CLINICAL ASPECTS

REPEATED CARDIAC ARRESTS IN A PATIENT WITH HYPOKALEMIC PERIODIC PARALYSIS AND TYPE 2 DIABETES MELLITUS

CORINA ROMAN FILIP1, AURELIAN UNGUREANU2

1“Lucian Blaga” University of Sibiu, 2Emergency County Hospital Sibiu

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Abstract: We present the case of a 42-year-old woman, brought into the emergency unit for severe muscle weakness predominantly in the lower limbs and cardiac arrhythmia, complicated with cardiac arrest responsive to resuscitation. Laboratory tests at admission revealed extremely low levels of potassium, severe respiratory acidosis and hyperglycemia. Four years prior this presentation, she was brought to the emergency room for shortness of breath and cardiac arrhythmia, flaccid tetraparesis after an episode of digestive infection. Soon after admission in the cardiology department, she developed sustained ventricular tachycardia, followed by asystole, but responsive to resuscitation manoeuvres. The potassium level was low and serum glucose level was increased. In both situations, the patient’s condition improved significantly after rebalancing the electrolytes and acid-base metabolism. It is to be mentioned that both episodes occurred after physical effort, consistent with a positive diagnosis of sporadic hypokalemic periodic paralysis. Associated type 2 diabetes mellitus raises the question of therapy management, knowing that the potassium homeostasis is modified by hyperglycemia and its complications, but also by insulin therapy. We consider this case as being particular by the nature of the cardiac events and pathological associations, in literature, such cases being rarely reported.

INTRODUCTION

Hypokalemic periodic paralysis is a heterogeneous group of muscle pathology, manifested by paroxysms of flaccid paralysis associated with serum abnormal levels of K+. The primary forms are due to mutations in the gene coding for Ca2+(CACNA1S), Na+(SCN4A), K+(KCNE3) channels.(1) The secondary forms are found in hyperaldosteronism, distal renal tubular acidosis, gastrointestinal massive losses, toxicity from cisplatin or amphotericin B. Features: attacks last from hours to days, without affecting the respiratory muscles, non-excitatory muscle with intrinsic muscular reflexes reduced or abolished, and inconstant occurrence of myotonic phenomena. (1)

CASE REPORT

We present the case of a patient with resuscitated cardiac arrest in the past, who presented to the emergency room for decrease muscle strength occurred predominantly in legs after physical exercise. During the routine examination and laboratory work-up, ventricular arrhythmias occur. Seconds later, the patient enters in a cardiac arrest but responded to electric shock with no residual neurologic deficits. At admission we found low potassium – K+ = 0,7mEq/L, Na+ = 133-137 mEq/L, Ca++ = 0,87 – 1,1 mmol/L, pH = 7,00 – 7,44, serum glucose = 500 mg/dL, CPK = 4761U/L, LDH = 750 U/L, AST/ALT = 213/257 U/L, WBC = 27500/mm, HGB = 12,4 g/dL. CT scan, cardiac and abdominal ultrasound showed no abnormalities. Neurological examination revealed a flaccid tetraparesis, abolished deep tendon reflexes, without sensory modification, no cranial nerve involvement.

The patient recognized the occurrence of similar episode of decreased muscle strength after physical effort. The diagnosis was made as hypokalemic periodic paralysis with flaccid tetraparesis, severe hypokalemia, type 2 diabetes, ventricular arrhythmias (ventricular fibrillation and tachycardia) (figure no. 1) and resuscitated cardiopulmonary arrest. The history of this patient revealed that four years ago, she was...
brought to the emergency room for flaccid tetraparesis after a sudden muscular effort in the context of food poisoning. Routine tests showed severe hypokalemia. Brought in the cardiology department for further cardiac investigations, ventricular arrhythmias (ventricular tachycardia and fibrillation) appeared causing cardiac arrests responsive to resuscitation manoeuvres.

Figure no. 1. ECG showing major electric disturbances due to severe hypokalemia

The differential diagnosis made in the neurology intensive care unit took into consideration an acute inflammatory polyradiculoneuritis, a recurrence of chronic inflammatory demyelinating polyradiculoneuritis, myasthenia gravis crisis, multiple sclerosis, stroke, vertebral trauma, but all were excluded on clinical and paraclinical grounds.

The specific resuscitation manoeuvres continued later in neurology ICU, with inotropic therapy, electrolyte replacement, antibiotics, prophylactic anticoagulant therapy, spironolactone, insulin therapy.

The evolution of the neurological syndrome after 4 days post resuscitation revealed no pathological findings (normal segmental muscle strength, symmetric deep tendon reflexes, plantar flexion response, without disturbance of sensitivity, coordination or speech. The patient is released on request and additional investigations were not possible. The treatment at home included acetazolamide and orally potassium substitution.

Discussion:

This paper outlines a case of sporadic hypokalemic periodic paralysis in the context of type 2 diabetes, with complex ventricular rhythm disturbances resulting in two cardiorespiratory arrests resuscitated.

Association of carbohydrate metabolism disorders cause problems in the context of severe hypokalaemia and insulin therapy per se can trigger hypokalemia crisis. In healthy subjects, insulin activates the sodium-potassium ATPase pump, causing a transient hypokalemia by K⁺ influx in cells, this being compensated by K⁺ channels activation.(2)

Insulin seems to potentiate depolarization of mutated Ca channels, they in turn disturbing membrane excitability by altering the conductance of K⁺ that grows and leads to muscle membrane hyperpolarisation.(3,4,5) In this case, insulin was administered after the rebalancing the ionic values. Hypokalemic periodic paralysis may be precipitated by physical exercise, or events that lower serum potassium levels, such as carbohydrate ingestion or the use of insulin in diabetics.

Failure to quickly identify the source of hypokalemia can be fatal, but the rapid reestablishment of potassium levels and the correction of metabolic and endocrine balance can be life-saving.(6,7) Literature gives few similar cases of association of diabetes and heart rhythm problems resulting in two cardiac arrests.

REFERENCES