Case Report

Severe Hypokalemia Mimicking Brain Death

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Abstract

We discuss a case of a 20-year-old female who presented with history of fever, vomiting, and decreased oral intake for 10 days followed by one episode of generalized tonic–clonic seizure and altered sensorium for 5–6 h. On arrival in the emergency room, she had Glasgow Coma Scale 3 (E1V1M1), both pupils fixed and dilated, low blood pressure, low oxygen saturation, and few gasping breaths. She appeared to be brain dead and was assumed to have a very poor prognosis. Investigations revealed severe hypokalemia. She had also suffered acute hypoxic-ischemic injury to the brain. However, she recovered and was discharged about 2 weeks later.

Keywords: Brain death, flaccid paralysis, severe hypokalemia

Introduction

Hypokalemia is a life-threatening condition, as it can lead to cardiac arrhythmias and respiratory muscle paralysis. We present a case of severe hypokalemia that mimicked brain death and initially led to the assumption of a poor prognosis.

Case Report

A 20-year-old female with no significant past comorbidities was brought to the emergency room (ER) in an unconscious state. She had history of intermittent fever, vomiting, and decreased oral intake for 10 days and numbness and weakness in all her limbs for 2 days. Outside, she was diagnosed and treated as a case of diabetic ketoacidosis (DKA) (antibiotics, fluids, insulin and other supportive treatment). However, she became drowsy and had an episode of generalized tonic–clonic seizure (GTCS). She was brought to our center the next day. Travel time was about 4–5 h.

On arrival in the ER, the patient was found to have few gasping breaths, hypotension (blood pressure = 70/40 mm Hg) and low oxygen saturation = 50% on room air. Central nervous system examination showed bilateral dilated pupils nonreactive to light, absent doll's eye reflex, bilateral mute plantar response, and absent deep tendon reflexes (Grade 0/5). The Glasgow Coma Scale (GCS) was E1V1M1. There was a witnessed second episode of GTCS. She was intubated, resuscitated with fluids and shifted to the Intensive Care Unit (ICU). Arterial blood gas analysis revealed high anion gap severe metabolic acidosis and severe hypokalemia (K^+ = 0.93 mmol/L, rechecked in serum) [Table 1]. Chest X-ray was within normal limits. Random blood sugar levels were 300–500 mg/dl. Outside electrocardiogram (ECG) showed junctional rhythm, U waves, wide QRS complexes, and nonspecific interventricular conduction delay [Figure 1]. Initial sequential organ failure assessment (SOFA) and acute physiology and chronic health assessment (APACHE II) scores were 9 and 30, predicting an ICU mortality of 40%–50% and 71%, respectively. After 3 h on mechanical ventilation, her pupils became normal sized and reactive to light and she had spontaneous respirations.

Next day, her GCS was E2VtM2. She had minimal spontaneous movements in both her upper limbs. APACHE II score was 27, predicting a mortality of 51%. Repeat ECG showed sinus tachycardia, U waves, nonspecific ST-T wave abnormalities, and ST-segment elevation in leads I and aVL [Figure 2]. The two-dimensional echocardiography suggested global left ventricular (LV) hypokinesia with poor LV ejection fraction (LVEF) of 15%–20%.

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Table 1: Arterial blood gas analysis and laboratory test results

ABG/test	Day 1	3 h later	Day 2	Day 3	Day 4	Day 5	Day 6 (R/A)	Reference range
ABG analysis								
pH	6.815	7.005	7.370	7.313	7.451	7.420	7.442	7.350-7.450
PO_2 (mm Hg)	526.1	132.2	141.4	147.7	146.4	125.6	80.7	80.0-100.0
PCO_2 (mm Hg)	40.4	28.5	23.0	18.2	24.2	26.9	27.4	35.0-45.0
P/F ratio	526.1	440	470	490	486	416	384.29	>300
Serum Na^+ (mmol/L)	140.5	146.5	146.0	148.1	136.0	132.3	132.2	135.0-148.0
Serum K^+ (mmol/L)	0.93	2.08	1.70	4.07	3.02	3.72	3.35	3.50-4.50
Serum Cl^- (mmol/L)	107.6	113.7	111.3	115.7	102.3	100.1	98.4	98.0-107.0
Serum Ca^{++} (ionized) (mmol/L)	1.226	1.271	0.941	0.805	0.851	0.942	0.926	1.120-1.320
Serum HCO_{3-} (mmol/L)	6.4	6.9	13.0	9.0	16.5	17.0	18.2	22-29
Base excess (mmol/L)	−27.7	−23.2	−10.4	−14.0	−6.0	−6.2	−4.9	−2-+2
Laboratory test results								
Serum creatinine (mg/dl)	2.49	4.34	4.88					0.6-1.5
Serum urea (mg/dl)	42.3		104.5					15-45
SGOT (U/L)	613.0							0-38
SGPT (U/L)	288.8			1189.6		1290.1	789.1	0-41
HIV/HBsAg/HCV	Negative							
Urinary ketones	Negative							
HbA1c	5.0							4-6=Nondiabetic 6-8=Controlled diabetic >8=Uncontrolled diabetic
Serum Mg^{2+} (mg/dl)		3.69						0.6-2.6
Serum iPO_4^{3-} (mg/dl)		2.30						2.3-4.7
Serum albumin (gm/dl)	2.85							3.5-5.2
INR	2.11			2.05			2.50	<1.3
PT (s)	28.1			26.7			33	
APTT (s)	71.3			48.2			121	22-34

PO_2: Partial pressure of oxygen; PCO_2: Partial pressure of carbon dioxide; iPO_4^{3-}: Ionized phosphate; SGOT: Serum glutamic oxaloacetic transaminase; SGPT: Serum glutamate-pyruvate transaminase; HIV: Human Immunodeficiency Virus; HBsAg: Hepatitis B surface antigen; HCV: Hepatitis C virus; HbA1c: Glycosylated hemoglobin; R/A: On room air; INR: International normalized ratio; PT: Prothrombin time; APTT: Activated partial thromboplastin time; LAB: Laboratory; P/F ratio: Ratio of partial pressure of arterial oxygen to fractional inspired oxygen

Magnetic resonance imaging (MRI) of the brain suggested acute hypoxic-ischemic injury with symmetrical abnormal signals in the medial aspect of bilateral thalami and hippocampi with mild diffuse cerebral edema. Imaging differential diagnosis was meningoencephalitis. Lumbar puncture was done [Table 2]. Due to progressively decreasing urine output, rising creatinine levels and persistent metabolic acidosis, hemodialysis was started. Blood cultures were negative, while urine culture showed insignificant growth of *Escherichia coli*.

Our working diagnosis was viral infection with multiorgan dysfunction syndrome (meningoencephalitis, myocarditis, acute liver injury, acute kidney injury, coagulopathy, dyselectrolytemia). Differential diagnoses were Guillain-Barre Syndrome (GBS) and DKA. She was treated with broad-spectrum antibiotics, steroids, anticonvulsants and other supportive treatment.

On Day 4, her GCS was E4VtM6. However, she had weakness in all four limbs (Medical Research Council Grading-Power = 3/5 in the upper limbs and 1/5 in lower limbs) and markedly decreased sensory perception in lower limbs. These recovered 10–12 days later. She was extubated on Day 5. Urine output improved to 20 ml/h by day 5 and normal by day 7. On Day 8, her LVEF was 30%–35%. She was shifted to ward on Day 10 and discharged with stable vitals on Day 16.

Table 2: Cerebrospinal fluid analysis

Test/characteristic	Result	Normal values
Opening pressure (cm H_2O)	13	10-20
Appearance	Clear	Clear
Color	Colorless	Colorless
Proteins (mg/dl)	89.5	15-45
Glucose (mg/dl)	127.9	50-80
WBC (number/microL)	0	0-5
HSV I and II PCR	Negative	Negative

WBC: White blood cells; HSV: Herpes simplex virus; PCR: Polymerase chain reaction

Discussion

Severe hypokalemia is defined as serum potassium level of <2.5 mEq/L. The symptoms of hypokalemia are nonspecific and predominantly related to muscular or cardiac function. Cardiac arrhythmias and acute respiratory failure from muscle paralysis are life-threatening complications that require

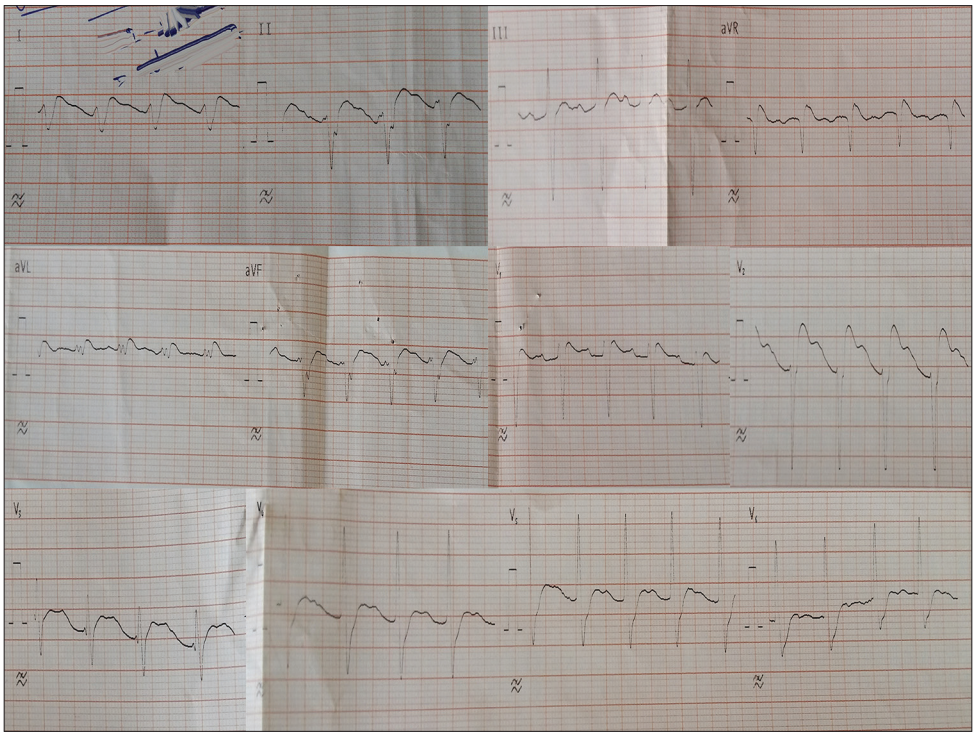

Figure 1: Electrocardiogram taken at outside center showing junctional rhythm, U waves, wide QRS complexes and nonspecific interventricular conduction delay

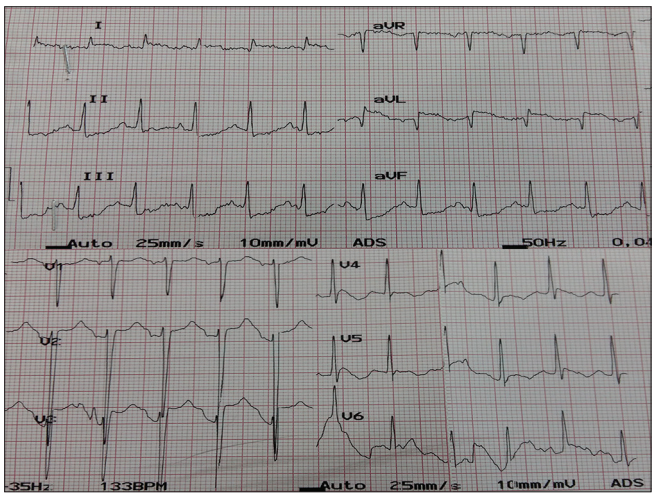

Figure 2: Electrocardiogram on Day 2 showing sinus tachycardia, U waves, nonspecific ST-T wave abnormalities and ST segment elevation in leads I and aVL

immediate diagnosis. Hypokalemia can cause flaccid paralysis, as well as gastric hypomotility and ileus.[1]

Brain death is defined as the irreversible loss of all functions of the brain including the brainstem. The three essential findings in brain death are coma, absence of brainstem reflexes, and apnea. The diagnosis of brain death is primarily clinical.[2]

There have been several reports of severe hypokalemia causing muscular paralysis. A case report by Aggarwal *et al*. described a patient presenting with quadriparesis and difficulty in breathing who was diagnosed with DKA and severe hypokalemia (K^+ = 1.3 mEq/L).[3] Belayneh and Kellerth reported bilateral paralysis of extremities diagnosed as thyrotoxic hypokalemic periodic paralysis.[4]

Our patient's presentation suggested brain death. Further workup revealed that the patient had severe hypokalemia possibly due to poor oral intake, vomiting, osmotic diuresis due to hyperglycemia, and administration of insulin, which are well-known causes of hypokalemia.[1] Diffuse flaccid muscle paralysis and its reversal with correction of hypokalemia suggest hypokalemia to be the primary cause of the paralysis.

On recovery, the patient admitted to previous episodes of weakness that resolved spontaneously. Hence, hypokalemic periodic paralysis could be another cause of hypokalemia, and a low serum potassium during an attack, excluding secondary causes, establishes its diagnosis.[5] However, this diagnosis could not be confirmed.

GBS mimicking brain death has also been reported.[6-8] These patients had a long recovery period, required prolong mechanical ventilation and in general had poor outcomes. In our case, the patient's presentation mimicking brain death, areflexia and albuminocytological dissociation in cerebrospinal fluid suggested GBS. However, because of the patient's rapid recovery with potassium correction, this diagnosis was considered less likely. Further, albuminocytological dissociation, while highly suggestive of GBS, is not specific for it.[9]

DKA with hypokalemia is a well-known entity.[3,10] In our case, the patient's elevated glucose levels, altered mental status and history of fever, vomiting, and decreased oral intake suggested DKA. However, the absence of urinary ketones and a normal HbA1C level rule out this diagnosis.

Limitations

The patient was lost to follow-up, and definite diagnosis could not be reached. Workups for toxic substrates and autoimmune diseases were not done.

Conclusion

Severe hypokalemia can lead to abolition of all muscular contractions and mimic brain death. Its rapid recognition and correction is essential for a favorable outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

References

1. Lederer E, Alsauskas ZC, Mackelaite L, Nayak V. Hypokalemia. New York: WebMD LLC; c1994-2018. Available from: https://emedicine.medscape.com/article/242008-overview. [Last updated on 2017 Oct 24; Last accessed on 2018 Mar 04].
2. Goila AK, Pawar M. The diagnosis of brain death. Indian J Crit Care Med 2009;13:7-11.
3. Aggarwal HK, Jain D, Kaverappa V, Yadav S, Jain P. Quadriparesis as a presenting manifestation of diabetic ketoacidosis: A rare case report. Diabetologia Croat 2013;42:5. Available from: http://www.idb.hr/diabetologia/13no2-3.pdf. [Last accessed on 2018 Mar 04].
4. Belayneh DK, Kellerth T. Thyrotoxic hypokalemic periodic paralysis in an African male: A case report. Clin Case Rep 2015;3:102-5.
5. Amato AA, Brown RH Jr. Muscular dystrophies and other muscle diseases. In: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J, editors. Harrison's Principles of Internal Medicine. 18th ed. United States of America: The McGraw-Hill Companies, Inc.; 2012. p. 3504-5.
6. Rajdev SK, Sarma D, Singh R, Uttam R, Khilnani P. Guillain Barre syndrome mimicking cerebral death. Indian J Crit Care Med 2003;7:50-2.
7. Kang BH, Kim KK. Fulminant guillain-barré syndrome mimicking cerebral death following acute viral hepatitis a. J Clin Neurol 2007;3:105-7.
8. Prasanna D, Madan N, Kapoor S. Fulminant Guillain-Barre syndrome mimicking clinical brain death: A rare condition with bad outcomes. J Med Cases 2015;6:320-1.
9. Ramachandran TS, Sater RA. Acute Inflammatory Demyelinating Polyradiculoneuropathy. New York: WebMD LLC; c1994-2018. Available from: https://www.emedicine.medscape.com/article/1169959-workup. [Last updated on 2017 Jun 08; Last accessed on 2018 Mar 04].
10. Jang TB, Chauhan V, Morchi R, Najand H, Naunheim R, Kaji AH, *et al.* Hypokalemia in diabetic ketoacidosis is less common than previously reported. Intern Emerg Med 2015;10:177-80.