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# Surgical intervention for upper extremity nerve compression related to arteriovenous hemodialysis accesses

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## Abstract

**Objective:** Chronic renal failure patients with arteriovenous hemodialysis access may exhibit pain and neurological complaints due to local nerve compression by the access conduit vessels of autogenous arteriovenous fistulas or the prosthesis of arteriovenous grafts. In this study, we have examined the results of surgical intervention for vascular access–related nerve compression in the upper extremity.

**Methods:** A single center retrospective study was performed of all patients referred for persistent pain and neurological complaints after vascular access surgery for hemodialysis. There were four brachial-cephalic, three brachial-basilic upper arm arteriovenous fistulas, and three prosthetic arteriovenous grafts. All patients had pain and sensory deficits in a distinct nerve territory (median nerve: 6; median + ulnar nerve: 1; medial cutaneous nerve: 1), and two patients had additional motor deficits (median nerve).

**Results:** A total of 10 patients (mean age: 59 years; range: 25–73 years; 2 men; 4 diabetics) were treated by surgical nerve release alone (2 patients) or in combination with access revision (8 patients). Mean follow-up was 23 months (range: 8–46 months). Direct complete relief of symptoms was achieved in six patients. Three patients had minor complaints, and one patient had a reoperation with good success.

**Conclusion:** Vascular access–related nerve compression is an uncommon cause for pain, sensory and motor deficits after vascular access surgery. Surgical nerve release and access revision have good clinical outcome with relief of symptoms and maintenance of the access site in the majority of patients.

## Keywords

Arteriovenous fistula, dialysis access, prosthetic grafts, nerve compression, surgical intervention, aneurysmorrhaphy

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## Introduction

Chronic renal failure (CRF) patients on intermittent hemodialysis treatment may exhibit a variety of neurological complaints as pain, motor and sensory deficits in their ipsilateral vascular access extremity. Perioperative nerve injury, ischemic monomelic neuropathy (IMN), and complex regional pain syndrome (CRPS) are considered complications of the initial vascular access creation.[1,2] In addition, systemic neuropathy resulting from uremia or diabetes mellitus may be present in these patients and are distinguishable from the above-mentioned neurological conditions because both upper and/or lower limbs are usually affected.[3] Hemodialysis access–induced distal ischemia (HAIDI) due to low peripheral perfusion after surgery may cause pain and eventually tissue loss. These patients usually have discoloration, coldness, and pain in the hand on exercise. Nerve compression and various

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**Table 1.** Pre- and postoperative access flow (Qa), digital pressure (DP), and digit/brachial index (DBI).

| Patient | Type of access | Time to intervention (months) | Preoperative DP (DBI) (mm Hg) | Postoperative DP (DBI) (mm Hg) | Preoperative Qa (mL/min) | Postoperative Qa (mL/min) | Follow-up (months) |
|---|---|---|---|---|---|---|---|
| 1 | UA loop PTFE | 4 | 140 (1.0) | 150 (0.97) | 2659 | 1320 | 19 |
| 2 | tBBAVF | 28 | 75 (0.71) | 70 (0.57) | 1545 | – | 4 |
| 3 | UA loop PTFE | 7 | 100 (0.98) | 122 (1.02) | 1215 | – | 23 |
| 4 | tBBAVF | 21 | 160 (0.89) | 145 (0.98) | 980 | 1060 | 8 |
| 5 | BCAVF | 14 | 110 (0.79) | 105 (0.7) | 1620 | 1430 | 17 |
| 6 | UA loop PTFE | 1 | 140 (0.88) | 135 (0.70) | 1340 | 1531 | 14 |
| 7 | BCAVF | 49 | 150 (1.0) | 129 (0.93) | 1514 | 1215 | 33 |
| 8 | BCAVF | 36 | 80 (0.72) | 55 (0.68) | 985 | – | 6 |
| 9 | eBBAVF | 75 | 125 (0.72) | 125 (0.74) | 1730 | 1890 | 19 |
| 10 | BCAVF | 108 | 105 (0.66) | 144 (1.03) | 3454 | 1246 | 14 |

UA AVG: upper arm arteriovenous graft; t/eBBAVF: transposed/elevated brachial-basilic arteriovenous fistula; BCAVF: brachial-cephalic arteriovenous fistula; DP: digital pressure; DBI: digit/brachial index; Qa: access flow.

entrapment syndromes such as carpal tunnel syndrome, ulnar and radial nerve entrapment occur significantly more in the dialysis population as compared to the general population.[4] However, it is unclear whether these syndromes are directly related to the presence of the vascular access. A rare cause for pain and neurological complaints is local nerve compression by the dilated vessels of the vascular access conduit or graft in dialysis patients with autogenous arteriovenous fistulas (AVF) and prosthetic arteriovenous grafts (AVG), respectively.

Hereby, we report on the results of surgical intervention for upper extremity vascular access–related nerve compression in hemodialysis patients. Because this was a retrospective patient study, a waiver from the institutional medical ethical committee was obtained.

## Patients and methods

From 1 January 2012 to 1 April 2018, 10 CRF patients with an arteriovenous hemodialysis access and symptoms of pain and nerve impairment in the ipsilateral upper extremity were referred to a tertiary vascular access center. There were three male and seven female patients with a mean age of 59 years (range: 25–73 years). Five patients had diabetes, and two patients had a history of coronary artery disease (CAD). Three patients had a prosthetic AVG in a looped configuration in the upper arm with anastomoses to the axillary artery and vein. Seven patients had either an autogenous brachial-cephalic (4) and brachial-basilic upper arm AVF with a transposed (2) or elevated (1) basilic vein. The majority of patients complained of persistent pain and sensory loss in a distinct nerve territory of the arm (median nerve: 6; median + ulnar nerve: 1; medial cutaneous nerve: 1). Two patients had sensory as well as motor deficits with numbness and loss of muscle strength in the hand.

Physical examination exhibited sensory loss in the medial forearm (medial cutaneous nerve), digits (median and ulnar nerve), and loss of muscle strength in index and long digits lumbricals (median nerve). There was no discoloration, coldness, or paresis of the hand, which might indicate low perfusion or monomelic neuropathy. Vascular access ultrasonography showed dilated and elongated inflow arteries as well as fistula outflow veins in the patients with either brachial-cephalic arteriovenous fistulas (BCAVF) or brachial-basilic arteriovenous fistulas (BBAVF). In addition, access flow and digital blood pressure measurements with and without access compression were performed to rule out high-flow access and/or low distal perfusion, indicating hand ischemia. The patients had a mean digital pressure (DP) measurement with open access of 118 mm Hg (range: 75–160 mm Hg) and mean digit/brachial index (DBI) of 0.84 (range: 0.66–1.0), indicating a sufficient distal perfusion. Access compression raised DP and DBI to a mean of 145 mm Hg and 0.95, respectively. The mean preoperative (10 patients) and postoperative (7 patients) access flows were 1704 and 1384 mL/min, respectively; 3 patients had a ligated access with central vein catheter (CVC) (Table 1; patient no.: 2, 3, and 8). Nerve conduction studies were performed in eight patients (normal in five and median nerve injury in three patients). Nerve conduction studies could not be performed in two patients, because of technical reasons (upper loop AVG with axillary anastomoses). Five patients had magnetic resonance imaging (MRI) of the upper extremity. In these subjects, a close relationship of the nerves to the vessels and prosthesis in the axilla (two patients) and elbow (three patients) was noticed. In the majority of patients, the artery was grossly dilated and/or elongated (Figure 1). MRI studies were not performed in five patients, because of the presence of a pacemaker (one patient) and claustrophobia (four patients).

### *Operative technique*

An S-shaped incision was made at the level of the elbow or a longitudinal incision dorsally of the biceps muscle in the upper arm. The upper arm BCAVF and BBAVF

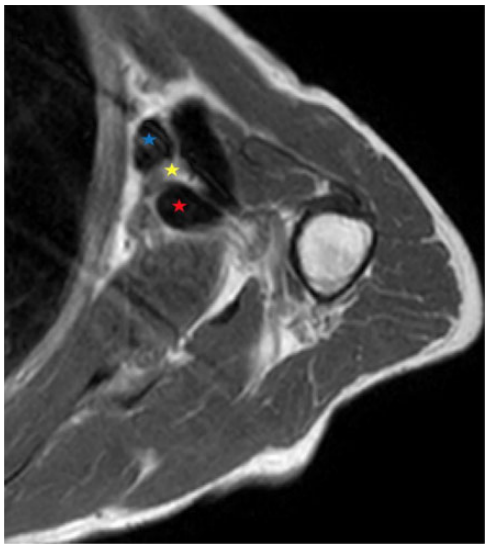

**Figure 1.** Magnetic resonance imaging of the upper arm in a patient with a brachial-basilic arteriovenous fistula. Notice the median nerve compression (yellow star) by the dilated and elongated brachial artery (red star) and basilic vein (blue star).

arteriovenous anastomoses and/or artery were dissected free from the surrounding tissues, and subsequently the median nerve was released by incising the medial intermuscular septum and bicipital aponeurosis over a distance of 5–10 cm. In the patient with ulnar nerve entrapment, the fascia over the nerve was incised over a distance of 5 cm at the level of the medial epicondyle of the elbow. Cutaneous nerve compression was relieved by dissecting the nerve away from the elevated basilic vein with transection of a single nerve branch for the purpose of adequate nerve mobilization. In this patient, a second stage operation was needed because of return of pain, and the vein was transected 1-cm proximal from the anastomosis, pulled away from the nerve, and reanastomosed to the vein stump. Axillary vessel exploration was performed with dissection of the artery-to-graft anastomosis and proximal prosthesis. Subsequently an end-to-end interposition was performed with a 5-cm segment of a 6-mm stretch expanded polytetrafluoroethylene (ePTFE; Gore-Tex, Flagstaff, AZ, United States) prosthesis between the initial graft (2 cm from the arterial anastomosis) to the distal graft, with a running polypropylene (Prolene) 6 × 0 suture. Revision of the venous aneurysmatic anastomosis vessels was performed with aneurysmorrhaphy to a vessel diameter of 10–15 mm. In addition, one patient with a BCAVF had reduction of the arteriovenous anastomosis to a length of 7 mm. AVF closure was performed by transection of the efferent vein 1-cm proximal of the arteriovenous anastomosis and running polypropylene (Prolene) 6 × 0 suture ligation of the vascular stump.

## Results

From 2012 to 2018, a total of 1471 vascular access–related operations were performed in a tertiary referral center. Of these, 55 operations were performed for HAIDI and 10 patients with vascular access–related upper extremity nerve compression were treated by surgical nerve release with or without vascular access revision. Acute onset nerve compression (<4 week after vascular access placement) was noticed in three patients with a prosthetic upper arm AVG, while seven patients (4 BCAVF; 3 BBAVF) encountered late onset nervous complaints after a mean period of 47 months (range: 14–108 months). Two patients underwent nerve release only (median and medial cutaneous nerve), three patients had median nerve compression release in combination with access revision (aneurysmorrhaphy; narrowing AV anastomosis), and two patients median nerve release with access closure (one patient had a functional renal transplant and the other patient continued hemodialysis with a CVC). The patients with an acute onset nerve compression due to upper arm looped AVGs were treated either by interposition of a stretch ePTFE graft (Gore-Tex, Flagstaff, AZ, United States) at the side of the arterial anastomosis (two patients) or graft explantation with median and ulnar nerve release (one patient). The latter patient received a tunneled CVC in the right internal jugular vein to continue dialysis treatment. DB indices remained stable after surgical intervention (mean: 0.83), while the mean access flow diminished to 1384 mL/min (range: 1060–1890 mL/min).

The outcome of surgical nerve release is listed in Table 2. Complete relief of symptoms was achieved in 6 of 10 patients, while minor residual symptoms remained in 4 patients.

There were no surgery-related complications. Dialysis treatment was successfully continued through the initial vascular access in seven patients. The patient with a brachial-basilic AVF with vein elevation and initial cutaneous nerve release had recurrence of symptoms with intractable pain during needling after a period of 28 months. Transposition of the basilic vein alleviated most of the complaints. The other patients were free of symptoms after a mean follow-up of 16 months (range: 4–33 months).

## Discussion

Pain in the upper extremity after vascular access surgery may be caused by a variety of issues, like arterial, venous, and nervous conditions. An adequate differentiation and diagnosis of these causes are of utmost importance for a successful treatment. Acute and late onset pain might be induced by, for example, a low peripheral perfusion, either causing HAIDI or IMN. In the latter situation not only pain but loss of sensibility and paresis urge to an

**Table 2.** Results of surgical intervention for vascular access–related nerve compression.

| Patient | Type of access | Intervention | Functional AVF | Outcome |
|---|---|---|---|---|
| 1 | UA AVG | Stretch ePTFE graft interposition arterial side | + | Partial relief |
| 2 | tBBAVF | Median nerve release<br>AVF closure/CVC | – | Complete relief |
| 3 | UA AVG | Median + ulnar nerve release; graft removal/CVC | – | Complete relief |
| 4 | tBBAVF | Median nerve release | + | Complete relief |
| 5 | BCAVF | Median nerve release<br>Aneurysmorrhaphy | + | Partial relief |
| 6 | UA AVG | Stretch ePTFE graft interposition arterial side | + | Complete relief |
| 7 | BCAVF | Median nerve release<br>Aneurysmorrhaphy | + | Complete relief |
| 8 | BCAVF | Median nerve release<br>AVF closure | – | Partial relief |
| 9 | eBBAVF | Cutaneous nerve release | + | Partial relief |
| 10 | BCAVF | Median nerve release<br>Aneurysmorrhaphy + anastomosis reduction | + | Complete relief |

UA AVG: upper arm arteriovenous graft; t/eBBAVF: transposed/elevated brachial-basilic arteriovenous fistula; BCAVF: brachial-cephalic arteriovenous fistula; CVC: central vein catheter.

acute intervention with access ligation. Acute ischemia usually occurs in diabetic patients with poor arm arteries and elbow AVFs, while HAIDI might develop slowly due to an increased access flow, inadequate collaterals, and steal in particular with BCAVFs. In these patients, flow reduction (banding; revision using distal inflow [RUDI] and/or bypass procedures: distal revascularization with or without interval ligation [DRIL]) may alleviate the symptoms.

Nerve problems in the extremities are not uncommon in patients undergoing hemodialysis. Many patients will suffer from symptoms of the underlying systemic neuropathy due to uremia or diabetes mellitus and may have complaints of pain and/or sensory loss, usually symmetrically in both extremities. Nerve injury during vascular access surgery might occur in particular with the creation of radial-cephalic wrist access and brachial-basilic upper arm AVF (sensory branch radial nerve and medial cutaneous nerve, respectively), but these are rarely debilitating.[5]

Another reason for pain and/or sensory deficits is nerve compression due to surrounding structures like ligaments and vessels, of which the carpal tunnel syndrome is the most common condition.[4] A rare condition is nerve compression by dilated and aneurysmatic vessels, exerting pressure on nervous structures, which may cause sensory and motor deficits. In a published series of 10 patients with true arterial aneurysms in the arm, all patients presented with painful masses and neurologic symptoms due to nerve compression. In addition, distal embolization caused digital ischemia in 50% of patients. Repetitive blunt vascular trauma was held responsible for aneurysm formation in five patients.[6,7] Upper limb false aneurysms are usually caused by penetrating trauma, a disrupted arteriovenous anastomosis, or develop after miscannulation of an AVF. The surgical procedures are aneurysmorrhaphy, excision with end-to-end arterial anastomosis, excision, and artery ligation with subsequently reversed autogenous vein interposition graft. The results of these surgical interventions are usually good, and median nerve deficits from compression return to normal after aneurysm repair in most patients.[8–11] True brachial artery aneurysms in dialysis patients are relatively uncommon and may occur many years after ligation of the vascular access, for instance after successful renal transplantation. The usual manifestation is an accidental finding of a pulsatile, painless, and asymptomatic mass in the upper arm. Complications include neurological complaints, aneurysm sac thrombosis, thromboembolic ischemic events, and disruption with bleeding. High access flow, immunosuppressive therapy after renal transplantation, and increased resistance following ligation of the AVF may accelerate the process of vascular dilation.[12–15]

An underestimated cause for neurological complaints in dialysis patients with a functional vascular access is the local nerve compression by the enlarging and often meandering inflow artery and outflow vein, which is the result of ongoing arterial and venous remodeling. In particular, BCAVF and BBAVF are prone to excessive vessel dilation and the high access flows from these AVFs are kept responsible for this phenomenon. A local dilation of the arteriovenous anastomotic complex in the elbow (BCAVF) adds to the risk of median nerve compression. In these patients, a combination of access flow reduction and aneurysmorrhaphy is indicated, to achieve a good and sustained long-term outcome.

In our study, four patients with BCAVF and BBAVF had relatively low DB indices and one patient had previously RUDI performed. Postoperative DBIs were similar in three patients, with complete relief of symptoms in two patients, indicating nerve compression as the major cause

**Table 3.** Literature of vascular access–related nerve compression syndrome.

| Author | No. of patients | Type of access | Symptoms | Territory |
|---|---|---|---|---|
| Reinstein et al.[16] | 3 | BBAVF/UA AVG | Sensory | Median and radial nerve |
| Ergungor et al.[17] | 1 | RCAVF | Sensory + motor | Median nerve |
| Sawin et al.[18] | 2 | BCAVF | Sensory + motor | Interosseous and radial nerve |
| Goldstein et al.[19] | 1 | UA AVG | Sensory + motor | Median nerve |
| Zerbi et al.[20] | 1 | RCAVF | Sensory + motor | Median nerve |
| Marzelle et al.[21] | 2 | BCAVF | Sensory | Median nerve |
| Kordzadeh et al.[22] | 4 | BCAVF | Sensory | Median nerve |
| Chang et al.[23] | 1 | LA AVG | Sensory | Median nerve |
| Matsuda et al.[24] | 1 | RCAVF | Sensory | Cutaneous nerve |
| Schilling et al.[25] | 1 | BCAVF | Sensory | Cutaneous nerve |

UA/LA AVG: upper arm/lower arm arteriovenous graft; BBAVF: brachial-basilic arteriovenous fistula; BCAVF: brachial-cephalic arteriovenous fistula; RCAVF: radial-cephalic arteriovenous fistula.

for symptoms. Eight patients had additional access revision or ligation. One might argue that aneurysmorrhaphy, anastomosis length reduction, or access ligation lowers flow and improve peripheral perfusion, but this was not proven by an increase in DBIs. In the patients without access revision, DBI remained similar (preoperative 0.80 vs postoperative 0.69), while patients with additional access interventions had a non-significant rise in DBI (0.75 vs 0.87). Only one patient (aneurysmorrhaphy + anastomosis length reduction) had a significant rise in DBI and decrease in flow, and in this case a concurrent ischemic steal syndrome might probably be the case.

Vascular access surgery, involving the axillary region, is more prone for the occurrence of nerve complications. The axillary artery is usually located behind the venous and nervous structures, and dissection can be more difficult and may cause vein or nerve damage. In addition, implantation of a rigid vascular access graft might compress the nerves if it travels from the depth (arterial anastomosis) to the superficial subcutaneous region in case of an upper arm looped prosthetic AVG with anastomoses to the axillary vessels. These patients will exhibit an acute onset of pain and neurological complaints, hindering the successful use of the newly created vascular access. Exploration of the arterial anastomosis in the axilla and creating a smooth loop with a stretch ePTFE interposition graft relieves the pressure on the nerve with subsequently disappearance of symptoms. In the three patients with axillary exploration in this study, no nerve injury at the access creation was found, only local nerve compression was noticed. It is of utmost importance to differentiate nerve problems from symptoms due to peripheral ischemia. Abnormal digital pressure measurements and determination of the DBI may reveal ischemia, while nerve conduction and MRI studies are indispensable to diagnose local nerve entrapment and compression.

Prevention of vascular access–related nerve compression is difficult to accomplish. In particular, in the elbow region, several structures are closely related to each other, and therefore either arterial or venous dilation may compress the median nerve in the long term. In patients with an upper arm loop AVG with axillary anastomoses, one might consider a wider loop of prosthetic graft around the nerve or the use of more stretchable grafts to prevent nerve compression.

The phenomenon of arteriovenous hemodialysis access–related nerve compression has been scarcely reported from the literature.[16–25] Most patients had elbow and upper arm autogenous AVFs with sensory deficits in the territory of the median nerve. The outcome of surgical nerve release was generally good with relief of symptoms and maintenance of the vascular access (Table 3).

## Conclusion

Acute and late onset upper extremity nerve compression is an underestimated cause for pain and neurological complaints in CRF patients with arteriovenous hemodialysis access. This condition should be differentiated from access-induced ischemia and/or IMN. Surgical nerve release with vascular access revision is the treatment of choice and results in an acceptable outcome with disappearance of complaints and functional vascular access in most patients.

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