Healthy Heart

Volume-9 | Issue-102 | May 5, 2018

Price : ₹ 5/-

Honorary Editor :
Dr. Vineet Sankhla

From the Desk of Hon. Editor:

Brugada Phenocopy (BrP) is a clinical entity in which patients present with an ECG pattern identical to either type 1 or 2 Brugada ECG patterns, yet it differs etiologically from true Brugada Syndrome(BrS). The defining feature of BrP is the absence of true congenital BrS. Therefore, a provocative testing with a sodium channel blocking agent such as ajmaline, flecainide or pro-cainamide will not reproduce the typical type 1 Brugada ECG pattern.

Two Independent Atrial rhythms can co-exist together, a condition called as atrial dissociation. The ECG finding of atrial dissociation is discussed in second ECG Challenge.

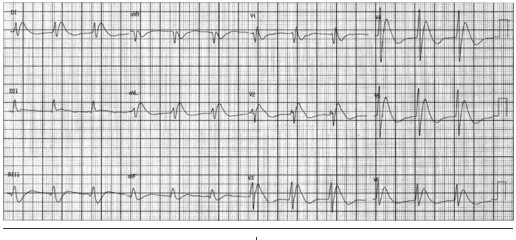
ECG CHALLENGES

Unusual ST-Segment Elevation in the Anterolateral Precordial Leads: Brugada Phenocopy

The patient is a 65-year-old male with history of type 2 DM, HTN, chronic smoking and prior stroke with residual left hemiparesis and aphasia. His medication included losartan, furosemide, simvastatin and met for min.

He was admitted to the emergency room in cardiac arrest and was quickly resuscitated with CPR and electric cardioversion maneuvers. Immediately after return of spontaneous circulation, a 12-lead ECG was performed (Figure 1).

Figure 1 ECG performed immediately after cardiopulmonary arrest reversion.



Based on the ECG, what is the most likely etiology of his cardiac arrest?

Response to ECG Challenge:

The first ECG shows accelerated junctional rhythm and a heart rate of 94 bpm, with J point and anterior ST elevation >2 mm. The correct diagnosis was acute ST elevation myocardial infarction. The patient underwent coronary angiography, which revealed proximal subocclusion of the LAD, with significant thrombus (Figure 2), successfully revascularized after placement of a DES. After the coronary intervention, we performed another ECG shown in Figure 3.

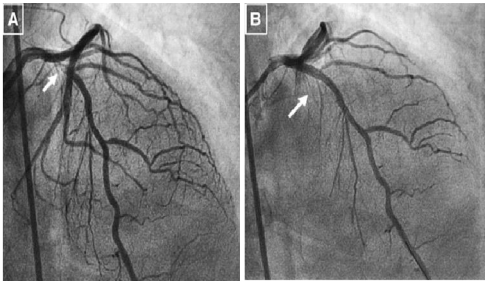


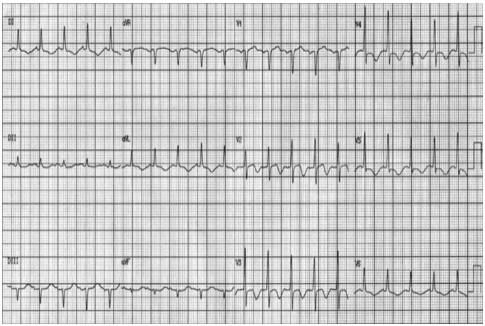
Figure 2

CAG with and without obstruction. LAD with 80% proximal obstruction with thrombus (A) and after DES placement (B).

Figure 3

ECG performed after PCI. Absence of Q wave infarction initially, loss of ST elevation with QRS narrowing. These changes are likely representative of reperfusion T wave inversions afterward. The postischemic diffuse T wave inversion is observed in the anterolateral wall, coincident with LAD territory, including its first diagonal branch (proximal LAD territory obstruction).

The initial ECG on presentation was compatible with ischemia-induced atypical Brugada Phenocopy (BrP)



Brugada Phenocopy Emerging as a New Concept

BrP is a clinical entity in which patients present with an ECG pattern identical to either type 1 or 2 Brugada ECG patterns, yet it differs etiologically from true Brugada Syndrome(BrS). The defining feature of BrP is the absence of true congenital BrS. Therefore, a provocative testing with a sodium channel blocking agent such as ajmaline, flecainide or procainamide will not reproduce the typical type 1 Brugada ECG pattern. The diagnostic criteria suggested for BrPs are the following (I–V are mandatory):

- Type 1 or 2 Brugada ECG pattern
- Underlying identifiable condition to explain the Brugada-like pattern on ECG
- The ECG pattern immediately resolves on resolution of the underlying condition
- Low clinical pretest probability of true BrS determined by a lack of symptoms and medical and family history
- Negative provocative testing with ajmaline, flecainide or procainamide
- Provocative testing is not mandatory if surgical right ventricular outflow tract manipulation has occurred within the last 96 hours
- Negative genetic screening (mutations are identifiable in only 20% to 30% of cases affected by true BrS)

The potential mechanisms and pathophysiology underlying BrP remain unclear. BrPs have been reported under a

multitude of clinical circumstances in the following distinct etiologic categories: metabolic conditions, endocrine disease (ie, hypopituitarism), electrolyte imbalances, mechanical compression (mediastinal tumors, pectus excavatum), ischemia-induced (the present case), myocardial and pericardial disease, acute pulmonary embolism and others. For a detailed list of conditions, please refer to the Educational Portal and International Registry on Brugada Phenocopies (www.brugadaphenocopy.com).

ECG CHALLENGE CASE -2

A 76-year-old man presented for a routine health evaluation. He reported no symptoms and the cardiovascular physical examination was within normal limits. He takes amlodipine for HTN. He was referred for a cardiology evaluation after the following ECG was performed.

Response to ECG Challenge

The ECG in Figure 1 shows 2 dissociated and independent atrial rhythms, a condition called atrial dissociation. An amplified view of the same ECG is shown in Figure 2, where the 2 rhythms are evident: one is a normal sinus rhythm (green arrows in lead II rhythm strip) at 60 beats/min with a QRS axis of +50 and a PR interval of 140 ms. The other is a rapid atrial rhythm (RAR) with a low-voltage P wave (red arrows in lead V1 rhythm strip) with a constant interval of 340 ms (178 beats/min). The 2 rhythms do not interfere with each other.

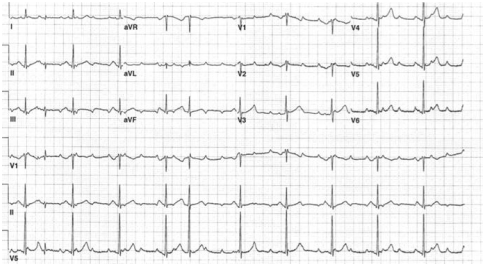


Figure 1 Initial ECG in an asymptomatic man with hypertension.

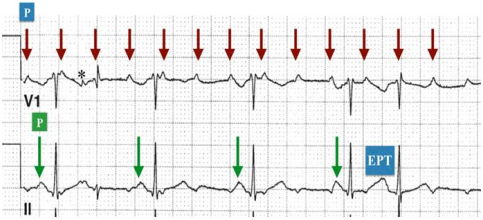


Figure 2

Atrial dissociation. Two dissociated and independent atrial rhythms are present on the same ECG. Top, A rapid atrial rhythm with red arrows tracking the P waves. Bottom, an independent basic rhythm (normal sinus rhythm) with green arrows tracking the P waves. The rapid atrial rhythm never conducts to the ventricles. Two ectopic premature beats are present: the first one (asterisk) conducts with aberrancy. The second one (EPT) overlaps on the T wave. Neither are related to the rapid atrial rhythm. EPT indicates ectopic P + T wave complex.

The second and sixth QRS complexes are premature and are preceded by ectopic atrial depolarizations not related to the RAR. The first premature QRS, with an aberrant conduction, is preceded by an ectopic atrial beat (asterisk), with a shorter interval with respect to the previous P wave of the RAR, in contrast with the constant P-P intervals of the RAR. The P wave of the RAR following the ectopic atrial beat overlaps on the conducted and aberrant QRS and there by is not recognizable.

The second premature QRS (sixth QRS complex) is preceded by an ectopic atrial beat, which overlaps on the previous T wave (ectopic P + T wave complex)

creating a visible notch and an apparent increase in voltage; the next P of the RAR overlaps on this conducted QRS. Again, the ectopic atrial beat does not interrupt the regular rhythm of the RAR.

In summary, the present ECG shows normal sinus rhythm and premature atrial contractions. There is a normal sinus P wave (green arrows) commanding the activation of the ventricles and another type of P waves (red arrows) representing the activation of only a confined and isolated portion of one atrium, generating a RAR. The letter has an entrant protective block, because it is not affected by the sinus activation, and also an exit block because its activation is not propagated to the rest of the atria and thereby to the ventricles. Some P waves of the RAR partially or totally overlap on the sinus P waves (Figure 3). However, they do not fuse. These findings are consistent with an ectopic, dissociated atrial rhythm.

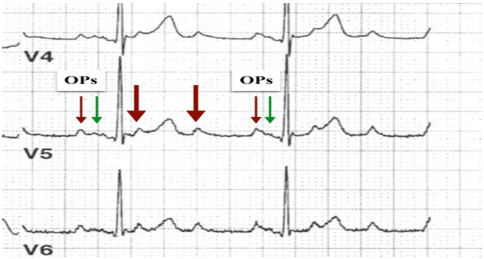


Figure 3

Overlapped P waves. Some P waves of the rapid atrial rhythm (red arrows) partially or totally overlap on the sinus P waves (green arrows). However, they do not fuse. OPs indicates overlapped P waves.

The existence of 2 independent P waves originating from 2 different areas of the atria has been described previously and is known as atrial dissociation. There are other scenarios where 2 independent P waves can be found in an ECG, such as in some patients with heart transplant. In such cases, 2 types of atrial tissue coexist: one is the remnant of the recipient's heart and the other from the donor's heart, initiating 2 different P waves that can be recorded, particularly when using additional ECG leads. These 2 P waves correspond to an independent activation of each isolated atrial tissue.

In our patient, given the benign nature of his ECG findings and lack of symptoms, no further testing or intervention was performed.

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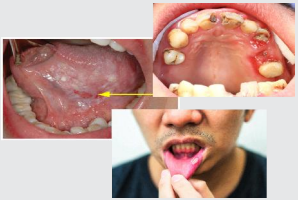

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Published from CIMS Hospital, Nr. Shukan Mall, Off Science City Road, Sola, Ahmedabad-380060.