Variations of Left Atrial Activation Patterns in Congestive Heart Failure

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We report about a case of a patient admitted to the Intensive Cardiac Care Unit for severe congestive heart failure which showed modification of P-wave morphology and duration, correlated with the clinical evolution. In the case here described, we show that ECG analysis, specifically P wave, allow us to assess the hemodynamic evolution of the acute decompensated heart failure patient.

Electrocardiographic examination is the first and the most simple and available diagnostic tool in the evaluation of patients with cardiac diseases. Usually, P-wave evaluation is not carefully assessed, in spite of very useful informations we can draw from its interpretation. We report about a patient admitted to our Intensive Cardiac Care Unit (ICCU) for severe congestive heart failure which showed peculiar modification of P-wave morphology and duration, well correlated with the clinical course.

Ann Noninvasive Electrocardiol 2012;17(2):153–155

interatrial block; congestive heart failure; Bachmann's bundle

CLINICAL CASE

A 45-year-old patient affected by Becker's muscular dystrophy, with severe left ventricle dysfunction and implanted with a cardiac defibrillator, was transferred from a secondary care hospital to our ICCU for the occurrence of severe acute decompensated heart failure poorly responsive to the usual medical therapy and for the evaluation of mechanical circulatory support or cardiac transplantation.

At admission in our hospital the patient presented a low-output syndrome and signs of congestive heart failure associated with *Staphylococcus aureus* sepsis from central venous catheter. An electrocardiogram (ECG) (12-lead ECG, Philips, Best, The Netherlands) revealed sinus rhythm, heart rate (HR) 90 bpm, interatrial block (P wave duration 165 msec), and a marked negative component of P wave in V₁ lead (Fig. 1); the echocardiogram showed mildly dilation of left ventricle (telediastolic diameter 68 mm, telediastolic volume 190 ml), left ventricle ejection fraction 0.25, moderate-to-severe mitral regurgitation, moderate tricuspi-

dal regurgitation, and systolic pulmonary artery pressure of 45 mmHg, severe biatrial dilation; NT-proBNP was 35,000 ng/L.

Therapy with inotropes (adrenalin and dopamine), sodium nitroprusside, i.v. diuretics and antibiotics was promptly started; the patients showed a slow and progressive resolution of symptoms and signs of systemic and pulmonary congestion and the infection was completely cleared up.

In the course of the hospital stay, along with the clinical improvement, the NT-proBNP decrease to 1240 ng/L and the electrocardiogram showed the decrease of P-wave duration (to 110 msec) and the reduction of the negative component of P wave in precordial V₁ lead and appearance of U waves, in spite of normal level of serum electrolytes (Fig. 2) when the patient was transferred to normal ward.

DISCUSSION

Under normal conditions, the electrical impulse generated in the sinus node is conducted through

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Figure 1. ECG at Admission. Interatrial block: Arrow indicates P-wave duration of 165 msec and a marked negative component of P wave in V₁ lead.

Figure 2. ECG at ICCU discharge. Arrow indicates the regression of interatrial block (P-wave duration of 110 msec), and reduction of the negative component of P wave in V₁ lead and the appearance of U waves.

the right atrium to the atrioventricular node by three internodal pathways, identified as anterior, middle, and posterior.¹ The anterior internodal pathways, the shortest one, give off a secondary branch, the Bachmann's bundle (BB), which course is along the superior quadrant of interatrial sulcus

and is responsible for the rapid propagation of the electrical impulse from the right to the left atrium² (Fig. 3).

In conditions such as valvular disorders, congestive heart failure, and hypervolemia, the increased atrial filling pressure can overstretch the interatrial

Figure 3. Scheme of internodal and interatrial pathways. Arrows indicate internodal and interatrial pathways and dashed arrow the Bachmann's bundle.

conduction pathways and determines a slowed conduction of the impulse to the BB, leading to a wide (duration > 110 msec), known as partial interatrial block.³

Recently, Holmqvist et al., using an electroanatomic mapping system, demonstrated the conduction of interatrial impulse through coronary sinus in case of interatrial block.⁴

When conduction in the interatrial bundle is blocked, the sinus impulse, in order to reach the left atrium, is forced to depolarize the right atrium descending to the area of atrioventricular junction, and activate the left atrial cavity exclusively in reverse mode, resulting in a biphasic P-wave morphology in inferior leads (advanced interatrial block).³

As previously showed by Ariyarajah et al.,⁵ the block in the interatrial conduction pathway can be reversible, and even the advanced interatrial block can regress to a partial IAB. Further-

more, diuretic therapy can reduce P-wave duration decreasing the atrium and interatrial pathways overstretch.⁶

In our patient the severe congestive status at admission determined the atrial overstretch with a significant increase in NT-proBNP level and the appearance of a partial interatrial block in the BB which resulted in a delayed left atrium activation and a prolonged and biphasic P wave in V₁. Clinical improvement following intravenous therapy was coupled with a decrease in NT-proBNP levels and the reduction of P-wave duration and regression of biphasic P-wave morphology in V₁.

In conclusion, the dynamic electroanatomical variations of left atrial activation pattern at standard ECG are correlated with atrial stretch due to hemodynamic imbalance; the evaluation of P-wave morphology and duration may allow us to evaluate the clinical course of the patients with acute congestive heart failure.

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