ABSTRACT

A 55-year-old diabetic woman suffered a posterior wall ST-elevation myocardial infarction. She developed contrast-induced nephropathy following coronary angiography. Acute fulminant uremic neuropathy was precipitated which initially mimicked Guillain-Barre Syndrome, hence reported.

**Key words:** Acute uremic neuropathy, contrast-induced nephropathy, Guillain-Barre syndrome

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 PRESENTATION

A 55-year-old woman presented with complaints of chest pain for 6 h associated with profuse sweating. She had a history of diabetes and hypertension for past 12 years, controlled on drugs. Her general physical examination and cardiovascular system examination were normal except for the presence of left ventricular fourth heart sound. 12-lead electrocardiography showed posterior wall ST-elevation myocardial infarction. Two-dimensional echocardiography revealed inferior, inferolateral and anterolateral wall hypokinesia, mild mitral regurgitation, and moderate left ventricular systolic dysfunction with left ventricular ejection fraction of 39%. Laboratory parameters were Hb = 11.8 g%, total leucocyte count = 11,000/mm³, random blood sugar = 265 mg%/DL, blood urea = 50 mEq/L, serum creatinine = 1.3 mg% (Cr Cl 54 ml/min), CKMB = 40 IU/L, serum sodium = 140 mEq/L, and serum potassium = 4 mEq/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 (i.v.) hydration pre- and post-coronary angiography, patient developed contrast nephropathy. Urine output decreased along with a rise in serum creatinine to 2.5 mg%/DL at 24 h.

The patient was kept under observation and i.v. hydration was continued. However, she noted slight weakness over 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. The following day, she felt paresthesia over both upper limbs 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
were 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 neither had any clinical signs of vasculitis such as rash, arthritis, or ocular involvement nor had a history of antecedent respiratory or gastrointestinal tract infection. Erythrocyte sedimentation rate was 76 mm/h and C-reactive protein was 68 mg/L. Cerebrospinal fluid (CSF) 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, 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 GBS, an acute inflammatory demyelinating polyradiculoneuropathy.

DISCUSSION

Peripheral neuropathy is a commonly encountered condition in clinical practice. Although 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 GBS.[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 and acute uremia points toward alternative diagnosis of acute uremic neuropathy. Although most commonly uremic polyneuropathy evolves over months, however, 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, more commonly in the 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.[4]

Our patient harbored CKD and suffered acute kidney injury following coronary angiography due to risk factors such as 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.

CONCLUSION

Acute polyneuropathy following coronary angiography is rare and the clinical picture can mimic GBS, so diagnosis should be made cautiously as the management of the two entities is entirely different.

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Conflicts of interest
There are no conflicts of interest.

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