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## Clinical Communications: Adults

### TETRAPARESIS AND FAILURE OF PACEMAKER CAPTURE INDUCED BY SEVERE HYPERKALEMIA: CASE REPORT AND SYSTEMATIC REVIEW OF AVAILABLE LITERATURE

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**Abstract—Background:** In severe hyperkalemia, neurologic symptoms are described more rarely than cardiac manifestations. We report a clinical case; present a systematic review of available literature on secondary hyperkalemic paralysis (SHP); and also discuss pathogenesis, clinical effects, and therapeutic options. **Case Report:** A 75-year-old woman presented to the emergency department complaining of tetraparesis. Her serum potassium level was 11.4 mEq/L. Electrocardiogram (ECG) showed a pacemaker (PMK)-induced rhythm, with loss of atrial capture and wide QRS complexes. After emergency treatment to restore cell membrane potential threshold and lower serum potassium, neurologic and ECG signs completely disappeared. An acute myocardial infarction subsequently occurred, possibly linked to tachycardia induced by salbutamol therapy. We reviewed 99 articles (119 patients). Mean serum potassium was 8.8 mEq/L. In most cases, ECG showed the presence of tall T waves; loss of PMK atrial capture was documented in 5 patients. In 94 patients, flaccid paralysis was described and in 25, severe muscular weakness; in 65 patients, these findings were associated with other symptoms. Concurrent renal failure was often documented. The most frequent treatments were dialysis and infusion of insulin and glucose. Eighty-seven percent of patients had complete resolution of symptoms. **Why Should an Emergency Physician Be Aware of This?:** Severe hyperkalemia is always a life-threatening medical emergency, as it can precipitate fatal dysrhythmias and paralysis. SHP should be considered in the differential diagnosis of neurologic signs and symptoms of uncertain etiology, especially in a subject with kidney failure

or who is taking medications that may worsen renal function. The presence of a PMK does not necessarily impede hyperkalemic cardiac toxicity. © 2015 Elsevier Inc.

**Keywords—**hyperkalemia; paralysis; pacemaker capture failure; kidney failure

#### INTRODUCTION

Severe hyperkalemia is a well-known life-threatening event that can lead to fatal cardiac dysrhythmias or neurologic derangements, such as muscle weakness and paralysis. Paralysis related to high serum potassium levels may be a recurrent and predictable syndrome due to a genetic disease (familial periodic paralysis) or an isolated, acute, and often undiagnosed event; the latter condition is known as secondary hyperkalemic paralysis (SHP). In clinical practice, neurologic symptoms are rarely seen, perhaps because cardiac manifestations begin earlier and are more frequently thought of and managed. We report a case where a patient presented with the chief complaint of hyperkalemia-induced paralysis and subtle, though very serious, cardiac abnormalities. In addition, we present a systematic review of available literature, discussing this condition together with the cardiac and neurologic effects of hyperkalemia, as well as its pathogenesis and therapeutic options.

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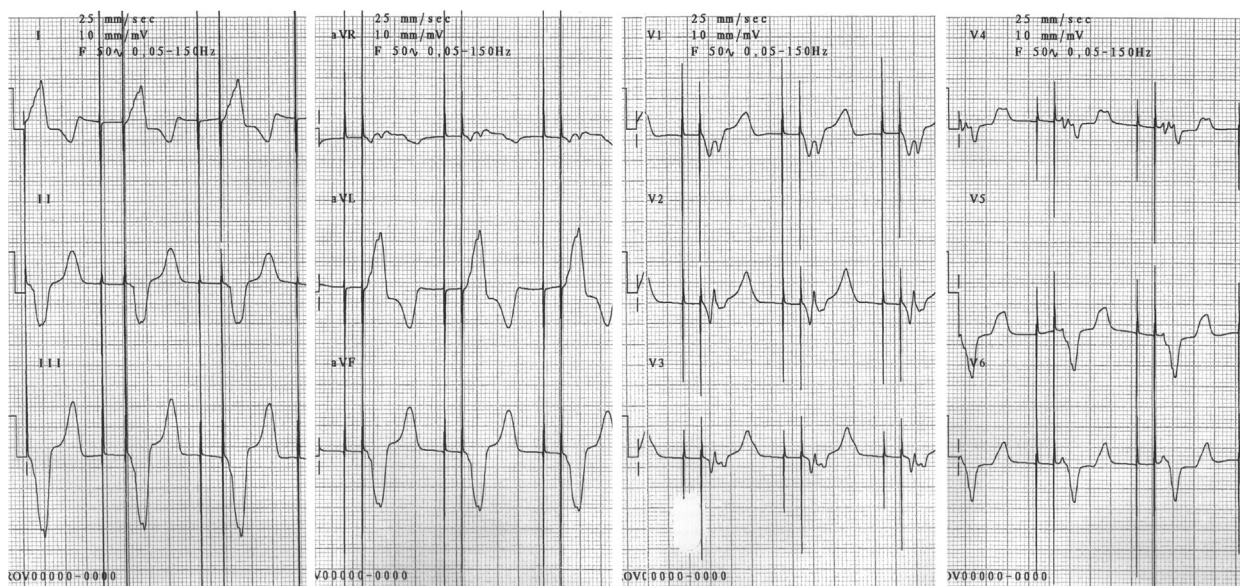
## CASE REPORT

A 75-year-old woman was sent to the emergency department by her general practitioner, who diagnosed “Transient ischemic attack. Drop attack. Patient unable to keep a standing position.” Her history revealed an acute myocardial infarction several years earlier, hypertension, sick sinus syndrome managed with a dual-chamber pacemaker (PMK), and mild chronic kidney disease. Her medications included acetylsalicylic acid (300 mg/d), benazepril (10 mg/d), amiloride/hydrochlorothiazide (5/50 mg/d), lecanidipine (10 mg/d), atorvastatin (20 mg/d), betahistine (8 mg/d), transdermal nitroglycerin, and occasional piroxicam and alprazolam. The patient was alert, oriented, and collaborative. She complained of progressive muscular weakness of a week’s duration, initially in both lower limbs and spreading to the upper limbs in the last 12 h. At the time of admission, she was unable to walk or stand up. Vital signs showed a blood pressure of 150/70 mm Hg, heart rate of 60 beats/min, respiratory rate of 18/min, peripheral oxygen saturation of 96% on room air, and skin temperature of 36.5°C. She denied injuries, as well as fever, vomiting, change in bowel habit, and use of drugs or medications other than those prescribed. On clinical examination, skin and oral mucosa were pale and dry. Heart, lung, and abdominal examinations were normal. On neurologic examination, the lower limbs were completely flaccid, while muscles of the upper limbs had some residual power and greatly decreased tone; she had extreme difficulty moving her fingers and was able to move the upper limbs on the plane, but was unable to raise them against gravity. She was areflexic. There was

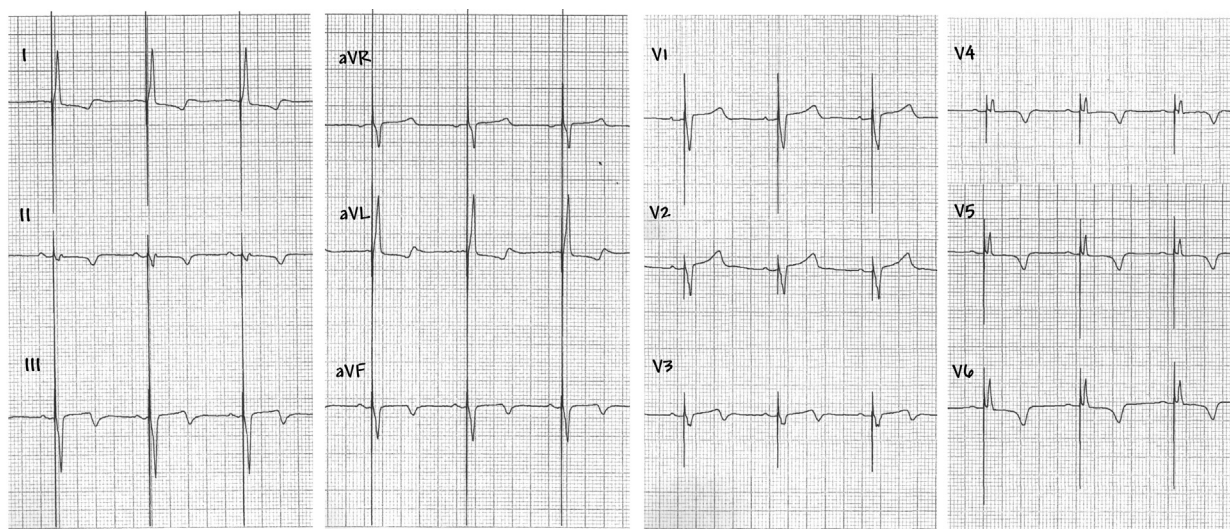
no sensory deficit and plantar responses were bilaterally absent. Facial and lingual motility were normal but speech was difficult and apparently dysarthric. Bladder catheterization revealed 200 mL dark amber urine. Blood tests showed 201 mg/dL urea, 4.4 mg/dL creatinine, 110 mEq/dL chloride, 136 mEq/L sodium, and 11.4 mEq/L potassium. Arterial blood gas analysis showed high anion gap (AG) metabolic acidosis (pH 7.30, pCO<sub>2</sub> 22.1 mm Hg, HCO<sub>3</sub> 10.6, base excess –14.0, AG 26.9). At the electrocardiogram (ECG), a PMK firing at 60 beats/min was noted, with loss of atrial capture and wide (about 240 ms) QRS complexes (Figure 1).

The patient was rapidly treated with an intravenous bolus of 10% calcium chloride (10 mL), infusion of 25 g glucose, and 10 IU insulin over 15 min, followed by nebulization of 15 mg salbutamol. At the same time, an infusion of 80 mEq sodium bicarbonate over 5 min was commenced, followed by an additional 80 mEq and 40 mg furosemide. After excluding ureteral obstruction with renal ultrasonography, a rapid infusion of 1000 mL normal saline was started. Finally, an enteral solution with 15 g polystyrene sulfonate was administered.

After about 40 min, neurologic signs almost completely disappeared, and only moderate generalized weakness persisted. Two hours after hospital admission, serum potassium was 6.6 mEq/L and ECG showed atrial fibrillation with mean heart rate of 105 bpm, ST segment depression, and negative T waves in the inferior and lateral leads. Four hours later, a third ECG (Figure 2) showed spontaneous atrial activity and a PMK spike with regular ventricular capture; an inferioseptal myocardial infarction was now evident and confirmed by



**Figure 1. Electrocardiogram on admission (K<sup>+</sup>: 11.4 mEq/L). Bicameral pacemaker, 60 beats/min; loss of atrial capture; very wide (about 240 ms) QRS complexes; tall and peaked T waves.**



**Figure 2.** Electrocardiogram after 6 h from admission ( $K^+$ : 5.5 mEq/L). Restoration of a spontaneous atrial rhythm, sufficient to not require the activation of pacemaker stimulation; normal PR interval; QRS narrowing (80 ms); reduction of T-wave amplitude; T-wave inversion in left precordial, lateral, and inferior leads.

troponin and echocardiography (akinesis of the inferior septum and the inferior wall, mildly depressed left ventricular systolic function with an ejection fraction of 55%). Glucose/insulin and polystyrene sulfonate were continued for 3 days, until normalization of serum potassium. A subsequent renal color-doppler ultrasound showed a severe stenosis (>90%) of the right renal artery. The patient was discharged home after 10 days with normal serum potassium and creatinine of 2.8 mg/dL.

## LITERATURE REVIEW

We performed a systematic review of available medical literature on SHP using PubMed, Scopus, and EBSCO databases. After excluding all cases of familial periodic paralysis, we found 101 articles reporting cases of SHP. Two articles were not reviewed because they were written in Japanese and Polish (1,2). Finally, we included 99 articles in our revision (the list is available as an online [Supplementary Appendix](#)). For one article (Teixeira, 2009; see [Supplementary Appendix](#)), the full text was not available, so we used only the data reported in the abstract. We found the first description of hyperkalemia-induced muscular weakness in an article published in 1943 (Finch, 1943; see [Supplementary Appendix](#)). Considering that many articles reported more than one case, and including our patient, we analyzed the most relevant information on 119 patients.

Most of the reported cases were male patients ( $n = 118$ ; 85 males [72%]; 33 females [28%]). Hyperkalemic paralysis seemed to affect a rather young population (mean age  $49.6 \pm 17$  years; median 51 years; range: 15–86 years). Mean age of females ( $54 \pm 17$  years; me-

dian 55 years; range: 18–86 years) was higher ( $p = 0.046$ ) than that of males ( $48 \pm 16$  years; median 51 years; range: 15–82 years).

The factors associated with the development of hyperkalemia in the reviewed literature are described in [Table 1](#). In most cases, concurrent chronic or acute renal failure was documented (75 cases [65.8%]), but the serum potassium was increased by concomitant potassium intake (by poisoning or excessive ingestion of potassium-rich foods), dehydration, drugs altering potassium renal reabsorption, or cell lysis.

Data on serum potassium were reported for 116 patients (97.5%). The serum potassium measured during hyperkalemic paralysis ranged from 5.6 to 12.3 mEq/L (mean  $8.8 \pm 1.2$  mEq/L; median 8.7 mEq/L).

**Table 1. Summary of Factors Associated with Development of Hyperkalemia (n = 115)**

Factor	n (%)
Chronic renal failure	39 (33.9)
Acute renal failure	37 (32.2)
Potassium intake (drug or feed)	25 (21.7)
Addison disease	15 (13.0)
Spironolactone	15 (13.0)
Dehydration	11 (9.6)
Hemolysis–cell lysis (cancer, rhabdomyolysis)	10 (8.7)
NSAIDs	7 (6.1)
ACE inhibitors	4 (3.5)
Hypoaldosteronism	4 (3.5)
Others*	13 (11.3)

ACE = angiotensin-converting enzyme; NSAIDs = nonsteroidal anti-inflammatory drugs.

\* Amiloride-hydrochlorothiazide ( $n = 3$ ); co-trimoxazole ( $n = 2$ ), diabetic ketoacidosis ( $n = 2$ ), eclampsia ( $n = 2$ ); arginine ( $n = 1$ ), Cushing syndrome ( $n = 1$ ), digoxin ( $n = 1$ ), and thalidomide ( $n = 1$ ).

One hundred and nine ECGs were described in the reviewed articles. Table 2 describes a summary of findings for 105 (96.3%) ECGs reported as pathologic. The most frequently reported ECG patterns were presence of tall peaked T waves without any other sign (n = 21 [20.2%]); association of absence of P wave plus wide QRS complexes and tall peaked T waves (n = 16 [15.4%]); wide QRS complexes plus tall peaked T waves (n = 13 [12.5%]); and first-degree atrioventricular block plus wide QRS complexes and tall peaked T waves (n = 7 [6.7%]).

Neurologic clinical findings reported in the reviewed literature are shown in Table 3. In 94 patients, a clinical picture of flaccid paralysis was described (56 cases had no other specification, 38 had an ascending pattern), and 25 patients were reported to have severe muscular weakness. Surprisingly, for one case, right hemiplegia was described. In many cases (n = 54 [45.3%]), paralysis or severe weakness was reported as a unique clinical feature, and in the remaining cases, these findings were reported as associated with other sign or symptoms.

The therapeutic options employed in the reviewed cases to treat SHP are shown in Table 4. The most frequently chosen patterns of treatment were dialysis alone (n = 12 [11.3%]); intravenous infusion of insulin and glucose (n = 8 [7.5%]); infusion of calcium and insulin/glucose (n = 6 [5.7%]); infusion of calcium and insulin/glucose followed by hemodialysis (n = 6 [5.7%]); and infusion of bicarbonate, calcium, and insulin/glucose, followed by hemodialysis (n = 5 [4.7%]).

Outcomes of the patient treatment were reported in 113 cases. Ninety-eight patients (86.7%) had complete resolution of symptoms, 1 (0.9%) experienced recovery of strength but unmodified sensory abnormalities, and 14 (12.4%) died. It should be noted that all cases of patients who did not survive were reported in articles dated from 1943 to 1966.

**Table 2. Summary of Electrocardiographic Pathologic Signs in Patients with Hyperkalemia and Abnormal Electrocardiogram (n = 105)**

ECG Sign	n (%)
Tall peaked T wave	75 (71.4)
Wide QRS complex	64 (61.0)
Absent P wave	34 (32.4)
First-degree AV block	14 (13.3)
Sinus or nodal bradycardia	11 (10.5)
IV conduction abnormalities	7 (6.7)
Sine waves	7 (6.7)
Others*	9 (8.6)

AV = atrioventricular; ECG = electrocardiogram; IV = intravenous.

\* ST segment abnormalities (n = 3): prolonged QT duration (n = 2), loss of pacemaker capture (n = 2); low amplitude QRS (n = 1), and short QT duration (n = 1).

**Table 3. Summary of Neurologic Clinical Findings of Patients with Hyperkalemic Paralysis (n = 119)**

Presenting Clinical Features	n (%)
Main sign/symptoms	
Flaccid tetraparesis	56 (47.1)
Ascending flaccid paralysis	38 (31.9)
Muscular weakness	25 (21.0)
Associated sign/symptoms	
Paresthesia, dysesthesia	26 (21.8)
Difficulty breathing	22 (18.8)
Lethargy	12 (10.3)
Sensory loss	7 (6.0)
Dysphagia, difficult in mastication	6 (5.1)
Dysarthria	6 (5.1)
Others*	16 (13.4)

\* Hyper-reflexia (n = 3), mental disorientation (n = 3), myalgia (n = 3); tremor (n = 2); fasciculation (n = 1), aphasia (n = 1), urinary retention (n = 1), bilateral facial palsy (n = 1), and hypertonia (n = 1).

## DISCUSSION

Although in the context of medical emergencies severe hyperkalemia is a rather common clinical condition, neurologic findings seem to occur much more rarely than cardiac symptoms, even if derived from similar pathophysiologic mechanisms. Based on Nernst equation, the ratio of extracellular to intracellular potassium concentration ( $K_e/K_i$ ) determines the value of the resting membrane potential; when  $K_e$  increases, the difference in membrane potential decreases and, accordingly, the activation of sodium conductance and the amplitude and propagation of the action potential are reduced. This phenomenon is clinically evident, especially for cells of nervous tissue and heart muscle.

**Table 4. Summary of Treatment of Patients with Hyperkalemic Paralysis (n = 109)**

Treatment of Hyperkalemia	n (%)
Insulin (i.v.)	68 (62.4)
Dextrose (i.v.)	63 (57.8)
Dialytic treatment*	49 (45.0)
Calcium chloride or gluconate (i.v.)	45 (41.3)
Bicarbonate (i.v.)	29 (26.6)
Isotonic sodium chloride or lactate (i.v.)	24 (22.0)
Sodium polystyrene sulfonate (e.n.)	19 (17.4)
Steroids	19 (17.4)
Salbutamol (inhalation)	11 (10.1)
Furosemide (i.v.)	8 (7.3)
Others†	20 (18.3)

e.n. = enteral administration.

\* Hemodialysis, hemofiltration, or peritoneal dialysis.

† Hypertonic sodium chloride (n = 2), mechanical ventilation (n = 2); acetazolamide (n = 1), aldosterone (n = 1), chlorothiazide (n = 1), digoxin (n = 1), distilled water (n = 1), isoprenaline (n = 1), levarterenol (n = 1), levulose (n = 1), mannitol (n = 1), metaraminol bitartrate (n = 1), neostigmine (n = 1), periston N (n = 1), strophanthin (n = 1), temporary pacing (n = 1), tutofusin (n = 1), and vitamin B (n = 1).

### Neurological Effects of Hyperkalemia

Neurologic manifestations occur more often with hyperthan hypokalemia, but the underlying mechanisms are unclear (3). As in myocytes, in nerve cells, a reduced transmembrane  $K_o/K_i$  ratio results in a decrease in the magnitude of resting membrane potential. Persistent depolarization inactivates sodium channels in the cell membrane, thereby producing a net decrease in membrane excitability that may manifest clinically as muscle weakness (4). The pathogenic process causing neuromuscular paralysis in SHP has been variably localized to muscle, neuromuscular junction, and nerve. There is accruing evidence that altered nerve excitability contributes significantly to muscle weakness by decreased function of the lower motoneuron axonal membrane (5–7). A motor nerve conduction study revealed patterns of response comparable to that observed in demyelinating syndromes (8–10). However, the exact pathogenesis of the slowed conduction velocities and conduction blocks needs further investigations in basic electrophysiology.

Hyperkalemic paralysis may have both a subacute (some days) or very acute (a few hours) onset (11). It typically begins as progressive muscular weakness and evolves as flaccid quadriplegia, usually with an ascending and symmetrical pattern, with the absence of tendon reflexes.

Differential diagnosis should include spinal cord injuries, central nervous system ischemia, botulism, drugs (eg, aminoglycoside antibiotics) and, above all, Guillain-Barré syndrome (in which paralysis has an ascendant progression, respiratory muscles are often affected, and a triggering event, such as fever, should be recognizable) and hyperkalemic periodic paralysis (12,13).

### Cardiac Effects of Hyperkalemia and ECG Findings

The effects of hyperkalemia on the heart are very dangerous and are related to both the absolute value of serum potassium, which modifies the  $K_o/K_i$  ratio, and the rapidity of the increase. Hyperkalemia reduces the resting potential and, consequently, restricts the difference between the resting and threshold potentials to a few millivolts, slowing the speed of conduction and decreasing the responsiveness to electrical stimulation. The consequences on atrial and ventricular myocardium, as well as sinoatrial node and conduction system, are known, and the corresponding ECG changes have been well described as appearing sequentially with rising serum potassium levels (Table 5) (14,15). Although these patterns may occur in only half the patients, their recognition is vital to achieve a rapid diagnosis and to start lifesaving treatment. However, the serum potassium level itself does not always predict ECG changes or degree of cardiotoxicity, and the presence of

**Table 5. Sequential Electrocardiogram Changes Related to Rise in Serum Potassium Levels (14,15)**

Serum Potassium (mEq/L)	ECG Abnormalities
>5.5	T wave: typical (22%): tall, narrow, peaked, tent-shaped; less frequent: tall, peaked, normal duration; peaked, normal duration and normal amplitude QTc: normal or decreased
>6.5	Prolonged QRS Prominent S waves in lead I and in left precordial leads Left or right axis deviation Diffuse ST segment elevation
>7	Decreased P wave amplitude PR prolongation
>8.8	Invisible P wave AV junctional or ventricular escape rhythm AV block of various degrees

AV = atrioventricular; ECG = electrocardiogram; QTc = corrected QT interval.

subtle ECG changes consistent with hyperkalemia should not be the only reason to treat a stable patient: a treatment based on ECG alone could result in errors or delays in almost 15% of cases (16,17).

ECG assessment may be even more complex in the presence of a PMK-induced rhythm. In the case of hyperkalemia, the impulse produced by a PMK may be inadequate because of the increase of depolarization myocardial threshold, which can depress the myocardial response to stimulation in terms of excitability and conductivity (18). In this situation, the PMK may work normally but might not be able to effectively capture the myocardium and induce the depolarization (“exit block”). The depression of intra-atrial or intraventricular conduction limits the PMK impulse propagation and may explain the failure of pacemaker capture, while reduction of sensing function is less common (19). The ECG typically reveals nonspontaneous atrial activity, ineffectual atrial stimuli, and widening of the paced QRS complexes. The loss of capture is typically transient, far away from the PMK implantation time and not due to lead dislodgement or atrial PMK output modifications (20). Because the atrial myocardium is more sensitive to hyperkalemia than ventricular myocardium, loss of atrial capture should be considered a serious sign of impending ventricular asystole and promptly treated (21). Apart from our patient, loss of atrial capture during stimulation with a bicameral PMK has been documented in the literature in only 4 patients with hyperkalemia (22–25).

### Etiopathogenesis of Hyperkalemia

Hyperkalemia is a known complication of therapy with angiotensin-converting enzyme inhibitors (ACE-Is), amiloride/hydrochlorothiazide, and spironolactone, mainly in

patients with renal failure (26–28). The association of potassium-sparing diuretics and ACE-Is is highly dangerous, even in patients with moderate renal insufficiency, as well as in the elderly (29). At therapeutic doses, calcium channel antagonists can cause hyperkalemia by blocking calcium channels and stopping production of aldosterone (30,31). As their role in potassium regulation disorders remains equivocal, these drugs should be used with high caution in patients with renal failure and hyperkalemia (4). In very rare cases, administration of statins can result in muscle toxicity and variable severity of injury to skeletal muscle (8,32). Due to prostaglandin synthesis inhibition, nonsteroidal anti-inflammatory drugs (NSAIDs) can cause hyperkalemia, producing a hypoaldosteronism hyporeninemic syndrome and inhibiting the release of renin (33). In patients with reduction of the circulating volume or with vascular kidney disease, NSAIDs also cause a reduction in renal blood flow and glomerular filtration rate. The risk of hyperkalemia is increased by concomitant use of NSAIDs and ACE-Is (34).

#### *Therapeutic Options and Outcomes*

Even in the presence of neurologic deficits or SHP, the treatment for hyperkalemia is based on a standard therapy. However, despite the potentially life-threatening complications of severe hyperkalemia, there is limited evidence to guide the treatment. Therapeutic options depend on both the level of potassium and cardiac and neurologic signs. Recently published guidelines suggest a five-step treatment strategy: 1) protect the heart; 2) shift  $K^+$  into cells; 3) remove  $K^+$  from the body; 4) monitor  $K^+$  and glucose; and 5) prevent recurrence, pointing out the priority of restoring an adequate difference between the resting and threshold potentials. Emergency treatment (the first two steps) should be given if the serum  $K^+$  is  $\geq 6.5$  mEq/L, with ECG changes, as well as when hyperkalemia is suspected on clinical grounds or based on ECG features (35). The life-saving first-line intervention in the more critical cases is an i.v. 10-mL bolus of 10% calcium chloride (immediately bioavailable and containing three times more calcium than gluconate). Its main electrophysiologic effect is to reduce the difference between the resting and threshold potentials by increasing the threshold potential, so that a near-normal potential difference can be restored (36). Because the effect of calcium on cell membrane ends in almost 30 min, in the presence of hyperkalemic paralysis, some authors suggested ensuring a continuous calcium chloride infusion after giving the initial bolus (37). To reduce serum potassium concentration, administration of insulin and glucose is the most widely used treatment, based on the assumption of potassium entering the cell along with glucose (38). Conversely, to date, sodium bicarbonate is not considered

a first-choice option because several studies report questionable efficacy (39–41). A more recently proposed therapeutic option consists of the administration of high doses of  $\beta_2$  agonists (e.g., 10–20 mg salbutamol via aerosol in 5 min) to induce hypokalemia as a side effect; however, their administration can cause other side effects, such as tremors and tachycardia (42). The reduction of serum potassium can be achieved by facilitating either renal (i.v. thiazide diuretics) or intestinal (oral or rectal polystyrene sulfonate) excretion. However, in the most serious cases, the treatment of choice is hemodialysis or ultrafiltration, which lowers the serum potassium much more rapidly.

#### *Epicrisis of Reported Clinical Case*

The presenting clinical picture was not clear. The clue to diagnosis went from ECG pattern of appropriate PMK spikes, but loss of atrial capture and wide QRS complexes. After emergency treatment and attainment of lower potassium levels, neurologic and ECG signs completely disappeared.

Hyperkalemia may have had a multifactorial etiology. The patients presented a reduction of circulating volume and a vascular kidney disease, and took drugs causing reductions in renal blood flow and glomerular filtration rate. A possible chronic salicylate intoxication was excluded on clinical grounds because the patient was fully alert and with normal mental state, was not febrile, and had no signs of pulmonary congestion.

The cause of the acute myocardial infarction (AMI) remains speculative. We hypothesize a potential role of salbutamol, which could have caused tachycardia and, subsequently, heart rate-dependent MI. A pre-existing MI that could have caused lowered kidney perfusion and sustained the hyperkalemia cannot be excluded with certainty, but is not probable because of the normal hemodynamic parameters and the absence of any evidence of low perfusion. Sodium bicarbonate can contribute to fluid overload, but in this case, pulmonary rales were not observed after the treatment, which also included furosemide and saline, so fluid overload can be confidently excluded. The echocardiogram with normal ejection fraction confirms this hypothesis.

#### **WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?**

SHP is a rare but potentially fatal clinical condition, the precise pathogenesis of which is unknown. High serum potassium levels may affect either muscle cell membrane or peripheral nerves. Even if mild chronic hyperkalemia can be an exception, high serum potassium levels should always be considered as a potentially life-threatening

condition. The effects can be catastrophic, especially when the increase in serum potassium is acute, as a rapid increase may precipitate fatal dysrhythmias and paralysis, potentially leading to respiratory muscle involvement and cardiac arrest. Hyperkalemia presenting with neurologic findings (like SHP) seems to have a low incidence, perhaps because hyperkalemia-induced cardiac toxicity usually becomes more evident before neurologic symptoms. Hyperkalemic paralysis should be kept in mind in the differential diagnosis of acute paralysis. In the presence of not clearly explainable neurologic signs and symptoms, hyperkalemia should always be considered, especially in elderly and dehydrated subjects, and those with kidney failure, even of a mild degree, particularly when the patient is taking medications that may worsen renal function. Great attention must always be paid when searching for characteristic ECG signs, associated or not with hemodynamic impairment; the presence of a PMK is not to be regarded as an obstacle to the identification of typical ECG alterations. Despite the severity of the clinical picture, if diagnosis and treatments are promptly and correctly carried out, the prognosis of SHP can be excellent. Early recognition with prompt treatment cannot only completely reverse paralysis, but can also prevent fatal cardiovascular complications.

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