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 of blood or due to acute noncompressive occlusion of the major proximal limb artery. Most reports about this complication appear in the neurology literature. IMN predominantly occurs in diabetic patients with evidence of peripheral neuropathy and atherosclerotic peripheral vascular disease. We report a case of ischemic monomelic neuropathy occurring in a patient with end stage diabetic nephropathy following PTFE (polytetrafluoroethylene) graft placement in proximal upper limb for chronic maintenance hemodialysis.

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 mononeuropathies occurring in an extremity following 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 neurophathy occurring in a patient with end stage diabetic nephropathy following polytetrafluoroethylene (PTFE) graft placement in proximal upper limb for chronic maintenance hemodialysis.

Case Report

A 58 year old female with 20 years history of type-I diabetes, 6 years history of ischemic heart disease and hypertension and known to have chronic renal failure for 2 years, was admitted with loose watery motions, gradually decreasing urine output, nausea, generalized weakness and generalized body swelling for the last ten days. She was on insulin, lisinopril, aspirin and nitrates. On examination her blood pressure was 160/100 mmHg, pulse 90/minute regular, jugular venous pressure 4.5 cm, pedal oedema and coarse bilateral crepitations in the lungs. Her laboratory investigations revealed Hb 12.4 gm/dl with normal white blood cell and platelet counts. Serum creatinine was 11.3 mg/dl, serum sodium 137 mEq/L, potassium 4.5 mEq/L, chloride 101 mEq/L, bicarbonate 18.3 mEq/L, calcium 9.8 mg/dl and phosphorus 4.8 mg/dl. 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 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 first hemodialysis via PTFE graft. Examination revealed decreased pinprick sensation along lateral aspect of left forearm with absent biceps and triceps reflexes. She also had difficulty in gripping objects with the left hand. Electrophysiological studies revealed sensory and motor axonal loss in the median, ulnar and radial nerve territories distal to the graft on left side. There was also evidence of sensory neuropathy. Unfortunately she refused to undergo any further surgical procedure with regards to her acute neuropathy. On follow up six months later she had wasting of thenar and hypothenar muscles of the left hand along with burning pain and tingling sensation.

Discussion

Ischemic monomelic neuropathy (IMN) is an infrequently recognized type of ischemic neuropathy produced by shunting of blood or due to acute noncompressive occlusion of the major proximal limb artery. Most reports about this complication appear in the neurology literature. The initial description was made by Bolton et al[5] in 1979, but it was not until 1983 when Wilbourn et al[2] introduced the term IMN.

However both Bolton and Wilbourn did not mention the etiology of renal failure in their patients. Riggs et al[4] in 1989 described upper extremity IMN occurring after shunt placement in diabetic patients with end stage renal disease and evidence of peripheral vascular disease. Pathophysiology of IMN has been attributed to ischemia secondary to diversion of large amount of blood away from distal forearm and hand following arterio-venous shunt formation in the proximal forearm. This results in irreversible ischemic damage leading to acute axonal loss in multiple peripheral nerves.[2,5,6] Electrophysiologic findings include axonal loss, showing low amplitude or absent responses to sensory and motor nerve stimulation with relatively pre-

served conduction velocities, and fibrillations and reduced motor unit recruitment on needle EMG. David A. Kaku et al reported in 1993 that conduction block occurs early in the course of upper extremity IMN, the resolution of which parallels clinical improvement following treatment.[7]

IMN is a serious complication of vascular access procedures that predominantly occurs in diabetic patients with evidence of peripheral neuropathy and atherosclerotic peripheral vascular disease.

To preserve neurological function an early recognition of this complication by neurologists, nephrologists and vascular surgeons is essential. The literature suggests that reduction of flow and shunting down the graft by techniques such as simple ligation or narrowing plication will help in retardation of the neuropathy.[8,9]

References

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