A right ventricular outflow tract focus triggering tachycardiomyopathy in a peripartum patient exposed to radiation and chemotherapy

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Case report

A 38-year-old patient affected by asymptomatic (NYHA I) mild left ventricle (LV) systolic dysfunction (ejection fraction 50%) secondary to previous chest ionizing radiation and chemotherapy exposure was referred to our institution due to functional class (NYHA IV) and left ejection fraction worsening (30%) 2 months after her second pregnancy and concomitant to numerous (>35 000/24 h) monomorphic ventricular ectopic beats (VEBs) from the right ventricle outflow tract (RVOT).

The patient had a ganglioneuroblastoma treated by high-dose radiotherapy and chemotherapy (doxorubicin, vincristine, and cyclophosphamide) when she was 2 years old. Then, a stable mild LV systolic dysfunction (ejection fraction 50%) persisted, not requiring cardiac therapy as she was completely asymptomatic. She was a former smoker until the age of 36 when she stopped as was giving birth to her first child. During the first pregnancy cardiac function remained stable.

A year later she became pregnant for a second time. During the second trimester, she complained of dyspnea (NYHA III) and systolic cardiac function deteriorated to ejection fraction 40%, also documented by brain natriuretic peptide increase beyond reference limit. She was therefore diagnosed with acute decompensated chronic heart failure and was given furosemide 25-mg bid, bisoprolol 1.25-mg bid, and nocturnal transdermal nitrates. Cardiac conditions improved and caesarean stenting. As VEBs did not decrease even after revascularization, she underwent catheter ablation of the RVOT focus (Fig. 1b: right and left lateral Carto, Biosense Webster, mapping system views localizing the ablated spot, red dots, at the septal portion of the RVOT; colours relate to activation during VEBs with red being early and blue-violet late activation). At 3-month follow-up, ECG (please note incomplete left bundle branch block, Fig. 1c) and 24-Holter recording (288 VEBs) documented complete resolution of the arrhythmia; at the same time, symptoms and ejection fraction were restored to basal (ejection fraction 50%, NYHA I).

Discussion

Cardiomyopathy during pregnancy is uncommon but potentially catastrophic to maternal health, accounting for up to 11% of maternal deaths.¹ Peripartum cardiomyopathy (PPCM) is currently defined on the basis of the presence of three criteria: I – development of symptomatic heart failure towards the end of pregnancy or in the following month her clinical conditions rapidly deteriorated (NYHA IV, ejection fraction 30%) despite maximal heart failure therapy.

At admission, accordingly to history of chest radiation exposure, even though the patient had never complained of angina, a coronary angiography was performed and revealed severe calcific proximal right coronary artery stenosis that was treated by percutaneous drug-eluted stenting. As VEBs did not decrease even after revascularization, she underwent catheter ablation of the RVOT focus (Fig. 1b: right and left lateral Carto, Biosense Webster, mapping system views localizing the ablated spot, red dots, at the septal portion of the RVOT; colours relate to activation during VEBs with red being early and blue-violet late activation). At 3-month follow-up, ECG (please note incomplete left bundle branch block, Fig. 1c) and 24-Holter recording (288 VEBs) documented complete resolution of the arrhythmia; at the same time, symptoms and ejection fraction were restored to basal (ejection fraction 50%, NYHA I).

The cause of PPCM has remained unclear. Recent research, however, suggests that PPCM could be a vascular disease (triggered by hormonal changes in late pregnancy) in which apoptosis in endothelial cells plays a central role.² On the other side, arrhythmias are the most common cardiac complication encountered during pregnancy in women with and without structural heart disease.³ The haemodynamic changes of pregnancy and postpartum have been well studied,⁴ and volume overload is the major contributor to arrhythmia development.
In the present patient, we observed two distinct haemodynamic deteriorations: whether the first (II trimester of the last pregnancy) seems due to worsening of a preexistent LV systolic dysfunction in a radiated heart or to PPCM, the second (2 months after delivery) appears, instead, to be a tachycardiomyopathy due to

Ventricular ectopic beats in patient (a) originating from the right ventricle outflow tract unresponsive to high-dose beta-blocker treatment (first bisoprolol 5 mg q.d., then metoprolol 100 mg bid); (b) after catheter ablation of the RVOT focus; and (c) at 3-month follow-up ECG.
numerous VEBs. If VEBs just happened to occur during postpartum or if the latter functioned as a trigger remains unanswered; in any case, the clinical situation was worsened by the underlying radiation and chemotherapy exposed heart. In fact, we believe the calcific proximal coronary stenosis is a ‘silent’ bystander contributing to worsening of symptoms during both haemodynamic deteriorations but probably not the cause of either of the two.

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**References**