

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Electrocardiogram Quiz – Case 9

A 71-year-old man presented to the emergency department of our hospital complaining of chest pain of 3 hours' duration. The patient mentioned a history of arterial hypertension under ramipril and atenolol and dyslipidemia under atorvastatin. He was hemodynamically stable with normal vital signs. Cardiac enzymes were positive and the diagnosis of a non-ST elevation myocardial infarction was set. The patient was admitted to the coronary unit for further investigation and treatment. The initial 12-lead surface ECG is depicted below.

Questions

- What abnormal ECG findings are present?
- What can we deduce about the patient's cardiac status?

Comment

Determining an underlying structural heart disease in an elderly man in the presence of a baseline left bundle branch block (LBBB)

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on surface ECG can be difficult. Ischemic or hypertensive heart disease or cardiomyopathy are plausible considerations. Especially the diagnosis of myocardial infarction, in the presence of LBBB can be considerably complicated. The reason is that LBBB alters both the early and the late phases of ventricular depolarization and produces secondary ST-T changes.

Our patient's ECG showed a borderline first degree atrioventricular block and a non-specific repolarization disorder. But the sharp notching of the upstroke of the S wave (0.04 sec) in the precordial leads told us more: this is the Cabrera sign. In patients with LBBB, the presence of the Cabrera sign in the precordial leads is considered to be reasonably specific and moderately sensitive for

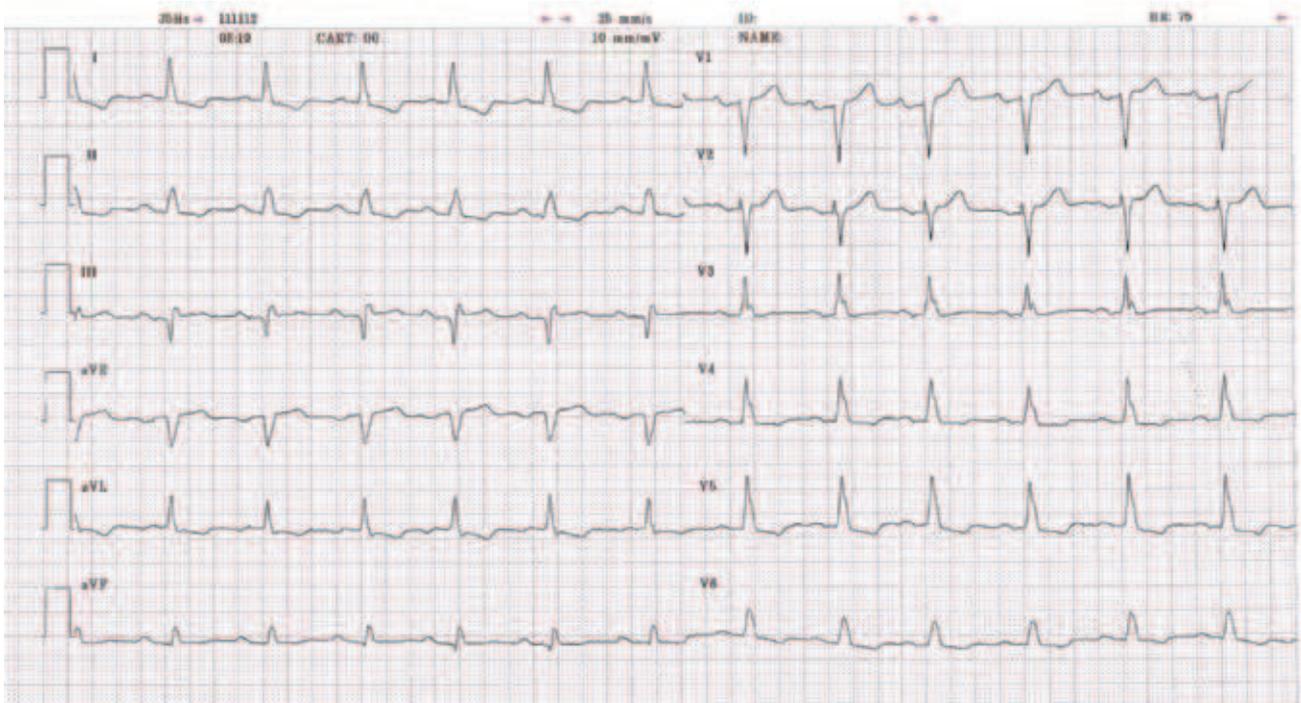


Figure 1

the presence of prior anterior myocardial infarction. There are three specific Cabrera sign variants; i.e. small, narrow r wave deforming the terminal QRS, series of tiny notches giving a serrated appearance along the ascending S wave, and similar series of late notches on QRS during epicardial pacing. In any case, notches are probably the result of a gross derangement of intraventricular conduction.

Several other signs have been proposed for the detection of myocardial ischemia and or infarction in the presence of LBBB. Chapman's sign is a notching ≥ 0.05 sec in the ascending limb of the R wave in lead I, aVL or V6. Furthermore, Sgarbossa et al, in 1996, described some ECG changes seen in those with LBBB and concomitant myocardial infarction and devised a point scoring system, known as the Sgarbossa criteria.

Moreover, among others, the aforementioned signs and criteria can be useful in the electrocardiographic diagnosis of myocardial infarction and ischemia in pacemaker patients. In this group of patients, ischemia diagnosis can be challenging due to initial electrical forces obscured by pacing stimuli, presence of ventricular fusion beats (with dual chamber devices), metabolic disorders (especially hyperkalemia), and cardiac memory phenomena.

Our patient underwent coronary angiography that revealed total occlusion of a dominant right coronary artery receiving collateral circulation from the left coronary tree. During his stay in the coronary unit he had an episode of sustained ventricular tachycardia which needed external defibrillation at 200 Joules. Finally, he received an automated implantable cardioverter-defibrillator and was discharged

with explicit instructions and medication.

In conclusion, the traditional view of impossible diagnosis of myocardial ischemia and or infarction in the presence of LBBB can be generally valid; however, the knowledge of specific signs on surface ECG could prove the opposite.

References

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