



Original Article

# Catheter Ablation for Premature Ventricular Complex Induced Cardiomyopathy: A Case Report and Brief Review of Literature

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## Abstract

Premature ventricular complex (PVC) induced cardiomyopathy is a potentially reversible left ventricular dysfunction induced by frequent PVCs. The left ventricular (LV) dysfunction improves once the PVCs are cured. We, hereby, present a case of symptomatic PVC induced cardiomyopathy in an adult male. PVCs were originating from left ventricle outflow tract. PVCs were mapped by 3D electro-anatomical mapping system and successfully ablated. The patient remained asymptomatic during the follow up appointments. A repeat echocardiogram obtained at three months showed that LV functions had normalized. (Indian J Cardiol 2022;25 (3-4):21-25)

## Case summary

A 53 years old male referred for PVC ablation. The patient was suffering with symptomatic PVCs for several months that progressed to LV dysfunction. Physical examination and biochemical tests were unremarkable. There was no family history of cardiac arrhythmia. Two separate 24-hour Holters documented high PVC burden of 25,000 and 30,000 per day on beta-blockers respectively. Coronary angiography confirmed normal epicardial coronaries. Cardiac magnetic resonance imaging showed no scar and LV ejection fraction of 41%. The presenting rhythm was normal sinus rhythm with frequent PVCs. The clinical PVCs are having inferior axis, QS in I/aVL/aVR, rS in V1 with transition in V3 (Figure 1). The EnsiteNavX 3D electroanatomical mapping system was used to define chamber geometry. A detailed geometric and activation map of the right ventricular outflow tract (RVOT) was created that demonstrated the early intracardiac electrograms (EGMs) -10msec pre-PVC at

posterior RVOT. Afterwards detailed geometric and activation map of the left ventricular outflow tract (LVOT) especially around cusps was created and demonstrated the early EGMs -35msec pre-PVC (Figure 2). The earliest signals were recorded from the left cusp near left main ostium. A 6 French Judkin's right 3.5 diagnostic catheter (Cordis, Miami, Florida, USA) was advanced and left main contrast injection was done to locate the safety of burns near left main ostium (Figure 3). Using 3.5 mm FlexAbility™ irrigated ablation catheter (Abbott, St. Paul, MN) radiofrequency applications delivered at left cusp eliminated the clinical PVCs. Additional radio-frequency burns were delivered to consolidate the lesion. Afterwards isoproterenol infusion was started and increased to a maximum dose of 10mcg/min. No PVCs seen till 30 minutes waiting period (Figure 4). The patient remained asymptomatic during follow up appointments, a repeat echocardiogram obtained at three months showed that LV functions had normalized (Figure 5).

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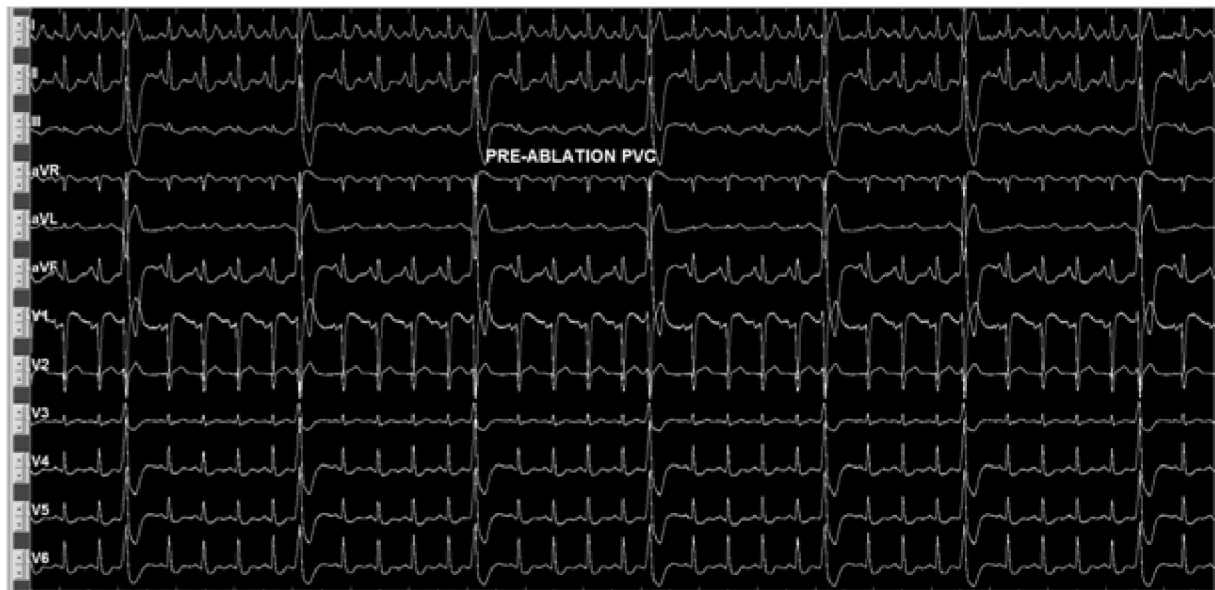


Fig. 1 : Baseline ECG showing frequent PVCs with inferior axis, QS in I/aVL/aVR, rS in V1 with transition in V3

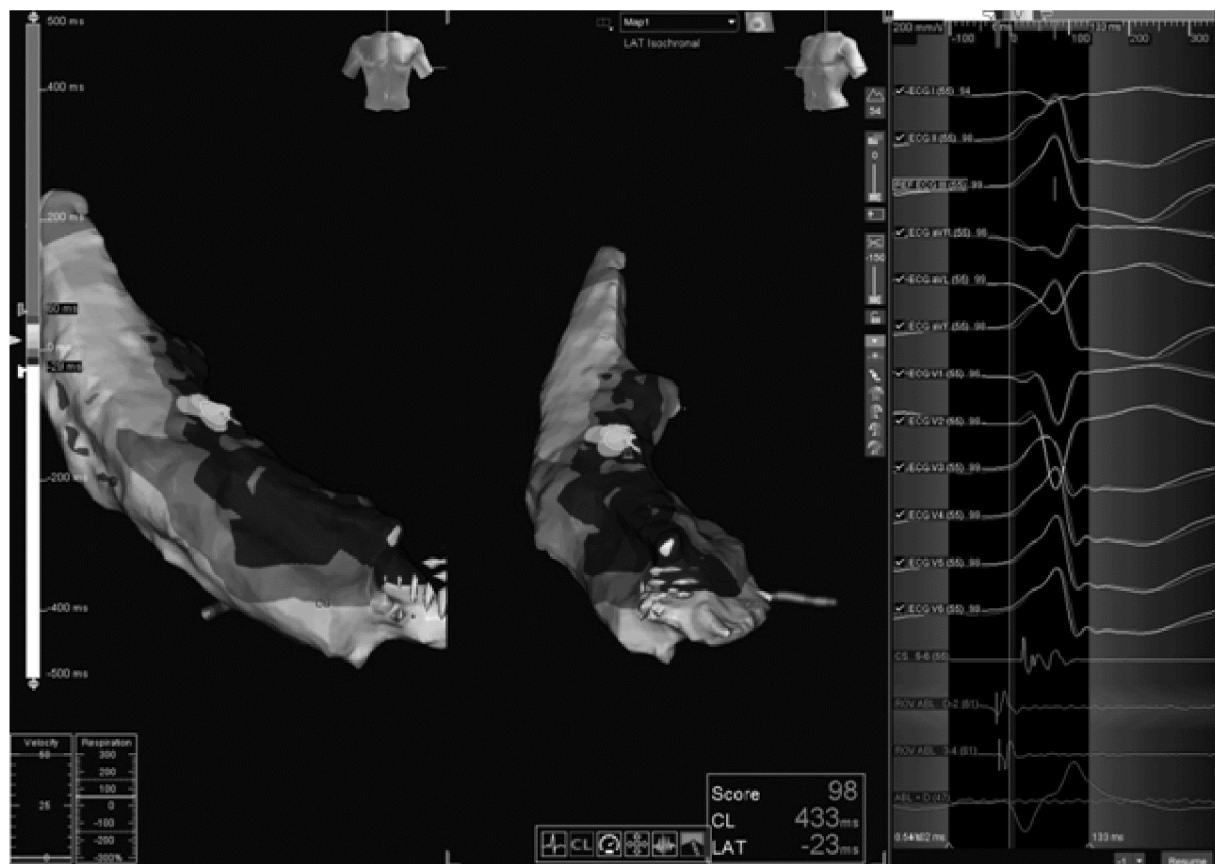


Fig. 2 : Activation map of LVOT showing early signals near left cusp and unipolar having QS configuration.



Fig. 3 : Left coronary injection to identify the proximity and safety of burn near left main ostium.

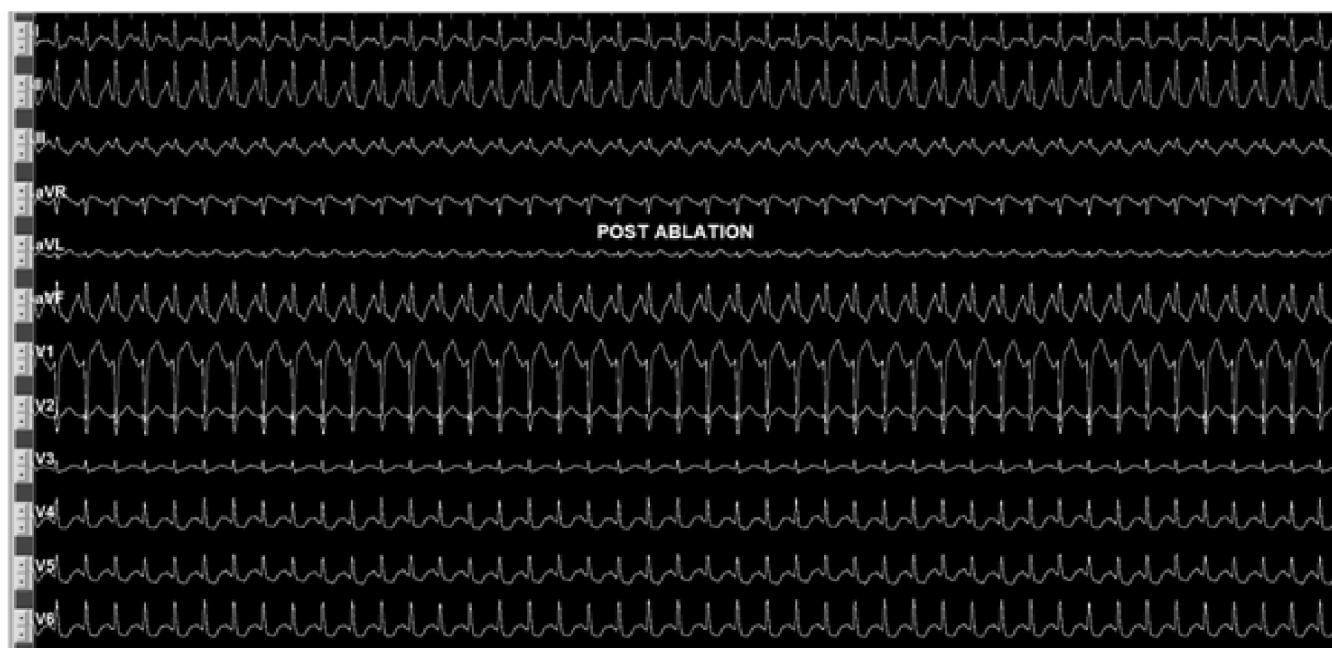


Fig. 4 : Post ablation ECG showing complete elimination of clinical PVCs.

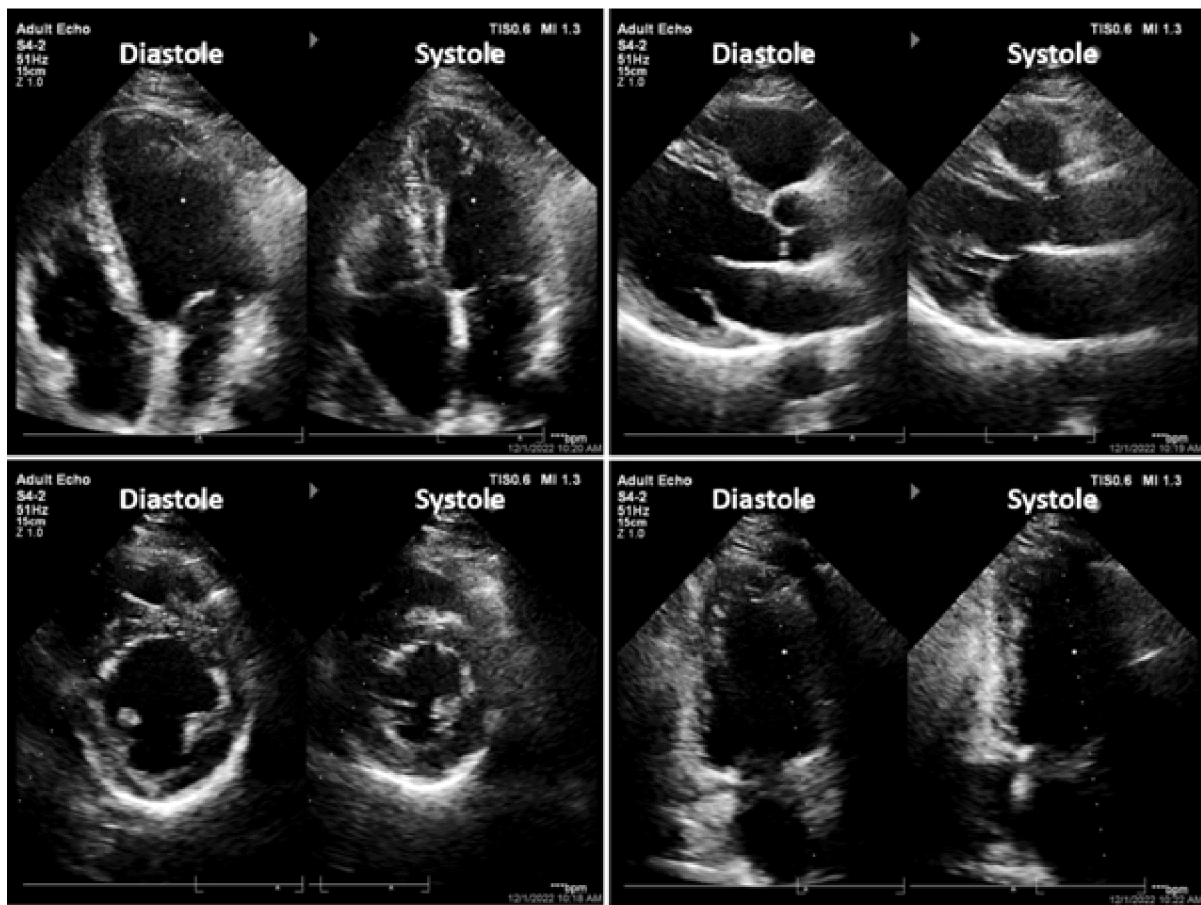


Fig. 5 : Follow up echocardiogram demonstrating normalization of left ventricular systolic function

### Discussion

PVCs are the commonest ventricular arrhythmia. In the absence of structural heart disease, they are often considered innocent bystander but in the presence of structural heart disease they have the potential to induce malignant ventricular arrhythmia. PVCs can occur by triggered automaticity or reentry. A stable coupling interval of the PVC with the preceding QRS that shortens with heightened sympathetic tone and prolongs with increased parasympathetic tone suggest triggered automaticity. A variable coupling interval may be seen in re-entrant PVCs<sup>1</sup>.

PVCs commonly arise from outflow tracts viz RVOT and LVOT, less commonly they can arise from HIS-Purkinje system, endocavitary structures like papillary muscle, moderator band, false tendons, annuli of aortic, pulmonary and atrioventricular valves. In structural heart disease PVCs can arise from areas of scar also.

Surface electrocardiogram (ECG) is the basic tool to identify and speculate the site of origin. 24-hour Holter is used to quantify PVC burden. Anti-arrhythmics are effective first line therapy for PVC suppression. Catheter ablation is needed if there is PVC induced cardiomyopathy, PVCs triggering ventricular fibrillation or symptomatic PVCs when medical therapy is ineffective or not tolerated<sup>1</sup>.

A high PVCs burden is independently associated with LV dysfunction<sup>2,3</sup>. The main reason of PVC-induced cardiomyopathy is LV dyssynchrony.

Ablation of outflow tract or fascicular PVCs is reportedly successful in 80% to 100% of cases<sup>4,5</sup>. In two-thirds of the patients undergoing PVC ablation due to PVC-mediated cardiomyopathy, LV function improves to normal within 4 months, although in some cases, it takes more than a year<sup>4-7</sup>. Hasdemir et al<sup>8</sup> reported that  $\geq 50\%$  of PVC-induced cardiomyopathy patients had  $\geq 25\%$  improvement in LVEF at 1 week after ablation.



### Conclusion

PVCs are generally considered innocent bystander and are only occasionally symptomatic. However, in certain cases may lead to congestive heart failure or sudden cardiac death. Majority of asymptomatic patients can be treated medically and in indicated patient can be cured by catheter ablation. Hence the risks and benefits of pharmacotherapy vs catheter ablation should be tailored according to the patient and PVC characteristics.

### References

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