Apical Ballooning Syndrome in a Patient with Significant Coronary Artery Disease after Single-Chamber Pacemaker Implantation

Musuraca G1*, Agostoni P2, Imperadore F1, Terraneo C3, Boldi E4, and M. Del Greco1

1Division of Cardiology, S. Maria del Carmine Hospital, Rovereto, Italy
2Division of Cardiology, St. Antonius Ziekenhuis, Nieuwegein, The Netherlands
3Division of Cardiology, Policlinico Hospital, Monza, Italy
4Division of Cardiology, Clinica S. Rocco di Franciacorta, Ome, Italy

Abstract

Apical ballooning syndrome (ABS, called also Takotsubo Cardiomyopathy) is an acute cardiac problem and a stress induced cardiomyopathy that may mimic acute myocardial infarction, and is typically observed in postmenopausal women after stressful events. We describe a case of a 79-year-old woman who complained of dyspnoea the day after single-chamber pacemaker implantation, but no chest pain. Echocardiography showed a left ventricle with akinesia of the apical and antero-lateral wall portions and severe reduction of global systolic function. The patient was treated with intravenous heparin, and intravenous nitrates. Coronary angiography showed two significant stenosis in the middle portion of the left anterior descending artery and in the proximal-middle tract of the right coronary artery. A ventricular angiogram showed a typical takotsubo-like shape of the left ventricle was observed. The clinical and echocardiographic picture normalized at discharge.

Keywords: Apical ballooning; Coronary artery disease; Pacemaker

Clinical Case

A 79-year-old woman with history of hypertension and no previous cardiac symptoms was admitted to our hospital for dizziness and paroxysmal third-degree atrioventricular (AV) block.

Preoperative evaluation revealed normal hematological tests, chest X-ray, and echocardiography. The EGC presented sinus rhythm to alternate first-degree AV block, right bundle branch block, and left anterior fascicular block. A single-chamber pacemaker (PM) with ventricular lead in the right ventricle (RV) apex was implanted without procedural complications. During the night following the implant, the patient complained of marked dyspnea. The ECG showed rhythm with triggering PM ventricular stimulation (Figure 1). PM inhibition showed the preexisting intraventricular conduction disturbance but not ST segment change. Chest X-ray excluded pneumothorax but showed bilateral pleural effusion and normal position of the PM lead in the RV. In echocardiogram, akinesia of the apical, antero-lateral portions of the left ventricle (LV) with reduced LV systolic function was observed. Cardiac biomarkers were slightly increased. The patient was treated with intravenous nitrates and intravenous heparin due to suspicion of acute coronary syndrome. Coronary angiography was performed and showed two significant stenoses in the middle portion of the Left Anterior Descending Artery (LAD) and in the proximal-middle tract of the Right Coronary Artery (RCA) (Figures 2–3), but no clear “culprit lesion”. The left ventricular angiogram confirmed the diagnosis of Apical Ballooning Syndrome (ABS) (Figure 4). In the following days, the clinical condition of the patient gradually improved. A control echocardiography before discharge showed normalization of the LV systolic function, and disappearance of the wall motion abnormalities except of the dyssynchrony due to RV stimulation. The patient was discharged without symptoms, with effective single-chamber RV stimulation, and normalized systolic LV function with an ejection fraction of 60%.
The ABS was first described in Japan in early 1990s, where it was named Takotsubo Cardiomyopathy, due to the similarity between the dilated LV and an ancient Japanese fishing pot used to trap (tsubo) octopuses (tako). Although the exact mechanism of this syndrome is unknown, it is thought to be due to an excess in catecholamine release, mainly norepinephrine, in stressful physical or psychological conditions and is thus named stress-induced cardiomyopathy [1]. Alternatively, it could be due to transient coronary occlusion by spasm or ruptured plaque [2]. Pacemaker implantation could be associated with a number of acute complications related to the procedure, the patient and/or the device itself [3]. Pacemaker implantation even if uncomplicated may be sufficiently stressful in individual patients to precipitate this syndrome as previously described in some cases [4–5]. In our case, there was no other cause for acute deterioration of LV function, and the diagnosis of apical ballooning syndrome was made with an echocardiogram and confirmed by angiography and ventriculography. The classical course of gradual and complete resolution of the regional wall abnormalities and normalization of the LVEF except the dysynchrony due to RV stimulation, at follow-up helped to confirm the diagnosis [6–7]. In our case, diagnosis may be more difficult because the absence of ECG changes that are masked by pacing [8]. We conclude that device implantation may represent a psychophysical stress that may be complicated by ABS. In our case, coronary artery disease was present but no culprit lesion was present, it was thus most probably a coincidental finding.

The most common ECG presentation of ABS is ST segment elevation in precordial leads suggesting anterior wall acute myocardial infarction. Concomitant ST segment elevation or absence of ST segment depression in inferior leads has been proposed as an adjunctive ECG abnormality distinguishing ABS [9]. It is also interesting to note that acute myocardial ischemia may be difficult to identify based on ECG analysis in PM patients paced in the apex of the RV. Several signs were proposed, but they are all insensitive [10]. Indeed, in symptomatic patients, the diagnosis should be based on new development of ST elevation [10–11].

Conclusion

In conclusion, ABS should be suspected after pacemaker implantation in patients presenting with angina-like symptoms and/or dyspnea, especially if a new development of ST modification and LV systolic dysfunction is present. The association between ABS and coronary artery disease, as in our case, is extremely rare in the medical literature.

References


Discussion

The ABS was first described in Japan in early 1990s, where it was named Takotsubo Cardiomyopathy, due to the similarity between the dilated LV and an ancient Japanese fishing pot used to trap (tsubo) octopuses (tako). Although the exact mechanism of this syndrome is unknown, it is thought to be due to an excess in catecholamine release, mainly norepinephrine, in stressful physical or psychological conditions and is thus named stress-induced cardiomyopathy [1]. Alternatively, it could be due to transient coronary occlusion by spasm or ruptured plaque [2]. Pacemaker implantation could be associated with a number of acute complications related to the procedure, the patient and/or the device itself [3]. Pacemaker implantation even if uncomplicated may be sufficiently stressful in individual patients to precipitate this syndrome as previously described in some cases [4–5]. In our case, there was no other cause for acute deterioration of LV function, and the diagnosis of apical ballooning syndrome was made with an echocardiogram and confirmed by angiography and ventriculography. The classical course of gradual and complete resolution of the regional wall abnormalities and normalization of the LVEF except the dysynchrony due to RV stimulation, at follow-up helped to confirm the diagnosis [6–7]. In our case, diagnosis may be more difficult because the absence of ECG changes that are masked by pacing [8]. We conclude that device implantation may represent a psychophysical stress that may be complicated by ABS. In our case, coronary artery disease was present but no culprit lesion was present, it was thus most probably a coincidental finding.

The most common ECG presentation of ABS is ST segment elevation in precordial leads suggesting anterior wall acute myocardial infarction. Concomitant ST segment elevation or absence of ST segment depression in inferior leads has been proposed as an adjunctive ECG abnormality distinguishing ABS [9]. It is also interesting to note that acute myocardial ischemia may be difficult to identify based on ECG analysis in PM patients paced in the apex of the RV. Several signs were proposed, but they are all insensitive [10]. Indeed, in symptomatic patients, the diagnosis should be based on new development of ST elevation [10–11].

Conclusion

In conclusion, ABS should be suspected after pacemaker implantation in patients presenting with angina-like symptoms and/or dyspnea, especially if a new development of ST modification and LV systolic dysfunction is present. The association between ABS and coronary artery disease, as in our case, is extremely rare in the medical literature.

References

