

Brugada electrocardiographic phenocopy in a patient with chronic Chagasic cardiomyopathy

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Electrocardiographic description

A 44 year-old male with a history of chronic Chagasic cardiomyopathy (CChC) (exclusively dromotropic form), systemic hypertension and centripetal obesity, presented for a routine electrocardiogram (ECG). There was no history of syncope or sudden death and no evidence of heart failure. Laboratory tests were within normal ranges. There was a positive serological test for Chagas' disease.

There was no family history of cardiovascular disease and both parents had normal ECGs.

His medication included enalapril 10 mg twice a day and hydrochlorothiazide 25 mg daily. His echocardiogram showed moderate concentric left ventricular hypertrophy (interventricular septum and posterior free wall of 14 mm) and signs of decreased ventricular compliance.

His 12-lead ECG showed (Fig. 1): sinus rhythm, heart rate 57 bpm, prolonged P-wave du-

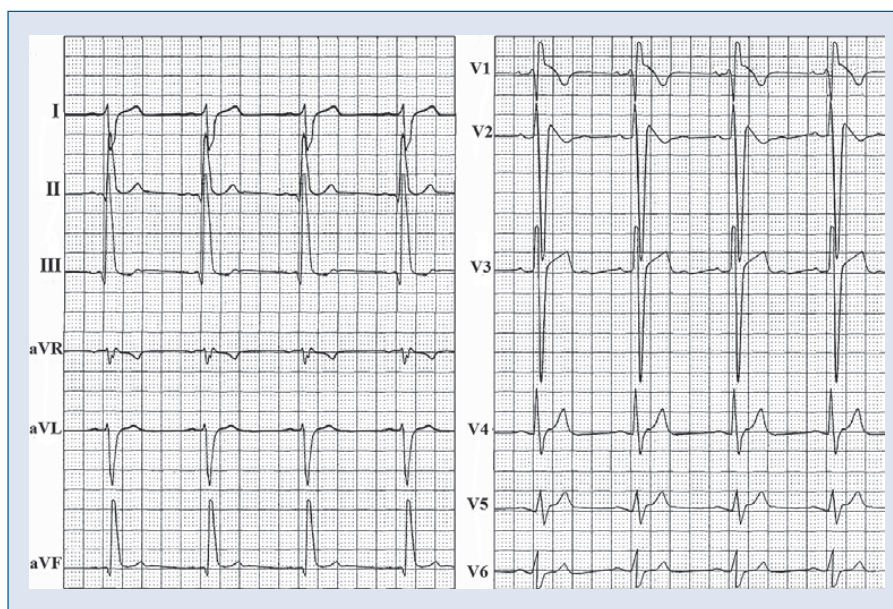


Figure 1. Sinus rhythm, heart rate 57 bpm, P-wave duration 120 ms, normal PR interval (160 ms), right QRS axis deviation ($+120^\circ$) and prolonged QRS duration (160 ms). Left posterior fascicular block: rS in lead I and aVL; qR pattern in leads II, III and aVF; R-wave in III > II, q-wave in III > q II. Complete right bundle branch block: QRS duration ≥ 120 ms; rsR' pattern in lead V1; broad S-wave in left leads I and aVL with a duration greater than 40 ms. QT interval 360 ms and QTc 350 ms. A Brugada-type 1 ECG pattern ('coved-type') can be seen in leads V1 and V2.

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Table 1. Brugada phenocopies previously described in the literature.

Drugs:	antiarrhythmic (class IA, IC, beta-blockers, calcium antagonists), nitrates, potassium channel openers, psychotropic agents (tricyclic and tetracyclic antidepressants, phenothiazine, selective serotonin reuptake inhibitors, lithium), anaesthetics (propofol, bupivacaine, lidocaine), anti-malarial agents, insulin, cocaine intoxication, histaminic H1 receptor antagonist dimonhydrinate, ketamine overdose
Electrolyte imbalance:	hyperkalemia, hyperkalcemia, severe hypokalemia
Acute ischemia:	right ventricular infarction
Hemodynamic changes that can affect the sodium channel:	fever, hypothermia, vagotonia
Neuromuscular disease:	myotonic muscular dystrophy or Steinert's disease, Friedreich's ataxia, Duchenne muscular dystrophy
Pectus excavatum	
Mechanical compression of RVOT:	mediastinal tumor, hemopericardium
Primary hyperparathyroidism	
Cardiomyopathies:	arrhythmogenic right ventricular dysplasia/cardiomyopathy, Chagas' cardiomyopathy, acute myocarditis
Brain injuries (subarachnoid hemorrhage)	
Left ventricular false tendons	
Pancreatitis, gallbladder disease	
Anaphylactic reaction/Kounis syndrome	

RVOT — right ventricular outflow tract

ration (120 ms), normal PR interval (160 ms), right QRS axis deviation (+120°) and prolonged QRS duration (160 ms). Left posterior fascicular block (LPFB) was observed: rS pattern in lead I and aVL, qR pattern in inferior leads, R-wave in III > II, q-wave in III > q II; complete right bundle branch block (RBBB). Additionally, a possible Brugada-type 1 ECG pattern ('coved type') in leads V1 and V2 was detected.

Does this patient have CChC and an asymptomatic Brugada syndrome?

Points to ponder

Brugada-type 1 ECG pattern can be seen in patients with Brugada syndrome, as well as in patients with other diseases, or secondary to drug or electrolytic disorders [1]. When the Brugada-type 1 ECG pattern can be explained by another disease (electrolyte imbalance, drug-induced, acute ischemia, increased insulin level, febrile states, hypothermia, mechanical compression of the RVOT, intracranial hemorrhage, etc.), the ECG is described as having a 'Brugada-like pattern' [2]. Recently, Pérez Riera et al. [3] introduced the term 'Brugada phenocopy' to describe the Brugada pattern that can be linked to a pre-existing and well known condition. They chose this term based on a previous definition of phenocopy: "an environ-

mental condition that imitates (copies) one produced by a gene". In their paper, the authors described a classic Brugada-type 1 ECG pattern in a patient intoxicated with propofol. In this particular case, the *environmental condition* was the infusion of propofol that triggered this particular ECG manifestation.

Cellular mechanism of ST-segment elevation in the so-called Brugada phenocopies could be explained by an intrinsically prominent I_{to} -mediated action potential (AP) notch in the epicardium but not in the endocardium, giving rise to a transmural voltage gradient [4].

The loss of the AP dome is produced by an outward shift of currents active at the end of phase 1 of the AP and in any interventions that increase outward currents (i.e. I_{to} , I_{K-ATP} , delayed rectifier potassium current I_{Ks} , I_{Kr}), or decrease inward currents: I_{Ca-L} , I_{Na} . Both conditions can accentuate ST-segment elevation leading to a Brugada phenocopy [4].

Drugs and clinical conditions capable of inducing transient ST-segment elevation in the right precordial leads may produce Brugada phenocopies. It remains uncertain whether the Brugada phenocopies are associated with a higher risk of malignant ventricular arrhythmias (similar to what happens with 'acquired Long QT syndromes') [4].

Table 1 summarizes the possible causes of Brugada phenocopies.

In the presented case, the patient had no history of sudden death, syncope or arrhythmia. There was no family history of Brugada syndrome. He was on no medication capable of inducing a Brugada phenocopy and had no electrolyte imbalance.

The patient had a conduction disorder (LPFB + + complete RBBB) which was compatible with CChC with ECG morphology in the right precordial leads resembling a Brugada ECG pattern.

Could he have both conditions?

In true Brugada syndrome, the width of the QRS interval duration is usually between 90 and 130 ms, but in the presented case the duration was 160 ms. It has been described that patients with Brugada syndrome could have left anterior fascicular block [5], but the presence of LPFB has not been described.

Left posterior fascicular block can be seen in CChC. However, it is not the most frequent conduction disorder.

In patients with true Brugada syndrome, an incomplete RBBB-like pattern, or even a complete atypical RBBB, can be present manifested by the lack of a broad S-wave in the left leads (I, aVL, V5, V6) [5].

In the presented case, there was a broad S-wave in the left leads indicating RBBB (in association with a LPFB, not described in true Brugada syndrome). Thus, Brugada-type 1 ECG pattern is less likely, and a Brugada phenocopy produced by conduction disorders associated with CChC is probably the correct diagnosis.

Conclusions

Brugada-type 1 ECG pattern can be seen in patients with well-known conditions, other than Brugada syndrome. These patterns are referred to as Brugada phenocopies. This is the first report of a patient with chronic Chagas' cardiomyopathy presenting as a Brugada phenocopy. The presence of a Brugada-type 1 pattern warrants exclusion of other medical conditions that can present with a very similar ECG.

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