Chronic Ischemic Monomelic Neuropathy after Arteriovenous Fistula Creation: A Unique Presentation of Vascular Steal.

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INTRODUCTION

We present the case of a 58 year old male with left upper extremity steal syndrome including ischemic monomelic neuropathy (IMN) 1.5 months after arteriovenous fistula creation. Surgical ligation of the distal fistula and creation of new fistula proximally resulted in complete resolution of his vascular steal symptoms almost immediately despite the chronicity prior to surgical presentation.

CASE DESCRIPTION

A 58 year old male presented for left upper extremity steal syndrome after three surgical salvage attempts at an outside institution in the preceding 1.5 months. He sought out a second opinion surgical consultation for reported severe rest pain for 22 days with loss of function and sensation of his ulnar distributed forearm and hand. He reported burning pain 10/10 radiating from left forearm to left shoulder and presented with contracted left 4th and 5th digits. His past medical history included hypertension, end stage renal disease on hemodialysis, hyperlipidemia, cerebrovascular accident with residual left sided weakness, and insulin dependent diabetes mellitus. His past surgical history included a right internal jugular tunneled dialysis catheter and left forearm fistula, subsequent tunneled dialysis catheter removal, distal revascularization interval ligation (DRIL) to left forearm fistula and two other salvage procedures to left forearm fistula, the details of which are unknown to our team. He reported a history of tobacco use, 1 pack per day for 35 years, for which he quit 3 years prior.

On physical exam at his initial consultation, our patient had a left forearm arteriovenous fistula with excellent thrill and bruit, exquisite tenderness to palpation, with intact pressure dressing to dialysis puncture sites. He had motor weakness with complete loss of active range of motion of 4th and 5th digits on the left hand, and decreased passive range of motion due to contracture of these digits. He also had complete loss of sensation of the ulnar distributed hand including 4th and 5th digits. His 1st through 3rd digits had full active range of motion and sensation, and he had 2+ capillary refill in all five digits. There were no scars presents on the medial forearm or ulnar distributed wrist or hand.

Vein mapping of the arm revealed a patent radial artery proximal to and at the anastomosis but occluded distal to the fistula anastomosis with peak velocities 227cm/sec proximal and 318cm/sec at the anastomosis. Venous outflow revealed peak systolic velocities at the anastomosis of 124cm/sec with no outflow stenosis. No deep venous thrombosis of the left internal jugular, subclavian, axillary or brachial veins was observed, and there was no thrombus in the basilic or cephalic veins of the left upper extremity. His venous diameter measurements were as follows:

Cephalic vein: Proximal humerus 4.4mm
Mid-humerus 5.4mm
Antecubital 6.1mm
Proximal forearm 5.7mm
Basilic vein: Proximal humerus 4.9mm
Mid-humerus 4.5mm
Antecubital 3.8mm
Upper forearm 2.8mm
Mid-forearm 2.6mm
Arterial measurements as follows:
Left brachial 191 cm/sec, 4.8mm in diameter
Left radial occluded distally

Based on our mapping and his surgical scarring it appeared the patient had a previous radio-cephalic fistula of the distal forearm prior to surgical salvage attempts. Therefore, he was taken to the operating room for surgical ligation of his fistula at the distal forearm. After the distal fistula was suture ligated and the wound was closed, attention was paid to the antecubital fossa for creation of a new brachiocephalic arteriovenous fistula. After careful lysis of adhesions through previous surgical scars a 7cm segment of the cephalic vein was dissected until freely mobile, and a 6mm opening in the brachial artery was anastomosed to a spatulated cephalic vein using 6-0 prolene suture in a running fashion. Adequate back-bleeding of both vessels was observed prior to completion of the anastomosis. There was evidence of an excellent thrill in the cephalic vein and strong palpable brachial and ulnar artery pulses distal to the anastomosis. Confirmation of this flow was completed using a Doppler probe with excellent bruit auscultated. Postoperatively, there was continued excellent thrill over the cephalic vein which was confirmed by bruit with Doppler ultrasound. A right internal jugular tunneled dialysis catheter was then placed prior to transfer to the post-anesthesia care unit (PACU).

Immediately post-operative in the PACU, the patient noticed full active range of motion of all digits on his left hand and returning sensation of the 4th and 5th digits. On post-operative day 1, his post-operative pain was controlled with as needed medications, he reported full resolution of previously reported burning pain, and had subjective improvements in sensation within the ulnar distribution. He was subsequently discharged after successful dialysis through his tunneled catheter. He was seen three weeks post-operative for staple removal and assessment of his fistula which continued to show excellent thrill and bruit with distal pulses intact. He demonstrated full range of motion of all digits on his left hand and had improved sensation of his ulnar hand with complete return to his baseline function. He was seen again at two months post-operative at which point he was cleared for dialysis through his arteriovenous fistula.

ACKNOWLEDGMENTS & DISCLOSURES

None of the authors listed for this publication have any financial support or conflicts of interest. We have informed consent from our patient for publication of his case in compliance with HIPAA.

DISCUSSION

Distal ischemic steal syndrome, or dialysis access steal syndrome, has been reported in 1-20% of patients with upper extremity access.[1] It can usually be diagnosed clinically, manifesting in a variety of symptoms including coolness, pallor, mild paresthesia, pain during dialysis, pain at rest, paralysis, contracture, ulceration, tissue necrosis, and loss of digits.

The extent of the ischemia can be classified into one of four stages[2]:
Stage I: pallor, cyanosis, or coolness of hand without pain
Stage II: pain during exercise or hemodialysis
Stage III: rest pain
Stage IV: ulcers, necrosis, or gangrene.

Critical ischemia is defined as being Stage III or Stage IV disease. If left untreated, critical ischemia may result in irreversible neurovascular compromise and the loss of fingers or the whole hand. Ischemic steal syndrome is caused by a significant decrease or reversal of blood flow through the arterial segment distal to the usually newly created vascular access because of the pressure differential created by the access site. This may be due to low resistance in the arteriovenous access or due to hypoperfusion secondary to distal arteriopathy.[1]

In addition to retrograde flow, ischemic symptoms may be caused or exacerbated by comorbidities such as diabetes, smoking, and peripheral vascular disease. It is often difficult to distinguish the extent of the role of each of these comorbidities in ischemic symptoms. Our patient had contributing comorbidities including diabetes and a previous history of cigarette smoking. Furthermore, in uremic diabetics with pre-existing neuropathy, steal syndrome can also cause decreased blood flow in the vasa nervorum, causing ischemic monomelic neuropathy (IMN) of the ulnar, radial and median nerves. Patients can develop severe sensorimotor dysfunction of the affected nerves without obvious tissue loss.[3] In such patients, ischemia that is not identified and reversed immediately may result in irreversible neural damage and permanent impairment of the involved extremity.[4,5]

A case report published the same month in which our patient presented represented a similar patient presentation after one month of IMN with slightly less contracture. After ligation of the fistula, the patient showed vast improvement in the median nerve distribution, however showed a slower return to function than the immediate results we experienced in our patient.[6] Another case reported in 2017, claimed IMN within hours of arteriovenous graft creation at the upper arm, subsequently ligated the graft on post-operative day 1 when the symptoms continued to worsen.[7]

CONCLUSIONS

We believe our patient had stage III vascular steal syndrome resulting in severe rest pain and ischemic monomelic neuropathy of his left upper extremity with at least 22 days of neural damage in the ulnar distribution prior to his consultation for second opinion. We immediately operated to surgically ligate the fistula and reverse his vascular steal. Given the current literature, we did not expect any functional return of his left hand or sensation in the ulnar distribution due to the chronic nature of his ischemic monomelic neuropathy, and we hoped only for improvement in his rest pain with ligation of his fistula. But to our surprise, immediately post-operative he demonstrated near complete motor function of his hand and improvement in sensation as early as post-operative day 1 with full return of motor and sensory function on the third post-operative week. Our patient provides a unique presentation of full functional and sensory return after more than three weeks of vascular steal, and should provide an important perspective regarding dialysis access salvage versus patient quality of life. The patients' functional status and pain levels should take precedence over salvage of an arteriovenous access site, and early ligation of the access should be completed prior to chronic IMN development. However, if a patient presents late along the IMN course, we recommend strong consideration of access ligation in order to attempt to regain full neurovascular function of the extremity as we experienced in our patient.

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