What is the Diagnosis?

CASE PRESENTATION

Syncope and QRS complex alternans

A 58-year-old man was hospitalized with syncope followed by fast restoration of consciousness (approximately, 15 seconds). The patient complained of fatigue or dyspnea on moderate efforts in the last seven months, which were aggravated by minimal activities over the last two weeks before hospitalization. He has a previous history of hypertension, myocardial infarction and coronary artery bypass grafting. Long-term medical therapy consisted of daily enalapril 10 mg, carvedilol 50 mg, furosemide 40 mg, atorvastatin 10 mg and aspirin 100 mg.

At the physical examination, the patient was in good general condition, without significant abnormalities. The blood pressure was at 132/84 mmHg and the heart rate at 62 bpm. Cardiac auscultation showed normophonic sounds, mild systolic murmur at a mitral focus and regular rhythm.

In the hospital, the patient complained of dizziness with ECG recording a sinus rhythm and second-degree atrioventricular block (2nd AVB) with alternation of right (RBBB) and left bundle branch block (LBBB) (Fig. 1). Serial ECG showed a period of 2:1 AV block and Wenckebach AV block (Fig. 2). The ECGs also demonstrated precordial Q wave (V1-V4) during RBBB pattern, which suggests an old anteroseptal infarction.

Figure 1. Second-degree atrioventricular block associated with right and left bundle brach block.
Chest X-ray showed an enlarged heart silhouette and metallic sternal suture. A transthoracic ECG was performed, which revealed an impaired left ventricular (LV) systolic function with a 25% LV ejection fraction (LVEF) (Simpson biplane), dilated LV (diastolic diameter of 72 mm), and mild mitral regurgitation.

Further evaluation for ischemic cardiomyopathy included cardiac magnetic resonance imaging (MRI) with gadolinium enhancement. This evaluation showed LV regional wall motion abnormalities and global dilatation of left chambers. There was reduced LV systolic function with a measured of LVEF of 22%. There was gadolinium enhancement of approximately 30% of the LV total mass. Delayed enhanced compatible with previous infarction was transmurally detected in the anterior and septal wall of the medioapical LV, and inferior and septal wall of the mediobasal LV. Absence of transitory perfusion abnormality or myocardial ischemia was also demonstrated by MRI.

The patient was submitted to cardiac resynchronization associated with defibrillator (TRC-D) with a good clinical response and recovery of functional class.

So, what is the mechanism of atrioventricular block?

**ANSWER**

The level of atrioventricular block was probably below of His bundle due to disease of the bundle branches with a fixed complete RBBB coupled to an intermittent abnormality of conduction through left bundle branch (LBB).

Baseline prolonged PR and HV intervals were presumed. Figure 3 represented variable degrees of slowed and blocked conduction in the His-Purkinje system. The P wave was intermittently blocked (2nd AVB) as AV conduction 2:1. Variable degree of slow conduction by the LBB coupled to fixed complete RBBB promoted different PR interval linked to the prolongation of HV interval and infraHissian AVB (Fig. 3a). The P wave was probably not conducted to the ventricle due to disease of the conduction system. However, there is a possibility that the P wave concomitant with the LBBB-QRS complex may be functionally blocked due to refractoriness of the His-Purkinje system, which was activated by the previous ventricular depolarization. Wenckebach phenomenon in the LBB occurred as show in Fig. 3b, which emphasizes the importance of the left branch in promoting AV blockade. Minimal PP variability was probably related to ventriculophasic sinus arrhythmia (Figs. 1 and 3a); the PP intervals, which contain a QRS complex, are shorter than the PP intervals that do not contain it.
Figure 3. Mechanism of atroventricular block, (a) with ECG showed in Fig. 1 and (b) with ECG showed in Fig. 2.

REFERENCE


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