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Original Research Paper

Radiodiagnosis

DIABETIC STRIATOPATHY: A RARE PRESENTATION OF NONKETOTIC HYPERGLYCEMIA

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ABSTRACT Type II Diabetes Mellitus is one of the most common non-communicable diseases with innumerable & potentially life threatening complications. In 2017, approximately 462 million individuals were affected by type 2 diabetes corresponding to 6.28% of the world's population (4.4% of those aged 15-49 years, 15% of those aged 50-69, and 22% of those aged 70+), or a prevalence rate of 6059 cases per 100,000. Over 1 million deaths per year can be attributed to diabetes alone, making it the ninth leading cause of mortality. The burden of diabetes mellitus is rising globally, and at a much faster rate in developed regions, such as Western Europe. The gender distribution is equal, and the incidence peaks at around 55 years of age. Global prevalence of type 2 diabetes is projected to increase to 7079 individuals per 100,000 by 2030, reflecting a continued rise across all regions of the world.[1] . Two of the common complications due to acute hyperglycaemia are Diabetic Ketocidosis & Non ketotic hyperosmolar coma which are considered a spectrum of the same complication due to low circulating levels of insulin leading to impaired glucose metabolism by insulin dependant tissues with rising levels of anti-insulin hormones like glucagon, cortisol & catecholamines due to intracellular starvation resulting in hypergylcemia & fatty acid breakdown & ketonemia.

Amongst the numerous complications of Type II Diabetes Mellitus, here we present a rare complication of acute hyperglycaemia and its radiological picture in the central nervous system. A 56 year old female patient with a history of Type II Diabetes Mellitus with Hypertension under long term medication came for a private consultation with a complaint of Right sided involuntary, random, irregular, flinging and flailing, rapid, non-patterned movements for past 7 days. The patient was advised for an urgent MRI of Brain which demonstrated high T1 signal & low T2/FLAIR intensity with no diffusion restriction of DWI & ADC map in left sided putamen & head of caudate nucleus. We illustrated a rare classical finding of acute hyperglycemic effect on brain in a case of long standing Type II Diabetes Mellitus despite being on medications.

KEYWORDS : Type II Diabetes Mellitus, Non-ketotic hyperosmolar coma

INTRODUCTION

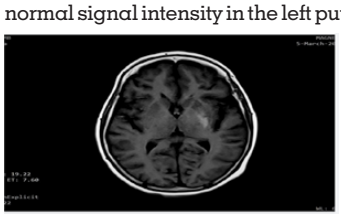
Type II Diabetes Mellitus is one of the most common non-communicable diseases in the world affecting both sexes.. Hyperosmolar hylerglycemic syndrome is a clinical condition that arise from a complication of diabetes mellitis. Hemichorea-hemiballismus is an unusual hyperkinetic movement disorder characterised by continuous involuntary movement of an entire limb or both limbs on one side of body. These movement are mostly irregular , variable in amplitude, and with no recognised pattern[2]. Bedwell first described hemichorea-hemiballismus associated with hyperglycemia that resolved as the blood glucose level were normalized. NHH is most frequently reported in elderly patients, who have type 2 diabetes. The majority of case published in the literature involve elderly female patients especially in asian subjects(1-4). The acute onset of hemiballismus-hemichorea is caused by focal lesion in contralateral basal ganglia, particularly the striatum.

CASE REPORT

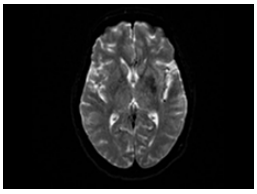
A 56 year old female patient was sent to radiology department with complaint of seven day history of involuntary movement of right upper and lower extremity. The movements increased with action , decreased with relaxation and disappeared during sleep . The patient's medical history included diabetes mellitus type 2 and hypertension. No history of parkinsonism or other neurological disorder was reported. Furthermore, the patient had no prior history of dopamine antagonist or estrogen medication use. A physical examination revealed transient weakness of right limbs. Therewas no evidence of sensory impairment, and cranial nerves were normal. The deep tendon reflexes were symmetrically hyperactive. Her skin was pink , warm and dry. Laboratory test revealed poorly controlled diabetes mellitus with random blood sugar 320mg/dl and hbA1c-8.1%.The urine examination was negative for ketones. The patient underwent MRI of the brain was performed 7 days later. After few days involuntary movement progressively decreased over the next few week . Ischemic or hemorrhagic stroke stroke represents the most common cause of disease[2-4] ; nonketotic hyperglycemia is the next most common cause. The pathophysiology of hemiballism-hemichorea triggered by hyperglycemia is controversial and poorly understood. In a few histopathologic studies, researchers have found gliosis, gemistocyte accumulation, and selective loss of neurons, without evidence of hemor-rhage or infarction[6-8]

Mr findings suggest that petechial hemorrhage cannot be responsible for the lesions since if the striatal T1 hyperintensity represents methemoglobin (at the subacute stage of hemorrhage), T2 hyperintensity should also be present; this was not the case in this patient or in most previously reported cases (7). Other authors proposed that the high-signal-intensity lesions on T1-weighted MR images could be related to manganese accumulation in gemistocytes, which would result from an increase in the activity of manganese superoxide dismutase[9-10]. This enzyme induction in gemistocytes (reactive astrocytes containing a rich protein content that usually appear during acute injury but later gradually shrink) can be triggered by multiple stimuli, including brain ischemia and hyperglycemia (9,10). Hence, the authors assumed that the acute putaminal dysfunction secondary to hyperglycemic or hyperosmolar insult could be associated with some degree of Wallerian degeneration of the internal white matter of the putamen with gemistocyte accumulation. Protein desiccation occurring in the course of Wallerian degeneration occiring in the course of Wallerian degeneration could explain the T1 - weighted hyperintensity in MR.

IMAGING FINDINGS- MR imgaing obtained 8 days after onset of hemiballismus hemichorea revealed strong hyperintense signal T1, hypointense in T2(FLAIR) noted in left putamen and head of caudate nucleus. Diffusion weighted images revealed normal signal intensity in the left putamen.



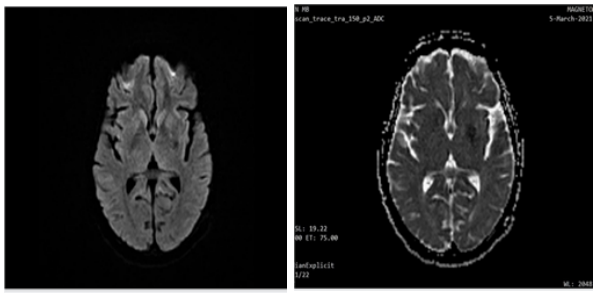
1.



2.

1.T1 weighted MR revealed hyperintense signal in left putamen and head of caudate nucleus

2.T2 weighted MR revealed hypointense signal in left putamen



3. DWI and ADC revealed normal signal intensity without obvious diffusion restriction

DIFFERENTIAL DIAGNOSIS-

The differential consideration for nonketotic hyperglycemia induced hemiballism-hemichorea include -

1. Hemorrhagic stroke /ischemic stroke
2. Complications of human immunodeficiency virus infection
3. vasculitis,
4. Central nervous system lupus,
5. Mass lesions
6. Multiple sclerosis
5. Thyrotoxicosis,
6. Pharmacologic therapy (ie, anticonvulsants, levodopa, oral contraceptives, and neuroleptics) (3).

The typical T1-weighted MR imaging characteristic of hyperglycemic hemiballismhemichorea is not specific to this condition and can be observed in other toxic, metabolic, or degenerative disorders. The most frequent conditions with bilateral T1 weighted hyperinteansity are chronic hepatic encephalopathy (5), followed by manganese toxicity during longterm parenteral nutrition (11), post–cardiac arrest encephalopathy, hypoglycemic coma, hypothyroidism, mild focal ischemia or chronic changes due to hypoxia, neurofibromatosis, Fahr disease or abnormal calcium metabolism, Wilson disease, and carbon monoxide poisoning (8). All these conditions usually involve basal ganglia on both sides of the brain converse to hyperglycemic hemiballism-hemicorea; however, a few cases of bilateral increased T1 signal intensity within the striatum have been reported in patients with hemiballism-hemichorea associated with hyperglycemia (7). In patients with hepatocerebral disease and manganese toxicity, the changes are seen mostly in the globus pallidus [8]

CONCLUSION

This patient, the characteristic MR findings and the medical history, including laboratory values, make all these diagnoses unlikely.Moreover, patients with hemiballismhemichorea not related to hyperglycemia have a different presentation compared with this patient, and these conditions largely can be excluded on the basis of patient history and laboratory findings.

Furthermore, In patients developing hemiballismus -hemichorea secondary to nonketotic hyperglycemia , the clinical course is usually favorable and symptoms tend to resolve spontaneously with normalization of hyperglycemia within hours days, weeks(2, 5).

Therefore in such poorly controlled diabetic patients with hemichorea hemiballismus the patients may present as acute manifestation of diabetic striatopathy with such radiological findings. Normalization of glucose is the primary therapy along with long term diabetic control(5)

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