic around elbow, 2 (1%) were natural vein transfer; saphenous vein graft, and one (0.5%) was radio-basilic in mid forearm. The **KDOQI [19]** recommended order of preference for placement of fistulae in patients with chronic kidney disease should be a forearm (radiocephalic) primary fistula, an elbow (brachiocephalic) primary fistula, a transposed brachial-basilic vein fistula, followed by arteriovenous graft (AVG) of synthetic or biological material. This comes in line with our results.

The present study showed that 104 (43%) of access failures occurred within the 1st three months after creation; early failure and 141 (57%) occurred after three months of creation; late failure. Of the 104 early failures, 94 (90%) were autogenous and 10 (10%) were synthetic. 84% (87) of early failures were due to failure of maturation, 8.5% (9) due to pseudoaneurysmal formation, 6.5% (7) 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% (5) were synthetic. 66% (94) of late failures were due to thrombosis, 16% (22) due to pseudoaneurysmal

formation, 13% (18) due to aneurysmal formation, and 5% (7) due to infection. Our results can be supported by **Chang et al. [20]** who reported that the major cause of AVF failure is progressive neointimal hyperplasia, which results in stenosis and subsequent thrombosis. **Stracke et al. [21]** also reported that the main cause of VA failure is outflow venous stenosis caused by vascular intimal hyperplasia and thrombosis, in turn triggered by platelet activation, endothelial cell injury, and vascular smooth muscle cell proliferation. Antiplatelet therapy has been suggested to prevent 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 2 cases (1%) were synthetic, and 7 failures (3%) were early and 94 failures (38%) were late. 64 case (25.5%) of access thrombosis were associated with access stenosis, 26 case (10.5%) with hypotension alone, 6 failures (2.5%) with external compression, 3 failures (1.5%) with wrong puncture, and 2 failures (1%) with central venous stenosis alone.

This come in agreement with **Masud et al. [23]** who stated that vascular access stenosis is a common complication that develops in a great majority of patients with an arteriovenous access and leads to access dysfunction. By restricting luminal diameter, this complication leads to a reduction in blood flow and places the access at risk for thrombosis. Similarly, the development of catheter-related fibroepithelial sheath also causes catheter dysfunction with its detrimental effects on blood flow. In other words, they concluded that, left untreated, the stenotic lesions go on to completely obliterate the lumen and occlude the blood flow and result in thrombosis of vascular access. Indeed, thrombosis of an arteriovenous access is a major cause of ultimate loss of vascular access. The current study showed that 34 case of access stenosis (14%) were associated with hypotension alone, 12 case (4.5%) with hypotension and central venous stenosis, 7 (2.5%) with central venous stenosis, 7 case (2.5%) with HTN, one case (0.5%) with arterial occlusive disease (in diabetic patient), and 3 cases (1.5%) with no obvious cause. Our results can be supported by **Tanaka et al. [13]** who reported that diastolic blood pressure (DBP) was significantly lower in the early VA failure group than in the without early VA failure group.

Also, **Manne et al. [24]** reported that AVFs (6.52%) failed because of intradialytic hypotension. Hypotension is one of the important causes of failure of vascular access. 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 2 (1%) were synthetic. 41 failures (16%) were associated with small vein diameter (less than 2.5 mm) 22 failures (9%) with hypotension alone, 17 failures (7%) with hypotension and central venous stenosis, 4 failures (1.5%) of unknown cause; (bad surgical technique?), 2 failures (1%) with central venous stenosis alone, and one failure (0.5%) with arterial occlusive disease (in diabetic patient).

In the previous reports, the diameter of the artery or vein is associated with the patency and maturation of VA **[25, 26, 27],** which support our findings. So, it was essential that the artery used in the creation of the fistula is able to adequately increase diameter allowing for the increased blood flow required to supply the fistula and distal tissues **[28]**.

Furthermore, **Siddiqui et al. [29]** reported that early fistula failure is usually due to thrombosis which can be triggered by hematoma, by low flow rates resulting from low blood pressure, or by a hypercoagulable state. Impairment of endothelial function is associated with decreased arterial remodeling and final venous lumen diameter.

Our results showed also that Pseudoaneurysmal formation was responsible for 31 access failures (13% of all failures) of which 27 case (11%) were autogenous and 4 cases (2%) were synthetic, and 22 (9%) were late and 9 (4%) were early. 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, difficult puncturing due to deeply seated access as in obese patients), 6 cases (2.5%) due to bad maturation of the access, 3 cases (1.5%) due to early puncture of the access before maturation, 3 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 5 cases (2%) without HTN. 15 cases (5.5%) were due to superimposed thrombosis and 3 cases (1.5%) were due leakage of blood; superimposed hematoma formation.

The aetiology of aneurysms is also ill defined. Pseudoaneurysms may be from the anastomosis and reflect leaking of blood outside the lumen perioperatively as a result of surgical technique or occur later as a complication of infection. Puncture of an AVF or arteriovenous graft (AVG) either as part of standard dialysis needling or from intervention can result in prolonged bleeding and pseudoaneurysm formation **[3]**. AVGs in particular are at risk if needling is too localized. **Cuadros et al. [30]** found an increased relative risk (2.5) of primary functional patency loss in their series. Hypertension increases vascular stiffness and promotes arteriosclerosis, which will decrease the blood flow across the anastomosis. Also, **Lin et al. [14]** reported that Hypertension significantly associated with AV access failure.

The current study showed that Infection was responsible for 8 access failures (4% of all access failures) of which 6 cases (3%) were synthetic and two cases (1%) were autogenous, and one was early (0.5%) and 7 were late (3.5%). 13 accesses with thrombosis and stenosis were subjected to interventions to correct access failure. 2 autogenous accesses (one brachiobasilic and one brachiocephalic) were undergoing percutaneous thrombectomy with balloon dilatation; PTA. The primary patency rates were 100% at 2months, 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).

11 accesses (5 autogenous and 6 synthetic) were undergoing 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 regain patency for 2 months, 4 accesses regain patency for 6 months, and 4 accesses regain patency for 12 months).

4 patients received permicath insertion, the primary patency rates were 100% at 6 months, 75% at 12 months, and 25% at 18 months. 3 patients received 4 lower limb AV accesses, (one synthetic and 3 autogenous) the patency rates were 50% at 2 months and 0% at 6 months. 3 patients received 3 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 subjected to portacath implantation which still functioning since 12 months.

A variety of thrombectomy procedures have been performed to achieve declotting of a thrombosed access. Mechanical as well as pharmacomechanical thrombolysis procedures can be successfully performed **[4, 31, 32]**. It is worth mentioning that the primary patency rates after declotting of a clotted vascular access are markedly reduced when compared with the primary patency following angioplasty of access stenosis **[4, 31]**.

Nassar et al. [32] reported that a clinically successful Percutaneous Thrombectomy was achieved in 91.9% of the total 520 cases studied. With a primary patency of 60%, 40%, and 17.7% at 3, 6, and 12 months, respectively. **Uflacker et al. [33]** reported a prospective randomized trial of surgical thrombectomy compared to mechanical thrombectomy with the Amplatz device in 37 patients. In this study, the primary patency at 30 days was 47% in the percutaneous group compared with 77% in the surgical group. Secondary patency in the percutaneous group was 68% at 30 days.

The systematic review and meta-analysis by **Chan et al. [34]** included 8 randomized, controlled trials and two retrospective cohort studies were included, comprising 806 (63%) and 466 (37%) participants in the surgical and wholly endovascular treatment arms respectively. There were no significant differences between endovascular and surgical therapy in the 30-, 60-, and 90-day primary nonpatency rates. In terms of secondary nonpatency rates, there were no significant differences between endovascular and surgical procedures at 30, 60, and 90 days. Endovascular procedures reported a significantly higher technical failure rate as compared with surgical thrombectomy (RR, 1.58; 95% CI, 1.06-2.37; P = .03). There was no significant difference in terms of minor and major complications.

Furthermore, **Aydın et al. [35]** reported that of 42 patients in the pharmacomechanical thrombectomy group, 41 (98%) had additional interventions and primary failure occurred in four patients (10%). Primary failure was seen in 15 (28%) patients in the surgical group. The primary patency rates at 6 and 12 months were significantly higher in the pharmacomechanical treatment group than the surgical group (85% vs. 67% and 78% vs. 55%, respectively; $p<0.05$).

Conclusion

- Creation and preservation of a healthy patent autogenous AV access in upper limb as long as possible represents the big challenge for ESRD patients health care providers due 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.
- 4 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 play the major role for repeated AV access failure. The most common cause for access thrombosis is venous stenosis and that for failure of maturation is the small vein diameter; less than 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 4 main concerns should be considered in every patient with repeated AV access failure before creation a new access:
    1. Correction of hypotension and central venous stenosis.
    2. Conservative management of access stenosis and thrombosis; surgical and percutaneous techniques.
    3. Trying conservative measures for correction of postoperative complications (such as infection, aneurysmal dilatation, pseudoaneurysmal formation) during early stages aiming at access preservation.
    4. 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 which predisposes to access failure.

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