EP CASE REPORT

Successful catheter ablation of ventricular ectopy in a young patient with implanted Melody valve

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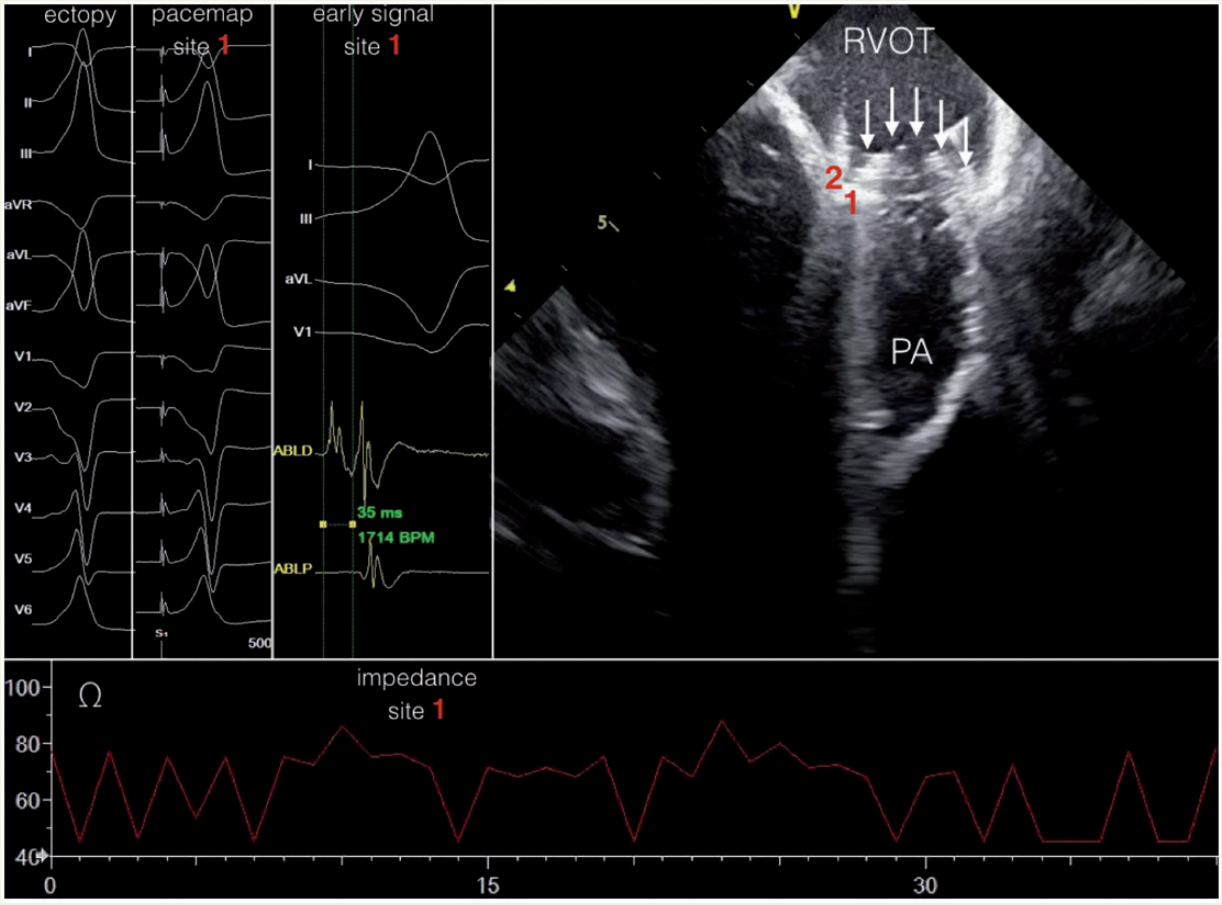
Case report

A 17-year-old male after transcatheter implantation of Melody pulmonary valve (TPV) (Medtronic) was admitted for catheter ablation (CA) of symptomatic premature ventricular complexes (PVCs—figure). The patient had a history of surgical correction of truncus arteriosus with aortic homograft at the pulmonary position at the age of 1 year. At the age of 4 years, he had another surgical procedure with exchange of homograft for Contegra valved conduit (Medtronic). Due to moderate stenosis and severe regurgitation of the valved conduit a TPV Melody was implanted at the age of 15. Series of echocardiographic follow-ups showed normal valve function, moderately dilated right ventricle (RV) with thickened walls and normal systolic function. The presence of monomorphic PVCs was already registered before the TPV implantation. Morphology of PVCs implied origin from the right ventricular outflow tract (RVOT). The daily burden of the ventricular



ectopy gradually increased from 10% to 30% after the TPV implantation. PVCs were highly symptomatic and were refractory to treatment with bisoprolol.

The ablation procedure was performed in general anaesthesia. Femoral veins and artery were used for vascular access and RVOT was mapped with an irrigated 4-mm tip catheter (Coolflex, SJM). Catheter manipulation was guided by intracardiac echocardiography (Siemens Medical Solutions) and NAVX (SJM) electroanatomical mapping system (EAM). Activation mapping and pace-mapping were used for allocation of PVC site of origin. The best pace-map and the earliest intracardiac bipolar electrogram (−35 ms) were found at the proximal end of the TPV stent (*Figure*, site 1). Ablation at that location was attempted, but was not successful due to frequent impedance changes (*Figure*, lower panel). Subsequently, ablation at a more proximal location underneath the stent, where the tip of the ablation catheter was not touching the stent (*Figure*, site 2), resulted in stable impedance drop and successfully abolished PVCs. There were no procedure-related complications. The patient was free of palpitations at the 3-month follow-up, and 24-h ECG monitoring revealed very sporadic PVCs.

Discussion

To the best of our knowledge this the first case report of a successful CA of symptomatic PVCs in the presence of the TPV. In general, PVCs originating from RVOT may be idiopathic in origin, but in patients with congenital heart disease, they may mark right ventricular impairment and volume overload. Potential mechanical irritation by TPV implantation may influence the burden of PVCs.[1] In our case, significant ventricular ectopy was already present before TPV implantation and it is difficult to estimate its effect on augmented PVC burden. Furthermore, there is available evidence that majority of new onset post-TPV implantation PVCs resolve during medium-term follow-up.[2] CA of ventricular ectopy arising from the RVOT is a routine procedure.[3] However, ablation can be challenging in the presence of the TPV valve. Specifically, contact of the stent with the ablation catheter can lead to shunting of the radiofrequency current and consequently ineffective ablation lesions. Current shunting can be detected as high-impedance variations during ablation. In our case, the successful ablation site was located under the edge of the stent, where stable impedance drop during ablation was achieved. Whether alternative energy source (i.e. cryoablation) could be successfully used within the stent itself is not known. Considering the technical challenges associated with ablation in the presence of a Melody valve, CA of PVCs originating form RVOT may be considered before the TPV implantation.

Conflict of interest: none declared.

References

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