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Case Report

Pericardial Effusion and Brugada Type 1 Electrocardiogram Pattern. An Another Case of Brugada Phenocopy

Aldo Coppolino* and Letizia Valeri

Department of Cardiology, S. Annunziata Hospital, Savigliano (CN), Italy

***Corresponding Author:** Aldo Coppolino, Department of Cardiology, S. Annunziata Hospital, Savigliano (CN), Italy.

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Abstract

Brugada type 1 ECG pattern is characterized by high take-off or highest point of the QRS-ST at least 2 mm over baseline and by coved type ST elevation followed by negative symmetrical T-wave in the right precordial leads (V1-V2).

The Brugada Syndrome associated with an increased hazard of sudden death can appear in patients with typical and diagnostic ECG changes and a history of symptoms (syncope, cardiac arrest).

Although several conditions can mimic Brugada ECG patterns, such as early repolarization, myocardial ischemia, electrolyte disorders or poor ECG filter, these clinical entities have been called Brugada Phenocopyies.

We report a case of Brugada Phenocopy in a 65-year-old man admitted to the emergency room because of syncope. ECG showed spontaneous and typical Brugada type 1 ECG, and echocardiography showed a massive pericardial effusion compressing the right ventricle. Brugada type 1 ECG disappeared after the pericardial drain, and we have not observed it anymore. The pericardial tamponade was the mechanism of syncope and the Brugada type 1 ECG pattern. Both had mimed Brugada Syndrome.

Keywords: Brugada Syndrome; Brugada Phenocopy; Electrocardiogram; Pericardial Effusion; Syncope

Abbreviations

ECG: Electrocardiogram; BrPs: Brugada Phenocopies; BrP: Brugada Phenocopy; BS: Brugada Syndrome; RVOT: Right Ventricular Outflow Tract

Introduction

In 1992 Brugada's brothers have described an inherited cardiac channelopathy called the Brugada Syndrome (BrS). It is associated with an increased hazard of sudden death, and principal elements are the history of symptoms (syncope, cardiac arrest) and diagnostic ECG type [1].

In 2012, Baranchuk proposed and classified the Brugada Phenocopy (BrP) as relative clinical entities that are etiologically distinct from true congenital BrS [2]. Typical ECG patterns are identical to BrS and BrP and define both. Despite BrS, BrP reveals various clinical, reversible and underlying conditions, and the symptoms could be different.

We report a case in which the patient developed a BrP having all characteristics of BrS.

Case Report

- D.S. was a 65-year-old man admitted to our emergency room because of syncope.
- It occurred in orthostatic position and without prodromes, causing head trauma.

- No family history of sudden death, no previous history of syncope and he no referred other cardiovascular risk factors.
- In the past, he experienced gastric cancer and subsequently underwent total gastrotomy and oesophageal-duodenal-ostomy.
- Afterwards, the patient developed a cancer relapse that caused stenosis of the duodenum and subsequent need for enteral nutrition.
- Upon arrival in the emergency room, he referred to dyspnoea, asthenia, contraction of diuresis, hypotension and no fever during previous days.
- He was not taking sodium channel blockers or tricyclic antidepressant drugs.
- The clinical examination showed tachypnea, pale and cold skin, signs of high jugular venous pressure, distant heart tones but no murmurs. The liver was enlarged and sore. The brachial blood pressure was 80/50 mmHg, peripheral pulse oximetry was 90%, body temperature was 36.8 °C. The chest X-ray showed pleural effusion at the right lung base and enlarged heart shadow.
- The main laboratory parameters were: Troponin T < 0.03 ng/dl, Creatinine 2.53 mg/dl; Hemoglobin: 10.3 g/dl, AST 461mg/dl, ALT 347 mg/dl, LDH 1036 mg/dl, Na: 131 mEq/L, K: 4.7 mEq/L, INR 1.62 pH: 7.42, pO2: 72 mmHg, pCO2: 34 mmHg, bicarbonates: 20 mg/dl, lactates: 1.9 mg/dl.
- The ECG, performed in the emergency room, showed a normal sinus rhythm, normal atrioventricular conduction spontaneous and a typical Brugada type 1 pattern in V2 lead (Figure 1).
- Clinically, there were two diagnostic hypotheses: Brugada Syndrome (a male patient with syncope and ECG with a typical coved pattern) and Cardiogenic Shock (symptoms and signs of multi-organ failure).
- Subsequently, we performed transthoracic echocardiography. The exam revealed normal left and right ventricular function, no significant valvular dysfunction but a massive pericardial effusion compressing the right ventricle (Figure 2).
- Promptly, we executed the pericardiocentesis to restore physiological pressure in the pericardial sack. We drained 1500 ml of bloody fluid, and the ECG recorded after the pericardial drain has shown that the Brugada type I pattern had disappeared (Figure 3). This pattern was not observed anymore in the following days.
- Unfortunately, the patient died a few days later because of advanced cancer.

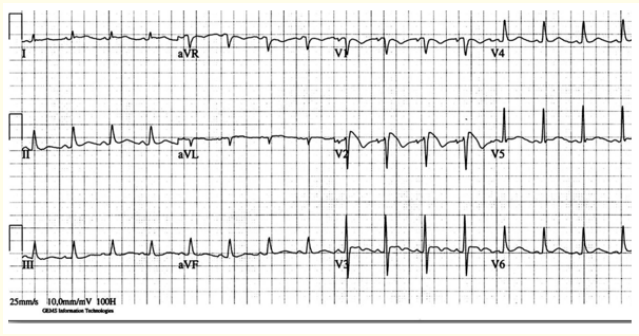

Figure 1: This is the ECG recorded on admission to the emergency room. It shows a typical coved type (type 1) of the Brugada pattern.

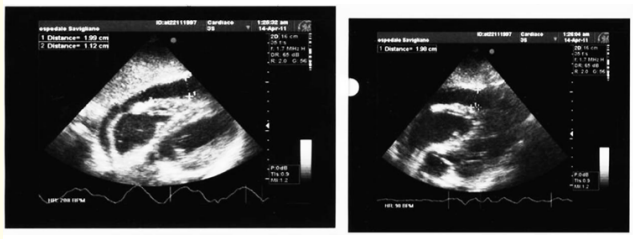

Figure 2: Echocardiogram. It shows large pericardial effusion.

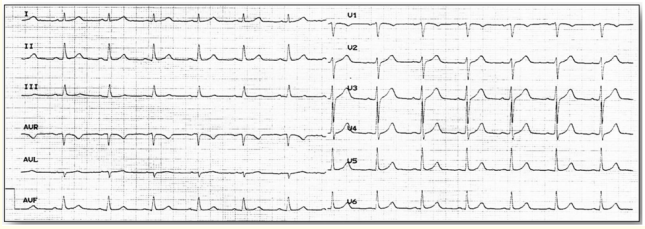

Figure 3: Ecg recorded at the end of the pericardial drain. The coved type pattern is disappeared.

Discussion

Brugada Phenocopy: diagnostic criteria and classification

Brugada Phenocopies (BrPs) are clinical entities that are etiologically distinct from true congenital Brugada Syndrome (BrS). BrPs show an ECG pattern as to either the type-1 or type-2 Brugada patterns. Still, they are elicited by various clinical circumstances in

association with an identifiable condition and, upon resolution, the ECG pattern normalizes.

BrPs is not due to the same sodium channelopathy as BrS, and the typical pattern ECG may be only transient while the underlying condition persists [2,3,5].

The term phenocopy serves as a reasonable and succinct description for all acquired Brugada-like ECG manifestations, already described in the Brugada Syndrome Consensus in 2005 [4].

Diagnostic criteria for BrPs are:

- Typical type 1 or type 2 Brugada ECG pattern.
- The presence of an underlying condition induces a Brugada ECG pattern.
- The resolution of the ECG pattern after eliminating the underlying condition.
- A low pretest probability for BrS as defined by lack of clinical symptoms, medical history and family history suggestive of BrS.
- Negative provocative testing with sodium channel blocker drugs.
- Negative genetic testing for SCN5A. This suggestion is not mandatory as it is possible to identify mutation only in 20%-30% of probands known to have BrS.

The first four criteria are mandatory [6,7].

According to the presenting ECG morphology, Anselm., *et al.* [8] had classified cases of BrPs in Type-1 and Type-2 BrPs and added three additional qualifiers to determine whether each case fulfils diagnostic criteria. Further qualifiers are classes A, B, and C.

Class A includes definitive BrPs with all mandatory diagnostic criteria, including a negative test with a sodium channel blocking agent.

Class B include cases so far suspected to be BrP, but the mandatory diagnostic criteria are incomplete. In these cases, the provocative test with a sodium channel blocking test agent is often impossible.

Class C includes cases highly suspected BrP with mandatory incomplete diagnostic. In particular, a provocative test with a sodium channel blocking test agent is not indicated or not justified.

The same authors also implemented an International Registry of Brugada Phenocopies to classify systematic cases [8,9].

Brugada phenocopy and brugada syndrome: ECG morphology

- The ECG patterns in BrP are identical as in BrS.
- They are type 1 “coved” and type 2 “saddleback” patterns and can be recording in standard precordial leads V1-V2 or having on second rib space.
- The type 1 pattern (coved type) has a high take-off or the highest point of the QRS-ST-segment at least 2 mm over the isoelectric baseline followed by a down-sloping concave or rectilinear ST-segment with a negative symmetric T-wave.
- The type 2 pattern (typical saddleback pattern) shows an ascending and short slope with a high take-off ≥2 mm at the end of QRS, followed by a concave or rectilinear downsloping ST segment. T wave is positive in V2 and with variable morphology in V1 following ST-segment [10].
- But the value of ECG in discriminating BrP and BrS is limited, and a systematic diagnostic approach is indeed warranted. It is impossible to distinguish BrP from BrS by ECG only. Moreover, recent methodologies, including β-angle and the triangle base, failed to accurately identify BrS and BrPs’ ECG patterns [11-14].
- In BrS, coved-type (type 1 ECG pattern) is only diagnostic, and specific symptoms are mandatory together as syncope, palpitations, aborted sudden death, family history of premature death [15].

Brugada phenocopy and clinical conditions

Some underlying clinical conditions can trigger ECG variations to BrPs.

Currently, the authors have grouped six categories into the International Registry of Brugada Phenocopies (www.brugadaphenocopy.com): 1) metabolic condition, 2) mechanical compression, 3) ischemia and pulmonary embolism, 4) myocardial and pericardial disease, 5) ECG modulation, 6) miscellaneous [8].

The leading group of cases reported into the registry

refers to metabolic conditions included hyponatremia [16], hypokalemia [17,18], hyperkalemia [19,20], hypothermia [21], diabetic ketoacidosis [22], and hypopituitarism [23]. Electrolyte disturbances cause a transient outward potassium current leading to the amplification of action potential notch, which facilitates loss of action potential dome in the right ventricular outflow tract (RVOT). These transient abnormalities generate voltage gradients between the endocardium and epicardium of RVOT, and the Brugada ECG pattern appears. This pattern disappears when the triggering metabolic condition is resolved.

Ischemia and pulmonary embolism are the second major group of cases. Ischemia induces a BrP ECG pattern by transient ion channel dysfunction through increased outward potassium current and decreased inward sodium current. It can occur in the context of acute occlusion of the right coronary artery with transient ischemia of the RVOT. At the same time, pulmonary embolism may induce BrP due to ischemia caused by the critical pressure overload on the right ventricle [24-29].

Mechanical compression is another reported clinical condition to BrP. Authors have described several cases of mechanical compression due to tumoral chest mass as mediastinal lymphoma, intracardiac tumours or metastatic carcinomas [30-32]. The hypothesis of the mechanism of the Brugada ECG pattern is the transmural repolarization heterogeneity induced by direct mechanical compression of the RVOT and the ischemia caused by the lesion pressure over the RVOT. Both can induce transient ion channel dysfunction.

Myocarditis and pericarditis are also among the causes of BrPs. Inflammation-induced ischemia and myocardial fibrotic changes may induce slow depolarization and repolarization heterogeneity. This heterogeneity is the proposed potential mechanism causing BrPs in this group [33,34].

ECG modulation is another and particular cause of BrP due to the improper application of high-pass filtering. Garcia-Niebla., *et al*. reported a female patient's case in which a non-recommended high-pass filter of 0.5 Hz produced a Brugada pattern [35]. Generally, the high-pass filter eliminates low-frequency noises during ECG recording, and the usual cut-off is 0.05 Hz. On ECG ST segment is a low-frequency component and can be distorted when applying a non-standard cut-off frequency value.

At last, some authors described cases of BrPs in the context of different underlying conditions not classified into previous groups. These incorporate a patient with Ebstein anomaly [36], intracranial haemorrhage [37], and following electrical cardioversion for atrial fibrillation [38].

Pericardial tamponade and our case report

- Cardiac tamponade is a clinical syndrome caused by fluid accumulation in the pericardial space, resulting in reduced ventricular filling and subsequent hemodynamic compromise.
- Symptoms vary with the acuteness and underlying cause of the tamponade.
- Patients may present with dyspnea, tachycardia, and tachypnoea.
- In more severe cases, it may occur syncope due to mechanical obstruction to ventricular ejection.
- The ECG recorded during cardiac tamponade often demonstrates tachycardia, low voltage of QRS and sometimes alternating electrical voltage of QRS.
- These electrical signs express the mechanical compression exerted by pericardial effusion.
- We believe that our case is peculiar as BrP. If on one side on admission in the emergency room the patient presented typical clinical signs of Brugada Syndrome as syncope and coved type ECG, on the other hand, the systematic approach allowed us to achieve the correct diagnosis.
- Effectively the patient displayed signs and symptoms of cardiogenic shock. He showed a massive pericardial effusion (underlying condition), a low pretest probability for Brugada syndrome (no previous clinical and family history), no fever, no assumption of sodium channel block drugs. Despite this, the ECG was not typical for cardiac tamponade.
- The massive pericardial effusion affected the right ventricle because of the mechanical compression on RVOT. It developed the "coved" ST segment on the right precordial leads mimicking the type 1 ECG Brugada pattern through changes of the trans-membrane ionic flow of epicardial cells.

- Upon the pericardiocentesis and the restoration of physiological pressure in the pericardial sack, the Brugada type I pattern disappeared, and we have not observed the ECG pattern anymore.
- According to Brugada Phenocopies diagnostic criteria and classification, we have considered our case Type 1 Class A because all four mandatory criteria were satisfied.
- Unluckily, the patient passed away a few days later, and we have not performed the provocative test with ajmaline.

Conclusion

This case report demonstrates that a massive pericardial effusion may mimic Brugada syndrome through syncope and type 1 ECG Brugada pattern. Therefore, the pericardial effusion with tamponade may be among several causes of Brugada Phenocopy.

Conflict of Interest

No conflict of interest to declare.

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