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Case Report

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Hypoglycaemia and Cardiac Failure: An Uncanny Association

Short running title: **Cardiac failure presenting as recurrent hypoglycaemia**

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Abstract

Hypoglycaemia in the intensive care unit is frequently encountered with varied causes. Here we report an extremely rare case of persistent hypoglycaemia due to congestive cardiac failure (CCF) and its successful improvement with management of CCF. An 81 years old man with chronic kidney disease and coronary artery disease presented with recurrent syncope, dyspnoea and swelling of body to the emergency room. The detailed laboratory work-up revealed diagnosis of CCF. He was managed with intravenous dextrose infusion but the hypoglycaemia episodes were persistent and recurrent. The hypoglycaemia episodes were corrected only after improvement of CCF, thus confirming the cause of hypoglycaemia to be CCF. CCF has been reported to cause hypoglycaemia mostly in children but extremely rare in adults. CCF though rare should be considered as one of the causes of persistent hypoglycaemia in adults. Literature search revealed few case series implicating CCF as cause of hypoglycemia.

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Keywords

hypoglycaemia; cardiac failure; uncanny association; congestive; diabetes mellitus; drug-induced; critical illnesses; organ failure

Introduction

Hypoglycemia is a common occurrence in the intensive care units (ICU). Persistent hypoglycaemia is one of the common end-stage manifestations in many diseases. The causes of hypoglycaemia include diabetes mellitus, drug-induced, critical illnesses like organ failure, certain hormonal deficiencies like cortisol or glucagon deficiency, non-islet cell tumour, malicious hypoglycaemia etc. The rare causes include congenital hyperinsulinism, insulin receptor mutation, inborn errors of metabolism, ectopic insulin secretion, autoimmune causes, cardiac failure etc [1-3]. Here we are reporting one such case of hypoglycemia due to congestive cardiac failure (CCF) which is of extremely rare occurrence.

Case report

An 81-year-old man, never smoker presented to the emergency room (ER) with altered sensorium, decreased food intake and irrelevant talking for two days. He had no history of fever, cough or shortness of breath. He had coronary artery disease (CAD) with history of percutaneous transluminal coronary angioplasty with stenting to left anterior descending artery and right coronary artery two months ago. His echocardiography done at that time showed a left ventricular ejection fraction of 25-30% with moderate mitral regurgitation and trivial tricuspid regurgitation. He was also diagnosed with chronic kidney disease (CKD) 3 months back. His prior blood urea was 137 mg/dl and serum creatinine were 3.5 mg/dl and he was on conservative management for CKD. There were no other comorbidities. At presentation in the ER, he was conscious but confused with Glasgow coma scale score of 12. His respiratory rate was 26/minute, pulse rate was

94/minute, oxygen saturation was 92% on room air and blood pressure was 140/90 mmHg. On auscultation, there were normal vesicular breath sounds and bilateral fine basal crepitations. The heart sounds were normal. His random blood sugar (RBS) in the ER was 35 mg/dl. The arterial blood gas analysis showed pH of 7.49, pCO2 of 26.8, pO2 of 52, and HCO3 of 20.9. His electrocardiogram had a normal sinus rhythm and troponin-T was negative. His chest radiograph showed cardiomegaly with bilateral middle and lower zone opacities in perihilar predominance indicating pulmonary oedema. He was given intravenous dextrose for correction of hypoglycaemia and then shifted to ICU with the provisional diagnosis of CCF with pulmonary edema vs bilateral pneumonia with sepsis and hypoglycaemia.

In the ICU he had similar episodes of hypoglycaemia which were recurrent and was managed by continuous infusions of 25% dextrose (table 1). His bedside lung ultrasound showed presence of bilateral closely spaced B lines and bilateral mild pleural effusion. The inferior vena cava diameter was 1.6 cm and there was no collapsibility. His screening echocardiogram revealed a left ventricular ejection fraction of 25-30%. His subsequent blood investigation results are shown in table 2. The computed tomography of chest showed bilateral ground glass opacities with septal thickening, bilateral pleural effusion and cardiomegaly suggestive of cardiogenic pulmonary oedema (figure 1). Thus, he was diagnosed with CCF and pulmonary edema with recurrent spontaneous hypoglycaemia. He was started on aggressive diuretic therapy with frusemide intravenous injection at a dose of 20mg thrice daily along with antibiotics (coamoxiclav and azithromycin) and other supportive therapy. With this treatment, the frequency of the hypoglycaemia episodes reduced gradually over 3 days (table 1). On day 4, his serum glucose levels improved and he started maintaining normoglycemia without any dextrose infusions. The patient improved during next few days along with resolution of chest radiograph abnormalities (figure 2). He was discharged on room air with treatment for CAD and CKD. He was counselled regarding possible hypoglycaemia and its management with advice on monitoring of RBS at home by glucometer. On follow up after 1 month in the out-patient department he was found to be doing well and had no further episodes of hypoglycaemia.

Day	Hypoglycemia episodes and the blood sugar level in mg/dl	Number of hypoglycaemia episodes
1	30, 26, 28, 40, 62, 69, 65	7
2	35, 20, 62, 37	4
3	20, 38, 50	3
4	RBS was never <70 mg/dl	0

Table 1

Parameters	Reference range	Result
Haemoglobin	11.5-16.6 g/dl	10.1 g/dl
Total leukocytes	4000-11000/cumm	10,600/cumm
Platelets	150000-400000/cumm	2,00,000/cumm
Total Bilirubin	0.1-1.2 mg/dl	0.8 mg/dl
Aspartate transaminase	0-35 IU/L	68 IU/L
Alanine transaminase	3-36 IU/L	72 IU/L
Alkaline phosphatase	44-147 IU/L	240 IU/L
Urea	10-40 mg/dl	170 mg/dl
Creatinine	0.7-1.3 mg/dl	3.9 mg/dl
Sodium	135-145 meq/L	136 meq/L
Potassium	3.5-5.5 meq/L	4.2 meq/L
Chloride	98-107 meq/L	108 meq/L
NT pro BNP	<125 pg/mL	>30,000 pg/ml
Procalcitonin	<0.1ng/mL	0.95 ng/mL

Table 2

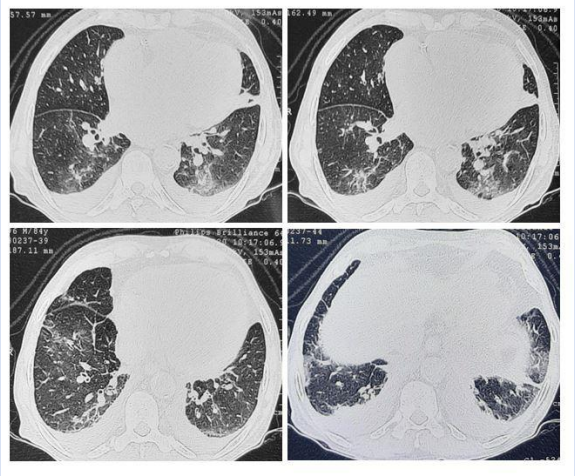

Figure 1

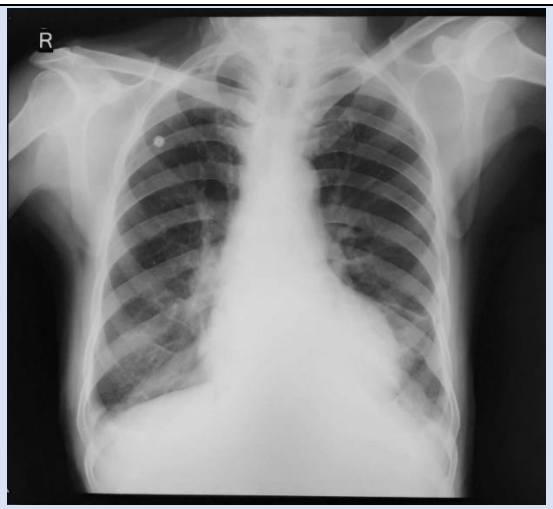

Figure 2

Discussion

Hypoglycaemia is defined as a plasma glucose concentration below 4 mmol/L (72 mg/dl) [4]. Occurrence of episodes of hypoglycaemia in critically ill patients is known [5]. It occurs due to septic shock related adrenal failure or fulminant hepatic failure. The cause of hypoglycaemia in critically ill patients is combination of insulin excess and inadequate nutritional supplementation along with decreased glucose production endogenously and increased glucose utilisation [5-7].

CCF is one of the rare and under-recognised causes of hypoglycaemia [8]. The mechanism of hypoglycaemia in CCF is poorly understood. Glucose and fatty acids are the main energy source for the myocardial cells [9]. Myocardial tissue preferentially uses glucose as the main source of energy in case of dilated or hypertrophic cardiomyopathy and during hypoxia or ischemia [10]. Hypoglycaemia induced plasma glucose fluctuations enhances reactive oxygen species production and exaggerated myocardial injury further leading to worsening of myocardial function [11,12]. The mechanism of hypoglycaemia in CCF is through the hepatic involvement. Liver injury in heart failure causes glycogenesis and gluconeogenesis leading to hypoglycaemia. Low calorie intake, increased glucose utilization by ischaemic tissues including the heart, insufficient peripheral circulation and malabsorption of nutrients due to gastrointestinal oedema are other possible mechanisms of hypoglycaemia in heart failure patients [13-15].

The clinical presentation of hypoglycaemia ranges from dizziness, sweating, palpitations, tremor, weakness, confusion, altered mental status to life-threatening complications like seizures and coma [4]. Immediate diagnosis and prompt glucose infusions is the key to successful treatment of hypoglycaemia and prevention of life-threatening complications. Prompt diagnostic approach towards the cause of the hypoglycaemia is necessary to prevent further such episodes.

Spontaneous hypoglycaemia associated with CCF is rarely reported in adults, though more cases have been reported in neonates and infants**. In a case report by H Khoury et al.,** an inverse relationship was found between serum insulin level and serum glucose level [16]. Hypoglycaemia resolved after alleviation of the CCF. In a study conducted by Benzing et al., acute CCF and hypoglycaemia were observed simultaneously in 27 patients with underlying heart disease and also found that constant intravenous infusion of glucose is the method of choice for the treatment of these patients rather than epinephrine or glucagon along with treatment of heart failure [15]. Hedayat et al., reported eleven cases of spontaneous hypoglycaemia in CCF in adults [13].

All the reported cases have found an association between cardiac failure and persistent hypoglycemia [13-17]. To the best of our knowledge this is the second case from India showing this interesting and important association of hypoglycaemia and CCF [17]. One recent clinical study in 2020 conducted by Y Teshima et al., found out that patients with chronic heart failure are at risk of developing hypoglycaemia even if they do not undergo any antidiabetic therapy [18]. It is possible that digging into the cause of hypoglycaemia is not given priority rather than its treatment. And even if found they may not be reported since there is paucity of evidence and knowledge regarding this association. Thus, we want to highlight that it is pertinent to find out the cause of hypoglycaemia in each case.

CKD is also an unusual cause of persistent spontaneous hypoglycaemia like CCF [19]. In CKD there is reduced glomerular filtration rate which leads to reduced clearance of insulin hence causes hypoglycaemia [20]. Our patient had both CKD and CCF. But in our case, we concluded that the cause of persistent hypoglycaemia as CCF as the hypoglycaemia resolved with the successful treatment of CCF and our patient was only on conservative management for his renal disease without any dialysis or acute management.

Conclusion

CCF should be considered as one of the causes of persistent spontaneous hypoglycaemia and thoroughly investigated. Timely suspicion and management can improve the outcomes by prevention of further episodes of hypoglycaemia and worsening of cardiac function by repeated episodes of hypoglycaemia.

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