CASE REPORT

BRUGADA PHENOCOPY IN HYPERKALAEMIA – A RARE ECG FINDING

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Hyperkalaemia is a potentially fatal clinical problem frequently seen in the emergency department (ED). It causes a spectrum of electrocardiogram (ECG) changes such as peaked T-waves, prolonged PR interval, widened QRS complexes, intraventricular/fascicular/bundle branch blocks, etc. Brugada Phenocopy (BrP) is a rare ECG finding seen in severe hyperkalaemia associated with the prevalence of malignant cardiac arrhythmias and all-cause mortality. Unlike Brugada Syndrome (BrS) it is a transient phenomenon and completely resolves with the normalization of hyperkalaemia. Here we report a Brugada Phenocopy (BrP) case observed in a middle-aged male with severe hyperkalaemia.

Keywords: Brugada Syndrome; Brugada Phenocopy; Hyperkalaemia; ECG; Emergency Department

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INTRODUCTION

Hyperkalaemia is a serum potassium level of >5.5 mEq/L and is a potentially fatal condition.[1] The presentation might range from being asymptomatic to severe hemodynamic, cardiac, and respiratory consequences.[2] The most common symptoms are weakness and fatigue but occasionally patient might present with frank ascending muscle paralysis and cardiac conduction abnormalities which ultimately lead to cardiac arrest. The impact of potassium on cardiac function is the major factor contributing to the mortality related to hyperkalaemia, which can be as high as 67 percent.[3,4] Hyperkalaemia depolarizes the cell membrane of the cardiac neural tissue and myocytes, slows ventricular conduction, and decreases the duration of the action potential. These changes produce the classic ECG findings seen in hyperkalemia.[5] Rarely, Brugada type 1, or type 2 ECG patterns can be seen in severe hyperkalaemia which is called Brugada phenocopies.[6,7] Brugada phenocopies are clinical entities that present with ECG patterns like "Brugada Syndrome" yet are induced by various clinical circumstances such as hyperkalemia.[8] BrP can be differentiated from BrS on ECG by the absence of P wave, marked QRS widening, and or abnormal QRS axis.[9] Here we discuss a case of Brugada phenocopy at serum potassium of 8.5 meq/L.

CASE PRESENTATION

A middle-aged man, presented to ED with two to three episodes of vomiting per day, progressive weakness of extremities for two days, and shortness of breath for one day. The weakness started in the lower extremities and progressively involved the upper extremities over two days. He had a history of chemotherapy for a recently diagnosed B-Cell chronic lymphocytic leukaemia with bendamustine one week before the onset of these symptoms. The history of syncope was negative. The family history of sudden cardiac death was negative. No history of addictions or illicit drug use was disclosed by the patient.

Physical examination showed blood pressure of 127/80 mmHg, Heart rate of 52 beats per minute, respiratory rate of 26 breaths per minute, oxygen saturation of 98% on room air, and temperature of 36.5C. He was awake, alert, and oriented to time, place, and person. Neurological examination showed decreased tone in the lower extremities, power of 3/5 in the upper extremities and 1/5 in the lower extremities, and reflexes of 1+ in all the extremities. Pupils were 2 mm in size bilaterally, reactive to light and accommodation. The touch sensation was decreased in all the extremities. The chest was clear to auscultation. The abdomen was soft and non-tender with absent bowel sounds.

In ED, 12-lead ECG showed bradycardia with tall, peaked T-waves, broad QRS complexes, and absent P waves. On a thorough analysis of the ECG, saddleback ST-segment elevation was appreciated in lead V2 and V3 (Figure-1).

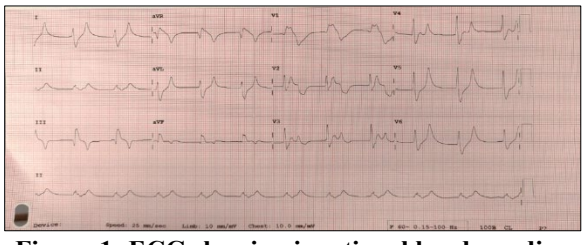

Figure-1: ECG showing junctional bradycardia, Tall peaked T-waves, Broad QRS complexes, Brugada Type 2 Pattern in V2-V3.

Venous blood gas analysis showed potassium of 8.2meq/L (normal range 3.5–5 meq/L) with pH of 7.18 (7.35–7.45) and bicarbonate of 8 meq/L (22–26 meq/L). The point-of-care blood glucose was 145 mg/dl. The rest of the relevant laboratory workup is given in Table 1.

Table 1: Laboratory Workup

Labs	Results	Normal range
Serum Creatinine	20.7 mg/dl	0.9–1.3 mg/dl
Blood Urea Nitrogen	123 mg/dl	6–20 mg/dl
Serum Sodium	120 meq/L	136–145 meq/L
Serum Potassium	8.5 meq/L	3.5–5.1 meq/L
Serum Bicarbonate	11.6 meq/L	20–31 meq/L
Serum Phosphate	20 mg/dl	2.4–4.5 mg/dl
Serum Calcium	6.2 mg/dl	8.6–10.2 mg/dl
Serum Uric Acid	26.7 mg/dl	3.5–7.2 mg/dl

He was nebulized with albuterol, intravenous calcium gluconate was given, and calcium gluconate infusion was started, followed by insulin and dextrose cocktails four times in a row and sodium bicarbonate 50 meq was also given. The ECG was repeated after medical treatment which showed complete resolution of the Brugada ECG pattern, yet peaked T-waves were present, at the serum potassium of 5.7meq/L (Figure-2).

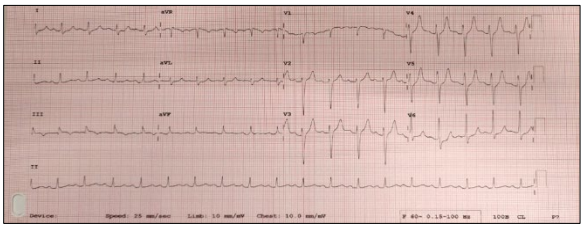

Figure-2: ECG showing resolution of Brugada Pattern.

Considering the diagnosis of tumour lysis syndrome, acute kidney injury with uraemia, hyperphosphatemia, and anticipating refractory hyperkalaemia, a left-sided double-lumen catheter was inserted, and one session of haemodialysis was done in the ED. The patient went through another session of haemodialysis and was discharged in hemodynamically stable condition after two days of admission.

DISCUSSION

Brugada syndrome (BrS) is an autosomal dominant channelopathy and the term was first coined in 1992 by the Brugada brothers.[10] BrS is a set of specific ECG changes with a high incidence of sudden cardiac death in patients with structurally normal hearts. Diagnosis of BrS is dependent on clinical features and typical ECG findings. Any one of the following clinical features: 1. Documented polymorphic ventricular tachycardia (VT), ventricular fibrillation (VF), 2. a family history of sudden cardiac death occurring in people of age less than 45 years, 3. family history of type 1 Brugada pattern ECG changes, 4. inducible VT during electrophysiology study, 5. unexplained syncope may be due to a tachyarrhythmia or nocturnal agonal respiration with specific ECG findings, including coved-type ST-segment elevation greater than 2mm in more than one of V1-V3 leads followed by a negative T wave with or without a drug challenge test in the 12-lead ECG.[10,11] More than 60 gene mutations encoding various ion channels have been identified, the most implicated is loss of function mutation in SCN5A, comprising 20–30% of BrS.

On the other hand, Brugada phenocopies (BrPs) are clinical entities with ECG patterns that are thought to be identical to those of true BrS but are produced by a variety of different underlying diseases like inferior/right ventricular myocardial infarction, pulmonary embolism, electrolyte derangements, and metabolic abnormalities.[12] It has been hypothesized that electrolyte disturbances, such as hyperkalaemia, hypokalaemia, hyponatremia, and hypocalcaemia, can amplify the transient outward current (I_{to}) mediated action potential notch and lead to the subsequent loss of the action potential dome in the epicardium of the right ventricle outflow tract (RVOT), which gives rise to a transmural voltage gradient and consequently produces the Brugada ECG pattern.[13] The clinical implication of BrP remains unknown, however recently a study demonstrated an increased risk of malignant arrhythmias associated with BrP.[14] Hyperkalaemia can produce classic ECG changes, however, hyperkalaemia with ECG changes of the Brugada type 2 pattern is extremely rare.

Why Should an emergency physician be Aware of this?

Hyperkalaemia might cause many ECG alterations, one of which is the crucial and rare Brugada phenocopy. It is critical to recognize this unusual clinical-morphological phenomenon and treat underlying problems such as hyperkalaemia. In the ED, it might be confused with Brugada Syndrome, therefore distinguishing BrP from BrS is critical because an implanted defibrillator is not indicated in BrP. Furthermore, to avoid additional examinations and cardiac procedures, it is important to look for reversible explanations of the Brugada pattern ECG, notably, type 2 and 3.

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AUTHORS CONTRIBUTION

NK: Conceived, designed, and edited the manuscript, and is responsible for the integrity of the research. FA: Review and final approval of the manuscript. JA: Data collection and manuscript writing. AM: Data collection and manuscript writing.

REFERENCES

1. Tran HA. Extreme hyperkalemia. South Med J 2005;98(7):729–32.
2. Hyperkalemia: Practice Essentials, Background, Pathophysiology [Internet]. [cited 2022 Jul 1]. Available from: https://emedicine.medscape.com/article/240903-overview
3. Management of severe hyperkalemia | Read by QxMD [Internet]. [cited 2022 Jun 27]. Available from: https://read.qxmd.com/read/18936701/management-of-severe-hyperkalemia
4. Chew HC, Lim SH. Electrocardiographical case. A tale of tall T's. Hyperkalaemia. Singapore Med J 2005;46(8):429–32.
5. Ameen M, Akbar G, Abbas N, Mirrani G. Hyperkalemia Induced Brugada Phenocopy: A Rare ECG Manifestation. Case Rep Cardiol 2017;2017:9464728.
6. Hunuk A, Hunuk B, Kusken O, Onur OE. Brugada Phenocopy Induced by Electrolyte Disorder: A Transient Electrocardiographic Sign. Ann Noninvasive Electrocardiol 2016;21(4):429–32.
7. Xu G, Gottschalk BH, Anselm DD, Benditt DG, Maheshwari A, Sreenivasan S, *et al*. Relation of the Brugada Phenocopy to Hyperkalemia (from the International Registry on Brugada Phenocopy). Am J Cardiol 2018;121(6):715–7.
8. Clinical manifestations of hyperkalemia in adults - UpToDate [Internet]. [cited 2022 Jun 29]. Available from: https://www.uptodate.com/contents/clinical-manifestations-of-hyperkalemia-in-adults
9. Mizusawa Y, Wilde AAM. Brugada Syndrome. Circ Arrhythm Electrophysiol 2012;5(3):606–16.
10. Brugada syndrome: Clinical presentation, diagnosis, and evaluation - UpToDate [Internet]. [cited 2022 Jun 30]. Available from: https://www.uptodate.com/contents/brugada-syndrome-clinical-presentation-diagnosis-and-evaluation#H3148310224
11. Brugada Phenocopy Definition, Diagnosis, and Differentiation From True Brugada Syndrome | PDF | Medical Diagnosis | Electrocardiography [Internet]. [cited 2022 Jun 30]. Available from: https://www.scribd.com/document/534837179/Brugada-Phenocopy-Definition-Diagnosis-and-Differentiation-From-True-Brugada-Syndrome
12. Littmann L, Monroe MH, Taylor L, Brearley WD. The hyperkalemic Brugada sign. J Electrocardiol 2007;40(1):53–9.
13. Rivera-Juárez A, Hernández-Romero I, Puertas C, Zhang-Wang S, Sánchez-àlamo B, Martins R, *et al*. Clinical Characteristics and Electrophysiological Mechanisms Underlying Brugada ECG in Patients with Severe Hyperkalemia. J Am Heart Assoc 2019;8(3):e010115.

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