Hypoglycemia detected during cardiac arrest of a non-diabetic patient with heart failure

Diyabetik olmayan kalp yetersizlikli hastada kalp durması sırasında hipogliseminin tespit edilmesi

İnci Tuğçe Çöllüoğlu, M.D., Hüseyin Dursun, M.D., Mete Yılmaz, M.D., Asım Oktay Ergene, M.D.

Deparment of Cardiology, Dokuz Eylul University Faculty of Medicine, İzmir

Summary– Hypoglycemia in non-diabetic patients with heart failure is a rare finding. It is thought to be caused by hepatic dysfunction secondary to chronic passive congestion, and reduced gluconeogenesis. In this report we present a 23-year-old man with a history of Duchenne muscular dystrophy hospitalized for decompensated heart failure and implantable cardioverter defibrillator shock. Laboratory examination on admission showed slightly elevated levels of hepatic enzymes. Despite the therapy for heart failure the patient's clinical status deteriorated, and sinus bradycardia and then cardiac arrest occured. After successful 5-minute cardiopulmonary resuscitation, an arterial blood sample revealed metabolic acidosis with a level of pH 7.04, bicarbonate 9.3, and glucose of 22 mg/dl. Acute treatment of metabolic acidosis with hypoglycemia was successfully performed. Based on this case, possible causes of unexplained hypoglycemia in non-diabetic patients with heart failure were described in a short literature review.

Özet– Diyabetik olmayan kalp yetersizlikli hastalarda hipoglisemi nadiren saptanır. Kronik pasif konjesyona ikincil gelişen karaciğer fonksiyon bozukluğuna ve azalmış glukoneogeneze bağlı olarak geliştiği düşünülmektedir. Bu yazıda, Duchene musküler distrofisi olan ve dekompanse kalp yetersizliği ve implante edilebilen kardiyoverter defibrilatör şoklaması nedeniyle hastaneye yatırılmış 23 yaşında bir olgu sunuldu. Başvuru sırasında yapılan laboratuvar incelemesinde karaciğer enzimlerinde hafif derecede yükselme mevcuttu. Kalp yetersizliği tedavisine rağmen klinik durumu bozulan hastada sinüs bradikardisi ve ardından kalp durması gelişti. Beş dakika süren başarılı kardiyopulmoner canlandırma sonrasında bakılan arter kan gazında pH 7.04, bikarbonat 9.3 meq/L ve glukoz 22 mg/dl tespit edildi. Akut metabolik asidoz ve hipoglisemi tedavisi başarılı bir şekilde uygulandı. Bu olgudan yola çıkarak, diyabetik olmayan kalp yetersizliği hastalarında açıklanamayan hipogliseminin muhtemel nedenleri kısa bir literatür derlemesi ile özetlendi.

Ischemic hepatitis is a clinical condition characterized by a massive and transient increase in serum transaminase levels. It is a usual complication of left- and right-sided heart failure. Hypoglycemia accompanies ischemic hepatitis in a small group of patients. However, its detection in a patient with no predisposition to hypoglycemia is a rare finding.

Abbreviations:

ALT	*Alanine transferase*
AST	*Aspartate transferase*
BUN	*Blood urea nitrogen*
Cr	*Creatinine*
ICD	*Implantable cardioverter defibrillator*
IV	*Intravenously*
K	*Potassium*

In this report, we describe the case of a patient with severe hypoglycemia detected during cardiac resuscitation, and thought to be induced by heart failure.

CASE REPORT

A 23-year-old man with a history of Duchenne muscular dystrophy was hospitalized in our coronary care unit with the diagnosis of implantable cardioverter defibrillator (ICD) shock and acute heart failure. Past medical history revealed a recent pulmonary embolism, and an ICD implantation for primary prevention

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Correspondence: Dr. İnci Tuğçe Çöllüoğlu. Dokuz Eylül Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Balçova, 35340 İzmir, Turkey.
Tel: +90 232 - 412 22 22 e-mail: tugcecolluoglu@windowslive.com

3 months previously. His blood pressure was 110/72 mmHg, heart rate 110/min with regular rhythm, respiratory rate 24/min, body temperature 36.8°C, and SO_2 by pulse oximetry 90% on room air. A grade 2/6 systolic regurgitant murmur was audible at the apex, but excess heart sounds were unclear. On pulmonary auscultation, sounds were heard with difficulty in the lower right lung field. Laboratory results on admission showed slightly elevated levels of alanine transferase (ALT) 75 U/L, aspartate transferase (AST) 73 U/L, and lactate dehydrogenase (LDH) 410 U/L, with a low level of sodium (Na) 130 mmol/L, and normal levels of blood urea nitrogen (BUN) 13.7 mg/dl, creatinine (Cr) 0.59 mg/dl, potassium (K) 4.0 meq/L, magnesium 0.86 mmol/L, and glucose 105 mg/dl. The patient was started on supplemental oxygen of 5 L/min, 150 mg/hour amiodarone intravenously (IV), and furosemide of 60 mg/day IV. Transthoracic echocardiography showed a left ventricular ejection fraction of 19%, dilatation of the left and right chambers of the heart, and a moderate degree of mitral regurgitation. Despite treatment, the patient still had shortness of breath with signs of volume overload in the following hours. Diuresis was ineffective, with only a 300 cc net negative fluid balance in 12 hours. Blood chemistry revealed Na 129 mmol/L, K 4.7 meq/L, BUN 22 mg/dl, Cr 1.25 mg/dl, AST 509 U/L, ALT 261 U/L. One hour after the patient had breakfast, his clinical status deteriorated, sinus bradycardia with pacemaker rhythm was observed and he had cardiac arrest afterwards. Cardiopulmonary resuscitation for 5 minutes with adrenalin and atropine of 1 mg/IV was performed and the patient returned with normal sinus rhythm. An arterial blood sample after resuscitation revealed metabolic acidosis with pH 7.04; bicarbonate 9.3 meq/L, pCO_2 37 mmHg, pO_2 110 mmHg and glucose 22 mg/dl. A control venous blood sample for glucose level also showed a glucose level of 30 mg/dl. A 50 ml of 30% glucose bolus was followed by 10% glucose over 4 hours; and 50 mEq/IV bolus of sodium bicarbonate with a maintenance of 10 mEq/hr administered. Control glucose, and bicarbonate levels were within normal limits. In the follow-up, the patient was extubated, and with intense heart failure therapy the functional class improved in a week time. No episode of hypoglycemia occurred again. Abdominal ultrasound showed a mildly enlarged liver with normal echogenity and normal vascularity. There was no evidence in the pancreas, pituitary, adrenals, or central nervous system of organic disease which might have led to the development of hypoglycemia. Hepatic enzymes returned to baseline values. And the patient externed home with medical therapy.

DISCUSSION

Ischemic hepatitis, a relatively infrequent disorder occurring in 0.16% to 0.50% of patients admitted to medical intensive care units, often follows episodes of hypotension or acute heart failure.[1] Patients present with rapidly elevated serum liver enzymes accompanied frequently by coagulopathy with a prolonged prothrombin time. The most frequent causes are left- and right-sided heart failure, chronic obstructive lung disease, and chronic renal failure.[1,2] During an acute episode, more than 90% of patients have transient deterioration of their renal functions. Hypoglycemia can accompany these findings, and in one report of 34 patients with ischemic hepatitis, hypoglycemia was reported in 11 (32.4%) patients. The glucose level was inversely correlated with the ALT level.[1]

Although the occurrence of hepatic hypoglycemia in non-diabetic patients with heart failure was first reported about 60 years ago, it is still a very rare finding.[3] Its recognition is difficult in these patients because the symptoms of hypoglycemia are non-specific and may be easily confused with other causes of abnormal behaviour, comatose state or cardiac arrest. Mellinkoff et al.[3] were the first to report 5 patients with coexisting heart failure among 20 patients with hepatic hypoglycemia. They stated that hepatic dysfunction secondary to chronic passive congestion was probably the cause of hypoglycemic episodes. In another study, the causes of cardiac arrest in patients with advanced heart failure and hospitalized for cardiac transplantation were reviewed, and 13 arrests with bradycardia were reported. Unexplained hypoglycemia (serum glucose level <20 mg/dl) was the detectable cause in 1 of these arrests.[5] Finally, Nomura et al.[2] described an unconscious patient with severe ischemic hepatitis caused by chronic pericardial effusion. In that case, the hypoglycemia was explained by reduced gluconeogenesis in the exhausted liver.

Furthermore, severe muscle diseases may cause hypoglycaemia, since the lower protein pool and turnover in these diseases cannot provide turnover of gluconeogenic amino acids to glucose during starvation. In these patients, muscle weakness and difficulty in

swallowing also affects the quality of life, resulting in irregular food intake and anorexia.[6] More frequent blood glucose measurements are required, especially when these patients have coexisting decompensated heart failure, and parenteral glucose infusion may be considered, if needed. In our patient, Duchenne muscular dystrophy may also have influenced the occurrence of the hypoglycemic attack.

Cardiac arrest in our patient was due to bradycardia arrest. It was not clear whether hypoglycemia was the main cause of the arrest or a coexisting finding. We think that amiodarone infusion might also have played a role in the hepatic dysfunction of an already congested liver. However, ischemic hepatitis was the main cause of hepatic failure and hypoglycemia in our patient, when we consider the progressively deteriorating clinical status from admission.

In conclusion, ischemic hepatitis and related complications should be kept in mind in patients with heart failure because coexisting hypoglycemia can be easily overlooked during cardiopulmonary resuscitation of a non-diabetic patient with advanced heart failure.

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Key words: Heart failure; hypoglycemia; ischemic hepatitis.

Anahtar sözcükler: Kalp yetersizliği; hipoglisemi; iskemik hepatit.