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ORIGINAL ARTICLE

Assessment of hyperosmolar hyperglycemic state with MRI

[1]Mohammed Hasan, [2]Vishal Saraswat

[1]Assistant Professor, Department of Radio Diagnosis, Major S D Singh Medical College & Hospital, Farukkhabad, Uttar Pradesh, India;
[2]Director & Chief Radiologist, Dr Vishal Imaging Centre, Aligarh, Uttar Pradesh, India

ABSTRACT:
Background: Hyperosmolar hyperglycemic state (HHS) is a complication of diabetes mellitus in which high blood sugar results in high osmolarity without significant ketoacidosis. The present study assessed MRI findings in patients with hyperosmolar hyperglycemic state. **Materials & Methods:** 65 patients of hyperosmolar hyperglycemic state of both genders were assessed for serum osmolality and blood urea nitrogen. All the patients underwent MRI on a 1.5 Tesla scanner. **Results:** Out of 65 patients, males were 30 and females were 35. Clinical findings were focal motor seizures in 23, multiple myoclonic jerks in 22, generalized tonic clonic seizures in 10 and complex partial seizures in 10 cases. The mean Na+/K+ level was 135.2/4.2 mmol/l, serum osmolarity was 302.4 mOsm/Kg, blood glucose level found to be 474.2 mg/dl and HbA1C level was 13.5%. The difference was non- significant (P> 0.05). **Conclusion:** Clinical findings were focal motor seizures, multiple myoclonic jerks, generalized tonic clonic seizures and complex partial seizures.
Key words: Hyperosmolar hyperglycemic state, seizures

Corresponding author: Vishal Saraswat, Director & Chief Radiologist, Dr Vishal Imaging Centre, Aligarh, Uttar Pradesh, India

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INTRODUCTION

Hyperosmolar hyperglycemic state (HHS) is a complication of diabetes mellitus in which high blood sugar results in high osmolarity without significant ketoacidosis.[1] Symptoms include signs of dehydration, weakness, leg cramps, vision problems, and an altered level of consciousness.[2] HHS occurs in patients with type 2 diabetes who can still produce insulin (as opposed to diabetic ketoacidosis [DKA], which occurs in persons with type 1 diabetes and some with type 2 diabetes).[3] Hyperglycemia-induced seizures are refractory to antiepileptic medications, account for 15–40% of seizures in patients with HHS, and are commonly focal motor seizures and epilepsia partialis continua.[4] HHS can be precipitated by infections, medications, nonadherence to therapy, undiagnosed diabetes, substance abuse, and coexisting diseases. Infections are the leading cause (57% of cases); pneumonia, often gram-negative, is the most common infection, followed by urinary tract infection and sepsis.[5] The hallmarks of HHS include profound dehydration, marked hyperglycemia, variable degrees of neurologic impairment, and mild or no ketosis. Although DKA and HHS have been described as distinct entities, one-third of patients exhibit findings of both.[6]

Characteristic magnetic resonance imaging (MRI) findings in hyperglycemia-induced seizures include focal altered signal intensity, i.e., subcortical T2 hypointensity with gyral hyperintensity involving the cortex and cortical or leptomeningeal postcontrast enhancement.[7] The present study assessed MRI findings in patients with hyperosmolar hyperglycemic state.

MATERIALS & METHODS

The present study comprised of 65 patients of hyperosmolar hyperglycemic state of both genders. All patients were informed regarding the study and their written consent was obtained.

Data such as name, age, gender etc. was recorded. The serum osmolality and blood urea nitrogen were calculated as follows- Serum osmolality (mOsm/kg = (2x (Na {mmol/L} + K {mmol/L}) + (BUN {mg/dl}/2.8) + (glucose {mg/dl}/18). Blood urea nitrogen (mg/dl) = Urea (mg/dl)/2.14. All the patients underwent MRI on a 1.5 Tesla scanner including conventional sequences (T1 weighted image [T1W], T2W, and fluid-attenuated inversion recovery [FLAIR]) and advanced sequences (diffusion-weighted imaging [DWI] and susceptibility-weighted angiography [SWAN]). Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 65		
Gender	**Males**	**Females**
Number	30	35

Table I shows that out of 65 patients, males were 30 and females were 35.

Table II Assessment of parameters

Parameters	Variables	Number	P value
Clinical findings	Focal motor seizures	23	0.18
	Multiple myoclonic jerks	22	
	Generalized tonic clonic seizures	10	
	Complex partial seizures	10	
Laboratory findings	Na+/K+ (mmol/l)	135.2/4.2	-
	Serum osmolarity (mOsm/Kg)	302.4	-
	Blood glucose (mg/dl)	474.2	-
	HbA1C (%)	13.5	-

Table II shows that clinical findings were focal motor seizures in 23, multiple myoclonic jerks in 22, generalized tonic clonic seizures in 10 and complex partial seizures in 10 cases. The mean Na+/K+ level was 135.2/4.2 mmol/l, serum osmolarity was 302.4 mOsm/Kg, blood glucose level found to be 474.2 mg/dl and HbA1C level was 13.5%. The difference was non- significant (P> 0.05).

Table III Assessment of MRI findings

Location	Variables	Findings
Cortex	T2/FLAIR	Hyperintense
	DWI	Restriction
	SWAN	Isointense
Subcortical white matter	T2/ FLAIR	Hypointense
	DWI	Isointense
	SWAN	Hypointense

Table III shows MRI findings in patients with hyperosmolar hyperglycemic state.

DISCUSSION

Hyperosmolar hyperglycemic state (HHS) is a life-threatening endocrine emergency that most commonly affects adults with type II diabetes mellitus. [8] Poor adherence to diabetes medication causes 21% of HHS cases. Other causes include myocardial infarction, cerebrovascular accident, pulmonary embolism, and mesenteric artery thrombosis. Psychoactive medications, especially second-generation antipsychotics, cause glucose elevations, insulin resistance, and diabetes independent of weight gain.[9] Older adults with type 2 diabetes are at higher risk of HHS because they often take dehydrating medications and may be unable to adequately communicate their symptoms if they live alone or in a nursing home. The mortality rate from HHS ranges from 10% to 50%, which is considerably higher than that of DKA. In children, the mortality rate from HHS may be as high as 60%.[10] Mortality predictors include age, degree of dehydration, hemodynamic instability (hypotension, absence of reflex tachycardia), degree of consciousness, infection, and a history of cancer.[11] The present study assessed MRI findings in patients with hyperosmolar hyperglycemic state.

We found that out of 65 patients, males were 30 and females were 35. Oh SH et al[12] found that females were affected more frequently than males. The mean serum glucose level measured after the onset of chorea was 481.5 mg/dl, HbA1c level was 14.4% and the serum osmolarity was 305.9 mmol/kg. 47 patients developed hemichorea. Six patients developed bilateral chorea, and magnetic resonance imaging (MRI) showed bilateral basal ganglia lesions. MRI showed that putamen was involved in all cases (isolated putamen=31 patients, additional basal ganglia lesions=22 patients). None had lesions confined to the caudate nucleus or the globus pallidus. In all, except one, the anterior limb of the internal capsule was spared. Follow-up MRI studies were performed in 22 patients. In most, hemichorea improved along with the disappearance of the lesions. In 39 patients, chorea had ameliorated completely. The remaining 14 cases showed some improvement during the follow-up period. The chorea recurred in seven patients.

We observed that clinical findings were focal motor seizures in 23, multiple myoclonic jerks in 22, generalized tonic clonic seizures in 10 and complex partial seizures in 10 cases. The mean Na+/K+ level was 135.2/4.2 mmol/l, serum osmolarity was 302.4 mOsm/Kg, blood glucose level found to be 474.2 mg/dl and HbA1C level was 13.5%. Lammouchi et al[13] in 24 patients found that posterior cerebral region was predominantly involved, with parietal

involvement in 83.3%, followed by occipital, frontal, and temporal involvement in 33.3% patients compared with occipital in 58.3%, parietal in 45.8%, and frontal and temporal in 16.6% of patients in previous literature. The subcortical T2 hypo-intensity was present in 83.3% of the patients, cortical hyper-intensity in all patients, and restricted diffusion in 66.6% of the patients compared with subcortical T2 hypo-intensity in 95.8% of the patients, cortical hyper-intensity in 62.5%, and restricted diffusion in 58.3% of the patients in previous literature. Although many etiologies present with subcortical T2 hypointensity, cortical hyperintensity, restricted diffusion, and postcontrast enhancement on MRI, the clinical setting of seizures in a patient with uncontrolled hyperglycemia, hyperosmolar state, and absence of ketones should suggest hyperglycemia-induced seizures to avoid misdiagnosis, unnecessary invasive investigations, and initiate timely management.

Most of the previous studies on MRI changes in peri-ictal period, post generalized tonic-clonic seizures, or status epilepticus reported subcortical T2 hyperintensity and in contrast, most studies reported subcortical T2 hypointensity in hyperglycemia-induced seizures. The exact pathophysiology leading to subcortical T2 hypo-intensity is not known and many hypotheses have been put forward. The transient deposition of free radicals and/or iron because of excitotoxic axonal damage during hyperglycemia-induced seizures and intracellular dehydration in glial and supporting tissues are postulated mechanisms for subcortical altered signal intensity.[14]

CONCLUSION

Authors found that clinical findings were focal motor seizures, multiple myoclonic jerks, generalized tonic clonic seizures and complex partial seizures.

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