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Samia Ejjebli *

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Case Report

# Cardiac Storm, Sepsis from Inoperable Osteitis, and Thromboembolic Complications in a Patient with Suspected Non compaction Cardiomyopathy: A Clinical Deadlock

**Samia Ejjebli *, Anas Elmahi, Alaa Altimimi, Salim Arous, Mohammed Ghali Bennouna, Abdennaser Drighil, Rachida Habbal**

Department of Cardiology, UHC Ibn Rochd, Casablanca, Morocco.

***Corresponding Author:** Samia Ejjebli, Department of Cardiology, UHC Ibn Rochd, Casablanca, Morocco.

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## Abstract

**Background:** Left ventricular non-compaction cardiomyopathy (LVNC) is a rare congenital disorder characterized by prominent myocardial trabeculations and deep intertrabecular recesses. It is often complicated by ventricular arrhythmias, thromboembolic events, and progressive heart failure. Electrical storm and systemic infections significantly worsen the prognosis in these patients.

**Case Presentation:** We report the case of a 40-year-old man with a history of dilated hypokinetic cardiomyopathy, chronic osteitis, and substance abuse, who presented with sustained ventricular tachycardia and cardiogenic shock. Echocardiography revealed severe left ventricular systolic dysfunction (LVEF 20%), apical thrombus, and features consistent with LVNC. Despite intensive antiarrhythmic therapy and multiple cardioversions, the arrhythmia proved refractory. The patient developed worsening sepsis from a chronic knee infection, followed by multiorgan failure, splenic infarction, and embolic stroke, ultimately culminating in death.

**Conclusion:** This case illustrates the catastrophic interplay between LVNC, electrical storm, and systemic infection. It underscores the importance of early recognition, aggressive rhythm and infection control, and the limitations of current therapeutic strategies in advanced cardiomyopathy complicated by systemic inflammation.

**Keywords:** left ventricle non compaction cardiomyopathy; electrical storm; sepsis; osteitis

## Introduction

Left ventricular non-compaction cardiomyopathy (LVNC) is a rare myocardial disorder characterized by prominent trabeculations and deep intertrabecular recesses, thought to result from arrested myocardial development during embryogenesis. It is often associated with systolic dysfunction, arrhythmias, thromboembolic events, and progressive heart failure. The clinical course can be further complicated by electrical storm defined as recurrent episodes of ventricular tachycardia or fibrillation within a short time frame which carries a high risk of mortality. Infections and systemic inflammation may act as additional stressors that exacerbate cardiac instability. We present the case of a 40-year-old man with dilated cardiomyopathy and features suggestive of LVNC, whose clinical deterioration was driven by a refractory electrical storm and concomitant sepsis, ultimately leading to multiorgan failure. This case underscores the complex interplay between arrhythmogenic cardiomyopathy and systemic inflammatory states in critically ill cardiac patients.

## Case presentation

Mr. A.M., a 40-year-old man, was admitted to the cardiology department with a background of dilated hypokinetic cardiomyopathy and presented with an acute clinical decompensation marked by severe palpitations. His medical history was already complex, including chronic substance abuse, a previous ischemic stroke, and a long-standing orthopedic complication a chronic osteitis following tibial surgery which made his case particularly challenging to manage from the outset.

The patient's symptoms began about six hours prior to admission, with a sudden onset of intense palpitations, profuse sweating, and vomiting. On arrival, he was tachycardic at 220 bpm, showing clear signs of poorly tolerated sustained ventricular tachycardia (figure 1).

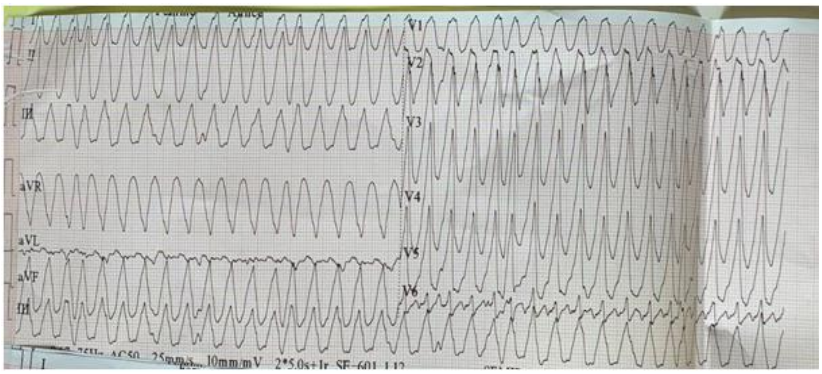


**Figure 1:** ECG after admission showing a ventricular tachycardia

Echocardiography confirmed a severely depressed left ventricular ejection fraction (LVEF) of 20%, with a markedly dilated left ventricle, prominent trabeculations, spontaneous echogenic contrast, and an apical thrombus—all features raising suspicion of a left ventricular non-compaction cardiomyopathy (LVNC) (figure 2 and 3).

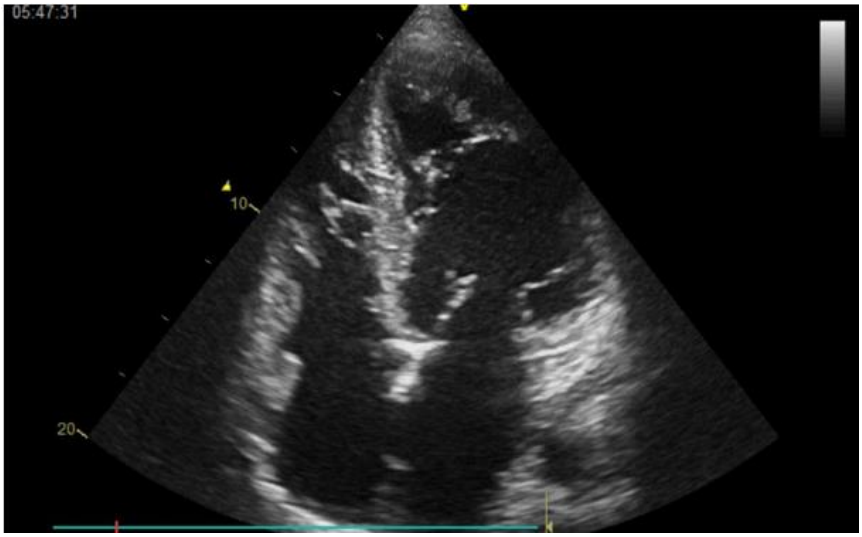


**Figure 2:** 4 cavity echocardiography showing the left ventricle trabeculations

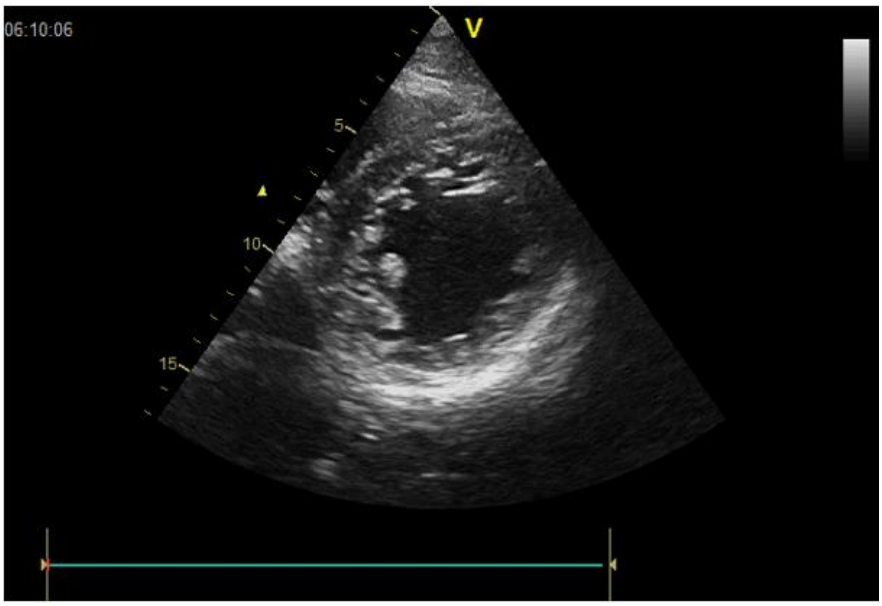


**Figure 3:** short axe Vue showing the Left ventricle trabeculations

In parallel, a purulent wound over the left knee (figure 4), presumed secondary to chronic osteitis, suggested ongoing systemic infection.

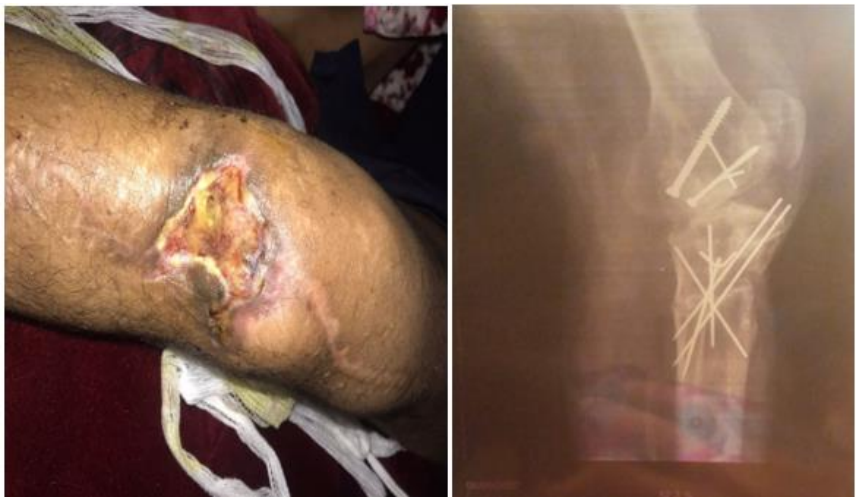

**Figure 4:** the wound of the left knee and the x-ray shows the material of the latest surgery

This compounded the difficulty of his management, especially in the context of a profoundly compromised cardiovascular system.

### Hemodynamic Instability and Refractory Arrhythmia (Days 1–2):

Upon admission, ECG confirmed a monomorphic wide-complex tachycardia consistent with VT. He was promptly treated with intravenous amiodarone (Cordarone) and underwent repeated synchronized electrical cardioversions, escalating up to 300J. Despite these measures, the arrhythmia persisted, and his blood pressure remained critically low, with peripheral hypoperfusion and altered mental status. These features characterized a full-blown electrical storm one that proved unresponsive to both pharmacologic and electrical therapy. This early phase of hospitalization was dominated by incessant arrhythmic relapses, an ominous sign that indicated both the severity of his underlying cardiomyopathy and the poor prognosis often associated with electrical storms in LVNC patients. Progressive

### Organ Dysfunction and Inflammatory Complications (Days 3–5):

By the third day, signs of organ dysfunction were beginning to accumulate. Renal function declined significantly: creatinine rose from 17.5 to 23 mg/L, and the estimated GFR fell to 32 mL/min/1.73m². Despite aggressive diuretic therapy with high-dose furosemide (Lasilix 60 mg TID), he remained oliguric. An ultrasound of the kidneys and bladder revealed no obstructive cause. Nephrology input ruled out the immediate need for dialysis but highlighted the need for close monitoring and further nephro-urological workup.

Meanwhile, the infectious focus near the knee worsened. Local cultures were obtained, and imaging was done, while empiric broad-spectrum antibiotics (Augmentin) were initiated. His inflammatory markers CRP >100 mg/L, leukocytosis suggested evolving sepsis, originating from an inoperable osteitic focus.

### Escalation to Multisystem Failure (Days 6–8):

Despite maximal medical therapy, the patient's condition continued to deteriorate. By Day 6, he was displaying classic signs of systemic inflammatory response syndrome (SIRS): persistent fever, elevated white cell count, and hemodynamic instability. His cardiovascular status remained precarious, with recurrent arrhythmias and hypotension requiring further cardioversions. On Day 8, the clinical situation worsened dramatically. He became hypotensive (BP 60/50 mmHg), with peripheral cyanosis and extreme agitation. An urgent cardioversion was performed yet again, but it was clear that the underlying pathophysiology had moved beyond rhythm disturbance alone. He now met full criteria for septic shock and cardiogenic shock.

A CT scan performed for abdominal pain revealed a splenic infarct—likely embolic in origin, considering his apical thrombus and arrhythmogenic substrate. Neurologically, he deteriorated with focal deficits, and imaging later confirmed a recent ischemic stroke. These events, together with anasarca, persistent oliguria, and escalating creatinine, pointed toward progressive multiorgan failure.

## Discussion

This case highlights the catastrophic interplay between advanced structural heart disease, arrhythmogenic instability, and systemic infection culminating in multiorgan failure. The patient, a 40-year-old man with known dilated hypokinetic cardiomyopathy, presented with features strongly suggestive of left ventricular non-compaction (LVNC)—a rare but increasingly recognized cardiomyopathy marked by prominent trabeculations and deep intertrabecular recesses resulting from arrested myocardial compaction during embryogenesis [1].

LVNC carries a high arrhythmic burden, with sustained ventricular tachycardia (VT) being a frequent and often life-threatening manifestation. In this case, the patient's electrical storm, defined as three or more episodes of VT/VF in 24 hours, was refractory to both pharmacological therapy and repeated synchronized cardioversion, underscoring the malignant arrhythmic potential of LVNC in end-stage disease [2]. Electrical storm is independently associated with increased mortality and often indicates advanced myocardial scarring and electrical instability [3]. Compounding the cardiac substrate was the presence of an apical thrombus, commonly observed in LVNC due to stagnant flow within the non-compacted myocardium, depressed LVEF, and endocardial dysfunction [4]. This likely served as the embolic source for the splenic infarct and recurrent ischemic stroke, illustrating the embolic risk inherent in this cardiomyopathy.

Concomitantly, the patient had a chronic purulent focus from osteitis of the knee, which evolved into a presumed source of sepsis. The resultant systemic inflammatory response added a second axis of pathophysiological stress. Septic shock in the context of underlying cardiogenic shock creates a vicious circle: inflammatory cytokines worsen myocardial depression and vasoplegia, while impaired cardiac output further compromises perfusion and host immune responses [5]. As his course progressed, the clinical picture evolved into multiorgan dysfunction syndrome (MODS) with acute kidney injury, neurological deterioration from embolic stroke, hepatic and splenic hypoperfusion, and systemic edema demonstrating the terminal trajectory in patients with end-stage cardiomyopathy complicated by infection and arrhythmias. Importantly, his refractoriness to conventional antiarrhythmic therapy and ineligibility for advanced heart failure interventions (e.g., ventricular

assist device, transplant) due to systemic infection and comorbidities contributed to the fatal outcome.

The coexistence of LVNC and chronic systemic infection is exceedingly rare in the literature, but this case underscores how extra myocardial factors like infection and inflammation can unmask or accelerate terminal decompensation in cardiomyopathy patients. Furthermore, it raises questions regarding earlier detection, aggressive rhythm control, prophylactic anticoagulation, and surgical debridement of chronic infections in similar high-risk patients.

## Conclusion

This case illustrates the lethal convergence of cardiac electrical instability, unresolved systemic infection, and thromboembolic events in a patient with advanced cardiomyopathy. The inability to control the rhythm disturbances, surgically treat the infectious source, or prevent embolic complications reveals the clinical limitations we still face in such scenarios. Early identification of high-risk features, multidisciplinary management, and possibly earlier consideration of mechanical circulatory support might be avenues for improving outcomes in future similar cases.

**Consent:** As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

**Ethical Approval:** It is not applicable.

**Competing Interests:** Authors have declared that no competing interests exist

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