

Bayés syndrome phenocopy in a patient with 1:1 atrial flutter and corrected tetralogy of Fallot

 Lucian Muresan,  Ronan Le Bouar,  Yasmine Doghmi,
 Crina Muresan,  Jacques Levy
 Department of Cardiology, "Emile Muller" Hospital; Mulhouse-France

Introduction

Bayés syndrome is currently defined as the finding of advanced interatrial block (a-IAB) on 12-lead ECG associated with atrial fibrillation (AF) (1). Several studies have demonstrated the high risk of developing atrial fibrillation for patients with a-IAB, which has practical consequences of anticoagulant administration (2-4).

Patients with tetralogy of Fallot have a high risk of developing supraventricular arrhythmias during their lifetime. The most common arrhythmias are represented by an atrial flutter, intra-atrial reentry, and atrial fibrillation (5, 6). Minimal data exist in the literature reporting the presence of a-IAB on the 12-lead ECG in patients with corrected tetralogy of Fallot and atrial flutter or atrial fibrillation, and even less about phenocopies of Bayés syndrome in these patients.

Case Report

A 56-year old male patient with past medical history of corrected tetralogy of Fallot (an initial palliative intervention at the age of 6 years and a second, complete repair, at the age of 12

years), percutaneous pulmonary valve implantation for severe pulmonary regurgitation at the age of 55 years, and one episode of paroxysmal atrial fibrillation who presented for palpitations and near-syncope. His medications included carvedilol 6.25 mg bid, eplerenone 25 mg od, perindopril 4 mg od, furosemide 20 mg od, Fluindione 20 mg od, and potassium supplements 600 mg tid. Clinical examination revealed a blood pressure of 107/71 mm Hg, heart rate of 260 bpm, SaO₂ of 97% breathing room air, and a temperature of 37 °C. His ECG recorded at admission and 5 minutes later are presented in Figures 1 and 2.

His echocardiogram revealed a mildly dilated left ventricle (ESD=55 mm) with severe LV systolic dysfunction, ejection fraction (EF) of 28% (Simpson), strain -8.6%, dilated right ventricle (48 mm at the level of the tricuspid annulus) and right atrium, sPAP of 33 mm Hg, and no pericardial effusion.

His biological workup showed no anemia, thyroid dysfunction, or electrolyte imbalance.

Considering his second ECG (Fig. 2), the wide QRS complex tachycardia was interpreted as atrial flutter with 1:1 A:V conduction. The patient was treated initially with IV amiodarone and oral carvedilol. An electrophysiological study was subsequently performed with the CARTO electro-anatomical mapping system, which demonstrated typical counterclockwise atrial flutter. Radiofrequency ablation of the cavotricuspid isthmus was performed, and the patient converted to sinus rhythm. The ECG in sinus rhythm at the end of the ablation procedure, recorded in the electrophysiology lab, is presented in Figure 3.

This ECG showed the presence of peculiar P waves, with an initial short and flat or positive component followed by a deep negative component in leads II, III, and aVF (best visible in lead II); positive in leads I and aVL, with a P-wave duration of 159 ms, suggesting the presence of a-IAB. However, on closer inspec-

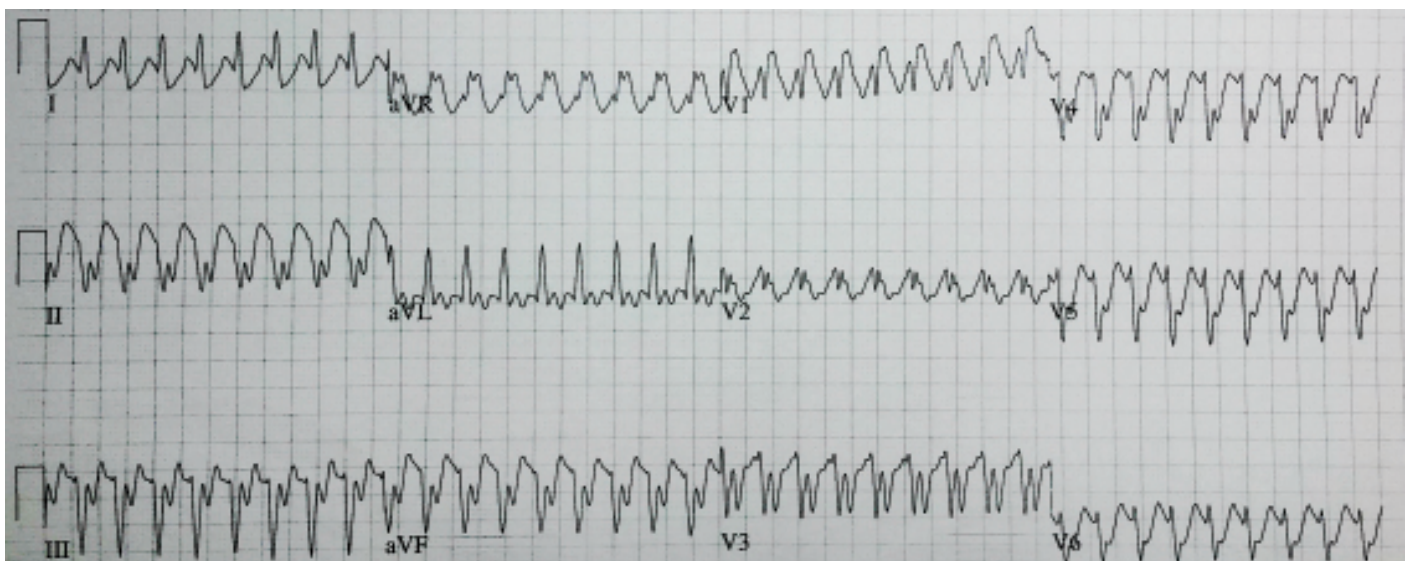


Figure 1. Wide QRS complex tachycardia with a heart rate of 260 bpm, right bundle branch block pattern, and left axis deviation. The differential diagnoses are ventricular tachycardia, supraventricular tachycardia with aberrancy, and pre-excitation syndrome

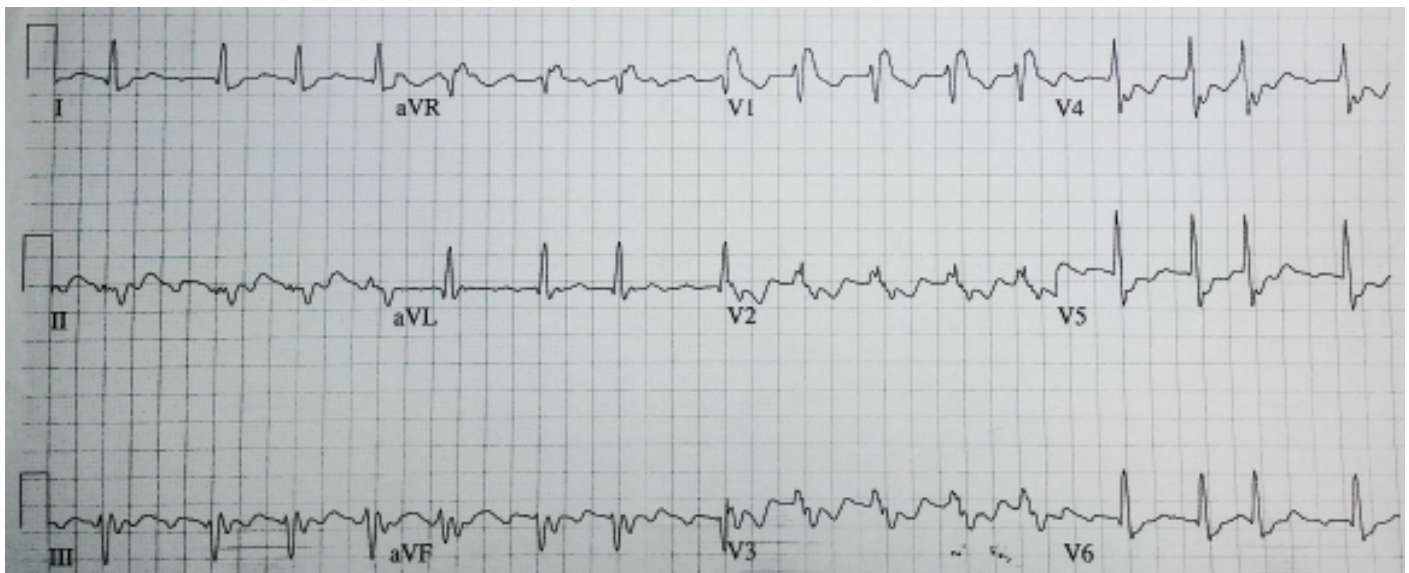


Figure 2. Twelve-lead ECG showing atrial flutter with 2:1 and 3:1 atrioventricular conduction, heart rate of 100 bpm, left axis deviation, and right bundle branch block

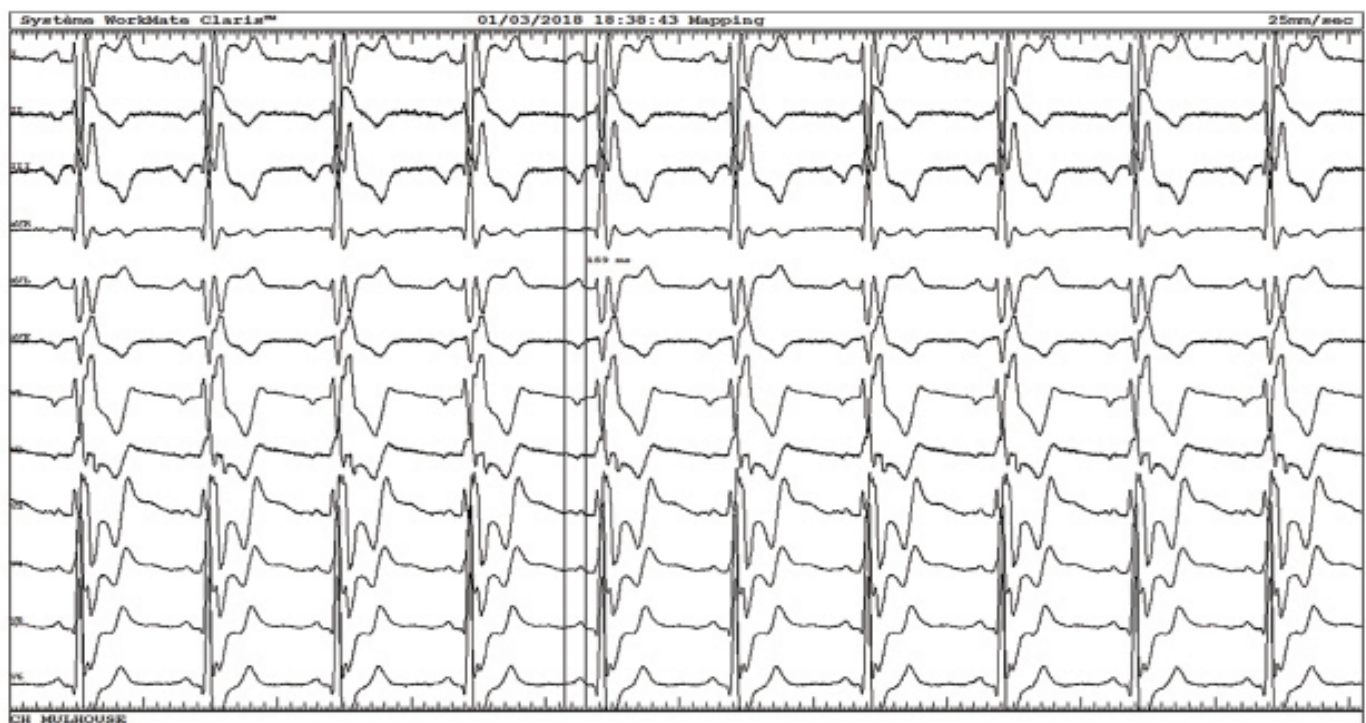


Figure 3. Twelve-lead ECG showing sinus rhythm, “north-west” axis deviation, and right bundle branch block. The P waves have an increased duration of 159 ms, with an initial short and flat or positive component followed by a deep negative component in leads II, III, and aVF, suggesting the presence of a-IB

tion, despite the presence of positive P wave in leads I and aVL, one could see that the morphology and the amplitude of the QRS complexes were different from the ECG in Figure 2, suggesting ECG electrode inversion.

A 12-lead ECG was recorded with the electrodes placed in the correct manner, which made the aspect of a-IB disappear (Fig. 4). The patient was discharged 48 hours later.

Discussion

We present the case of a Bayés syndrome phenocopy explained by a 12-lead ECG recorded with misplaced electrodes, masquerading as a-IB post catheter ablation of atrial flutter with 1:1 A:V conduction in a patient with corrected tetralogy of Fallot.

Table 1. Explanation of the incorrect placement of the ECG electrodes responsible for the “Bayés syndrome” phenocopy aspect in Figure 3

Electrode	Normal position	Modified position
Right arm (red color electrode)	Responsible for recording DI (with left arm electrode), DII (with left leg) and aVR (unipolar recording)	V1 (red color electrode)
Left arm (yellow color electrode)	Responsible for recording DI (with right arm electrode), DIII (with left leg electrode) and aVL (unipolar recording)	V2 (yellow color electrode)
Left leg	Responsible for recording DII (with left arm electrode), DIII (with right arm electrode) and aVF (unipolar recording)	Left arm
Right leg	Also called the indifferent electrode	unchanged
V1	4 th intercostal space, right parasternal	Right arm
V2 (yellow color electrode)	4 th intercostal space, left parasternal	Left leg (yellow color electrode)
V3	Halfway between electrode V2 and V4	unchanged
V4	5 th intercostal space, on the mid-clavicular line	unchanged
V5	5 th intercostal space, on the anterior axillary line	unchanged
V6	5 th intercostal space, on the mid-axillary line	unchanged

A correct 12-lead ECG recording requires standard placement of electrodes on the patient's limbs and chest, as follows: lead I is a bipolar recording of the electrical current traveling from the positive electrode (left arm) to the negative electrode (right arm). Lead II records the electrical activity between the left leg (positive electrode) and the right arm (negative electrode), and lead III records the electrical activity between the left leg (positive electrode) and the left arm (negative electrode). Leads aVR, aVL, and aVF are “augmented” unipolar recordings from the right arm, left arm, and left leg, respectively. The fourth limb electrode—the “indifferent electrode”—is placed on the patient's right leg. Any reversal between each of these two electrodes will create a typical change in the morphology of the P wave, QRS, and T wave. Therefore, reversing the two electrodes of lead I (right arm and left arm) will determine an inversion of polarity of the P wave and QRS such that the P wave and QRS complex become negative, a switch occurs between leads II and III, as well as between aVR and aVL, with the lead aVF staying unchanged. A reversal of the electrodes of lead II (right arm to left leg) will determine an inversion of the polarity of the P waves and QRS in lead II, with a switch between leads I and III and also between leads aVR and aVF, with lead aVL remaining unchanged. A reversal of the electrodes of lead III (left arm to left leg) will determine an inversion of the polarity of the P waves and QRS in lead III, a switch between leads I and II, as well as between aVL and aVF, with lead aVR remaining unchanged. With electrode reversal of either right arm to right leg, left arm to right leg, or bilateral arms to leg reversal, leads II, III, lead I, respectively, become isoelectric, which is not the case in our patient. Notably, the left leg to right leg reversal does not change the ECG aspect.

With these in mind, by examining Figures 3 and 4, one can observe that in Figure 3 lead I looks like lead aVF of Figure 4, lead II

looks like the mirror image of lead I of Figure 4, lead III is like the mirror image of lead II of Figure 4, aVR is like lead aVL of Figure 4, aVL is like lead aVF of Figure 4, and lead aVF is like aVR of Figure 4. However, no simple or combined limb lead electrode reversal is compatible with such an ECG aspect. A closer look at the P wave morphology in V1 and V2 reveals that the P wave has a slightly different morphology in Figure 4 compared with Figure 3, suggesting that the electrodes V1 and V2 have been misplaced too. The only misplacement combination of electrodes giving such an aspect on ECG is a switch between the right arm electrode and V1, placement of the V2 electrode on the left leg, placement of the left leg electrode on the left arm and placement of the left arm electrode in the position of V2 (Table 1), which is an unusual, albeit possible, misplacement pattern of the ECG electrodes, as explained probably by the fact that the reversed electrodes typically have the same color (right arm electrode and V1: red; left arm electrode and V2: yellow). Notably, the correct placement of the limb electrodes leads to the disappearance of the a-IAB pattern (Fig. 4).

Even though incorrectly recorded, we consider the ECG in Figure 3 is valuable because it mimicked Bayés syndrome and created a false aspect of a-IAB. Therefore, a Bayés syndrome phenocopy can occur because of a not-so-rare technical error, that is, incorrect limb electrode placement.

Conclusion

This case emphasizes the fact that a thorough inspection of the 12-lead ECG is warranted when providing care to patients and is, in our knowledge, one of the first reported cases of Bayés syndrome phenocopy.

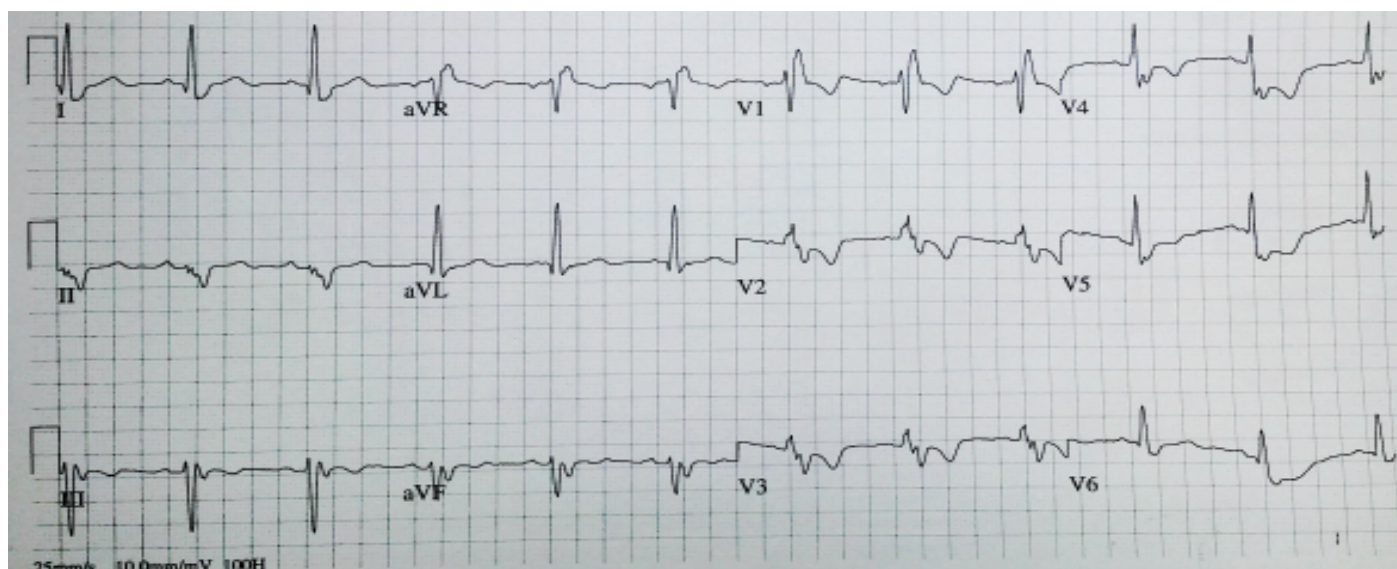


Figure 4. Twelve-lead ECG recorded with correctly-placed ECG electrodes, revealing sinus rhythm with a different morphology of the P wave and QRS complexes compared with Figure 3, with positive P waves in leads II, III, and aVF; positive in leads I and aVL; left axis deviation; right bundle branch block

Informed consent: Informed consent was obtained from the patient prior to the creation of the manuscript.

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Address for Correspondence: Lucian Muresan, MD,
Department of Cardiology,
"Emile Muller" Hospital;
20 Avenue du Docteur René Laennec, 68100,
Mulhouse-France
Phone: 00 33 389 646464
Fax: 00 33 389 647635
E-mail: lmure_san@yahoo.com
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Acute left main coronary artery occlusion following transcatheter aortic valve replacement without obvious risk factors of coronary obstruction

İD Beytullah Çakal, İD Sinem Çakal, İD Oğuz Karaca, İD Bilal Boztosun
Department of Cardiology, İstanbul Bağcılar Medipol Mega University Hospital; İstanbul-Turkey

Introduction

Transcatheter aortic valve implantation (TAVI) has emerged as a prominent alternative for patients with severe aortic stenosis, who are deemed to be at a greater risk for surgical interventions (1). However, extreme caution is recommended against the lethal complications, such as coronary obstruction (CO) or annulus rupture, associated with this procedure that need to be overcome urgently (1). Herein, we report an occlusion of acute left main coronary artery (LMCA) after TAVI, which was treated with triple stenting, due to immediate stent recoils.

Case Report

A 79-year-old woman, who was diagnosed with severe aortic stenosis, was referred to our clinic for the evaluation of TAVI. Because she had accompanying diseases including anemia and chronic obstructive pulmonary disease, the heart team concluded that the patient was at a high risk for surgery (a logistic