Case Report

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An Unusual Presentation of Recurrent Hypoglycemia

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Abstract

Hypoglycemia is defined as random blood sugar equal to or lower than 60mg/dl. The most common cause is medications such as sulfonylurea, biguanides and insulin. Other causes include liver disease, certain tumors, kidney disease, severe infections and starvation. It can be a very common presentation in elderly patients with altered mental status who are on polypharmacy. Investigating recurrent hypoglycemia can be a challenge. Whilst the obvious focus is to rule out an underlying endocrine etiology, a thorough history and recognition of factitious cause is important and worth bearing in mind. This can be difficult to diagnose and often, can only be ruled out by extensive investigations and exclusion of other causes . Patients with clinical hypoglycemia unawareness are at high risk of severe hypoglycemia that requires third-party assistance. Hypoglycemia is less frequent in type 2 diabetes than it is in type 1. Population-based data indicate that the overall event rate for severe hypoglycemia (requiring the assistance of another individual) in insulin-treated type 2 diabetes is approximately 30 percent of that in type 1 diabetes (35 versus 115 episodes per 100 patient-years). In this case a young non diabetic female presented to ED in a state of altered mental status with recurrent hypoglycemia, the cause for which was thought to be sepsis and ultimately diagnosed as fulminant hepatic failure. In fulminant hepatic failure there as altered mental status with coagulopathy in setting of acute liver disease. Neurotoxins like ammonia and glutamine with cytokines produce cytogenic and vasogenic effects which leads to cerebral oedema and thus altered sensorium. Patient presents in a state of hepato cellular dysfuction, encephalopathy and cerebral oedema, infections or multi organ failure. The case emphasizes the importance of appropiate history taking and correct differential diagnosis establishment in order to achieve good outcome of a patient with fulminant hepatic failure.

Keywords: Hypoglycemia; Diabetes; Hepatic Failure; Altered Sensorium.

Introduction

Hypoglycemia is defined as random blood sugar equal to or lowers than 60mg/dl. The most common cause is medications such as sulfonylurea, biguanides and insulin. Other causes include liver disease, certain tumors, kidney disease, severe infections and starvation. It can be a very common presentation in elderly patients with altered mental status who are on polypharmacy. Investigating recurrent hypoglycemia can be a challenge. Whilst the obvious focus is to rule out an underlying endocrine etiology, a thorough history and recognition of factitious cause is important and worth bearing in mind. This can be difficult to diagnose and often, can only be ruled out

by extensive investigations and exclusion of other causes. In a setting of endogenous insulin deficiency (type 1 and advanced type 2 diabetes), one episode of hypoglycaemia reduces both counterregulatory hormone responses to and subjective awareness of subsequent hypoglycaemia, thus impairing physiological defences against hypoglycaemia. This phenomenon may lead to a vicious cycle of recurrent hypoglycaemia and glucose counterregulatory failure, of which hypoglycaemia unawareness (i.e. the inability to perceive symptoms of hypoglycaemia) is the clinical representative.

The underlying mechanism of hypoglycaemia-induced counterregulatory failure has not yet been disclosed. Patients with clinical hypoglycaemia unawareness are at high risk of severe hypoglycaemia that requires third-party assistance. Hypoglycemia is less frequent in type 2 diabetes than it is in type 1. Population-based data indicate that the overall event rate for severe hypoglycemia (requiring the assistance of another individual) in insulin-treated type 2 diabetes is approximately 30 percent of that in type 1 diabetes (35 versus 115 episodes per 100 patient-years) and that event rates for hypoglycemia requiring professional emergency medical treatment range from 40 to 100 percent of those in type 1 diabetes .

In this case a young non diabetic female presented to ED in a state of altered mental status with recurrent hypoglycemia, the cause for which was thought to be sepsis caused by fulminant hepatic failure. In fulminant hepatic failure there as altered mental status with coagulopathy in setting of acute liver disease. Hepatic encephalopathy occurring within 8 weeks of onset of illness defines fulminant hepatic failure. The common cause is either viral hepatitis or toxin mediated. Neurotoxins like ammonia and glutamine with cytokines produce cytogenic and vasogenic effects which leads to cerebral oedema and thus altered sensorium.

Patient presents in a state of hepato cellular dysfuction, encephalopathy and cerebral oedema, infections or multi organ failure. Altered mental status with coagulopathy in setting of acute liver disease. Hepatic encephalopathy occurring within 8 weeks of onset of illness defines fhf.

Laboratory studies show higher levels of transaminase (>1000), with mixed hyper bilirubinemia, elevated ammonia with prolonged pt, aptt, metabolic acidosis and increased lactates. Many therapies for management like insulin and glucagon to stimulate regeneration, prostaglandin-E, corticosteroids, hemofiltration, charcoal hemo-perfusion, plasma exchange have been tried but the best results are achevied by liver tranplantation.

The case emphasises the importance of appropiate history taking and correct diffrential diagnosis establishment in order to achieve good outcome of a patient with fulminant hepatic failure.

Case History

36 year old female presented to ed with c/o decreased responsiveness since 1day associated with 2-3 episodes of vomiting since morning following which she became drowsy.

The airway was maintainable by using a naso pharyngeal device, breathing labored with a respiratory rate of 32/m, saturating at 100% on room air. She had a heart rate of 77 beats per minute and blood pressure of 110/70mmhg.

Her Glasgow coma scale reading was E4V1M5, pupil bilaterally reactive, RBS of 44 mg/dl.

50% of dextrose given i.v bolus.

POC done include ECG and ABG.

On further history taking she was known to be a case of psychosis, was taking medications 4 months ago along with some pain killers.

Icterus was noted on HEENT examination, chest was bilateral clear, CVS- S1, 2 heard with no murmur, abdomen was soft, tenderness was noted over right hypochondrium with hepatomegaly, bowel sounds heard, CNS examination revealed decreased left side body moment and her plantars were bilateral extensors.

Her LMP-9/12/14 (5d/28d), last delivery-9yrs ago, Copper t – in situ.

On repeat vitals, her heart rate, blood pressure, saturation, respiratory rate were all similar except her blood sugar level which was noticed to be 450mg/dl after 50% dextrose.

Her ECG showed normal sinus rhythm.

ABG shows :- ph- 7.4, pco2- 24.6, po2- 112 on 4l of o2, Na- 117 meq/l, k- 5.5 meq/l, hco3- 15.3, LAC- 5.9.

In view of above investigations differentials of CVA, Sepsis due to hepatic cause, Isulinoma and drug over dose were made for which ct brain plain and ct abdomen along with complete blood count, renal and liver profile, viral markers were sent.

The ct brain and abdomen revealed a normal study.

Before shifting the patient to ICU her vitals were

rechecked this again revealed similar parameters except rbs of 95mg/dl.

Patient was started on i.v. antibiotics and maintainece fluids.

In the ICU she was managed symptomatically.

Her CBC revealed hb-10.7m/dl, platelets-421 , rbc-3.13, mcv- 101.7

Mch- 34.3, TLC-39.6*10-9/l, neutropils-79%, eosinophils- 1%, lympocytes- 6% .

Liver function test were bilirubin- total-7.6mg/dl (direct- 3mg/dl, indirect- 4mg/dl), total proteins- 5.5 mg/dl, albumin-2.7 mg/dl, globulin-2.8 mg/dl, sgot-452 iu/l, sgpt- 1025 iu/l, alk phosphate- 230 iu/l.

Renal function test na- 124.8 mmol/l, k- 4.4 mmol/l, cl- 101.3 mmol/l

S.urea- 12mg/dl, s.creat- 36mg/dl.

Urine routine- normal, urine for tox- not sinificant, s. markers- hbsag- negative, hiv- negative, hcv-negative, Hep E- positive, S.Ammonia – 183microgm, APTT- 86.7.

Final Diagnosis:

Severe sepsis

Hepaitis E

Hepatic encephalopathy

Fulminant hepatic failure

Discussion

In ED if a patient presents with recurrent hypoglycemia apart from ruling out the other causes of altered mental status e should also think about the underlying liver pathology and detailed liver profile should be sent.

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