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Brugada syndrome with type 2 pattern. Case report

Síndrome de Brugada con patrón tipo 2. Reporte de un caso

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ABSTRACT

Brugada syndrome is an inherited cardiac channelopathy that leads to malignant ventricular arrhythmias and sudden cardiac death, despite the absence of signs of structural heart disease. The syndrome's characteristic electrocardiographic pattern is rare and may be the only clinical manifestation, which complicates identification. This syndrome accounts for between 4 and 12% of cases of sudden cardiac death and up to 20% in patients with structurally normal hearts. It predominantly affects men, with a frequency of eight to 10 times compared to women, and an estimated prevalence of one to five cases per 10,000 individuals. The *SCN5A* gene is the most associated mutation, identified in 11 to 28% of cases. Syncope is the most relevant clinical sign, although it is not always present. Currently, diagnosis and risk stratification remain a clinical challenge due to the lack of consensus on reliable prognostic markers. Studies such as FINGER and PRELUDE have questioned the usefulness of inducibility in electrophysiological studies as a prognostic criterion, limiting its applicability in clinical practice. This report describes the case of a young asymptomatic male patient who has no relevant personal or family medical history and presents an electrocardiographic pattern consistent with Brugada syndrome. This finding poses a significant clinical challenge regarding risk assessment, highlighting the need for more precise criteria for the management of these patients.

RESUMEN

El síndrome de Brugada es una canalopatía cardiaca hereditaria sin signos de enfermedad estructural del corazón que conduce a arritmias ventriculares malignas y muerte súbita cardiaca. Su patrón electrocardiográfico característico, aunque raro, puede ser la única manifestación clínica, lo que dificulta la identificación. Este síndrome representa entre 4 y 12% de los casos de muerte cardiaca súbita y hasta 20% en pacientes con corazones estructuralmente normales. Afecta predominantemente a hombres, con una frecuencia de ocho a 10 veces en comparación con las mujeres, y una prevalencia estimada de uno a cinco casos por cada 10,000 individuos. El gen SCN5A es la mutación más asociada, identificada en 11 a 28% de los casos. El síncope es el signo clínico más relevante, aunque no siempre está presente. Actualmente, el diagnóstico y estratificación del riesgo siguen siendo un desafío clínico debido a la falta de consenso sobre marcadores pronósticos confiables. Estudios como FINGER y PRELUDE han cuestionado la utilidad de inducibilidad en los estudios electrofisiológicos como criterio pronóstico, limitando su aplicabilidad en la práctica clínica. Este informe describe el caso de un hombre joven y asintomático que no tiene antecedentes médicos personales o familiares relevantes y presenta un patrón electrocardiográfico consistente con el síndrome de Brugada. Este hallazgo plantea un desafío clínico significativo en cuanto a la evaluación del riesgo, subrayando la necesidad de criterios más precisos para el manejo de estos pacientes.

Abbreviations:
BS = Brugada Syndrome
ECG = Electrocardiogram
SD = Sudden Death
TCAs = Tricyclic Antidepressants

INTRODUCTION

The Brugada-type electrocardiographic pattern is a rare manifestation that indicates a predisposition to ventricular arrhythmias. It is a cardiac channelopathy without structural heart disease. Its clinical

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relevance lies in its ability to cause sudden death in young patients with healthy hearts. It is known to be inherited in an autosomal dominant manner, with the *SCNA5* gene being the most common.[1]

Brugada Syndrome (BS) accounts for between 4 and 12% of sudden cardiac deaths and up to 20% in structurally healthy hearts.[2] It is eight to 10 times more common in men, with an estimated global prevalence of one to five cases per 10,000 people. Genetic variants are identified in 11 to 28% of cases, with the *SCNA5* gene being the most common.[3,4] Syncope is the most prominent feature in the clinical history. Family history is extremely important, particularly the presence of type 1 BS in relatives or a family history of sudden death in individuals ≤ 45 years of age.[5]

Studies such as FINGER have been reported, whose main contribution was to establish that medical history is the most powerful predictor. It was determined that the rate of arrhythmic events is significantly higher in patients with previous syncope or sudden cardiac arrest, while patients who do not report symptoms have a notably low risk (0.5% per year). In addition, it was the first to point out that variables such as gender, family history or inducibility in the electrophysiological study did not have solid positive predictive value. In addition, the PRELUDE study, which focused specifically on the usefulness of programmed electrical stimulation, recorded decisive results by demonstrating that the inducibility of arrhythmias in the laboratory does not reliably identify patients at risk of spontaneous arrhythmic events, clearly corroborating the low predictive value observed in the FINGER study. This study also proposed new and more promising risk markers, such as a short ventricular refractory period (< 200 ms) and QRS fragmentation potential on baseline electrocardiogram (ECG).[6,7]

As risk factors for BS, given that it is an autosomal dominant hereditary disease, the most common mutation is in the *SCN5A* gene, which encodes the α subunit of the cardiac sodium channel INa. Mutations reduce sodium flow, especially in the epicardium of the right ventricle, causing voltage gradients that manifest as ST-segment elevation. Recent studies have highlighted the importance of non-modifiable risk factors that are intrinsic to the patient, specifically the type of *SCN5A* mutation, regardless of clinical symptoms. This evidence underscores that the genetic substrate may be a predictor of malignant arrhythmic events. The most consistent non-modifiable risk factor is male sex. Arrhythmic events and Sudden Death (SD) also manifest predominantly in males, suggesting that male sex hormones (androgens) may have a modulatory effect on sodium channel function, exacerbating the phenotype. In the geographical context, although cases have been reported worldwide, the highest prevalence rates have been identified in Southeast Asian populations, where the disease is endemic and known by local names, e.g., Lai Tai in Thailand, which means Sudden Unexplained Nocturnal Death Syndrome (SUNDS).[8,9]

The diagnosis of BS is based on the identification of the distinctive electrocardiographic pattern and its correlation with clinical risk factors. The Brugada consensus established a precise classification of ECG patterns. Only type one has definitive diagnostic value. The patterns observed on the ECG are type 1 (dome or shark fin): concave or straight descending ST segment elevation of ≥ 2 mm (≥ 0.2 mV) in V1 and/or V2, followed by a negative T-wave. This pattern is the only diagnostic one. Type 2 (saddleback): J-point elevation ≥ 2 mm (greater than the PR segment), followed by a descending ST-segment that remains ≥ 1 mm above the isoelectric line and ends with a positive or biphasic T-wave. This pattern is suspicious. Type 3: type 1 or type 2 morphology, but with ST-segment elevation < 1 mm. Nonspecific pattern.

Type two pattern, known as saddleback morphology, is of clinical interest. It is characterized by a J point elevation ≥ 2 mm in V1 and/or V2, and an ST-segment that descends toward the isoelectric line but remains elevated ≤ 1 mm, creating the initial convex appearance followed by the characteristic saddleback concavity. This finding, although suggestive, has a low positive predictive value on its own, as it can be confused with normal repolarization variants, especially in athletes. Likewise,

as associated clinical criteria, it has been documented to have survived an episode of cardiac arrest secondary to ventricular fibrillation or polymorphic ventricular tachycardia, a history of unexplained syncope understood as a self-limiting arrhythmic event, nocturnal agonal respiration, which are episodes of abnormally noisy or labored breathing during sleep, and a family history of type 1 Brugada pattern in relatives or a family history of sudden death ≤ 45 years of age.[10,11]

This article presents the case of a symptomatic patient with no family or personal history who presents the characteristic electrocardiographic pattern of the syndrome. This finding poses a challenge in risk stratification and clinical decision-making.

CASE PRESENTATION

This is a 26-year-old man, originally from Chiapas, who currently resides in Villahermosa, Tabasco, Mexico. Occupation: undergraduate student. He sought medical services at the Juárez Autonomous University of Tabasco (UJAT) due to frequent spontaneous palpitations at rest, occurring three to five times per month, occasionally associated with mild dizziness but without syncope. In the physical examination, the vital signs were: blood pressure 111/67; heart rate 87 bpm; respiratory rate 18 bpm; temperature 36.6 ºC; oxygen saturation ($SatO_2$) 98%; weight 66.5 kg and height 1.69 m. Relevant personal and family history: denies any cardiovascular disease. No family history of sudden death or hereditary heart disease. Current illness: palpitations began approximately three months ago, with no apparent triggering factors. The episodes are short-lived and not related to physical exertion. The patient also reports generalized fatigue and occasional episodes of dizziness without loss of mental clarity. He denies chest pain, dyspnea, syncope, diaphoresis, fever in the last six months, or neurological symptoms. He has not received previous medical treatment. General condition: patient conscious, alert, oriented in all three neurological spheres. Cardiovascular examination: rhythmic heart sounds, no murmurs or rubs, peripheral pulses present and symmetrical.

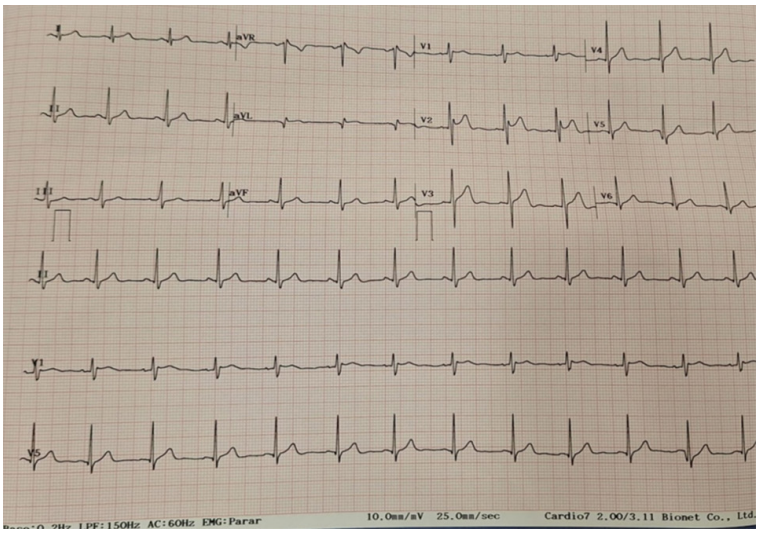

Figure 1: Standard 12-lead ECG calibrated at 25 mm/sec and 10 mm/mV; 3 channels + 3 rhythm channels. A standard 12-lead ECG is observed at rest in sinus rhythm, with a normal cardiac axis at 60º.

Studies

Baseline ECG: ST-segment elevation with saddleback morphology in leads V1-V2, with positive T-wave, suggestive of type 2 Brugada pattern.

The ECG documented ST-segment elevation in leads V1-V2 with a 1 mm notch and a positive T-wave consistent with the «saddleback» pattern characteristic of type 2 BS. The remainder of the ECG showed no evidence of atrioventricular block, bundle branch block, or atrial or ventricular hypertrophy *(Figure 1)*.

24-hour Holter monitoring

24-hour Holter monitoring, which recorded: a) a minimum heart rate of 46 bpm; b) a maximum heart rate of 137 bpm; c) a maximum RR interval of 1.422 s; and d) isolated premature atrial contraction.

The Holter review documented the following: basal sinus rhythm with an average of 74 bpm, a minimum of 46 bpm, and a maximum of 137 bpm; no atrial fibrillation was documented. Negative study for complex arrhythmias. No pauses longer than 2 seconds *(Figure 2)*.

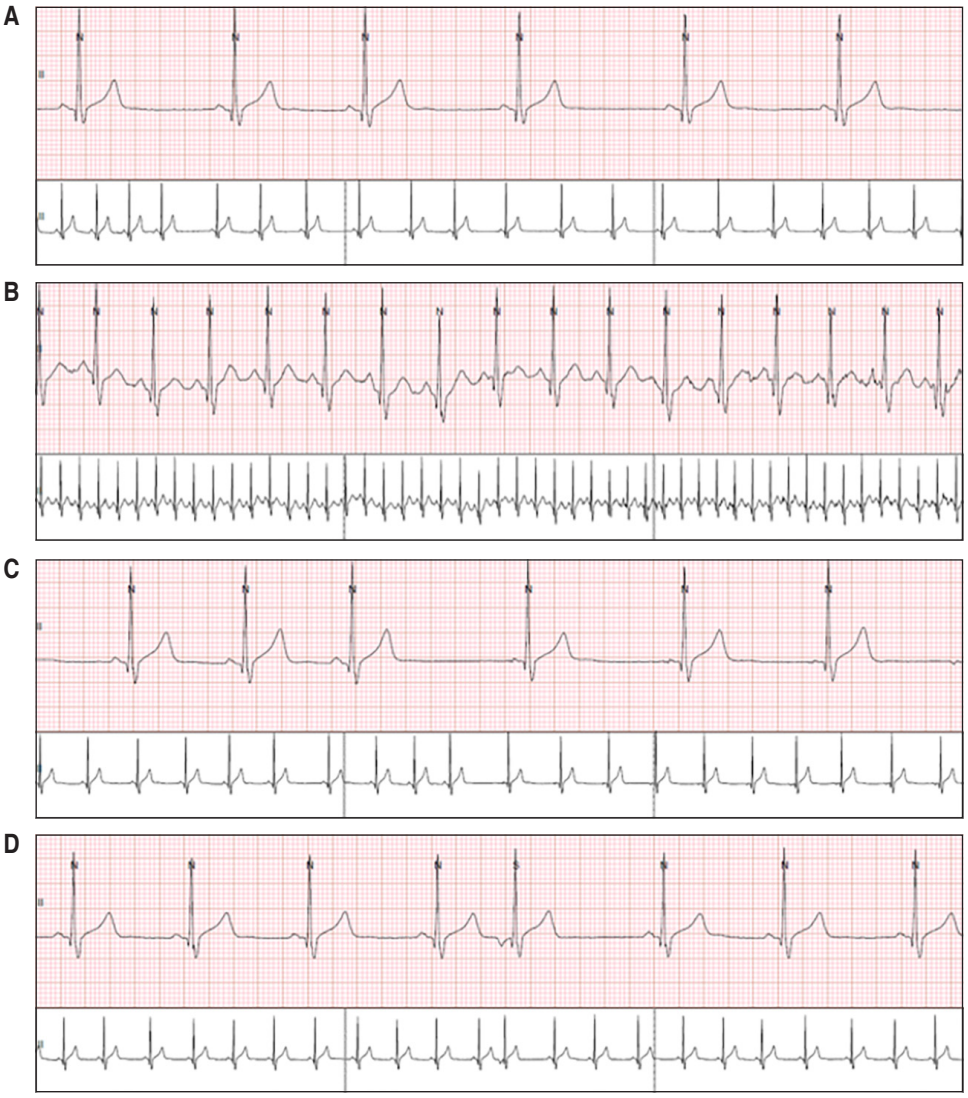

Figure 2:

24-hour HOLTER, which recorded: **A)** the lowest HR of 46 beats per minute (bpm), **B)** the highest Heart Rate (HR) of 137 bpm, **C)** the longest RR interval of 1.422 sec, and **D)** the isolated premature atrial contraction.

Laboratory tests

Blood cytometry: leukocytes 7,400/µL, rest of white blood cell count normal. Red blood cell count: Hb: 15.4 g/dL, HCT: 46.1%, MCV: 99.8 fL, MCH: 33.3 pg, RBC: 4.9 × 10^6/mm^3, Platelets: 316 × 10^3/mm^3, MPV: 6.8% (low, range: 7.4-10.4). Serum electrolytes: sodium: 147.5, Potassium: 4.40, Serum chloride: 101.9 (unit: mmol/L). Lipid profile: cholesterol: 189.9, LDL: 99, HDL: 56.5, Triglycerides: 171 (high) (unit: mg/dL). Liver profile: total serum proteins: 7.23 g/dL, Globulin: 2.455 serum albumin: 4.77 g/dL, A/G ratio: 1.943, total bilirubin: 0.477 mg/dL, indirect B: 0.399 mg/dL, direct B: 0.078 mg/dL, SGOT: 34.7 U/L, SGPT: 51.7 U/L (elevated, 0-40.0), GGT: 21.4 U/L, ALP: 208.5 U/L, LDH: 308.54 U/L. Blood chemistry: glucose: 97.6, UREA: 22.1, BUN: 10.3, creatinine: 0.91, uric acid: 3.3 (low, 3.6-7.7) (unit: mg/dL). Urinalysis: performed as part of the initial approach. No relevant findings were found.

Diagnostic impression

Probable *de novo* type 2 Brugada syndrome (electrocardiographic pattern) under investigation. Frequent palpitations and dizziness without evidence of hemodynamic compromise or loss of consciousness.

DISCUSSION

A convex ST-segment elevation of more than 2 mm in the precordial leads (V1-V3) followed by a positive or biphasic T-wave that creates a «saddleback» appearance on the electrocardiogram, suggests a type 2 Brugada pattern, which has a lower prevalence than type 1. It has been reported that the effect of mutated sodium channels can lead to the expression of the pattern in conditions of increased risk, such as fever, electrolyte imbalances, vagal stimulation, psychosis, medications that act on sodium ion channels, and drug use. It is understood that BS can be dynamic, presenting with a type 2 pattern accompanied by significant symptoms and without any physiological condition, such as fever or potassium disturbances (hyperkalemia), that may be sufficient to manifest the pattern and progress to the more dangerous type 1 pattern. Therefore, it is important to closely monitor patients to avoid a more unfavorable prognosis. The role of electrophysiological studies remains controversial, as evidence in reports indicates that they have high sensitivity for identifying patients at higher risk of arrhythmic events, while their specificity remains low.[12]

Although this pattern is highly suggestive of BS, it is essential to establish a thorough differential diagnosis, as numerous entities, including primary pathological conditions and transient or drug-induced phenomena (Brugada phenocopies), can mimic this morphology.[13] Differentiation is crucial, given that the prognosis and therapeutic management vary dramatically. The Brugada pattern is classified as a channelopathy without obvious structural heart disease. However, other cardiomyopathies and channelopathies may present ST-segment elevation in V1-V2: Early Repolarization Syndrome (ERS): ERS, especially the variant affecting the right precordial leads, may manifest with J-point and ST-segment elevation, which can be confused with the type 2 pattern. The differential diagnosis is based on the specific morphology and the absence of other clinical criteria for Brugada syndrome. Wolff-Parkinson-White (WPW) syndrome: although it is a ventricular pre-excitation, the presence of delta waves can mask or influence the morphology of the ST-segment, creating false elevations in the right precordial leads. Sick sinus syndrome: although it is primarily a conduction disorder, its presence or the influence of related drugs can complicate the ECG and generate patterns like saddleback. And Arrhythmogenic Right Ventricular Dysplasia (ARVD): this condition is characterized by the replacement of myocardial tissue with adipose and fibrous tissue, predominantly in the right ventricle. It can cause repolarization abnormalities that resemble the Brugada pattern.[14] However, ARVD is distinguished by epsilon waves and/or right ventricular dilation/dysfunction.

Brugada phenocopies and acquired (reversible) causes. Brugada phenocopies are ECG patterns that meet the Brugada criteria (type 1, 2, or 3) but are secondary to reversible or induced conditions, disappearing once the underlying cause is resolved. It is estimated that these phenocopies constitute a significant percentage of initially diagnosed patients. Electrolyte disturbances: Elevated potassium levels can affect repolarization and depolarization, creating patterns that resemble ST elevation. Similarly, elevated calcium levels can also alter action potentials, contributing to ECG abnormalities.[12] Fever/ Febrile states: increased body temperature can unmask a type 1 or type 2 pattern, especially in silent BS carriers. Fever amplifies sodium channel dysfunction.

In the context of a symptomatic patient with a type 2 pattern, active management of reversible factors such as fever and immediate discontinuation of inducing drugs (sodium blockers, tricyclic antidepressants (TCAs), cocaine) is strongly recommended before performing the pharmacological provocation test for a definitive diagnosis of BS. Reversibility of the ECG pattern after elimination of the acquired cause would confirm a phenocopy and not underlying genetic BS.[15,16]

CONCLUSIONS

This clinical case provides valuable lessons in both clinical and educational settings on how to approach Brugada syndrome.

From a clinical perspective, it highlights the importance of maintaining a high index of diagnostic suspicion in patients with seemingly nonspecific symptoms such as dizziness and palpitations, especially when a type 2 Brugada electrocardiographic pattern is identified. The absence of physiological factors that explain these findings reinforces the need for a thorough evaluation using 24-hour Holter monitoring, intentional search for malignant ventricular arrhythmias, and complete biochemical analysis to rule out secondary causes or precipitating factors.

In the educational context, this case exemplifies the complexity of differential diagnosis in cardiac channelopathies and highlights that type II electrocardiographic patterns, although less specific than type I, should not be underestimated. It provides an opportunity to reinforce knowledge about risk stratification in Brugada syndrome, emphasizing that the presence of associated symptoms significantly increases the risk of fatal arrhythmic events.

The systematization of the diagnostic approach presented serves as an educational model for the methodical analysis of similar cases, promoting clinical practice based on the integration of electrocardiographic findings, clinical manifestations, and complementary studies. Finally, this case reinforces the need for structured follow-up protocols and early consideration of preventive measures, including the possible indication of an implantable cardioverter defibrillator in symptomatic patients with a strong suspicion of Brugada syndrome.

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