Introduction

It is well known that coronary spasm and atherosclerotic lesions are closely related.1 When the vasospastic episode involves the right coronary artery, ischaemia-related bradyarrhythmias can occur. This phenomenon has been attributed both to the ischaemia of the atrioventricular (AV) node secondary to impaired blood flow via the AV nodal artery and to the Bezold–Jarisch reflex due to an increased vagal tone secondary to ischaemia. We report a case of recurrent syncopes associated with bradyarrhythmias due to vasospasm of the right coronary artery diagnosed by the loop recorder examination.

Case Summary

A 44-year-old man with recurrent syncopes referred to our centre for a new syncopal episode. The medical history was unremarkable; no cardiovascular risk factors were reported. Two months earlier, he was evaluated in another centre where he underwent electrocardiogram (ECG) (Figure 1A) and echocardiography examination, which were normal and exercise ECG, which not able to achieve a significant heart rate (80%) and did not show symptoms or any ECG changes. Then, he was discharged after the implantation of a loop recorder device (Medtronic LINQ, Minneapolis, MN, USA). On admission, the loop recorder evaluation showed recurrent episodes of junctional rhythm (30–35 b.p.m.), lasting a few minutes, with marked ST elevation and a more pronounced negative T wave compared with basal electrogram.

Figure 1 (A) Basal 12-lead electrocardiogram recorded on admission shows mild and aspecific repolarization abnormalities in the inferior leads. (B) Template of the basal morphology of a normal QRS as shown by the loop recorder. (C) Episode of junctional rhythm (30–35 b.p.m.) and marked ST elevation with negative T wave visualized by the loop recorder. (D) Twelve-lead electrocardiogram showing marked ST elevation in inferior leads and reciprocal ST depression in the anterior leads.
Figure 1B and C) consistent with transient transmural myocardial ischaemia. During the hospital stay, the 12-lead ECG recorded during another pre-syncope confirmed a marked ST elevation in the inferior lead with reciprocal ST depression in the anterior leads (Figure 1D). The coronary angiogram was performed the day after showing an atherosclerotic plaque resulting in mild-moderate stenosis (30–40%) in the middle segment of right coronary artery; no other obstructive lesions were observed. Pharmacological therapy with diltiazem was titrated up to long-acting dosage of 300 mg, once daily. After 4 months, the patient is well and he hasn’t had syncopes anymore.

Discussion

Bradyarrhythmias and syncope can be associated with occlusion or spasm of the right coronary artery. Coronary vasospasm mostly affects sites of atherosclerosis resulting in episodes of myocardial ischaemia. In addition, this phenomenon can be associated with fatal ventricular arrhythmias and sudden cardiac death. In our case, no chest pain or ventricular arrhythmias occurred during the coronary spasm but ischaemia-related junctional rhythm resulted in marked bradycardia and recurrent syncopes. The novel generation of loop recorder devices such as Medtronic LINQ (Minneapolis, MN, USA) have considerably improved the quality of cardiac signal allowing a better definition of P waves and QRS morphologies. In our case, the QRS morphology during the coronary spasm, compared with the basal one, showed the evidence of transmural myocardial ischaemia, then confirmed later with the 12-lead ECG. Therefore, this case firstly shows a diagnosis of coronary spasm achieved through the analysis of a loop recorder previously implanted for unexplained syncopes. This case highlights a possible role of the loop recorder in the morphological analysis of the QRS beyond the traditional purpose of cardiac rhythm analysis, obviously in those cases when the electrograms are stored by the loop recorders for specific reasons (in our case the marked bradycardia).

Conflict of interest: none declared.

References