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Original Article

Radiology Section

Aetiologies of Subcortical T2 Hypointensity on MRI in Patients Presenting with Seizures or Visual Symptoms: A Cross-sectional Study

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ABSTRACT

Introduction: Magnetic Resonance Imaging (MRI) findings in seizures associated with hyperglycaemia, particularly non ketotic hyperglycaemia, are distinctive and can aid diagnosis. Characteristic features include focal subcortical T2-weighted hypointensity, with or without cortical hyperintensity, reflecting underlying metabolic disturbances.

Aim: To analyse patients presenting with seizures or visual symptoms who demonstrate subcortical T2 hypointensity on MRI and by correlating these radiological patterns with clinical and laboratory findings.

Materials and Methods: This cross-sectional study was conducted in the Department of Radiodiagnosis, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India, between 2020 and February June 2022. Patients presenting with seizures and/or visual disturbances who demonstrated focal subcortical T2 hypointensity on MRI were included. Imaging was performed on 1.5T and 3T scanners. Sequences analysed included T2, Fluid Attenuated Inversion Recovery (FLAIR), Diffusion Weighted Imaging (DWI), Apparent Diffusion Coefficient (ADC), and Susceptibility Weighted Imaging/Gradient Echo (SWI/GRE).

Results: Twenty-two patients were evaluated. Hyperglycaemia was confirmed in nine patients (serum glucose 384-520 mg/dL; HbA1c 7.1-14%). MRI consistently showed subcortical hypointensity on T2-weighted, FLAIR, and SWI/GRE sequences, with corresponding reduced signal on DWI. No haemorrhage was detected. The parieto-occipital lobes were predominantly affected (77.8%), while frontal and temporal lobes were less frequently involved (22.2%). One patient demonstrated complete resolution of abnormalities on follow-up after glycaemic control. The remaining 13 patients had alternative diagnoses, including infarcts, meningitis, metastases, cerebritis, and Sturge-Weber syndrome.

Conclusion: Subcortical T2 hypointensity, especially in the parieto-occipital regions, is a subtle but reliable MRI marker of hyperglycaemia-related seizures. Recognition of this finding facilitates accurate diagnosis, prevents misinterpretation as stroke or infection, and enables timely initiation of metabolic correction. Isolated subcortical T2 hypointensity in the parieto-occipital regions should alert radiologists to hyperglycaemia as an underlying cause of seizures or visual symptoms, allowing timely metabolic correction and complete reversibility of MRI findings.

Keywords: Diabetes mellitus complications, Magnetic resonance imaging, Parieto-occipital hypointensity

INTRODUCTION

Subcortical T2-weighted hypointensity on MRI, particularly in the parieto-occipital regions, represents a subtle but clinically significant finding that may be easily overlooked or misinterpreted. This imaging feature can mimic a variety of pathologies, including ischaemia, infection, and neoplasm, often leading to diagnostic uncertainty. In recent years, increasing evidence has associated this imaging abnormality with hyperglycaemia, particularly in the non ketotic state [1-3]. Hyperglycaemia-induced neurological dysfunction encompasses a wide clinical spectrum, ranging from confusion, focal deficits, and visual disturbances to seizures and hemichorea [4,5]. Among these, seizures and visual hallucinations—especially those arising from occipital lobe involvement—are increasingly recognised as characteristic manifestations of non ketotic hyperglycaemia [6,7]. The associated imaging abnormalities are diverse and may include cortical T2 hyperintensity, restricted diffusion, striatal signal alterations, or focal subcortical hypointensity [8-10].

Previous studies by Raghavendra S et al., and Seo DW et al., have described reversible subcortical hypointensity on T2-weighted and FLAIR sequences in patients with hyperglycaemia-related seizures [8,11]. Hiremath SB et al., further reported this feature in 83.3% of their cohort, suggesting its potential diagnostic value [3]. However, the pathophysiological basis remains incompletely understood. Proposed mechanisms include γ-Aminobutyric Acid (GABA) depletion leading to neuronal hyperexcitability, intracellular dehydration due to hyperosmolarity, and mineral deposition within the subcortical white matter [5,6]. Importantly, these lesions have been shown to reverse with appropriate glycaemic control, indicating that the changes are metabolic rather than structural [12,13]. Despite these observations, subcortical T2 hypointensity is not pathognomonic for hyperglycaemia and may also occur in conditions such as ischaemia, infection, and neoplasia [14,15]. Differentiating hyperglycaemia-related imaging changes from these mimics is critical, as management strategies differ substantially.

This diagnostic ambiguity underscores the need for comprehensive evaluation of subcortical T2 hypointensity within a broader clinical context. The present study aimed to address this gap by analysing patients presenting with seizures or visual symptoms who demonstrate subcortical T2 hypointensity on MRI and by correlating these radiological patterns with underlying clinical and laboratory findings. The novelty of this study lies in its comparative approach—evaluating hyperglycaemia-induced subcortical hypointensity alongside other causes such as infarction, infection, neoplasia, and congenital syndromes. By delineating the imaging and clinical differences among these entities, this study seeks to enhance diagnostic accuracy, improve recognition of hyperglycaemia-related changes, and prevent misinterpretation of this subtle but distinctive MRI finding.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Radiodiagnosis, Mahatma Gandhi Medical College and Hospital, Jaipur, Rajasthan, India, from June 2020 to February 2022. The study was approved by the Institutional Ethics Committee

(IEC No.: /MGMC&H/IEC/JPR/4988), and written informed consent was obtained from all participants prior to inclusion.

Inclusion criteria: Patients presenting with seizures and/or visual disturbances who demonstrated focal subcortical T2 hypointensity on MRI were included in the study.

Exclusion criteria: Patients with known causes of subcortical hypointensity, such as calcification, haemorrhage, neoplasm, infection, or prior infarction, were excluded from the study.

Sample size estimation: A total of 22 patients fulfilling the inclusion criteria were enrolled during the study period. The sample size was based on the number of consecutive eligible patients presenting within the defined time frame.

Study Procedure

All MRI examinations were performed on 1.5T and 3T scanners using a standardised brain protocol. The following sequences were analysed: T2-weighted, FLAIR, DWI, ADC maps, and SWI or GRE sequences.

Clinical and laboratory correlation was performed in all cases, including blood glucose and HbA1c estimation. Cerebrospinal fluid (CSF) analysis was obtained when clinically indicated.

The following imaging and clinical parameters were evaluated:

- Distribution and laterality of subcortical T2 hypointensity;
- Associated cortical and subcortical signal alterations on other MRI sequences;
- Presence of diffusion restriction or susceptibility effects;
- Correlation with clinical and metabolic parameters, including blood glucose and HbA1c levels.

STATISTICAL ANALYSIS

Data were compiled and analysed using Microsoft Excel (Microsoft 365, Microsoft Corp., Redmond, WA, USA) for descriptive statistical analysis. Results were expressed as frequencies and percentages.

RESULTS

During the study period, a total of 10,264 brain MRI examinations were performed, of which 1,196 patients presenting with seizures and/or visual disturbances were reviewed. Among these, 22 patients demonstrated focal subcortical T2 hypointensity and fulfilled the inclusion criteria. Of the 22 patients, 15 presented with seizures, four with visual disturbances, and three with both symptoms.

Hyperglycaemia was identified as the underlying cause in nine patients (40.9%), confirmed through a combination of radiological, clinical, and laboratory findings. Among these, three patients had known type 2 diabetes mellitus, while six patients were newly diagnosed based on elevated serum glucose and HbA1c levels. All nine patients demonstrated poorly controlled diabetes, with serum glucose levels ranging from 384-520 mg/dL and HbA1c between 7.1-14%. Glycosuria without ketonuria was observed in eight of nine patients. One patient underwent EEG, which was within normal limits. Clinical and laboratory details of the hyperglycaemic patients are summarised in [Table/Fig-1].

In the hyperglycaemia group, subcortical T2 hypointensity was predominantly located in the parieto-occipital regions (n=7) and posterior temporal lobes (n=2) [Table/Fig-2]. These lesions typically showed absence of diffusion restriction and no associated enhancement or mass effect. The remaining 13 patients (59.1%) were diagnosed with alternative aetiologies, including acute infarcts (n=7), leptomeningeal metastases (n=2), diffuse meningitis (n=2), cerebritis (n=1), and Sturge–Weber syndrome (n=1).

In acute infarcts, subcortical T2 hypointensity was noted in the frontoparietal or middle cerebral artery territories, typically adjacent to cortical diffusion restriction with reduced ADC values, distinguishing them from hyperglycaemia-related cases.

Age (years)/ sex	Clinical presentation	Blood glucose (mg/dL)	HbA1c (mg/ dL)	Urinary investigations	
				Sugar	Ketones
30/M	Generalised Tonic–Clonic Seizures (GTCS), coloured vision, occasional blackout, and visual obscuration	384	14	+++	++
44/M	Transient visual obscuration and spotter in the eyes are associated with decreased vision and headache	407	10	+++	Nil
48/M	Right-sided weakness, transient visual obscuration, and headache	520	8.2	+++	Nil
64/F	Decreased vision and occasional blackouts and headache	375	7.4	++	Nil
48/M	Decreased vision, focal seizure, and headache	402	8.0	+++	Nil
46/M	Right-sided weakness of upper limb, focal seizures	365	7.1	++	Nil
51/M	Known case of multiple myeloma. Loss of vision in the left eye	500	9.2	+++	Nil
45/F	Focal seizures and headache	450	8.2	+++	Nil
55/M	Focal seizures and decreased vision	492	8.7	+++	Nil

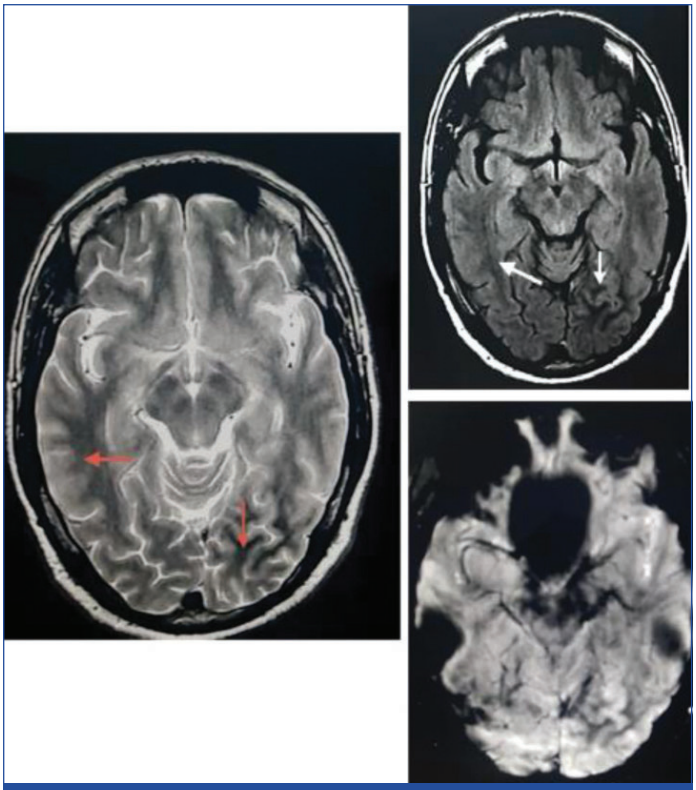
[Table/Fig-1]: Clinical and laboratory characteristics of hyperglycaemic patients with seizures.

Patients	Location involved	Subcortical white matter		
		T2/Flair	DWI	SWAN
1	Right parieto-occipital	Hypo	Isointense	Hypointense
2	Bilateral parieto-occipital	Hypo	Isointense	Hypointense
3	Left parieto-occipital	Hypo	Isointense	Hypointense
4	Left temporo-parieto-occipital	Hypo	Isointense	Hypointense
5	Left perirolandic region	Hypo	Isointense	Hypointense
6	Left parieto-occipital lobe	Hypo	Isointense	Hypointense
7	Left parieto-occipital lobe and precentral gyrus	Hypo	Isointense	Hypointense
8	Bilateral parieto-occipital lobe	Hypo	Isointense	Hypointense
9	Right parieto-occipital lobe	Hypo	Isointense	Hypointense

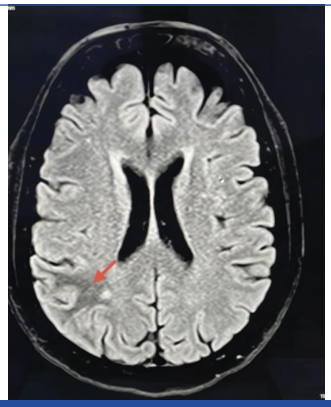
[Table/Fig-2]: MRI findings in all nine hyperglycaemic patients.

Leptomeningeal metastases and meningitis demonstrated leptomeningeal or pachymeningeal enhancement along with T2-weighted hypointensity and adjacent parenchymal oedema, features absent in the hyperglycaemia group. The cerebritis case showed patchy cortical-subcortical hyperintensity with restricted diffusion and contrast enhancement. In Sturge–Weber syndrome, subcortical T2 hypointensity was observed beneath gyriform calcifications in the occipital lobe, consistent with chronic venous stasis. CSF analysis confirmed viral meningoencephalitis in the cerebritis case, tuberculous meningitis in one patient, and viral meningitis in another.

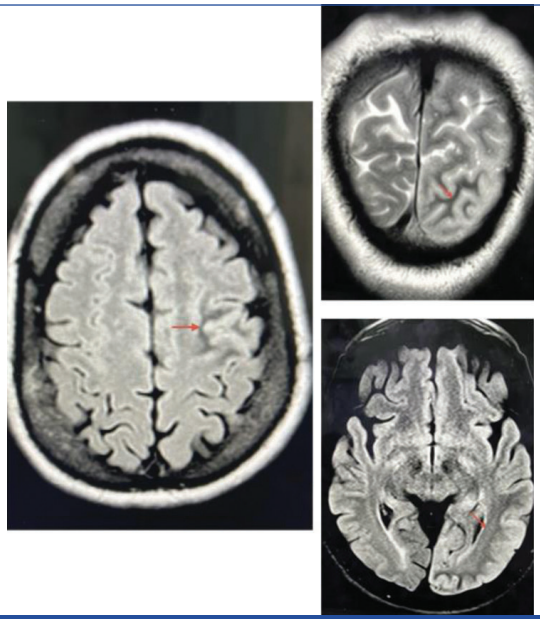
In the nine hyperglycaemic patients, MRI demonstrated subcortical hypointensity on T2-weighted and FLAIR sequences, consistently corresponding with hypointensity on SWI/GE. DWI showed reduced signal intensity in the same regions. No susceptibility-related blooming or haemorrhage was noted. Parieto-occipital lobes were involved in seven patients (77.8%) [Table/Fig-3-5], while the frontal and temporal lobes were each affected in two patients (22.2%) [Table/Fig-6]. Among the nine hyperglycaemic patients, five exhibited left-sided predominance of hypointensity, while four had right-sided predominance. Among the remaining 13 patients, infarcts were left-sided in four patients and right-sided in three; other pathologies were bilaterally present. Follow-up imaging in one patient showed complete resolution after glycaemic control [Table/Fig-7].



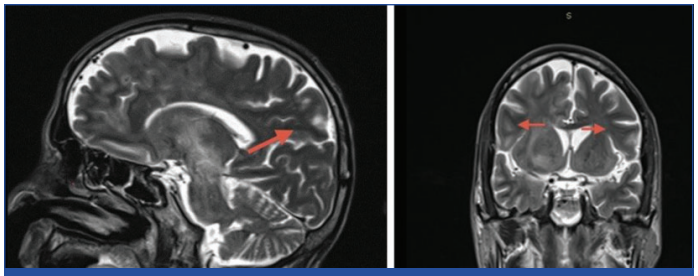
[Table/Fig-3]: MRI brain (axial T2-weighted image), FLAIR image and GRE image showing subcortical hypointensity in bilateral parieto-occipital lobes with no susceptibility/blooming artefact.



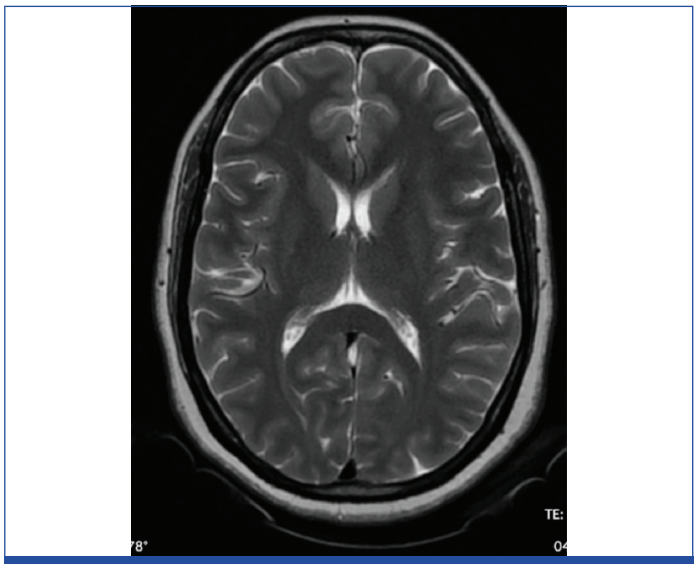
[Table/Fig-4]: MRI brain (axial FLAIR image) of another patient showing subcortical hypointensity in right parietal lobe.



[Table/Fig-5]: MRI brain (axial T2-weighted and FLAIR image taken at different levels) of a patient with newly diagnosed diabetes mellitus showing areas of T2 weighted hypointensity in left parieto-occipital regions.



[Table/Fig-6]: MRI brain (sagittal and coronal T2-weighted image) of a patient with viral meningoencephalitis showing areas of altered signal intensity in right thalamus, midbrain, pons and right capsuloganglionic area along with areas of T2 weighted hypointensity in right frontoparietal lobe.



[Table/Fig-7]: Follow-up MRI after glycaemic control demonstrating resolution of previously seen abnormalities.

DISCUSSION

Hyperglycaemia may present with a wide range of neurological manifestations, many of which are subtle and non specific. Occipital lobe seizures are particularly noteworthy, often accompanied by visual disturbances such as coloured or complex visual hallucinations, blurred vision, headache, confusion, and homonymous hemianopia [1,2].

In this study, one patient presented with generalised tonic–clonic seizures, two with focal seizures, four with isolated visual symptoms, and two with a combination of focal seizures and visual disturbances. These findings are consistent with earlier reports describing occipital lobe seizures and visual hallucinations as common presenting features of hyperglycaemia-related encephalopathy [Table/Fig-8] [3,8,11-13].

All hyperglycaemic patients demonstrated subcortical T2 hypointensity, a finding strongly associated with hyperglycaemia and well documented in prior studies [3,8,11-13]. Hiremath SB et al., reported subcortical hypointensity in 83.3% of hyperglycaemic patients [3], while Lee EJ et al., observed this feature in all their patients [13]. The posterior cerebral predominance observed in present cohort (77.8%) aligns with prior studies by Putta SL et al., and Goto H et al., which demonstrated predominant occipital lobe involvement in non ketotic hyperglycaemia, attributed to the selective regional susceptibility of the visual cortex to metabolic disturbances [14,15].

The pathophysiology remains incompletely understood. One hypothesis suggests that hyperglycaemia leads to GABA depletion, reducing inhibitory neurotransmission and lowering the seizure threshold, whereas ketone bodies in ketoacidosis may exert a protective effect by enhancing GABA synthesis [4]. Another proposed mechanism involves neuronal dehydration and altered GABA metabolism in hyperosmolar states, contributing to excitability [5]. Chronic hyperglycaemia itself may also have a direct proconvulsant effect [6].

Study (Year published)	Place of study	Objective of the study	Subcortical T2 SI	
			Hypointense	Normal
Seo DW et al., (2003) [11]	Department of Neurology, Samsung Medical Centre, Sungkyunkwan University School of Medicine, Seoul, South Korea.	To report cases of partial status epilepticus associated with NKH and characterise the MRI findings specifically transient subcortical T2/FLAIR hypointensity near seizure focusâ"and discuss possible mechanisms (free radicals/iron deposition.	3 (100)	0
Raghavendra S et al., (2007) [8]	Department of Neurology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, Kerala, India	To prospectively evaluate clinical and MRI abnormalities in patients with non ketotic hyperglycaemia (NKH) presenting with seizures, and identify characteristic imaging findings (specifically subcortical T2 hypointensity) in this context.	4 (100)	0
Chen CC et al., (2011) [12]	Taiwan	To prospectively evaluate the clinical and MRI abnormalities in six patients with NKH complicated by simple or complex partial seizures, focusing on imaging features such as subcortical T2 hypointensity, contrast enhancement, restricted diffusion, and spectroscopy findings.	5 (83.3)	1 (16.7)
Lee EJ et al., (2016) [13]	Department of Radiology (and Neurology) at Dongguk University Ilsan Hospital, Goyang-shi, South Korea	To describe the characteristic MRI abnormalities in hyperglycaemia-induced seizures and to evaluate the diagnostic value of contrast-enhanced FLAIR imaging in identifying these changes.	11 (100)	0
Hiremath SB et al., (2019) [3]	Department of Radiodiagnosis, Pushpagiri Institute of Medical Sciences and Research Centre, Tiruvalla, Kerala, India; and Department of Radiodiagnosis, Krishna Institute of Medical Sciences, Karad, Maharashtra, India; (with one author affiliated with The Ottawa Hospital, Ottawa, Ontario, Canada)	To highlight the typical Magnetic Resonance Imaging (MRI) findings in hyperglycaemia-induced seizures and compare the results with similar previous studies with a brief mention of pathophysiological mechanisms.	5 (83.3)	1 (16.7)
Gupta S et al., 2026	Department of Radiodiagnosis, Mahatma Gandhi Medical college and Hospital, Jaipur, Rajasthan, India	The present study aims to address this gap by analysing patients presenting with seizures or visual symptoms who demonstrate subcortical T2 hypointensity on MRI, and by correlating these radiological patterns with underlying clinical and laboratory findings.	9 (40.90)	13 (59.10)

[Table/Fig-8]: Summary of published studies describing subcortical T2 hypointensity in hyperglycemia-related seizures [3,8,11-13].

The subcortical T2 hypointensity observed in these patients likely reflects transient metabolic derangements, including excitotoxic injury, intracellular dehydration, or mineral deposition. Sasaki F et al., demonstrated T2 hypointensity in the occipital subcortex of a patient with non ketotic hyperglycaemia, supporting the iron deposition hypothesis [7]. The reversibility of these changes on follow-up imaging further supports a functional rather than structural basis. Other associated imaging features may include cortical T2 hyperintensity, striatal involvement, and leptomeningeal enhancement [8]. PET and MR spectroscopy studies have demonstrated cortical hypermetabolism and altered metabolite ratios, respectively [9,10]. Iron deposition has also been suggested as a contributor to T2 hypointensity [11].

However, it is crucial to recognise that subcortical T2 hypointensity is not specific to hyperglycaemia. In this study, 13 of 22 patients were diagnosed with alternative aetiologies, including acute infarcts (n=7), leptomeningeal metastases (n=2), diffuse meningitis (n=2), cerebritis (n=1), and Sturge–Weber syndrome (n=1). Therefore, while posterior-predominant subcortical T2 hypointensity in a patient with uncontrolled hyperglycaemia and seizures or visual symptoms serves as a strong diagnostic clue, it is not pathognomonic. Similar imaging findings may also occur in vascular, infectious, neoplastic, and other metabolic conditions, each with distinct underlying mechanisms.

In acute ischaemic infarcts, subcortical T2 hypointensity may be observed adjacent to the infarcted cortex, particularly in the frontoparietal or middle cerebral artery territories. The pathophysiologic basis involves acute cytotoxic Oedema, which leads to restricted diffusion and increased intracellular water, potentially causing magnetic susceptibility changes and signal loss on T2-weighted or SWI sequences. Additionally, deoxyhaemoglobin accumulation from sluggish venous drainage in ischaemic tissue may contribute to the observed hypointensity. The presence of restricted diffusion, vascular territorial distribution, and subsequent encephalomalacia helps differentiate infarction from hyperglycaemia-related lesions, which typically lack diffusion restriction and resolve with glycaemic control [16].

In infectious meningoencephalitis, subcortical T2 hypointensity is less common but may occur due to inflammatory infiltration, microvascular congestion, and paramagnetic free radical accumulation within the affected tissue [17]. Bacterial or tuberculous meningitis may show meningeal enhancement and adjacent parenchymal Oedema, sometimes accompanied by restricted diffusion or CSF abnormalities. In viral cerebritis, neuronal necrosis and microhaemorrhages can cause focal hypointensity on SWI or T2 images [18]. In this study, patients with meningitis and cerebritis demonstrated meningeal enhancement and CSF evidence of infection, features not observed in hyperglycaemia-related lesions.

In leptomeningeal metastases, subcortical T2 hypointensity may result from tumour cell infiltration of the leptomeninges, leading to secondary venous congestion, parenchymal Oedema, and microhaemorrhages. The hypointensity is often accompanied by leptomeningeal enhancement and may involve multiple cortical regions. Pathophysiologically, tumour-induced microangiopathy and local hypoxia contribute to susceptibility effects [19,20]. Contrast enhancement and multifocal distribution distinguish these cases from the typically non enhancing, posteriorly located lesions of hyperglycaemia.

In Sturge-Weber syndrome, subcortical T2 hypointensity represents the deposition of paramagnetic minerals (iron and calcium) secondary to chronic venous stasis beneath leptomeningeal angiomatosis. This results in gyriform calcifications with adjacent white matter volume loss and signal drop-out on T2 and SWI images. Unlike hyperglycaemia, which causes reversible metabolic changes, these hypointense regions in Sturge-Weber syndrome are chronic and irreversible [21].

Clinical context, laboratory data (including blood glucose and CSF analysis), and diffusion-weighted or contrast-enhanced MRI findings play a key role in distinguishing these conditions. Radiologists should consider hyperglycaemia when encountering isolated subcortical T2 hypointensity, particularly in the parieto-occipital regions, in patients presenting with seizures or visual symptoms. Correlation with serum glucose and ketone status is essential to avoid misdiagnosis and unnecessary invasive testing. Prompt recognition can lead to complete reversibility of MRI abnormalities with appropriate glycaemic control.

Limitation(s)

This study was limited by its small sample size, the lack of uniform contrast-enhanced MRI in all patients, and heterogeneity in imaging data, which restricted the evaluation of certain imaging patterns. A larger multicentric cohort with serial follow-up imaging could further validate these observations.

CONCLUSION(S)

Subcortical T2-weighted hypointensity, particularly in the parieto-occipital regions, is a subtle but valuable imaging feature in patients with hyperglycaemia. When observed in isolation without structural abnormalities, it strongly suggests a hyperglycaemia-related Aetiology. Recognition of this feature is crucial for distinguishing hyperglycaemia from mimics such as ischaemia, encephalitis, meningitis, and metastasis. Early identification enables timely metabolic correction, prevents unnecessary interventions, and improves patient outcomes.

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