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Case Report

Fever-Induced Brugada-Pattern Electrocardiogram

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ABSTRACT

Background: The Brugada syndrome is a type of cardiac arrhythmia frequently overlooked because of the dynamic character of the condition. Because it tends to progress into ventricular arrhythmias, it is a disorder that, if left untreated, carries the risk of being deadly. Not only is it essential for the practicing clinician to understand the situations that can disclose the concealed Brugada syndrome, but it is also essential for patients to understand these circumstances so that they can be educated to seek medical assistance quickly. This study aimed to describe the diagnosis and management of fever-induced Brugada pattern electrocardiogram

Case presentation: Male in his 42-year-old with a history of intermittent fever for four days before hospital admission. High-degree fever was only relieved by taking antipyretics and was accompanied by nausea and muscle and joint pain. Upon arrival at the emergency department, he denied any complaints of chest pain or discomfort, shortness of breath, orthopnea, PND, leg swelling, palpitation, or syncope. A chest radiograph showed normal cardiac and pulmo (Figure 1); a first electrocardiogram showed Sinus Rhythm, HR 112 bpm, regular, FA normal, HA normal, P wave normal, PR interval 160 msec, QRS 80 msec, QTc 326 msec, Coved ST elevation at lead V1 (1 mm), V2 (3 mm), T inversion at lead V1-V2, suggesting sinus tachycardia with type II Brugada pattern.

Conclusion: A Brugada pattern can be exposed to several stimuli, but fever is particularly potent. To assist urgent or emergency follow-up in cardiology, Emergency physicians must be informed of specific ECG findings based on the patient's clinical risk factors. The emergency doctor must be able to tell the difference between this pattern and a typical variation of RBBB, as a delayed diagnosis can have dire consequences.

1. Introduction

A hereditary condition known as the Brugada syndrome can result in sudden cardiac death (SCD) and life-threatening ventricular tachyarrhythmias. It is a diverse genetic disease. It is a form of cardiac illness passed down in an autosomal dominant manner and is caused by defective ion channels. The electrocardiogram pattern of those with this disease is typical, and they are more likely to experience potentially fatal ventricular arrhythmias. Alterations in the electrocardiogram can be transitory and are occasionally revealed by acquired diseases such as fever and electrolyte imbalances, making the diagnosis more difficult. Nevertheless, Brugada-like electrocardiogram abnormalities are still possible to induce even without congenital malfunction of ion channels. This is something that needs to be kept in mind. They conducted an in-depth analysis of the reports, determined an etiologic classification, and posed hypotheses regarding the probable causes, paving the way for a scientific investigation of the phenomenon.[1] To diagnose, observing ST-segment elevation greater than 1 mm in precordial leads (specifically V1-V3) located in the 4th, 3rd, or 2nd intercostal spaces is necessary. Three distinct types of Brugada-type electrocardiogram (ECG) patterns have been described According to the guidelines established in 2013 and 2015, a type 1 pattern alone is adequate to diagnose Brugada Syndrome (BrS). However, for type 2 and type 3, a conversion to a type 1 pattern is required by administering class I antiarrhythmic drugs. In addition to this conversion, one of the following criteria must also be met: Arrhythmia-related syncope, ventricular fibrillation that has been diagnosed, nocturnal agonal breathing, and a history of sudden cardiac death in people under the age of 45 without any apparent causes, or the presence of coved-type electrocardiogram (ECG) changes in family members who have passed away. [2] However, the ECG patterns are only temporary. They can be brought on by several factors, including but not limited to fever, medications, electrolyte disorders, or Right ventricular outflow tract mechanical compression. It is unknown at this time what percentage of people with induced BTEP develop malignant arrhythmias.[3]

2. Case Report

An Asian man in his 42s with a history of intermittent fever for four days before hospital admission. A high-degree fever could only be relieved by taking antipyretics and was accompanied by nausea and

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muscle and joint pain. The patient did not complain of bleeding symptoms such as petechiae, nosebleeds, gum bleeding, or black tarry stool. Because of the fever, the patient went to public health and was given an antipyretic. Because the symptom persisted, on Saturday, June 17, 2023, the patient returned to public health, and a rapid dengue test (IgG and IgM anti-dengue) was performed that showed a positive result. The patient was advised to go to the hospital if the fever was not relieved. Because the fever persisted, the patient went to an ER in a private hospital. The patient was in hemodynamic condition with blood pressure 110/70 mmHg, Heart rate 100 bpm, RR 24 tpm, Temperature 38.8 C, and SaO2 99% on NC 4 lpm. The ECG showed ST segment elevation at leads V1 and V2, and the patient was assessed for STEMI. He was given Aspirin 320 mg, Clopidogrel 300 mg, Atorvastatin 40 mg, Paracetamol 1 gram, Omeprazole 40mg, and Ondansetron 4mg. The patient was then referred to our hospital for further management.

Upon arrival at the emergency department (ED), he denied any complaints of chest pain or discomfort, shortness of breath, orthopnea, PND, leg swelling, palpitation, or syncope. On examination, His heart rate was 94 bpm regular, his respiratory rate of 24 tpm, his blood pressure of 119/77 mmHg with MAP (83 mmHg), a temperature of 37.0°C, and an oxygen saturation level of 99% with room air. Physical examination did not show rhonchi and murmur in this patient. Laboratory tests revealed a thrombocyte count 122.000, increased transaminase OT/PT 94/119, and a positive IGM/IGG dangue.

A chest radiograph showed normal cardiac and pulmo (Figure 1). A first electrocardiogram showed Sinus Rhythm, HR 112 bpm, regular, FA normal, HA normal, P wave normal, PR interval 160 msec, QRS 80 msec, QTc 326 msec, Coved ST elevation at lead V1 (1 mm), V2 (3 mm), T inversion at lead V1-V2, suggesting sinus tachycardia with type II Brugada pattern (Figure 2),. An echocardiogram showed normal RA, RV, LA, and LV dimensions, Systoli. LV Function was normal EF biplane 64%, mitral regurgitation trivial, global normokinetic (Figure 3).

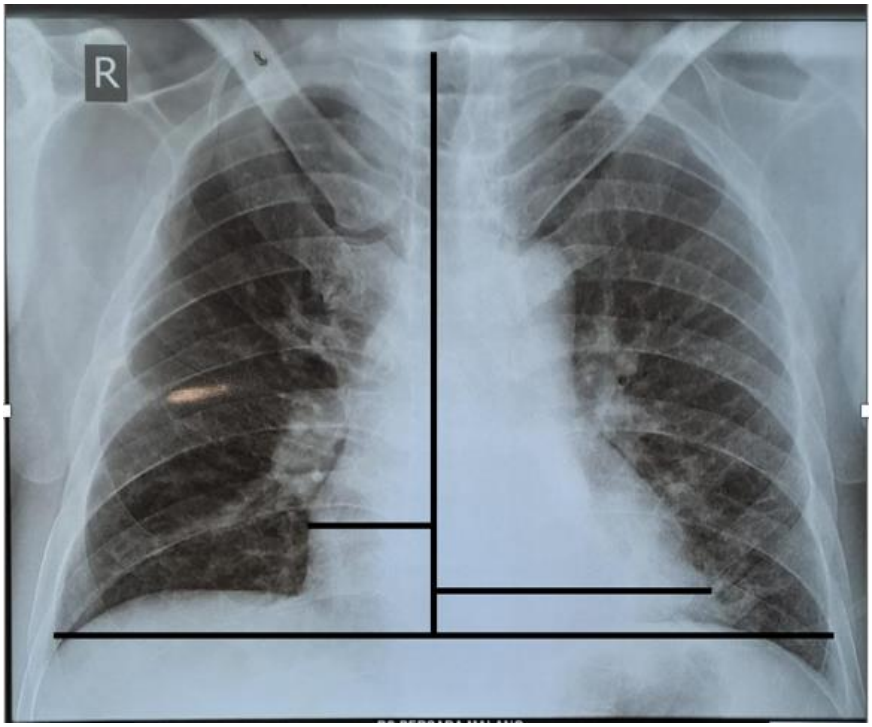

Figure 1. The chest radiograph showed normal results.

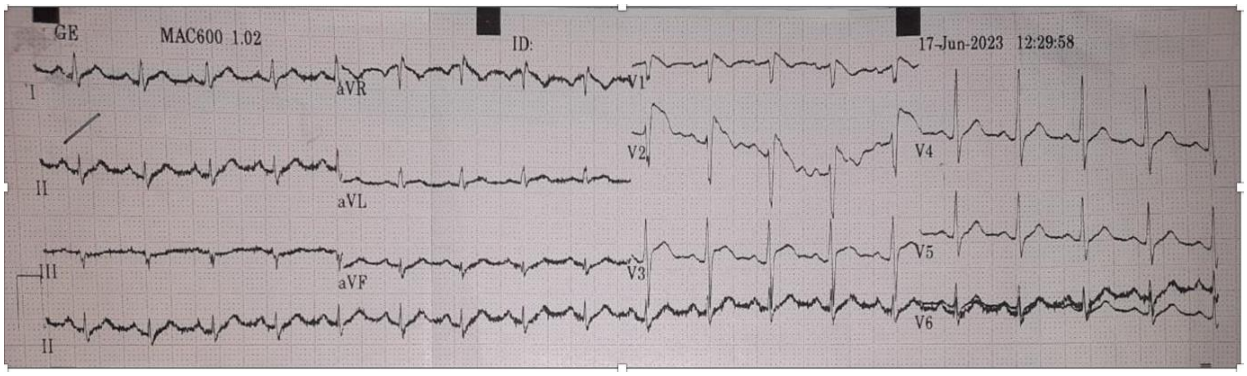

Figure 2. Initial electrocardiogram on First medical contact Sinus Rhythm, HR 112 bpm, regular, FA normal, HA normal, P wave normal, PR interval 160 msec, QRS 80 msec, QTc 326 msec, Coved ST elevation at lead V1 (1 mm), V2 (3 mm), T inversion at lead V1-V2.

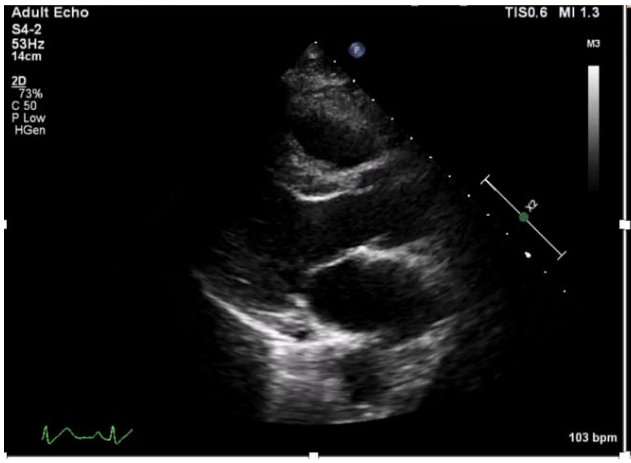

Figure 3. Parasternal long axis view on TTE revealing did not found structural heart disease.

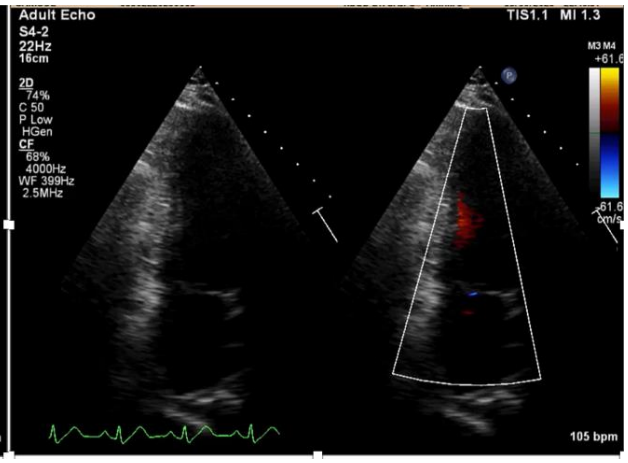

Figure 4. 4ch view on TTE revealing a mitra regurgitation trivial.

In the ER patient with a temperature of 37.0, we evaluated the electrocardiogram and Sinus rhythm of 95 bpm, FA N, HA N; PR interval of 120 ms; QRS complex of 80 ms, QTc of 403 ms, and inverted T at leads V1 and V2 (figure 5). we obtained ST changes at lead V1 and V2 ST elevation back to baseline. The patient was suspicious of STEMI at the private hospital, our evaluation of the cardiac enzyme marker was normal troponin I 0.4, and we did not find signs of acute coronary syndrome in this patient. We stopped giving double antiplatelets, and the patient continued observation at the cardiovascular care unit. At the CVCU, the patient complained of fever again, with a temperature of 38.9 Celcius. We evaluated ecg Sinus Rhythm, HR 112 bpm, regular, FA normal, HA normal, P wave normal, PR interval 160 msec, QRS 80 msec, QTc 326 msec, Coved ST elevation at leads V1 (1 mm), V2 (3 mm), and T inversion at leads V1-V2 Conclusion: Sinus tachycardia with type II Brugada pattern. At CVCU, consultation with a cardiac electrophysiologist was suggested to avoid fever and drugs that cause QT prolongation.

2. Discussion

Brugada-type ECG patterns (BTEP) The genetic disorder under consideration is associated with a distinct electrocardiogram (ECG) observation, namely the presence of either persistent or temporary elevation of the ST segment in the right precordial leads (V1, V2), with or without concurrent right bundle branch block. Fever is recognized as a contributing factor in the Brugada-type ECG pattern (BTEP) manifestation. Numerous case reports have extensively documented the association between fever and Brugada syndrome (BS). These reports have observed that individuals with elevated body temperature often exhibit Brugada-type electrocardiographic patterns (BTEP) and are at an increased risk of experiencing sudden cardiac death or ventricular arrhythmias.[4] These changes in the ECG are dynamic, and they are frequently hidden. However, they may become visible if specific triggers are present, such as fever, intoxication (which can be caused by substances such as Alcohol, cocaine, or cannabis), vagal stimulation, electrolyte imbalance, anesthetics (propofol, bupivacaine), psychiatric drugs (amitriptyline, lithium), and sodium channel blockers are some of the substances that might cause this.[6] The fever-induced form of the Brugada syndrome is becoming an increasingly well-known condition. According to Amin et al.,[6], a type I Brugada ECG pattern was 20 times more prevalent in patients with fevers than those without. Patients who have symptomatic Brugada syndrome account for approximately 18% of all cases of sudden cardiac arrest were shown to be precipitated by fever. This translated to a prevalence of 2%, compared to an estimated 0.05% prevalence of asymptomatic Brugada syndrome in the overall population. They also discovered that the patients often fell into the age range of 30 to 60 years old and that 87% of the patients were of the male gender, which was a finding that was also observed in earlier studies.[5] Fever-induced Brugada syndrome has been linked to factors such as lower age, male gender, and the effect of temperature on mutant sodium channels, which reduces sodium current and consequently delays conduction. Consequently, as the degree of hyperthermia increases, also contributes to the problem is an imbalance in the ionic current also contributes to the problem, which causes the action potential to have a deeper notch, further generating ventricular arrhythmias. Fever-induced Brugada syndrome fever increases the incidence of cardiac arrest, with the Type 1 Brugada pattern having a higher prevalence than the Type 2 Brugada pattern.[6]

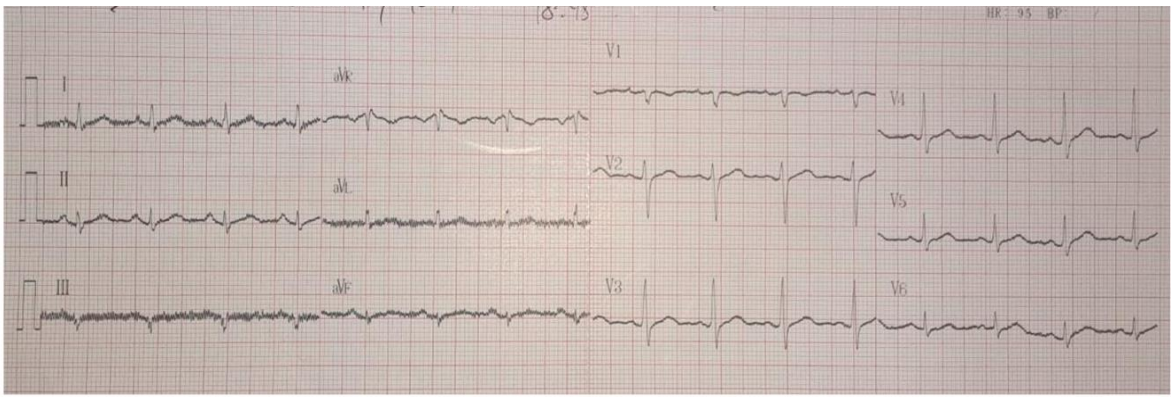

Figure 5. Second electrocardiogram with temp 37 celcius was Sinus rhythm 95 bpm, FA N, HA N; PR interval 120 ms; QRS complex 80 ms; QTc 403 ms; inverted T at lead V1 and V2 .

In the case we looked at, the patient was a 42-year-old man with a fever. In cases of Brugada syndrome, several ECG patterns have been described. The patient exhibited characteristics of type 1. A covered pattern is shown by an ST-segment increase of 2 mm (0.2 mV) slowly decreasing. An asymmetric negative T wave in the right precordial leads follows after that. No clear rI wave can be seen. ECG demonstrates that type 2 (saddle-back pattern) begins with a positive wave (rI that is 2 mm (0.2 mV) from the isoelectric line. A minimum ST rise of 0.5 mm (0.05 mV) is then followed by a positive/flat T wave in V2 and a variable T wave in V1. Brugada syndrome is mainly diagnosed based on how it looks. In addition to the unique ECG pattern, one of the following clinical conditions must be met: (a) a previous diagnosis of ventricular tachycardia (VT) or ventricular fibrillation (VF), (b) a history of sudden cardiac passing in the family, (c) a history of Brugada syndrome within the family, (d) breathing in agony when sleeping, or (e) the ability to cause VT/VF during an electrophysiological study.[7]

According to the 2013 HRS/EHRA/APHRS expert consensus statement, BrS can be diagnosed by finding a type 1 ECG that happens independently or because of a drug. In the report from the 2016 HRS/APHRS/EHRA/SOLAECE J Wave Syndrome Consensus Conference, a point-scoring system for diagnosing BrS was suggested. This system would include clinical characteristics. The Shanghai Score System was notable because it said The significance of a spontaneous type 1 electrocardiogram (ECG) pattern outweighs other factors in diagnosing Brugada Syndrome (BrS). One thing that made this scoring system stand out was that it used real-world methods, parameters from an electrocardiogram, and diagnostic information gleaned from a patient interview. Based on the total score, likely and/or certain BrS, possible BrS, and a nondiagnostic result were all ruled out.3.5 points: likely or Brugada syndrome (BrS) is specific; two to three points: maybe Brugada syndrome: two points for not being diagnostic. In this case, we used Shanghai Score System to determine that the score was 3. which means possible Brugada syndrome.[8]

Based on clinical findings, it is recommended to do a pharmacologic challenge using a sodium channel blocker when there is a suspicion of BrS. Still, there is no spontaneous type 1 ST-segment elevation present. On the website www.brugadadrugs.org, you can also find a list of the agents used for this purpose. The test is only regarded positive if it results in an ECG pattern of type 1, and it ought to be terminated if frequent ventricular extrasystoles or other arrhythmias are observed, as well as an increase in the width of the QRS complex by more than 413% compared to its baseline value. A drug challenge is unnecessary in asymptomatic patients with type 1 ECG under baseline settings. This is due to the lack of extra diagnostic value that the challenge would provide. Additionally, it is not advised to use these provocative drug tests when it has been demonstrated that fever can cause type I ECG changes.[9]

So far, ICDs and drugs have been the only ways to help people in the BrS. Recommendations for the treatment of BrS, based on the 2013 HRS/EHRA/APHRS The 2015 ESC guidelines for the diagnosis and management of patients with hereditary primary arrhythmia syndromes, for the treatment of patients with ventricular arrhythmias, for the prevention of sudden cardiac death, and the treatment of patients with ventricular arrhythmias. These recommendations are based on the existing research and the Task Force members' clinical expertise. [11] As with all of these suggestions, they will need to be tested repeatedly in new studies. In BrS, the best way to stop arrhythmias is through education and changes in living. Patients should know the triggers and moderators that can cause malignant rhythms. ECG signs of BrS have recently been linked to a strong S-wave in Lead I. In this patient, Fever should be treated aggressively with antipyretics, and people should avoid things that shouldn't be used. (see www.brugadadrugs.org). We instructed that If the patient is experiencing a high temperature, it is recommended that they get an electrocardiogram. Family members may be referred to receive training in cardiopulmonary resuscitation, and they may be recommended to consider obtaining an automated external defibrillator for use in the house.[10] There are not often malignant ventricular arrhythmias in asymptomatic individuals with BrS, and are typically unrelated to physical activity. Therefore, Participation in athletics may not be prohibited despite the existence of these patterns. It is interesting to observe. Nevertheless, it has been shown that the Brugada pattern is improved directly after physical activity, most likely due to increased vagal tone. This is something to keep in mind.[11]

3. Conclusion

Up to 10% of annual mortality has been attributed to undiagnosed Brugada syndrome. Several stimuli can expose a Brugada pattern, but fever is particularly potent. To assist urgent or emergency follow-up in cardiology, Emergency physicians must be informed of specific ECG findings based on the patient's clinical risk factors. The emergency doctor must be able to tell the difference between this pattern and a typical variation of RBBB, as the delayed diagnosis can have dire consequences.

4. Declaration

4.1 Ethics Approval and Consent to participate
This study was approved by local Institutional Review Board, and all participants have provided written informed consent prior to involvement in the study.

4.2. Consent for publication
Not applicable.

4.3 Availibility of data and materials
Data used in our study were presented in the main text.

4.4 Competing interests
Not applicable.

4.5 Funding Source
Not applicable.

4.6 Authors contributions
Idea/concept: SA. Design: TYN. Control/supervision: IP, HM, AR. Data collection/processing: IP Analysis/interpretation: TYN, IP. Literature review: IP. Writing the article: TYN. Critical review: IP, HM, AR. All authors have critically reviewed and approved the final draft and are possible for the content and similarity index of the manuscript.

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