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RESEARCH ARTICLE

Silent Culprit: Young Stroke Unmasking Left Ventricular Non-Compaction Cardiomyopathy

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Abstract: ***Background:*** Stroke in young adults is an uncommon but devastating event that warrants investigation for rare etiologies beyond traditional vascular risk factors. Among these, Left Ventricular Non-Compaction Cardiomyopathy (LVNC), a rare congenital myocardial disorder, is an underrecognized source of cardioembolic stroke. LVNC is characterized by prominent ventricular trabeculations and deep intertrabecular recesses that predispose to stasis, thrombus formation, and systemic embolism. This case highlights the presentation of LVNC unmasked by an acute ischemic stroke in a previously healthy young woman. ***Case Presentation:*** A 19-year-old female presented with sudden onset of left-sided weakness, facial deviation, and slurred speech for 30 minutes. She was hemodynamically stable and had a National Institutes of Health Stroke Scale (NIHSS) score of 12, indicating a moderate stroke. Non-contrast CT brain showed no hemorrhage with an ASPECTS score of 9, making her eligible for thrombolysis. She received intravenous alteplase (0.9 mg/kg) within 2 hours of onset, leading to rapid neurological improvement (NIHSS 2 at 24 hours). Subsequent imaging ruled out hemorrhagic transformation. Further evaluation revealed T-wave inversions on ECG, and echocardiography demonstrated left ventricular hyper-trabeculation with ejection fraction (EF) of 40%. Cardiac magnetic resonance imaging (CMR) confirmed LVNC with apical aneurysm, fulfilling the Petersen criterion (non-compacted to compacted ratio >2.3). Thrombophilia and autoimmune profiles were negative. She was discharged on oral anticoagulation (warfarin, target INR 2-3) and heart-failure therapy, achieving full functional recovery (modified Rankin Scale = 1) at 3-month follow-up. ***Discussion:*** This case illustrates the diagnostic and therapeutic challenges in young stroke. LVNC often remains asymptomatic until an embolic event reveals the underlying pathology. The deep trabecular recesses and apical aneurysm act as potential sources for thrombus formation, even in the absence of arrhythmias or overt cardiac dysfunction. Our patient represents the ideal clinical sequence-timely thrombolysis, comprehensive etiologic evaluation, and targeted management-resulting in excellent neurological and cardiac outcomes. The case reinforces the value of NIHSS and ASPECTS scoring in acute decision-making and highlights the need to include cardiac imaging (Echocardiography and CMR) in the etiological evaluation of stroke in young adults without conventional risk factors. Literature suggests that stroke is often the first manifestation of LVNC, and delayed diagnosis increases recurrence risk. The present case supports these findings and emphasizes the effectiveness of anticoagulation in secondary prevention. ***Conclusion:*** Left Ventricular Non-Compaction Cardiomyopathy is a rare but clinically important cause of embolic stroke in the young. Early recognition through multimodal cardiac imaging is essential for guiding anticoagulation and preventing recurrence. This case exemplifies how structured acute stroke management combined with thorough etiological evaluation can uncover rare cardiomyopathies and significantly improve outcomes. Clinicians should maintain a high index of suspicion for LVNC in cryptogenic or cardioembolic strokes in young adults to ensure early diagnosis and timely intervention.

Keywords: Young stroke, Left ventricular non-compaction cardiomyopathy, Cardioembolic stroke, Thrombolysis, Anticoagulation.

INTRODUCTION

Stroke in young adults represents a growing clinical concern, accounting for approximately 10-15% of all ischemic strokes worldwide. Unlike elderly patients, in whom traditional vascular risk factors such as hypertension, diabetes mellitus, and atherosclerosis predominate, strokes in young adults often stem from unusual and heterogeneous causes-including cardioembolic, prothrombotic, autoimmune, and vascular disorders. The socioeconomic burden is disproportionately high because of the long-term disability in a population otherwise at the peak of productivity and reproductive potential. Therefore, identifying uncommon etiologies, especially structural cardiac abnormalities, is crucial for optimizing secondary prevention and improving outcomes.[1]

Among cardiac causes of young stroke, Left Ventricular Non-Compaction Cardiomyopathy (LVNC) has emerged as a rare but increasingly recognized entity. LVNC is a distinctive form of cardiomyopathy characterized by excessive trabeculations and deep intertrabecular recesses within the left ventricular (LV) myocardium, resulting from an arrest in the normal compaction process during embryogenesis. This results in a bilayered myocardium-an outer thin compacted layer and an inner thick non-compacted layer with prominent trabeculations communicating with the LV

cavity. The morphological abnormality predisposes to left ventricular systolic dysfunction, ventricular arrhythmias, heart failure, and thromboembolic complications, including stroke.[2]

The clinical spectrum of LVNC is broad and may range from asymptomatic individuals incidentally diagnosed on imaging to those presenting with heart failure, sudden cardiac death, or systemic embolism. While genetic inheritance-both familial and sporadic-has been described, the disorder often escapes early recognition due to its variable phenotype and overlapping features with other cardiomyopathies such as dilated, hypertrophic, and restrictive subtypes. With advances in cardiac imaging, particularly echocardiography and cardiac magnetic resonance imaging (CMR), the identification of LVNC has become more feasible. The Petersen criterion on CMR (non-compacted to compacted ratio >2.3 in end-diastole) remains the standard reference for diagnosis, though different thresholds proposed by Jacquier, Grothoff, and Stacey have led to variations in reported prevalence.[3]

The thromboembolic potential in LVNC is multifactorial. The deep recesses of the non-compacted myocardium promote blood stasis, creating a nidus for thrombus formation. Reduced ejection fraction, ventricular aneurysm, atrial fibrillation, and regional wall motion abnormalities further amplify embolic risk. Notably, several cases have documented cardioembolic stroke as the first manifestation of LVNC, even in patients with preserved ejection fraction and sinus rhythm. The reported prevalence of stroke among LVNC cohorts varies widely (0-38%), underscoring the heterogeneity of diagnostic criteria and follow-up durations. Still, the possibility of embolism in structurally abnormal yet compensated ventricles signifies a “silent threat” often overlooked until catastrophic events such as stroke occur.[4]

A Study by *et al.* (20)[5] encompassing over 5,000 patients estimated a pooled thromboembolism prevalence of 6.2% in adults and 2.6% in pediatric populations, highlighting that although rare, embolic events in LVNC carry significant morbidity. Similarly, *et al.* (20)[6] found higher percentages of non-compacted LV volume among young adults with cryptogenic ischemic stroke compared with controls, reinforcing the role of LVNC as an underdiagnosed cause of cryptogenic stroke.

Diagnostic awareness among clinicians remains limited. Symptoms such as exertional dyspnea, palpitations, or syncope are often misattributed to benign causes in young adults. Furthermore, conventional transthoracic echocardiography may fail to visualize apical trabeculations adequately. Hence, comprehensive evaluation of young stroke should routinely include cardiac imaging-particularly in the absence of common risk factors-to rule out structural causes like LVNC, patent foramen ovale (PFO), or valvular pathologies.

The importance of this case lies in its demonstration of LVNC presenting as the *first and only manifestation* in a previously healthy young woman who developed an acute ischemic stroke. Early recognition permitted timely thrombolysis with excellent neurological recovery, followed by identification of the underlying cardiomyopathy through echocardiography and CMR. The case underscores the role of systematic stroke evaluation protocols employing NIHSS (National Institutes of Health Stroke Scale) and ASPECTS (Alberta Stroke Program Early CT Score), along with extended etiological work-up tailored for young patients.

RESULTS AND OBSERVATIONS:

A 19-year-old previously healthy female presented to the Emergency Department with sudden-onset deviation of the mouth to the right, slurred speech, and weakness of the left upper and lower limbs, noticed 30 minutes prior to arrival. She had no history of hypertension, diabetes, smoking, alcohol, or drug use. There was no prior cardiac illness, rheumatic fever, or family history of cardiomyopathy or early sudden death.

Initial Evaluation

On arrival, she was conscious, alert, and oriented, with stable vital parameters (BP 118/76 mmHg, pulse 86 bpm, SpO_2 98% on room air). Neurological examination revealed left hemiparesis (power 3/5), left facial weakness (UMN type), and mild dysarthria, without sensory loss or visual field deficits. NIHSS score was 12, indicating a moderate stroke severity. Cardiovascular and respiratory examinations were unremarkable, and no murmurs or gallops were detected.

Emergency Investigations

Initial work-up included:

- Random blood glucose, CBC, renal and liver function tests, coagulation profile - all within normal limits.
- Non-contrast CT brain showed no intracranial hemorrhage. The ASPECTS score was 9, with subtle insular ribbon loss but preserved middle cerebral artery (MCA) territory.

Given her young age, short onset-to-door time (30 min), and absence of contraindications, she was deemed a candidate for intravenous thrombolysis.

Treatment and Immediate Course

She received intravenous alteplase (rt-PA, 0.9 mg/kg) administered within two hours of onset. The infusion was uneventful, and her neurological status improved markedly-NIHSS decreased to 4 at 24 h and 2 at discharge. A follow-up CT brain at 24 h revealed no hemorrhage or infarct progression, with resolution of the hyperdense MCA sign (Figure 2).

Extended Etiological Evaluation

Given the absence of conventional stroke risk factors, further evaluation for cardioembolic and systemic causes was initiated.

- Electrocardiogram (ECG) revealed T-wave inversions in leads V1-V6, I, II, III, and aVF, suggesting underlying structural heart disease (Figure 1).
- Transthoracic echocardiography (TTE) demonstrated marked left ventricular hyper-trabeculation, predominantly at the apex and mid-ventricular segments, with an ejection fraction (EF) of 40% (Figure 3). No valvular lesions, vegetations, or intracardiac thrombus were seen.
- Cardiac Magnetic Resonance Imaging (CMR) confirmed LV non-compaction cardiomyopathy with apical aneurysm, fulfilling Petersen's diagnostic criterion (NC/C ratio >2.3 in diastole). There were patchy myocardial wall edemas in apical inferior, anterior, and septal regions, and slow turbulent flow within the apical cavity (Figures 4A-4C).
- Autoimmune and thrombophilia screening (ANA, antiphospholipid antibodies, protein C, protein S, factor V Leiden) was negative.

No carotid or vertebral dissection was identified on CT angiography, ruling out large-vessel vasculopathy.

Differential Diagnosis

Differentials for embolic stroke in the young included:

- Paradoxical embolism via PFO (excluded by TTE),
- Rheumatic or infective endocarditis (no vegetations),
- Dilated or hypertrophic cardiomyopathy, and
- LVNC, confirmed on CMR as the final diagnosis.

Final Diagnosis

Ischemic stroke secondary to cardioembolism from Left Ventricular Non-Compaction Cardiomyopathy with apical aneurysm.

Hospital Course and Outcome

Following thrombolysis, she was monitored in the stroke unit. Anticoagulation with oral warfarin (target INR 2-3) was initiated after 48 hours, bridged with low-molecular-weight heparin. Standard heart-failure therapy comprising bisoprolol 2.5 mg OD, enalapril 5 mg OD, and furosemide 20 mg PRN was introduced. She underwent supervised physiotherapy and speech rehabilitation.

At discharge (day 7), her neurological deficits had nearly resolved (NIHSS = 2), and she was functionally independent (modified Rankin Scale = 1). Follow-up at three months revealed no recurrent stroke or cardiac symptoms. Repeat echocardiography demonstrated stable LV function with persistent trabeculations.

Table 1: Clinical Course and Investigations

Parameter	Finding
Age/Sex	19-year-old female
Symptoms	Left hemiparesis, facial deviation, dysarthria
Onset-to-door time	30 min
NIHSS (admission)	12
ASPECTS	9
Treatment	IV alteplase within 2 hours
NIHSS (24h)	4
NIHSS (discharge)	2
CT 24h	No haemorrhage , shows hyperdense MCA sign
Echocardiography	LV hypertrabeculation, EF 40%
Cardiac MRI	LVNC with apical aneurysm
Outcome	mRS 1 at 3 months

Table 2: Major Causes of Young Stroke (18-50 years)

Category	Examples
Cardioembolic	LVNC, rheumatic heart disease, atrial fibrillation, PFO, ventricular aneurysm
Arterialdissection	Carotid/vertebral artery dissection
Prothrombotic	Antiphospholipid syndrome, protein C/S deficiency, factor V Leiden
Vasculitis	Takayasu arteritis, lupus, primary CNS vasculitis
Other	Cocaine/amphetamine use, migraine-related infarcts, HIV

Table 3: Selected Case Reports of Stroke due to LVNC

Author/Year	Age	Presentation	Management	Outcome
Norton PT. (2012)[7]	28	Recurrent embolic stroke	Anticoagulation after recurrence	Recovery
Abraham RL *et al.* (2009)[8]	35	MCA stroke needing thrombectomy	Stenting + anticoagulation	Good
Camen S *et al.* (2200)[9]	30	Initially cryptogenic stroke	Delayed anticoagulation	Recurrent TIA
Present Case, 2025	19	First embolic stroke	Early thrombolysis + anticoagulation	Excellent (mRS 1)

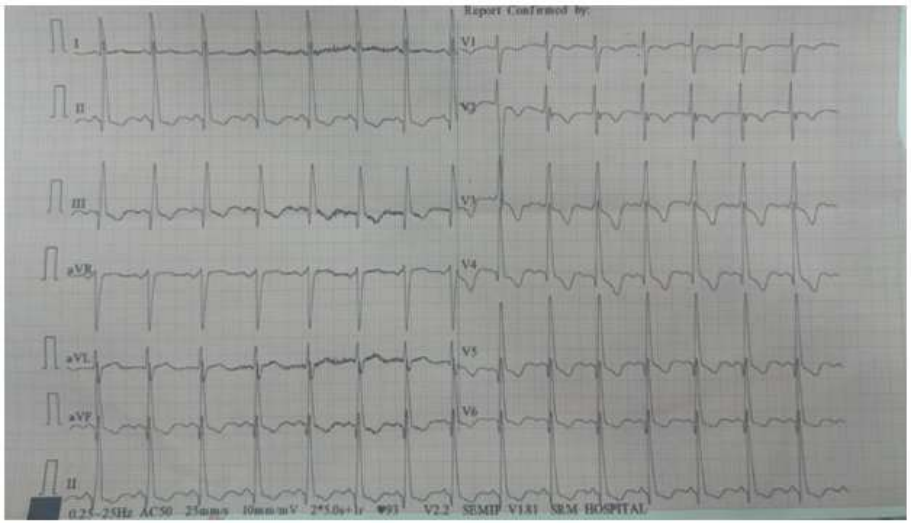


Figure 1: ECG showing diffuse T-wave inversions (V1-V6, I, II, III, aVF) suggestive of underlying LV structural abnormality.

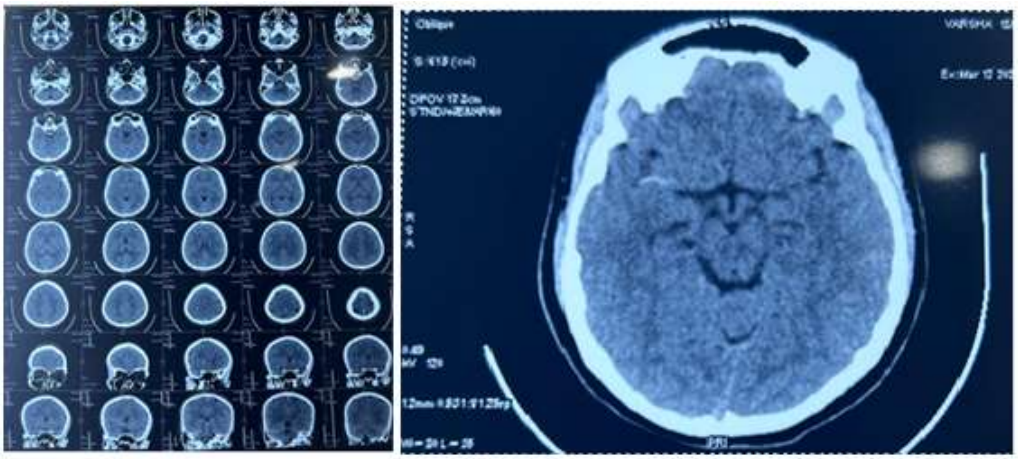


Figure 2: CT brain showing hyperdense right MCA sign pre-thrombolysis (ASPECTS 9).

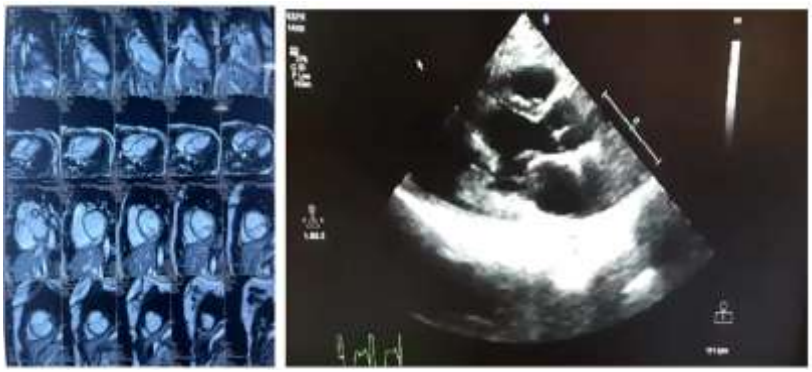
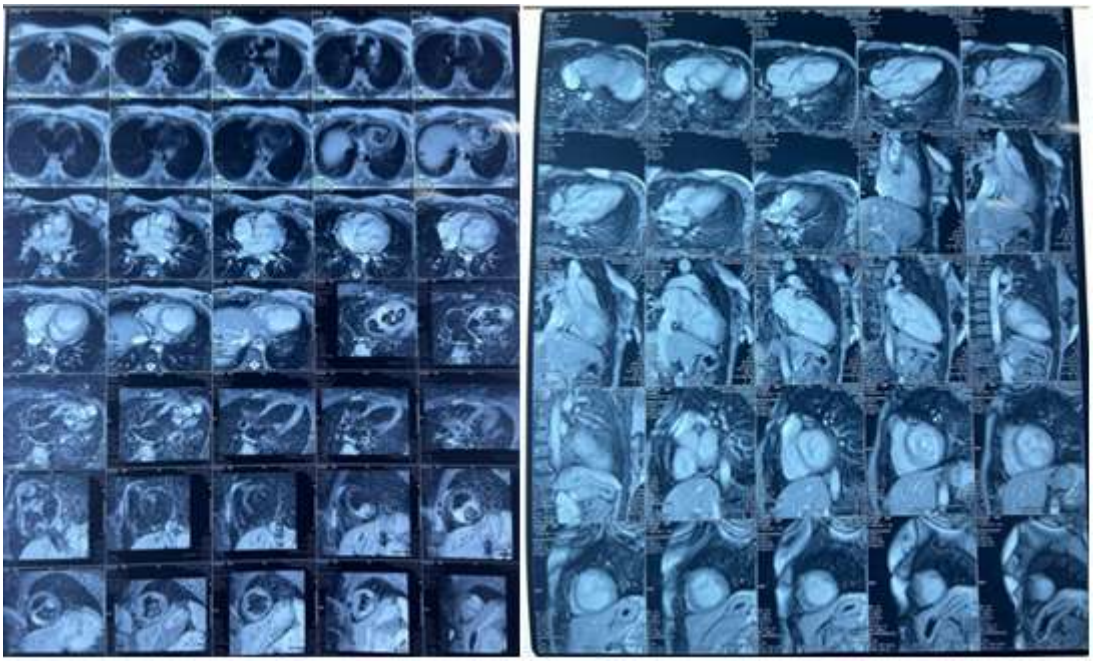


Figure 3: Echocardiogram demonstrating hyper-trabeculated LV with deep intertrabecular recesses.



Figures 4A-4B: CMR revealing non-compacted LV apex, hypokinesia, apical aneurysm, and slow flow turbulence.

DISCUSSION

This case underscores a rare but clinically significant etiology of ischemic stroke in young adults-Left Ventricular Non-Compaction Cardiomyopathy (LVNC)-and demonstrates the importance of systematic diagnostic evaluation in atypical stroke presentations. The patient, a previously healthy 19-year-old female with no vascular risk factors, presented with acute focal neurological deficits, was appropriately thrombolysed, and subsequently found to have LVNC on echocardiography and cardiac MRI. This case highlights how a structural cardiac abnormality, often silent, may serve as a "hidden embolic source" that only manifests after a catastrophic neurological event.

Clinical and Pathophysiological Interpretation

LVNC is characterized by a bilayered myocardium consisting of a compacted epicardial and a non-compacted endocardial layer with deep trabecular recesses that communicate with the LV cavity. This morphologic abnormality, believed to result from arrested myocardial compaction during embryogenesis, predisposes to myocardial dysfunction, arrhythmia, and thromboembolism. In our patient, the presence of apical aneurysm and moderate LV systolic dysfunction (EF 40%) provided an anatomic substrate for mural thrombus formation and subsequent embolization. However, thromboembolism in LVNC can occur even with normal ejection fraction, suggesting that mechanical stasis within recesses is itself thrombogenic. The embolic mechanism in LVNC stems from low shear stress and regional blood stagnation within trabecular spaces. Additionally, associated ventricular arrhythmias or regional wall hypokinesia may further impair blood flow, predisposing to clot formation. Our case fits this pathophysiologic model-absence of arrhythmia but presence of apical aneurysm contributed to embolization into the MCA territory, culminating in acute ischemic stroke.

Comparison with Existing Literature

Stroke as an initial manifestation of LVNC remains uncommon but increasingly reported. Ambulatory EC. (2009)[4] reviewed adult and pediatric LVNC cases and noted stroke prevalence ranging from 0-38%, depending on diagnostic criteria and comorbidity profiles. Similarly, Cecchin F et al. (2010)[6] reported a 15% incidence of thromboembolic events in adults with LVNC, with poorer prognosis among those with reduced EF. Our patient aligns with these findings, presenting with moderate systolic dysfunction and single embolic episode, yet achieving full neurological recovery due to timely thrombolysis.

Large registry analyses have further clarified the epidemiological context. MacKnight JM. (2022)[10] analyzed over 2,500 LVNC patients and reported a major adverse cardiovascular event (MACE) rate of 1.6% per patient-year, encompassing stroke, systemic embolism, and sudden cardiac death. Likewise, Finsterer J. (2010)[11] in a meta-analysis of more than 5,000 subjects found pooled thromboembolism prevalence of 6.2% in adults and 2.6% in pediatric cohorts, confirming that LVNC is not benign and carries an appreciable embolic risk.

Recent case reports reinforce this trend. Pepi M et al. (2010)[12] described recurrent embolic strokes in a 28-year-old male where LVNC was diagnosed only after recurrence, highlighting the risk of missed early identification. Williams G.et al. (2016)[13] presented a 35-year-old female with MCA stroke requiring thrombectomy; LVNC was subsequently confirmed on

imaging, emphasizing the role of multimodal diagnostics in young stroke evaluation. Aramaki K et al. (2005)[14] similarly reported a cryptogenic stroke in a 30-year-old that later revealed LVNC, underscoring the frequency of delayed recognition. In contrast, the present case demonstrates the ideal diagnostic sequence-early identification, prompt thrombolysis, and targeted secondary prevention-leading to an excellent outcome (mRS 1).

CONCLUSION

This case illustrates how Left Ventricular Non-Compaction Cardiomyopathy can silently predispose to catastrophic embolic stroke in young adults. Early recognition, facilitated by systematic stroke assessment (NIHSS, ASPECTS) and advanced cardiac imaging, enables appropriate acute and preventive therapy. The patient's complete neurological recovery and absence of recurrence reinforce that timely thrombolysis, followed by anticoagulation and heart-failure management, can dramatically improve prognosis.

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