INTERESTING ELECTROCARDIOGRAM

COMPLEX CASE STUDY

Supraventricular Bigeminy-induced Conduction Delay in the Atria and Right Bundle Branch

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Case presentation

A 68-year-old gentleman with history of type 2 diabetes mellitus was diagnosed with non-ischemic dilated cardiomyopathy. Left ventricular ejection fraction was 20% and the etiology of the cardiomyopathy was presumed to be secondary to viral infection. He developed persistent atrial fibrillation (AF). He was treated with heart failure medications and a rate-control strategy for AF and oral anticoagulation. On follow-up visits, a marked improvement in heart failure symptoms was noted (ramipril 10 mg once daily, carvedilol 50 mg twice daily, spironolactone 25 mg once daily, and digoxin 0.25 mg once daily). Left ventricular ejection fraction improved to 32%. He was referred to the Arrhythmia Clinic for assessment of electrical cardioversion for symptomatic persistent AF. He underwent successful synchronized cardioversion without AF recurrences at 4 months’ follow-up.

ECG description

The immediate post-cardioversion 12-lead electrocardiogram (ECG) showed sinus rhythm with incomplete right bundle branch block (IRBBB) and left anterior fascicular block (LAFB) (Figure 1). An atrial bigeminy was also apparent. The P-wave duration is 110 ms in sinus beats and 130 ms in the premature atrial contractions (PAC). The coupling interval is almost fixed at 700 ms (± 20 ms), the compensatory pause presents some variation but it is around 1280 ms (± 80 ms). The PACs seems to be originated in the peri-sinus region, as the first component of the P-wave is positive in the inferior leads. The morphology of the P-wave in leads II, III, and aVF may suggest, at first glance, an advanced interatrial block (IAB). However, the diagnosis of advanced (third degree) IAB is difficult to confirm, as in lead aVF the P-wave presents two positive bimodal components. In order to confirm the diagnosis of advanced (third degree) IAB; the P-wave has to be positive–negative (+/−) in leads III and aVF, and positive–negative or positive–isodiphasic in lead II. This implies that the second component of the atrial activity is around −30° (caudo-craneal conduction). The PR interval of the sinus beat is 160 ms and the PR interval of the PAC is 200 ms. The QRS of the sinus beat depicts IRBBB+LAFB with a duration of 110 ms, and the QRS of the conducted PAC depicts complete right bundle branch block (CRBBB) + LAFB.

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Points to ponder

This is an interesting 12-lead ECG as it depicts two different conduction delays as a consequence of a PAC. The first one is the development of a partial interatrial block that is defined as a P-wave duration >120 ms. It cannot be labeled as advanced because a clear positive-negative morphology is not seen in lead aVF. Advanced interatrial block has been linked to very frequent development of AF constituting an arrhythmic syndrome now called “Bayes syndrome”. However, partial IAB has also been linked to higher incidence of AF and AF recurrences after therapy. Advanced IAB indicates structural and electrical remodeling that serves as substrate for supraventricular arrhythmias. Post-cardioversion IAB was previously identified as a possible risk factor for AF recurrence after electrical cardioversion.

Can we determine in this case whether IAB represents a dynamic functional (rate-related) conduction delay as we usually see it in the distal bundles? Unfortunately, we cannot be sure about this, as the origin of the PAC may play an essential role in the morphology of the P-wave. As we explained above, the origin of the PAC is in the higher right atrium (first component is positive in the inferior leads) and conducting from right to left as it is negative in lead aVR and positive in lead aVL. However, the “crista terminalis” can act as an electrical barrier before the impulse reaches the Bachmann bundle, thus giving the impression that all the conduction delay occurs in the Bachmann bundle. If the stimulus crosses the Bachmann with slow conduction, the resultant P-wave will be notched and longer than 120 ms, however; the positive/negative component in the inferior leads will not be present. In summary, we can determine that this PAC conducts with partial to advanced IAB (giving a minimum negative component in lead II) activating the left atrium in a caudal-cranial direction, most likely through the coronary sinus, however; we cannot confirm whether this delay is rate-dependent or origin-dependent.

It is also difficult to confirm the physiologic or pathologic nature of the CRBBB in the conducted PAC. In fact, the conduction disturbance complies with the “aberrancy rule,” but there are two facts that cannot be overlooked. An IRBBB is present in sinus beats denoting a mild (first degree) conduction defect in the right bundle. Furthermore, the coupling intervals of the PAC and those of the premature R-R intervals are relatively long.
leading to a pathologic phase 3 aberrancy in the right bundle branch, as it was explained decades ago. Analysis of surface 12-lead ECGs still provides interesting intellectual exercises that are crucial to the understanding of cardiac electrical problems.

References