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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 years old patient affected by asymptomatic (NYHA I) mild left ventricle systolic dysfunction (ejection fraction, EF 50%) secondary to previous chest ionizing radiation and chemotherapy exposure was referred to our Institution due to functional class (NYHA IV) and left EF worsening (30%) two months after her second pregnancy and concomitant to numerous (>35000/24 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, cyclophosphamide) when she was two years old. Since then a stable mild left ventricle systolic dysfunction (EF 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 his first child. During the first pregnancy cardiac function remained stable.

A year later she remained pregnant for a second time. During the second trimester she complained dyspnea (NYHA III) and systolic cardiac function deteriorated to EF 40%, also documented by Brain Natriuretic Peptide (BNP) increase beyond reference limit. She was therefore diagnosed 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 section was performed at 34 weeks due to obstetric indication. Following delivery the patient was given enalapril 5 mg bid, bisoprolol was up titrated to 5 mg q.d. and furosemide was stopped due to complete symptoms relief and documentation of EF restoration (to basal EF 50%) and. Two months later, however, she presented with palpitations due to numerous monomorphic VEBs originating from the RVOT (Figure 1a),

unresponsive to high dose beta-blocker treatment (first bisoprolol 5 mg q.d, then metoprolol 100 mg bid). In fact, in the following month her clinical conditions rapidly deteriorated (NYHA IV, EF 30%) despite maximal CHF therapy.

At admission, accordingly to history of chest radiation exposure, even though the patient had never complained 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 (Figure 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, Figure 1c) and 24-Holter recording (288 VEBs) documented complete resolution of the arrhythmia; at the same time symptoms and EF were restored to basal (EF 50%, NYHA I).

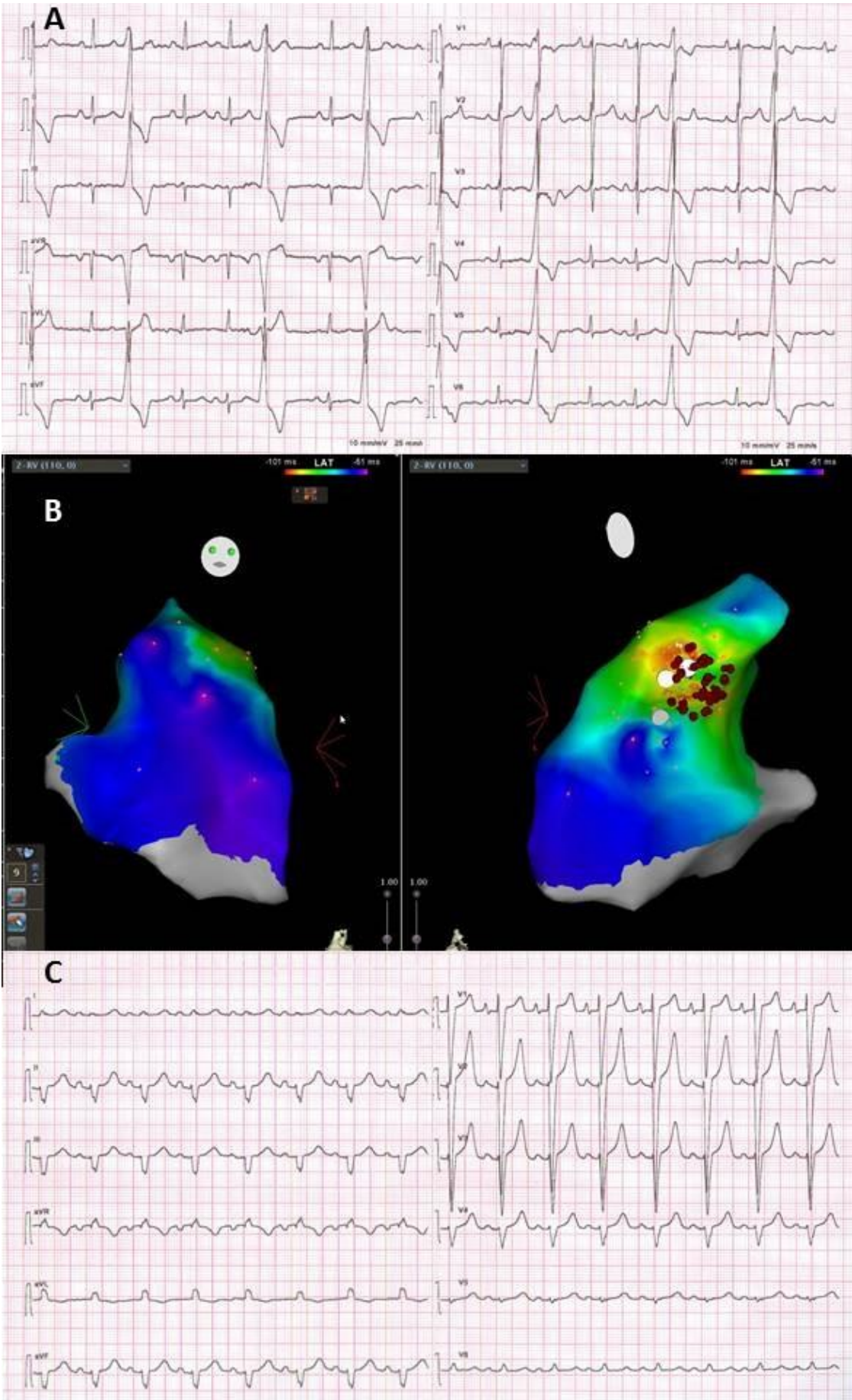
Discussion

Cardiomyopathy during pregnancy is uncommon but potentially catastrophic to maternal health, accounting for up to 11% of maternal deaths¹. Peripartum cardiomyopathy (PPCM) is currently defined based on the presence of three criteria: I - development of symptomatic heart failure toward the end of pregnancy or in the months following delivery (usually 3-4 months); II - the absence of an identifiable cause of heart failure; and III - left ventricular systolic dysfunction (LV ejection fraction <45% on echocardiogram)². 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 role³. On the other side arrhythmias are the most common cardiac complication encountered during pregnancy in women with and without structural heart

disease⁴. The haemodynamic changes of pregnancy and postpartum have been well studied⁵ and volume overload is the major contributor to arrhythmias 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 pre-existent left ventricle systolic dysfunction in a radiated heart or to PPCM, the second (two months after delivery) appears, instead, to be a tachycardiomyopathy due to numerous VEBs. If VEBs just happened to occur during postpartum or if the later functioned as 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 a “silent” bystander contributing to worsening of symptoms during both haemodynamic deteriorations but probably not the cause of either of the two.

Figure 1.



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