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Case Report

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A Rare Case of Hypoglycemic Unawareness in a Patient With Chronic Congestive Heart Failure

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ABSTRACT

Hypoglycemia and hypoglycemic unawareness is usually a complication of tight glycemic control in insulin-dependent diabetes mellitus. To our knowledge, this is the first case report of hypoglycemic unawareness subsequent to cardiac failure induced hypoglycemia of prolonged duration. Through this case report, we aimed to highlight that hypoglycemia should be suspected as a cause of decreased level of consciousness in patients suffering from recurrent congestive cardiac failure , as it can be easily overlooked and the condition can easily be reversed by simply administering adequate glucose.

Introduction

Hypoglycemic unawareness is a failure to develop or recognize autonomic warning symptoms before the development of neuroglycopenia. This event usually occurs in patients with insulin-dependent diabetes mellitus, insulinoma, and psychiatric disorders treated with repetitive insulin-induced hypoglycemia [1-3].

Here we report a case of hypoglycemia resulting from recurrent episodes of Congestive Heart Failure and subsequent hypoglycaemic unawareness. It is pertinent to mention that the cause of mental obtundation, in this

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case, could not be attributed to hypoglycemia till serum chemistry unmasked the cause.

Case Report

A 90-year-old male was admitted in the emergency ward of our hospital with a 5-day history of breathlessness, anorexia, cough, vomiting, generalized body swelling, and decreased urinary output. The patient was a known case of hypertension on erratic treatment and gave a history of similar episodes of generalized body swelling and decreased urinary output in the past few months for which he used to seek frequent admissions at a local hospital. He used to receive diuretics off and on for symptomatic relief. General physical examination revealed an irregular pulse of 90 beats per minute with a blood pressure of 80/60 mm Hg. Moreover, the patient was drowsy but arousable (Glasgow Coma Scale [GCS]=11/15). Besides his raised jugular venous pressure, the patient had pedal edema with peripheral and central cyanosis.

Chest examination revealed scattered rales with diminished air entry in both the infrascapular regions. Also, the patient had cardiomegaly and tender hepatomegaly. Investigations revealed a normal hemogram and kidney function tests. However, arterial blood gas analysis at admission revealed a pH of 7.17, $PaCO_2$ of 29 mm Hg, PaO_2 of 34 mm Hg, and HCO_3 of 10.6 mmol/L. Moreover, liver function test revealed serum bilirubin of 2.5 mg/dL with a total protein of 7.4 g/dL, AST (aspartate aminotransferase) of 94 U/L, ALT (aminotransferase) of 88 U/L, and alkaline phosphatase of 140 U/L. However, venous blood sugar was only 20 mg/dL. The insulin and C peptide levels were within the normal range. Electrocardiogram and chest skiagram showed atrial fibrillation and bilateral pleural effusion, respectively. Biochemical analysis of pleural fluid revealed a transudative effusion with a sugar level of 15 mg/dL. Serum lactate level was elevated, too. Except for bilateral pleural effusion, ascites, congested hepatomegaly with congested inferior vena cava, abdominal ultrasonography was unremarkable.

Echocardiography revealed features of hypertensive cardiovascular disease with mild mitral regurgitation and severe tricuspid regurgitation with enlarged right atrium and ventricle with right ventricular systolic pressure of 50 mm Hg and borderline left ventricle functions. Upper GI endoscopy, CT scan of the brain, and CSF analysis were unremarkable. The HbA1C of the patient was 4.8%. According to the above clinical and laboratory findings, a diagnosis of hypertensive heart disease with hypoglycemia was made.

Upon administration of 25% dextrose infusion, the patient's sensorium improved. Subsequently, he was maintained on 5% dextrose infusion and encouraged to take oral dextrose which helped maintain his blood sugar in a healthy range. Pressor support to keep blood pressure followed by diuretic therapy was instituted to relieve him of his congested state.

Discussion

Hypoglycemia is seen in patients suffering from chronic Congestive Heart Failure due to multiple causes. Congestive Heart Failure for a significant duration leading to chronic passive congestion of liver is common amongst all these patients. None of these cases had evidence of organic disease of the pancreas, pituitary, adrenals, or central nervous system which might have led to the development of hypoglycemia [4].

The possible pathogenesis of hypoglycemia in patients with severe cardiac failure includes hepatic congestion, hypoxia, exhaustion, and gluconeogenic precursor defects. Suppression of gluconeogenesis leads to elevated serum lactate levels [5]. Other reasons contributing to hypoglycemia include hypoxemia, inadequate carbohydrate intake, and hypotension secondary to circulatory collapse [4].

Furosemide administration can also cause hypoglycemia [5]. The likely contributory causes of hypoglycemia in our patient were congestive hepatomegaly, hypotension, hypoxemia, inadequate intake, and furosemide administration. The partial or complete inability to perceive hypoglycemia, at which the typical warning symptoms occur, is defined as hypoglycemic unawareness. Patients suffering from hypoglycemic unawareness either do not realize neuroglycopenic symptoms or become aware of lower plasma glucose levels (high thresholds). Physiological hypoglycemia is defined as a fall in plasma glucose concentration below 65 mg/dL (3.5 mmol/L) [6]. Hepburn et al. categorized hypoglycemia unawareness into following groups [7]: normal awareness, partial awareness, and total loss of awareness.

Counter-regulatory hormones (glucagon and epinephrine) are released at a plasma glucose level of 65-68 mg/dL. Autonomic symptoms begin to appear around the blood sugar level of 58 mg/dL. At 54 mg/dL level, the cognitive function deteriorates as reflected by delayed

reaction time and impaired ability to perform other psychomotor tasks [6].

There is a possibility that repetitive episodes of hypoglycemia due to chronic heart failure in our patient damaged his glucose-sensitive neurons. Besides, there is a reduced autonomic response and decreased adrenergic sensitivity in the elderly accounting for the absence of autonomic symptoms in response to hypoglycemia [6].

The expression of Glucose Transporters (GLUT-1) as well as the neuron-specific Glucose Transporters (GLUT3) located in the capillaries of the blood-brain barrier increases in response to recurrent hypoglycemia. As such, there is no diminution in the brain glucose uptake due to frequent hypoglycemia. As a result, there is less neuroglycopenia in future hypoglycemic episodes, and the need to generate the counter-regulatory responses and the autonomic symptoms to alert the subject about hypoglycemia is no more required [8]. This chain of reactions explains as to how the brain gets adapted to hypoglycemia which in turn causes impaired awareness of hypoglycemia in subsequent hypoglycaemic episodes.

Conclusion

Hypoglycemia should be strongly considered in a patient of chronic Congestive Heart Failure with acute deterioration in sensorium where other causes of mental obtundation have been rolled out by appropriate investigations.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles were considered in this article. The participant was informed about the purpose of the research and its implementation stages; He was also assured about the confidentiality of his information; Moreover,He was allowed to leave the study whenever he wish, and if desired, the results of the research would be available to him.

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Conflict of interest

The authors declared no conflict of interest.

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