Case Report

Nepalese Heart Journal 2025; Vol 22(2), 83-86

Refractory True Torsade de Pointes after Adalimumab Therapy in a Patient with Crohn's Disease Successfully Treated by Stellate Ganglion Block: A Case Report

Vijay Yadav[1], Ratna Mani Gajurel[1], Chandra Mani Poudel[1], Sanjeev Thapa[1], Bhawani Manandhar[1], Kundan Raj Pandey[1], Dhiraj Poudel[1], Ninadini Shrestha[2]

[1] Department of Cardiology, Manmohan Cardiothoracic Vascular and Transplant Center, Institute of Medicine, Tribhuvan University, Nepal
[2] Department of Anesthesiology, Maharajgunj Medical Campus, Institute of Medicine, Tribhuvan University, Nepal

Corresponding Author:
Vijay Yadav
MBBS, MD, DM Cardiology
Post-Doctoral Fellowship in Cardiac Electrophysiology and Pacing
Department of Cardiology, Manmohan Cardiothoracic Vascular and Transplant Center, Institute of Medicine, Tribhuvan University, Nepal
ORCID ID NO: 0000-0002-3837-3828
Email: vjmedicine451@gmail.com

Cite this article as: Yadav V, Gajurel RM, Poudel CM, Thapa S, Manandhar B, Pandey KR, Poudel D, Shrestha N. Refractory True Torsade de Pointes after Adalimumab Therapy in a Patient with Crohn's Disease Successfully Treated by Stellate Ganglion Block: A Case Report. Nepalese Heart Journal. 2025;22(2):83–86.

Submission Date: : 7 June, 2025
Acceptance date: 20 October, 2025

Submitted using Care checklist

Abstract

Several studies have reported the occurrence of new-onset cardiomyopathy and ventricular arrhythmias (VAs) after the administration of Adalimumab. We report the case of a 20-year-old male who presented himself with the features of dilated cardiomyopathy and a left ventricular ejection fraction of 10-15% after two weeks of administration of adalimumab. During the hospital stay, he developed multiple episodes of VAs (true torsade de pointes) in the form of electrical storm, which were refractory to both pharmacological and electrical interventions. He eventually underwent ultrasonographic guided left-sided stellate ganglion block with 6 mL of 0.2% bupivacaine and 40 mg of methylprednisolone injection. There was no recurrence of ventricular arrhythmias for 72 hours after the procedure.

Key words: Adalimumab, True Torsade de Pointes, Electrical storm, Stellate Ganglion Block

DOI: https://doi.org/10.3126/nhj.v22i2.85801

Introduction

Cardiotoxicity, leading to cardiomyopathy followed by tachyarrhythmias, is one of the life-threatening side effects of adalimumab, which is associated with poor quality of life and substantial mortality[1]. Stellate Ganglion Block (SGB) can reduce myocardial sympathetic tone that results in improved short-term control of ventricular arrhythmias and a reduced need for defibrillation. A successful SGB can increase arrhythmia free days, giving more time to stabilize the patient and allowing for therapy that is more definitive. We report the case of a 20-year-old male with adalimumab-induced cardiomyopathy who presented himself with refractory ventricular tachycardia, which was successfully managed with a left-sided stellate ganglion block.

Case Presentation

A 20-year-old male, a diagnosed case of Crohn's colitis for the last 5 months who had received a total of three doses of Adalimumab (80 mg s/c for consecutive 2 days followed by 80 mg s/c after 2 weeks) in another center, presented himself to the emergency room of the Manmohan Cardiothoracic Vascular and Transplant Center (MCVTC) with complaints of progressive dyspnea with orthopnea, palpitations, and easy fatigability for a week. His family history was negative for sudden cardiac death. His vital parameters were normal. Auscultation revealed fine crepitations over the bilateral lung field. His complete blood picture, renal and liver function tests were normal. N-Terminal Pro-Brain Natriuretic Peptide (NT–proBNP) was significantly elevated at 13,500 pg/ml.

The baseline 12-lead electrocardiogram (ECG) revealed normal sinus rhythm with a rate of 88 bpm, PR and QTc intervals of 140 ms and 484 ms, respectively. T wave inversion was present in the anterior precordial leads (Figure 1). A 2D echocardiogram (ECHO) demonstrated dilated all cardiac chambers with global hypokinesia of the left ventricular wall and severe left ventricular systolic dysfunction with an ejection fraction (LVEF) of 10–15% - findings consistent with dilated cardiomyopathy (DCM). His previous ECHO before the start of adalimumab revealed a normal LVEF of 65%. CT-CAG revealed normal course and calibers of major coronary arteries.

Upon admission, the patient was managed in a step-wise approach with intravenous furosemide and nitroglycerin, oral Ramipril, Spironolactone, Dapaglifozin, and other supportive measures. However, his condition deteriorated over the course of time. He developed a sudden drop in his Glasgow Coma Scale (E1V1M1) with an increase in oxygen requirement following a generalized tonic-clonic seizure. He subsequently suffered a pulseless electrical activity (PEA), requiring cardiopulmonary resuscitation (CPR) and defibrillation for pulseless ventricular tachycardia (VT), leading to intubation and mechanical ventilation, and was transferred to the Coronary Care Unit (CCU). During his CCU stay, the patient experienced recurrent episodes of pulseless VT and ventricular fibrillation (VF), necessitating further CPR, defibrillation, and antiarrhythmic therapy with intravenous (IV) Lidocaine infusion. Owing to such a VT storm, he was administered oral propranolol, IV magnesium sulphate, IV calcium gluconate, and adequate sedation. After gradual clinical stabilization, the patient was successfully extubated on the seventh day of ICU admission.

However, the patient developed recurrent episodes of ventricular tachycardia, requiring multiple electrical cardioversions at 150–200 Joules. We could obtain the VT ECG strips only from the attached cardiac monitor while he remained in the CCU. The ECG showed frequent ventricular extrasystoles in the form of bigeminy with coupling interval (CI) of more than 400 ms. The tachyarrhythmia started from a ventricular extrasystole with CI of 480 ms, falling in the middle of the T wave (R-on-T phenomenon), subsequently degenerating into polymorphic VT - likely mechanism being Phase 3 early-afterdepolarization (Figure 2). We labelled the tachyarrhythmia as True Torsade de Pointes (TdP) owing to both long QTc and long coupling interval (> 400 ms) – thus differentiating it from pseudo TdP.

Given the refractory nature of his ventricular arrhythmias, he underwent a left-sided stellate ganglion block. The patient was made to extend the neck to the right, and the USG-guided transverse short-axis view was made. In this view, the thyroid gland, the carotid artery, the compressible internal jugular vein, the prevertebral fascia (PVF), Chassaignac's tubercle (anterior tubercle of C6 vertebrae) and the oval shaped structure of the longus colli muscle were visualized (Figure 3-Left). Following which, a 25-gauge and 4 cm needle was inserted via the lateral paracarotid approach between the prevertebral fascia and the ventral surface of longus colli muscle (subfascial injection). Visualization of the needle tip as an echogenic dot on the screen was confirmed. The correct position of the needle was further confirmed by the expansion of local anesthetics beneath the prevertebral fascia and above the longus colli. Finally, 6 mL of 0.2% bupivacaine with 40 mg of methylprednisolone injection was given after confirmation of negative aspiration for blood (Figure 3-Right). The procedural success was assessed by the development of signs of ipsilateral Horner's syndrome. Following this intervention, the patient remained free of VT for the next 72 hours. He was discharged on guideline-directed medical therapy for heart failure with reduced ejection fraction and oral propranolol as a maintenance therapy for VT.

He is planned for Intracardiac Defibrillation (ICD) implantation on a later date after the patient and his family members' approval.

Discussion

We report a case of new-onset heart failure with refractory polymorphic VT (True TdP) in a 20-year-old boy who was recently initiated on Adalimumab for Crohn's colitis. Adalimumab is a fully human, recombinant immunoglobulin G anti-TNF-alpha with high affinity that is used to treat various autoimmune conditions, including Crohn disease[2]. It is postulated that the development of cardiomyopathy and worsening of heart failure in patients with anti-TNF-alpha (Adalimumab) is due to suppression of cardioprotective concentration of TNF-alpha, making cardiomyocytes susceptible to apoptosis, increased oxidative stress, and selective cytotoxicity to cardiac myocytes[3]. In our case, an ischemic cause for acute decompensated heart failure was ruled out by coronary angiogram. Other reversible causes of dilated cardiomyopathy (DCM), like infections, incessant tachycardia, thyrotoxicosis, and alcohol related causes, were ruled out too from the history and laboratory tests. Finally, we attributed the cause of his DCM to cytotoxic effects in the cardiac myocyte induced by adalimumab.

Complex changes in the electrical properties of ventricular cardiomyocytes occur in DCM that predispose to ventricular arrhythmias (VAs). Prolongation of action potential duration, a characteristic feature in DCM, is associated with a significant delay in repolarization that may increase the susceptibility to malignant VAs by mechanisms such as triggered activity or reentry[4]. In general, early afterdepolarization (EAD) occurs in association with action potential prolongation[5]. Therefore, the ventricular hypertrophy and heart failure can lead to EAD that can cause polymorphic VT in the form of Torsade de Pointes[6]. In our patient, the baseline QTc was 484 ms, and hence the mechanism of his VT was thought to be due to EAD.

Torsade de Pointes, first coined by Dessertenne[7], refers to a polymorphic VT that results from long QT. A coupling interval greater than 400ms in the background of prolonged QT is referred to as True TdP. If the QT is prolonged with a CL less than 400ms, it is termed as pseudo TdP[8]. In our case, the polymorphic VT started with an extra-systole with a coupling interval of 480 ms on the background of a slightly prolonged baseline QT interval. The arrhythmia was hence labelled as True TdP.

Although drug-refractory ventricular tachycardia (VT) is amenable to catheter ablation and surgical sympathectomy, neither of them may be immediately feasible in critically ill and hemodynamically unstable patients. An emerging treatment option is percutaneous stellate ganglion block (SGB), which creates temporary sympathetic interruption to treat refractory ventricular arrhythmias[9]. The stellate ganglion is a fusion of the inferior cervical and first thoracic sympathetic ganglions at the level of C7 transverse process. Right or left SGB are a specific type of peripheral nerve block that targets the sympathetic blockade of neuronal impulses using the injection of local anesthetic and steroids into nerve bundles in the cervical area[10]. The American Heart Association and American College of Cardiology (ACC/AHA) guidelines also recommend a class IIB indication for autonomic neuromodulation in patients with ventricular arrhythmia refractory to pharmacological management and for whom catheter ablation is not feasible[11]. A comprehensive meta-analysis of 22 case reports/series published between 1974 and 2017 identified only 35 patients treated with SGB for VT/VF. Left SGB was performed in 30 of these cases, and bilateral SGB in 5 cases. Either unilateral or bilateral SGB reduced the number of VT/VF episodes and internal and/or external defibrillation events for 24 h after blockade. The reduction in VT/VF after SGB was independent of the etiology of ventricular arrhythmia, with comparable reduction of VT/VF episodes and defibrillation events seen with ischemic and non-ischemic cardiomyopathy and with monomorphic and polymorphic VTs[12]. In 2019, Tian et al[13]. reported the use of SGB with bupivacaine alone or combined with

lidocaine in 30 patients with refractory ventricular arrhythmias, with half receiving left SGB and the other half receiving bilateral SGB. Around 60% of the patients were free from VT at 24 hours and the ICD interrogation showed 92% reduction in VA episodes 72 hours after SGB. Similarly, in 2020, Fudim et al[14] reported the use of bilateral SGB in 20 patients with refractory ventricular arrhythmias following which 45% of them had no recurrence of VT/VF for the next 48 hours, 20% had no recurrence for the rest of hospitalization, and 65% were ultimately discharged from the hospital. In yet another recently published multinational and multicenter study in 2024[15], it was shown that SGB was associated with a consistent reduction of treatment-refractory VT/VF and associated shocks at 24 and 48 hours after application. In our case too, there was no recurrence of VT with the requirement of shocks for the next 72 hours after SGB.

Conclusion:

This case report is one-of-a-kind in that the stellate ganglion block for drug-refractory VT was performed for the first time at our institute. We found out that the SGB was very effective in reducing VT/VF occurrence, thereby significantly reducing the need for defibrillation shocks and allowing the treating cardiologists to decide upon further definitive management.

Declaration of patient consent

The authors certify that they have obtained consent from the parent for publication of clinical information and the investigation report.

Acknowledgements:

None

Financial support and sponsorship:

Nil

Conflict of interest:

None declared

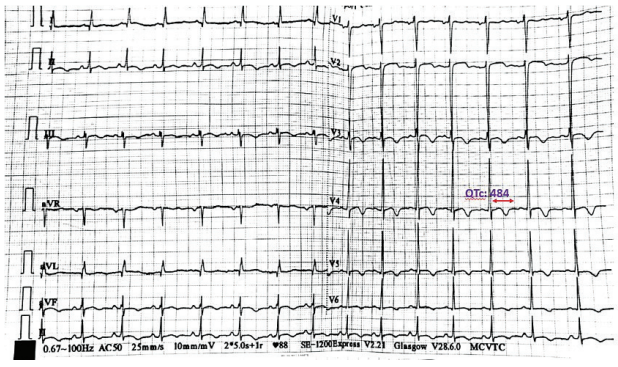

***Figure 1**. Baseline ECG with QTc of 484 msec*

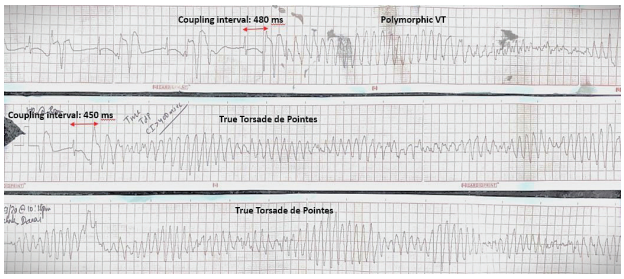

***Figure 2.** Long coupled VPCs (480/450 msec) initiating true torsade de pointes*

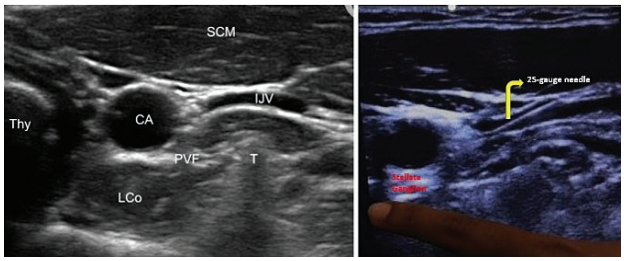

***Figure 3.** (Left) Transverse short-axis view of USG neck. (Right) A yellow arrow shows a 25-gauge 4 cm needle piercing the lateral aspect of neck. A finger points at the stellate ganglion area where it was blocked.*

Thy (Thyroid gland), SCM (Sternocleidomastoid muscle), IJV (Internal Jugular Vein), CA (Carotid Artery), PVF (Prevertebral Fascia), T (Chassaignac's Tubercle), LCo (Longus Colli muscle)

References :

1. Toufaily AJAC, Reports CC. Severe cardiomyopathy induced by adalimumab administration for crohn's disease. 2020;5:1035-6. https://doi.org/10.15761/JCCR.1000129
2. Vena GA, Cassano N. Drug focus: adalimumab in the treatment of moderate to severe psoriasis. Biologics : *targets & therapy.* 2007;1(2):93-103.
3. Grillo TG, Silveira C, Quaglio AEV, Dutra RM, Baima JP, Bazan SGZ, et al. Acute heart failure as an adverse event of tumor necrosis factor inhibitor therapy in inflammatory bowel disease: A review of the literature. *World journal of cardiology.* 2023;15(5):217-28. https://doi.org/10.4330/wjc.v15.i5.217
4. Mages C, Gampp H, Syren P, Rahm AK, André F, Frey N, et al. Electrical Ventricular Remodeling in Dilated Cardiomyopathy. *Cells.* 2021;10(10). https://doi.org/10.3390/cells10102767
5. Lerman BB, Markowitz SM, Cheung JW, Thomas G, Ip JE. Ventricular Tachycardia Due to Triggered Activity: Role of Early and Delayed Afterdepolarizations. *JACC Clinical electrophysiology.* 2024;10(2):379-401. https://doi.org/10.1016/j.jacep.2023.10.033
6. Roden DM. Drug-induced prolongation of the QT interval. *The New England journal of medicine.* 2004;350(10):1013-22. https://doi.org/10.1056/NEJMra032426
7. Dessertenne F, Coumel P, Fabiato A. [Ventricular fibrillation and twisted peaks]. *La Presse medicale*. 1969;77(6):193-6.
8. Rosso R, Hochstadt A, Viskin D, Chorin E, Schwartz AL, Tovia-Brodie O, et al. Polymorphic ventricular tachycardia, ischaemic ventricular fibrillation, and torsade de pointes: importance of the QT and the coupling interval in the differential diagnosis. *European heart journal.* 2021;42(38):3965-75. https://doi.org/10.1093/eurheartj/ehab138
9. Fudim M, Boortz-Marx R, Patel CB, Sun AY, Piccini JP. Autonomic Modulation for the Treatment of Ventricular Arrhythmias: Therapeutic Use of Percutaneous Stellate Ganglion Blocks. *Journal of cardiovascular electrophysiology.* 2017;28(4):446-9. https://doi.org/10.1111/jce.13152

10. Kirkpatrick K, Khan MH, Deng Y, Shah KB. A Review of Stellate Ganglion Block as an Adjunctive Treatment Modality. *Cureus.* 2023;15(2):e35174. https://doi.org/10.7759/cureus.35174

11. Al-Khatib SM, Stevenson WG, Ackerman MJ, Bryant WJ, Callans DJ, Curtis AB, et al. 2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Journal of the American College of Cardiology.* 2018;72(14):e91-e220.

12. Fudim M, Boortz-Marx R, Ganesh A, Waldron NH, Qadri YJ, Patel CB, et al. Stellate ganglion blockade for the treatment of refractory ventricular arrhythmias: A systematic review and meta-analysis. *Journal of cardiovascular electrophysiology*. 2017;28(12):1460-7. https://doi.org/10.1111/jce.13324

13. Tian Y, Wittwer ED, Kapa S, McLeod CJ, Xiao P, Noseworthy PA, et al. Effective Use of Percutaneous Stellate Ganglion Blockade in Patients With Electrical Storm. *Circulation Arrhythmia and electrophysiology.* 2019;12(9):e007118. https://doi.org/10.1161/CIRCEP.118.007118

14. Fudim M, Qadri YJ, Waldron NH, Boortz-Marx RL, Ganesh A, Patel CB, et al. Stellate Ganglion Blockade for the Treatment of Refractory Ventricular Arrhythmias. *JACC Clinical electrophysiology.* 2020;6(5):562-71. https://doi.org/10.1016/j.jacep.2019.12.017

15. Chouairi F, Rajkumar K, Benak A, Qadri Y, Piccini JP, Mathew J, et al. A Multicenter Study of Stellate Ganglion Block as a Temporizing Treatment for Refractory Ventricular Arrhythmias. *JACC Clinical electrophysiology.* 2024;10(4):750-8. https://doi.org/10.1016/j.jacep.2023.12.012