



## YOUNG ISCHEMIC STROKE AS 1ST PRESENTATION OF LEFT VENTRICULAR NON-COMPACTION WITH FATAL RECURRENT EMBOLIC STROKE

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### ABSTRACT

**Background:** Left ventricular non-compaction (LVNC) is a rare cardiomyopathy characterized by excessive ventricular trabeculations and deep intertrabecular recesses. Thromboembolic complications, including ischemic stroke, are uncommon but potentially life-threatening. **Case Presentation:** A 26-year-old previously healthy man presented with sudden-onset left-sided hemiparesis and Broca's aphasia. Brain computed tomography demonstrated an acute ischemic infarct. Transthoracic echocardiography revealed a dilated left ventricle with reduced ejection fraction (30%), excessive apical trabeculations consistent with LVNC, and multiple mobile left ventricular thrombi. Despite supportive management, the patient developed recurrent neurological deterioration with respiratory failure and died. **Conclusion:** This case highlights LVNC as an important but often overlooked cause of cryptogenic ischemic stroke in young adults. Early cardiac imaging is essential for identifying LVNC and associated ventricular thrombi, enabling timely anticoagulation and potentially preventing recurrent embolic events.

### KEYWORDS :

### INTRODUCTION

Left ventricular non-compaction (LVNC) is an uncommon myocardial phenotype characterized by excessive left ventricular trabeculations and deep intertrabecular recesses. [1] [2] With advances in echocardiography and cardiac magnetic resonance imaging, its recognition has increased in recent years. [2] [3] The European Society of Cardiology lists LVNC among "unclassified" cardiomyopathies, whereas the American Heart Association includes it among primary congenital forms [4]. Several diagnostic criteria have been proposed, but there is currently no gold standard. Clinically, LVNC may present with heart failure, ventricular arrhythmias, systemic thromboembolism, and sudden cardiac death. [1] [2] [3] Ischemic stroke is an uncommon but important manifestation, resulting from thrombus formation due to blood stasis within the non-compacted myocardium, left ventricular dysfunction, or associated atrial arrhythmias. [2] [6] [7] We report a young patient who presented with acute ischemic stroke as the initial manifestation of previously undiagnosed LVNC, highlighting the importance of considering an underlying cardiac etiology in young adults with cryptogenic stroke. [7]

### Case Presentation

A 26-year-old previously healthy man presented to the emergency department with sudden-onset left-sided hemiparesis and Broca's aphasia of six hours' duration.

Patient was out of the window for any chemical or mechanical thrombectomy. There was no history of hypertension, diabetes mellitus, dyslipidemia, smoking, alcohol consumption, illicit drug use, or prior cerebrovascular events. His family history, including a three-generation pedigree, was unremarkable for congenital heart disease, inherited cardiomyopathies, premature sudden cardiac death, or thromboembolic disorders.

On admission, the patient was hemodynamically stable, with a pulse rate of 74 beats/min, blood pressure of 120/70 mmHg, and oxygen saturation of 97% on room air. General physical and cardiovascular examinations were unremarkable, with no audible cardiac murmurs or signs of heart failure.

Neurological examination revealed Broca's aphasia with left-sided upper and lower limb weakness (Medical Research Council grade 3/5) and an extensor plantar response on the affected side. The initial electrocardiogram demonstrated normal sinus rhythm without evidence of atrial fibrillation or conduction abnormalities. Basic investigation of this patient was normal. (figure 1,2) An urgent non-contrast computed

tomography (CT) scan of the head with CT angiography was performed, which revealed ischemic infarct of right frontoparietal region. (Figure 3). As part of the evaluation for ischemic stroke in a young patient without conventional vascular risk factors, transthoracic echocardiography was performed. Two-dimensional echocardiography demonstrated a dilated left ventricle with a left ventricular end-diastolic diameter of 63 mm and moderately reduced left ventricular systolic function with an ejection fraction of 30%.

Multiple prominent trabeculations with deep intertrabecular recesses were observed predominantly involving the apical and mid-ventricular segments, and color Doppler imaging demonstrated blood flow within these recesses, consistent with left ventricular non-compaction. The diagnosis of LVNC was supported by an end-systolic non-compacted to compacted (NC/C) myocardial ratio  $>2:1$ , fulfilling the Jenni echocardiographic criteria (Figure 5). [8]. In addition, multiple mobile thrombi were visualized within the left ventricular cavity (Figure 4). Based on these findings, a diagnosis of left ventricular non-compaction cardiomyopathy complicated by left ventricular thrombus and cardioembolic ischemic stroke was established.

Despite supportive management, the patient's neurological status deteriorated on the third day of hospitalization. He developed complete paralysis of the affected limbs, with muscle power declining to 0/5, followed by aspiration leading to acute hypoxemic respiratory failure requiring endotracheal intubation and mechanical ventilation. Despite aggressive supportive treatment, the patient's clinical condition continued to worsen, and he succumbed approximately 12 hours later due to progressive respiratory failure secondary to massive ischemic stroke.

Table 1. Baseline laboratory investigations

| Investigation          | Result              | Reference Range         |
|------------------------|---------------------|-------------------------|
| Hemoglobin             | 13.6 g/dL           | 13–17 g/dL              |
| Total leukocyte count  | $6 \times 10^9/L$   | $4–11 \times 10^9/L$    |
| Platelet count         | $233 \times 10^9/L$ | $150–450 \times 10^9/L$ |
| Serum sodium           | 137 mmol/L          | 135–145 mmol/L          |
| Serum potassium        | 3.8 mmol/L          | 3.5–5.0 mmol/L          |
| Blood urea nitrogen    | 13 mg/dL            | 7–20 mg/dL              |
| Serum creatinine       | 1.0 mg/dL           | 0.7–1.3 mg/dL           |
| Random blood glucose   | 138 mg/dL           | 70–140 mg/dL            |
| Liver function tests   | Normal              | Within reference range  |
| Thyroid function tests | Normal              | Within reference range  |

**Figure 1** All routine haematological, biochemical, and metabolic investigations were within normal limits



**Figure 2** Twelve-lead electrocardiogram demonstrating sinus rhythm with diffuse T-wave inversions in the inferior and anterolateral leads.

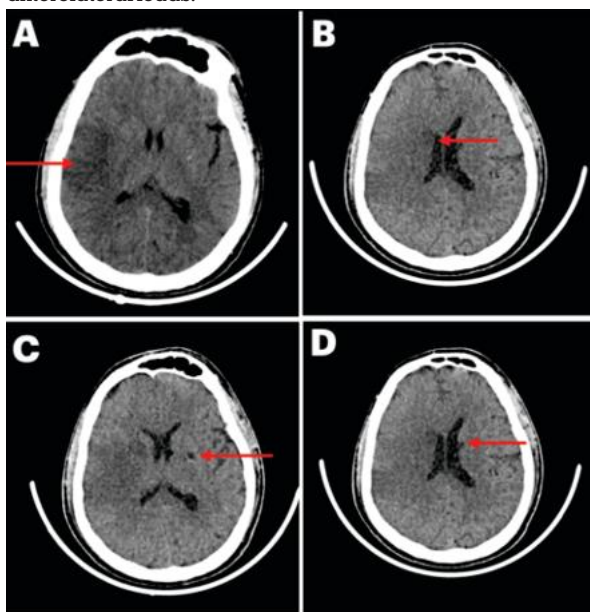


**Figure 3** Non-contrast axial CT scan of the brain demonstrating : (A) ill-defined hypodensity with loss of gray-white differentiation in the right frontoparietal parasylvian region, consistent with an acute ischemic infarct; (B) ill-defined hypodensity involving the body of the right caudate nucleus; (C) Chronic lacunar Infarct involving the left Globus Pallidus; (D) Chronic lacunar infarct involving the body of the left caudate nucleus.

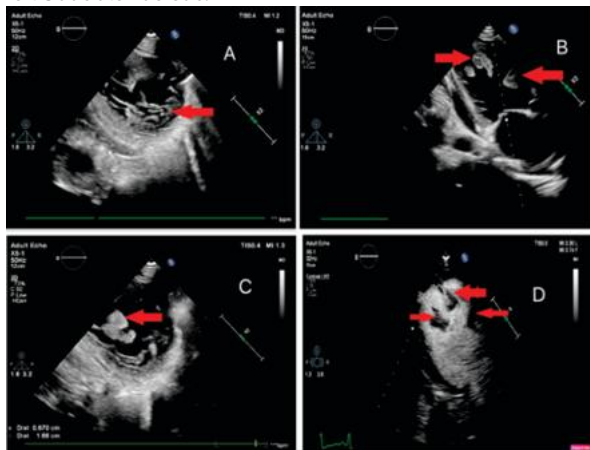


**Figure 4** Transthoracic echocardiography demonstrating Left Ventricular Non-Compaction (LVNC) with multiple left ventricular thrombi. (A) Parasternal short-axis view demonstrating a two-layered myocardium with thin and compacted outer layer and thick, spongy, and heavily "non-

compacted" inner layer. (B) Apical two-chamber view demonstrating multiple mobile thrombi within the left ventricular cavity. (C) Parasternal short-axis view demonstrating a large left ventricular thrombus. (D) Contrast enhanced echocardiography confirming multiple left ventricular thrombi.

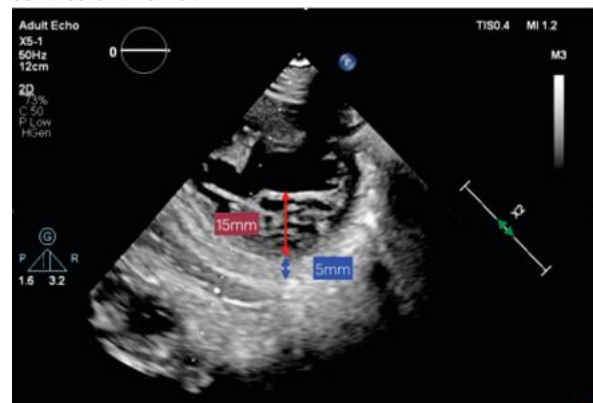


**Figure 5** Parasternal short-axis view of the left ventricle at end-systole demonstrating a two-layered myocardium with a non-compacted to compacted (NC/C) myocardial ratio >2:1, consistent with the Jenni echocardiographic criteria for left ventricular non-compaction.

## DISCUSSION

Left ventricular non-compaction (LVNC) is a rare heart disease characterized by a two-layered myocardium with excessive trabeculations and deep intertrabecular recesses resulting from incomplete myocardial compaction during embryogenesis. Although LVNC has been reported as the third most frequently diagnosed cardiomyopathy, its prevalence in the adult population remains low, estimated at approximately 0.05%.[4] Despite increasing recognition with advances in echocardiography and cardiac magnetic resonance (CMR), LVNC continues to represent one of the most controversial entities in contemporary cardiology. Recent evidence suggests that LVNC is best regarded as a structural phenotype within a cardiomyopathic continuum rather than a single discrete disease, emphasizing the importance of integrating imaging findings with the overall clinical context rather than relying solely on myocardial morphology.[5],[10],[16]

One of the greatest challenges in clinical practice is the absence of universally accepted diagnostic criteria for LVNC. Multiple echocardiographic and CMR-based diagnostic criteria have been proposed, including those described by Chin, Jenni, Petersen, and Stöllberger, each emphasizing different morphologic characteristics and imaging parameters.[8],[13],[14],[3] Consequently, the reported prevalence of LVNC varies considerably depending on the diagnostic criteria employed. The recent JACC Cardiovascular Imaging Expert Panel highlighted that excessive left ventricular trabeculation may also be observed in healthy individuals, athletes, pregnant women, and patients with other cardiomyopathies; therefore, isolated hypertrabeculation should not be considered synonymous with LVNC.[10] Similarly, Arbustini et al. proposed that LVNC should be viewed as a phenotypic trait shared by several myocardial diseases rather than an independent cardiomyopathy in every patient, further illustrating the ongoing diagnostic uncertainty.[16]

Thromboembolic complications remain among the most devastating manifestations of LVNC and are attributed to blood stasis within the deep intertrabecular recesses, impaired left ventricular systolic function, atrial arrhythmias, and left ventricular thrombus formation.[3],[4],[5] Cerebral embolism may be the initial clinical presentation, particularly

in young adults without conventional vascular risk factors.[6],[17] Pöyhönen et al. demonstrated a significant association between excessive left ventricular trabeculation and cryptogenic ischemic stroke in young adults, suggesting that LVNC should be considered during the evaluation of unexplained ischemic stroke.[17] Similarly, Finsterer and Stöllberger reviewed the neurological manifestations of LVNC and emphasized that ischemic stroke represents one of the most important extracardiac complications of this disease.[6]

Our patient illustrates this uncommon but clinically significant presentation. A previously healthy young male presented with acute ischemic stroke and was subsequently found to have severe left ventricular systolic dysfunction, classical echocardiographic features of LVNC, and multiple mobile left ventricular thrombi. Despite supportive management, he developed recurrent neurological deterioration followed by aspiration and respiratory failure, ultimately resulting in death. Similar presentations have been reported only rarely in the literature, including cases described by Benyounes et al., Nacanabo et al., and Romero-Barajas et al., all highlighting ischemic stroke as the first manifestation of previously undiagnosed LVNC.[7],[11],[9]

Current management of LVNC is largely extrapolated from heart failure and thromboembolic guidelines because disease-specific evidence remains limited. The 2023 ESC Guidelines recommend individualized management based on left ventricular systolic function, documented ventricular thrombus, atrial fibrillation, previous systemic embolism, ventricular arrhythmias, and family history rather than trabecular morphology alone.[2] Although anticoagulation is generally recommended in patients with documented left ventricular thrombus or previous embolic events, robust evidence guiding the management of asymptomatic patients or those with preserved systolic function is still lacking. [2],[5],[15]

This case highlights an important clinical message. In young adults presenting with cryptogenic ischemic stroke, particularly in the absence of traditional vascular risk factors, clinicians should maintain a high index of suspicion for an underlying cardioembolic source. Routine transthoracic echocardiography should include careful assessment of left ventricular morphology, and the presence of excessive trabeculations, reduced systolic function, or intracavitary thrombi should prompt consideration of LVNC. Early recognition may facilitate timely anticoagulation and appropriate heart failure management, potentially preventing recurrent embolic events. Further prospective studies are required to establish standardized diagnostic criteria and evidence-based treatment strategies for this challenging cardiomyopathy. [2],[5],[10],[16]

## CONCLUSION

Non-compaction of the left ventricle is a complex condition requiring early and appropriate management. The case presented highlights the diagnostic and therapeutic challenges associated with non-compaction of the heart, a phenomenon that is still poorly understood, with complications that can significantly affect the patient's quality of life. Treatment must be personalised, combining optimal management of heart failure, prevention of thrombus and treatment of stroke.

Multidisciplinary management, combining advanced cardiac imaging and neurovascular monitoring, is essential to optimise the management of this complex condition.

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