

Sudden cardiac arrest induced by hypoglycemia and hypokalemia in a chronic alcoholic patient

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Abstract: A 44-year-old alcoholic patient was admitted to the Cardiac Intensive Care Unit due to sudden cardiac arrest in the mechanism of asystole. Additional examinations revealed hypoglycemia 13 mg/dl and hypokalemia 3.27 mmol/l. Thanks to prompt resuscitation and intravenous infusion of 40% glucose and potassium, the condition of the patient improved. Based on the case reported, causes of sudden cardiac death in alcoholics are discussed.

Key words: sudden cardiac arrest, hypoglycemia, hypokalemia, chronic alcoholic disease

INTRODUCTION

Sudden cardiac death (SCD) is an unexpected death due to cardiac causes occurring in a short time period (generally within 1 h of symptom onset) in a person with known or unknown cardiac disease. Most cases of SCD are related to cardiac arrhythmias. Hypokalemia, hypomagnesemia, and hypocalcemia are the causes of acquired long QT syndrome and sudden cardiac death [1]. In the case described, sudden cardiac arrest was caused by hypoglycemia and hypokalemia induced by alcohol and diarrhea.

Hypoglycemia induced by alcohol was first described by Brown and Harvey in 1941 in six chronic alcoholic persons [2]. Studies demonstrate that hypoglycemia is associated with cardiac disturbances and an increased incidence of death [3]. In the Framingham study, heavy drinking was apparently associated with unexpected sudden death – i.e. sudden death in the absence of prior evidence of ischaemic heart disease [4]. The basic arrhythmogenic effects of alcohol are still insufficiently delineated. Subclinical heart muscle injury from chronic heavy use may be instrumental in producing patchy delays in conduction. The hyperadrenergic state of drinking and withdrawal may also contribute, as may electrolyte abnormalities, impaired vagal heart rate control, repolarization abnormalities with prolonged QT intervals, and worsening of myocardial ischaemia or sleep apnea [5].

CASE REPORT

A 44-year-old patient with long-term history of alcoholism – the patient has been drinking alcohol for about 20 years – was admitted to the Cardiac Intensive Care Unit due to sudden cardiac arrest after consumption of high amounts of alcohol. Cardiac and respiratory arrest occurred at the patient's

home. During the 15-min resuscitation, the ambulance team intubated the patient, administered heart massage and artificial ventilation. The electrocardiography recording showed asystole followed by bradycardia. On admission to hospital, arterial pressure was found to be 90/40 mm Hg. The level of glycemia was low – 13 mg/dl and of potassium 3.27 mmol/l. The patient received atropine, an infusion of sodium and potassium chloride, sodium bicarbonate, 40% glucose and iv dopamine; his hemodynamically efficient circulatory function was restored. The patient's family reported a week-long history of diarrhea prior to admission. On physical examination, the patient was found to be unconscious, emaciated and cachectic. The eyeballs were directed right and upwards. The pupils were wide, even, and initially did not react to light. The thoracic cavity was asthenic. Vesicular murmur was heard above the lung fields; additionally, rales were detected at the base. The peripheral artery pulse was weak. The heart rate was regular, accelerated to 100/min. Abdominal integuments were soft, without pathological resistances. Liver and spleen were not enlarged. Meningeal and Babinski's signs were negative. The upper limbs were spastically contracted at the elbows.

Laboratory tests revealed the presence of anemia with hemoglobin 10.2 g/dl, RBC 3.10 M/uL, MCV 100 fl and leucocytosis 12.08 K/uL. Renal parameters were normal; however, the results of hepatic enzymes – ASPAT 228 U/l, ALT 56 U/l, and INR 1.3 were suggestive of postalcohol hepatic damage. The HBs antigen was negative. The titer of HBs and HCV antibodies were also negative. Arterial blood gasometry showed metabolic acidosis, pH 7.2 (norm 7.35-7.45) with deficiency of BE –7.5 mEq/L (norm –3-3 mEq/L), the bicarbonate level 19.4 mEq/l (norm 21-27 mEq/l). The level of procalcitonine was normal – 0.35 ng/ml (norm below 2) at increased concentration of CRP – 67 mg/l. The troponin level was normal. Toxicological tests for methanol and glycol were negative. The heat CT scan did not disclose lesions whereas the chest radiogram revealed fibroindurative changes in the right lung apex and inflammatory changes in the left lung lower lobe. Sputum culture showed the presence of *Pseudomonas aeruginosa*.

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The patient was mechanically ventilated for two days and his own efficient respiration was restored. The patient was extubated and respired spontaneously. During the following days the patient was conscious and reacted to questions. Physical examination did not show any features of paresis. The next day, the patient – in good general condition – was transferred to the Department of Internal Diseases for further treatment. The patient answered questions logically and carried out personal hygiene- and eating-related activities unaided. The repeated electrocardiography showed sinus heart rhythm accelerated to 100/min. The follow-up laboratory tests revealed normal serum levels of glucose and electrolytes.

The ultrasound scan showed an enlarged liver and numerous calcifications in the pancreas suggestive of chronic pancreatitis. Echocardiography disclosed proper sizes of heart cavities with accelerated function. Antibiotic therapy was administered (amoxicillin with clavulanic acid), oral omeprazole and potassium chloride. Since the treatment of pneumonia was difficult, the patient underwent the QuantiFERon test. The concentration of interferon gamma indicated *M. tuberculosis* infection. Based on the chest X-ray picture and elevated interferon gamma, the patient – still in good condition – was sent to the Phthisiology Department for further diagnostic procedures and treatment.

DISCUSSION

Sudden cardiac death (SCD) accounts for 250,000 deaths in the United States every year; therefore, identification of modifiable risk factors for SCD continues to be an important goal for public health. Risks differ for SCD compared with other forms of coronary heart disease death. One modifiable risk factor, alcohol consumption, may have a differential effect on the risk of sudden *versus* non-sudden coronary heart disease (CHD) death. It is well established that heavy alcohol consumption (>5 drinks/d) is associated with an increased risk of SCD [6].

Heavy consumption of alcohol can lead to negative cardiovascular outcomes, such as higher blood pressure, cardiomyopathy, heart failure and sudden cardiac death. Regular heavy ethanol consumption has been associated with a type of non-ischemic dilated cardiomyopathy termed alcoholic cardiomyopathy (ACM). In general, alcoholic patients consuming > 90g of alcohol a day (approximately seven to eight standard drinks per day) for over 5 years are at risk for the development of asymptomatic ACM, which is clinically expressed as an impairment of left ventricular function (non-symptomatic stage). Those who continue to drink may become symptomatic and develop signs and symptoms of heart failure (symptomatic stage) [7].

The patient after consumption of alcohol was admitted to the Cardiac Intensive Care Unit due to sudden cardiac arrest with symptoms of hypoglycemia.

The clinical manifestations of hypoglycemia traditionally fall into two groups: neurovegetative and neuroglycopenic manifestations. The neurovegetative manifestations result from the release of hormones antagonizing insulin (glucagon, adrenaline) and most commonly include: anxiety, irritability, hyperhidrosis, tachycardia, asthenia, pallor and mydriasis. The neuroglycopenic manifestations develop in patients with markedly reduced glucose levels and most commonly affect cognitive functions and may include convulsions, plantar

response, abnormal deep tendon reflexes, and even loss of consciousness and coma [8].

Alcoholic hypoglycemia seems to develop mainly due to starvation, absent liver glycogen stores and impaired hepatic gluconeogenesis, although the explanation for the latter remains unclear. Starvation in the course of alcoholism may cause inhibition of pyruvate dehydrogenase. The consequent accumulation of pyruvate might promote the conversion of glyceraldehyde-3-phosphate to diphosphoglycerate, thereby inhibiting gluconeogenesis [9]. Alcoholic ketoacidosis also develops as a result of starvation or vomiting, or both, during a recent drinking bout. The pathogenesis of alcoholic ketoacidosis is not clear; a major contributing factor is the 'counter-regulatory' hormonal response to hypoinsulinemia, which mobilizes free fatty acids in the liver. Both alcoholic hypoglycemia and alcoholic ketoacidosis are associated with excess consumption of alcohol, food deprivation, and the increased ratio of reduced to unreduced nicotinamide adenine dinucleotide, the coenzyme system involved in ethanol oxidation [10].

There is evidence that heavy drinking can induce cardiac arrhythmias and ventricular tachycardia, which are both mechanisms associated with sudden death. However, there is no consistent evidence from population studies that heavy drinking is associated with an increased risk of sudden death. Heavy drinking raises blood pressure and about 10% of hypertension in middle-aged men may be attributed to heavy drinking [11]. In a prospective study of Finnish men aged 40–64 followed for five years, the sudden death rate increased with increasing alcohol intake and was more pronounced in the older men (over 50 years) [12]. In a five year follow up of 269,755 American men aged 20–65 employed in the telephone industry, heavy drinking (>5 drinks a day) was associated with an increased risk of arrhythmic death but not with death from circulatory failure [13].

In the case described, sudden cardiac arrest was caused by chronic alcohol consumption and diarrhea, which together with malnutrition led to hypoglycemia and hypokalemia. Such coexisting electrolyte and metabolic disorders resulted in the development of cardiac arrhythmia with consequent cardiac arrest in the mechanism of asystole.

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