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NEUROIMAGING IN DIABETIC PATIENTS IN ACUTE EMERGENCY SETTING : IMPLICATIONS FOR HYPERGLYCEMIA AND HYPOGLYCEMIA

Radio-Diagnosis

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ABSTRACT

Diabetes mellitus is a leading global health concern, with its prevalence rising alongside increasing urbanization, sedentary lifestyles, and poor dietary habits. As one of the major metabolic disorders, diabetes is associated with both chronic and acute complications. Among the acute complications, significant alterations in blood glucose levels—specifically hyperglycemia and hypoglycemia—can lead to neurocognitive dysfunction and neurological emergencies. In emergency settings, patients with extreme fluctuations in blood glucose levels often present with altered mental status, seizures, or focal neurological deficits, prompting immediate medical intervention. Neuroimaging plays a crucial role in diagnosing and differentiating the underlying cause of neurological symptoms, as well as in guiding therapeutic strategies. It was a prospective study done at a tertiary care center in southern Rajasthan in a duration of 1 year between Jan 2024- Jan 2025. We collected three cases/patients who presented with glucose level abnormalities like uncontrolled hyperglycemia or severe hypoglycemia in emergency at our centre and underwent MRI brain. MRI brain findings and relevant clinical and lab data were noted. Neuroimaging is an indispensable tool in the evaluation of diabetic patients presenting with acute neurological symptoms in emergency settings, especially those related to extreme fluctuations in blood glucose. Both hyperglycemia and hypoglycemia can lead to significant cerebral consequences, and timely neuroimaging can help clinicians distinguish between glucose-related pathophysiological changes and other more serious neurological conditions. Neuroimaging aids in identification of the cause of encephalopathy and helpsus to differentiate hypoglycemic/hyperglycemic encephalopathy from hypoxic ischemic encephalopathy or acute hypertensive encephalopathy or infarct.

KEYWORDS

Non-ketotic hyperglycemia, acute hypoglycemic encephalopathy, Neuroimaging, hyperglycemic hyperosmolar state, Hypoxic ischemic encephalopathy.

INTRODUCTION:

The brain is a glucose glutton, consuming more than half of body's total glucose. Because the brain does not store excess as glycogen, CNS function is highly dependent on a steady, continuous supply of blood glucose.

Glucose must be actively transported across blood brain barrier that occurs with the help of a transporter protein known as GLUT-1.

Both hyper and hypoglycemic can cause brain injury with hyperglycemia being relatively more common cause. 1

There may be various neurological manifestations due to deranged glucose metabolism ranging from mild, reversible focal deficits to status epilepticus, coma and death.

Hyperglycemia And Hypoglycemia In Diabetic Patients:-

1. Hyperglycemia

In diabetic patients, hyperglycemia, especially in the context of diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state (HHS), can lead to severe neurological manifestations. Symptoms of severe hyperglycemia may include confusion, lethargy, stupor, or coma. The elevated glucose levels, combined with dehydration, electrolyte imbalances, and acidosis in DKA or HHS,exacerbate cerebral edema, causing neurological deterioration.

2. Hypoglycemia

On the other hand, hypoglycemia (typically defined as a blood glucose level of less than 70 mg/dL) can also cause acute neurological symptoms, ranging from irritability, confusion, and agitation to seizures or even coma. The brain is highly sensitive to glucose fluctuations, and a rapid decline in glucose levels can disrupt normal neuronal activity, leading to significant clinical symptoms.

In both scenarios, neuroimaging is often necessary to rule out other causes of acute neurological changes, such as ischemic stroke, hemorrhage, or seizures, and to assess the potential structural or functional consequences of the glucose abnormality.

Neuroimaging Modalities In Acute Diabetic Emergencies

Various neuroimaging techniques are utilized to evaluate diabetic patients with acute neurological symptoms. The most common imaging modalities include:

1. CT Scan (Computed Tomography)

The CT scan is typically the first-line imaging modality in acute emergency settings due to its rapid availability and ability to rule out major structural abnormalities like intracranial hemorrhage or ischemic stroke. CT imaging, however, is less sensitive for detecting subtle brain edema or metabolic changes associated with hyperglycemia and hypoglycemia.

- **Hyperglycemia:** In cases of severe hyperglycemia, non- contrast CT may demonstrate signs of cerebral edema, particularly in patients with DKA or HHS. However, these changes are often subtle and may not be visible until the edemais advanced.
- **Hypoglycemia:** In hypoglycemic patients, CT scans are generally unremarkable unless other complications, such as stroke, are present. Severe hypoglycemia can lead to diffusion abnormalities in certain brain regions, which may be apparent in advanced imaging modalities.

2. MRI (Magnetic Resonance Imaging)

MRI is more sensitive than CT for detecting brain tissue abnormalities and can provide more detailed information about cerebral edema, ischemia, and metabolic changes related to extreme glucose fluctuations. MRI is particularly useful in identifying subtle changes in the brain that may occur due to prolonged hyperglycemia or hypoglycemia.

- **Hyperglycemia:** MRI can reveal signs of cerebral edema or ischemia, especially in patients with HHS or DKA.

Diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) sequences are valuable in detecting brain changes even in the absence of gross structural damage.

- **Hypoglycemia:** MRI is particularly useful in detecting the neurovascular and structural consequences of severe hypoglycemia, such as ischemic lesions or selective neuronal loss

in regions like the hippocampus, basal ganglia, and cerebral cortex. Diffusion-weighted imaging (DWI) may reveal areas of restricted diffusion corresponding to hypoglycemic-related ischemia.

3. Diffusion Tensor Imaging (DTI)

Diffusion Tensor Imaging, a sophisticated MRI technique, can be used to evaluate the integrity of white matter pathways. In both hyperglycemic and hypoglycemic emergencies, DTI can provide insights into microstructural changes that might not be apparent on conventional MRI scans. DTI is particularly valuable in detecting subtle damage to the brain's white matter tracts,which could be implicated in cognitive dysfunction or motor deficits.

4. Positron Emission Tomography (PET) And Functional MRI (fMRI)

PET and fMRI are more advanced neuroimaging techniques that measure brain activity and metabolism. These modalities are generally not used in routine emergency settings but may be considered for research or specialized clinical investigations to understand the functional implications of glucose dysregulation in the brain. Both techniques can assess alterations in cerebral metabolism, with PET scans detecting glucose hypometabolism in areas affected by prolonged hyperglycemia or hypoglycemia.

A. Hypoglycemic Encephalopathy

Hypoglycemia indicates low blood sugar levels and acute hypoglycemic brain injury is known as hypoglycemic encephalopathy1. Most cases of adult hypoglycemia occur as a result of diabetes treatment with insulin and sulfonylureas.

Glucose insufficiency results in impaired oxygen utilization and compromised intracellular energy production. Indirect effects of hypoglycemia occur secondary to accumulation and release of excitatory neurotransmitters, which in turn accentuates the degree of hypoglycemic brain injury. Various clinical presentations include seizures, mental confusion and coma. The residual neurologic deficits are correlated with the severity of cortical injury1. NECT can show symmetrical hypodense parietal and occipital lobes. The putamina appear hypodense whereas the thalami are spared. Diffuse cerebral edema with near total sulcal effacement and blurred grey white matter interface can be seen. On MR imaging, the earliest changes are seen on DWI sequences. The involved regions show restricted diffusion with corresponding ADC hypointensity. The extent of theseabnormalities depends on severity and duration of hypoglycemia. Changes are usually bilateral, though bilaterally asymmetric, or, rarely, unilateral lesions, may occur. Reduction in ADC values has been shown to follow establishment of cerebral isoelectricity, a process that may be asynchronous.2 T2 /Flair hyperintensity in the parietooccipital cortex and basal ganglia is typical of acute hypoglycemic encephalopathy.

Differential Diagnosis Of Hypoglycemic Encephalopathy Include

1. **Hypoxic Ischemic Encephalopathy:-** In contrast to hypoglycemic encephalopathy, thalami and cerebellum are often affected in HIE.
2. **Acute Cerebral Ischemia Infarction:-** wedge shaped, involving both cortex and underlying white matter.
3. **Acute Hypertensive Encephalopathy:-** It typically affects parietooccipital cortex but spares most of the underlying white matter and rarely restricts on DWI. Neuropathologic studies have demonstrated that the cerebral cortex, hippocampus, and basal ganglia are commonly affected sites in severe hypoglycemia. However, cerebellum and the brain stem are usually spared, while the occipital cortex, dorsofrontal cortex, and hippocampus may be more resistant to prolonged hypoglycaemia.3-4

B. Hyperglycemic Encephalopathy

The brain is protected by high glucose exposure because of blood-brain barrier which reduces brain exposure to two-thirds that of the peripheral blood level.

Types:-

1. Chronic Hyperglycemic Brain Injury:-

Hyperglycemiainduced brain injury can be chronic or acute. Patients with DM2 have accelerated arteriolosclerosis and lipohyalinosis with silent infarcts, brain volume loss, and decreased cognitive functioning. MR shows increased numbers of T2/FLAIR subcortical andperiventricular hyperintensities, especially in the frontal WM, pons, and cerebellum. DTI demonstrates loss of microstructural integrity with decreased FA. Elevated myoinositol on MRS reflects gliosis, an indicator of brain injury1.

2. Acute Hyperglycemic Brain Injury:-

Two acute conditions are associated with hyperglycemia: DKA and HHS. DKA and HHS are both caused by reduction in the net effective action of circulating insulin.

A. Diabetic Ketoacidosis (DKA):- Although DKA can occur in patients with both DM1 and DM2, it is quite uncommon in DM2. DKA is defined as acidosis with a venous pH less than 7.3 or serum bicarbonate concentration less than 15 mmol/L in the presence of serum glucose concentration more than 11 mmol/L. DKA is characterized by glucosuria, ketonuria, and ketonemia.

Imaging in patients with acute DKA is nonspecific, with vasogenic cerebral edema the most common abnormality. Younger age, severe acidosis, hypocapnia, and dehydration have all been cited as risk factors for DKA- related cerebral edema. If severe cerebral edema develops and is aggressively treated, survivors may show imaging evidence for bilateral (central) descending transtentorial herniation1.

B. Hyperglycemic hyperosmolar non ketotic encephalopathy (HHS):- HHS occurs almost exclusively in patients with DM2. The clinical laboratory criteria for HHS include plasma glucose concentration > 33.3 mmol/L, serum bicarbonate concentration

> 15 mmol/L, absent or minimal ketonuria and ketonemia, effective serum osmolality > 320 mOsm/kg, and the presence of stupor or coma. Unlike DKA, seizures are common. Imaging in HHS is uncommon. T2/FLAIR hypointensity in the parietooccipital WM and transient lesions of the corpus callosum splenium have been reported. Patients treated for HHS with rapid correction of the hyperosmolar state may alsodevelop osmotic demyelination with typical findings of central pontine myelinolysis1.

C. Hyperglycemia-Induced Hemichorea (HIHH)-Hemiballismus:- Also called delayed-onset diabetic striatopathy, the syndrome of hyperglycemia-induced hemichorea-hemiballismus (HIHH) is characterized by non-patterned and involuntary unilateral movements.

HIHH usually occurs in elderly patients, more often affects female patients, and may be the first ("unmasking") symptom of DM2. It can present as an acute, life-threatening manifestation of DM or develop 1-4 weeks after the hyperglycemic event when blood sugar has been controlled. MR findings are virtually pathognomonic of HIHH. T1 shortening in the basal ganglia contralateral to the hemichorea-hemiballism with sparing of the thalamus is characteristic . In two thirds of HIHH cases, T2* GRE or SWI demonstrates hypointense signal within the affected striatum. The signal abnormality probably represents manganese or zinc deposition, not calcium or hemorrhage.1

MATERIALS AND METHODS:-

We collected three cases/patients who presented with uncontrolled hyperglycemia or severe hypoglycemia in emergency at a tertiary care centre in Northern part of India and underwent MRI brain. MRI brain findings and relevant clinical and lab data were noted.

Cases

Case 1:-A male patient 78 years old, known case of Diabetes mellitus type 2, chronic liver disease and portal hypertension came after prolonged starvation in unconscious state. His blood glucose levels were 38mg/dl on presentation. His GCS Score was 6/15 (E1V2M3). MR showed symmetrical hyperintense signals in bilateral basal ganglia, medial aspect of left thalami, bilateralhippocampus and along the cortex of the cerebral hemisphere with sparing of the white matter, brainstem and cerebellum on T2 and FLAIR sequence and shows restriction on diffusion, suggesting of acute hypoglycemic encephalopathy.

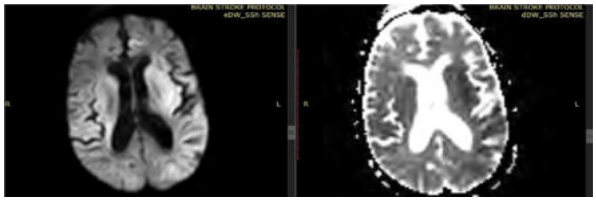

Figure 1A:- DWI Sequence **Figure 1B:-** ADC Sequence

Figure (1A and 1B):- MR Sequences DWI And ADC Axial

Images- Showing Symmetrical restricted diffusion in parietooccipital region and basal ganglia.

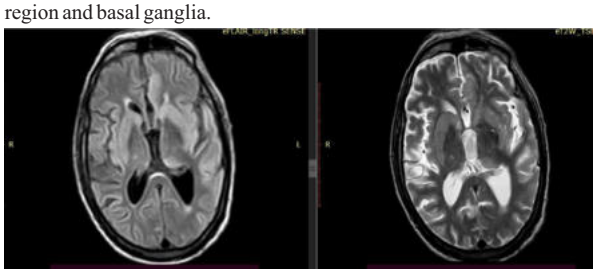

Figure 2A:-FLAIR Sequence **Figure 2B:-**T2W Sequence

Figure 2a And 2b :- MR sequences FLAIR AND T2 Axial images showing symmetrical hyperintense signals in the bilateral basal ganglia, medial aspect of left thalami and along the cortex of the cerebral hemisphere with sparing of the white matter.

Case 2:- A female patient aged 43 years presented in emergency department with 2 episodes of seizures. Patient had also complaint of double vision and blurring of vision. Patient was conscious but disoriented. Patient was known case of type II diabetes mellitus. Patient had history of anaemia. Patient's vitals were as follows:- Temperature was afebrile. Respiratory rate was 20/min. BP was 115/70 mmHg. Pulse rate was 95/min and SPO2 was 98% . Overall GCS was E4 V3 M5(patient responded to words and localised motor response was present).ECG and ABG were normal. Random blood sugar was 343 mg/dL. Patient was then admitted in ICU. MRI findings showed true gyriform pattern diffusion restriction in parietooccipital region predominantly on right side. T2 /FLAIR hypointensity was seen in bilateral parieto-occipital region with gyri appearing oedematous and thickened. Cortex was predominantly involved and the grey-white matter differentiation was intact. MRI venogram was normal. The probable diagnosis was kept hyperosmolar hyperglycemic encephalopathy.

Figure 3A and 3B, Figure 4A and 4B

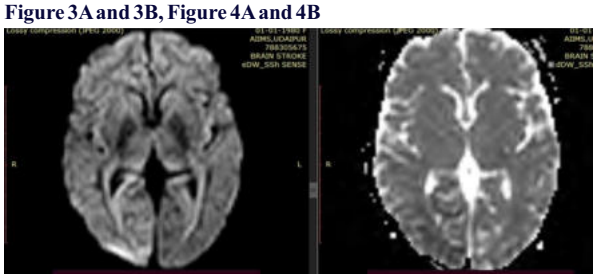

Figure 3A:-DWI Sequence **Figure 3B:-**ADC Sequence

Figure (3A And 3B):- MR SEQUENCES DWI AND ADC AXIAL IMAGES showing true gyriform pattern diffusion restriction in parietooccipital region predominantly on right side.

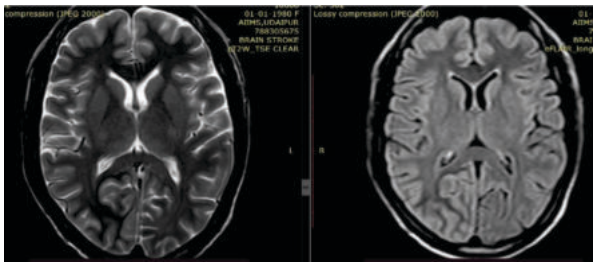

Figure 4A:-T2W Sequence **Figure 4B:-** FLAIR Sequence

Figure 4A And 4B:- MR sequences FLAIR AND T2 Axial images showing hypointensity in bilateral parieto-occipital region with Gyri appearing oedematous and thickened. Cortex is seen predominantly involved and the grey-white matter differentiation is seen intact.

Case 3:- A male patient aged 44 years presented in emergency with a history of diabetes mellitus controlled by oral glibenclamide and metformin with seizures. His family had noticed that he had been drowsy and had become Unarousable an hour before the seizure. His MR report suggested Symmetrical hyperintense signals in the bilateral basal ganglia with diffuse cerebral edema, likely acute metabolic/ toxic encephalopathy, probably acute hypoglycemic encephalopathy.

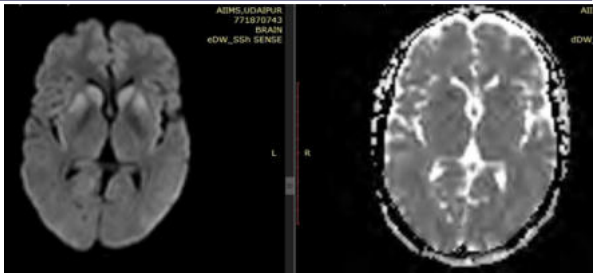

Figure 5A:-DWI Sequence **Figure 5B:-**ADC Sequence

Figure 5A And 5B:- MR Sequences DWI And ADC Axial

Images:- Showing diffusion restriction in bilateral caudate and putamen.

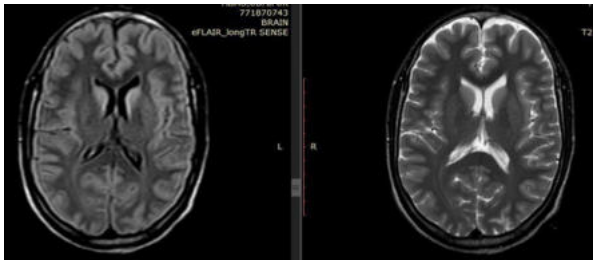

Figure 6A:-FLAIR Sequence **Figure 6B:-** T2W Sequence

Figure 6A And 6B :- MR sequences FLAIR AND T2 Axial images showing symmetrical hyperintense signals in the bilateral basal ganglia with diffuse cerebral edema.

DISCUSSION:

The main source of energy for the neurons of the brain is glucose.The adult brain makes only 2% of the total body weight but it utilizes around 20% of the total energy derived from glucose.6

Significant variations in plasma glucose levels, be it hypoglycemia or hyperglycemia, can present with myriad clinical manifestations and may mimic stroke or epilepsy.1 This case series reported three cases showing neuroimaging findings of uncontrolled hyperglycemia and severe hypoglycemia.

MR imaging can help in demonstrating the signal abnormalities in cortex and subcortical regions as well as basal ganglia abnormalities of hyperglycemia induced hemichorea, hemiballismus syndrome. Hemichorea-Hemiballismus is more common in elderly patients with non-insulin dependent diabetes and is more prevalent in Asians. The condition is characterized by an involuntary, nonrhythmic movement disorder that involves one side of the body. The abnormal movements may be continuous or intermittent, they disappear during and they are attributed to focal lesions in the contralateral basal ganglia. The leading cause of hemichorea-hemiballismus is an ischemic or hemorrhagic stroke1. Non-ketotic hyperglycemia (NKHG) is the next most common cause 7 of HC-HB and correction of hyperglycemia is usually curative. The clinical spectrum of NKHHS encompasses symptoms mimicking more frequently posterior circulation stroke, although the involvement of anterior brain areas is not uncommon8. Alternatively, a metabolic etiology is suggested by the frequent finding of peculiar MRI abnormalities9. These include the presence of subcortical T2-FLAIR hypointensities, which represent the most frequent alteration , and of disseminated cortical lesions withincreased DWI signal and restricted ADC. T2-FLAIR hypointensities have been related to free radical damage, iron accumulation, and early cortical ischemia 10-11, which can be promoted by either hyperosmolarity or hyperglycemia. An infarct show loss of grey white matter differentiation and more of peripheral involvement as compared to non- ketotic hyperglycemic encephalopathy.

As in our case series, patient (CASE 2) with hyperglycemic encephalopathy showed hypointensity on T2 /FLAIR in bilateral parieto-occipital region. The findings were similar to a study done by Serene et al 12who demonstrated a patient who had presented with focal seizures in which magnetic resonance imaging revealed subcortical T2/FLAIR hypointensity in bilateral occipital regions, which has been described to be characteristic for hyperglycemia-related seizures.Few other studies 14, 15, 16 have shown have also shown subcortical T2 hypointensity in seizures with NKH. The other associated abnormalitiy noted along with focal subcortical T2

hypointensity was cortical gyral swelling which was also seen in patients included in study by Raghavendra S et al 9. The DWI in this case showed restricted diffusion that suggested 'cytotoxic edema'. The focal cytotoxic edema could be related to seizures, focal ischemia or hyperviscosity 17. Restricted diffusion has been recently reported in seizures with NKH 15 and diabetic ketoacidosis 18.

Hypoglycemia can be induced by overuse of insulin or oral hypoglycemic agents or renal failure 19.The pathomechanism of HE still remains unclear. One of the pathogenetic mechanisms for diffusion restriction in HE that have been proposed includes energy failure. Severe hypoglycemia induces neurochemical changes. Glucose deprivation leads to arrest of protein synthesis in many regions, incomplete energy failure and loss of ion homeostasis, cellular calcium influx, and intracellular alkalosis.

Consequently, neuroactive amino acid (aspartate) release into the extracellular space occurs and results in selective neuronalnecrosis, predominantly in the cerebral cortex, caudoputamen, and hippocampus.[20,21,22,23]. However, protein synthesis in the cerebellum, brain stem, and hypothalamus remains unaffected because of the greater activity of the glucose transport mechanisms.[23,24,25]

Brain MR imaging is a useful technique to evaluate severe HE. Some authors have reported DWI and ADC findings of HE [20,21,22] According to previous reports, the lesions show reversible cytotoxic edema at the cerebral cortex26 ,splenium21, and internal capsule22.In our case series, patients with acute hypoglycemic encephalopathy, showed hyperintensity on T2/FLAIR with diffusion restriction in the parietooccipital cortex and basal ganglia (case 1 and case 3). The findings are similar to the findings obtained in the study by G. Bethla et al2 .

In patients with diabetic emergencies, one of the main roles of neuroimaging is to differentiate between glucose-related changes and other potential neurological causes of altered mental status. Key considerations include:

1. **Acute Stroke:** Diabetic patients are at an increased risk of stroke, and acute ischemic stroke may present with symptoms similar to severe hypoglycemia or hyperglycemia. CT and MRI are essential for distinguishing between ischemic stroke and other causes of altered mental status.
2. **Seizures:** Seizures due to hypoglycemia can be difficult to differentiate from those caused by other neurological conditions. MRI may show subtle changes in areas of the brain susceptible to hypoglycemic injury, such as the hippocampus, while CT may fail to detect such abnormalities.
3. **Cerebral Edema:** As mentioned, both hyperglycemia (especially in DKA or HHS) and hypoglycemia can contribute to cerebral edema. The exact mechanisms are complex and may involve osmotic shifts, metabolic disturbances, and vascularchanges. MRI sequences such as T2-weighted images, FLAIR, and DWI can help assess the presence and extent of cerebral edema in these patients.
4. **Structural Abnormalities:** Rarely, neurological symptoms in diabetic patients may be unrelated to glucose fluctuations, requiring neuroimaging to rule out structural abnormalities like tumors, infections (e.g., meningitis or encephalitis), or vascular malformations.

CONCLUSION:-

Neuroimaging is an indispensable tool in the evaluation of diabetic patients presenting with acute neurological symptoms in emergency settings, especially those related to extreme fluctuations in blood glucose. Both hyperglycemia and hypoglycemia can lead to significant cerebral consequences, and timely neuroimaging can help clinicians distinguish between glucose-related pathophysiological changes and other more serious neurological conditions. Neuroimaging aids in identification of the cause of encephalopathy and helps us to differentiate hypoglycemic/hyperglycemic encephalopathy from hypoxic ischemic encephalopathy or acute hypertensive encephalopathy or infarct. While CT remains the first-line modality for rapid assessment, MRI offers superior sensitivity and specificity for detecting brain changes associated with both acute hyperglycemia and hypoglycemia. In all cases, neuroimaging should be part of a comprehensive diagnostic approach that includes clinical assessment, laboratory tests, and timely therapeutic interventions to optimize patient outcomes.

Abbreviations

DKA -Diabetic KetoacidosisHE- Hypoglycemic encephalopathy
HHS- Hyperosmolar Hyperglycemic state
DM2 – Diabetes mellitus type II
DWI- Diffusion-weighted imaging
HC-HB – Hemi chorea – hemi ballismus
NKHG - Non-ketotic hyperglycemia
NKHHS- Non-ketotic Hyperosmolar Hyperglycemic state
HIHH - Hyperglycemia-induced hemichorea-hemiballismus

'Statements and Declarations' :-

Not applicable

Ethical Considerations:-

Not required as it was an observational study.

Consent To Participate:-

Informed written consent was taken from all the 3 patients.

Declaration Of Conflicting Interest:-

'The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article'.

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REFERENCES:-

1. Osborn's Brain imaging, pathology and anatomy
2. G. Bathla, B. Policeni, A. Agarwal Neuroimaging in Patients with Abnormal Blood Glucose Levels American Journal of Neuroradiology May 2014, 35 (5) 833-840; DOI: 10.3174/ajnr.A3486
3. Kang EG, Jeon SJ, Choi SS, Song CJ, Yu IK. Diffusion MR Imaging of hypogly-cemic encephalopathy. AJNR. 2010;31:559–64. doi:10.3174/ajnr.A1856.
4. Finelli PF. Diffusion-weighted MR in hypoglycemia coma. Neurology. 2001;57:9 33–35. doi: 10.1212/wnl.57.5.933-a.
5. Adeyinka A, Kondamudi NP. Hyperosmolar Hyperglycemic Syndrome. [Updated 2023 Aug 12].
6. Mergenthaler P, Lindauer U, Dienel GA, Meisel A (2013) Sugar for the brain: the role of glucose in physiological and pathological brain function. Trends Neurosci 36(10):587 –597
7. Priola AM, Gned D, Veltri A, et al. "Case 204: Nonketotic Hyperglycemia-induced HemiballismHemichorea." Radiology 2014; 271:304-308.
8. Rossi S, Romoli M, Urbinati G, Benini M, Russo M, D'Anna L, Abu-Rumeileh S, Sacco S, Querzani P, Foschi M. Acute stroke-like deficits associated with nonketotic hyperglycemic hyperosmolar state: an illustrative case and systematic review of literature. Neurol Sci. 2022 Aug;43(8):4671-4683. doi: 10.1007/ s10072-022-06088-7. Epub 2022 Apr 28. PMID: 35482160; PMCID: PMC9349111.
9. Raghavendra S, Ashalatha R, Thomas SV, Kesavadas C. Focal neuronal loss, reversible subcortical focal T2 hypointensity in seizures with a nonketotic hyperglycemic hyperosmolar state. Neuroradiology. 2007;49(4):299–305. doi: 10.1007/s00234-006-0189-6.
10. Ida M, Mizunuma K, Hata Y, Tada S. Subcortical low intensity in early cortical ischemia. AJNR Am J Neuroradiol. 1994;15(7):1387–93.
11. Selim MH, Ratan RR. The role of iron neurotoxicity in ischemic stroke. Ageing Res Rev. 2004;3(3):345–53. doi: 10.1016/j.arr.2004.04.001.
12. Panneer, Serene & Jain, Ayush. (2024). Neuroimaging in uncontrolled hyperglycemia: a case series and literature review. Egyptian Journal of Radiology and Nuclear Medicine. 55.10.1186/s43055-024-01195-1.
13. Lee, I, MR Imaging Findings of Hyperglycemic Encephalopathy. Radiological Society of North America 2012 Scientific Assembly and Annual Meeting, November 25 - November 30, 2012 ,Chicago IL.
14. .Seo DW, Na DG, Na DL, Moon SY, Hong SB (2003) Subcortical hypointensity in partial status epilepticus associated with non ketotic hyperglycemia. J Neuroimaging 13:259–263
15. Lavin PJ (2005) Hyperglycemic hemianopia: a reversible complication of non-ketotic hyperglycemia. Neurology 65:616–619
16. Wang CP, Hsieh PF, Chen CC, et al (2005) Hyperglycemia with occipital seizures: images and visual evoked potentials. Epilepsia 46:1140–1144
17. Chu K, Kang DW, Kim DE, Park SH, Roh JK (2002) Diffusion-weighted and gradient echo magnetic resonance findings of hemichorea-hemiballismus associated with diabetic hyperglycemia: a hyperviscosity syndrome? Arch Neurol 59:448–452
18. Placidi F, Floris R, Bozzao A, et al (2001) Ketotic hyperglycemia and epilepsia partialis continua. Neurology 57:534–537
19. Lo L, Tan AC, Umapathi T, et al . Diffusion-weighted MR imaging in early diagnosis and prognosis of hypoglycemia. AJNR Am J Neuroradiol 2006;27:1222–24
20. Hasegawa Y, Formato JE, Latour LL, et al. Severe transient hypoglycemia causes reversible change in the apparent diffusion coefficient of water. Stroke1996;27:1648–56
21. Kim JH, Roh JH, Koh SB . Reversible injury of internal capsule and splenium in a patient with transient hypoglycemic hemiparesis. Cerebrovasc Dis 2006;22:282–83
22. Albayram S, Ozer H, Gokdemir S, et al. Reversible reduction of apparent diffusion coefficient values in bilateral internal capsules in transient hypoglycemia-induced hemiparesis. AJNR Am J Neuroradiol 2006;27:1760–62
23. Auer RN, Siesjo BK. Hypoglycaemia:brain neurochemistry and neuropathology. Bailleires Clin Endocrinol Metab 1993;7:611–25
24. Kiessling M, Xie Y, Kleihues P . Regionally selective inhibition of cerebral protein synthesis in the rat during hypoglycemia and recovery. J Neurochem 1984;43:1507–14
25. Auer RN . Progress review: hypoglycemic brain damage. Stroke 1986;17:699–708
26. Lo L, Tan AC, Umapathi T, et al. Diffusion-weighted MR imaging in early diagnosis and prognosis of hypoglycemia. AJNR Am J Neuroradiol 2006;27:1222–24