

Electrolyte disturbance induced syncope and seizures in an elderly male- A rare combination of presentation

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Abstract

Electrolyte imbalances are common in elderly population. Ageing alters the milieu interior and impairs homeostatic mechanisms in such a way that geriatric population have a prolong and exaggerated response to stress. Thus, an event--pathological or traumatic--which produces a trivial change in plasma electrolytes of young people may produce major oscillations of plasma levels in the elderly, in clinical practice most electrolyte disturbances in old age are iatrogenic in origin. Adaptation to dehydration in elderly are impaired. So events like vomiting or diarrhea can acutely precipitate electrolyte levels and can cause serious manifestations in elderly. We present a case of 72 year old male who presented with severe hypokalemia, hypomagnesemia, cardiac rhythm disturbances and generalized tonic clonic seizures (GTCS).

Keywords: Electrolytes, Vomiting, Cardiac rhythm, GTCS.

Introduction

Hypokalemia and hypomagnesaemia are associated with cardiac dysrhythmias. Syncope from arrhythmia most commonly occurs in ventricular tachycardia, which accounts for 11% of all cases.¹ Changes in the extracellular potassium level profoundly affects the function of the cardiovascular system and may provoke fatal complications such as QT prolongation, ventricular arrhythmia and even cardiac arrest. Electrolyte disturbances like dysnatraemias, hyocalcemia, hypomagnesemia can also change the seizure threshold of brain leading to convulsions. Again the elderly patients who already have a vulnerable brain are susceptible for permanent damage, if not treated properly. So it is of utmost importance to assess and address dehydration and renal impairment in elderly to avoid sudden electrolyte imbalances there by avoiding catastrophic effects of dyselectrolemias.

Case Report

A 68 year old male presented to the casualty with complaints of abnormal movements involving right arm and disorientation of 30 minutes duration. There was no history of trauma, headache, vomiting, diplopia. There was history of acute non bloody diarrhea 8 to 10 episodes throughout the previous night after he had dinner at a marriage ceremony. On the initial examination patient was drowsy with intermittent tonic movements of the right arm. The patient was intermittently responding to deep painful stimuli. Pulse was 150 beats /min thready, Blood pressure – 80/50 mm Hg. Pupils were normal size reacting to light, bilateral planters were flexor. Skin turgor was lost. In view of seizure like activity, the patient was given iv diazepam 5 mg to which the abnormal movements responded.

Urgent investigations revealed, serum glucose – 150 mg/dl, serum sodium – 142 meq/l, serum potassium – 1.8 meq/l (severe hypokalaemia), serum calcium – 10 mg/dl, kidney and liver function tests were normal. ECG revealed monomorphic ventricular tachycardia (Fig. 1). Immediately, the patient was given inj. amiodarone 300mg bolus over 10mins and was shifted to medicine ICU. Serum potassium was corrected with iv 40 meq potassium chloride which was given over 4 hours. Volume challenge with normal saline was given for hypotension. The seizure like activity stopped. It was presumed that prolonged syncope due to ventricular tachycardia may have triggered syncope induced pseudoseizures or true seizures were triggered by prolonged cerebral hypoxia due to persistent ventricular tachycardia. Subsequent ECGs of the patient showed features of hypokalemia. (Fig. 2, 3). Intravenous ciprofloxacin and metronidazole was added to the treatment regimen. Six hours later the patient had generalised tonic clonic convulsions which lasted for 4 minutes. He was managed with iv diazepam, iv phenytoin sodium. CT scan brain was done which was normal. Repeat serum potassium was 2.2 meq/l and serum magnesium 0.7 meq/l (severe hypomagnesaemia). The spot urine potassium- to - creatinine ratio was 1 mEq/ gram suggesting hypokalemia due to gastrointestinal loss (in our case vomiting). Patient was further infused with 20 meq KCL over 2 hours and 2 grams of magnesium sulphate was given iv bolus followed by 1 gm/hour infusion for next 4 hours and magnesium levels were corrected to 2.6 meq/l and potassium level was corrected to 4.2 meq/l. Anti epileptics were continued for next 24 hours and was gradually tapered.

The diarrhea subsided by the next day. No further seizure activity occurred. Vitals signs were stable. The patient was discharged after 6 days.

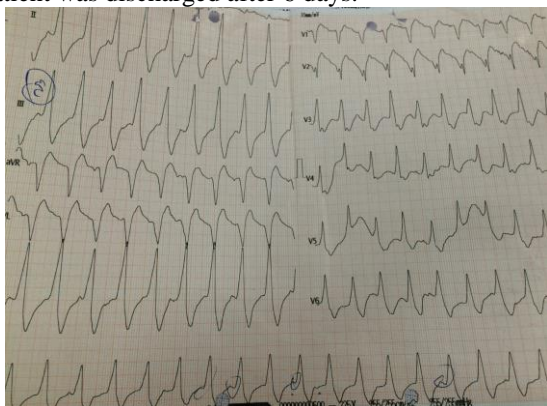


Fig. 1: ECG showing monomorphic ventricular tachycardia

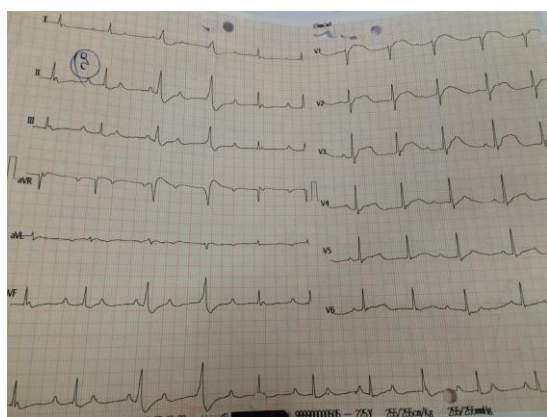


Fig. 2: Variable block, QT interval prolongation, U waves and Interpolated VPC's

Discussion

Electrolyte imbalances like hyponatremia, hypocalcemia, hypomagnesemia are potential causes of seizures. Hypokalemia and hyperkalemia rarely cause seizures.^{2,3}

On the contrary, hypokalemia causes profound ECG changes. The various ECG changes can be:

1. Decreased T wave amplitude
2. ST segment depression
3. T wave inversion
4. PR interval prolongation
5. U waves

The various arrhythmias associated with hypokalemia are:⁴⁻⁷

1. Sinus bradycardia
2. AV blocks
3. ventricular tachycardia
4. ventricular fibrillation
5. torsades de pointes

Ventricular tachycardias both mono and polymorphic can cause syncope that may lead to cerebral hypoxia and seizure like activity. The initial presentation of our patient may be explained by this phenomena, but later our patient had true generalized tonic clonic convulsions which was presumed to be due to severe hypomagnesemia. Sodium disorders, hypocalcemia and hypomagnesemia all can cause generalized tonic clonic, focal (partial) or other types of seizures. The rapidity of electrolyte imbalance increases the risk of seizures hence it is difficult to give cut-off of electrolyte level below which seizures may manifest.^{8,9}

A complete serum biochemistry evaluation for electrolyte levels including sodium, calcium and magnesium is a must to identify the cause of seizure, specially for patients with first episode of seizure.¹⁰ Further it is of much more importance to evaluate electrolyte status in an elderly patient who presents with seizure. As 15 to 30% of acute symptomatic seizures in elderly patients are because of toxic metabolic causes.¹¹ In a review of 375 patients with status epilepticus, metabolic disorder was the primary etiology in 10% and mortality was 40%.¹²

Hypomagnesemia, is defined as a plasma magnesium less than 1.6 mEq/L with a level less than 0.8 mEq/L regarded as severe hypomagnesemia. Magnesium causes depolarization of neuronal network and bursts of action potential by its membrane stabilizing effect and via N-methyl-D-aspartate glutamate receptors.¹³ It also prevents membrane depolarization as a voltage gated dependant calcium channel antagonist.¹⁴

Hypomagnesemia is often symptomatic with serum ionized Mg^{2+} below 1.2mg/dl, presenting as CNS hyperexcitability, cardiac arrhythmias and neuromuscular irritability. GTCS occur in severe hypomagnesemia ($Mg < 1$ mEq/L).²

Treatment of severe symptomatic hypomagnesemia especially with seizures is done by injectable magnesium sulfate 1 to 2 gm over 5 min intravenous followed by 1 to 2 gm per hour iv as infusion for few hours, it can be repeated if seizure persists, reduction of dose required in patients with renal insufficiency.¹⁵ Anti epileptic drugs (AED) as stand alone are ineffective if electrolyte imbalance persists. Electrolyte disorder requires early identification and correction to control seizures and prevention of permanent brain damage. AED is not necessary if the cause for electrolyte imbalance has been corrected.¹⁶

Conclusion

The physicians should therefore be aware of the existence of acute seizures due to electrolyte disturbances and have an understanding of the underlying medical conditions leading to electrolyte imbalance, for this may provide the means of controlling the disease and initiate a rapid and appropriate therapy especially in elderly age group.

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