

Severe Hyperkalemia Masquerading as Acute Ischemic Stroke: A Case Report

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What's Known

- Severe hyperkalemia manifests as symmetrical ascending weakness. Only two cases of hemiparesis associated with hyperkalemia have been reported.
- In the context of hyperkalemia, major contributing factors in the development of focal neurologic deficits are end-stage renal disease, recreational drug abuse, and abnormalities in the medium to large vasculature of the central nervous system.

What's New

- For the first time, we report hemiparesis and severe hyperkalemia in a patient without major contributing factors to the development of focal neurologic deficits.
- Investigation of electrolyte imbalance before the administration of intravenous thrombolysis is recommended in patients with a sudden onset of focal neurologic deficits associated with electrocardiographic abnormalities.

Abstract

Severe hyperkalemia usually presents as cardiac or neurologic manifestations. We report a case of a 63-year-old Caucasian woman, who was admitted to Namazi Hospital, affiliated to Shiraz University of Medical Sciences (Shiraz, Iran) in August 2019. The patient suffered from left-sided weakness and slurred speech for one hour prior to admission. Initially, the patient was treated for acute ischemic stroke, and an intravenous recombinant tissue plasminogen activator (IV-rTPA) was prescribed. However, further investigations showed severe hyperkalemia. Hemiparesis and slurred speech improved significantly with appropriate management of hyperkalemia.

To the best of our knowledge, this is the first case of hyperkalemia masquerading as acute ischemic stroke without evidence of concomitant central nervous system malignancies, large vessel atherosclerosis, or recreational drug abuse. Stroke mimics due to hyperkalemia should be considered in any patient with simultaneous sudden onset of focal neurologic deficits and tall peaked T waves, particularly in the context of renal failure and a history of potassium-sparing drug use.

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Keywords • Hyperkalemia • Ischemic stroke • Paresis • Tissue plasminogen activator

Introduction

Drug-induced hyperkalemia is an electrolyte imbalance that most frequently occurs in patients with renal insufficiency. It can be life-threatening if not diagnosed and treated promptly.¹ Cardiac arrhythmias and neurologic manifestations are the most common consequences of severe hyperkalemia but can be reversed with appropriate treatment in about 90% of cases.² Symmetrical ascending weakness is the most common neurologic presentation of hyperkalemia.³ Hemiparesis is a very rare presentation of hyperkalemia and to the best of our knowledge, thus far only two cases have been reported in the literature.^{4,5} Effiong and colleagues have reported a case of reversible hemiplegia in the context of severe hyperkalemia, end-stage renal disease, and recreational drug abuse.⁴ Moreover, Lee and colleagues reported a case of hemiparesis due to hyperkalemia, end-stage renal disease, and extracranial carotid artery stenosis.⁵ We herein report a rare case of severe hyperkalemia masquerading as acute ischemic stroke. In contrast to previously reported cases,^{4,5} our patient did not have a medical history of end-stage renal disease, recreational drug abuse, or abnormalities of the medium to large-sized blood

vessels of the central nervous system. The present study aimed to improve timely diagnosis and treatment strategies in patients with stroke mimics due to metabolic disturbances.

Case Presentation

In August 2019, a 63-year-old Caucasian woman was referred to the emergency department of Namazi Hospital, affiliated with Shiraz University of Medical Sciences, Shiraz, Iran. She complained of left-sided weakness and slurred speech for one hour prior to admission. Her medical history was consistent with hypertension and ischemic heart disease. Drug history consisted of clopidogrel 75 mg once daily (Actoverco, Iran), metoprolol 25 mg twice daily (Razi, Iran), spironolactone 100 mg twice daily (Iran Hormone, Iran), and losartan 25 mg once daily (Shafa, Iran). Her blood pressure and pulse rates were 90/60 mmHg and 35 beats per minute, respectively. Neurological examination revealed mild dysarthria and left-sided hemiparesis. Based on the scoring system proposed by the Medical Research Council (MRC),⁶ the patient had muscle power of 4/5 and 2/5 in the left upper and lower extremities, respectively. Otherwise, the neurological examination was unremarkable. The brain computed tomography (CT) scan was normal and her National Institute of Health Stroke Scale (NIHSS) score⁷ was 7 points. The patient had severe sinus bradycardia on cardiac monitoring. Electrocardiography (ECG) was performed, which confirmed sinus bradycardia, and showed PR prolongation, widening of QRS complexes, and tall peaked T waves (figure 1). Therefore, we performed emergency arterial blood gas (ABG) and biochemistry sample tests including potassium, blood urea nitrogen (BUN), and creatinine. Considering a clinical suspicion of acute ischemic stroke, the NIHSS score, and eligible time window for receiving intravenous recombinant tissue plasminogen activator (IV-rTPA), IV-rTPA therapy at a dose of 0.9 mg/Kg was prescribed despite abnormalities in ECG. However, no favorable response was obtained from the therapy during the infusion

period. ABG and biochemistry values were pH 7.18 (normal: 7.35-7.45), pCO₂ 38 mmHg (normal: 35-45), HCO₃ 13.8 mEq/L (normal: 22-26), BUN 42mg/dL (normal: 8-23), creatinine 2.3 mg/dL (normal: 0.7-1.2) glomerular filtration rate (GFR) 27.6 mL/min/1.73m² (normal: ≥90), and potassium 9 mEq/L (normal: 3.5-5). Based on previous laboratory test results obtained two months earlier, the patient had normal BUN and creatinine levels; resulting in the diagnosis of acute renal failure. Immediate treatment with calcium gluconate 10% (10 cc over 5-10 min, intravenous (IV) slow infusion), dextrose/insulin (dextrose 10%: 5 ml/Kg IV, IV bolus of regular insulin: 0.1 U/Kg), and kayexalate oral powder (0.3-1 g/Kg every 6 hours) was initiated. While these drugs were being administered, neurological deficits and ECG abnormalities began to improve dramatically. At post-treatment sampling, her potassium level was still 9 mEq/L necessitating emergency hemodialysis. After a single hemodialysis session, the follow-up laboratory data showed marked improvement in serum BUN (30 mg/dL), creatinine (1.6 mg/dL), and potassium (6.4 mEq/L) levels. Both brain magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) were performed to rule out the possibility of acute ischemic stroke with certainty. The results showed a few symmetrical periventricular hyper signal lesions in fluid-attenuated inversion recovery (FLAIR) sequence without diffusion restriction in diffusion-weighted imaging (DWI) and normal angiogram (figures 2A-C). In addition, color Doppler sonography of the carotid and vertebral arteries of both sides and transthoracic echocardiography (TTE) were normal. To rule out a cardioembolic source for the development of focal neurologic deficits, transesophageal echocardiography (TEE) was performed, which showed no abnormalities. Upon hospital discharge, a 7-day ECG Holter monitor test was performed, which showed neither atrial fibrillation nor atrial flutter. Biochemistry values continued to decrease and the serum BUN, creatinine, and potassium levels upon discharge from the hospital were 21 mg/dL, 1.4 mg/dL, and 4 mEq/L, respectively.

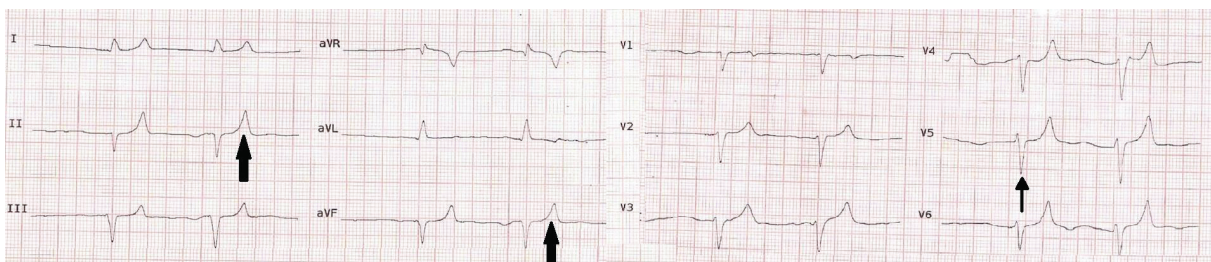


Figure 1: Electrocardiography (ECG) shows severe bradycardia, very low amplitude P waves, increased PR interval, widening QRS complexes (thin black arrow), and tall peaked T-waves (thick black arrows).

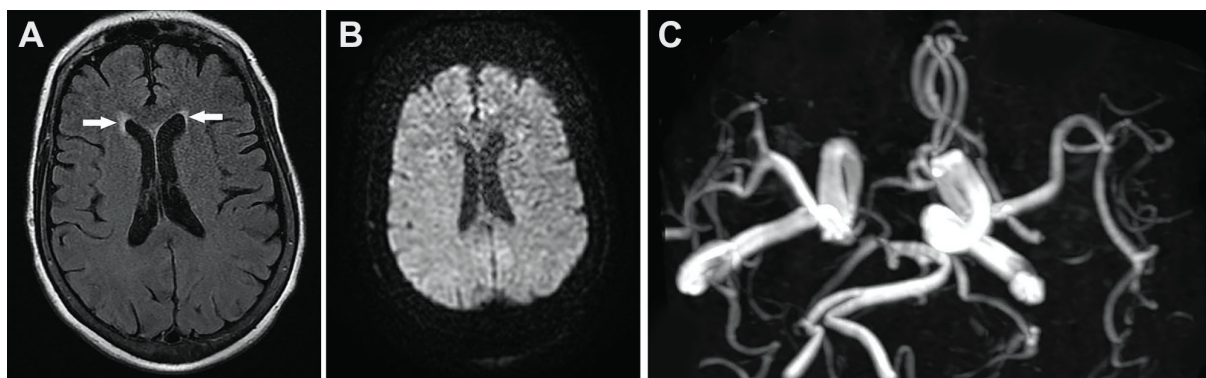


Figure 2: (A) Fluid attenuated inversion recovery (FLAIR) magnetic resonance (MR) image shows a few symmetrical periventricular high signal lesions (white arrows). (B) The diffusion-weighted image is normal. (C) Brain MR angiogram shows no significant stenosis in intracranial arteries.

After hemodialysis, her left-sided weakness, dysarthria, and ECG abnormalities were completely resolved. The patient did not show any new neurological symptoms during 18 months of clinical follow-up and her biochemical laboratory test results were within the normal range. Written informed consent was obtained from the patient for the anonymous publication of her data in this case report.

Discussion

In this report, a case of severe hyperkalemia masquerading as acute ischemic stroke is presented. It highlights various pathogenesis and therapeutic approaches for patients with the sudden onset of focal neurologic deficits associated with hyperkalemia. Risk factors for hyperkalemia include male sex, non-black race, diabetes mellitus (DM), low body mass index (BMI), active smoking, history of coronary heart disease or stroke, low GFR, use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or potassium-sparing diuretics.⁸

Stroke mimics (SMs) are non-vascular conditions that can present as a sudden onset of focal neurologic deficits such as cerebrovascular disease. The reported incidence of SMs ranges from 4.8% to 31%. The most common etiologies of SMs include conversion disorders followed by seizures, migraine, toxic-metabolic disorders, brain tumors, and infections.^{9, 10} Hemiparesis is a very rare presentation of hyperkalemia and only two cases have been reported in the literature so far. The focal neurologic deficits in these earlier reported cases occurred in the context of end-stage renal disease with concomitant stenosis in the central nervous system (CNS), large-sized blood vessels, or recreational drug abuse that cause reversible cerebral vasoconstriction.^{4, 5} However, our patient had no history of end-stage

renal disease or recreational drug abuse. In addition, CNS vasculature evaluation showed no evidence of stenosis or atheromatous plaque formation in the medium- to large-sized blood vessels. Regarding the etiopathogenesis of focal neurologic deficits in hyperkalemia, several etiologies including uremia,¹¹ history of cerebrovascular accident (CVA), or underlying CNS large vasculature abnormalities have been proposed.^{4, 5} However, in our case, it seems that these previously reported mechanisms do not explain “why” and “how” asymmetrical focal neurologic deficits have occurred. Therefore, we propose an additional etiology that would justify our observations. There is a strong relationship between potassium and acid-base disturbances. According to Boscardin and colleagues, acid-sensing ion channels-1 α (ASIC-1 α) exist in CNS and reach a steady state of desensitization at pH around 7.2.¹² The function of this ion channel is to mediate the influx of sodium into intracellular space and generate action potentials. Therefore, the acidic environment puts the neuronal cells in a stunned state resulting in transient loss of nervous system functions. As a result, we hypothesized that in addition to its direct effect, hyperkalemia can indirectly affect CNS via acid-base disturbances. Further studies, with more patients with hyperkalemia associated with a focal neurologic deficit, are required to substantiate the pathophysiology of this disease. Neurologists should be aware of this rare neurological presentation of severe hyperkalemia and consider it in the case of any patient, who presents a sudden onset of neurological deficit accompanying ECG abnormalities on cardiac monitoring.

The overall complication rate of IV-rTPA infusion in SMs, including symptomatic intracranial hemorrhage and orolingual edema, is very low (<1%). Evidently, intravenous thrombolysis is a safe treatment modality in SMs.¹⁰

However, some etiologies of SMs (e.g., toxic-metabolic disturbances) need timely intervention, such as double lumen catheter insertion and hemodialysis, but should be delayed, if the patient receives IV-rTPA. Therefore, differentiating SMs due to toxic/metabolic disturbances from true cerebrovascular events is crucial in the acute phase.

As a limitation of the study, brain and cervical CTA could not be performed to better visualize extra- and intracranial arteries, since the patient was suffering from renal failure.

Conclusion

IV-rTPA therapy should be delayed in patients with focal neurologic deficits having ECG abnormalities compatible with severe hyperkalemia. In such circumstances, sample biochemical analysis should be performed first and as a matter of urgency.

Conflict of Interest: None declared.

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