IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 21, Issue 10 Ser.5 (October. 2022), PP 08-11
www.iosrjournals.org

An interesting case of Hemichorea-hemiballism

Dr. Siddhant Singh Chandel[1], Dr. Nandakumar[2]
[1](Post graduate Resident, Department of Radio-diagnosis, Dr. B. R. Ambedkar Medical College and Hospital/ Rajiv Gandhi University of Health Science, India)
[2](Professor and Head, Department of Radio-diagnosis, Dr. B. R. Ambedkar Medical College and Hospital/ Rajiv Gandhi University of Health Science, India)

Abstract
Characterized by acute or subacuteneuroclinical triad of hemichorea-hemiballism, hyperglycaemic state and unique abnormalities limited to the striatum on neuroimaging nonketotic hyperglycemia chorea-balismus is a rare metabolic syndrome secondary to hyperglycemic condition. It is a complication of poorly controlled diabetes mellitus and clinically mimics a cerebrovascular event which requires prompt action. Imaging helps in identification and diagnosis of this rare condition which sometimes can be confusing clinically. We report this case of nonketotic hyperglycemic chorea-ballismus in a 52 years old male with history of diabetes mellitus and typical radiological findings to familiarize clinicians to this unusual disorder which can mimic otherclinical conditions.

Keywords: *Nonketotic hyperglycemia, T1 hyperintense basal ganglia lesion, hemichorea-hemiballismus*

Date of Submission: 03-10-2022 Date of Acceptance: 17-10-2022

I. Introduction

Hemichorea/hemiballism is a neurological syndrome often reported in elderly patients with diabetes mellitus and is characterized by brief abrupt irregular involuntary movements on one or both side of the body. These movements mainly involve the upper extremity. This syndrome is prevelant in asian population and has female predominance and is associated with vascular insult near subthalamic nucleus[1]. Various predisposing factors include neurodegenerative diseases, cerebrovascular disease, immunological disorders, neoplastic diseases, infections and metabolic syndromes [2]. Majority of the patients recover after correction of blood sugar levels. The exact pathophysiology behind the imaging findings isnot very well known but some posulated mechanisms include hyperviscosity secondary to hyperglycaemia, leading to regional blood-brain barrier disruption and metabolic damage, decreased gamma-aminobutyric acid (GABA) availability in the striatum secondary to the non-ketotic state[3]. We report this case of acute onset hemichorea-hemoballismus in a male patient with poorly controlled type II diabetes mellitus.

II. Case Presentation

A 51 year-old male, known case of pulmonary tuberculosis on anti-tubercular therapy since 12 months presented to the emergency department with 20 days history of acute involuntary random movements of left upper and lower limbs, left sided weakness, giddiness and cloudiness and was suspected to have stroke. On examination, there was left upper and lower limb chorea, mild left sided deficit and flattening of nasolabial fold, fundoscpy was normal. Random blood sugar on admission was 500 mg/dl. Blood count, liver function tests, renal function tests, inflammatory markers, thyroid function tests, electrocardiogram were normal.

Initially non contrast enhanced CT brain was performed to rule out hemorrhagic stroke but was normal, MRI brain was subsequently obtained demonstrating heterogeneously T1 hyperintensityinvolving right caudate and lentiform nucleus tracking to the medial aspect of right thalamus and anterior part of right half of midbrain.T2 weighted images shows heterogenoushyperintense signal in the right basal ganglia region. There is no diffusion restriction or blooming on diffusion and gradient echo images respectively.

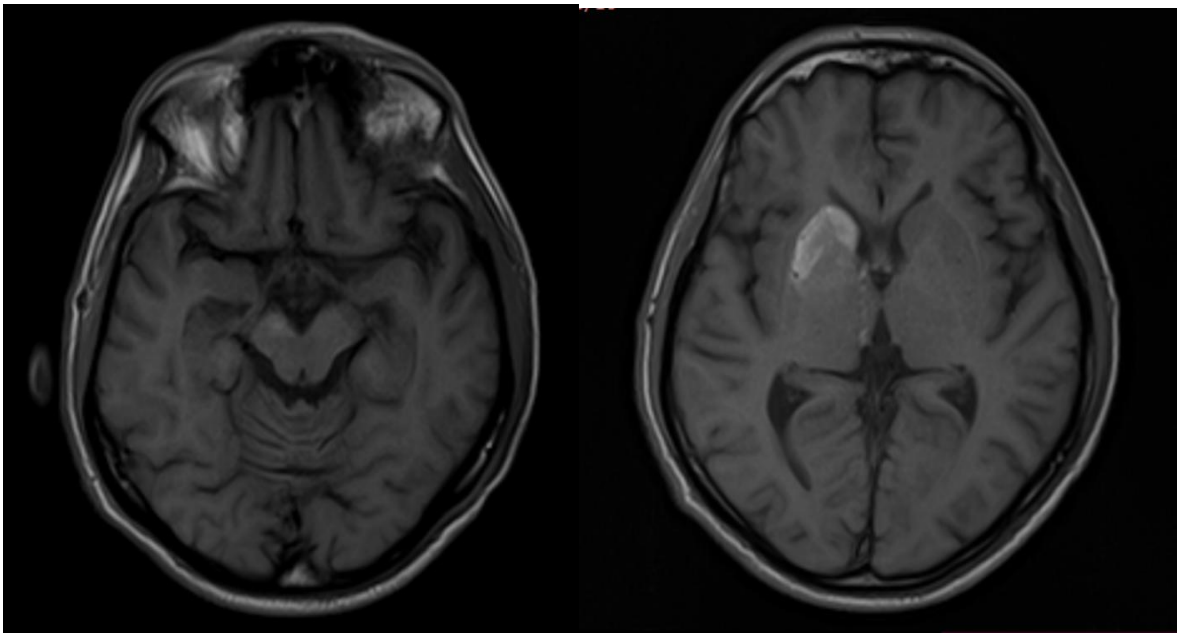

Figure 1: Non contrast MR at the level of basal ganglia and midbrain demonstrate T1 hyperintensity in right lentiform nucleus, caudate nuclei and right half of anterior midbrain

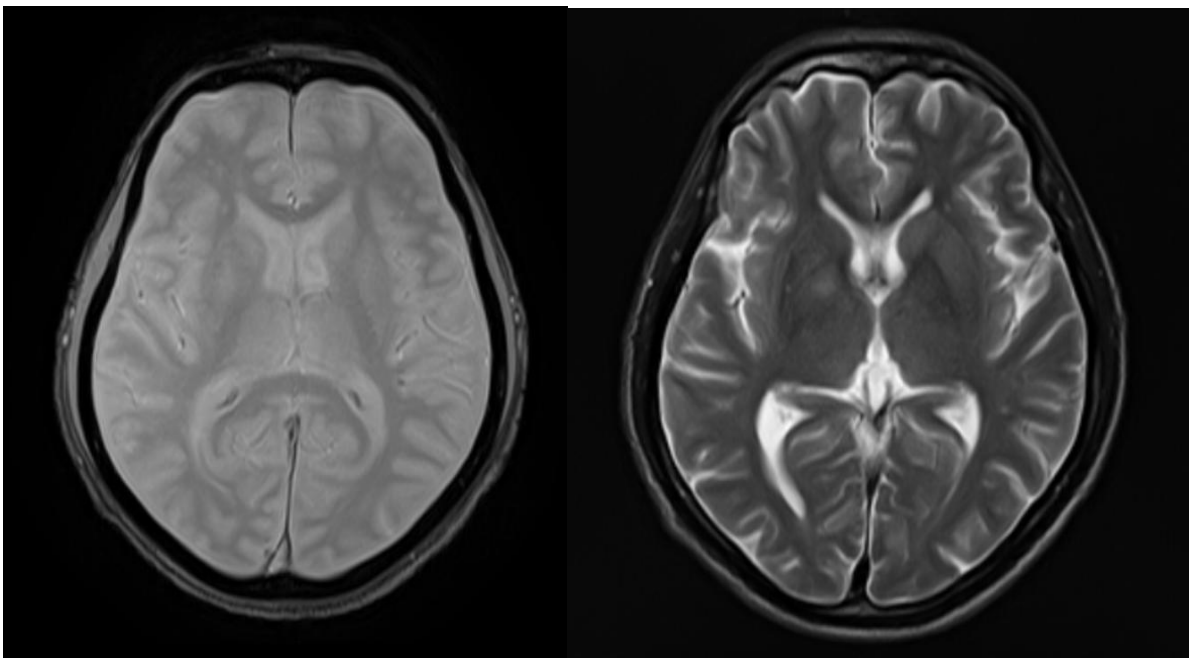

Figure 2 :T2 weighted images demonstrate heterogenous intermediately hyperintensesignal in right basal ganglia region, no blooming on gradient echo images.

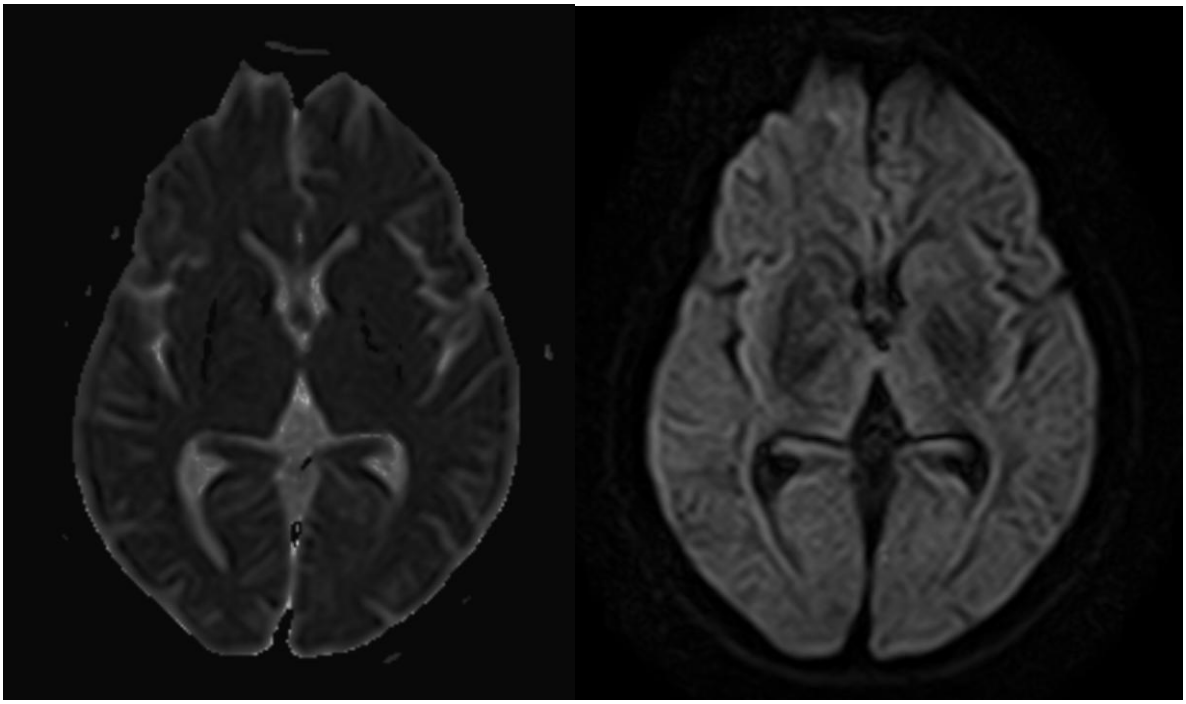

Figure 3 :Diffusion weighted images and corresponding ADC images at the level of basal ganglia were unremarkable .

Supported by the laboratory tests, these imaging findings consistent with nonketotic hyperglycemia in the context of poorly controlled diabetes. The patient was later was started on insulin regimen and his symptoms dramatically improved with neurologic exam returning to normal at normal serum glucose levels.Hence presumed etiology for his initial neurologic symptoms was a global neurologic dysfunction secondary to hyperglycemia. After a week of hospitalization, the patient was discharged and instructed to follow-up.

III. Discussion:

Nonketotic hyperglycemia is primarily due to poorly controlled blood sugar levels in type II diabetes mellitus patients who classically present with the clinical finding of Hemichorea-hemiballism .The majority of reported cases are reported in Asians with a female predominance often affecting the elderly, most commonly in the seventh decade.

Many hypotheses have been made explain the mechanism of the disease including dopaminergic hypersensitivity, vascular insufficiency leading to transient ischemia to basal ganglia and shifting of cerebral metabolism to the anaerobic pathway causing reduction in both GABA and acetylcholine with metabolic acidosis leading to basal ganglia dysfunction[4].

In literature, nonketotic hyperglycemia is associated with a characteristic appearance of unilateral or asymmetric lesions in basal ganglia typically contralateral to the side of the patient's presenting symptoms.

The most commonMRI finding associated with nonketotic hyperglycemia include high signal intensity basal ganglia and putamen on T1-weighted brain contralateral side. T2-weighted images have more variable finding with the majority of basal ganglia lesions are hypo-intense or iso-intense to background normal. On SWI, there are mixedresults with some demonstrating susceptibility artifact while others don't.

Lai, P.H. at el., evaluated neuroimaging of 10 patients with nonketotic hyperglycemic hemichorea in primary diabetes mellitus and CT and T1-weighted MR images showed lesions of the putamen and caudate unilaterally or bilaterally.

Lin, J.J. and colleagues report 7 similar patients with CT showing an increased density in the contralateral putamen and/or caudate and MRI revealing abnormal hyperintensity on T1-weighted and hypointensity on T2-weighted images. Lin, J.J. and colleagues followed up 7 patients and the lesions on CT and MRI showed complete resolution within 3 months and 11 months, respectively [5].

In another literature of 19 out of 22 patients who had a follow-up brain MRI after an interval of 2 months the high signal intensity basal ganglia lesions resolved along with the improvement in chorea. Another case demonstrates that 8 out of 9 patients underwent follow up MRI scans within an interval of 1–18 months and the hyperdense striatal lesions had disappeared completely or near-completely[6].

IV. Conclusion

Non-ketotic hyperglycemia is a rare treatablecondition mimicking stroke clinically as patients present with acute onset of unilateral chorea symptoms which can be be confused with an acute vascular event resulting in unnecessary investigations and interventions.Characteristic MRI findings with appropriate clinical history and lab findings help in timely diagnosis of this disease and helps physician to initiate appropriate treatment. This disease has good prognosis as compared to acute cerebrovascular injury and sypptms resolve after correction of elevated blood sugar levels

Acknowledgement
The study was supported by Department of Internal Medicine and Principal Dr. B. R. Ambedkar medical college, Bangalore, Karnataka
Conflicting interests: None

References

Bibliography

[1]. Oh SH, Lee KY, Im JH, et al. Chorea associated with non-ketotic hyperglycemia and hyperintensity basal ganglia lesion on T1-weighted brain MRI study: a meta-analysis of 53 cases including four present cases. J NeurolSci 2002;200:57–62.
[2]. Jagota P, Bhidayasiri R, Lang AE. Movement disorders in patients with diabetes mellitus. J NeurolSci 2012;314:5–11.
[3]. Narayanan S. Hyperglycemia-induced hemiballismushemichorea: a case report and brief review of the literature. J Emerg Med. 2012;43 (3): 442-4.
[4]. Oh SH, et al. Chorea associated with non-ketotic hyperglycemia and hyperintensity basal ganglia lesion on T1-weighted brain MRI study: a meta-analysis of 53 cases including four present cases. J Neurol Sci. 2002;200(1–2):57–62.
[5]. Lin JJ, et al. Presentation of striatal hyperintensity on T1-weighted MRI in patients with hemiballism-hemichorea caused by non-ketotic hyperglycemia: report of seven new cases and a review of literature. J Neurol. 2001;248(9):750–5.
[6]. Chromoblastomycosis. Urol Cutaneous Rev. 1946;50(6):341–3.

Dr. Siddhant Singh Chandel, et. al. "An interesting case of Hemichorea-hemiballism." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 21(10), 2022, pp. 08-11.