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# Ischemic Monomelic Neuropathy: Diagnosis, Pathophysiology, and Management

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## INTRODUCTION

There is an approximate growth of 3% to 4% of patients undergoing dialysis every year as a result of increased pool of chronic kidney disease (CKD) from various medical problems such as diabetes, hypertension, and cardiovascular diseases. Arteriovenous (AV) fistula use increased from 27.9% to 55% between 1998 and 2007 (Figure 1).[1] By December 2013, in 62.5% of prevalent dialysis patients an AV fistula was being used.[2] In 1966, Brescia *et al*. introduced the endogenous AV fistula, a revolutionary therapeutic modality for the management of renal patients. Most common complications of vascular access can be divided into hemodynamic and mechanical complications. Complications from hemodynamic alterations include venous hypertension, arterial steal syndrome, and high-output cardiac failure. Mechanical complications include pseudo-aneurysm, which may develop from a puncture hematoma, degeneration of the wall, or infection. A rare complication that can develop after hemodynamic alteration of vascular access is ischemic monomelic neuropathy (IMN).

## CASE PRESENTATION

A 59-year-old African American man was admitted for creation of vascular access for dialysis. Past medical history included diabetes mellitus complicated by peripheral neuropathy and retinopathy, end-stage renal disease (ESRD) from diabetes and hypertension, a history of dialysis for about 7 years, multiple deep venous thromboses in the upper extremities at AV fistula sites, hypertension, hyperlipidemia, chronic pancreatitis, anemia, and previous smoking (half a pack per day for 20 years).

Physical examination showed a temperature of 98 °F, blood pressure 104/59 mm Hg, pulse 99 beats/min, respiratory rate of 16/min, and 98% saturation on room air. Extremities had bilateral edema 1+. The lower extremity pulses were difficult to determine because of the edema. The rest of the examination findings were normal. Laboratory values on admission were as follows: white blood cells 8.4, hemoglobin 10.4, platelets 274, and hematocrit 32. The basic metabolic panel included sodium 134 mEq, potassium 4.3, chloride 98, bicarbonate 20, blood urea nitrogen 33, and creatinine 4.0 mg/dl. Liver function tests include total protein is 8.8, albumin is 4.5, bilirubin 0.7, alkaline phosphate 269, AST 50, ALT 31. Coagulation tests showed an international normalized ratio (INR) of 1.2, prothrombin time (PT) 12.1 seconds, and activated partial thromboplastin time (aPTT) 45.9 seconds.

### Hospital Course

The patient was started on heparin for bridging anticoagulation, as INR was subtherapeutic. He had a right femoral catheter for regular dialysis. After great saphenous vein mapping was performed, the patient underwent a left superficial femoral artery to vein loop graft without any perioperative complications. On postoperative day 0, patient he complained of pain at the incision site and weakness of left lower extremity. His pain was controlled with acetaminophen/oxycodone, and it was thought that his weakness could be from surgery or anesthetics or from his posture. On postoperative day 1, he complained of worsening weakness and numbness of left lower extremity.

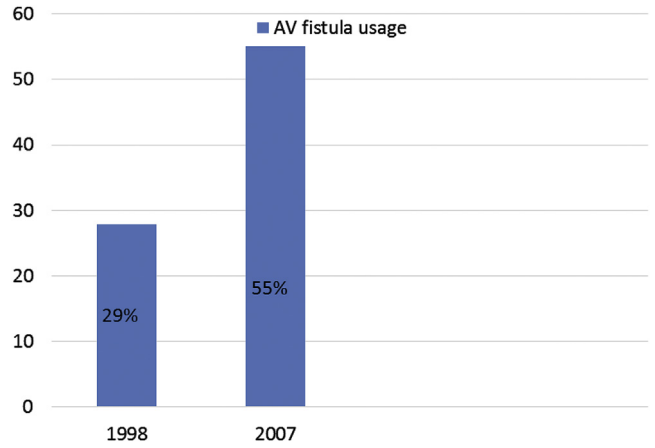


**Figure 1.** Arteriovenous (AV) fistula usage.

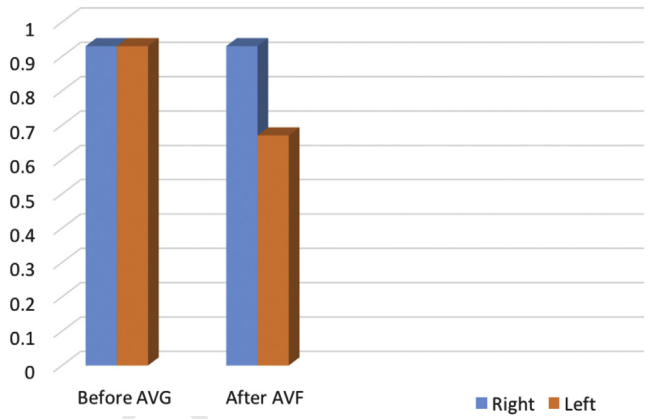


**Figure 2.** Ankle-brachial index (ABI) before and after graft placement. AVF, ■■■; AVG, ■■■. Q6

The neurology department was consulted at that time. On examination the patient had edema at the incision site, and the leg was warm to the touch. Neurological examination showed normal mental status, speech, language, and cranial nerves. The motor system showed normal bilateral upper extremity strength. The lower extremities showed left foot dorsiflexion of 1/5, left foot plantar flexion 3/5, and right dorsiflexion and plantar flexion of foot 5/5. Left hip flexion was 3/5 when the patient was lying on the bed and was 1/5 when he was sitting on the bed. The foot invertors were 0/5 and the foot evertors 0/5. Sensation to light touch, cold, pinprick, and temperature were decreased on the left from the lower third of the leg to the foot and were normal on the right side. Sensation to light touch and cold were normal on the right side. Coordination test results were normal. Pulses could not be determined because of chronic edema.

Ultrasound of the left thigh, computed tomography of the abdomen and pelvis, and magnetic resonance imaging of the spine did not explain the causes of the above-mentioned clinical findings. Ultrasound showed a patent AV graft and no hematoma. The ankle-brachial index (ABI) was 0.93 on the right and 0.67 on the left, and decreased on the left side following graft placement. The ABI before placing the AV graft was 0.93 in both legs (Figure 2). Current analog tracings were consistent with tibial obstructive disease on the left. The right leg was normal. Physical therapy was brought on board, as well as assistance with ambulation, and the patient was found to have a high stepping gait on the left. Vitamin D levels were normal. Foot drop and weakness after surgery without lumbar radiculopathy pointed to ischemic monomelic neuropathy (IMN), which was a diagnosis of exclusion.

After diagnosing IMN clinically, we evaluated the patient's risks and benefits of closing versus not closing the AV graft. He did not have any other site for creating another AV access for dialysis, and closing the AV graft would not completely resolve the problem. We planned to manage the foot drop by physical therapy and ankle–foot arthrosis. The patient's ambulatory status was somewhat impaired due to arthritis and general medical decline for some months prior to the vascular access surgery. He and his family agreed with conservative management and decided not to close AV graft. He was discharged with follow-up neurology consultation for electromyography to evaluate the extent of damage, and also followed up with vascular surgery for assessing the AV graft.

## DISCUSSION

Our patient had neurological symptoms confined to the left lower extremity, weakness, and a foot drop complication. The differentials were IMN, lumbar plexopathy, nerve compression by hematoma or by edema, spinal cord pathology, and iatrogenic nerve injury.

Ultrasound, computed tomography of the abdomen and pelvis, and magnetic resonance imaging of the spine ruled out any of above differential diagnoses. The ABI were done before and after surgery and clearly showed decreased ABI on left after surgery, which was due to a physiological steal phenomenon. The patient did not have vascular ischemic symptoms. The recent vascular access surgery and neurological symptoms of foot drop, numbness, and weakness were suggestive of nerve damage at the level of the mid-thigh or leg or both. As there was no nerve compression, the most likely possibility was IMN.

Ischemic neuropathy of upper limb nerves after dialysis surgery was first reported by Bolton *et al.* in 1979.[3] The term "ischemic monomelic neuropathy" was introduced in 1983 by Wilbourn, a neurologist at the Q2 Cleveland Clinic, and was intended to distinguish isolated ischemia of the arterial supply to multiple nerves of a single extremity.[4] The term refers to the combination of ischemia and neuropathy in a single limb





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