JURNAL KEDOKTERAN DIPONEGORO
(*DIPONEGORO MEDICAL JOURNAL*)
Online : http://ejournal3.undip.ac.id/index.php/medico
E-ISSN : 2540-8844
DOI : 10.14710/dmj.v15i5.54093
JKD (DMJ), Volume 15, Number 5, September 2026 : 502-508

TYPE 1 BRUGADA ELECTROCARDIOGRAM PATTERN IN PATIENT WITH RECURRENT HYPOKALEMIA : A CASE REPORT

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Keywords:
Phenocopy,
Brugada syndrome,
Hypokalemia,
Potassium.

Received: 12 October 2025
Revised: 01 March 2026
Accepted: 01 March 2026
Available online: 01 September 2026

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ABSTRACT

Background: Brugada syndrome is a rare and potentially fatal arrhythmogenic disease characterized by ST-segment elevation in the right precordial lead.[1–3] The Brugada electrocardiogram pattern can be triggered by different conditions, such as hypokalemia. Hypokalemia is characterized by a decreased level of potassium in the blood. The causes of hypokalemia vary, leading to different therapeutic approaches depending on the mechanism. Hypokalemia is known to potentially trigger arrhythmias. Therefore, recurrent hypokalemia with the electrocardiogram pattern of Brugada raises questions about their correlation. **Case Illustration:** A 22-year-old male presented to the Emergency Room at Sultan Fatah General Hospital with weakness in all four extremities. An electrocardiogram was performed, which revealed an elevation of the ST-segment in leads V1-V3. Further history-taking indicated no clinical signs or symptoms of arrhythmia, syncope, or sudden death in the patient's complaint, past medical history, or family history. Physical examination showed motor strength in the upper and lower extremities with a symmetrical value of 4 on both the right and left sides. Laboratory tests confirmed severe hypokalemia. The patient was admitted and received parenteral and oral potassium correction. **Conclusion**: Hypokalemia can trigger arrhythmias, one of which is the type 1 Brugada electrocardiogram pattern. Understanding the relationship between hypokalemia and Brugada syndrome can help medical professionals identify, manage, and educate patients and their families about the condition. This will ensure that the patients receive appropriate treatment for the underlying cause and enable early detection of the risk of sudden death.

INTRODUCTION

Brugada syndrome is a fatal arrhythmia that can cause sudden death without any structural abnormalities. This inherited condition is autosomal dominant and is characterized by ST-segment elevation in leads V1-V3 on an electrocardiogram (ECG).[1,2] The SCN5A gene mutations are responsible for causing an ion channel disorder in this syndrome (channelopathy). Many factors can induce the Brugada ECG pattern, one of which is hypokalemia.[1]

Hypokalemia is an electrolyte imbalance marked by a potassium level below 3.5 mmol/L. The etiology of hypokalemia includes a decrease in potassium intake, increased potassium excretion through renal or gastrointestinal excretion (due to diarrhea, vomiting, excessive diuretic and laxative use), and potassium redistribution into cells. Increased excretion is the most common cause.[3,4] Hypokalemia is often asymptomatic; however, symptoms such as muscle weakness and fatigue may occur. In severe hypokalemia, muscle pain and cramps can also occur. Furthermore, severe hypokalemia can lead to heart function disturbances, including the onset of ventricular arrhythmias.[4]

CASE ILLUSTRATION

A 22-year-old male presented to the Emergency Room at Sultan Fatah General Hospital at 5 A.M., complaining of general weakness one day before admission. The weakness started in the evening and was primarily felt in both the right and left thighs, accompanied by muscle pain during physical activity. There was no fever, vomiting, or diarrhea (although he experienced some mild nausea, which had caused

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a slight decrease in appetite). Previously, he had not engaged in heavy physical activities (academic activities were solely conducted online).

In February 2020, he had a similar complaint due to vomiting. However, the condition improved after taking medication for the symptoms. He denied having syncope, palpitations or other symptoms related to heart disease. There were no similar symptoms, syncope, palpitations, or sudden death in his family history. He took anti-nausea medication to reduce the nausea.

During the examination, he was fully alert and able to communicate effectively. His blood pressure was 120/89 mmHg, heart rate was 88 beats per minute, respiratory rate was 22 breaths per minute, body temperature was 36.5 degrees Celsius and oxygen saturation was 99% in room air. The heart examination showed a regular rhythm of heart sounds without a murmur, and the lung examination was unremarkable. There was epigastric pain in the abdomen. The motor strength of both upper and lower extremities was assessed as 4 out of 5, symmetric on the right and left sides, with the interpretation that the patient can oppose gravity but cannot withstand moderate to maximal resistance from the examiner. Laboratory tests showed a potassium level of 1.41 mmol/L and blood urea was slightly increased (49 mg/dL). There were no abnormalities in the heart and lungs on the chest x-ray. The ECG revealed sinus rhythm with ST-segment elevation with the coved type in leads V1 – V3 and a corrected QT interval (QTc) of 647 msec **(Figure 1)**.

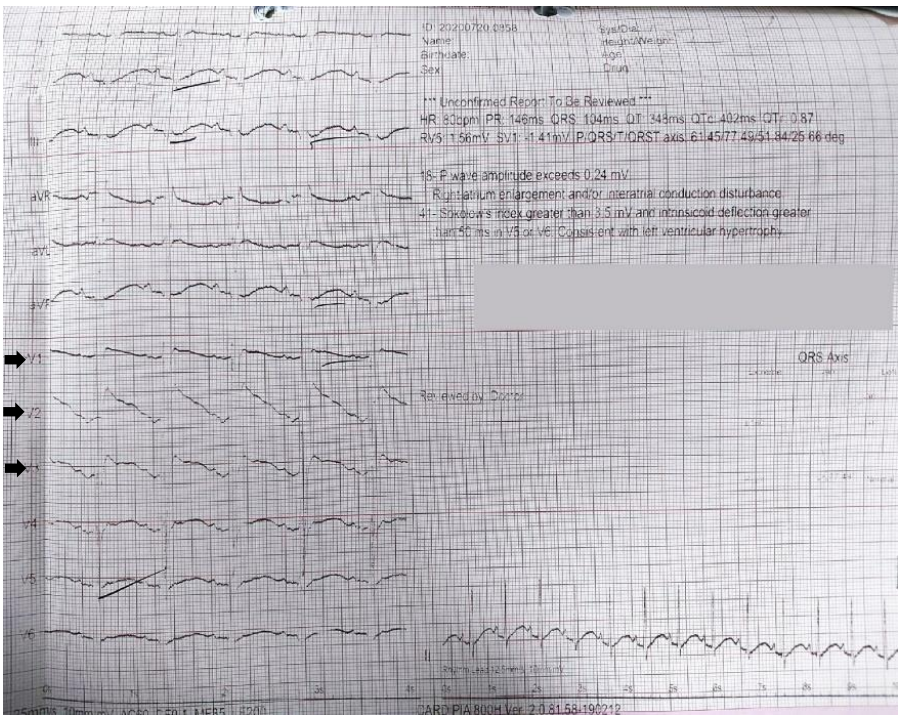

Figure 1. Electrocardiogram at first admission (ST-segment elevation shown with arrows)

The patient received a potassium chloride (KCl) infusion along with Ondansetron 4 mg twice a day, Ranitidine 50 mg twice a day, and KSR 600 mg three times a day. He was admitted for observation. During the hospital stay, his electrolyte levels were evaluated after intravenous administration of KCl, and his clinical condition was monitored. He was referred to the nutrition department for consultation. On the fourth day, the patient was discharged with clinical improvement and prescribed KSR 600 mg three times a day and Vitamin B Complex once daily. He was scheduled for a follow-up at the Internal Medicine Outpatient Clinic Sultan Fatah General Hospital and referred to a hospital with more comprehensive facilities for the evaluation of hypokalemia and a type 1 Brugada ECG pattern. However, he did not go to the referral hospital.

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Three months later, the patient returned to the Emergency Room with a similar complaint, but he also experienced slight palpitations in his chest. He was taking KSR 600 mg three times a day. During the physical examination, he was fully alert and able to communicate effectively. His blood pressure was 130/88 mmHg, heart rate was 81 beats per minute, respiratory rate was 24 breaths per minute, body temperature was 36.5 degrees Celsius, and oxygen saturation was 99 to 100% in room air. His motor strength was assessed as 4 out of 5. Laboratory tests revealed severe hypokalemia (1.73 mmol/L). The ECG was similar to the first admission **(Figure 2)**. No abnormality was found on the chest x-ray.

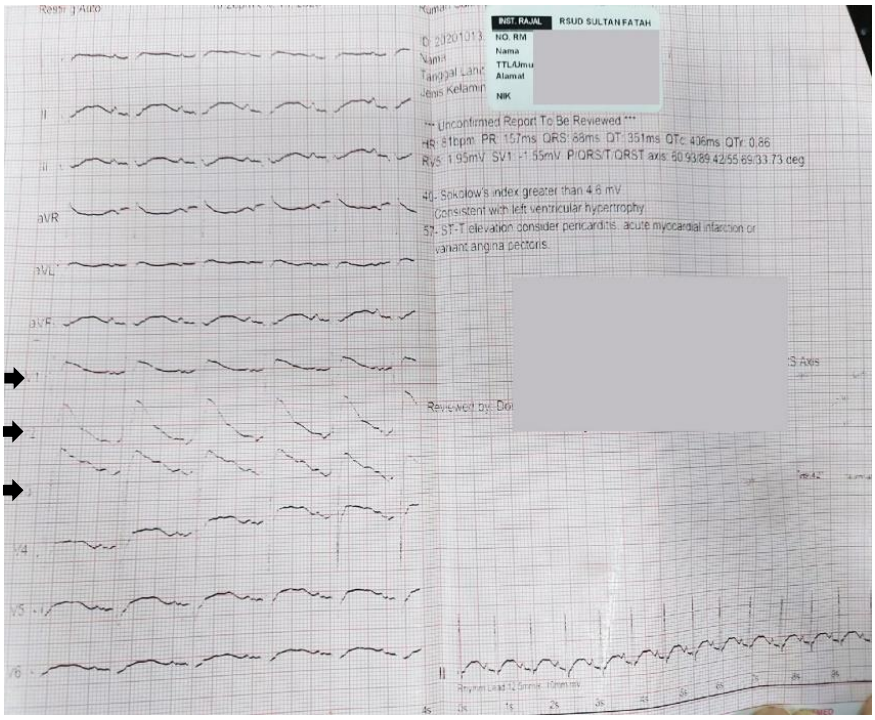

Figure 2. Electrocardiograph at second admission (ST-segment elevation shown with arrows)

He was re-admitted and received a KCl infusion, Omeprazole 40 mg twice a day, KSR 600 mg three times a day, and was encouraged to consume a high-potassium diet. His clinical condition was monitored, and potassium levels were evaluated. He did not have health insurance at the time and declined for futher diagnostic evaluation due to financial issue. He requested discharged with clinical improvement and a referral was planned for evaluation at a hospital with more comprehensive facilities. For the same reason, the patient did not return for follow-up at the Outpatient Clinic.

DISCUSSION

Brugada syndrome contributes to sudden death in young individuals (under 50 years of age) in 4 to 12% of all cases, with a prevalence of 8 to 10 times higher in men than in women.[5] The highest prevalence of Brugada syndrome is found in Asia, particularly in Southeast Asia, with a rate of 3.7 per 1000. The reason Asia has the highest number of cases remain unknown.[6] According to Bezzina et al[7], this condition is attributed to the discovery of haplotype B (HapB) in the Asian ethnic chromosome rather than in Caucasians or Africans. HapB is linked to heart conduction disorders, and its high prevalence in Asians may suggest a link to Brugada syndrome.

Brugada syndrome is hereditary, and it is caused by genetic mutations. SCN5A was the first genetic mutation linked to this anomaly. Currently, more than 350 genetic mutations related to Brugada syndrome have been reported. These mutations are associated with abnormalities in sodium, potassium, and calcium channels, leading to cell dysregulation. However, only 35% of Brugada syndrome cases are definitively due to genetic factors. Of the genetically related cases,

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30% are caused by genetic mutations in SCN5A, and 5% are due to a combination of other genetic mutations. Thus, 65% of cases do not originate from genetic mutations. This may be due to various factors, including copy number variations in the SCN5A gene, pathogenic mutations in unknown genes, or epigenetic factors that lead to variations in characteristic expression within families with Brugada syndrome.

Brugada syndrome has three ECG patterns. Type 1 is characterized by ST-segment elevation with a coved (upward-concave) pattern of ≥ 2 mm followed by negative T-waves in the right precordial leads (V1-V3). Type 2 is characterized by ST-segment elevation with a saddleback pattern, and Type 3 exhibits ST-segment elevation of ≤ 1 mm with a coved or saddleback pattern. Of these three types, Type 1 or coved type is considered to be diagnostically significant.[2] The ECG pattern of Brugada can be induced by consuming sodium channel blockers, fever, vagotonic agents, α-adrenergic agonists, β-adrenergic blockers, tricyclic or tetracyclic antidepressants, first-generation antihistamines (dimenhydrinate), a combination of glucose and insulin, hyperkalemia, hypokalemia, alcohol and cocaine toxicity.[1] Brugada syndrome may manifest at a young age as ventricular tachycardia (VT), ventricular fibrillation (VF), syncope, or can be asymptomatic throughout life. It is often accidentally detected during medical check-ups, so many cases remain unreported.[8]

There is one terminology that is different from Brugada syndrome, namely Brugada phenocopy. What distinguishes it from true Brugada syndrome is the presence of a specific cause that triggers ECG changes, and when this cause is addressed, the ECG returns to normal (sinus rhythm). Additionally, in Brugada phenocopy cases, no history of cardiac arrest, syncope, or sudden death is found in the patient or their family, and the results of provocation tests using sodium channel blockers, such as Ajmaline or Flecainide, are negative.[9] The recommended therapy for patients with high-risk arrhythmias and risk of sudden death in Brugada syndrome is the placement of an Implantable Cardioverter Defibrillator (ICD). For asymptomatic patients with a low risk of arrhythmias, an ICD is not recommended and may even be risky, especially for young, active individuals. Therefore, personalized screening and therapy adjustment based on the patient's risk are necessary.[2]

Hypokalemia is an electrolyte imbalance marked by a potassium level below 3.5 mmol/L. The most common cause of hypokalemia is the loss of potassium in the blood through the renal or gastrointestinal systems. Another cause is the redistribution of potassium into cells and the insufficient intake of potassium-containing nutrients.[5–7] The causes of potassium redistribution into cells are insulin, β-adrenergic receptor agonists, alkalosis, and potassium influx into muscles in cases of Hypokalemic Periodic Paralysis (HypoPP).[3,4]

HypoPP is one of the primary periodic paralysis disorders with a prevalence of 1:100,000, caused by genetic mutations in the calcium channels on chromosome 1 or the sodium channels on chromosome 17. HypoPP is characterized by weakness in one or both limbs, with the absence of tendon reflexes during attacks, accompanied by triggers such as high carbohydrate consumption, rest after exercise, or stress. The onset of attacks typically occurs in the first or second decade of life, with attack duration lasting more than 2 hours. There is often a family history of genetic abnormalities in the skeletal calcium and sodium channels. Clinical improvement was observed following potassium consumption, and diagnostic tests confirmed low potassium levels. This condition must be confirmed not to be caused by secondary hypokalemia/hyperkalemia (renal disorders, adrenal gland issues, thyroid gland issues, renal tubular acidosis, excessive use of diuretics or laxatives) or due to a decrease in muscle potential response caused by repetitive stimulation.

Evaluating the signs and symptoms of hypokalemia is crucial because it can be life-threatening in certain conditions. Patients may be asymptomatic, but they may also complain of weakness, fatigue, muscle pain, palpitations, and even arrhythmias. Physical examination for hypokalemia focuses on changes in the ECG and neurological abnormalities (general or specific muscle weakness). Generally, in hypokalemia, the ECG is characterized by a decrease in the amplitude of the T wave, depression of the ST-segment, inversion of the T wave, prolongation of the PR interval, and the formation of U wave. Arrhythmias that can occur in hypokalemia include sinus bradycardia, VT or VF, and Torsades de pointes.[3] Hypokalemia also enhances

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the function of I_{to} (which is located in the right ventricular outflow tract (RVOT) of the epicardium) during phase 1 of the action potential. This leads to rapid repolarization and creates a transmural voltage gradient between the epicardium and endocardium. This factor contributes to the formation of the characteristic ST-segment elevation in Brugada syndrome, along with the inhibition of intracellular sodium entry (either due to genetic mutations or medication influence). This mechanism supports the hypothesis of the Brugada ECG pattern, which involves repolarization (phase 2 reentry) and depolarization.[1,10,11]

There have been reports indicating that Brugada ECG findings can be seen in cases of hypokalemia. One such report was published by Gazzoni et.al[12] in their case study on the Brugada phenocopies induced by hypokalemia in HypoPP cases. According to this report, the Brugada ECG pattern cannot be considered non-threatening, even in the absence of genetic abnormalities. This is due to recent studies, which show that the temporary Brugada ECG pattern can result in life-threatening events, as also illustrated in the study conducted by Juntila and colleagues. They reported findings on the risk of fatal arrhythmias leading to sudden cardiac death with Brugada ECG pattern in patients without a history of Brugada syndrome. Out of the 47 people who were studied, 5 individuals had the Brugada ECG pattern due to electrolyte imbalances. One of them experienced sudden cardiac death. Due to these findings, Juntilla et. al[13] concluded that ECG changes in acute conditions, including electrolyte imbalances, should be considered as a risk factor for life-threatening arrhythmias. Therefore, the discovery of Brugada ECG pattern and potential arrhythmia-triggering risk factors should be aggressively treated.

Patients with hypokalemia, especially those with genetic disorders such as HypoPP, are at risk of fatal arrhythmia. Quoting from "Letter to the Editor: Possible Brugada Phenocopy Induced by Hypokalemia in a Patient with Congenital Hypokalemic Periodic Paralysis," Anselm et. al[14] added that the type 1 Brugada ECG pattern in HypoPP may be due to hypokalemia at that time or there may be sodium channel abnormalities in skeletal (HypoPP) or myocardial (Brugada syndrome). Therefore, provocation tests become crucial to assess whether there is an involvement of ion channel abnormalities in the myocardium or not.[14,15]

In this particular case, a 22-year-old Asian male presented with weakness in all four extremities, without the presence of common precipitating factors for hypokalemia, such as excessive fluid loss. He had previously experienced similar symptoms due to vomiting, which were improved with medication. There was no family history of weakness in all four extremities or cardiovascular disease related. Notable findings from examinations included reduced motor strength in all four extremities, Brugada type 1 ECG pattern, severe hypokalemia and reccurence within three months without any identifiable precipitating factors. Based on these, it was suspected to be HypoPP as it met the clinical criteria for HypoPP according to the 87th European Neuromuscular Centre (ENMC) International Workshop in 2000. This suspicion was further supported by the Brugada type 1 ECG findings, suggesting possible involvement of overlapping channel disorders, although hypokalemia itself can trigger ECG abnormalities. However, confirmation of channelopathy was not feasible due to limited supportive facilities, and the absence of a cardiologist and cardiac electrophysiologist, necessitating referral to a hospital with comprehensive facilities. At the peripheral hospital setting, initial management and patient and family education about the condition should be provided to increase the patient's awareness of their condition.

The dynamic nature of the Brugada pattern on ECG, which can sometimes appear asymptomatic, makes it very challenging to estimate the overall incidence rate of Brugada syndrome.[8] Indonesia itself does not have incidence data for Brugada syndrome, primarily because the disease is challenging to identify, especially since most cases are asymptomatic. With the highest prevalence of Brugada syndrome in the Southeast Asian population and Indonesia being the most populous country contributing 40% to the Southeast Asian population, awareness of Brugada syndrome, especially among emergency room doctors, would greatly assist patients in receiving appropriate care, including considerations for implantable cardioverter-defibrillator (ICD) placement.[1,8]

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CONCLUSION

Hypokalemia can cause Brugada patterns to appear on an ECG, making it one of the triggering factors. Recurrent hypokalemia during acute attacks with a type 1 Brugada ECG pattern indicates potential overlapping channel disorders in the myocardium and skeletal muscle, which requires further evaluation. This evaluation is necessary due to the high risk of arrhythmias, which, if not promptly detected, can lead to sudden death. Clinicians must have a basic knowledge of the clinical symptoms, ECG findings, and supportive examinations encountered in patients with Brugada syndrome or Brugada phenocopy. This knowledge helps ensure patients to receive the specialist or subspecialty services they need. In this case, the patient is still considered to have a Brugada phenocopy since there are no symptoms such as syncope, arrhythmia, or sudden death in the family, and it has not been confirmed by provocation tests or genetic testing.

ETHICAL APPROVAL

Written informed consent was obtained from the patient for the publication of this case report.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

FUNDING

No specific funding was provided for this article.

AUTHOR CONTRIBUTIONS

Writing—original draft preparation, review and editing, Melinda Veronica.
Supervision, Yana Suryana.

ACKNOWLEDGEMENTS

This work was supported by Sultan Fatah General Hospital, Demak, Central Java.

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