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RESEARCH ARTICLE
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SUBCORTICAL T2 HYPOINTENSITY IN SEIZURES WITH A NONKETOTIC HYPERGLYCEMIC HYPEROSMOLAR STATE

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ABSTRACT:
Imaging features in seizures associated with a nonketotic hyperglycemia (NKH). A elderly female, known case of type 2 diabetes mellitus, presents with involuntary movements in left upper limb since 3 days. The MRI results revealed the presence of subcortical T2 hypo-intensity in the parieto-occipital white matter, along with cortical enhancement. Areas of limited diffusion were visible using Diffusion Weighted Imaging (DWI). Encephalitis, cancer, and hemorrhagic infarct were among the radiologic differential diagnoses that were first evaluated, creating a difficult diagnostic situation.

BACKGROUND:NKH is a rather typical consequence of type 2 diabetes, particularly in people who are 50 or older. T2 hyper intensity has been reported to follow seizures on several occasions. Our patient, however, had localized T2 hypo intensity that exhibited strong clinic electrographic correlation.

CASE STUDY:-48year-old women arrived to our health care center with complaints of involuntary movements 3 days back. She developed involuntary movements of left upper limb, which was insidious in onset, progressive, 6-7 episodes per day. It was not associated with loss of consciousness, vomiting, blurring of vision. She also complaints of right sided headache. Patient is a known of type 2 diabetes mellitus, not on any medications. Patient is also known case of hypertension.

INVESTIGATIONS: Tests for liver function, kidney function, and complete blood count were all normal. Patient did not have ketonuria.
Fasting blood sugar – 302 mg/dl
Postprandial blood sugar -505 mg/dl
HbA1C -14.8%
Serum Osmolality -294 mOsmol/l
EEG – Normal awake record

MRI -MRI study of the brain was performed for further evaluation. The MRI revealed normal T1 -W imaging. Right parietooccipital white matter has focal subcortical T2 hypo intensity on T2-W and FLAIR imaging, covering hyper intense cortex. DWI research revealed concentrated regions of constrained diffusion .On contrast examination, focal cortical contrast enhancement of overlying cortex was noted. (Fig 1to 3)

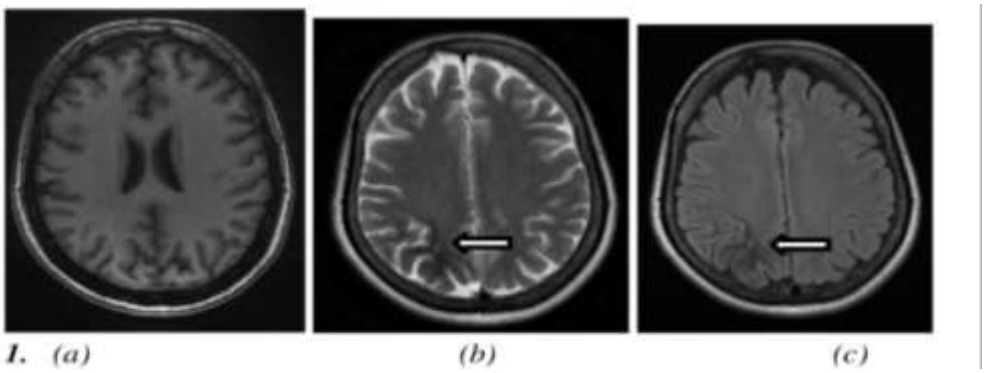

Figure 1.(a) Normal T1-W imaging ,(b) T2-W ()FLAIR images shows subcortical T2 hypointensity in right parietooccipital white matter

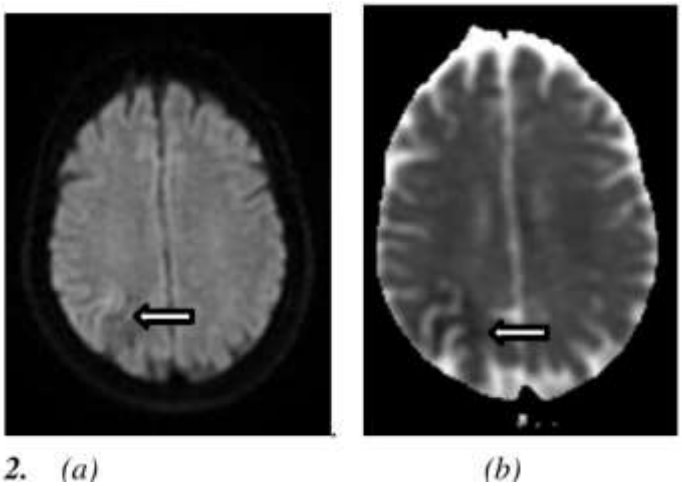

Figure 2. (a) DWI and (b)ADC map show the area of restricted diffusion

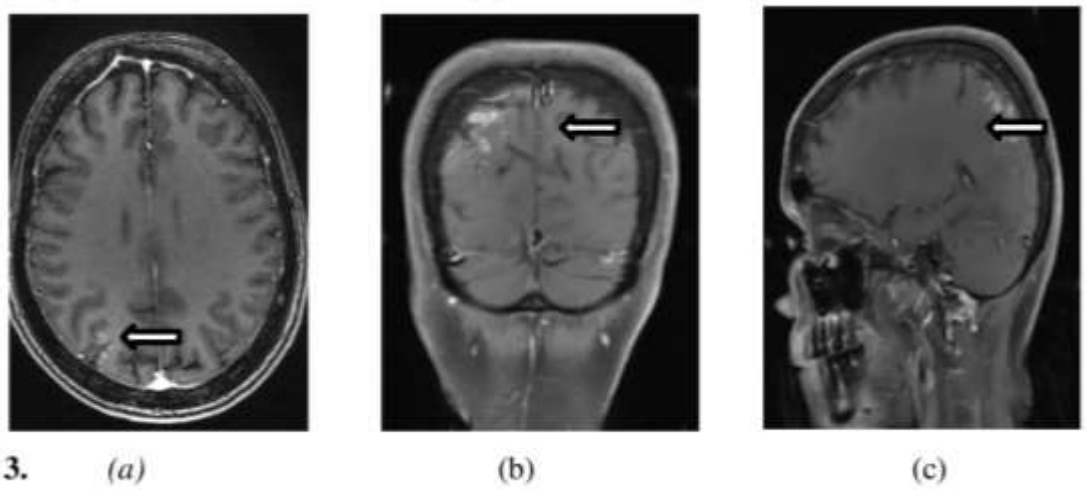

Figure 3. T1 post contrast images in (a)axial ,(b)coronal and (c) sagittal images show overlying cortical enhancement in posterior parietal regi

DIFFERENTIAL DIAGNOSIS:

Encephalitis, cancer, and hemorrhagic infarct were among the radiologic differential diagnoses that were first evaluated, creating a difficult diagnostic situation.

The following are some reasons of T2 hypointensity: Viral encephalitis, meningitis, leptomeningeal metastases, hemorrhagic infarct, and hypoxia insult are characteristics of acute presentation.

Multiple sclerosis, Sturge-Weber syndrome, Moya Moya disorder , tuberous sclerosis, and hemimegalencephaly are characteristics of the chronic presentation.

TIP: Do not list diagnosis, please. We are interested in learning how the final diagnosis was determined. This area has to be extensively explored because it is frequently the most crucial. All valid diagnosis must be supported.

TREATMENT:

TIP: Include both pharmaceutical and non-pharmacological treatments, such as supportive care, exercises, and operations.

RESULT and FOLLOW UP:

Participant was monitored until complaints subsided and the MRI abnormalities disappeared.There was a minimum of six months of follow-up available.During the follow-up, her diabatic state was evaluated and tracked.

Declaration -

- Ethics approval and consent to participate – Patients consent was taken completely.
- Consent for publication – All authors give permission for the publication
- Availability of data and materials – Data was made available by the corresponding author.
- Competing interests – No competing interest was recorded.
- Funding – There is no funding for the research.
- Authors' contributions - 1. Dr. Adarsh B Hampole : Conceptualization, formal analysis, resources,

DISCUSSION:

NKH is a rather typical consequence of type 2 diabetes, particularly in people who are 50 or older. Participant first experienced a focal seizure. Another crucial medical indicator of NKH is a reversible field deficit. Multiple reports of T2 hyper intensity following seizures exist. [1-3]. The individual we treated had localized T2 hypo intensity that was well correlated on the clinic electrographic scan. Recent research has linked seizures with NKH to subcortical T2 hypo intensity. Other clinical situations such viral encephalitis, meningitis, hemorrhagic infarct, leptomeningeal metastases, and hypoxia also elicited T2 hypo intensities on MRI. In chronic conditions such Sturge-Weber syndrome, tuberous sclerosis, multiple sclerosis, moya-moya illness, and hemimegalencephaly, subcortical hypo intensity can also arise, although it does not resolve quickly. Uncertainty surrounds the true pathophysiology of this momentary subcortical hypointensity. In this patient, the DWI revealed constrained diffusion, which supported cytotoxic edoema. Seizures, localised ischemia, or hyperviscosity may all be contributing factors to the focal cytotoxic edoema. Because the subcortical abnormalities were temporary, it is possible that the original cortical illness was the cause. [4]. The buildup of transitory free radicals in the subcortex as a result of excitotoxic axonal damage during seizures is one potential mechanism. The occipital and nearby regions were the only ones to have medical and imaging alterations. Similar tendencies have also been seen recently.

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