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Case Report

# Incessant fascicular VT presenting as cardiogenic shock with multi-organ dysfunction syndrome

Ankur C. Thummar (MD, DM Cardiology)*, Ganesh Patil (MD), Ashar Khan (MD, DM Cardiology), Yash Y. Lokhandwala (MD, DM Cardiology), Ajay U. Mahajan (MD, DM Cardiology), Pratap J. Nathani (MD, DM Cardiology)

*Lokmanya Tilak Municipal Medical College & General Hospitals, Sion, Mumbai, India*

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ABSTRACT

We report a rare clinical presentation of incessant idiopathic fascicular ventricular tachycardia (FVT), presenting as multi-organ dysfunction (MOD) syndrome with cardiogenic shock. Our patient was a 19-year-old male who presented with slowly progressive dyspnea from New York Heart Association (NYHA) II to NYHA IV at the time of presentation, palpitations, and dilated cardiomyopathy due to drug-refractory FVT. The patient was in cardiogenic shock with raised central venous pressures and required inotropic support for maintaining systolic blood pressure above 90 mmHg. The MOD was seen in the form of deranged liver and kidney parameters. Echocardiography showed a dilated left ventricle (LV, 58 mm at end-diastole, 52 mm at end-systole) and decreased ejection fraction (20%). Electrocardiography showed a wide-QRS tachycardia (QRS 140 ms, cycle length 440 ms), with RsR' in lead V1 and a QRS axis of −60°. After stabilization with ventilation, inotropic support, and cautious use of diuretics, an electrophysiologic study was performed. A Purkinje potential with early local ventricular activation was recorded from the LV inferoseptal region. The tachycardia was ablated at this site with radiofrequency (RF) energy (40 W for 35 sec). Over a 3-month follow-up, the patient remained asymptomatic and the LV size and function returned to normal.
<Learning objective: To highlight a rare and unreported complication of fascicular ventricular tachycardia presenting as multi-organ dysfunction and its reversion with radiofrequency ablation.>


## Introduction

Most tachycardiomyopathies result from incessant atrial arrhythmias or atypical atrioventricular re-entry tachycardias. Incessant fascicular ventricular tachycardia (VT) leading to a tachycardia-mediated cardiomyopathy has been documented previously [1,2]. A clinical picture of congestive heart failure has been previously published in some children [3,4]. The extreme presentation as cardiogenic shock with multi-organ dysfunction (MOD) has not been reported to date.

## Case report

A 19-year-old male previously healthy, presented with a 2 month history of progressive dyspnea, from New York Heart Association (NYHA) II to NYHA IV at the time of presentation. He had felt frequent episodes of fast, regular palpitations, which were sometimes associated with presyncope, for which the patient had not taken any treatment. He presented with a history of a short febrile illness with upper respiratory tract infection 1 month prior to the onset of dyspnea. At presentation, he had a pulse rate of 136 beats/min; blood pressure, 80/60 mmHg; and respiratory rate, 28/min. There was a tender hepatomegaly and a raised jugular venous pressure. The extremities were cold and he was oliguric. On auscultation, a gallop rhythm at the apex and extensive bilateral fine crepitations were present. Chest X-ray demonstrated cardiomegaly with congested lung fields. The 12-lead electrocardiogram (Fig. 1) showed a wide-QRS tachycardia with QRS duration of 140 ms, cycle length 440 ms, with RsR' in lead V1 and a QRS axis of −60°. Echocardiography (Fig. 2) showed marked dilatation of the left ventricle (LV) and severely compromised LV contractility [LV ejection fraction (EF) 0.2]. The hepatic enzymes were massively elevated: aspartate transaminase 6420 U/L and alanine transaminase 7890 U/L. The serum creatinine was 2.5 mg/dL and troponin-T

* Corresponding author at: Department of Cardiology, Casualty Building, 2nd Floor, LTMMC & General Hospital, Sion, Mumbai 22, Maharashtra, India.
Tel.: +91 7498994924; fax: +91 02224076100/02224031202.
*E-mail address:* drankurt@gmail.com (A.C. Thummar).

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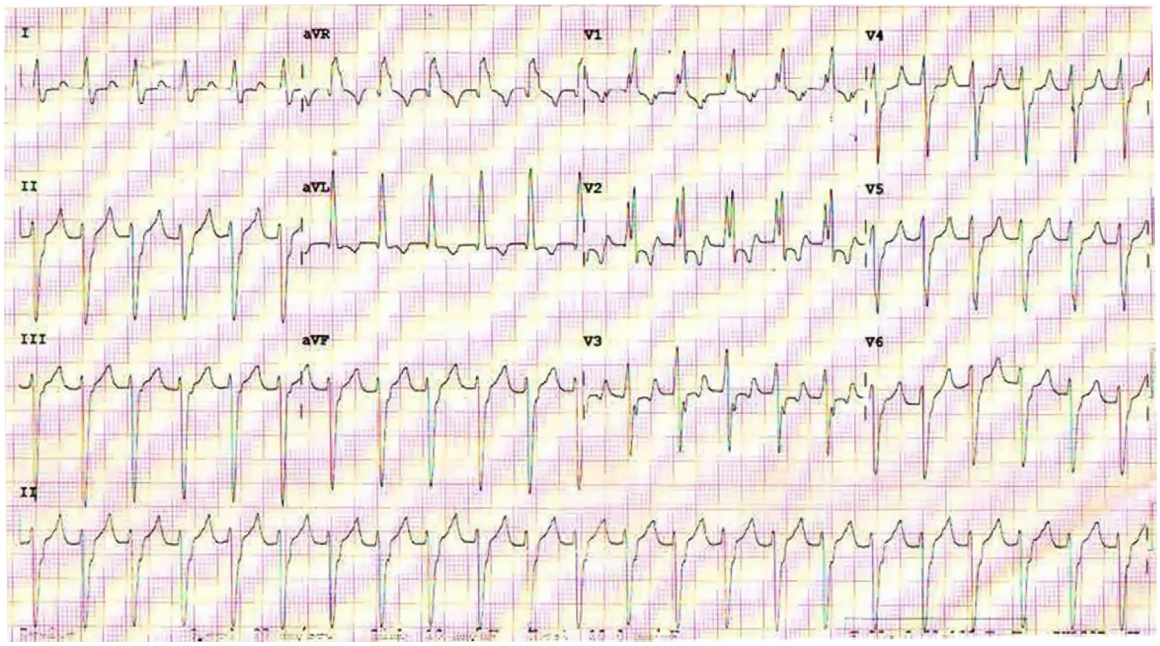


**Fig. 1.** Surface electrocardiogram of ventricular tachycardia showing right bundle branch block-like pattern with left-axis deviation. There were VA conduction with 1:1 VA relationship.

(0.11 μg/L) was also elevated. The human immunodeficiency virus, hepatitis B surface antigen, hepatitis C virus, dengue fever virus antibody immunoglobulin M, and influenza H1N1 tests were negative. Cardiac magnetic resonance imaging with contrast enhancement did not show delayed hyperenhancement, infiltration, or scar.

The patient was initially given 5 mg (0.1 mg/kg) verapamil as an intravenous bolus over 2 min. As VT was not terminated with the initial dose, after 15 min, another 5 mg intravenous bolus over 2 min was given. However, VT could not be terminated despite two boluses of intravenous verapamil. Hence, intravenous metoprolol and amiodarone were tried, but the tachycardia persisted. Multiple attempts of direct current cardioversions were also ineffective. The patient was started on non-invasive ventilation with inotropic support and cautious use of diuretics. After a duration of 48–72 h, his condition stabilized with the previously mentioned supportive measures. Subsequently, he was taken up for electrophysiologic studies and catheter ablation. There were 1:1 VA relationships; atrial pacing at a shorter cycle length was conducted with a normal QRS, proving the mechanism to be VT (Fig. 3). The VT mechanism was likely abnormal automaticity. This was evidenced by (i) the incessant nature and (ii) later on (even during the electrophysiologic study) the gradual spontaneous termination and reinitiation, *without initiating ectopy* (Fig. 4). The LV was mapped; a Purkinje potential with early local ventricular activation was recorded from the LV inferoseptal region. Radiofrequency energy at this site terminated the VT within 35 s. After this no VT was inducible, even with isoprenaline.

The follow-up echocardiography performed after 1 week of ablation showed a slight decrease in the LV dimension with an increase in the LVEF to 0.35. The patient was discharged on

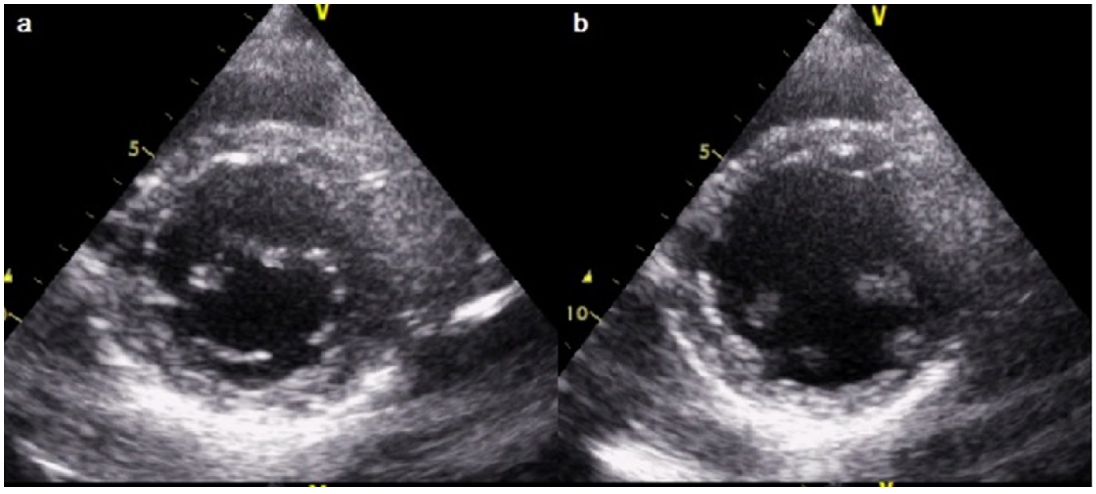


**Fig. 2.** 2D echocardiogram short-axis views at mid-cavity level showed a dilated left ventricle with poor contractility. (A) End-systolic frame. (B) End-diastolic frame.