Endotext

DIABETIC STRIATOPATHY

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Received July 15, 2024

ABSTRACT

Acute onset de novo movement disorders are increasingly being reported in the settings of hyperglycemia, particularly from Asian countries. Although hemichorea-hemiballism is the most common and classically described movement semiology in association with hyperglycemia, various other hyperkinetic (choreoathetosis, dystonia, tremors, akathisia, restless leg syndrome etc.) and hypokinetic (parkinsonism) movement disorders are recognized. Diabetic striatopathy (DS) is defined as the disease phenomenon characterized by either choreo-ballistic movement or suggestive signature changes in striatum on imaging or presence of both. DS is generally considered as the complication of long-standing, poorly controlled non-ketotic hyperglycemia with acute hyperglycemic surge, though it can also be the first presentation of previously undiagnosed diabetes. Thus, it is recommended to test for capillary blood glucose in every patient presenting with de novo acute onset movement disorders of any semiology irrespective of past history of diabetes. It is important to recognize that normal brain imaging does not exclude the diagnosis of DS (clinically isolated DS) because nearly 50% cases may not have any characteristic neuroradiological stigmata. There is also high prevalence of clinical-neuroradiological discordance in DS cases. Thus, while managing such patients' priority should be imparted on bedside identification of the movement semiology accurately and aggressive treatment of hyperglycemia rather than ordering expensive neuroradiological investigation. Generally diabetic movement disorder carries excellent prognosis. The majority of cases rapidly resolves with insulin therapy alone with or without use of adjunctive neuroleptics.

INTRODUCTION

Although a myriad of neurological complications resulting from chronic micro- and macroangiopathy and acute metabolic perturbations in diabetes mellitus (DM) had been well documented, structured studies on acute-onset movement disorders among patients with DM were surprisingly left out until recently (1,2). Movement disorders can manifest either as the first manifestation of undiagnosed DM or in later advanced stages of the disease (3-7). Genesis of these abnormal movements can directly be attributed to hyperglycemia or hypoglycemia, and may result from diabetic complications such as vasculopathy and neuropathy (2,8). Moreover, there are syndromes or conditions which can present as movement disorders alongside DM (8,9). Aggressive glycemic control is

known to alleviate abnormal movements in most of the cases (1,2,8). Among all the movement semiologies discussed in literature, hemichorea-hemiballism is most frequently reported (1,2). Diabetic striatopathy (DS) is an umbrella term referring to a hyperglycemic condition associated with both or either one of the two following conditions: (1) acute onset chorea-ballism; (2) striatal hyperdensity on computed tomography (CT) or striatal hyperintensity on T1-weighted magnetic resonance imaging (MRI) (1,2,10). We herein briefly summarize the movement disorders in DM keeping DS at the center of discussion. Epidemiological and clinical spectrum, pathophysiology, neuroradiological conundrums, and available treatment are discussed. We also have tried to shed light upon the knowledge gaps in understanding of this particular disease that need to be addressed.

EPIDEMIOLOGY- MAGNITUDE OF THE PROBLEM

At present there is no prospective epidemiological study to assess the incidence or prevalence data available regarding movement disorders in diabetes. Few retrospective analyses with weak study methodology showed the prevalence of DS was in order of 1% or even less (11-13). On the other hand, a prospective study by Dubey et al revealed that 17.4% patients were diabetic among 552 patients presented with acute onset movement disorders and its mimics (including epilepsia partialis continua in a movement disorder clinic (1).

A systematic review of 176 patients observed that the lion share of DS cases was reported from Asian countries (2). Multiple factors like easy accessibility to healthcare, poor compliance to drugs, ethnicity, or genetic susceptibility might play roles, but it definitely requires more exploration. However, a study by Shafran et al revealed that DS was actually underdiagnosed in western populations leading to its underreporting (11).

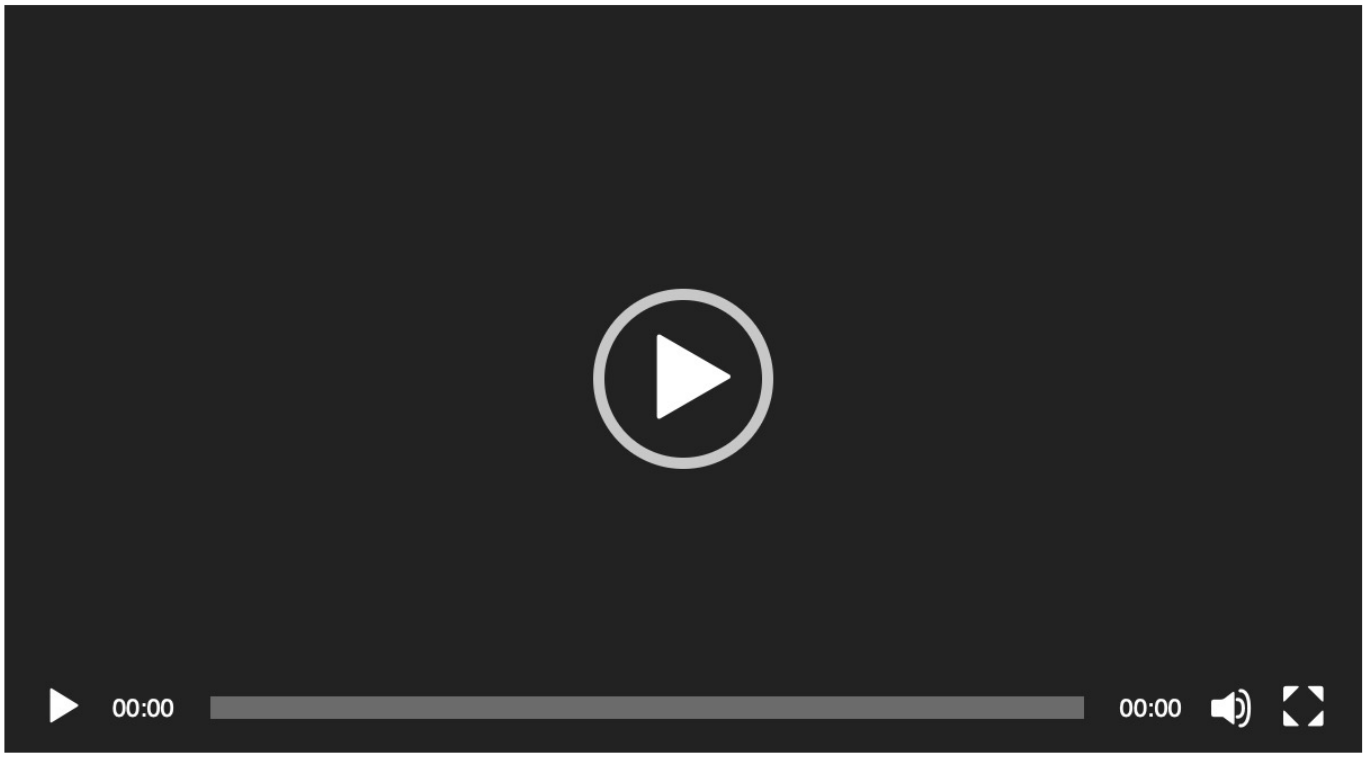
Acute onset movement disorders in diabetes had been reported in a wide range of age groups ranging from first to ninth decade (2). The mean age of the patients was generally sixth to seventh decade observed in different case series or systematic reviews (2,14-19). Two studies from India reported a relatively younger mean age (fifth decade) of presentation (1,20). Chen et al analyzing only the cases of hemichorea-hemiballism with ketotic hyperglycemia also found a median age of 54 years (21).

Across different studies over the years, notably, a woman preponderance (nearly 2 times in most of the studies) of hyperglycemic hemichorea-hemiballism movements had been observed (2,14-21). The exact reason for this female predominance or the role of biological sex on hyperglycemia-induced acute movement disorders needs further study. Some have postulated that increased dopaminergic receptor sensitivity secondary to estrogen deficiency in the striatum among postmenopausal women might make them susceptible to hyperkinetic movement disorders (17,21). In contrast with this global scenario, the authors' largest clinical series from India revealed a slight male predominance (52.5%) which needs further confirmation by replication in other independent studies (1).

CLINICAL PRESENTATION- SPECTRUM OF MOVEMENT DISORDERS IN DIABETES

Among different movement semiologies described among diabetics (table-1), hemichorea-hemiballism is the most common and classically described (1-8). See video 1-6 for different movement disorders associated with hyperglycemia.

To view the videos online, please click here.



1. 1. Video-1: Left upper limb monochorea as the presenting manifestation of dia…	**1:12**
2. 2. Video-2: Left hemichorea with dystonia in a 64-year-old patient with type 2 diabete…	0:35
3. 3. Video-3: Acute onset right upper limb monoballism in a 72-year-old male. He was …	0:05
4. 4. Video-4: Right lower limb monoballism for last 3 months in a 48-year-old female wi…	1:41
5. 5. Video-5: Left hemichorea with dystonia and left hemifacial spasm in a 60-year-old …	0:31
6. 6. Video-6: Diaphragmatic myoclonus (duration of 3 days) in a 75-year-old lady with …	0:27

Table 1. Different Movement Semiologies Observed Among Patients with Diabetes	
Choreic and ballistic movements	**Non-choreoballistic movements**
Choreoballism- hemi / mono / generalized Pure chorea- hemi / mono / generalized Pure ballism- hemi / mono / generalized Choreoathetosis	Tremors Hemifacial spasm Parkinsonism Myoclonus- focal, action, diaphragmatic, opsoclonus-myoclonus Dystonia Restless leg syndrome Ataxia Dyskinesia- Paroxysmal kinesigenic dyskinesia, paroxysmal non-kinesigenic dyskinesia, paroxysmal exertional dyskinesia

Dubey et al (1) in their largest clinical series showed that non-choreic, non-ballistic movements were present among 30.5% of 59 cases. Therefore, an immediate capillary blood glucose (CBG) measurement in all patients with any sort of acute onset movement disorders is of pivotal importance before ordering other costly and time-consuming investigations. Bilateral clinical involvement was identified in 37.2% of all patients and was significantly more common in non-choreoballistic movement disorders than choreoballism (1). In an analysis by Chua et al bilaterality was documented in 9.7% DS cases (2), whereas bilaterality was even more frequently (19.5%) observed in the series described by Dubey et al (1). The latter was the only study which systematically assessed both the hyperglycemia-associated choreoballism and non-choreoballistic movement disorders. It observed no statistically significant differences regarding demographic or clinical variables between these two types of movement disorders except bilaterality and delay in diagnosis (more frequent in non-choreoballism than choreoballism) (1).

Many a times seizures can mimic hyperkinetic movement disorders or sometimes both may coexist (3). Most commonly epilepsia partialis continua mimics a movement disorder. It is not conventionally categorized as a movement disorder; but is rather a type of simple focal motor status epilepticus with frequent repetitive muscle jerks, usually arrhythmic, that continues over prolonged periods. Moreover, the epilepsia partialis continua patients have electroencephalographic changes. The differentials to be considered are stroke, associated opposite hemispheric structural defect/s, and space-occupying lesions. Non-ketotic hyperglycemia is a well-known cause of reversible epilepsia partialis continua (22,23).

CORRELATION WITH MARKERS OF GLYCEMIA AND DIABETIC COMPLICATIONS

Movement disorders have been described in different types of DM, including type 1, type 2 and type 3c diabetes (1,2,5). DS is generally a complication of long-standing DM with poorly controlled glycemic status flared up by an acute hyperglycemic surge in a non-ketotic milieu. In the cohort of Dubey et al the mean duration of DM was 9.8 years and movement disorders were the presenting manifestation of previously undiagnosed DM in three cases (5.1%) (1). Patient-level meta-analysis of previously published cases has found a higher number (17%) of DS cases having previously undiagnosed DM (2). This discrepancy could be due to lack of screening or publication bias. Nonetheless, it is recommended to measure blood glucose levels at presentation among all patients with acute onset movement disorders

irrespective of their past glycemic status. Importantly, the majority of the patients with DS bears the stigmata of other chronic microvascular diabetic complications (1).

PATHOPHYSIOLOGY- HOW METABOLIC MICROVASCULAR EFFECTS INFLUENCE MACRO-MOVEMENTS

From the various previously reported speculative pathophysiological basis of DS, Dubey et al (10) proposed "ominous octet" of pathogenesis of DS, which includes sequential occurrence of following factors: 1) gemistocytopathy, 2) petechial hemorrhage, 3) methemoglobin deposition, 4) mineral (calcium and magnesium) deposition, 5) cytotoxic edema, 6) myelinolysis, 7) gliosis, and 8) atrophy. The hyperglycemic state results in hyperosmolarity and hyperviscosity leading to reduced cerebral blood flow causing insult to the striatal astrocytes which are exquisitely sensitive to ischemia. These tumescent reactive astrocytes are known as gemistocytes, the most consistent finding gathered from limited number of biopsy studies (2).

Interestingly enough, genesis of majority of hyperglycemic movement disorders occurs in the background of non-ketotic milieu (1,2). In non-ketotic hyperglycemia brain metabolism is shifted towards the alternative anaerobic pathway in Krebs cycle causing depletion in gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter. This leads to attenuated inhibition of the subthalamus by the medial globus pallidus resulting in hyperkinetic movements. Conversely, GABA can be readily re-synthesized from acetoacetate, which is in abundance in the ketotic milieu (8). Hence, in the latter state, hyperkinetic movements rarely occur unless some other sinister mechanisms (such as cerebrovascular insufficiency or ultrastructural changes in basal ganglia) are at play (1).

NEURORADIOLOGICAL AND CLINICAL CONUNDRUM OF DS

Although the sensitivity of MRI was observed to be higher than CT scan to detect DS (95.3% vs. 78.9%), the need for CT can't be obviated in cases of negative MRI scans. There is plenty of cases where mismatch (defined as the complete absence of anomaly in basal ganglia on one imaging modality, but not the other) and incompatibility (defined as the difference in locations of striatal anomalies between CT and MRI) exist (2). Hyperdensity on CT or hyperintensity on T1-weighted MRI in contralateral (to the side of the abnormal movements) putamen surrounding edema or mass effect, along with hyperglycemia and hemichorea-hemiballism movements, is pathognomonic of DS (10). Putamen is the most commonly involved striatal structure, whereas isolated caudate or globus pallidus or subthalamic nucleus involvement seem to be less frequent (1,2). A significant portion of cases shows concomitant affliction of all three striatal components (putamen, caudate, and globus pallidus) (2). The reason behind putaminal vulnerability to hyperglycemia and how the same anatomical lesion causes such wide arrays of movement disorders remain elusive.

The pathological basis behind the striatal hyperintensity on T1-weighted MRI and hyperdensity on CT scan in these patients can be proved by histopathological evidence of petechial hemorrhages causing accumulation of methemoglobin (2). Unfortunately, this theory of microhemorrhages behind T1 hyperintensity can't be substantiated well on corresponding gradient-echo images. In contrast, accumulation of gemistocytes due to ischemic events and neuronal dysfunction may partially explain the striatal hyperintensity on T1-weighted MRI, but not hyperdensity on CT (2,10). Few DS cases have documented restricted diffusion in diffusion-weighted imaging sequence (24). Advanced imaging modalities such as MR volumetry, spectroscopy, functional MRI, positron emission tomography (PET), single photon emission CT (SPECT), susceptibility weighted MR, perfusion imaging etc. although not routinely done in clinical practice for diagnosis of DS, might unveil its intricate pathophysiological basis (2,10).

Previous studies showed that in different case series patients with choreo-ballistic movements did not have suggestive neuroimaging findings in 5-45% cases (clinically isolated DS) (2,14-17,25). Study focusing on both choreo-ballistic and non-choreo-ballistic movements revealed that only 44% cases had changes in brain MRI (1). This wide variability had been attributed to the varied use of MRI or CT and non-homogenous neuroradiological definition of DS applied among various studies (1,2,10). Moreover, neuroradiological changes lag behind the clinical manifestations. Nevertheless, it underscores the importance of initiating management by recognizing this disease phenomenon on the basis of clinical symptomatology (presence of acute onset movement disorders with concurrent hyperglycemia) without waiting for neuroimaging (1). On the contrary, 2% of patients may show radiological striatal lesions without any clinically manifested movement disorders (radiologically isolated DS) (2,26-28). There are also plenty of reports of clinical-radiological discordance or inconsistency in DS (1,2,14). Thus, striatopathy with clinically manifested movement disorders (symptomatic DS) can be subdivided into two groups, i.e., 1) concordant: bilateral involuntary movements with bilateral DS, or unilateral involuntary movements with contralateral DS (6); and 2) discordant: bilateral involuntary movements with unilateral DS or unilateral involuntary movements with bilateral or ipsilateral DS (10,29-31). This frequently observed clinical-radiological dissociation in DS is apparently contradictory with the classical concept of neurological localization of lesion-manifestation and requires further studies with newer neuroimaging modalities (1,10). Due to the controversial and ambiguous nature of the term "diabetic striatopathy" in literature (2), we had previously proposed a three-subset classification (10) (figure- 1).

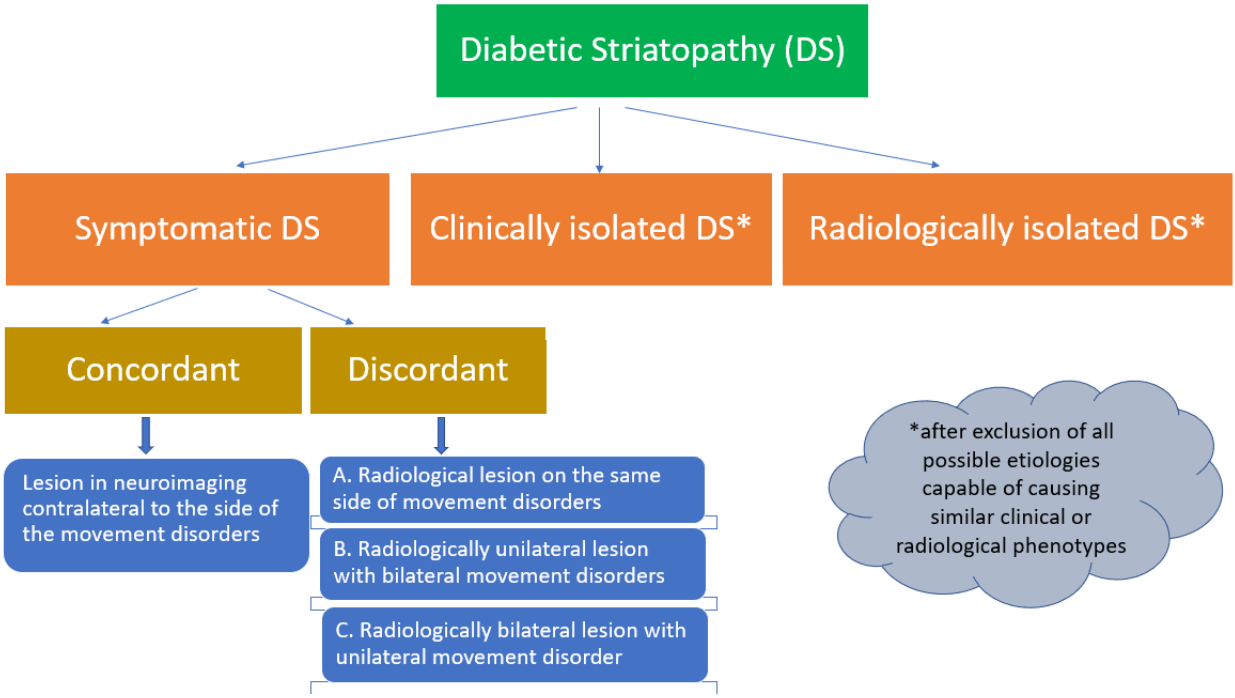


Figure 1. Dubey's classification schema of Diabetic Striatopathy (Adapted from: Dubey S, Biswas P, Ghosh R, Chatterjee S, Ray BK, Benito-León J. Neuroimaging of Diabetic Striatopathy: More Questions than Answers. Eur Neurol. 2022;85:371-6.)

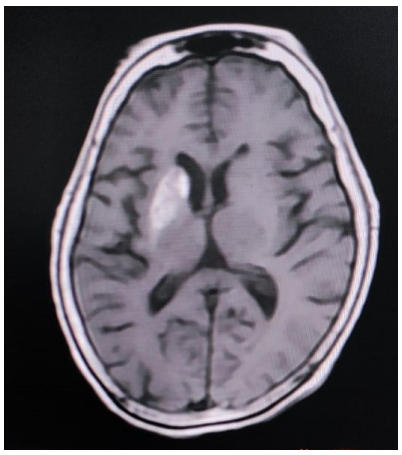


Figure 2. Right striatal hyperintensity on T1 weighted MRI in a 56-yeald-old lady with previously undiagnosed diabetes presented with left hemichorea-hemiballism persisting for 1 week. Blood glucose was 453 mg/dl. Movement disorders abated with management of hyperglycemia with insulin therapy alone.

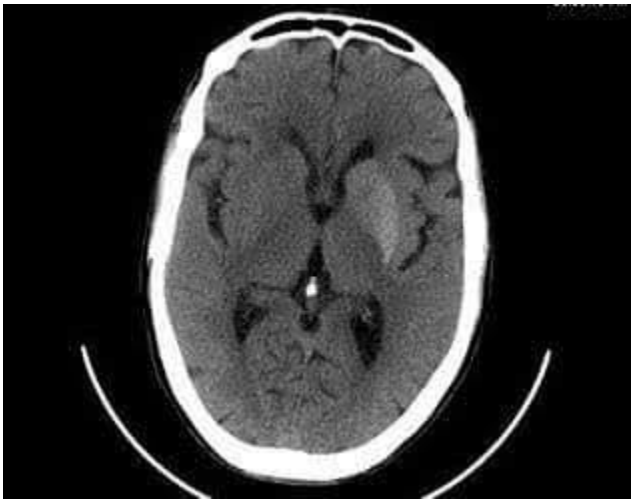


Figure 3. Left striatal hypodensity on non-contrast CT scan in a 68-year-old gentleman with diabetes presented with right hemichorea. Blood glucose was 356 mg/dl and HbA1c was 15.2%. Movement disorder was partially improved with glycemic control and needed haloperidol for complete recovery.

TREATMENT AND PROGNOSIS

Intensive management of hyperglycemia with insulin remains the pivotal measure to treat movement disorders associated with hyperglycemia (1,2). Some authors have speculated worsening of involuntary movements on aggressive lowering of blood glucose (analogous to diabetic retinopathy) (32-35), but this needs clarification by further reports. According to past studies, from one-fourth to almost half of the patients recover with insulin therapy alone (1,2,16,17) with a higher recovery rate in ketotic hyperglycemia cases (21). Additional therapies such as haloperidol, tetrabenazine, risperidone, tiapride (ballism and chorea), levodopa (parkinsonism), trihexyphenidyl, clonazepam (dystonia), pramipexole (restless leg syndrome), propranolol (tremor), carbamazepine (hemifacial spasm) etc. have been used with varying success rates (1,2). Whether the requirement of additional drugs may be attributed to late presentation or diagnostic delay needs further study (1,2,15,20). Surgical interventions such as pallidotomy,

ventrolateral thalamotomy, transcranial magnetic stimulation, and globus pallidus internus deep brain stimulation had been tried for intractable symptoms (2,36-38).

In the study by Dubey et al (1), treatment of movement disorders was documented and followed up for at least three weeks. Patients who recovered fully from all involuntary movements within seven days were regarded as early responders, while the rest were taken as late responders. In that series the majority (47.5%) of the patients had early and complete resolution of symptoms, 28.8% responded late but had a complete reversal, while 23.7% cases recovered partially. Interestingly, in Chua et al's analysis recovery was earlier among patients on glucose-therapy only (2 days) compared to those receiving additional anti-chorea medications (14 days), although median pre-treatment lag period was identical between those two groups (4 days) (2). Overall, the previous literature showed that recovery rate varied from 76.4% to 100% (2,14-21), which could be attributable to heterogeneity in the definition of recovery (clinical and/or neuroradiological) and duration of follow-up employed across different studies. During recovery, as expected, symptomatic improvement precedes abolition of neuroradiological stigmata. Minimum time period for radiological reversal noted in study by Chua et al were 10 days on CT and 60 days on MRI. On follow-up MRI scans progressive increase in striatal hyperintensity to reach its maximum limit was noted at around 90 days, whereas the mean periods of complete radiological reversal were around 60 and 180 days on CT and MRI, respectively. The median duration of discernible changes on CT and MRI were 24 and 120 days respectively (2). However, it is not at all uncommon to come across cases demonstrating persisting striatal anomalies on follow-up neuroimaging for months irrespective of symptomatic recovery (2,3,39). Currently there is paucity of studies which longitudinally evaluate the evolution of radiological changes over the course of disease process.

Despite having limited data regarding long-term follow-up, nearly 20% cases of DS clinically recurred even after initial resolution of striatal anomalies., which underscored the importance of periodic neuroradiological surveillance even after initial recovery. Recurrence rate did not differ across different treatment modalities (i.e., with or without additional use of anti-chorea medications) employed (2).

CONCLUSION

Acute onset or de novo movement disorder is one of the important neurological complications of DM, most prevalent but not limited to Asian population. Unfortunately, it is still less well-recognized among physicians, diabetologists, and endocrinologists leading to its diagnostic delay and probably poorer prognosis. Although DS and other movement disorders are generally complications of poorly controlled long-standing type 2 diabetes in the non-ketotic hyperglycemia state among elderly, it may be the first presentation of diabetes. Hence, clinicians must be aware of this entity so that crucial time is not wasted and readily available glucose measurement are ordered when dealing with such patients irrespective of their past glycemic status. Exact pathophysiological mechanisms, genetic basis, radiological correlates, and the explanation for the seemingly discordant clinical-radiological picture in hyperglycemia-induced movement disorders remain elusive. Much work needs to be done to determine the optimal management and prognostic indicators of this emerging disease entity.

ACKNOWLEDGMENTS

Informed consent was obtained from all patients whose videos are included in this chapter.

REFERENCES

1. Dubey S, Chatterjee S, Ghosh R, Louis ED, Hazra A, Sengupta S, et al. Acute onset movement disorders in diabetes mellitus: A clinical series of 59 patients. Eur J Neurol. 2022;29:2241-8.
2. Chua CB, Sun CK, Hsu CW, Tai YC, Liang CY, Tsai IT. "Diabetic striatopathy": clinical presentations, controversy, pathogenesis, treatments, and outcomes. Sci Rep. 2020;10:1594.
3. Chatterjee S, Ghosh R, Ojha UK, Diksha, Biswas P, Benito-León J, et al. Recurrent facial focal seizure with chronic striatopathy and caudate atrophy- a double whammy in an elderly woman with diabetes mellitus. Neurohospitalist. 2022;12:147-50.
4. Chatterjee S, Ghosh R, Kumari R, Ojha UK, Benito-León J, Dubey S. Faciobrachial myoclonus as the presenting manifestation of diabetic keto-acidosis. Tremor Other Hyperkinet Mov (N Y). 2021;11:9.
5. Ghosh R, Roy D, Chatterjee S, Dubey S, Swaika BC, Mandal A, et al. Hemifacial spasm as the presenting manifestation of type 3c diabetes mellitus. Tremor Other Hyperkinet Mov (N Y). 2021;11:14.
6. Ghosh R, Dubey S, Roy D, Ray A, Pandit A, Ray BK, et al. Choreo-ballistic movements heralding COVID-19 induced diabetic ketoacidosis. Diabetes Metab Syndr. 2021;15:913-7.
7. Dubey S, Chatterjee S, Mukherjee D, Ghosh R, Sengupta S, Lahiri D, et al. “Dancing belly” in an old diabetic lady. J Family Med Prim Care. 2020;9:2580-2.
8. Jagota P, Bhidayasiri R, Lang AE. Movement disorders in patients with diabetes mellitus. J Neurol Sci. 2012;314:5-11.
9. Chakraborty PP, Ray S, Bhattacharjee R, Ghosh S, Mukhopadhyay P, Mukhopadhyay S, et al. First Presentation of Diabetes as Diabetic Ketoacidosis in a Case of Friedreich's Ataxia. Clin Diabetes. 2015;33:84-6.
10. Dubey S, Biswas P, Ghosh R, Chatterjee S, Ray BK, Benito-León J. Neuroimaging of Diabetic Striatopathy: More Questions than Answers. Eur Neurol. 2022;85:371-6.
11. Shafran I, Greenberg G, Grossman E, Leibowitz A. Diabetic striatopathy- Does it exist in non-Asian subjects? Eur J Intern Med. 2016;35:51-4.
12. Ryan C, Ahlskog JE, Savica R. Hyperglycemic chorea/ballism ascertained over 15 years at a referral medical center. Parkinsonism Relat Disord. 2018;48:97-100.
13. Ottaviani S, Arecco A, Boschetti M, Ottaviani E, Renzetti P, Marinelli L. Prevalence of diabetic striatopathy and predictive role of glycated hemoglobin level. Neurol Sci. 2022;43:6059-65.
14. Gómez-Ochoa SA, Espín-Chico BB, Pinilla-Monsalve GD, Kaas BM, Téllez-Mosquera LE. Clinical and neuroimaging spectrum of hyperglycemia-associated chorea-ballism: systematic review and exploratory analysis of case reports. Funct Neurol. 2018;33:175-87.
15. Cosentino C, Torres L, Núñez Y, Suárez R, Vélez M, Flores M. Hemichorea/hemiballism associated with hyperglycemia: report of 20 cases. Tremor other hyperkinet mov (N Y). 2016;6:402.
16. Guo Y, Miao YW, Ji XF, Li M, Liu X, Sun XP. Hemichorea associated with nonketotic hyperglycemia: clinical and neuroimaging features in 12 patients. Eur Neurol. 2014;71:299-304.
17. Lee SH, Shin JA, Kim JH. Chorea-ballism associated with nonketotic hyperglycaemia or diabetic ketoacidosis: characteristics of 25 patients in Korea. Diabetes Res Clin Pract. 2011;93:e80-3.
18. Oh SH, Lee KY, Im JH, Lee MS. Chorea associated with nonketotic 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;200:57-62.
19. Lee BC, Hwang SH, Chang GY. Hemiballismus-hemichorea in older diabetic women: a clinical syndrome with MRI correlation. Neurology. 1999;52:646-8.
20. Prabhu S, Ramya N. Movement disorders and diabetes: a study of South India. Internet J Neurol. 2012;14:1-5.
21. Chen C, Zheng H, Yang L, Hu Z. Chorea-ballism associated with ketotic hyperglycemia. Neurol Sci. 2014;35:1851-5.
22. Paiboonpol S. Epilepsia partialis continua as a manifestation of hyperglycemia. J Med Assoc Thai. 2005;88:759-62.
23. Shrivastava V, Burji NP, Basumatary LJ, Das M, Goswami M, Kayal AK. Etiological profile of epilepsia partialis continua among adults in a tertiary care hospital. Neurol India. 2013;61:156-60.
24. Chu K, Kang DW, Kim DE, Park SH, Roh JK. Diffusion-weighted and gradient echo magnetic resonance findings of hemichoreahemiballismus associated with diabetic hyperglycemia: a hyperviscosity syndrome? Arch Neurol. 2002;59:448–52.
25. Chen C, Zheng H, Yang L, Hu Z. Chorea-ballism associated with ketotic hyperglycemia. Neurol Sci. 2014;35:1851-5.
26. Choi JY, Park JM, Kim KH, Park JS, Shin DW, Kim H. Radiographic basal ganglia abnormalities secondary to nonketotic hyperglycemia with unusual clinical features. Clin Exp Emerg Med. 2016;3:252-5.
27. Hsu JL, Wang HC, Hsu WC. Hyperglycemia-induced unilateral basal ganglion lesions with and without hemichorea. A PET study. J Neurol. 2004;251:1486-90.
28. Hansford BG, Albert D, Yang E. Classic neuroimaging findings of nonketotic hyperglycemia on computed

tomography and magnetic resonance imaging with absence of typical movement disorder symptoms (hemichorea-hemiballism). J Radiol Case Rep. 2013;7:1-9.
29. Danve A, Kulkarni S, Bhoite G. Non-ketotic hyperglycemia unmasks hemichorea. J Community Hosp Intern Med Perspect. 2015;5:27825.
30. Lin JJ. Ipsilateral putamen hyperintensity on T1-weighted MRI in non-ketotic hyperglycemia with hemiballism-hemichorea: a case report. Parkinsonism Relat Disord. 2001;7:319-21.
31. Fong SL, Tan AH, Lau KF, Ramli N, Lim SY. Hyperglycemia-associated hemichorea-hemiballismus with predominant ipsilateral putaminal abnormality on neuroimaging. J Mov Disord. 2019;12:187-9.
32. Lim KX, Khaing Zin T, Yu Z, Peh WM. Delayed Presentation of Hemichorea in Diabetic Striatopathy. Cureus. 2022;14:e30219.
33. Lizarraga KJ, Adams D, Post MJD, Skyler J, Singer C. Neurovascular uncoupling after rapid glycemic control as a trigger of the diabetic-uremic striatopallidal syndrome. Parkinsonism Relat Disord. 2017;39:89-90.
34. Ando Y, Kadoya M, Kodera T. Involuntary Movements During Treatment for Hyperglycemia. AACE Clin Case Rep. 2022;9:21-2.
35. Cho HS, Hong CT, Chan L. Hemichorea after hyperglycemia correction: A case report and a short review of hyperglycemia-related hemichorea at the euglycemic state. Medicine (Baltimore). 2018;97:e0076.
36. Son BC, Choi JG, Ko HC. Globus Pallidus Internus Deep Brain Stimulation for Disabling Diabetic Hemiballism/Hemichorea. Case Rep Neurol Med. 2017;2017:2165905.
37. De Vloo P, Breen DP, Milosevic L, Lee DJ, Dallapiazza RF, Hutchison WD, et al. Successful pallidotomy for post-hyperglycemic hemichorea-ballism. Parkinsonism Relat Disord. 2019;61:228-30.
38. Kaseda Y, Yamawaki T, Ikeda J, Hayata M, Dohi E, Ohshita T, et al. Amelioration of persistent, non-ketotic hyperglycemia-induced hemichorea by repetitive transcranial magnetic stimulation. Case Rep Neurol. 2013;5:68-73.
39. Lucassen EB, Delfyett WT, Stahl MC. Persistent Hemichorea and Caudate Atrophy in Untreated Diabetic Striatopathy: A Case Report. Case Rep Neurol. 2017;9:299-303.