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RESEARCH ARTICLE

A CASE OF UNUSUAL PRESENTATION OF HYPEROSMOLAR HYPERGLYCEMIC STATE (HHS)

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ABSTRACT

Introduction: Hyperglycaemia is an important diagnostic differential and has been reported to cause focal neurological deficits masquerading as stroke. Discussion of hyperglycaemia as a stroke mimic has been sparse in the era of discussion weighted imaging, but remains an important mimic. **Objective:** To create awareness that hyperglycaemia should be considered in patients presenting to the Ed with focal neurological symptoms. **Case Presentation:** A 98 Year old Female was brought to the ED with C/o Altered Sensorium, one episode of seizure, Right sided Upper and Lower Limb Weakness, Aphasia Since around 30 minutes prior to arrival to the ED. Patient is known case of Hypertension, Diabetes Mellitus Type II, and Coronary Artery Disease – Post – PTCA, Non – Compliant to medications. On examination, patient had a GCS of E4V1M4, Pupils werebilaterally 2mm and sluggishly reacting, her random blood glucose was 719 mg/dL. Her CNS examination revealed the power in her right Upper and Lower Limbs was 0/5, whereas herleft upper and lower limbs had a power of 5/5, deep tendon reflexes –on the Right Side were Muteand brisk on the left side. Plantar reflex onright side was Mute and left side was extensor. Patient's Blood Gas Analysis, revealed Lactic Acidosis. Urine Ketones was Negative. The patient was found to have a High Serum Osmolality of 315mmol/Kg. In view of clinical findings, patient was suspected to have a Hyperosmolar hyperglycaemic state (HHS) with Stroke and as per the Stroke Protocol, Non – Contrast Computed Tomography (NCCT) Brain, followed by MRI Brain was done which was not conclusive of any acute changes. The patient was immediately started on Intravenous Fluids for management of HHS, along with Insulin infusion. Neurology Consultation, Endocrinology and Cardiology Consultation were taken. Patient's Neurological Status Improved after 2 hours of management in the ED.She was admitted in the Intensive Care Unit (ICU).Patient improved over the course of her stay in the hospital and was discharged on the 5th day with follow-up advice. **Conclusion:** In conclusion, our patient had a hyperglycaemic hyperosmolar state (HHS) induced focal neurological deficit, which presented as transient ischaemic attack (TIA). We would state that in a patient presenting with focal neurological deficit and hyperglycemia, hyperglycaemic hyperosmolar state (HHS) induced focal neurological deficit should be considered as a differential diagnosis.

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INTRODUCTION

Hyperosmolar hyperglycemic state (HHS) is one of two serious metabolic derangements that occur in patients with diabetes mellitus (DM).[15, 16] It is a life-threatening emergency that, although less common than its counterpart, diabetic ketoacidosis (DKA), has a much higher mortality rate, reaching up to 5-10%.[15] Won Frerichs and Dreschfeld first described the disorder around 1880. HHS is most commonly seen in patients with type 2 DM who have some concomitant illness that leads to reduced fluid intake, as seen, for example, in elderly institutionalized persons with decreased thirst perception and reduced ability to drink water.[20] HHS is characterized by hyperglycemia, hyperosmolarity, and dehydration without significant ketoacidosis. Most patients present with severe dehydration and focal or global neurologic deficits.[17, 21, 22] HHS was previously termed hyperosmolar hyperglycemic nonketotic coma (HHNC); however, the terminology was changed because coma is found in fewer than 20% of patients with HHS.[17, 18, 19] Hyperglycemia is an important diagnostic differential and has been reported to cause focal neurological deficits masquerading as stroke. Discussion of hyperglycemia as a stroke mimic has been sparse in the era of diffusion weighted imaging, but remains an important mimic.

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CASE REPORT

A 98 Year old Female was brought to the ED with C/o Altered Sensorium, Right sided Upper and Lower Limb Weakness, Aphasia Since around 30 minutes prior to arrival to the ED. H/o 1 episode of Tonic Clonic Movements of the Limbs, was also Present, just prior to onset of her Symptoms. Prior to the onset of symptoms, patient was doing her household work. No H/o Vomiting, LOC, Fever, Trauma. Patient is known case of Hypertension, Diabetes Mellitus Type II, and Coronary Artery Disease – Post – PTCA, Non – Compliant to medications.

PRIMARY SURVEY

- Airway – Patent
- Breathing – RR : 16/minute, SpO2 – 100 % on RA
- Circulation – HR : 85/ min, BP – 90/60 mmHg, Temp: Afebrile
- Disability – GCS : E4V1M4, Pupils – B/L 2mm, Sluggish GRBS : 719 mg/dL,

SECONDARY SURVEY

HEENT-No Pallor, Icterus Cyanosis, Lymphadenopathy, JVP - Not Raised, Tongue: Dry, Left Sided Gaze

CHEST

On Inspection – No Deformity or Scar Mark seen. No swelling or lump.
Bilaterally Equal Chest Rise Seen
On Palpation – Equal Chest Rise, No Tenderness

On Percussion – Bilaterally Resonant on Percussion

On Auscultation – Bilaterally Equal Vesicular Breath Sound. No Added Sounds Heard.

CVS – First and Second Heart sound audible, no murmur or friction rub sound

CNS – Altered Sensorium,
Power – Right Upper and Lower Limbs: 0/5,
Left Upper and Lower Limbs – 5/5,
Deep tendon Reflexes – Right Side: Mute, Left: Brisk,
Plantar – Right Side: Mute, Left: Extensor
Sensation, Gait and Cerebellar Signs – Could not be assessed

ABDOMEN

Inspection -No Scar, No Swelling, Umbilicus - Normal in Position, No Engorged Vein. Hernial Orifices - Normal
Palpation - Soft, No Tenderness No palpable mass felt and normal bowel sound.
Percussion – No Dullness, Fluid Thrill or Shifting Dullness
Auscultation – Bowel Sound Heard. No Tenderness, No Bruit.
External Genitalia – Normal

EXTREMITIES – Within Normal Limit

MANAGEMENT IN ED

In View of the Findings, Point of care (POC) Tests was sent which included, Electrocardiogram (ECG) which was suggestive of a Normal Sinus Rhythm. Patient's Blood Gas Analysis, revealed Hyperlactemia with lactates of 2.8, pH was 7.30, HCO3 -28.1, Na+ - 128, K+ - 4.Urine Ketones was Negative. The patient was found to have a High Serum Osmolarity of 315mmol/Kg.

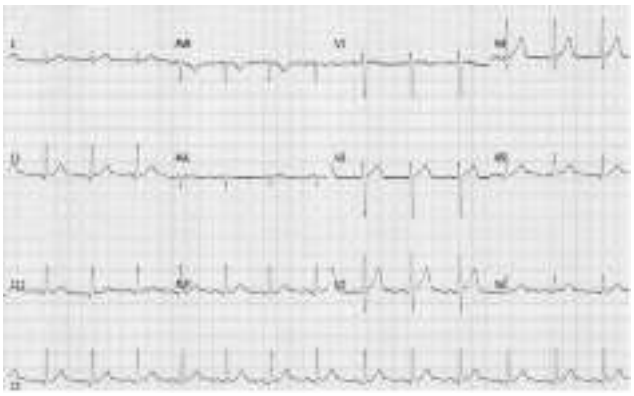

Fig 1. Electrocardiogram

In view of clinical findings, patient was suspected to have a Hyperosmolar hyperglycemic state (HHS) with Stroke and as per the Stroke Protocol, Non – Contrast Computed Tomography (NCCT) Brain, followed by MRI Brain were done, both of which were suggestive of, Chronic Microvascular Ischemic changes in Bilateral Fronto – Parietal White Matter. Age Related Cerebral Atrophy. No Acute Infarct or Haemorrhage was seen. In view of the Hyperosmolar hyperglycemic state (HHS) patient was immediately started on Intravenous Fluids, 2 liters of 0.9% Normal Saline (NS), was given as a bolus followed by at a rate of 200ml per hour, after a 2- D ECHO was suggestive of a Normal Study with a Ejection Fraction of 55%. Neurology Consultation was taken, in view of the Neurological Deficit. A Blood Gas Analysis was repeated after 2 L IVF – 0.9 % NS, which showed Lactates has reduced to 2.0, pH was 7.35, HCO3 was 24.4, Na+ improved to 134, K+ had reduced to 3.3mEq/L. Endocrinology Consultation was taken in view of HHS, and was advised for Injection Human Insulin 6 U IV stat; followed by Injection Insulin Infusion at 6 U/Hr, along with Injection KCL at 40 mEq in 500 ml NS Over 4 Hours, as the Serum Potassium (K+) was 3.3 mEq/L.

Re Evaluation after 2 L of IVF – NS (around 1 Hour), after ED Management

Primary Survey

- Airway – Patent
- Breathing – RR : 16/mt, SpO2 – 100 % on RA
- Circulation – HR : 70/ min, BP – 100/70 mmHg, Temp: Afebrile
- Disability – GCS : E4V2M5, Pupils – B/L 2mm, Reactive, GRBS : 428 mg/dL
- GCS : E4 V4 M5-6 (after 1 and Half Hours in the ED)

Secondary Survey

HEENT – Tongue : Dry. No Nystagmus. No Fixed Gaze
CHEST – Air Entry Bilaterally Equal and Clear.Heart sounds S1and S2 Heard. Abdomen – Soft, Bowel Sounds - Normal, No Tenderness, Guarding, Rigidity

CNS – Agitated, Altered Sensorium, Not Obeying Verbal Commands,

Power – Right Upper and Lower Limbs: 4/5,
Left Upper and Lower Limbs – 5/5,

Patient responded well to the fluid resuscitation and Insulin Therapy. Patient's Neurological Status Improved within 2 hours for presentation to the ED, with return of active movements in Right Upper and Lower Limbs, Confused Speech, yet not obeying commands. Cardiology Consultation was taken in view of the patient's past history of PTCA and was empirically started on antiplatelets, statins and antihypertensive drugs. Patient was admitted in the Intensive Care Unit (ICU). An Electroencephalogram (EEG) was done the following morning, in view of the history of Abnormal Tonic Clonic Movements, which was suggestive of only Moderate Slowing in the Background, no epileptiform discharges were seen, thereby Seizure was ruled out, after discussion with the Neurologist. Patient improved symptomatically over the course of her stay in the hospital, and was discharged on the 5th Day post admission with discharge and follow up advice.

DISCUSSION

Neurological impairment in the setting of hyperglycemic states with or without ketoacidosis has long been known.[1, 2, 3] Through the 1980s there were many articles written about neurological manifestations of hyperglycemia with most of these cases involving a combination of coma or seizures without ketoacidosis.[1, 4-6] Focal neurological deficits are often reported, including aphasia, homonymous hemianopsia, hemiparesis, or hyperreflexia, but usually in the setting of post-ictal Todd's paresis or where post-ictal state could not be ruled out.[1, 2, 6] In some of these early cases with diabetic coma and focal neurological signs intravascular thrombosis and brain ischemia later was found at autopsy.[1,7] Subsequent discussion of focal neurological presentations has been sparse after the advent diffusion weighted imaging. As per the history given by the patient's son, our patient had an episode of general clonic tonic seizure; therefore the hemiparesis could also have been post-ictal Todd's palsy. The patient's EEG findings are non-specific and likely secondary to her hyperglycemia.

Maccario and others noted that in their hyperglycemic patient with EEG slowing in the affected hemisphere was seen without clinical evidence of seizures.[1,6] In studies of the effects of hyperglycemia on EEGs it has been demonstrated that slowing can be a result of hyperglycemia and that the slowing seen associated with hyperglycemic states may take days to resolve.[1, 8] Seizure is a metabolically active state which generates increased perfusion on perfusion imaging.[1, 9, 10] However, there are reports of post – ictal hyperperfusion and hypoperfusion mimicking ischemia.[1, 11-14] This disconnect between normal CBV with increased TTP and MTP with loss of function is consistent with flow-metabolism coupling, blood flow adjusting to energy demands. In our patient's case hyperglycemia was associated with temporary neuronal dysfunction in the left hemisphere based on EEG slowing in the absence of vascular compromise. The lower perfusion reflects lower demand from metabolically inactive tissue rather than hypoperfused tissue at risk of infarction.

Conclusion

In conclusion, that our patient had a hyperglycaemic hyperosmolar state (HHS) induced focal neurological deficit, which presented as transient ischaemic attack (TIA). We would state that in a patient who presents with focal neurological deficit and hyperglycemia, hyperglycaemic hyperosmolar state (HHS) induced focal neurological deficit should be considered as a differential diagnosis.

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