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Internal Medicine Section

Diabetic Amyotrophy (DA) with Diabetic Neuropathic Cachexia (DNC): An Underrated Combination

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ABSTRACT

Amongst the myriad neuropathic variants of diabetes mellitus, Diabetic Amyotrophy (DA) and neuropathic cachexias are rare complications. DA was first defined in the year 1980 and goes by the name 'Bruns-Garland syndrome and diabetic lumbosacral radiculoplexopathy'. Diabetic Neuropathic Cachexia (DNC) is a rare form of peripheral neuropathy. It is characterised by weight loss, sleep disorders, severe muscle wasting involving the femoral regions bilaterally associated with burning pain and allodynia.

Keywords: Allodynia, Diabetes mellitus, Lumbosacral, Plexopathy

The DA is characterised by an asymmetric, lower extremity-predominant symptom complex that occurs in cases of poorly controlled diabetes mellitus. The symptom complex usually localises to the anterior horn cells and the proximal girdle muscles. The lifelong incidence of DA in middle-aged to elderly patients with diabetes is approximately 1% [1,2].

A 44-year-old male presented with complaints of tingling and numbness in both lower limbs up to his knees for one year and a relatively new onset of severe pain in his left leg associated with weakness for six months. Apart from the bilateral tingling sensations, this unilateral left leg pain was sharp and burning in nature and extended from the left foot to the upper part of the left thigh. It was continuous throughout the day. The pain was aggravated with even a light touch to the leg. The patient was diagnosed case of diabetes for eight years and on irregular treatment with oral anti diabetics (Metformin 500 mg twice and Voglibose 0.2 mg twice before meals. The Visual Analogue Scale (VAS) score of the left leg pain was 8 points. He had progressive difficulty in getting up from a squatting position, getting up from a chair and climbing steps. There was a history of weight loss of 7 kg over the last six months.

On examination, the patient was emaciated. Body Mass Index (BMI) was 17.4 kg/cm2 (48 kg/166 cm). Pulse was 106/min, regular with well-felt peripheral pulses. Jugular Venous Pressure (JVP) was normal and oedema was absent. Cardiovascular, respiratory and per abdominal examination was normal.

Neurologic examination revealed normal higher functions, intact cranial nerves. Motor system examination revealed bilateral quadriceps wasting [Video-1]. Tone was normal. Power in both ankle dorsiflexion and plantar flexion was grade 4+, Knee extensor grade 3, knee flexor grade 4, hip flexors grade 3 and extensor grade 4+ [Video-1] [3].

Bilateral ankle and knee reflexes were lost. There was difficulty in getting up from supine to sitting and getting up from the squatting position [Video-2]. His abdominal muscles were lax and there was visible wasting of intercostal muscles, though fasciculations were not visible or elicitable [Video-3]. Sensory examination was consistent with glove and stocking anaesthesia. Vibration senses were lost up to the ankle.

Investigations revealed Haemoglobin as 11 gram%, Fasting blood sugar- 188 mg%, Post prandial blood sugar- 311 mg%, Glycosylated Haemoglobin (HbA1c): 8.8%. Kidney function test revealed a Urine Albumin to Creatinine Ratio (UACR) of 45 mg/gram of creatinine. Serum urea was 44 mg/dL and serum creatinine was 1.6 mg/dL. The estimated Glomerular Filtration Rate (eGFR) calculated by the Chronic Kidney Disease Epidemiology Collaboration formula (CKD-EPI) was 54 mL/min/1.73 meter square body surface area. Serum vitamin D and B12, Creatine phosphokinase, and thyroid function test results were normal. Nerve conduction study revealed sensory and motor axonal neuropathy. Sensory nerve action potential of the saphenous and sural nerves was absent. The distal latency of the compound action potential of the bilateral femoral nerves was prolonged (7.33-8.42 msec). Positive sharp wave and fibrillation potentials of bilateral iliopsoas, adductor magnus, and rectus femoris muscles were detected during needle Electromyography (EMG) examinations. These electrophysiologic findings were suggestive of diabetic lumbosacral plexopathy and distal sensory polyneuropathy [4]. Lumbar Magnetic Resonance Imaging (MRI) spine revealed mild discal protrusion at L3-L4, L4-L5 levels in the midline.

Patient was started on Insulin Glargine 8 units once at 8 pm and insulin Actrapid six units thrice before meals and Tab. Metformin 500 mg twice a day and the sugar levels were controlled. For painful neuropathy, Tab. gabapentin 300 mg TID, alpha lipoic acid 600 mg and 25 mg amitriptyline were initiated. At the end of four weeks, his pain diminished in intensity, and VAS scores reduced from eight to six points. However, the neurologic examination did not improve.

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