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Case Report

Ischemic Monomelic Neuropathy: a Complication of Vascular Access Procedure

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ABSTRACT. Ischemic monomelic neuropathy (IMN) is an infrequently recognized type of ischemic neuropathy produced by shunting blood or acute occlusion of a major proximal artery in the extremities. IMN predominantly occurs in diabetic patients with evidence of peripheral atherosclerotic vascular disease and neuropathy. We report a case of ischemic monomelic neuropathy occurring in a diabetic patient with end-stage renal disease following the placement of polytetrafluoroethylene (PTFE) graft as a vascular access in the proximal upper arm for chronic hemodialysis.

Key words: Hemodialysis, Diabetes, Grafts, Ischemia, Chronic renal failure.

Introduction

To maintain normal structure and function, peripheral nerves need adequate supply of oxygenated blood.[1] Welbourn et al[2] were the first to use the term ischemic monomelic neuropathy to describe multiple distal mono-neuropathies that result in an extremity from shunt placement or proximal embolic occlusion in a major artery.[3] This complication occurs exclusively in diabetic patients, particularly those with peripheral neuropathy and evidence of atherosclerotic peripheral vascular disease.[4]

We report a case of ischemic monomelic neuropathy, which occurred in a patient with end-stage diabetic nephropathy following polytetrafluoroethylene (PTFE) graft placement in the proximal upper arm as a vascular access for chronic hemodialysis.

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Case

A 58-year-old woman with 20-year history of type-I diabetes, 6-year history of ischemic

heart disease and hypertension and 2-year history of chronic renal failure, was admitted with a 10-day complaint of loose watery motions associated with gradually decreasing urine output, nausea, generalized weakness and generalized body swelling. She was on insulin, lisinopril, aspirin and nitrates.

On examination, the blood pressure was 160/100 mm Hg, pulse 90/minute and regular, jugular venous pressure 4.5 cm, and there was pedal edema and coarse bilateral crepitations in the lungs.

The laboratory investigations revealed hemoglobin level 124 gm/L with normal white blood cell and platelet counts. Serum creatinine was 1334μmol/L, serum sodium 137 mmol/L, potassium 4.5 mmol/L, chloride 101 mmol/L, bicarbonate 18.3 mmol/L, calcium 1.96 mmol/L and phosphorus 2.2 mmol/L. Chest x-ray was consistent with pulmonary edema. She was started on hemodialysis via double lumen right internal jugular catheter. Due to poor peripheral vasculature, a left brachio-cephalic arterio-venous bridge PTFE graft was placed as a permanent access for dialysis. A week after discharge, she presented with numbness and burning sensation of the left arm extending from just above the cubital fossa down to the lateral aspect of the hand. These symptoms worsened after the first hemo-dialysis via the PTFE graft.

Examination revealed decreased pinprick sensation along the lateral aspect of the left forearm with absent biceps and triceps reflexes. The patient also had difficulty in gripping objects with the left hand. Electro-physiological studies revealed sensory and motor axonal loss in the median, ulnar and radial nerve distributions distal to the graft. Unfortunately, the patient refused to undergo any further surgical interventions. Six months later, she had worsening of the condition manifested by wasting of the thenar and hypothenar muscles of the left hand associated with burning pain and stinging sensation.

Discussion

Ischemic monomelic neuropathy (IMN) is an infrequently recognized type of ischemic neuropathy produced by shunting blood or acute occlusion of a major proximal artery in the extremities. Most reports about this complication appeared in the neurology literature.

In 1989, Riggs et al[4] described IMN in the upper extremity that resulted from a shunt placement in diabetic patients with end-stage renal disease and evidence of peri-pheral vascular disease and neuropathy.

The pathophysiology of IMN has been attributed to ischemia secondary to the diversion of large amount of blood away from distal forearm and hand following the creation of the arterio-venous shunt in the proximal arm. This results in irreversible ischemic damage leading to an acute axonal loss in multiple peripheral nerves.[2,5,6]

The usual electrophysiologic findings include axonal loss, showing low amplitude or absent responses to the sensory and motor nerve stimulation, relatively preserved conduction velocities, fibrillations and reduced motor unit recruitment of the needle electro myograph (EMG). Kaku et al[7] reported that conduction block could be an early sign of reversible injury in the course of the upper extremity IMN and that the resolution of this sign paralleled the clinical improvement following treatment.

To preserve the neurological function, the early recognition of this complication by neurologists, nephrologists and vascular surgeons is essential. The reduction of the flow in the A-V graft shunt by techniques such as simple ligation or narrowing plication helps to retard of the neuropathy.[8,9]

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