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Brugada pattern associated with febrile gastroenteritis: A case report

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Case Report

ABSTRACT

The Brugada pattern is an electrocardiographic manifestation that mimics true Brugada syndrome, potentially triggered by transient and reversible conditions. This case describes a 66-year-old Indonesian male with acute gastroenteritis, characterized by mild hypokalemia (3.2 mmol/L) and fever, who exhibited a Brugada type 1 ECG pattern. The patient experienced over 10 episodes of diarrhea, more than seven episodes of vomiting, and a fever of 39 °C, with no cardiac symptoms or family history of sudden cardiac death. The electrocardiogram (ECG) showed coved-type ST-segment elevation greater than 2 mm, accompanied by inverted T waves in leads V1-V3, which is indicative of a Brugada type 1 pattern. Laboratory results confirmed mild hypokalemia and leukocytosis, while echocardiography and chest X-ray showed no structural abnormalities. The patient received a comprehensive treatment plan including mineral sorbitol infusion, paracetamol, cefixime, potassium supplementation, and notably, cilostazol—a phosphodiesterase-3 inhibitor not previously documented for gastroenteritis-associated Brugada pattern. After three days of treatment, the Brugada pattern resolved. This case offers important insights for clinical practice in Indonesia by considering Brugada pattern in febrile gastroenteritis with ST-segment elevation, even with mild hypokalemia. It also raises the possibility that the combination of electrolyte correction and cilostazol may facilitate ECG normalisation. Further studies are needed to evaluate the therapeutic potential of cilostazol and to improve diagnostic accuracy in differentiating transient Brugada patterns from Brugada syndrome.

INTRODUCTION

Brugada patterns are electrocardiographic manifestations that resemble actual Brugada syndrome (BrS) and are frequently caused by reversible conditions. Brugada phenocopy (BrP) has been associated with metabolic problems, including hypokalemia. Although febrile conditions such as gastroenteritis can induce Brugada-like ECG patterns, they are currently excluded from the BrP classification due to differing pathophysiological mechanisms and prognostic implications. This case describes an uncommon Brugada type 1 ECG pattern in a 66-year-old Indonesian male with febrile gastroenteritis and mild hypokalemia (3.2 mmol/L), which was treated with a combination of electrolyte correction and cilostazol.[1]

This case is particularly noteworthy as it occurred with only mild hypokalemia, despite Brugada pattern being previously associated with severe electrolyte imbalances (K+ <2.6 mmol/L) as reported in prior literature.[2] It suggests that other factors, such as systemic inflammation from gastroenteritis, may contribute to the ECG changes.[3] Diarrheal diseases often precipitate electrolyte disturbances, but cardiac screening is rarely prioritized.[4] This case highlights the importance of

integrating ECG screening into the management of febrile gastroenteritis in Indonesia, where diarrheal illnesses are endemic. The effective application of cilostazol, usually designated for congenital Brugada syndrome, to alter the Brugada pattern in this metabolic scenario provides fresh therapeutic perspectives, notably relevant in Indonesia, where diarrheal diseases continue to be a primary cause of hospitalization.[5] This report enhances Indonesian medical practice by illustrating the importance of vigilant ECG monitoring, even in mild electrolyte disturbances resulting from gastroenteritis, and suggesting that cilostazol may have broader applications in cases of reversible Brugada pattern.

CASE DESCRIPTION

A 66-year-old man came to the hospital with complaints of diarrhea for one day. The diarrhea was accompanied by nausea, vomiting, and fever that had begun several hours earlier. The patient reported experiencing diarrhea with a stool frequency exceeding 10 times and vomiting more than 7 times within the last 24 hours. The stool was yellow, liquid in consistency, and without any dregs, mucus, or blood. The patient denied any history of fainting, history of heart attacks, and family history of sudden cardiac arrest. Physical examination showed the following vital signs: blood pressure of 140/80 mmHg; pulse 110 times per minute; 24 breaths per minute; SpO_2 98%; temperature of 39^{o}C with anthropometric data, body weight 54 kg and height 161 cm (BMI 22.84, with normal impression). Cardiopulmonary examination revealed no abnormalities on inspection, palpation, and auscultation. Abdominal examination revealed the impression of increased bowel sounds with a frequency of 22 per minute, with signs of mild to moderate dehydration, including dry mouth, thirst, and headache.

An ECG examination revealed ST segment elevation with a coved morphology above 2 mm, accompanied by inverted T waves in the precordial leads V1–V3, which suggests Brugada type 1 syndrome, as shown in Figure 1 (A). Echocardiography showed normal cardiac chamber dimensions, normal left ventricular geometry, normokinetic wall motion, and normal global and segmental systolic function of the left ventricle, with an ejection fraction (EF) of 56%. Diastolic function of both the left and right ventricles was within normal limits. The mitral and aortic valves demonstrated normal anatomy and function, while the tricuspid valve showed mild regurgitation with a tricuspid valve gradient (TVG) of 17 mmHg. No pericardial or pleural effusion was detected. The chest X-ray findings were normal, with clear lung apices, no infiltrates or masses, normal bronchovascular markings, sharp costophrenic angles, smooth diaphragm, and a cardiothoracic ratio (CTR) of less than 0,5. Blood laboratory examinations showed leukocytosis, with a leukocyte level of $13.6x10^3$/uL. High-sensitivity Troponin (HsTroponin) level was within normal level (<1.5 ng/l). Electrolyte analysis showed a slight decrease in chloride (97 mmol/L), a normal sodium level (136 mmol/L), and mild hypokalemia with a potassium level of 3.2 mmol/L.

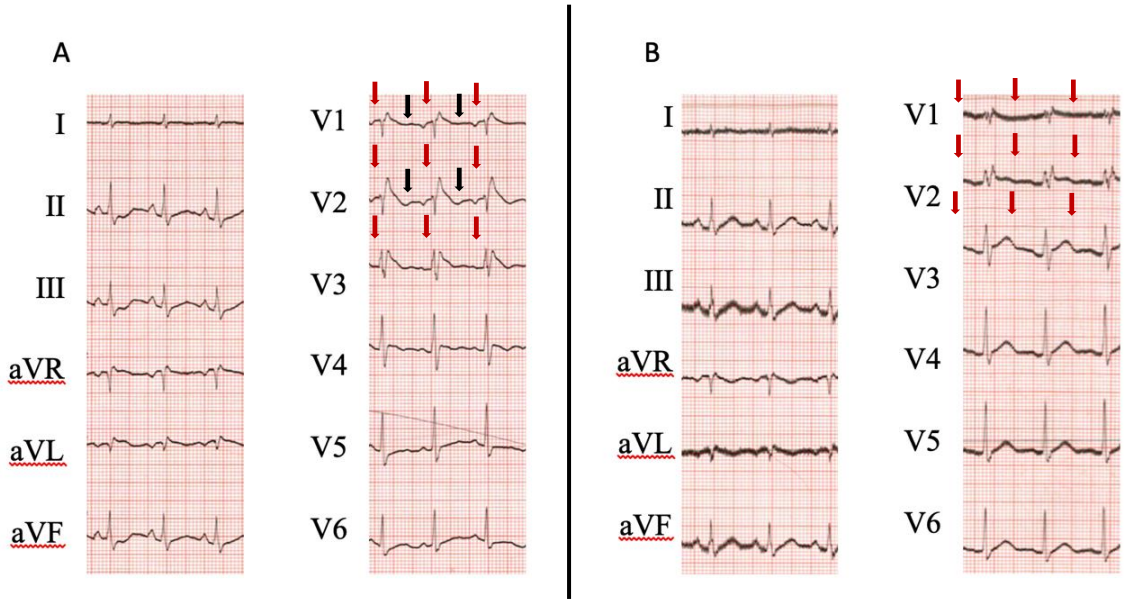

Figure 1. (A) ECG during acute gastroenteritis reveals a coved-type ST segment elevation in precordial leads V1-V3 (red arrows), followed by an inverted T wave (black arrows), consistent with a Brugada type 1 pattern. (B) Follow-up ECG three days later, after clinical resolution of the infection and correction of electrolyte imbalance, demonstrating normalization of the ST segment and T waves (red arrows).

Based on the overall diagnostic process, the patient was diagnosed with a Brugada pattern triggered by acute gastroenteritis. The patient was treated with mineral sorbitol infusion at 60 cc per hour as a fluid maintenance, paracetamol 500 mg three times daily, cefixime 200 mg twice daily, potassium chloride tablet 600 mg once daily, and cilostazol 100 mg once daily. Cilostazol is used to normalize ST-segment elevation patterns observed on the patients' ECGs.[6] This case report demonstrates the efficacy of cilostazol in the treatment of Brugada pattern. It has been shown to reduce coved-type ST-segment elevation and prevent the occurrence of ventricular fibrillation (VF). After three days of therapy, the patient showed significant improvement, with resolution of diarrhea, vomiting, and fever. Follow-up ECG also showed normalization of the ST-segment, as shown in Figure 1 (B).

DISCUSSION

This case demonstrates a rare presentation of Brugada pattern triggered by mild hypokalemia (K+ 3.2 mmol/L) and systemic inflammation due to acute gastroenteritis, with rapid ECG normalization following a combination therapy of potassium chloride tablet 600 mg and cilostazol (100 mg). According to the current BrP classification, febrile-induced Brugada ECG patterns are excluded due to potential overlap with unmasked BrS. Although this case fulfilled several BrP diagnostic criteria, the presence of fever excludes this case from formal classification. Nevertheless, the transient and reversible nature of the ECG pattern, along with the absence of clinical or family history of BrS, supports the notion of a Brugada pattern rather than true congenital Brugada syndrome.[1]

The underlying mechanisms of the clinical manifestations in this case likely involve two pathways: hypokalemia and systemic inflammation marked by fever. Increased fluid excretion due to gastroenteritis can lead to electrolyte imbalances, a known trigger for Brugada pattern. A 2020 study by Mosav et al. reported that among 437 patients with gastroenteritis, 344 (78.7%) experienced moderate dehydration, 90 (20.8%) severe dehydration, and 3 (4.4%) mild dehydration.

Additionally, 41.6% had sodium imbalances and 17.2% potassium imbalances.[7] In our case, electrolyte analysis revealed hypokalemia (3.2 mmol/L). Hypokalemia reduces extracellular potassium levels, enhancing the transient outward potassium current (I_{to}) in the right ventricular outflow tract (RVOT), which deepens the action potential notch. This amplifies the transmural voltage gradient, producing the characteristic coved-type ST-segment elevation observed in Brugada pattern.[8]

Hyperthermia is a well-established trigger for the Brugada pattern, as it disrupts cardiac ion channels and may reveal the ECG pattern of Brugada.[9] This phenomenon has been documented in cases where fever or hyperthermia exhibited ST-segment elevations typical of BrS[10] The underlying mechanism involves mutations in the *SCN5A* gene, which encodes cardiac sodium channels, resulting in reduced sodium currents and altered cardiac repolarisation.[11] The combined effects of hypokalaemia, which is frequent in gastroenteritis, and inflammatory mediators enhance the transient outward potassium current (I_{to}). This results in a deepened action potential notch in the right ventricular outflow tract (RVOT), thereby producing the characteristic coved-type ST-segment elevation associated with the Brugada pattern.

The diagnostic criteria for BrP include: (1) the presence of a type 1 or 2 Brugada pattern on ECG examination; (2) an identifiable and reversible underlying condition; (3) resolution of the Brugada ECG pattern after treating the underlying condition; (4) absence of clinical symptoms, prior illnesses, or a family history suggestive of Brugada syndrome, sudden cardiac death, or structural heart disease. Additional supportive criteria include (5) a negative provocative test using sodium channel blockers (e.g., procainamide, flecainide, or ajmaline), and (6) a negative *SCN5A* gene mutation result. A diagnosis of BrP is generally considered valid when the first four criteria are met, while the remaining two serve as optional but supportive evidence..[12,13]

In our case, four major criteria for BrP were met: (1) a Brugada type 1 ECG pattern with coved-type ST-segment elevation ≥2 mm in leads V1–V3; (2) an identifiable and reversible clinical trigger—febrile gastroenteritis with mild hypokalemia; (3) complete resolution of the ECG pattern following clinical recovery and electrolyte normalization; and (4) absence of personal or familial history of Brugada syndrome, sudden cardiac death, or structural cardiac disease. However, it should be noted that according to the current classification, fever-induced Brugada patterns are excluded from BrP due to potential overlap with unmasked Brugada syndrome. Despite this, the transient and fully reversible nature of the ECG changes in this patient, along with the absence of other clinical indicators suggestive of Brugada syndrome, supports a diagnosis of a Brugada pattern that closely resembles BrP.[12]

The administered combination therapy, which included mineral sorbitol infusion, paracetamol, cefixime, potassium supplementation, and cilostazol, effectively targeted both the symptoms and the underlying aetiology of the patient's condition. The mineral sorbitol infusion restored fluid volume and corrected electrolyte imbalances, normalising cardiac electrical conduction and resolving the Brugada pattern, while reducing arrhythmia risks associated with hypokalaemia.[14] Paracetamol played a crucial role in controlling fever, which can exacerbate cardiac ion channel dysfunction by destabilizing sodium channels (*SCN5A*) and promoting additional ST-segment elevation.[10] Potassium chloride tablets effectively addressed the patient's hypokalaemia (3.2 mmol/L), thereby preventing serum potassium fluctuations that may disrupt cardiac repolarisation.[15] Cefixime, a third-generation cephalosporin, targets bacterial pathogens in the gastrointestinal tract, thereby reducing systemic inflammation and metabolic stress that exacerbate the Brugada pattern.[16] Cilostazol, a phosphodiesterase-3 inhibitor, stabilises the ECG through the elevation of intracellular cAMP, enhancement of calcium influx via L-type channels, and inhibition of the transient outward potassium current (I_{to}), resulting in the normalisation of ventricular repolarisation.[17] This comprehensive, multimodal approach underscores the importance of addressing both metabolic triggers (such as hypokalemia and infection) and the electrophysiological substrate (I_{to} modulation) in the effective management of Brugada pattern.

This gastroenteritis-associated Brugada pattern provides new insights compared to existing medical literature. First, the adjunctive use of cilostazol, a phosphodiesterase-III inhibitor, introduces a novel therapeutic dimension. Cilostazol is generally utilised in congenital Brugada syndrome to mitigate arrhythmias through the enhancement of cAMP-mediated L-type calcium currents.[6,17] In this context, it is used to accelerate ECG normalisation, likely by counteracting the dominance of transient outward potassium current (I_{to}) and stabilising repolarisation.[18] Second, the combination of cilostazol with electrolyte correction, previously unreported in Brugada pattern triggered by gastroenteritis, contrasts with prior management that solely on electrolyte repletion.[19] The multidisciplinary approach combining infection control through cefixime and cardiovascular stabilization using cilostazol appears in contrast to fatal Brugada pattern cases in pulmonary tuberculosis with AIDS, where antibiotic therapy alone proved inadequate for managing cardiac ion channel dysfunction.[5,20]

The limitations of this case are the absence of a sodium channel blocker provocation test, which is necessary to exclude true Brugada syndrome definitively. Nevertheless, the absence of arrhythmia symptoms and a family history of sudden cardiac death provide support to the Brugada pattern diagnosis. Additionally, this case report is inherently limited by its retrospective and single-case design, which restricts generalizability and precludes definitive causal inference.

According to the National Consensus on Acute Diarrhoea Management (2024), 4.3% of diarrhoea cases occur across all age groups in Indonesia.[21] This statistic highlights the importance of ECG screening in febrile gastroenteritis accompanied by electrolyte imbalances, even in cases of mild hypokalaemia. Although fever-induced Brugada patterns are excluded from the current formal classification of Brugada phenocopy, cases like this illustrate the need for clinical vigilance. This case presents two primary insights: (1) febrile gastroenteritis with mild electrolyte disturbances may unmask Brugada ECG features, and (2) cilostazol may accelerate ECG normalization in certain cases, requiring further research in a larger cohort to validate its efficacy.

CONCLUSION

This case highlights the importance of including Brugada patterns in the differential diagnosis of patients presenting with febrile gastroenteritis and mild hypokalemia, accompanied by ST-segment elevation on ECG. It emphasizes that the potential impact of systemic inflammation on cardiac electrophysiology should not be overlooked. Although fever-induced patterns are excluded from current BrP classification, the resolution of ECG abnormalities following electrolyte correction and cilostazol administration suggests a potential therapeutic benefit. These findings highlight the need for further investigation into the role of cilostazol and the management of transient Brugada patterns associated with systemic inflammatory states..

CONFLICT OF INTEREST

There are no conflicts between the patient and the authors. We attached the patient's informed consent.

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The authors said gratitude to the patient and his family for their contribution to this case report

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are not publicly accessible due to patient confidentiality and institutional restrictions. However, the authors can provide access to anonymized data upon reasonable request. Requests for data access can be submitted to the corresponding author via email, subject to approval by the institutional ethics committee.

SUPPLEMENTARY MATERIALS

The supplementary material comprises laboratory results, chest X-rays, echocardiography and electrocardiograms, both prior to and following therapy.

AUTHOR CONTRIBUTIONS

LCE conceptualized and defined the study objectives, coordinated the manuscript preparation, and oversaw the writing process. AMMA conducted the literature review, analyzed clinical data, and evaluated the diagnostic criteria for Brugada syndrome and Brugada phenocopy, contributing significantly to the discussion section. IP was responsible for the interpretation of diagnostic studies, including electrocardiograms and echocardiograms, and took part in the critical review and editing of the manuscript to ensure accuracy and clarity. All authors contributed collaboratively to the completion of this case report on Brugada phenocopy triggered by acute gastroenteritis.

DECLARATION OF USING AI IN THE WRITING PROCESS

The authors acknowledge the use of AI-assisted technologies during the writing process of this manuscript. All AI-generated content was thoroughly reviewed, verified, and edited by the authors to ensure accuracy, clarity, and alignment with the scientific objectives of the study. The final manuscript reflects the authors' intellectual contributions and interpretations.

LIST OF ABBREVIATIONS

BrP: Brugada phenocopy; BrS: Brugada syndrome; ECG: Electrocardiogram; EF: Ejection Fraction; TVG: Tricuspid Valve Gradient; CTR: Cardiothoracic Ratio; HsTroponin: High-Sensitivity Troponin; VF: Ventricular Fibrillation; I_{to}: Transient Outward Potassium current; RVOT: Right Ventricular Outflow Tract

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