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Early repolarization associated with accelerated atrioventricular conduction (short PR interval) and incomplete right bundle branch block: postulated mechanisms

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Abstract A 36-year-old male patient, who was admitted with atypical chest pain and had normal physical examination and laboratory findings, including on an echocardiogram and a maximal exercise treadmill/thallium test, is described. His electrocardiograms (ECGs) revealed ST-segment elevation in leads V_1 through V_1, suggestive of early repolarization variant (ERPV); in addition, there was evidence of accelerated atrioventricular conduction (short PR interval) and incomplete right bundle branch block (IRBBB). It is postulated that these 2 features represent ECG "correlates" of ERPV, which is characterized by accelerated repolarization and depolarization, and are due to a rapid conduction through all or some component(s) of the atria/AV-node/Hiss bundle/left bundle branch/left ventricle "chain," leading to a short PR interval and early and accelerated activation of the left ventricle, with resultant IRBBB. In addition to this being a case report, it constitutes a speculation that all the noted ECG findings are related and occurred in the context of ERPV; in all scientific fairness, it is possible that the accelerated atrioventricular conduction and IRBBB could have also occurred in a patient who happened to have ERPV. Finally, the occasional occurrence of morbidity and mortality in a patient with ERPV does not mitigate (at least until we know more) the time-honored belief that the ERPV is, after all, a benign ECG variant.

Keywords: ECG; Early repolarization; Normal variants; PR interval; Accelerated AV conduction; Right bundle branch block

Introduction

Two trenchant review articles by Boineau[1,2] and 3 responding editorials [3-5] have rekindled the concern whether the early repolarization variant (ERPV) is always a truly "normal variant" or an electrocardiographic (ECG) sign of occasional underlying pathology. As the sole ECG reader at my hospital, exposed to a large number of ECGs (>25 000 annually), I have noted, and puzzled about, the several ECG correlates of the ERPV,[1,2] beyond the ST-segment elevation (STE). I have been particularly bewildered by the slightly slurred onset of QRS complexes, simulating the delta waves of the Wolff-Parkinson-White syndrome and the short duration of the QRS complexes (<70 milliseconds)[1] in some patients with ERPV. More frequently, I have noted the peaked T waves, inverted T waves, and short QTc intervals in some young male subjects with ERPV, which promptly automated ECG interpretation systems[6] to include in the printout the diagnoses of "consider metabolic abnormalities" (implying hyperkalemia), "consider myocardial ischemia," and "consider hypercalcemia," respectively. In addition, patients with ERPV have tall R waves, reduced angle of the ascending limb of R waves, rapid QRS intrinsicoid deflection, prominent J waves (shown to be a repolarization event), reciprocal ST-segment depression (STD) in aVR, multiple peaks of T waves, and U waves.[1,7] More complex ECG patterns are also noted when ERPV coincides with left ventricular hypertrophy (LVH).[1] The present communication aims at bringing attention to 2 other heretofore undescribed ECG correlates occasionally found in subjects (patients) with ERPV: (1) accelerated atrioventricular (AV) conduction (AAVC; short PR interval), similar to what is seen in Lown-Ganong-Levine syndrome (LGLS),[8] and (2)

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incomplete right bundle branch block (IRBBB). An illustrated case of such a patient we cared for recently with ERPV and these 2 ECG correlates is presented, and speculation about the underlying mechanism is provided.

Case report

A 36-year-old man was admitted to the hospital with the complaints of atypical chest pain and palpitations. The chest pain was described as "squeezing" or "pressurelike," was of unclear duration, waxed and waned, occurred at rest, and was relieved by walking. There was no associated dyspnea, nausea, perspiration, or weakness. He smoked 1 pack of cigarettes daily and used cocaine daily for the past 3 years. There was no history of any medical illnesses or prior hospitalizations. On physical examination, heart rate was 75 beats per minute, systemic blood pressure was 108/55 mm Hg, and respirations were 12/min^{-1}; on auscultation, the lungs were clear, there was a splitting of the S_1 and S_2 with slight widening on deep inspiration and an increase in the intensity of the S_1. Laboratory testing values were normal, including a serum calcium level of 9.0 mg/dL (reference range, 8.5-10.5 mg/dL). Results of a chest radiograph and a 2-dimensional echocardiogram were normal, with the latter revealing no evidence of LVH or noncompaction and a left ventricular ejection fraction of approximately 60%. The admission ECG (Fig. 1) showed a heart rate of 65 beats per minute; a PR interval of 109 milliseconds; an IRBBB with an rSR' pattern in leads V_1 through V_3; with a QRS duration of 110 milliseconds; a QT of 439; a QTc of 456; frontal axes of the P waves, QRS complexes, and T waves of 64°, 59°, and 63°, respectively; and STE in leads V_1 through $V6_6$, suggestive of ERPV. There appeared to be a merging of R' and the J wave in lead V_1. He exercised for 13 minutes and 30 seconds on the Bruce protocol; reached a peak heart rate of 173 beats per minute, which represented 94% of the his predicted maximal heart rate, and peak blood pressure of 132/72 mm Hg; and had a normal ECG response and thallium-201 images at peak exercise and redistribution phases. The resting ECG before commencement of exercise (Fig. 2) also showed evidence of ERPV with STE in V_1 through V_6, short PR interval, an rSr' in leads V_1 and V_1, and a micro deltalike wave early in the inscription of the R wave of lead V_3. Some changes in the appearance of the ECGs in Figs. 1 and 2 are due to the use of the Mason-Likar stress ECG hook-up in the latter and probable variation in the placement of the precordial leads. Although there were no automated measurements in the exercise ECGs, the duration of the PR intervals appeared shortened. The STE in lead V_2 was of the "western saddle" variety (Figs 2 and 4).[1,2]

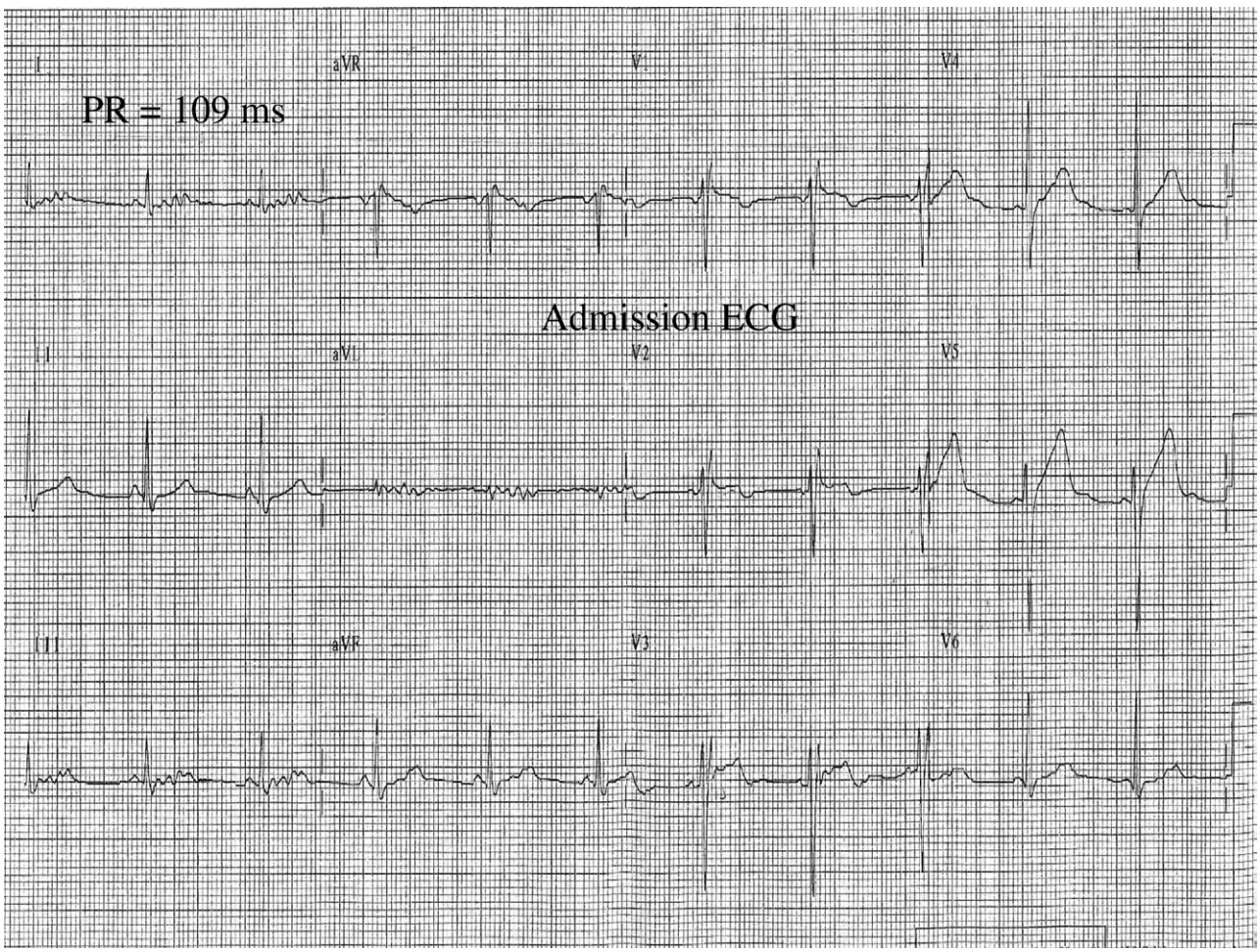

Fig. 1. Admission ECG; for details, see text.