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DIABETIC STRIATOPATHY CLINICAL, RADIOLOGICAL PROFILE AND OUTCOMES – A PROSPECTIVE STUDY FROM TAMIL NADU.

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ABSTRACT **Background:** Diabetic striatopathy (DS) is an uncommon hyperglycemia-related movement disorder characterized by hemichorea–hemiballismus and striatal imaging abnormalities, with limited contemporary Indian data. **Objective:** To describe the clinical profile, neuroimaging findings, and outcomes of DS in a tertiary care center in Tamil Nadu. **Methods:** This prospective observational case series included adults presenting between September 2023 and September 2025 with acute or subacute movement disorders associated with hyperglycemia. Clinical, laboratory, neuroimaging, treatment, and outcome data were analyzed descriptively. **Results:** Thirteen patients (mean age 73.2 ± 7.8 years; 84.6% male) were included. All had poorly controlled type 2 diabetes mellitus (mean HbA1c 12.2 ± 1.9%) with non-ketotic hyperglycemia. Hemichorea–hemiballismus predominated. Neuroimaging abnormalities were seen in 92.3%, most commonly contralateral putaminal T1 hyperintensity, with right-sided predominance. All improved with glycemic correction; residual chorea persisted in 30.8% and relapse in 15% at six months. **Conclusion:** DS predominantly affects elderly patients with poorly controlled diabetes. While short-term outcomes are favorable, residual and recurrent symptoms highlight the need for sustained glycemic control and follow-up.

KEYWORDS : Diabetic striatopathy; Hemichorea; Hemiballismus; Hyperglycemia; Basal ganglia; Movement disorders

INTRODUCTION

The term diabetic striatopathy(DS) was coined around 2009 [1] and refers to hyperglycemia presenting with (1) acute or subacute onset chorea/ballism and or (2)striatal hyper density in CT or T1 hyperintensity in MRI [2].The disease however has been well described since decades .It is the second most common cause of hemichorea /hemiballismus [3] and has been given various names including "hyperglycemic non ketotic hemichorea hemiballismus" ,"diabetic hemiballismus/hemichorea" etc .It was considered to be a disease affecting only Asian population ,elderly females and strictly in the setting of non ketotic hyperglycemia[4] .However recent studies particularly from Indian have described an increasingly heterogenous picture wherein the gender ratio in the largest study was nearly equal and cases were described even in ketotic hyperglycemia [5].Interestingly ,other movement disorders including tremor ,dystonia ,hemifacial spasm and parkinsonism are being described in the spectrum of diabetic striatopathy . In India, where diabetes mellitus is highly prevalent and often poorly controlled, DS is likely underdiagnosed and underreported, with existing literature largely limited to isolated case reports. Awareness of this entity among neurologists is essential to avoid unnecessary investigations and to ensure timely metabolic correction.

In this case series, we describe the clinical presentation, neuroimaging features, and outcomes of patients with DS to a tertiary care neurology center in India, highlighting the spectrum of neurological manifestations and response to treatment.

METHODOLOGY

This prospective observational case series was conducted at the Department of Neurology of a tertiary care referral center in Tamil Nadu, India. Medical records of patients evaluated between September 2023-september 2025 were reviewed.

Adult patients (≥18 years) presenting with acute or subacute onset movement disorders temporally associated with hyperglycemia were included. Hyperglycemia was defined as random blood glucose >200 mg/dL and/or HbA1c ≥6.5%. Eligible movement disorders included chorea, Ballismus, dystonia, tremor, myoclonus, or mixed phenomenology .Hemiballismus was defined as "involuntary, violent, coarse and wide-amplitude movements involving ipsilateral arm and leg" [6] and hemichorea was defined as brief, abrupt, irregular, unpredictable, non-stereotyped movements affecting one side of the body[7] .The remaining movements were defined by their standard MDS definitions .Patients with either clinical or radiological fulfillment of DS criteria were included[2] . Patients with structural brain lesions unrelated to hyperglycemia (stroke, tumor, infection, demyelination), drug-induced or hereditary movement disorders, alternative metabolic encephalopathies, or incomplete clinical or imaging data were excluded.

All standard lab parameters were collected .All patients underwent neuroimaging with CT and/or MRI brain. MRI sequences reviewed included T1-weighted, T2-weighted, FLAIR, DWI, ADC, and SWI where available. Imaging was evaluated for basal ganglia involvement, laterality, and signal characteristics. Radiological assessment was performed independently by a neurologist and a radiologist, with consensus agreement for discrepant findings.

Treatment details, including glycemic control measures and symptomatic therapy for movement disorders, were recorded. Outcomes assessed included time to clinical improvement after correction of hyperglycemia, degree of symptom resolution at discharge, and status at follow-up where available.

Statistical analysis was descriptive. Continuous variables were summarized as mean ± standard deviation or median with interquartile range, and categorical variables as frequencies and percentages. Clinical–radiological correlations and prognostic factors were analyzed narratively due to the small sample size.

RESULTS

A total of **13 patients** were included in this case series of diabetic striatopathy. The **mean age** at presentation was **73.2 ± 7.8 years**, indicating a predominantly elderly cohort. There was a marked **male predominance**, with **11 males (84.6%)**and **2 females (15.4%)**.

All patients had **type 2 diabetes mellitus (T2DM)**. Diabetes was **newly diagnosed at presentation** in **2 patients (15%)**, while the remaining had a prior diagnosis. The cohort demonstrated **poor long-term glycemic control**, with a **mean HbA1c of 12.2 ± 1.9%**. The **mean duration of diabetes** among known diabetics was **3.04 ± 3.15 years**. All patients presented with **non-ketotic hyperglycemia**, and none had evidence of diabetic ketoacidosis.

The **mean duration from symptom onset to hospital presentation** was **2.92 days**. The **predominant movement phenomenology** was **hemichorea–hemiballismus**. With respect to laterality, movements were **left-sided in 5 patients**, **right-sided in 3 patients**, and **bilateral in 4 patients**. One patient had oromandibular dyskinesia.

Neuroimaging abnormalities suggestive of diabetic striatopathy were identified in **12 patients (92.3%)**. Of these, **6 patients showed**

characteristic findings on CT brain, and **6 on MRI brain**. One patient had **normal CT imaging** despite classical clinical features. **MRI lesions predominantly involved contralateral T1 hyperintensity in contralateral putamen ,followed by caudate hyperintensity .**

All patients received treatment in the form of hydration and insulin therapy ,12 of our patients were started on Haloperidol and clonazepam .One each on sodium valproate and tetrabenazine .Mean time to improvement was 3.3 days with all patients reporting improvement ,but only one patients being completely asymptomatic .At 1 month follow up 8 patients had compete recovery and 5 patients had recovered only partially with residual mild choreoathetosis .At 6 months 4 patients had residual chorea ,but not the disabling hemiballismus .2 of these patients had relapse of severe hemichorea hemiballismus due to poor drug compliance and hyperglycemia .Interestingly both their MRI brain taken at readmission had showed T2 FLAIR hyperintensities in basal ganglia suggestive of ischemic changes.

Table 1. Patient Characteristics Of Diabetic Striatopathy Cohort

Characteristic	Value
Number of patients, n	13
Age (years)	
Mean ± SD	73.2 ± 7.8
Sex, n (%)	
Male	11 (84.6%)
Female	2 (15.4%)
Type of diabetes, n (%)	
Type 2 diabetes mellitus	13 (100%)*
Newly diagnosed diabetes at presentation, n (%)	2 (15%)
HbA1c (%), mean ± SD	12.2 ± 1.9
Hyperglycemic state, n (%)	
Non-ketotic hyperglycemia	13 (100%)*
Mean duration of DM	3.04 (SD=3.15)
Time from symptom onset to presentation (days)	2.92 days
Laterality of movement disorder	Left -5 right -3 bilateral -4 1-oromandibular dyskinesia
Common movement phenomenology	Hemichorea / Hemiballismus
Positive imaging findings	12 (92.3 %)[CT-6 MRI -6, 1 CT normal)
Side of lesion in imaging	Right -9 (75%)left -3(25%)

DISCUSSION

A variety of conditions can cause acquired hemichorea-hemiballismus syndrome. These include structural damage to subcortical and cortical structures (cerebrovascular disease, infection, trauma, neoplasia), inflammatory disorders(paraneoplastic ,autoimmune) metabolic (nonketotic hyperglycemia), or drug induced (dopamine agonist or phenytoin). **Diabetic striatopathy was a rare treatable neurological manifestation of hyperglycemia which if not identified and treated promptly may lead to residual chronic hemi chorea, sometimes even requiring surgical treatment [8,9,10].The exact pathology is yet to be elucidated ,however there is histopathological evidence of(1) petechial hemorrhages causing accumulation of methemoglobin, and (2) presence of gemistocytes (reactive astrocytes)[11],[12] .There is also increasing evidence that DS may be a diabetic vasculopathy as evidenced by small vessel ischemic changes in striatum noted on follow up [13].**

In this case series, diabetic striatopathy (DS) predominantly affected elderly patients with severe chronic hyperglycemia, presenting acutely with hemichorea–hemiballismus in a non-ketotic milieu. The mean age in our cohort (73.2 years) is comparable to that reported by Nadig et al. [14] (70.2 years) and aligns with pooled international data identifying DS as a disorder of the sixth to eighth decades of life.

A key distinguishing feature of our study is the marked male predominance (84.6%). Nadig et al. reported a female predominance (female: male ratio 1.5:1), consistent with earlier literature . This is attributed to the estrogen depleted state in post menopausal women leading to increased dopaminergic sensitivity .However, Dubey et al.[5] , in their large Indian clinical series of 59 patients, observed a slight male predominance (52.5%) and probably indicates that the female predominance might have been a selection bias and needs further prospective studies incorporating functional imaging to arrive at a definite conclusion [15].All patients in our series had type 2 DM with very poor glycemic control (mean HbA1c 12.2%). This exceeds the mean HbA1c reported by Nadig et al. (10.98%) and is comparable to the upper range described in Dubey et al. and pooled reviews, reinforcing chronic hyperglycemia as the dominant risk factor for DS . Newly diagnosed diabetes at presentation occurred in 15% of our patients, lower than the 27% reported by Nadig et al., but within the spectrum described by Dubey et al. and international series.

Neuroimaging abnormalities were identified in 92.3% of our patients, predominantly contralateral putaminal T1 hyperintensity with frequent caudate involvement. While Nadig et al. reported striatal abnormalities in approximately 87% of patients, Dubey et al. ,with much larger cases emphasized that only 44% of patients in their broader movement-disorder cohort had MRI changes, underscoring the phenomenon of clinically isolated DS.

Another interesting finding in our study was the predominance of right striatopathy with left hemichorea Ballismus.Even in those with bilateral movements the severity of left hemichorea was more significant and had corresponding right striatopathy alone [92 % with right striatopathy] .A similar side predilection was noted by Nadig et al ,whereas side of lesion was not mentioned by Dubey et al . Although several other series have reported a predominance of left-sided hemichorea [2] , there is no established biological explanation. Reviews have suggested that hemispheric vulnerability arising from metabolic or dopaminergic asymmetry may contribute; however, this remains speculative and may be a reporting bias arising from smaller sample sizes in most series.

Clinical outcomes were favorable, with a mean time to improvement of 3.3 days-faster than the 6.3 days reported by Nadig et al. At six months, residual chorea persisted in 30.8% of our patients, and relapse occurred in 15%, closely mirroring recurrence rates (15–20%) described by Dubey et al. and other international literature [4].Interestingly two of our patients who ended up having chronic chorea ,when re-imaged showed non homogenous T2 FLAIR hyperintensities in bilateral putamen and caudate suggesting microvascular injury .These cases strengthens the “double hit hypothesis” regarding diabetic striatopathy resulting from an acute metabolic insult on top of preexisting striatopathy .[16] ,[17].

CONCLUSION

Diabetic striatopathy is an important, potentially reversible neurological complication of poorly controlled type 2 diabetes mellitus, predominantly affecting the elderly. In this prospective case series from Tamil Nadu, DS most commonly presented as acute hemichorea–hemiballismus in the setting of severe non-ketotic hyperglycemia, with characteristic striatal neuroimaging abnormalities in the majority of patients. Our cohort demonstrated a striking male predominance and a right-sided striatal predilection, contributing to the evolving understanding of the demographic and radiological heterogeneity of this entity.

Prompt metabolic correction resulted in rapid clinical improvement in all patients, highlighting the reversibility of the acute phase. However, a substantial proportion developed residual or recurrent chorea, particularly in association with poor glycemic control and imaging features suggestive of microvascular injury, supporting a “double-hit” pathophysiological mechanism. Increased awareness of diabetic striatopathy among clinicians is essential to facilitate early diagnosis, avoid unnecessary investigations, and prevent long-term neurological sequelae through aggressive metabolic management and follow-up.

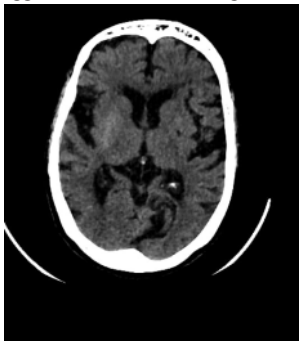

Figure 1. CT brain image of a 70 years old female with acute left

hemichorea hemiballismus showing right striatal hyperdensity.
Source : department of radiodiagnosis DSMC ,Siruvachur

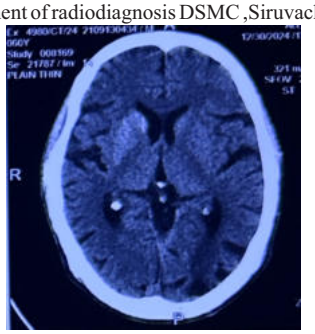

Figure 2 CT brain plain of 61 years male with bilateral chorea but left > right ,showning right striatal hyper density. Incidental chronic infarct in left MCA territory noted.
Source : department of radiodiagnosis DSMC ,Siruvachur

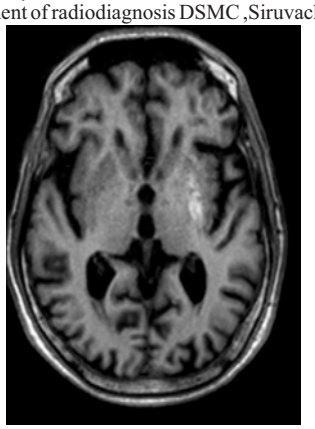

Figure 3: MRI brain showing T1 hyperintense lesion in left putamen in an 82 years male with right hemichorea hemiballismus . Source : department of radiodiagnosis DSMC ,Siruvachur

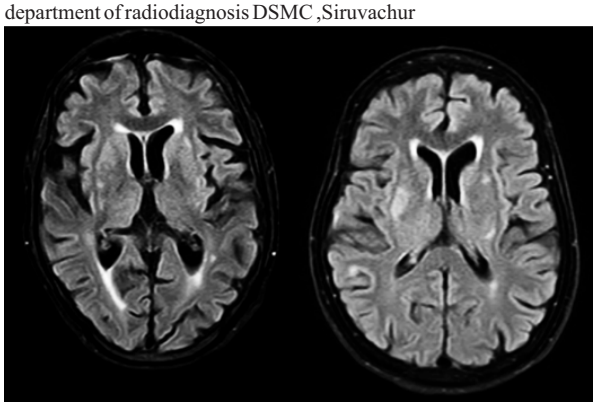

Figure 4,5: MRI brain images of the two patients who developed chronic chorea showing small vessel ischemic changes in bilateral striatum . Source : department of radiodiagnosis DSMC ,Siruvachur

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