Neurological improvement following revision of vascular graft remnants in the upper extremity

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ABSTRACT

Remnant vascular grafts may result in significant neurological deficits owing to compression of adjacent neural structures. We report this finding in two cases after extracorporeal membrane oxygenation decannulation and removal of an arteriovenous fistula in the upper extremity. In both cases, removal of the graft, patch arteriotomy, and external neurolysis resulted in significant recovery of neurological function. We review the preoperative workup, diagnostic studies, and technical approach to treatment in an effort to increase recognition among vascular and cardiovascular surgeons and to demonstrate a safe and effective management option through a multidisciplinary approach. (J Vasc Surg Cases Innov Tech 2024;10:101539.)

Keywords: Vascular graft; Peripheral nerve; Compression; Multidisciplinary; Neurolysis

Rates of extracorporeal membrane oxygenation (ECMO) have increased over the years, and arteriovenous fistulas provide vascular access commonly for hemodialysis patients.[1,2] Despite the life-saving nature of these vascular interventions, the close proximity of blood vessels and nerves may lead to nerve injury with resultant pain, weakness, or sensory change. Direct injury to a nerve may occur owing to needle puncture or manual pressure.[3-6] In other cases, neurological deficit may result from compression owing to the presence of a mass, such as a hematoma, pseudoaneurysm, or vascular graft.[7] In such cases, nerve decompression with or without removal of the offending mass can yield clinical improvement.[8]

Cannulas are removed regularly once the need for ECMO has resolved, and arteriovenous grafts for hemodialysis may be removed for infection or other reasons. It is common practice to leave behind a graft remnant, or stump, rather than remove the graft fully in an effort to avoid damaging the parent blood vessel. We report unique clinical scenarios in which an ECMO access device and arteriovenous graft were removed primarily, but the stumps of vascular graft that remained resulted in severe upper extremity neurological deficits. In both cases, removal of the remnant graft, patch arteriotomy, and nerve decompression resulted in significant neurological improvement. We review the clinical signs and symptoms suggestive of nerve compression after primary graft removal, diagnostic studies, timing of referral, and a multidisciplinary surgical strategy for treatment. Both patients provided consent for publication.

CASE 1

A 69-year-old woman with a history of Takotsubo cardiomyopathy was admitted to the hospital after ECMO decannulation of the right axillary artery. The patient reported significant weakness and pain in the right upper extremity. On examination 6 months after surgery, the patient had no intrinsic hand movement and minimal grip strength with a complete wrist drop. A Tinel's sign was noted over the prior incision in the infraclavicular region. The patient had a palpable radial pulse. A duplex ultrasound of the right upper extremity arteries showed patent vessels with no pseudoaneurysm, hematoma, or deep venous thrombosis. Electromyography (EMG) and a nerve conduction study (NCS) showed severe injury of the ulnar innervated muscles. Magnetic resonance imaging (MRI) of the right brachial plexus showed a small area of T2 hypointensity inferior to the right clavicle (Fig 1). There was signal abnormality at the infraclavicular brachial plexus level, which likely encompassed the right brachial plexus divisions and/or cords. Surgical exploration was recommended.

Under general endotracheal anesthesia, the vascular surgeon opened the previous incision and a 2-cm bulbous remnant of the ECMO graft was encountered at the right axillary artery. The approximate location of the compression is noted in Fig 2 (case 1). Vessel loops were placed around the axillary artery proximally and distally to the graft. An external neurolysis of the cords and branches of the brachial plexus in the region was performed by the neurosurgeon. Continued splaying of

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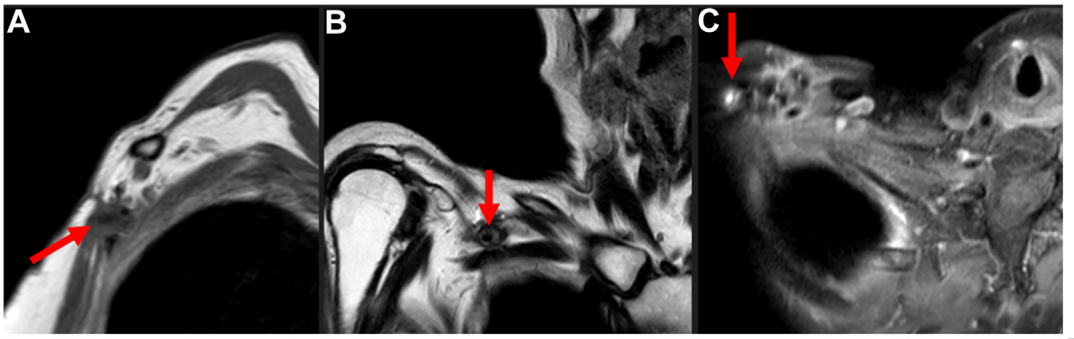

Fig 1. Magnetic resonance imaging (*MRI*) of the brachial plexus showing neural involvement. A red arrow shows a 2-cm circular region of hypointensity inferior to the right clavicle in close proximity to the brachial plexus on sagittal **(A)** and coronal **(B)** T1-weighted sequences. There is an associated focus of abnormal contrast enhancement on T1 contrasted axial sequence **(C)**.

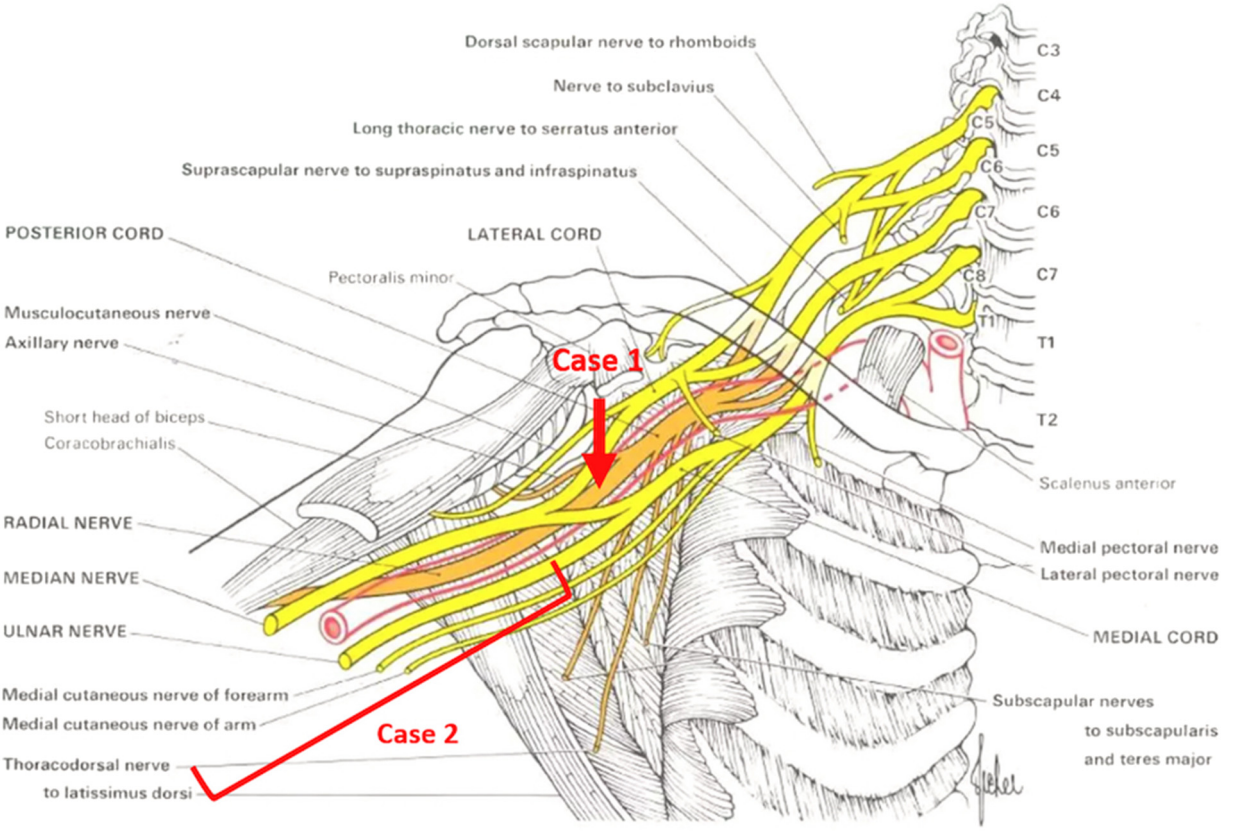

Fig 2. Anatomical relationship between the brachial plexus and vasculature. Sites of neural compression are noted in case 1 in the infraclavicular region along the axillary artery (*arrow*) and in case 2 between the axilla and antecubital fossa along the brachial artery (*bracket*). (Adapted from Figure 3 in Aids to the Examination of the Peripheral Nervous System, Fifth Edition; Saunders Elsevier, 2010.)

the neural elements owing to mass effect from the remnant graft was noted. The old graft was resected. The resultant arteriotomy was closed with a bovine pericardium patch. It was sewn such that the patch was flush with the axillary artery (Fig 3). No other compression sites along the brachial plexus were identified. The wound was closed. The patient reported improvement at her 2-week follow-up visit. At the 6-month follow-up visit, she had near complete return of hand intrinsic, grip, and wrist extension strength with mild clawing of her fourth and fifth digits. She had complete relief of preoperative pain. She had mild residual numbness in her fingers. She has returned to driving and reports significant improvement in quality of life.

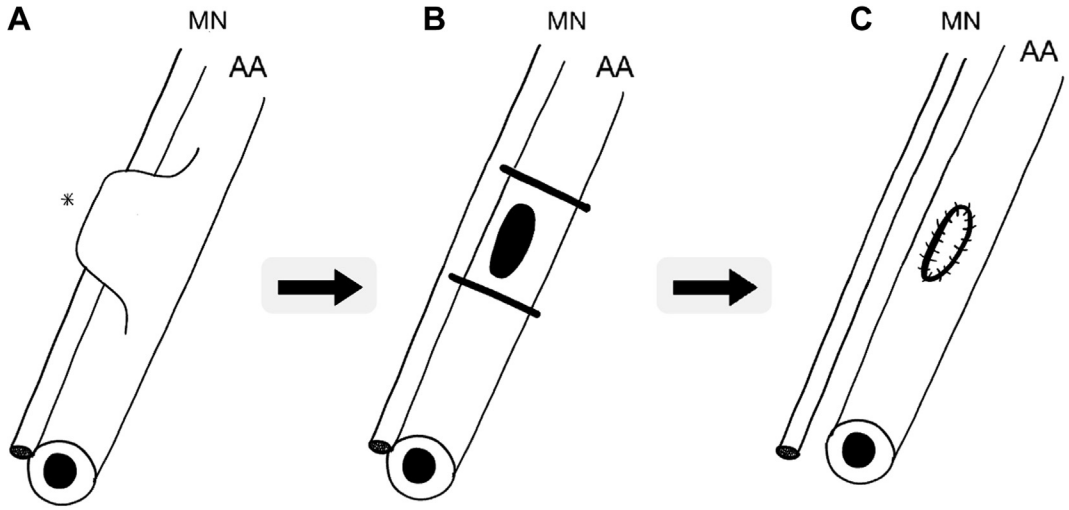

Fig 3. Schematic of surgical intervention. **(A)** The remnant graft arising from the brachial artery (BA) is compressing the median nerve (*MN*) (*). **(B)** Clamps (*solid black rectangles*) are applied across the BA on either side of the graft, and the graft is excised leaving an arteriotomy (*black oval*). **(C)** A patch is sewn in place to close the arteriotomy and the clamps are removed. An external neurolysis of the median nerve is performed and the nerve compression is resolved. *AA*, Axillary artery,

CASE 2

A 49-year-old woman with a history of end-stage renal disease on hemodialysis, diabetes mellitus type 1, and hypertrophic cardiomyopathy presented with left hand weakness and pain after upper arm arteriovenous graft partial removal owing to concern for infection. On examination 1 year after graft removal, the patient had weakness primarily in the median nerve innervated muscles with minimal grip strength. She had moderate weakness of hand intrinsic muscles. The patient had two focal Tinel's signs, one at the proximal and one at the distal end of the incision, which extended from the region of the axilla to the region of the antecubital fossa. She had a palpable radial pulse. An upper extremity duplex ultrasound study showed no arterial insufficiency in the left arm. EMG showed active denervation of the median and ulnar nerve-innervated hand muscles. A small stump of bovine pericardium graft on the brachial artery had been left in place after surgery. Owing to concern that the graft remnants may be compressing the median nerve, surgical exploration was recommended.

Under general endotracheal anesthesia, the vascular surgeon opened the prior incision overlying the biceps muscle. Bovie cautery was used to dissect down to the underlying fascia, and a handheld nerve stimulator was used to identify any nerves within scar tissue. External neurolysis of the median nerve was performed by the neurosurgeon, and a 2-cm firm mass at the proximal end of the incision and a 4-cm bulbous pulsatile mass at the distal end were noted. The approximate location of compression is noted in Fig 2 (case 2). Both masses were overlying the median nerve, which seemed to be normal in appearance and caliber, except for a slightly diminished size at the proximal and distal locations in contact with graft remnants. Scar tissue along the proximal brachial vein at the site of a previous venous graft anastomosis was dissected. Several Prolene sutures were removed. No further compression was noted along the median nerve at this site. At the level of the antecubital fossa, the residual stump of the arteriovenous graft was excised from the brachial artery, and a bovine pericardium patch angioplasty was performed such that the patch was flush with the surface of the artery. After removal of the remnant graft, there was no compression of the median nerve. The incision was closed in a standard fashion. At the 6-month follow-up, her grip strength had normalized and her hand intrinsic strength significantly improved. She continued to experience mild pain and sensory loss. She was able to grip and drink from a cup, which she was not able to do before surgery.

DISCUSSION

In this case series, we aim to raise awareness among vascular and cardiovascular surgeons that remnant grafts can be sources of symptomatic nerve compression. However, subsequent surgery to remove the graft and decompress the neural elements can be done safely and yield significant improvements in neurological function. In both cases, a vascular graft had been partially, rather than completely, removed in an effort to avoid injury to the parent vessel. Mass effect from the residual graft led to compression on the adjacent neural elements, and surgical treatment involved removal of the mass effect by excision of the remnant graft and placement of a vascular patch. To our knowledge, this report is the first of removing residual vascular grafts following ECMO or

dialysis in the setting of persistent neurological deficits associated with subsequent neurological improvement.

In the postoperative period after partial graft removal, the vascular or cardiovascular surgeon can assess for multiple symptoms and signs in the upper extremity that may indicate ongoing neural compression by a residual vascular graft. The median nerve runs closest, with vascular structures in the upper arm, and pain or sensory alteration in the lateral palm and palmar surface of the first three digits is suggestive of median nerve involvement. Grip weakness and the inability to make an OK sign with the thumb and index finger may be present.[9] The ulnar nerve is in close proximity and injury to the nerve may manifest as pain or sensory alteration in the medial palm and fourth and fifth digits. Ulnar nerve dysfunction is associated with an inability to cross the fingers on examination.[10] A Tinel's sign was paramount in both cases in helping to localize the site of nerve compression. This sign is present when percussion over the irritated nerve results in an electric, shock-like sensation traveling distally along the course of the affected nerve. Of note, depending on the relationship of the nerve to the prior skin incision, the Tinel's sign may be directly over or adjacent to the surgical incision used for placement or partial removal of the vascular graft. Tapping over the palpable graft remnant in both patients resulted in reproduction of their presenting pain. If there is more than one Tinel's sign, the locations can be marked preoperatively, and each site should be explored intraoperatively, such as in case 2, where compression was noted at two locations.

In addition to history and examination findings, studies such as EMG/NCS and MRI can help to corroborate the presence of neural involvement and aid in localizing the site of compression. EMG/NCS may confirm the distribution of affected nerves, such as the median and ulnar, without musculocutaneous involvement, suggesting injury at the level of the cords and/or branches. Combined EMG and ultrasound have been shown to help localize the injury to the level of the brachial plexus cords, branches, or peripheral nerve.[11,12,13,14] In case 1, MRI of the brachial plexus showed abnormalities at the level of the infraclavicular brachial plexus. However, because MRI of the brachial plexus is not routinely obtained after vascular procedures, it is unclear if findings represent scar or expected postoperative changes vs pathological compression. In our cases, we found that the abnormal brachial plexus imaging provided further support for the diagnosis, as the findings fit with the overall clinical context. Should a patient develop neurological findings after partial graft removal with symptoms in a distribution of one or more nerves, especially in the presence of a Tinel's sign, referral to a peripheral nerve surgeon is recommended.

The time between symptom onset, referral, and surgical intervention was evaluated. For several reasons, such as sedation or hypoperfusion issues, patients following cardiovascular or vascular procedures may have an impaired level of consciousness after initial surgical intervention, which can complicate early detection of a neurological deficit.[15] A neurological deficit after surgery may or may not improve over time.[16] A neurapraxia, or temporary injury resulting in demyelination without axonal damage, will recover within 3 months typically. However, an axonotmesis type injury may or may not recover spontaneously or in the setting of ongoing neural compression. Therefore, waiting ≥3 months to assess for spontaneous recovery is recommended.[16] These recommendations do not apply to cases of neurotmesis in which a nerve is transected, because spontaneous reinnervation cannot occur and early intervention is indicated.[17] Despite there being >6 months between neurological symptom recognition and nerve decompression in cases 1 and 2 and the severity of symptoms, both patients showed near complete neurological recovery rapidly after surgery for residual graft removal and external neurolysis. These findings suggest that a delay in diagnosis or treatment is not a contraindication to treatment and is not necessarily associated with poor outcome.

Our report has limitations. First, although graft removal and patch placement were performed safely in both cases, it is unclear if injury to the parent vessel may occur in a larger patient series. For revision surgery, the benefits of potential improvements in pain, weakness, and sensory loss were felt to outweigh the risk of vascular injury associated with graft removal. We assert that this determination is best made in a multidisciplinary fashion involving vascular and neurosurgery. To decrease the risk of future neurological deficit after the index surgery involving partial graft removal, the surgeon may similarly weigh risk vs benefit of trimming the residual graft as flush as possible to the parent vessel to minimize mass effect. Second, other conditions may explain symptoms after graft removal surgery, such as cervical radiculopathy, ischemic monomelic neuropathy, complex regional pain syndrome, or peripheral neuropathy; however, the lack of neck pain and hand discoloration or coldness, anatomical distribution of symptoms, presence of Tinel's sign, and supportive studies argued against alternate diagnoses. Last, although neurolysis alone may have resulted in improvement in patient symptoms, owing to ongoing significant mass effect exerted by the graft remnant following neurolysis in each case with resultant distortion of the adjacent neural elements, it was not felt to be sufficient, and graft removal and patch angioplasty were therefore also performed.

CONCLUSIONS

Neural compression by residual vascular grafts after ECMO or hemodialysis procedures can result in neurological deficits that may significantly improve by removal of the remnant graft and subsequent nerve decompression.

The distribution of symptoms and presence of a Tinel's sign suggests nerve involvement. Vascular and cardiothoracic surgeons should have an increased awareness of neurological deficits in the setting of a vascular graft remnant. A multidisciplinary approach can be taken to surgical treatment for safe and effective reversal of neurological symptoms.

DISCLOSURES

None.

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