



Ischemic Monomelic Neuropathy Post-AV Access Surgery: A Warm Ischemic Hand Missed!

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Keywords: AV access surgery; Ischemic monomelic neuropathy; Haemodialysis access induced distal ischemia; Distal access steal syndrome.

Abstract

Introduction: Ischemic Monomelic Neuropathy (IMN) is a rare complication after Arteriovenous (AV) fistula creation for hemodialysis. It is marked by motor weakness and sensory changes, often without major hand ischemia. We report a case of IMN after Brachiocephalic Fistula (BCF) creation in an AVF-naïve upper limb.

Case report: This is a case of 55-year-old Indian woman with long standing diabetes, hypertension, dyslipidemia, and chronic kidney disease who suffered from ischemic monomelic neuropathy following left brachiocephalic fistula creation. Diagnosis was established on basis of predominant neurological symptoms without overt signs of tissue ischemia. Following revascularization with BCF ligation, weakness and numbness persisted. Nerve conduction study confirmed axonotmesis of multiple nerves. At 6-month follow-up, recovery was suboptimal.

Discussion: Pathophysiology related to acute-on-chronic reductions in blood flow, where chronic microvascular damage to perineural vessels lowers the ischemic threshold of peripheral nerves, leading to axonal injury. It typically presents soon after AV fistula creation with diffuse motor and sensory deficits involving radial, median, and ulnar nerves. Unlike dialysis access steal syndrome, IMN presents with a warm hand, preserved capillary refill, and palpable distal pulses despite significant neurologic dysfunction.

Conclusion: IMN is a rare but serious nerve complication post-AVF; early diagnosis and intervention are key to preventing permanent disability.

Introduction

Ischemic Monomelic Neuropathy (IMN) is a rare complication associated with the creation of Arteriovenous (AV) fistula for hemodialysis. It is a distinct clinical entity characterized by predominantly neurologic symptoms such as motor weakness and sensory changes, often in the absence of critical ischemia of the hand [1]. This devastating complication of AV fistula can result in irreversible neurological deficit even with timely inter-

vention. We hereby report a case of ischemic monomelic neuropathy following creation of a Brachiocephalic Fistula (BCF) for vascular access on an AVF-naïve upper limb.

Case Report

A 55-year-old Indian woman with underlying diabetes mellitus, hypertension, dyslipidemia, and Chronic Kidney Disease (CKD) approaching End-Stage Renal Failure (ESRF) presented to AVF clinic for assessment for vascular access surgery.



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Vascular mapping was done; left proximal cephalic vein of 2.5 cm and a left brachial artery of 4 cm were good for left brachiocephalic fistula creation. Surgery performed was successful with good venous distension and thrill post-creation, with no immediate complications.

Unfortunately, she experienced rest pain with increasing severity as well as progressive numbness and weakness of the affected hand just 1-day post-surgery. She also noted gradual swelling over her forearm and hands.

However, she presented to us only after one week, hoping that the symptoms would resolve spontaneously. On examination, there were no skin changes in the forearm or wrist. She was found to have a solid left radial pulse, and the left hand was warm to touch with normal capillary refilling time. There was loss of sensation over palmar aspect of the left hand and all fingers. There was no wrist drop. The range of motion of left fingers were reduced with poor fingers flexion and extension. She has motor and sensory deficits in the area of ulnar, median and radial nerve distribution.

A bedside ultrasound demonstrated a mildly calcified left radial and brachial arteries, with normal Doppler signal. The left BCF was found to be patent, measuring 5.07 mm in diameter with a flow of 722 ml/min. The diagnosis of ischemic monomelic neuropathy was made, and the patient proceeded with ligation of the left BCF on the same day.

During subsequent clinic visit, patient no longer experienced rest pain, with palpable radial and brachial pulses. However, she suffered from persistent weakness of left fingers flexion and extension, along with numbness predominantly over palmar aspect of the hand. Nerve Conduction Study (NCS) was performed, revealing a marked reduction in Compound Muscle Action Potential (CMAP) in all 3 distal branches of radial, median and ulnar nerves as well as absence of sensory amplitude further confirming the diagnosis. At 6-month follow-up, her left-hand motor functions and sensory recovery only improved marginally despite complaint to left hand and forearm physiotherapy and rehabilitation.



Figure 1: Well healed left brachiocephalic fistula creation scar.

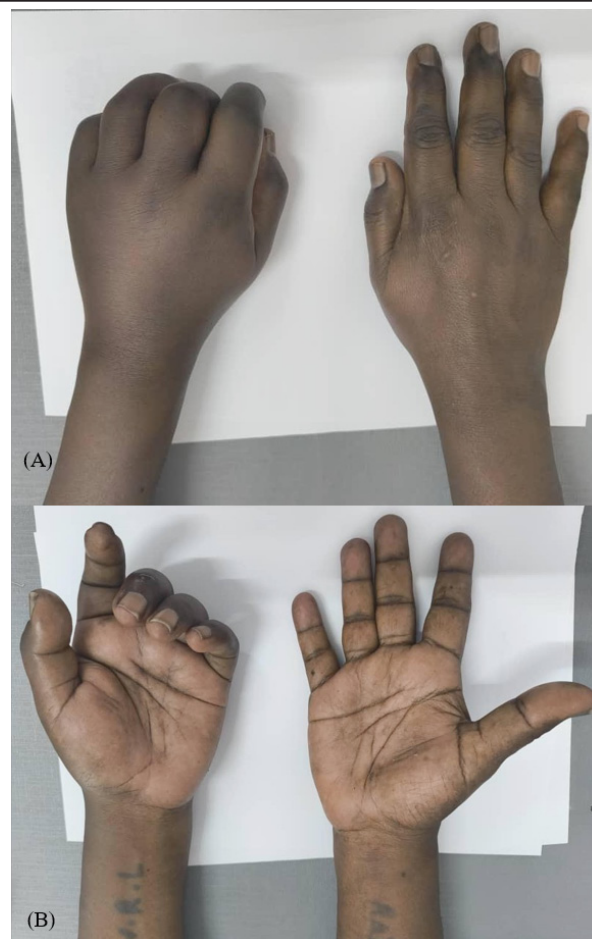


Figure 2: Patient unable to extend the left fingers fully (A) dorsal view, (B) palmar view.

Discussion

Chronic Kidney Disease (CKD) stage five, previously referred to as End-Stage Renal Disease (ESRD), represents a state of irreversible kidney failure, defined by a sustained Glomerular Filtration Rate (GFR) of less than 15 mL/min. In Malaysia, the incidence and prevalence of CKD stage five have been steadily increasing over the past two decades [2].

An Arteriovenous (AV) fistula is considered the gold standard for long-term haemodialysis access, owing to its superior longevity and lower risk of infection compared to other vascular access options. However, the creation of an AV fistula is not without potential complications, which are generally classified into mechanical and hemodynamic categories.

Mechanical complications include pseudoaneurysm formation and vascular wall degeneration, often secondary to repeated puncture trauma or infection. Hemodynamic complications, on the other hand, may involve venous hypertension, arterial steal syndrome, or high-output cardiac failure due to altered blood flow dynamics.

There are two variants of upper limb ischemia after vascular access surgery or Hemodialysis Access-Induced Distal Ischemia (HAIDI). Dialysis Access Steal Syndrome (DASS) is a more common form of HAIDI, as compared to a rarer variant, which known as IMN.

Based on KDIGO 2019, IMN is defined as a distinct entity from AV access steal based on the unique clinical presentation of this disease [3]. IMN is characterized by axonopathy or multifocal axonal loss affecting both motor and sensory branches of multiple distal nerves. It typically arises due to a reduction in ar-

terial blood flow, often as a result of blood being diverted away from the major arteries following AV fistula creation [4]. It is considered rare due to the extensive collateral arterial supply to the vasa nervorum, which typically protects peripheral nerves from ischemia. The pathophysiological mechanism underlying IMN is postulated to result from acute-on-chronic alterations in perfusion. Chronic microvascular damage to the perineural vessels is believed to lower the ischemic threshold of the affected nerves, rendering them more susceptible to ischemic injury [5,6]. As a result, even a modest reduction in blood flow may be sufficient to trigger ischemic neuropathy in these compromised nerves. This condition is most commonly associated with brachial artery-based AV fistulas, as the brachial artery serves as the primary arterial supply to the forearm and hand [7].

Currently, there are no well-established predictive factors for the development of IMN. However, it has been observed to occur more frequently in females and in patients with diabetes, although the exact reasons for such predisposition remain unclear. In the present case, the patient profile aligns with these commonly reported characteristics [8].

Symptom onset in IMN is typically acute, occurring shortly after AV fistula creation. Neurological symptoms often predominate and may occur in the absence of overt tissue ischemia. IMN generally presents as diffuse sensory and motor deficits involving multiple peripheral nerve territories—most commonly the radial, ulnar, and median nerves. Patients usually report pain, paresthesia, and numbness in the affected limb, accompanied by significant motor weakness. Common findings include poor wrist extension, impaired function of the intrinsic hand muscles, and difficulty with thumb opposition. Importantly, vascular findings are usually preserved: the hand remains warm, capillary refill is intact, and distal pulses or Doppler signals are present—features that help distinguish IMN from DASS [7,9]. In our patient, the clinical profile strongly favored a diagnosis of IMN. She had an immediate onset of pain and neurologic symptoms following AV fistula creation, with no associated skin changes or signs of tissue ischemia. The left hand remained warm, capillary refill was preserved, and a strong radial pulse was palpable. In contrast to DASS, which typically presents with diminished or absent radial pulse, delayed capillary refill, cold hands, digital discoloration, and symptoms that improve with manual compression of the AV access. Also, in DASS, ischemia tends to affect skin and muscle tissue more than nerves.

Once the diagnosis of IMN is established, immediate surgical intervention is required to avoid permanent nerve damage and long-term functional impairment. Closure of the AV fistula remains the most widely recommended treatment for IMN [10]. Alternative strategies, such as access banding or Proximalization of Arterial Inflow (PAI), have also been described in selected cases with acceptable outcomes.

Despite numerous alternatives available, the prognosis of IMN remains variable even with prompt intervention; some patients experience complete recovery of neuromuscular function, whereas others may have persistent deficits. For this reason, patients should be counselled pre-operatively about the risk of incomplete neurological recovery even after timely and adequate treatment, and this should be explicitly documented in the informed consent process.

In our case, surgical ligation of the AV fistula was performed. Unfortunately, the patient presented to us beyond the optimal window for revascularisation, resulting in residual neurological deficits despite technically adequate intervention. This highlights the significance of early recognition, high clinical suspicion, and prompt management in IMN to maximise the likelihood of functional recovery and minimise long-term disability.

Conclusion

IMN, though rare, remains a significant complication following AV fistula creation. Clinicians need to maintain a high index of suspicion for IMN, as early recognition and prompt management can be limb-saving and may prevent irreversible disability. Unlike typical ischemic complications, IMN is more commonly associated with nerve injury rather than overt tissue ischemia. Our case underscores the critical importance of timely intervention and early presentation to optimize neurological recovery and minimize morbidity. Furthermore, it contributes valuable insight to the existing literature by adding to the limited pool of reported cases, thereby supporting future reference, improving clinical awareness, and enhancing management strategies for better patient outcomes.

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