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Case Report

Acute onset monoparesis in newly diagnosed patients with type 2 diabetes mellitus with poor glycemia

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ABSTRACT

In newly diagnosed patients with diabetes with poor glycemia, presentation is sometimes acute onset monoparesis, which eventually improved with good glycemic control. To report an unusual patient with newly diagnosed diabetes whose initial manifestation was acute onset monoparesis. A 58-year old female patient with new onset type 2 diabetes mellitus with diabetic ketoacidosis (DKA) who presented to us with acute onset monoparesis (right lower limb) lower motor neuron (LMN) type without bladder involvement, secondary to poor glycemia which eventually resolved with good glycemic control.

Keywords: Diabetes, Monoparesis, Poor glycemia

INTRODUCTION

Diabetic neuropathy, which occurs in about 50% of individuals with long standing type 1 and type 2 diabetes mellitus, manifests as a diffuse neuropathy (distal symmetrical polyneuropathy and/or autonomic neuropathy), a mononeuropathy or radiculopathy/ polyradiculopathy. As with other complications of diabetes mellitus (DM), the development of neuropathy correlates with the duration of diabetes and glycemic control. Additional risk factors include high body mass index; smoking; associated cardiovascular disease, elevated triglycerides and hypertension. Both myelinated and unmyelinated nerve fibers are lost. Raff et al.[1] studied the association of ischemic mononeuropathy with diabetes mellitus. Mononeuropathy is less common than polyneuropathy in DM and presents with pain and motor weakness in the distribution of a single nerve. Kelkar suggested that mononeuropathy in diabetes may be caused by an immune mediated vasculopathy and sometimes the presentation can be sub-acute.[2] In this case report, we describe a patient with new onset Type 2 DM with DKA who presented to us with acute onset monoparesis (right lower limb) LMN type without bladder involvement, secondary to poor glycemia.

CASE REPORT

A 58-year old female presented to the medicine OPD with history of fever 15 days back, recorded maximum 101°F, lasted for a few days with complaints of low backache, weakness and numbness in right lower limb for last 10 days. Weakness had rapid onset with progression to maximum severity and patient was not able to bear weight. Patient also complained of polyuria and polydipsia for 10 days and constipation for last 5 days. The patient consulted a local practitioner who advised MRI spine which came out to be normal. Patient got her blood sugar checked which came out to be high. She was referred to Acharya Shri Chander College of Medical Sciences, Sidhra, Jammu for the same (blood sugar-682mg/dl and HbA1C- 12.2%).

On clinical examination, she had normal vitals. Chest and cardiovascular system findings were non-contributory. Abdomen was distended, bowel sounds sluggish. Neurological examination, Glasgow Coma Scale -15/15, higher mental function was normal, normal bulk in all four limbs, tone decreased in right lower limb, bilateral plantar down going. Power in right (upper limb- Grade 5, lower limb- grade 2) and left (upper limb- grade 5, lower limb- grade 5). Deep tendon reflexes of right side (biceps: 2+, triceps: 2+, knee: absent, ankle: +, supinator: +) and left side (biceps: 2+, triceps:2+, knee: +, ankle: +, supinator: +). Proprioception was normal. Sensation present bilaterally both lower limbs, slight decreased in right lower limb (impaired vibration).

Results of laboratory investigations were normal complete blood count, random blood sugar- 588mg/dl (high), HbA1c-12.5% (high), Serum(S) Sodium- 129 mEq/L, S. potassium- 4.5 mEq/L, S. calcium- 9.4 mg/dl, S. urea- 98 mg/dl, S. creatinine- 1.4 mg/dl, arterial blood gas analysis revealing- ph: 7.32, PCO_2:30 mmHg, PO_2:71 mmHg, HCO_3: 16 mmol/L indicative of metabolic acidosis. Urine routine- albumin traces, glucose: +++, ketones: large (indicative of ketonuria), Urine for microalbumin- 16 (negative), Ophthalmological examination demonstrated no evidence of retinopathy, viral markers non-reactive, X-ray Chest and abdomen- Normal, ECG- normal sinus rhythm, ECHO- normal sized cardiac chambers with LV diastolic dysfunction with ejection fraction- 65%, USG- cholelithiasis, grade 1 fatty liver, MRI Brain and spine was normal with no signs of myelopathy or compression of nerve roots. CRP-negative, ANA- 0.82 (negative), and P-ANCA-2.53 (negative) to rule out any vasculitic neuropathy, NCV report was normal.

Patient was managed with insulin infusion and intravenous fluids. Over 2-3 days when glycemia improved weakness started improving, and eventually patient started walking within 3 days. Blood sugar at the time of discharge was 130 mg/dl and patient went walking home with grade 5 power in all limbs.

DISCUSSION

The clinical presentations of diabetic neuropathy are more protean and varied. Multiple pathogenetic factors include metabolic factors like high blood glucose, advanced glycation end products (AGE), sorbitol, and abnormal lipid levels along with ischemia, insulin imbalance and nerve fiber degeneration. Hyperglycemia induces increase in sorbitol levels, decrease in nitric oxide and glutathione levels, increase in free radicals due to AGE, oxidative stress, generation of cytokines leading to increased inflammation and decrease in growth factor levels, all contributing to ischemic reperfusion injury, microangiopathy and peripheral neuropathy. Polyol pathway leads to osmotic damage to nerve cells, reduction in myoinositol and increased production of superoxide, hydrogen peroxide and hydroxyl radical. Higher intracellular glucose leads to increased production of triose phosphates leading to activation of protein kinase C via diacyl glycerol which damages the capillaries, its permeability and contractibility and destroys the nerve function. There is impaired fatty acid metabolism (dihomogammalinolenic and arachidonic acid) in nerves; these are required for normal nerve structure and conduction. Mononeuropathy in diabetes could present in form of peripheral mononeuropathy where there is single nerve damage due to compression or ischemia; occurring in wrist in form of carpal tunnel syndrome or in elbow or foot in form of unilateral foot drop; the other presentations being cranial mononeuropathy where cranial nerves are involved associations being unilateral pain, paralysis of eye muscle along with double vision or mononeuritis multiplex. Popovic et al presented a case of 74 years old man who was admitted to emergency department of military medical academy complaining of moderate pain and weakness of right arm, neurological examination showing weakness in several muscle groups in right arm, most notable right-hand grip weakness.[3] Laboratory findings were glucose level of 15 mmol/L, HbA1C of 9.3%, with elevated triglycerides. Electromyography showed plexopathy in C6, C7 and C8 levels on right side, MRI cervical spine was normal with no signs of myelopathy or compression of nerve roots. Patient was managed with metformin and alpha-lipoic acid and within three days neurological deficit was significantly reduced. In our study we described a quite similar patient with new onset type 2 diabetes with DKA with acute onset monoparesis (isolated right lower limb weakness) LMN type without bladder involvement, secondary to poor glycemia and which resolved with proper management of poor glycemia.

Pica et al also reported a similar case with bilateral branchial plexopathy as an initial presentation in a newly-diagnosed, uncontrolled diabetes mellitus.[4] Brachial plexopathy has seldom been associated with diabetes mellitus and could present as a rare subtype of the diabetic neuropathies. Santillan et al proposed a case study of 39-year old patient who presented with severe, bilateral and asymmetrical, axon-loss branchial plexopathies occurring in the midst of diabetic ketoacidosis.[5] Massie et al studied the occurrence of isolated diabetic cervical radiculoplexus neuropathy and found its clinical features and pathological alterations similar to diabetic lumbosacral radiculoplexus neuropathy due to ischemic injury and microvasculitis.[6] Sander et al proposed that diabetic amyotrophy is a disabling illness that is distinct from other forms of diabetic neuropathy, and characterized by weakness followed by wasting of pelvifemoral muscles, either unilaterally or bilaterally, with associated pain.[7] Rydberg, et al did a longitudinal study to explore potential associations between diabetes mellitus and entrapment neuropathies during long term follow up and found hyperglycemia to affect the median and ulnar nerves differently.[8] Kocer et al examined different methods of

studying sural nerve conduction in a group of patients with impaired glucose tolerance supporting the idea that impaired glucose tolerance is a transitional state before diabetes and also the importance of dorsal sural nerve latencies for early detection of neuropathy.[9] Ogawa et al reported a case of a 48-year old man with a 14-year history of type 2 diabetes with proliferative diabetic retinopathy and distal symmetrical diabetic polyneuropathy and within a span of 8 months, he sub acutely developed difficulty in both shoulder movement and trouble standing up from a squatting position with severe bilateral shoulder and thigh pain.[10] His unbearable symptoms were partially alleviated by administration of selective serotonin reuptake inhibitor, fluvoxamine maleate. All these studies were more or less in accordance with our case study.

Regarding our case report, patient was newly diagnosed diabetes with DKA, presentation being acute onset monoparesis (right lower limb) with normal bulk, hypotonia, grade 2 power (moving right lower limb with gravity eliminated), preserved reflexes excluding right knee reflex, plantar down going and slightly decreased sensation (vibration impaired) in right lower limb. Neuroimaging of brain and spine being normal and all causes of vasculitis weakness being ruled out. We managed the patient with intravenous fluids and insulin infusion. Progressively when glycemia started to control, monoparesis started resolving, power improved to grade 3 on first day, then to grade 4 on 2nd day and finally patient started walking on 3rd day when patient had fully controlled glycemia levels. The possible etiology being a rare acute presentation in diabetes with poor glycemia pathogenic mechanism being oxidative stress, free radical and cytokine production, activation of protein C and osmotic damage to nerve cells due to hyperglycemia.

CONCLUSION

This case report highlights the importance of considering less common presentation such as acute onset monoparesis in newly diagnosed type 2 diabetes mellitus patients with poor glycemia with clearly excluding the other possible causes of isolated monoparesis. Diagnosing and treating the etiology by strict control of lood sugar can alleviate the weakness and prevent morbidity in patients with type 2 diabetes mellitus.

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