Cureus
Part of SPRINGER NATURE

Open Access Case Report

Blocking the Sympathetic Storm: Adjunctive Left Stellate Ganglion Block for Refractory Ventricular Tachycardia After Inferolateral Myocardial Infarction

Review began 10/05/2026
Review ended 10/13/2026
Published 10/18/2026

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DOI: 10.7759/cureus.118126

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Abstract

Electrical storm following myocardial infarction (MI) can be difficult to control when antiarrhythmic drugs and cardioversion fail. We describe a 53-year-old man with hypertension and previously undiagnosed diabetes who presented with an inferolateral ST-segment elevation MI caused by total occlusion of the proximal left circumflex artery, which was treated with primary percutaneous coronary intervention and drug-eluting stent placement. He then developed marked leukocytosis with a systemic inflammatory response and acute kidney injury (peak creatinine, 4.03 mg/dL), which resolved with empirical antibiotic therapy and supportive renal care. On day 6, he experienced recurrent syncope associated with sustained monomorphic ventricular tachycardia (VT) that persisted despite intravenous amiodarone and lidocaine, electrolyte correction, and repeated cardioversion, necessitating sedation and mechanical ventilation. Repeat coronary angiography demonstrated a patent stent. VT ceased following an ultrasound-guided left stellate ganglion block (SGB) using 1% lidocaine and dexamethasone, which resulted in Horner syndrome. Antiarrhythmic therapy was continued, and the patient was extubated within 24 hours. Speckle-tracking echocardiography demonstrated severely reduced lateral and anterior strain (global longitudinal strain, -12.0%). A patent stent in the culprit vessel and sustained monomorphic arrhythmia made a scar-related substrate the leading consideration. The close temporal association between the block and cessation of VT suggests that sympathetic blockade may have contributed to arrhythmia control, although the continuation of antiarrhythmic therapy precludes firm causal conclusions. Left SGB may serve as a rapid bridge to definitive treatment in patients with refractory electrical storm.

Categories: Cardiology, Internal Medicine, Anesthesiology
Keywords: acute kidney injury, autonomic modulation, electrical storm, mechanical dispersion, myocardial infarction, percutaneous coronary intervention, speckle-tracking echocardiography, stellate ganglion block, ventricular tachycardia

Introduction

Electrical storm is defined as three or more episodes of sustained ventricular arrhythmia within 24 hours [1]. After myocardial infarction (MI), it carries a high in-hospital mortality, particularly when episodes recur despite antiarrhythmic drugs, beta-blockade, electrolyte correction, and deep sedation [1]. Heightened sympathetic tone both triggers ventricular tachycardia (VT) and makes it harder to suppress [2]. Much of the efferent sympathetic supply to the ventricles passes through the stellate ganglia, so injecting local anesthetic around the left stellate ganglion produces temporary, reversible cardiac sympathetic blockade that can be performed at the bedside. For drug-refractory monomorphic VT storm, current guidelines recommend catheter ablation and suggest that autonomic modulation may be considered when ablation is ineffective or not possible [1]. Percutaneous stellate ganglion block (SGB) has therefore been used mainly as rescue or bridging therapy rather than as a substitute for definitive treatment [2-5]. We report a patient in whom monomorphic VT storm developed four days after primary percutaneous coronary intervention (PCI) for an occluded left circumflex artery (LCx), despite a patent stent, while he was recovering from a systemic inflammatory illness with acute kidney injury (AKI). VT ceased after left SGB with short-acting lidocaine while antiarrhythmic therapy continued. The case illustrates the bedside distinction between stent failure and a scar-related mechanism, and the use of SGB as a bridge when conventional therapy has failed.

Case Presentation

A 53-year-old man with a two- to three-year history of hypertension presented to the emergency department in the evening with chest pain that had begun earlier that day. He had no known drug allergies. Point-of-care testing showed a creatine kinase-MB level above 80 ng/mL, a troponin I level of 9.65 ng/mL, and a B-type natriuretic peptide (BNP) level of 216 pg/mL. The electrocardiogram (ECG) showed ST-segment elevation in leads II, III, aVF, and V6, with ST-segment depression in V1 and tall T waves in V2-V3, consistent with an inferolateral infarction extending to the posterior wall (Figure *1A*). By hospital day 2, Q

waves had developed in the inferior leads, accompanied by ST-segment elevation and T-wave inversion in leads II, III, aVF, I, aVL, and V6 (Figure *1B*).

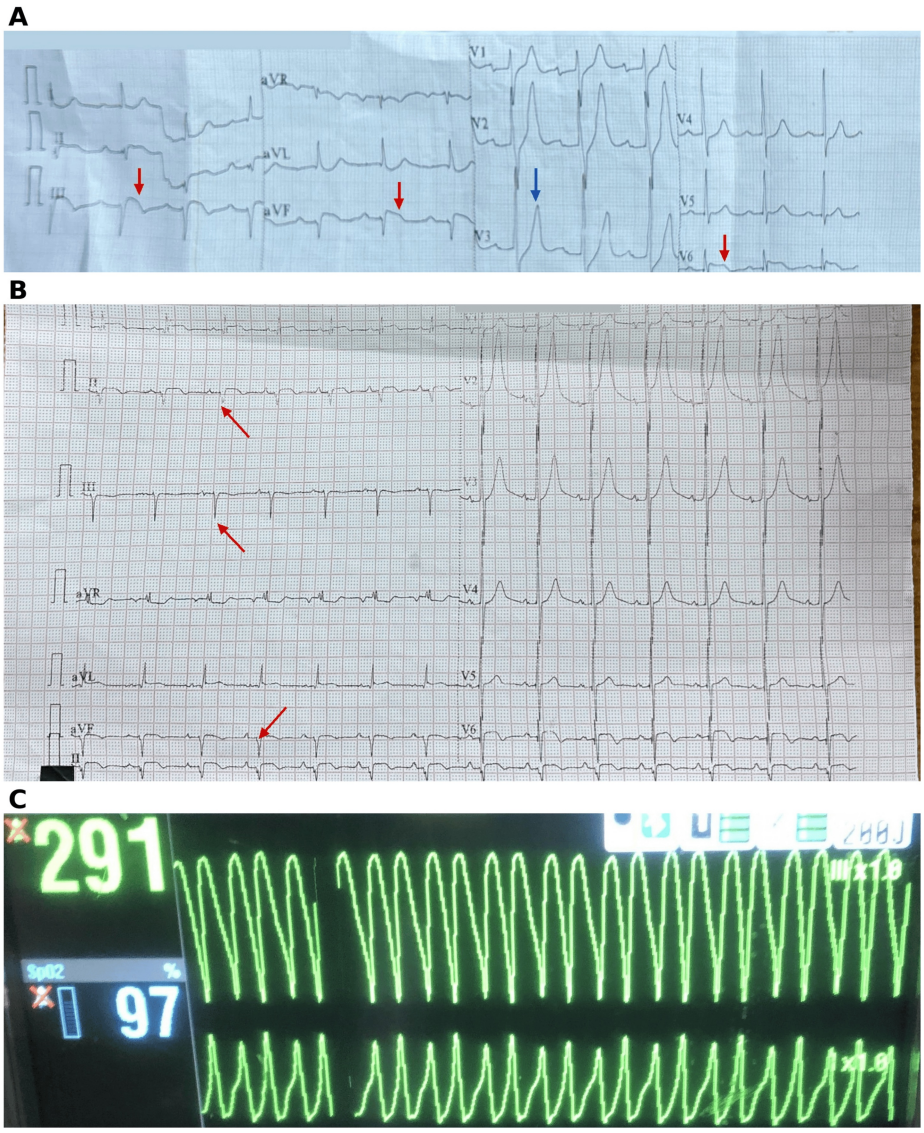


FIGURE 1: ECGs

(A) Presenting ECG showing ST-segment elevation in leads II, III, aVF, and V6 (red arrows in leads III, aVF, and V6), with ST-segment depression in V1 and tall T waves in V2-V3 (blue arrow in V3), consistent with an inferolateral infarction extending to the posterior wall. (B) Day 2, before PCI, showing inferior Q waves (red arrows in leads II, III, and aVF), with ST-segment elevation and T-wave inversion in leads II, III, aVF, I, aVL, and V6. (C) Defibrillator monitor recording during the VT storm, showing regular monomorphic wide-complex tachycardia at a displayed rate of 291/min in two monitoring leads (upper trace, lead III), with 200 J selected for cardioversion. Identifying information has been removed.

VT, ventricular tachycardia; PCI, percutaneous coronary intervention; ECG, electrocardiogram

Blood tests at presentation showed a leukocyte count of 12.49×10³/μL, with 84% neutrophils; hemoglobin of 14.6 g/dL; platelet count of 137×10³/μL; creatinine of 0.96 mg/dL; and potassium of 4.77 mmol/L. Glycated hemoglobin was 9.1%, indicating previously undiagnosed diabetes mellitus. Tests for HIV, hepatitis B surface antigen, and hepatitis C antibodies were negative. Echocardiography before catheterization showed a left ventricular ejection fraction (LVEF) of approximately 50% by visual estimation, hypokinesia of the posterior and lateral walls, and mild mitral regurgitation.

Coronary angiography on the morning of hospital day 2 showed total occlusion of the proximal LCx, judged to be the culprit lesion, with moderate non-culprit disease: 60% stenosis of the mid left anterior descending artery (LAD) and 50%-60% stenosis of the proximal first diagonal branch (Figure *2*). Primary PCI of the LCx was performed that afternoon through the right femoral artery under local anesthesia, with 7,000 IU of

unfractionated heparin and activated clotting time monitoring. Using a 6-Fr XB 3.5 guide catheter, the occlusion was crossed with a Pilot 50 guidewire (Figure *3A*) and predilated with a non-compliant balloon at 12 atm. A 2.5 mm × 28 mm drug-eluting stent was deployed at 14 atm (Figure *3B*) and postdilated with a 2.75 mm × 12 mm non-compliant balloon at 16 atm, restoring thrombolysis in myocardial infarction (TIMI) grade 3 flow (Figures *3C*, *3D*). A tirofiban infusion was administered after the procedure, and treatment with aspirin, ticagrelor, rosuvastatin 40 mg, and metoprolol was initiated. The moderate mid-LAD stenosis was managed medically, with further assessment planned at follow-up.

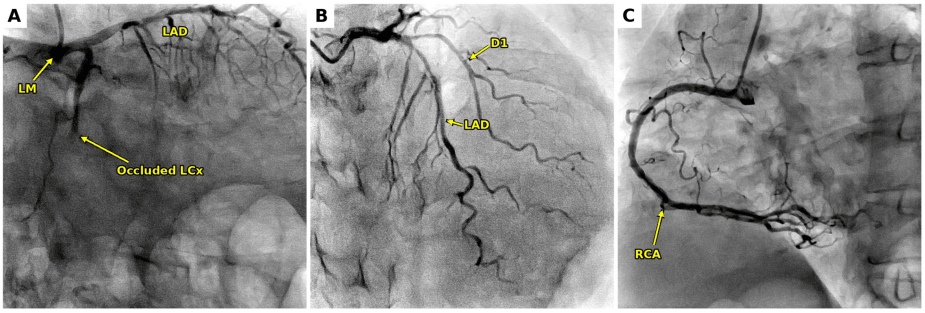


FIGURE 2: Diagnostic coronary angiography

(A) Right anterior oblique 41°, caudal 35°: LM and LAD, with the proximal LCx ending abruptly (arrow), consistent with total occlusion of the culprit vessel. (B) Anteroposterior, cranial 24°: LAD and first diagonal branch (D1); the moderate mid-LAD (60%) and proximal D1 (50%-60%) stenoses were managed medically. (C) Left anterior oblique 37°, cranial 26°: RCA.

LM, left main; LAD, left anterior descending artery; LCx, left circumflex artery; RCA, right coronary artery

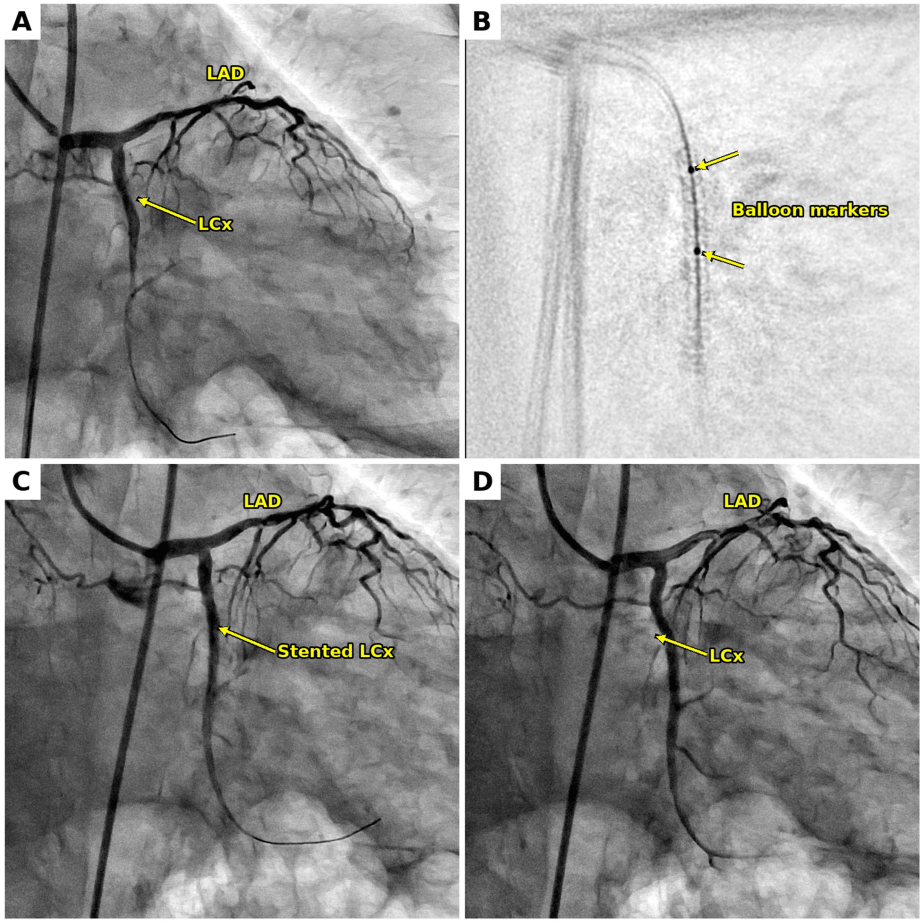


FIGURE 3: Primary PCI of the LCx

(A) Working projection (right anterior oblique 12°, caudal 24°) after the guidewire crossed the occluded proximal LCx (arrow); the distal vessel is small and poorly opacified. (B) Stent-enhancement image in the same projection; arrows indicate the radiopaque balloon markers in the proximal LCx. (C) Same projection after stenting, showing a widely patent stented LCx (arrow) with TIMI grade 3 flow. (D) Final angiogram (left anterior oblique 6°, caudal 11°) after guidewire removal (arrow, LCx). Identifying information embedded in the original images has been cropped.

LAD, left anterior descending artery; LCx, left circumflex artery; TIMI, thrombolysis in myocardial infarction; PCI, percutaneous coronary intervention

Leukocytosis with neutrophilia was already present at presentation, before angiography. By the evening of the procedure, the patient had developed a marked systemic inflammatory response: his leukocyte count had risen to 23.51×10^3/μL (82% neutrophils), C-reactive protein (CRP) was 272.6 mg/L, and serum sodium had fallen to 129 mmol/L. He also had a cough. Creatinine rose from 0.96 mg/dL to a peak of 4.03 mg/dL on day 4. The AKI occurred after contrast exposure in the setting of a marked systemic inflammatory response and was considered multifactorial [6]. He was treated empirically with meropenem and teicoplanin at renally adjusted doses, intravenous N-acetylcysteine with fluids, oral sodium bicarbonate, and furosemide guided by volume status. Nephrotoxic drugs were avoided. Creatinine fell to 0.84 mg/dL by day 6, whereas CRP declined more slowly, to 216.96 mg/L on day 6 and 142.14 mg/L on day 7. Procalcitonin on day 7 was 2.10 ng/mL. The laboratory course is summarized in Table *1*.

Parameter	Presentation	Day 2 (after PCI)	Day 4	Day 6 (VT storm)	Day 7
Creatinine (mg/dL)	0.96	1.1	4.03	0.84	0.91
Leukocytes (×10³/µL)	12.49	23.51	13.83	10.16	-
CRP (mg/L)	-	272.63	-	216.96	142.14
Procalcitonin (ng/mL)	-	-	-	-	2.1
BNP (pg/mL)	216	-	-	1020	-
Sodium (mmol/L)	137.2	129	133	133.4	136.3
Potassium (mmol/L)	4.77	3.93	5.22	3.62	3.86
Magnesium (mg/dL)	-	-	-	-	2.25

TABLE 1: Laboratory course

PCI: percutaneous coronary intervention; VT: ventricular tachycardia; CRP: C-reactive protein; BNP: B-type natriuretic peptide

On day 6, the patient experienced recurrent syncope caused by sustained monomorphic VT. The defibrillator monitor recorded a regular wide-complex tachycardia at 291/min (Figure *1C*), and episodes recurred in rapid succession, meeting the definition of electrical storm. A 12-lead ECG during VT was not available. By then, creatinine had returned to 0.84 mg/dL, but CRP remained 216.96 mg/L, potassium was 3.62 mmol/L, and BNP had risen to 1,020 pg/mL. Treatment consisted of intravenous lidocaine (100 mg bolus, followed by 2 mg/min), repeated 150 mg boluses of intravenous amiodarone followed by an infusion, magnesium sulfate, calcium gluconate, and oral mexiletine 150 mg twice daily, together with repeated synchronized cardioversion. Tirofiban and nicorandil infusions were restarted until repeat angiography could be performed. VT continued to recur at short intervals, so the patient was sedated with midazolam and fentanyl and mechanically ventilated. Repeat angiography on day 7 showed a widely patent LCx stent with TIMI grade 3 flow, excluding stent thrombosis. Between episodes, the ECG showed sinus rhythm with QT prolongation during amiodarone therapy (corrected QT interval up to 489 ms). Electrolytes were monitored closely, and serum magnesium on day 7 was 2.25 mg/dL.

Because antiarrhythmic drugs and repeated cardioversion had failed and the stent was patent, the anesthesia team was consulted for cardiac sympathetic blockade. Immediately before the block, the patient was sedated and mechanically ventilated, and VT continued to recur despite the antiarrhythmic regimen described above. On day 8, a left SGB was performed under ultrasound guidance. A 5 cm Stimuplex needle was advanced just below the carotid artery to the plane superficial to the longus colli muscle, and 6 mL of 1% lidocaine containing 4 mg of dexamethasone was injected. No complications occurred, and the patient remained hemodynamically stable during the procedure. Left-sided Horner syndrome confirmed the effectiveness of the block. VT ceased following the block and did not recur during the remainder of the hospital stay. Amiodarone, mexiletine, and metoprolol were continued. Sedation was withdrawn, and the patient was extubated within 24 hours.

By day 10, the patient was hemodynamically stable (blood pressure, 124/80 mmHg; heart rate, 90/min; oxygen saturation, 98% on room air) and free of arrhythmia, with no further inflammatory or renal complications. He was receiving aspirin, ticagrelor, rosuvastatin, metoprolol 25 mg twice daily, amiodarone 200 mg twice daily, and mexiletine 150 mg twice daily. A secondary-prevention implantable cardioverter-defibrillator (ICD) was advised but could not be implanted during the index admission because of financial constraints; implantation is planned. Electrophysiology review, including consideration of catheter ablation, and reassessment of the LAD stenosis were also planned at follow-up.

Echocardiography on day 11 showed an LVEF of 40% by the single-plane Simpson method and 44% by speckle tracking, lower than the visual estimate of about 50% before PCI, with a global longitudinal strain of -12.0% (Figure *4*). Strain was relatively preserved in the septal, anteroseptal, and inferior segments but was severely reduced or paradoxical (positive) in the anterolateral and basal anterior segments, with abnormality centered on the lateral wall supplied by the LCx. Peak strain dispersion, which reflects heterogeneity in the timing of regional contraction, was 79 ms. Key events are summarized in Table *2*.

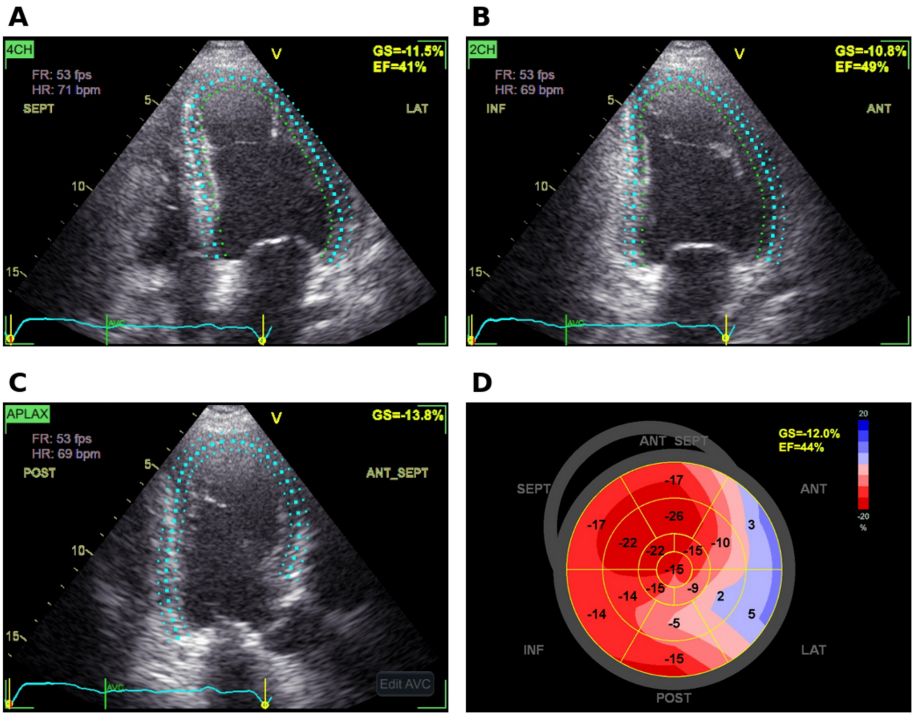


FIGURE 4: Speckle-tracking echocardiography

(A) Apical four-chamber view (longitudinal strain: -11.5%). (B) Apical two-chamber view (-10.8%). (C) Apical long-axis view (-13.8%). (D) Bullseye map (global longitudinal strain -12.0%, LVEF 44%). Strain is relatively preserved in the septal, anteroseptal, and inferior segments and severely reduced or paradoxical (positive values, blue) in the anterolateral and basal anterior segments, an abnormality centered on the lateral wall supplied by the occluded LCx.

LVEF, left ventricular ejection fraction; LCx, left circumflex artery

Time point	Events
Presentation (evening)	Chest pain since earlier the same day; inferolateral ST elevation; troponin I 9.65 ng/mL; glycated hemoglobin 9.1%
Day 1	Admitted to the coronary care unit; LVEF about 50% with posterior and lateral hypokinesia
Day 2	Angiography: proximal LCx occlusion (culprit), mid-LAD 60%, proximal first diagonal 50%-60%; primary PCI to the LCx (2.5 × 28 mm drug-eluting stent), TIMI grade 3 flow; evening leukocytes 23.51×10³/μL, CRP 272.6 mg/L
Day 4	AKI, creatinine 4.03 mg/dL; empirical antibiotics and renal supportive care
Day 6	Recurrent syncope from sustained monomorphic VT (electrical storm); repeated synchronized cardioversion; intravenous lidocaine and amiodarone, magnesium sulfate, calcium gluconate, oral mexiletine; metoprolol continued; BNP 1020 pg/mL, potassium 3.62 mmol/L
Day 7	VT still recurring at short intervals; sedation (midazolam, fentanyl) and mechanical ventilation; check angiography: patent stent, TIMI grade 3 flow
Day 8	Before block: sedated, ventilated, VT recurring despite antiarrhythmic therapy. Ultrasound-guided left SGB (6 mL of 1% lidocaine with dexamethasone 4 mg); Horner syndrome; no complications. After block: VT ceased; antiarrhythmic drugs continued
Day 9	No VT; sedation withdrawn; extubated within 24 hours of the block
Day 10	No VT; hemodynamically stable on room air; secondary-prevention ICD advised
Day 11	Echocardiography: LVEF 40%-44%, global longitudinal strain -12.0%

TABLE 2: Timeline of clinical events, treatment, and arrhythmia response

LVEF: left ventricular ejection fraction; LCx: left circumflex artery; LAD: left anterior descending artery; PCI: percutaneous coronary intervention; TIMI: thrombolysis in myocardial infarction; CRP: C-reactive protein; AKI: acute kidney injury; VT: ventricular tachycardia; BNP: B-type natriuretic peptide; SGB: stellate ganglion block; ICD: implantable cardioverter-defibrillator

Discussion

VT storm began on day 6, after the inflammatory and renal complications had peaked. The key question at the bedside was whether the arrhythmia reflected recurrent ischemia due to stent failure, which would have required urgent repeat PCI, or a scar-related mechanism. Several observations favored the latter. The VT was sustained and monomorphic; it began several days after reperfusion rather than within the first 48 hours, when ventricular arrhythmias are usually attributed to acute ischemia, and repeat angiography showed a widely patent stent with TIMI grade 3 flow [1]. Strain imaging also showed regional mechanical dysfunction centered on the lateral wall, the territory of the occluded LCx; impaired strain in the basal anterior segment may reflect overlap with the diagonal territory or the LAD stenosis. A scar-related substrate was therefore considered likely. This remains an inference: a contribution from the moderate non-culprit LAD stenosis could not be completely excluded, and without a 12-lead ECG during VT, cardiac magnetic resonance imaging, or electrophysiological mapping, a specific re-entry circuit could not be demonstrated.

Speckle-tracking echocardiography also provided a measure of arrhythmic risk. Mechanical dispersion, the variation in time to peak strain across segments, was greater in post-infarction patients with documented ventricular arrhythmias than in those without, and in a prospective multicenter study, it predicted arrhythmic events independently of LVEF [7,8]. The peak strain dispersion of 79 ms in this patient is close to the value reported in post-infarction patients with recurrent arrhythmias (85±29 ms) [7]. Strain imaging cannot identify scar or localize a VT circuit in the same way as late gadolinium enhancement imaging or electroanatomical mapping. However, it demonstrates regional mechanical dysfunction corresponding to the infarct territory and may provide supportive evidence of the structural substrate when those modalities are unavailable.

Left SGB interrupts efferent sympathetic outflow to the heart and may reduce sympathetically driven arrhythmia triggers and dispersion of repolarization in the infarct border zone. In an early study of 49 patients with electrical storm after acute MI, sympathetic blockade, including left SGB, was associated with lower mortality than antiarrhythmic therapy guided by advanced cardiac life support [9]. Systematic reviews of published case series found significant reductions in ventricular arrhythmia burden and shocks after SGB [2,10]. In one of these reviews, acute MI was the trigger in 15 of 38 patients [10]. Single-center and multicenter series have since reported fewer arrhythmia episodes and defibrillations after the procedure [3,4]. In a prospective multicenter study of 131 patients with refractory electrical storm, percutaneous SGB reduced treated arrhythmic events by at least half within 12 hours in 92% of patients [5]. These studies

report group-level reductions in arrhythmia burden; a single case cannot establish efficacy, and our patient's response should be interpreted as an individual clinical course rather than evidence that SGB works. The 2022 European Society of Cardiology guidelines recommend catheter ablation for monomorphic VT storm that does not respond to antiarrhythmic drugs and state that autonomic modulation may be considered when ablation is ineffective or not possible (class IIb) [1]. In our ventilated patient, SGB could be performed immediately at the bedside, and Horner syndrome provided a simple clinical indication of a successful block. VT ceased following SGB after conventional antiarrhythmic and electrical therapies had failed. The close temporal association suggests a clinically important contribution from sympathetic blockade, although causality cannot be established because amiodarone, mexiletine, and beta-blockade were continued, the patient remained sedated, and his systemic illness was resolving. Most published series used long-acting agents such as bupivacaine or ropivacaine [4,10]. The persistence of arrhythmia control beyond the expected duration of action of lidocaine in our patient is compatible with interruption of a self-perpetuating cycle of sympathetic activation but may also reflect the cumulative effects of concomitant therapy.

The systemic illness may have lowered the arrhythmic threshold, but we regard it as a possible contributor rather than the cause of the storm. Renal function had recovered by the time VT began, whereas the inflammatory response persisted (CRP, 216.96 mg/L; procalcitonin, 2.10 ng/mL), and potassium was in the low-normal range. Systemic inflammation can increase circulating catecholamine levels; these factors may promote ventricular arrhythmias and complicate antiarrhythmic drug dosing. However, VT ceased on day 8 while inflammatory markers remained elevated (CRP, 142.14 mg/L on day 7), so the inflammatory state alone does not explain the timing of the storm. The inflammatory illness and electrolyte disturbances were therefore managed alongside the arrhythmia rather than regarded as its sole cause.

This report has several limitations. Only a two-lead monitor recording of the VT was available, so the exit site could not be localized from QRS morphology. Neither electrophysiological mapping nor cardiac magnetic resonance imaging was performed; therefore, the proposed scar-related mechanism remains unconfirmed. The case notes record repeated cardioversions but not the exact number of VT episodes or shocks, so the arrhythmia burden before and after each intervention could not be quantified. Because VT ceased while antiarrhythmic drugs, beta-blockade, and sedation were continued, the contribution of SGB cannot be separated from that of concomitant therapy. The cause of the systemic inflammatory response was not established, and the patient was treated empirically with broad-spectrum antibiotics. Finally, follow-up is limited to the index admission. A secondary-prevention ICD has been advised but not yet implanted, and longer-term follow-up, including evaluation for catheter ablation, is needed to determine whether arrhythmia control is durable.

Conclusions

In this patient, refractory monomorphic VT storm after primary PCI ceased following left SGB, administered alongside continued antiarrhythmic therapy. Because this is a single case involving concurrent treatments, the response cannot be attributed to the block alone and should not be generalized. In selected patients with refractory electrical storm, left SGB may be considered an adjunctive treatment and a temporary bridge when conventional therapy has failed, but not a substitute for definitive management, such as catheter ablation or an ICD when indicated. When the culprit stent is patent, and the VT is monomorphic, a scar-related substrate is a reasonable working hypothesis, although residual ischemia due to non-culprit disease should not be dismissed. Strain imaging may provide supportive, but not confirmatory, evidence of such a substrate when cardiac magnetic resonance imaging or electroanatomical mapping is unavailable.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Mohit Goyal, Shriya Razdan, Om Shankar

Acquisition, analysis, or interpretation of data: Mohit Goyal, Shriya Razdan, Soumik Ghosh, Rajpal Prajapati

Drafting of the manuscript: Mohit Goyal, Shriya Razdan

Critical review of the manuscript for important intellectual content: Mohit Goyal, Soumik Ghosh, Rajpal Prajapati, Om Shankar

Supervision: Soumik Ghosh

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Acknowledgements

The authors thank the Department of Anesthesiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi, India, for performing the stellate ganglion block. De-identified clinical data and images are available from the corresponding author upon reasonable request.

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