Factors contributing to patency after aneurysmorrhaphy and outflow repair in arteriovenous fistula aneurysm treatment

Sunil Siri Pathamanathan, MBBS[1], Nurul Nadiah Nazurah Binti Mohd Ali, MD[1], Muhammad Zaid Haritha Mohamed Fauzi, MBBS[2], Ismazizi Zaharudin, MD[3]

[1]Department of General Surgery, Hospital Kuala Lumpur, Malaysia, [2]Department of General Surgery, Pulau Pinang Hospital, Malaysia, [3]National Heart Institute, Malaysia

ABSTRACT

Introduction: Vascular access-related aneurysms (VARA) are a complication of arteriovenous fistulas. Repair techniques have been described in the literature with varied outcomes.

Materials and Methods: We conducted a prospective cohort study on patients who had VARA repair over 41 months. The indication for repair was an aneurysmal arteriovenous fistula (AVF) at risk of haemorrhage or difficulty in cannulation. Pseudoaneurysms, infected AVF and bleeding VARA were excluded. All patients underwent outflow stenosis treatment when present, followed by aneurysmorrhaphy. They were monitored periodically over 12 months, measuring functional primary and cumulative patency and access flow. We studied the patient demography, access flow and presence of outflow stenosis. Access flow was measured from the brachial artery (Qa) as a surrogate using ultrasonography. A Kaplan-Meier survival analysis was used to predict the primary and cumulative patency at 12 months and factors contributing to 12-month patency were analysed.

Results: A total of 64 patients were recruited for this study, of whom 58 completed the study. Most of the participants were male (67%) with a median age of 45 years. Forty-six patients (79.3%) had brachiocephalic fistula (BCF) aneurysms. Thirty-nine (67.2%) had preexisting outflow stenoses that required intervention. All patients underwent an aneurysmorrhaphy, of whom 12% had a cephalic arch vein transposition due to severe stenosis. Primary patency at 12 months was 86%, whereas the cumulative patency rate was 95%. Patency was significantly associated with younger age and showed a positive trend with higher pre-intervention Qa. Symptomatic recurrent stenosis developed in 17.2% of the cohort.

Conclusion: Improving the patency of VARA entails the treatment of outflow stenosis and aneurysmorrhaphy. Surveillance is important to detect and treat recurrent outflow stenoses. The outcome is better among younger patients with pre-interventional access flow as measured in the brachial artery as a surrogate.

KEYWORDS:
Arteriovenous fistula, aneurysmal arteriovenous fistula, vascular access, aneurysm, vascular access-related aneurysm, aneurysmorrhaphy, outflow stenosis

INTRODUCTION

The incidence of end-stage renal failure in Malaysia is increasing at an alarming rate. Aneurysms at the access sites of arteriovenous fistulas (AVF) used for dialysis are among the morbidities of this disease. Vascular access-related aneurysms (VARA) are not uncommon, with reported incidences ranging from 5-60%.[1] This wide range may be attributed to the varying definitions of VARA used in the literature. VARA is defined as a localised dilatation of the access vessel involving all wall layers, and a common threshold is 18mm.[2] Aneurysms develop due to a combination of high wall shear stress (WSS) induced by high flow, outflow stenosis and wall weakening due to multiple cannulations.[1,3]

Most VARA are asymptomatic and do not require any intervention. However, up to 31% of VARA require surgery due to haemorrhage or dysfunction.[4] Both European Society of Vascular Surgery (ESVS) and National Kidney Foundation's Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines recommend surgical revision in symptomatic VARA.[5,6] Balaz et al., conducted a meta-analysis on VARA repair, either with aneurysmorrhaphy alone or combined with staplers or the use of sizing mandrels.[7] They found that the 12-month primary patency rate was 45-95%, with a pooled rate of 82%.[7] We are reporting the results of VARA repair at our centre, and studied the factors contributing to patency in our series.

MATERIALS AND METHODS

This was a prospective study of adult patients who underwent VARA repair over 41 months, from 1st July 2017 to 31st November 2020, at Kuala Lumpur Hospital (HKL), Malaysia. Our study received ethical approval from the National Medical Research and Ethics Committee (NMRR ID-23-00037-IVH). All patients with native VARA and an unhealthy access vessel wall at the risk of rupture were included. These

This article was accepted: 01 April 2024
Corresponding Author: Muhammad Zaid Haritha bin Mohamed Fauzi
Email: zaidmeister@yahoo.com

included VARA with ulcers with eschar or thinned overlying skin. These patients were referred to our clinic from their respective dialysis centres because of concerns about rupture. Thinned skin was defined as white paper-like skin with loss of subcutaneous fat and normal tissue turgor due to scarring. Patients with pseudoaneurysms, infected fistulas, bleeding VARA, or those who presented with a herald bleed were excluded. In contrast to true aneurysms (VARAs), pseudoaneurysms have a wall defect and are more commonly associated with arteriovenous grafts. We excluded pseudoaneurysms because their pathophysiology is different, and the diseased segment is usually short. All procedures used to treat preexisting peripheral and central outflow stenoses were recorded. Outflow stenoses were categorised into two: central vein (axillary, subclavian, brachiocephalic vein and superior vena cava) and cephalic arch stenoses. All patients underwent preoperative Doppler ultrasound examination to measure the access flow at the brachial artery (Qa1) as its surrogate marker.[8]

All patients underwent central venography under local anaesthesia. Patients with flow-limiting outflow stenoses underwent angioplasty repair using a noncompliant balloon catheter. If complex cephalic arch stenosis was detected, cephalic to axillary vein transposition (CAT) was performed before VARA repair. Patients with central venous occlusion that was not amenable to angioplasty underwent surgical venous bypass. All the patients received a temporary dialysis catheter (TDC) on the contralateral side.

All procedures were performed with consent under general anaesthesia and prophylactic antibiotics without systemic heparin. The scarred or ulcerated skin was excised while creating a cutaneous flap. The venous limb was mobilised and a combination of aneurysmectomy and aneurysmorrhaphy was performed depending on the aneurysm length. Aneurysmectomy was performed between the clamps using the back-wall technique with polypropylene 5/0-6/0 sutures.

During aneurysmorrhaphy, the AVF inflow was clamped temporarily before clamping the aneurysmal segment longitudinally along the venous limb axis. These clamps were applied at a level to match the proximal and distal non-aneurysmal venous limb segments, after which the inflow clamps were released. The excess aneurysmal venous wall was resected above the clamps, leaving a cuff for aneurysmorrhaphy anastomosis in two layers. The repaired vessel was then anchored laterally with the ends of the suture to the bed without overt twisting to allow the native wall to lie anteriorly beneath the cutaneous flap.

Completion venography was performed, and any residual flow-limiting stenosis was repaired endovascularly. The wound was closed with a vacuum drain. Patients were observed in the ward for one week and dialysed using TDC. The TDC was removed after discharge once the repaired VARA was successfully used.

Assessments were made at 2 weeks and 1, 4, 6, and 12 months after surgery by measuring access flow wound and aneurysm recurrence. Qa2 was defined as the last access flow measured during the follow-up. A central venogram was performed if there was clinical suspicion of outflow stenosis and the patient was treated accordingly. Cumulative patency was defined as the time from successful AVF cannulation after VARA repair until access abandonment/end of the study. Primary patency was defined as the time until intervention to maintain patency. Both parameters were measured in months along with the type of intervention.

Statistical analyses were performed using the IBM SPSS Statistics (version 26). Categorical variables were analysed using Fisher's exact test, whereas continuous variables were analysed using the Mann-Whitney U test against a binary outcome and we accepted a p-value for statistical significance of ≤0.05. Kaplan-Meier survival analysis was used to estimate primary and cumulative patency rates.

RESULTS

Over the study period, 64 patients were recruited for the study. Of these, 58 were included in the final analysis. Six patients were excluded from the analysis due to loss to follow-up (4) and deaths unrelated to surgery (2). There were 39 men (67.2%), and the median age of the cohort was 45 years (range=20-75 years). Most of the VARA configurations were BCF (79.3%), and the median fistula age was eight years. The median access flow (Qa1) was 2.2L/min, whereas <7% had a flow of less than 1L/min. None of the patients showed clinical evidence of high-output heart failure. All patients in our cohort had Valentini type 3 VARA with thinned skin or superficial ulcers.[5] The decision regarding the risk of rupture was at the discretion of the operating surgeon. Thirty-nine patients had pre-existing outflow stenosis that required intervention (67.2%), of which 61.5% were at the cephalic arch and 19.0% required surgical reconstruction either by CAT or surgical venovenous bypass using a polyester graft. Most outflow stenoses were associated with BCF (87.2%), and all venovenous bypasses were performed for BCF VARA associated with central occlusion (Table I).

Recurrent outflow stenosis developed in ten patients who were treated with balloon angioplasty. No recurrence of VARA ulcers or skin thinning was observed during the 12-month surveillance period. We performed a sub-analysis of access flow before and after intervention (Qa1 and Qa2), defining high flow as >2L/min, which was present in 61.3% of the cohort before repair. As a large number of these patients had follow-up via telehealth (38.9%), we were unable to record their respective Qa2 and did not proceed with the sub-analysis. Among those who attended the clinic in person (n=22), flow reduction was observed in 63.6% of the patients.

The complications were monitored during the study period. Perioperative access thrombosis developed in 3.5% of patients, occurring in two patients with prior complex outflow intervention. One patient had a cephalofemoral vein bypass created to treat central venous occlusion a month before, whereas the second underwent a CAT and central vein PTA. Both patients underwent perioperative thrombectomy and their access remained patent after 12 months. One patient had a postoperative haematoma that

Table I: Demographic and intervention of VARA repair cohort

Characteristics (n=58)	n (%)	n (median) (interquartile range)
Patient age,years		45 (26) (range 20-75)
Gender		
Male	39 (67.2)	
Female	19 (32.8)	
Age of fistula, years		8 (5) (range 2-19)
VARA configuration		
BCF	46 (79.3)	
BBF	6 (10.3)	
RCF	6 (10.3)	
Pre-intervention brachial artery flow as a surrogate for access flow, L/min		2.2 (1.7) (range: 0.6 to 3.9)
Total outflow stenosis	39 (67.2)	
Central outflow stenosis	15	
Treatment modality		
Central conventional balloon venoplasty	11	
Vein-vein bypass	4	
Cephalic arch stenosis	28	
Treatment modality		
CAS conventional balloon venoplasty	21	
CAT	7	
TDC time, week	3 (0)	

Note: BCF = brachiocephalic fistula; BBF = brachiobasilic fistula; RCF = radiocephalic fistula; CAS = cephalic arch stenosis; CAT = cephalic arch transposition; VARA = vascular access related aneurysm; TDC = temporary dialysis catheter.

Table II: Comparison between the demographic data and cumulative patency at 12 months of repair

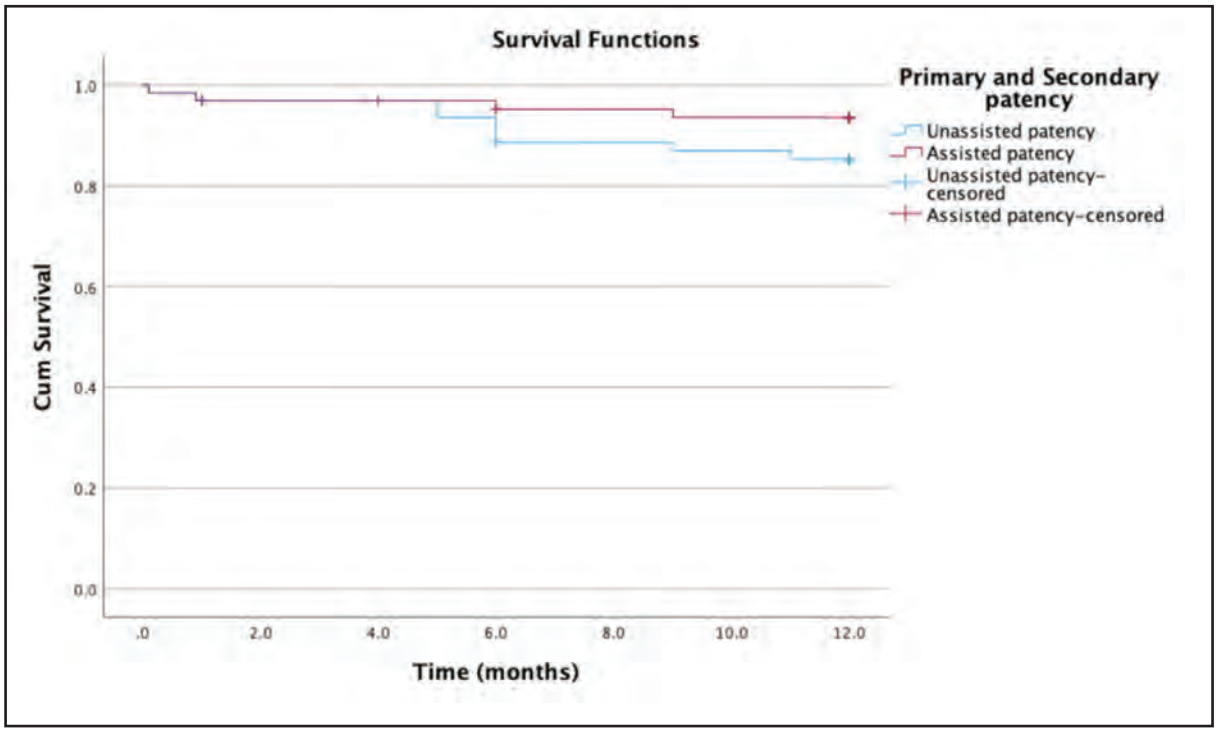
Characteristics	Loss of access n(%) (n=3)	Cumulative patency n(%) (n=55)	p-value
Patient age, year (median)	68.50 (9.19)	44.03 (13.99)	0.02
Gender			
Male	1 (2.6)	38 (97.4)	0.25
Female	2 (10.5)	17 (89.5)	
Age of fistula, year (median)	7.50 (3.54)	7.67 (3.39)	0.95
Fistula configuration			
BBF	0	5 (9.1)	0.44
BCF	2 (66.6)	46 (83.6)	
RCF	1 (33.4)	4 (7.3)	
Fistula flow, L/min (median)	1.15 (0.78)	2.43 (1.06)	0.08
Outflow stenosis	3	36	0.54
Thrombotic event	0	2 (3.64)	1

Note: BCF = brachiocephalic fistula; BBF = brachiobasilic fistula; RCF = radiocephalic fistula. The p-values reported are from univariate analysis.

Table III: Comparison of VARA aneurysmorrhaphy/aneurysmectomy results in the literature

Study	Year	n	Mandrel used	CVS before VARA repair -%	High Qa before VARA repair-%	Unassisted Patency 1y-%	Assisted Patency 1y-%
Hossny et al.[21]	2014	14	yes	Excl.	29*	NA	86
Nezakatgoo et al.[22]	2018	102	yes	NA	NA	NA	NA
Wan et al.[23]	2019	41	yes	Excl.	NA	95	100
Woo et al.[12]	2010	19	yes	20	NA	92.9	NA
Shigala et al.[24]	2014	31	yes	68	29***	65	74
Almehmi et al.[25]	2012	36	no	NA	NA	NA	NA
Patel et al.[10]	2015	48	no	90	NA	73	100
Wang et al.[11]	2017	185	no	71	8**	45	98
Our study		58	no	67	60**	84	94

Note: VARA = venous access-related aneurysm, Qa = access flow, CVS = central venous stenosis, 1y = 1 year, NA = not applicable. *High flow was defined as >1.5L/min. **High flow was defined as >2L/min. ***High flow associated cardiac failure



Time (Months)	0	4	6	12
Number at risk: primary	64	59	52	50
Number at risk: cumulative	64	59	56	55

Fig. 1: Kaplan-Meier curve showing cumulative and primary patency of VARA after repair over 12 months.

required emergency evacuation and haemostasis and the AVF was salvaged. No catheter-related bloodstream infection events were associated with TDC.

Kaplan-Meier survival analysis at 6 and 12 months showed that the primary patency rates were 88.7% and 85.3%, and the cumulative patency rates were 95.2% and 93.5%, respectively (Figure 1). One patient died from a cardiac event during the postoperative period and the second after surgery for intestinal obstruction five months after VARA repair, resulting in an adjusted 1-year patency of 94.8%, a 3-day mortality rate of 1.7%, and an overall mortality rate of 3.4% during the 12-month study period. We found that repaired VARA among younger patients (median age = 44 years) was statistically more likely to remain patent for one year (p=0.02). VARA with a higher access flow was more likely to remain patent one year after repair, although the difference was not statistically significant (p=0.08) (Table II). All the repaired VARA with high Qa1 were patent at 12 months, excluding two due to loss of follow-up.

DISCUSSION

There are various techniques for VARA treatment, although the principles should include exclusion of the aneurysm to reduce the risk of rupture, treatment of outflow stenosis to reduce recurrence, and improvement of access to real estate, as most VARA have unhealthy overlying skin and are tortuous. Strategies employed in VARA repair include external prosthetic mesh, staplers, grafts or mandrels.[7] Synthetic grafts offer shortened operative time but are associated with poor patency.[9] Staplers offer the benefit of speed, however, the device cost is a deterrent in our centre. Relining VARA with stent grafts has been described, though issues with sizing, seal and cannulation relegate this modality as a temporising measure instead.[5] Our experience in repairing VARA has led us to practice aneurysmorrhaphy, as it does not use a prosthetic graft and is cost-saving with regards to operative consumables.

The patency rate of our series compares favourably with the literature on VARA aneurysmorrhaphy.[7] We identified two studies that had similar patient characteristics (Table III). In the series by Patel et al., they selectively performed single- or two-stage repairs after routine fistulography,[10] whereas Wang et al., performed partial aneurysmectomy for all their VARA patients.[11] Both groups had a high proportion of outflow angioplasty, although they did not have many high-flow VARA. In a report by Wang et al., most angioplasties before and after VARA repair were for stenosis at the cephalic arch (33% and 23%, respectively).[11] Both studies reported far lower rates of TDC (2% and 23%, respectively) compared to our study. Neither categorised the VARA morphology, and it is most likely that those that required TDC had a complex VARA, that is, type 3. Sigala et al., found a similarly high proportion of outflow stenosis, though their repair employed a mandrel.[3] Woo et al., reported excellent primary patency rates in their cohort, although a surprisingly small proportion of patients had outflow stenosis.[12] All other studies on VARA aneurysmorrhaphy either excluded or did not detail the central stenosis.

In this report, we highlight the role of access flow and outflow stenosis in the pathogenesis of VARA. In the literature, outflow stenosis is present in 78% of VARA, whereas the incidence in our cohort was 67%. The location of stenoses varies depending on the AVF configuration.[13] Treating these stenoses reduces recurrence and aids in wall integrity during aneurysmorrhaphy by reducing the wall tension. We employed a similar approach to outflow stenosis as Patel et al.,[10] whereby all patients underwent a fistulogram before VARA repair.

Rajput et al.,[13] found that apart from the arm cephalic vein, most stenoses among BCF VARA were located at the cephalic arch. In our study, CAS was found in 85.3% of BCF VARA with outflow stenosis and 2.9 times more likely than a central disease. This preponderance is attributed to multiple factors including altered flow and WSS, extrinsic effects of the chest wall fascia, venous valves and possibly arch morphology.[14] In our experience, complex CAS is best treated with CAT as this also negates the effects of the chest fascia and arch morphology.[15] In our study, recurrent central stenoses were repaired with balloon angioplasty instead of stents due to cost and concerns of extrinsic compression with stent fracture and inadvertent coverage of collaterals.[5,6]

The second factor we highlight is the access flow. Sixty-one percent of our cohort had an access flow of more than 2L/min. High access flow promotes outflow stenosis and aneurysm formation.[16] Various techniques have been described to reduce fistula flow with the intent of reducing aneurysmal progression/recurrence.[17,18] Based on the literature, flow-limiting procedures should be incorporated in the repair of high-flow VARA. We found that VARA with high Qa1 tended to have better patency after aneurysmorrhaphy than those with Qa1 <1L/min (Table II).

A challenge in aneurysmorrhaphy is to estimate an appropriate neoluminal size. We employed an individual approach whereby the neolumen matched the outflow non-aneurysmal vessel, thus promoting laminar flow. An added benefit of avoiding mandrel use is prevention of prolonged vessel clamping and systemic anticoagulation. We avoided perioperative systemic heparin administration due to the risk of bleeding-related complications.[19] Our study had two intraoperative thrombotic events occurring after clamping, both among patients with complex outflow diseases. We currently employ a selective approach to systemic anticoagulation instead of not-at-all.

Given the complexity of treating VARA, detecting the disease at an earlier stage will improve patient morbidity and outcome. Primary prevention measures include avoiding 'general area cannulation' which is a known risk factor for developing AV aneurysms. Instead, healthcare personnel at dialysis centres should practice buttonhole or rope ladder techniques.[5] As discussed earlier, outflow stenosis is a risk factor for VARA and may manifest with raised venous pressure, prolonged bleeding from puncture sites after dialysis or a pulsatile AVF and patients with these signs should be referred by their primary physician or dialysis centre personnel. Late referrals may lead to aneurysm development, loss of access, rupture, and exsanguination. Early detection based on a high level of suspicion will expedite management, whereby the vascular access team can discuss with the patient treatment options, including salvage or creation of a new AVF while the existing AVF is still usable.

LIMITATIONS

Not all patients completed our face-to-face clinical surveillance because of logistics. Instead, many had telehealth follow-up. Others who were not contactable (6.0%) formed further bias due to the loss of follow-up. The statistical data in this study were analysed using univariate analysis. As the number of patients with loss of patency was low, we were unable to make accurate conclusions regarding the factors that contributed to this. Our findings were more likely to be affected by the treatment of outflow disease than by aneurysmorrhaphy. Finally, we were not able to control cannulation techniques, as they were performed in external centres.

CONCLUSION

VARA is highly associated with outflow stenosis and a high Qa. Good 1-year cumulative patency may be achieved among younger patients and those with higher pre-interventional brachial arterial flow, measured as a surrogate for access flow. The treatment strategy is multifaceted, and treating outflow disease is the cornerstone of managing VARA, whereas surveillance for recurrence is paramount. Early detection at dialysis centres can expedite earlier referrals and potentially salvage AVFs.

CONFLICT OF INTEREST

None

FUNDING

None

ACKNOWLEDGEMENTS

The authors thank the late Zainal Ariffin Azizi, Department of Surgery, Hospital Kuala Lumpur for his mentorship during the inception of this study.

REFERENCES

1. Inston N, Mistry H, Gilbert J, Kingsmore D, Raza Z, Tozzi M, et al. Aneurysms in vascular access: state of the art and future developments. J Vasc Access 2017; 18(6): 464-72.
2. Balaz P, Björck M. True aneurysm in autologous hemodialysis fistulae: definitions, classification and indications for treatment. J Vasc Access 2015; 16(6): 446-53.
3. Sigala F, Kontis E, Saßen R, Mickley V. Autologous surgical reconstruction for true venous hemodialysis access aneurysms—techniques and results. J Vasc Access 2014; 15(5): 370-5.
4. Al-Thani H, El-Menyar A, Al-Thani N, Asim M, Hussein A, Sadek A, et al. Characteristics, management, and outcomes of surgically treated arteriovenous fistula aneurysm in patients on regular hemodialysis. Ann Vasc Surg 2017; 41: 46-55.
5. Lok CE, Huber TS, Lee T, Shenoy S, Yevzlin AS, Abreo K, et al. KDOQI clinical practice guideline for vascular access: 2019 update. Am J Kidney Dis 2020; 75(4): S1-164.

6. Schmidli J, Widmer MK, Basile C, de Donato G, Gallieni M, Gibbons CP, et al. Editor's choice – vascular access: 2018 clinical practice guidelines of the European Society for Vascular Surgery (ESVS). Eur J Vasc Endovasc Surg 2018; 55(6): 757-818.
7. Baláž P, Rokošný S, Bafrnec J, Whitley A, O'Neill S. Repair of aneurysmal arteriovenous fistulae: a systematic review and meta-analysis. Eur J Vasc Endovasc Surg 2020; 59(4): 614-23.
8. Zamboli P, Fiorini F, D'Amelio A, Fatuzzo P, Granata A. Color Doppler ultrasound and arteriovenous fistulas for hemodialysis. J Ultrasound 2014; 17(4): 253-63.
9. Chang J, Hejna EE, Terranella SL, Trawczynski MH, Hollinger EF, Jensik SC, et al. Graft repair of arteriovenous fistula aneurysms is associated with decreased long-term patency. Am J Surg 2022; 224(1): 136-40.
10. Patel MS, Street T, Davies MG, Peden EK, Naoum JJ. Evaluating and treating venous outflow stenoses is necessary for the successful open surgical treatment of arteriovenous fistula aneurysms. J Vasc Surg 2015; 61(2): 444-8.
11. Wang S, Wang MS. Successful use of partial aneurysmectomy and repair approach for managing complications of arteriovenous fistulas and grafts. J Vasc Surg 2017; 66(2): 545-53.
12. Woo K, Cook PR, Garg J, Hye RJ, Canty TG. Midterm results of a novel technique to salvage autogenous dialysis access in aneurysmal arteriovenous fistulas. J Vasc Surg 2010; 51(4): 921-5.e1.
13. Rajput A, Rajan DK, Simons ME, Sniderman KW, Jaskolka JD, Beecroft JR, et al. Venous aneurysms in autogenous hemodialysis fistulas: is there an association with venous outflow stenosis? J Vasc Access 2013; 14(2): 126-30.
14. Sivananthan G, Menashe L, Halin NJ. Cephalic arch stenosis in dialysis patients: review of clinical relevance, anatomy, current theories on etiology and management. J Vasc Access 2014; 15(3): 157-62.
15. Sigala F, Saßen R, Kontis E, Kiefhaber LD, Förster R, Mickley V. Surgical treatment of cephalic arch stenosis by central transposition of the cephalic vein. J Vasc Access 2014; 15(4): 272-7.
16. Miller GA, Hwang WW. Challenges and management of high-flow arteriovenous fistulae. Semin Nephrol 2012; 32(6): 545-50.
17. Miller GA, Goel N, Friedman A, Khariton A, Jotwani MC, Savransky Y, et al. The MILLER banding procedure is an effective method for treating dialysis-associated steal syndrome. Kidney Int 2010; 77(4): 359-66.
18. Berard X, BrizziV., Mayeux S, Sassoust G, Biscay D, Ducasse E, et al. Salvage treatment for venous aneurysm complicating vascular access arteriovenous fistula: use of an exoprosthesis to reinforce the vein after aneurysmorrhaphy. Eur J Vasc Endovasc Surg 2010; 40(1): 100-6.
19. Smith GE, Souroullos P, Cayton T, Harwood A, Carradice D, Chetter IC. A systematic review and meta-analysis of systemic intraoperative anticoagulation during arteriovenous access formation for dialysis. J Vasc Access 2016; 17(1): 1-5.
20. Hossny A. Partial aneurysmectomy for salvage of autogenous arteriovenous fistula with complicated venous aneurysms. J Vasc Surg 2014; 59(4): 1073-7.
21. Nezakatgoo N, Kozusko SD, Watson JT, Empting R, Shahan CP, Rohrer MJ. A technique for the salvage of megafistulas allowing immediate dialysis access. J Vasc Surg 2018; 68(3): 843-8.
22. Wan Z, Lai Q, Zhou Y, Chen L, Tu B. Partial aneurysmectomy for treatment of autologous hemodialysis fistula aneurysm is safe and effective. J Vasc Surg 2019; 70(2): 547-53.
23. Almehmi A, Wang S. Partial aneurysmectomy is effective in managing aneurysm associated complications of arteriovenous fistulae for hemodialysis: case series and literature review. Semin Dial 2012; 25(3): 357-64.