Hyperglycemia-induced seizures and blindness

Sir,

We read with great interest the article titled "Hyperglycemia-induced seizures-Understanding the clinico-radiological association" by Hiremath *et al*. in the October-December 2019 issue of Indian Journal of Radiology and Imaging.[1] The article is highly informative and describes the mechanism and imaging manifestations of hyperglycemia-induced seizures. In this article, we describe a similar recent case with imaging on admission and follow-up images.

A 28-year-old woman, known case of exogenous Cushing's disease, presented to the emergency services with three episodes of generalized tonic-clonic seizures, followed by

blindness. Random blood glucose was 612 mg/dL. HbA1c levels were performed with a value of 8.6%. Urine was negative for ketones. Blood pressure was 110/76 mmHg, without any history of fever, and a negative vasculitic workup. Fundoscopic examination revealed no significant abnormality, raising the possibility of cortical blindness. MRI of the brain was performed, which revealed multifocal areas of cortical hyperintensity on T2 Weighted Images and corresponding restricted diffusion in bilateral parietal, occipital, and temporal lobes was seen. There was relative sparing of the frontal lobes. However, unlike prior studies, subcortical hypointensity was not seen. There was no restricted diffusion in the subcortical white matter, and the subcortical white matter did not show any susceptibility on susceptibility weighted imaging (SWI). Basal ganglia were normal. Imaging differentials at this point were posterior reversible encephalopathy syndrome (PRES), post ictal changes, and hyperglycemia-induced changes. PRES was ruled out due to restricted diffusion in the cortex, and absence of appropriate clinical findings. History of three episodes of seizures without any status epilepticus ruled out post ictal changes. Based on above imaging findings, a possibility of hyperglycemia-induced seizures and associated cortical blindness was raised. Follow-up imaging was recommended and performed after adequate control of blood glucose. Follow-up MRI after 10 days, when blindness persisted, showed significant resolution of the cortical restricted diffusion and hyperintensity. Even at this point, no T2/SWI hypointensity was seen in the subcortical white matter. Thus, a final diagnosis of hyperglycemia-induced seizures and associated cortical blindness was made. Recent studies,[2] including the article in your journal[1] have demonstrated cortical T2 hyperintensity with restricted diffusion and subcortical T2 hypointensity, predominantly in the occipital lobes. Our case showed similar findings [Figure 1], barring the subcortical T2 hypointensity, which is considered specific for hyperglycemia.[3] The pathophysiology behind this T2 hypointensity is unclear; however, the most reliable evidence states it to be due to the transient accumulation of free radicals and iron, which is secondary to cortical ischemia.[4] This finding is transient, which could be the possible explanation why it was not present in our case, as the MRI was performed 9 h after the event. The fact that the findings resolved with control of blood glucose levels, with cortical blindness points toward hyperglycemia as the etiology. Occipital lobe seizures have been reported with hyperglycemia,[5] which was also present in our case. Other minor imaging findings which have also been described include post gadolinium enhancement of the involved area. Other neurologic presentations of hyperglycemia include hemiballismus-hemichorea, which appears on imaging as T1 hyperintensity in the contralateral corpus striatum.

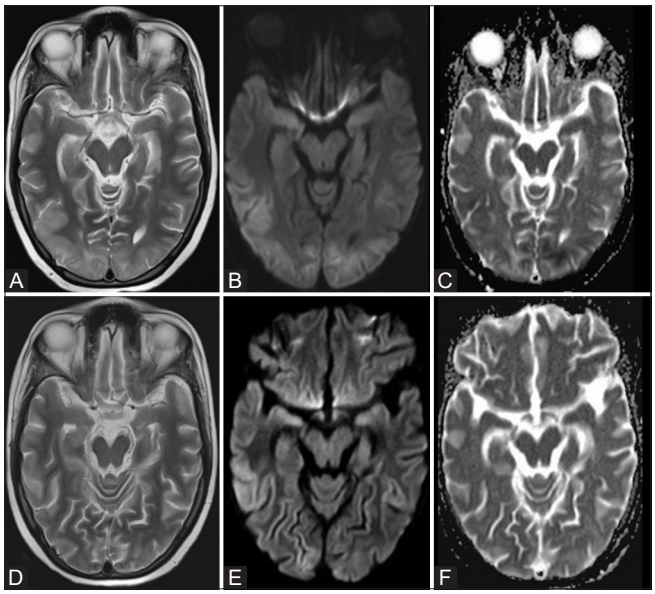

Figure 1 (A-F): (A-C) Images of the MR Brain on admission. (D-F) Follow-up MR images after 10 days. (A) T2 axial images showing cortical thickening with symmetric subcortical hyperintensities in the bilateral temporo-occipital lobes. (B) Diffusion weighted images showing cortical restricted diffusion in affected areas. (C) Corresponding Apparent Diffusion Coefficient (ADC) maps showing low values. (D) T2 axial images showing significant reduction in the cortical thickening. (E and F) DWI and ADC, respectively, showing near-complete resolution of the previously seen findings

Hence, we see that hyperglycemia can present with occipital lobe seizures, cortical blindness and imaging findings are variable, with predominant involvement of the cortex and subcortical white matter. In the appropriate clinical setting, parieto-occipital/occipital cortical hyperintensity should alert suspicion for hyperglycemia-induced changes.

Integration of clinical data with imaging findings and follow-up imaging are helpful in arriving at a definitive diagnosis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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