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CASE REPORT

Confounding elements in the recognition of severe hyperkalemia. A case report

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Introduction: Severe hyperkalemia is a life-threatening condition that demands a rapid diagnosis and prompt treatment. The following case report highlights the possible complexity in the clinical presentation of this condition and the importance of a thorough assessment of patients that do not provide the classical clinical findings.

Case presentation: A 40-year-old male called the emergency services for low blood pressure and an overall altered state. Upon arrival, the prehospital team found a patient with shock signs that was complaining of feeling unwell and reported profuse diarrhea starting three days prior. Additionally, the patient also had muscle weakness and occasional spastic movements of the limbs and face. During the assessment, the patient went suddenly into cardiac arrest, life support (including the management of hyperkalemia) was immediately started and return of spontaneous circulation was soon obtained. Point-of-care blood testing established the diagnosis of severe hyperkalemia. Definitive in-hospital care consisted mainly of fluid resuscitation, circulatory support, hemodialysis and antimicrobial therapy and the patient was discharged 16 days later with no neurological impairment.

Conclusions: Although atypical, hyperkalemia can be caused by diarrhea in certain circumstances and its clinical manifestations can be misleading. Thus, keeping a broad clinical perspective and early use of blood tests can ensure proper treatment of life-threatening hyperkalemia.

Keywords: hyperkalemia, hypokalemia, cardiac arrest, atypical

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Introduction

Severe hyperkalemia (defined as serum potassium levels over 6.5 mmol/l) is the consequence of several pathologies and an adverse effect to a number of medical treatments [1,2]. As one of the reversible causes of cardiac arrest [1], its recognition and treatment can be lifesaving. The following case report highlights the possible complexity in the clinical presentation of this condition and the importance of a thorough assessment of patients that do not provide the classical clinical findings.

Case presentation

A 40-year-old male called the emergency services for self-measured low blood pressure and an overall altered state. Upon arrival, the prehospital critical care team found a conscious, but visibly agitated patient with signs of respiratory effort and pale, cold skin. Additionally, he presented with generalized muscle weakness and occasional spastic movements of the limbs and face. His medical history included a well-controlled type 2 diabetes under Metformin treatment and hypertension treated with Rilmenidine and Bisoprolol, with no prior kidney disease. The patient reported multiple diarrheic stools over the past three days. His vitals showed hypotension (88/62 mmHg), a slight bradycardia (56 beats per minute), mild hypoxemia (oxygen saturation of 93%) and a serum glucose level of 147 mg/dl. The 12-lead EKG exposed a sinus bradycardia with non-specifically peaked T waves in three leads (II, V3, V4). Early into the primary assessment, the patient suddenly went into cardiac arrest by pulseless electrical activity, advanced life support was initiated and return of spontaneous circulation was obtained after three minutes. A point-of-care arterial blood gas test was carried out and it revealed a significant lactic acidosis (pH=6.83; Lactate=12.47 mmol/l) and severe hyperkalemia (K^+=7.3 mmol/l) along with a marked acute renal dysfunction (Creatinine=16.2 mg/dl, Urea=125 mg/dl). The treatment of hyperkalemia was started as per the European Resuscitation Council's current guidelines (with calcium gluconate, insulin-glucose infusion, sodium bicarbonate)[1]. During the prehospital care and transport, the patient went on repeating the cardiac arrest twice, each time being successfully resuscitated and eventually handed over to the emergency department. Stool sampling allowed for the conduction of a coproculture, which established the diagnosis of acute Campylobacter jejuni gastroenteritis. Definitive in-hospital care consisted mainly of fluid resuscitation, circulatory support through vasoactive medication, hemodialysis and antimicrobial therapy. The patient was discharged after 16 days with no neurological impairment.

Discussions

In the vast majority of medical literature, abundant diarrhea is listed as a frequent cause of hypokalemia [1,3], rather than hyperkalemia due to potassium loss via the gastrointestinal tract. On the other hand, diarrhea is also responsible for dehydration and loss of bicarbonate, thus being capable of causing hypovolemic shock and metabolic

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acidosis [4,5]. Moreover, if left untreated, it can lead to prerenal kidney failure [6], which, along with the metabolic acidosis, are well established causes of increased serum potassium levels [1,2]. Taking these different pathophysiological mechanisms into account, one can observe how diarrhea shifts from being a cause of hypokalemia to being a precipitator of hyperkalemia. Additionally, in this patient, the Metformin treatment that he was following seems to have also contributed to the worsening of the metabolic acidosis by raising lactate levels in the blood (Metformin-associated lactic acidosis) [4,7] and consequently to the aggravation of the hyperkalemia.

A further point to explore in the pathophysiologic chain of events leading up to the patient's condition is the general state of his renal function. It is well known that patients with diabetes are at risk for developing chronic kidney disease and can have their potassium excretion (but also the elimination of other metabolic products, such as urea) consequently impaired [8]. With chronic renal dysfunction being one of the common causes of hyperkalemia [2], but also of uremia and its complications, a differential diagnosis had to be formulated, all the more so as the uremic syndrome can present itself with abundant diarrhea [9,10]. In this regard, the patient's recent medical records clarified that his kidney function was previously unaffected (KDIGO stage G2A1 with no structural abnormalities) and his wife confirmed his strict compliance with the antidiabetic treatment. Furthermore, an obstructive uropathy was ruled out in the emergency department through the ultrasonographic assessment of the kidneys and the reason behind the diarrhea was found out to be a gastroenteritis through a Campylobacter jejuni infection.

Put together, these observations suggest that the kidney injury of the patient was acute in onset and unlikely to have been the cause of an uremic syndrome responsible for the signs and symptoms recorded. Rather, the altered renal function tests were repercussions of the hypovolemic shock and cardiac arrest.

Another aspect of discussion lies in the clinical manifestations of hyperkalemia in this particular case. Classically, hyperkalemia presents itself with flaccid paralysis or paresis, paresthesia in the extremities and nausea [1,2,11]. The EKG changes associated with this condition are bradycardia with or without atrioventricular blocks of various degree [1,11-13], P wave flattening, broad QRS complexes, ST segment modifications and peaked T waves [1,2,11]. Instead, the manifestations of hypokalemia include spastic paralysis or paresis, fatigue, ST segment modifications, T wave flattening, prolonged QT interval and emergence of U waves [1,3]. The two opposing conditions (hyperkalemia and hypokalemia) could be hence considered somewhat similar in appearance, yet multiple clinical and electrocardiographic criteria could technically still differentiate them (etiology, type of paresis/paralysis, heart rate, P wave amplitude, atrioventricular conduction, QRS complex length, T wave amplitude, presence/absence of U waves). Nevertheless, this case illustrates how both clinical and electrocardiographic findings can be misleading in the absence of arterial blood gas testing, as the patient had a presentation which rather emulated hypokalemia - recent history of profuse diarrhea, spastic paresis, a slight sinus bradycardia with normal P waves (that could have been explained by the Bisoprolol treatment) - while being hyperkalemic.

Conclusion

Both hyper- and hypokalemia are reversible causes of cardiac arrest, and their early detection and management are fundamental in the management of the critically ill patient in whom hydro-electrolytic imbalances are suspected. Although most of the time clearly distinguished by etiology, clinical signs and electrocardiographic elements, the two conditions can have equivocal and even overlapping manifestations. A broad clinical perspective, a good understanding of acid-base disturbances, knowledge of less common adverse effects to medication and rapidly available blood testing (in prehospital settings included) can help avoid misdiagnosing one condition for the other and ultimately improve patient outcome.

Authors' contribution

RSC (Conceptualization; Data curation; Writing – original draft)
SET (Formal analysis; Writing – review & editing; Supervision)
TA (Data curation; Formal analysis; Writing – review & editing)
TI (Formal analysis; Writing – review & editing; Supervision)

Conflict of interest

None to declare.

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