A Case Report of Neuroglycopenic Coma With Diffuse Cortical Involvement
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ABSTRACT

Hypoglycemia is reversible if recognized and treated promptly, delayed recognition leads to neuroglycopenia with poor outcome. White matter involvement is most commonly observed in neuroglycopenia. Superficial grey matter involvement is also observed in neuroglycopenia. Involvement of superficial cortical structures will have dismal outcome.

KEY WORDS:
Hypoglycemia, delayed recognition, neuroglycopenia

1. INTRODUCTION

Hypoglycemia is a metabolic abnormality that triggers a series of physiological, psychological and behavioral responses. The clinical manifestations include neuroglycopenic symptoms such as inability to concentrate, drowsiness, confusion, speech abnormality and in coordination due to brain fuel deprivation and neurogenic or autonomic symptoms such as palpitations, tachycardia, diffuse weakness, anxiety and hunger. Prolonged hypoglycemia can result in neuronal dysfunction and death, with deficits ranging from measurable cognitive impairments to aberrant behavior, seizures and coma. In sustained hypoglycemia, the integrity of cerebral neurons is not preserved. The initial changes are seen in mitochondria, first in dendrites followed by stoma. Ultimately there is rupture of cell membranes and cell death. DWI of patients with acute hypoglycemia has shown high-intensity signals in numerous different locations of the brain. Although cortex, basal ganglia, and hippocampus seem to be the brain tissues most vulnerable to hypoglycemia, the underlying mechanism of the distribution pattern of the high-intensity signals remains unclear. Asymmetry of the lesions might be explained by the asynchronous onset of electro-cerebral silence between the hemispheres. Involvement of neostriatum and diffuse cortical lesions are associated with poor outcome.

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2. GLASSGOW COMA SCALE:

The GCS is divided in three assessment parameters:

Eye opening (score 1 to 4): spontaneous eye opening when standing next to the patient’s bed or even during procedures receives a score of 4. Eye opening by verbal stimulus using the simple commands such as “open your eyes”, sometimes-continuous verbal stimulus is needed, the score given is 3. Eye opening with painful stimulus, applied by the examiner, in regions as nail bed and by supraorbital pressure, the score given is 2. No eye opening even after application of all previous described stimuli, the score given is 1.

Verbal response (score 1 to 5): patient is oriented in time, space and aware of the self, he/she is able to answer accordingly simple questions such as “Do you know where you are right now?”, “Are you aware of what has happened?”; the score given is 5. Patient can answer questions, but incoherently, he/she is disoriented and confused, the score given is 4. A score of 3 is given for patients who answer do not match questions. The need of painful stimulus that answers are incomprehensible songs, e.g., moaning, groaning, the score given is 2. No response even after application of all previous described stimuli, the score given is 1.

Motor response (score 1 to 6): score of 6 is given for patient who obey simple commands, such as “raise your arm or leg”, “Move your feet or hands” with adequate motor response. After a painful stimulus, patient find the origin and try to remove what is causing the pain, the score given is 5. After a painful stimulus, the patient is able to find the pain and move the limb by flexion, however, he/she is not able to remove the source of...
pain, the score given is 4. A score of 3 is given for the patient who motor response is by flexion movement, evidenced by decortication response, therefore, presenting arms flexed, or bent inward on the chest, hands clenched into fists, and legs extended and feet turned inward. A score 2 is given for patients that motor response is by extensor movement and decerebrate posture in which neck is extended, arms are rigid extended close to elbows, legs are extended on knees level, and feet in plantar flexion. A score 1 is given for patient who does not present no motor response even after application of all previous described stimuli.

3. CASE PRESENTATION

A 31-year-old male patient brought to causality with loss of consciousness and seizures. Patient’s attenders gave history of consumption of alcohol and retired to bed the day before his presentation. Attendees noticed excessive snoring in the morning after few hours he developed seizures. He is brought in unconscious state to causality on the same day. At the time of presentation GCS is 3/15. Vitals: pulse rate: 96 bpm, blood pressure: 90/60 mm of Hg, Spo2: 88% with room air and 96% with 4L of oxygen. On examination, CV S and GIT: normal, RS: Coarse crepitations present, CNS: Bilateral pin point pupils, not reacting to light, hypotonia with absent DTR in all limbs. Planter’s bilateral no response. Investigations: Random blood sugar: 39 mg/dl, Total counts: 7,500 cells/mm3, differential counts: polymorphs 78%, lymphocytes 18%, Serum Creatinine: 2.3 mg/dl, serum electrolytes: Na+ 149 mmol/l, K+ 3 mmol/L, Cl-114 mmol/L. CT brain plain is normal. Patient diagnosed as Neuroglycopenia with aspiration pneumonia and treated accordingly.

On Day 2, GCS is 3/15, patient developed high grade fever, vitals: pulse rate: 102 bpm, blood pressure: 110/70 mm of Hg, temperature: 102°F, Spo2: 94% with 4L of oxygen, CV S and GIT: normal, RS: Coarse crepitations present, CNS: Bilateral pin point pupils, not reacting to light, hypotonia with absent DTR in all limbs, neck stiffness and extensor plantar response observed. Fundus is normal. CSF analysis showed: Total counts: 5 cells/mm3, differential count: lymphocytes 100%, proteins: 20 mg/dl, sugars: 187 mg/dl, adenosine deaminase levels: 4.6 U/L. On day 3, Patient Glasgow come scale is 3/15, vitals: pulse rate: 96 bpm, blood pressure: 90/60 mm of Hg, Spo2: 88% with room air and 96% with 4L of oxygen, CV S and GIT: normal, RS: Coarse crepitations present, CNS: Anisokoria is noticed. Pupil’s right- 5 mm, left- 1 mm not reacting to light. The same day at 6 pm on examination anisokoria on being not reversed, after exclusion of space occupying lesion, pilocarpine drops (0.025%) were installed and after 45 minutes pupils were examined and constriction of right pupil was noted with no alteration in size of left pupil, indicating involvement of ciliary ganglion, probably due to chronic alcoholism. Patient couldn’t be revived and expired on day 4.

4. DISCUSSION:

Delayed treatment of hypoglycemia results in neurological sequelae like seizures, speech abnormalities, ataxia, stroke like symptoms and loss of consciousness. Hypoglycemia was initially reported to predominantly involve the cortex, neostriatum, and hippocampus in many reported predominant white matter involvement, mainly affecting the centrum semiovale, corona radiata, internal capsule and splenium of the corpus callosum. In fact, involvement of the white matter is now thought to be earlier and more common than grey matter involvement. The thalamus, brainstem and cerebellum are invariably spared and this may help to differentiate hypoglycemia from hypoxemic injury which often involves the thalamus. On the basis of topographic distribution of signal abnormalities, three imaging patterns have been described. These include 1) predominant grey matter involvement affecting the cortex, neostriatum, hippocampus. 2) Predominant white matter involvement affecting the periventricular white matter, internal capsule and splenium of corpus callosum. 3) mixed pattern involving both grey and white matter. The cerebellum is less prone to hypoglycemic insult; it may relate to greater efficiency of glucose transporter, thus making hypoglycemia induced cerebellar dysfunction a less common complication of severe hypoglycemia. Taguchi, et al. reported the brain magnetic resonance imaging findings of a patient with hypoglycemic encephalopathy, which included increased reversible signal intensity in the left temporal and occipital gray matter, right putamen, both posterior limbs of internal capsule and triangular area of lateral ventricle. Patients with focal involvement of internal capsule or corona radiata or splenium usually have good prognosis, these lesions resolve promptly after restoration of blood glucose, though tend to follow clinical symptom resolution. Patients with extensive white matter involvement show variable response. The prognosis in these cases varies between complete recovery and persistent vegetative state Involvement neostriatum and diffuse cortical lesions portend dismal outcome. Failure of lesions to regress on follow up imaging is also associated with poor prognosis.

5. CONCLUSION

Hypoglycemia is reversible if recognized and treated promptly. Delayed recognition leads to neuroglycopenia with poor outcome. White matter involvement is most commonly observed in neuroglycopenia. Superficial grey matter involvement is also observed in neuroglycopenia. Involvement of superficial cortical structures will have dismal outcome.

CONFLICTS OF INTEREST

The authors do not have any conflict of interest.
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