CASE REPORT

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Diabetic Striatopathy as a Presenting Symptom in Newly Diagnosed Type 2 Diabetes Mellitus: A Case Report

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Abstract

Diabetic striatopathy is a rare, acute neurological complication of diabetes mellitus which presents with non-ketotic hyperglycemia and involuntary movements, specifically hemichorea or hemiballismus. Striatal abnormalities on neuroimaging have been reported in most, but not all cases. This case report aims to increase awareness that diabetic striatopathy can be a presenting symptom of diabetes mellitus.

A 92-year-old Filipino female with no history of diabetes presented with acute onset of focal clonic flexion of the left upper extremity for a few hours, which progressed to right hemifacial spasm. She was diagnosed with a hyperosmolar hyperglycemic state and was treated accordingly. Cranial CT scan findings were unremarkable. There was an immediate resolution of her neurologic symptoms after the correction of hyperglycemia.

The diagnosis of diabetic striatopathy highlights the importance of increasing awareness and understanding of this condition among clinicians to prevent delayed diagnosis and treatment. Screening for hyperglycemia is advisable for patients with involuntary movements. The prognosis for diabetic striatopathy is good with prompt glycemic control in most cases.

Key words: hyperkinetic movement disorder, nonketotic hyperglycemia, involuntary movement, neurologic complication

INTRODUCTION

The central and peripheral nervous system can be affected by diabetes mellitus. There are different associated neurologic complications such as neuropathy, coma and seizures. Abnormal movements, which are found in increased frequency among persons with diabetes, can occur as the initial presentation of diabetes in those with poor glycemic control. The exact pathophysiology is still unknown.[1]

Diabetic striatopathy (DS) is a rare acute hyperkinetic movement disorder due to nonketotic hyperglycemia. The reported prevalence rate of DS is 1 in 100,000. Despite the very dramatic and obvious clonic flexion of the extremity, the rarity of the disorder and the unfamiliarity of physicians with this condition hinders the recognition of the symptom as DS, leading to misdiagnosis.[2] Diabetic striatopathy usually occurs in elderly Asian women with type 2 diabetes mellitus with poor glycemic control. It is rarely seen in type 1 diabetes mellitus.[3]

CASE PRESENTATION

A 92-year-old Filipino nun from Manila was admitted due to involuntary movements of the left upper extremity and right hemifacial spasm. These symptoms, noted as myoclonic movements of the left upper extremity with hemifacial spasm described as twitching of the right side of the face started acutely 12 hours prior to admission while she was sleeping. There was no loss of consciousness and the patient was able to recall the events.

Hypertension was noted in the past medical history. There was no known history of diabetes. Six months ago, she was diagnosed with allergic dermatitis and was given oral steroids with tapering doses. During physical examination at the emergency room, an episode of myoclonic movements of a few seconds involving the left upper extremity was observed. Amidst such episode, she was coherent and oriented to time, place and person with intact recent and remote memory. Manual muscle testing of all extremities revealed a full range of motion against gravity

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with minimal resistance. No sensory deficit was observed on all extremities. All deep tendon reflexes were +2. Vital signs were normal. Weight was 43 kilograms. No signs of dehydration were seen.

At the time of the occurrence of the involuntary movements, capillary blood glucose taken using a glucometer was noted to be high. Confirmatory venous blood glucose testing performed in the laboratory yielded a value of 671 mg/dL. Laboratory results were as follows: HbA1c of 13.40%, serum ketones less than 0.5 mmol/L, serum osmolality of 328 mOsm/kg and serum WBC of 16,000/µL. Urinalysis revealed: glucose +3, negative for ketones, positive nitrite, WBC of 43/hpf and bacteria of 69/hpf. The blood pH was 7.489 and arterial bicarbonate was 24.4 mmol/L. Creatinine was 103.6 umol/L with estimated glomerular filtration rate was 43 ml/min/1.73 m^2. The patient was managed as a case of hyperglycemic hyperosmolar state initially with hydration with intravenous fluid, and administration of intravenous insulin glulisine. Insulin glargine 20 units subcutaneously once daily was eventually started. Ceftriaxone was started for the urinary tract infection. Bedside electroencephalogram was unremarkable. Plain cranial CT scan revealed no evidence of acute hemorrhage, acute or chronic infarct or focal mass lesion. The basal ganglia were physiologically calcified bilaterally. MRI was not performed due to lack of consent.

The patient was discharged with improved glycemic control and with no recurrence of hemiballismus nor hemifacial spasm. Fixed doses of insulin glulisine pre meals and linagliptin 5 mg tablet once daily were given as home medications.

DISCUSSION

Hyperglycemia can present with abnormal movements. The movements tend to acutely develop on one side of the body and usually disappear when the patient sleeps. Other neurologic deficits include weakness, pyramidal tract signs and hypotonia.[1]

Diabetic striatopathy is an uncommon complication of type 2 diabetes mellitus which manifests as involuntary movements such as hemichorea or hemiballism. The associated risk factors were elderly Asian females with type 2 diabetes mellitus with poor glycemic control. The mean age of patients affected according to a meta-analysis was 71 years of age. Signs of diabetic striatopathy include non-ketotic hyperglycemia, unilateral limb dyskinesia and striatal abnormalities on neuroimaging.[3] Unilateral chorea is commonly seen in patients with hyperglycemia compared to bilateral chorea. Not all patients with diabetic striatopathy manifest with involuntary movements, some have impaired consciousness instead.[4] Neuroimaging tests typically reveal a contralateral basal ganglia lesion. Magnetic resonance imaging is more sensitive in detecting these lesions with a sensitivity of 95% while CT scan has a lower sensitivity of 78%.[5] Common neuroradiologic findings in diabetic striatopathy are striatal hyperdensity on CT scan and striatal hyperintensity on T1-weighted MR imaging.[6] However, it is also not uncommon for DS cases to have an unremarkable neuroimaging.[7,8] In a study by Dubey et al which included 59 persons with diabetes with acute dyskinesia, majority of the patients (55.9%) did not have any abnormal striatal findings in neuroimaging tests.[7]

The pathophysiology of diabetic striatopathy is still unclear but mainly involves different mechanisms in the basal ganglia, an important structure involved in the production of movement in the body. Two proposed theories are the microvascular disease theory and infection or autoimmune inflammatory response theory.[3] In the microvascular disease theory, uncontrolled hyperglycemia causes patchy necrosis and edema, lymphocyte infiltration, reactive astrocytosis, with macrophage and erythrocyte extravasation in basal ganglia.[9] The second theory implicates the role of infection and autoimmune inflammatory response in basal ganglia dysfunction. Some studies have shown that in DS cases, there was lymphocytic infiltration around the blood vessels of the basal ganglia.[10]

Another theory explores the imbalance in the biochemical substrates in the brain. In non-ketotic hyperglycemia, the brain metabolism shifts to an anaerobic pathway which utilizes and depletes GABA, consequently decreasing acetylcholine levels. This decrease in GABA and acetylcholine, which are normal substrates in basal ganglia circuitry, causes basal ganglia dysfunction leading to involuntary movements.[11]

Treatment is mainly control of hyperglycemia. It has a good prognosis, with resolution of involuntary movements once there is adequate glycemic control. Only 13% of patients have been reported to have recurrence of involuntary movements associated with hyperglycemia.[12]

VIDEO OF THE CASE

CONCLUSION

There is an increasing number of patients with diabetes diagnosed incidentally because of the onset of complications. As clinicians, we should be aware of the rare neurologic complications of uncontrolled diabetes mellitus to be able

to properly diagnose and treat the patient. In our case, the patient had no previous history of diabetes mellitus and was incidentally diagnosed because of her admission for involuntary movements. Diabetic striatopathy has a good prognosis hence proper diagnosis can lead to resolution of involuntary movements upon attaining blood sugar control.

Ethical Consideration
Patient consent was obtained before submission of the manuscript.

Statement of Authorship
All authors certified fulfillment of ICMJE authorship criteria.

CRediT Author Statement
PDCS: Conceptualization, Methodology, Investigation, Resources, Data Curation, Writing – original draft preparation, Visualization; **TFR:** Conceptualization, Investigation, Resources, Data Curation, Writing – review and editing; **JMIC:** Conceptualization, Investigation, Resources, Data Curation, Writing – review and editing, Supervision, Project administration.

Data Availability Statement
No datasets were generated or analyzed for this study.

Author Disclosure
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