departments, insufficient support from local bodies, family preference for private facilities hindering cooperation.

The National Medical Commission has mandated FAP for medical students from the first year and continued throughout the curriculum. The majority of colleges preferred to divide students into teams and rotate them with the other preclinical departments. Since the NMC criteria states that the villages adopted have to be beyond the field practice area, colleges may have difficulty in complying to the 5 family per student norm.

Arumugan *et al*.[5] posted the entire batch for FAP wherein students were divided into 3 batches which was further divided into smaller teams. Prior sensitization was carried out for the students, faculty, paramedical staff, etc. in all the medical colleges in the current study, which was comparable to the study by Langde *et al*.[6] and Arumugan *et al*.[5] As suggested by Vanikar *et al*.[2] it is essential to introduce FAP to the villagers and the stakeholders by addressing Gram Sabha's to sensitize the villagers and gain their confidence and acceptance.

Vanikar *et al*.[2] recommended that at least 10 visits must be planned in the first professional year which was followed by 36 (72%) of the colleges. They also insist that students must be trained to interact with the families prior to the community exposure. There should be at least one ASHA worker with every 25 students who will be entrusted with the responsibility of training students to facilitate interaction with the families and gain their confidence.[2]

We observed that the major hurdles encountered in the smooth implementation of the program were mainly due to lack of transport and logistics, shortage of manpower, language barrier, lack of interest and clinical knowledge in the students. Similar concerns were reported by Yalamanchali *et al*.[7] and Langde *et al*.[6] It was apparent that proper planning, coordination and support was crucial to facilitate smooth implementation of the programme, this was comparable to the views expressed by Langde *et al*.[6]

Conflict of Interest. None declared.

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Movement disorders in diabetes mellitus: Our observations

We have read with interest the case report by Nahid *et al*.,[1] regarding diabetic striatopathy (DS) in an adult with ketotic hyperglycaemia. We commend the authors for their contribution and wish to elucidate several vital aspects.

1. Contrary to the authors' depiction of DS as a rare and 'life-threatening manifestation' of diabetes, we contend that the condition's relative rarity and under-recognition stem from anecdotal evidence such as case reports/series and a handful of retrospective studies.[2–5] Thus, comprehensive analyses on long-term prognosis, recurrence, the emergence of comorbid conditions, and mortality trends are pending.[2,3] From our observations, while DS affects the quality of life if untreated or mismanaged, fatalities specifically attributed to DS have not been reported.
2. In defining DS, the authors align with Chua *et al*. (2020).[5] However, a newer and broadly accepted hypothesis by Dubey *et al*. (2022)[6] encompasses symptomatic DS, clinically isolated DS, and radiologically isolated DS, challenging the necessity of striatal hyperintensity on T1-weighted magnetic resonance imaging (MRI) and contralateral movement disorder as definitive criteria.[6] This underscores the prevalence of clinical–radiological discordance and indicates that diagnosis extends beyond mere blood analysis and neuroimaging findings.[6]
3. The report does not address the lack of awareness among neurologists—and by extension, general physicians and endocrinologists—regarding the spectrum of acute onset *de novo* non-choreoballistic movement disorders associated with DS, which are as prevalent as the classically described hemichorea–hemiballism.[2–10] This oversight by Nahid *et al*.[1] underscores the need for broader recognition of DS's neurological manifestations.
4. Although the authors correctly identify poorly controlled chronic glycaemia as a pivotal risk factor for DS and related movement disorders,[1] this does not account for the instances where rapid correction of hyperglycaemia precipitates or exacerbates movement disorders.[2,3] This suggests that both chronic hyperglycaemia and acute glycaemic fluctuations, potentially due to disruption of the basal ganglia circuitry and failure of striatopallidal blood flow autoregulation, contribute to the pathophysiology of DS.[6]
5. We question Nahid *et al*.'s interpretation[1] of T1 hyperintensity in Figure 2 of their manuscript, proposing instead that the imaging might depict a T2-weighted fluid-attenuated inversion recovery (FLAIR) sequence. The distinction between T2-FLAIR and T1-weighted images, particularly in cerebrospinal fluid darkness and grey–white matter contrast, is critical.[11] Furthermore, we challenge the purported clinical–radiological corroborative intensity changes over the right striatum.[6]

Hyperglycaemia-induced movement disorders often demonstrate remarkable reversibility with correction of hyperglycaemia alone.[2–10] We aim to amplify the imperative for clinicians to employ a broader differential diagnosis approach for abnormal limb movements in patients with DS, emphasizing the criticality of precise movement semiology identification, stringent and prompt blood glucose management, and the utilization of appropriate neuroimaging for expedited diagnosis and prevention of complications. Such diligence can facilitate early diagnosis, circumvent severe complications, reduce unnecessary expenditures, and prevent diagnostic and therapeutic errors.

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