Isolation of pulmonary vein and superior vena cava for paroxysmal atrial fibrillation in a young adult with left ventricular non-compaction

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We report a 19-year-old male patient with left ventricular non-compaction who presented with atrial fibrillation (AF) and ventricular tachycardia. Ventricular tachycardia was induced by AF with rapid ventricular response, but was prevented by electrical isolation of the pulmonary veins and superior vena cava.

![Figure 1](https://example.com/image1.png)

**Figure 1** Top: wide QRS tachycardia (200 bpm) induced by paroxysmal atrial fibrillation with rapid ventricular response. Bottom: discharges from the left superior pulmonary vein (LSPV) and superior vena cava (SVC) that initiated paroxysmal atrial fibrillation were observed with straight multipolar catheters placed in the SVC, LSPV, and left inferior pulmonary vein (LIPV). Discharges from the other three PVs were also recorded but are not included in the polygraph record.
Introduction
Left ventricular non-compaction (LVNC) is a rare congenital cardiomyopathy characterized by prominent trabeculations and deep intra-trabecular recesses in the left ventricular (LV) wall.\textsuperscript{1,2} The clinical presentation of LVNC includes heart failure, arrhythmias, and thromboembolic events.\textsuperscript{1,2} The diagnosis and management of life-threatening ventricular arrhythmia are particularly important because they correlate with prognosis. We report a case of ventricular tachycardia (VT) induced by atrial fibrillation (AF). Electrical isolation of the pulmonary veins (PVs) and superior vena cava (SVC) aborted these arrhythmias.

Case report
A 19-year-old man was admitted to our hospital for the management of a series of pre-syncopal attacks associated with AF and VT. The first episode of atrial tachycardia (AT) occurred at age 7, which was treated with oral propranolol, and the same treatment was continued for the next 12 years without recurrence of AT. On admission, a Holter recording identified frequent episodes of AF (maximum heart rate, 272 bpm) immediately followed by VT. Transthoracic echocardiography showed prominent trabeculations and deep intra-trabecular recesses in the LV wall. The LV end-diastolic diameter and fractional shortening were 60 mm and 23%, respectively. The left atrium was not dilated. Serum level of brain natriuretic peptide was 18.9 pg/mL.

Electrophysiological study showed no evidence of dual atrioventricular nodal pathway or atrioventricular accessory pathway. Maximal 1:1 atrioventricular conduction was observed at a rate of 272 bpm. Intravenous infusion of isoproterenol (2 \textmu g/min) elicited AF and AT followed immediately by VT due to rapid ventricular response (Figure 1), necessitating multiple direct-current shocks. The focal repetitive firing that initiated AF and AT originated from all four PVs and SVC (Figure 1). Electrical isolation was achieved by segmental antral radiofrequency ablation (single Lasso technique) applied to all four PVs and SVC. After the procedure, neither AF nor VT was induced by the maximal stimulation protocol even with isoproterenol. Repeated Holter monitoring revealed no tachyarrhythmia during the next 3 years of follow-up.

Discussion
Supraventricular tachycardia due to atrioventricular accessory pathway is one of the major complications in childhood LVNC.\textsuperscript{1} In contrast, the incidences of VT and AF increase with age in adult LVNC patients.\textsuperscript{2} One interesting finding in our patient was that AF-induced VT developed at a young age. Given the fact that AF in our patient was initiated by ectopic beats from the PVs and SVC, the mechanism of AF might be relatively common. However, considering the rarity of AF among young adults, the pathology of LVNC might be implicated even in atrial arrhythmogenicity. Considering that LV contraction was reduced, probably reflecting ventricular myocardial damage, progressive ischaemia and subsequent scar tissue in the non-compacted lesion could be potential arrhythmogenic substrates for VT.\textsuperscript{2} Enhanced atrioventricular conduction and rapid ventricular response might also play important roles in the development of VT, as described in a previous report in another structural heart disease.\textsuperscript{3} Unfortunately, the precise mechanism of VT could not be determined due to the unstable condition. Thus, the correlation between LVNC and the development of double tachycardia remains to be investigated. Our report highlights the importance of AF ablation by PVs (and SVC) isolation in preventing AF and hence VT episodes.

Conflict of interest: none declared.

References