Acute Fulminant Uremic Neuropathy Following Coronary Angiography Mimicking Guillain Barre Syndrome

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Abstract
A 55 yr old diabetic lady suffered a posterior wall STEMI. She developed Contrast induced nephropathy following coronary angiography. Acute fulminant uremic neuropathy was precipitated which initially mimicked Guillain Barre Syndrome, hence reported.

Introduction
Uremic neuropathy is predominantly a distal symmetrical sensorimotor polyneuropathy, most often affecting the lower limbs. It characteristically progresses over the course of months, but can occasionally take a faster course, wherein, Guillain Barre Syndrome (GBS) and vasculitic neuropathy are its close differentials.

Case Report
A 55 year old lady presented with complaints of chest pain for 6 hours associated with profuse sweating. She had past history of diabetes and hypertension for past 12 years, controlled on drugs. Her general physical examination and cardiovascular system examination was normal except for presence of left ventricular fourth heart sound.12-lead electrocardiography showed posterior wall ST elevation myocardial infarction. 2D echocardiography revealed inferior, inferolateral and anterolateral wall hypokinesia, mild mitral regurgitation and moderate left ventricular systolic dysfunction with left ventricular ejection fraction of 39%. Her laboratory parameters were Hb=11.8gm%, TC= 11000/mm³, random blood sugar-265mg%, blood urea=50 meq/L, serum creatinine=1.3mg% (Cr Cl 54 ml/min), CKMB= 40 IU/L, serum sodium=140meq/L and serum potassium= 4meq/L. She was managed with thrombolytic therapy and guideline directed medical treatment). Coronary angiography showed triple vessel disease. Despite adequate glycemic control and adequate intravenous hydration pre and post coronary angiography, patient developed contrast nephropathy. Her urine output decreased along with rise in serum creatinine to 2.5mg% at 24 hours. Patient was kept under observation and i.v. hydration was continued. But she noted slight weakness over her both lower limbs below the ankle joint. Weakness progressed gradually and 2 days later, she was not able to move her lower limbs at all. Following day she felt paresthesia over her both upper limb associated with weakness. On examination, both the lower limbs were flaccid and power was zero. Deep tendon and plantar reflexes were absent bilaterally in lower limbs. Upper limbs also were hypotonic with power of 2/5 of both distal and proximal muscles. There was vibratory and pressure sensory loss in lower limbs below the knee and patchy sensory loss in upper limb. Higher mental functions were normal and Cranial nerves were intact. There was no involvement of bowel or bladder. Respiratory muscles were not involved. Of note, the patient did neither have any clinical signs of vasculitis such as rash, arthritis, or ocular involvement nor had a history of antecedent respiratory or gastrointestinal tract infection. ESR was 76 mm/hr and CRP was 68 mg/L. Cerebrospinal fluid examination revealed a normal cell count and elevated protein concentration. Nerve conduction studies showed very low amplitude potentials in the nerves of both the lower and upper limbs with prolonged distal latencies. Electromyographic examination showed evidence of acute partial active denervation over proximal and distal muscles examined with no evidence of demyelination. On day 6, her serum creatinine started decreasing with associated improvement of neuropathy. After 15 days, serum creatinine declined to 1.5 mg% and power in both upper and lower limbs improved to 4/5 but patchy sensory loss persisted and patient was discharged from the hospital with a diagnosis of resolving acute uremic neuropathy. The clinical picture though mimicked Guillain Barre Syndrome (GBS), an acute inflammatory demyelinating polyradiculoneuropathy.

Discussion
Peripheral neuropathy is a commonly encountered condition in clinical practice. Though, diabetes mellitus and excessive alcohol use coupled with a poor diet are the most common causes of peripheral neuropathy, onset is insidious and progression is slow in these conditions. The most common cause of acute muscle weakness associated with peripheral neuropathy in adults is Guillain-Barre syndrome.1 The diagnosis of GBS is made by recognizing the pattern of rapidly evolving paralysis with areflexia, absence of fever or other systemic symptoms, and characteristic antecedent events. Rapidly progressing acute peripheral neuropathy in our patient favored GBS but coexistence of preexisting chronic kidney disease (CKD) with superimposed contrast induced neuropathy (CIN) and acute uremia point towards alternative diagnosis of acute uremic neuropathy. Although most commonly uremic polyneuropathy evolves over months, there have been reports of severe fulminant motor neuropathies, sometimes associated with sepsis.2,3 CSF protein levels are usually normal but may be elevated in patients with severe uremic polyneuropathy. The most significant abnormality on electrophysiologic study is a reduction in the amplitude of compound motor and sensory action potential. Both motor and sensory conduction velocities are reduced and late reflexes (H reflex and F wave) become abnormally prolonged.

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more commonly in lower extremities. There is a high correlation between declining creatinine clearance and reduction in conduction velocities. Polyneuropathy is not seen in new onset acute renal failure, but when present systemic vasculitis is the underlying mechanism. Recovery often occurs in two phases, initial rapid improvement over days to weeks and then more protracted improvement over period of months.1

Our patient harboured CKD and suffered acute kidney injury following coronary angiography due to risk factors like diabetes, poor left ventricular function and preexisting CKD. The acute worsening of renal function led to fulminant polyneuropathy which improved in concordance with the renal parameters. Acute polyneuropathy following coronary angiography is rare and clinical picture can mimic GBS, so diagnosis should be made cautiously as the management of the two entities is entirely different.

References