Vol. 7, No. 7, August 2026
E-ISSN:2723 – 6692 P-ISSN:2723– 6595
http://jiss.publikasiindonesia.id/

Cardioembolic Stroke as tche Initial Manifestation of Previously Undiagnosed Moderate Mitral Stenosis with Atrial Fibrillation: A Case Report

Dian R. Hasibuan[1]*, Dharma Yuda[2], Anwari Delmi[3], Sanggap I. Sitompul[4], Yusuf Galenta[5]
Muara Teweh Regional Hospital, Indonesia[1]
Doris Sylvanus General Hospital, Indonesia[2,4,5]
Tk. IV Guntung Payung Regional Hospital, Indonesia[3]
Email: dianrosikinhasibuan@gmail.com[1]*, dharma.yida6@gmail.com[2], anwari.delmi5@gmail.com[3], Sanggap@med.upr.ac.id[4], yusufgalenta@gmail.com[5]

Keywords:
Cardioembolic stroke; atrial fibrillation; mitral stenosis; ischemic stroke; valvular heart diseasee.

Abstract
Cardioembolic stroke remains one of the most severe complications of atrial fibrillation (AF), particularly in patients with structural heart disease such as mitral stenosis (MS). Moderate-to-severe MS contributes to left atrial enlargement and blood stasis, thereby increasing the risk of thromboembolism even in the absence of visible intracardiac thrombus on imaging.1,2 Early identification of underlying valvular abnormalities in patients presenting with ischemic stroke is essential for optimizing secondary prevention.3 A 40-year-old woman was referred to a tertiary healthcare facility for echocardiographic evaluation following hospitalization for suspected nonhemorrhagic stroke associated with atrial fibrillation and a urinary tract infection. She initially presented with dysarthria, aphasia, and dysphagia. Physical examination revealed stable hemodynamics with central cranial nerve VII and XI paresis. Electrocardiography demonstrated atrial fibrillation with a controlled ventricular response. Brain computed tomography showed infarction in the left frontal lobe and left corona radiata, with additional lacunar infarcts in the midbrain and pons. Transthoracic echocardiography revealed moderate mitral stenosis with a mitral valve area of 1.44 cm^2, mild mitral regurgitation, left atrial dilation, mildly reduced left ventricular systolic function, reduced right ventricular systolic function, and minimal pericardial effusion, without evidence of intracardiac thrombus or vegetation. The patient received multidisciplinary management involving neurologists and cardiologists, including antiplatelet therapy, a beta-blocker, a statin, diuretics, and supportive care, resulting in gradual neurological improvement. This case highlights ischemic stroke as the first clinical manifestation of previously undiagnosed moderate mitral stenosis accompanied by atrial fibrillation. The coexistence of AF and left atrial enlargement likely contributed to the cardioembolic risk despite the absence of a demonstrable intracardiac thrombus.

INTRODUCTION

Cardioembolic stroke accounts for a substantial proportion of ischemic strokes and is associated with greater morbidity, recurrence, and mortality than other stroke subtypes (Cozza et al., 2026; Ip et al., 2022; Johansen et al., 2023; Kato et al., 2024; Myrmel et al., 2025; Perera et al., 2022). Atrial fibrillation (AF) is among the strongest predictors of cardioembolic events

because impaired atrial contraction promotes blood stasis and thrombus formation within the left atrium, particularly the left atrial appendage.

Mitral stenosis (MS) is an important structural cardiac abnormality associated with atrial remodeling and thromboembolic complications (Abd Elkareem et al., 2023; Autherith et al., 2026; Campora et al., 2024; Giannini et al., 2022; Molnar et al., 2023; Rudiktyo et al., 2025). Progressive obstruction of transmitral flow increases left atrial pressure and promotes left atrial dilation, endothelial dysfunction, and atrial fibrillation, all of which contribute to systemic embolic risk. Even moderate MS may substantially increase the risk of ischemic stroke, particularly when accompanied by atrial fibrillation.

The diagnosis and severity assessment of mitral stenosis are primarily established by echocardiography, which enables evaluation of the mitral valve area, transmitral gradients, chamber dimensions, and associated valvular abnormalities (Manzo et al., 2023; Pala et al., 2026; Rolando et al., 2025; Sadeghpour & Behjati, 2026; Silva et al., 2024). Comprehensive cardiac evaluation is therefore important in younger patients presenting with ischemic stroke, particularly when conventional vascular risk factors are absent. We report a case of ischemic stroke as the initial clinical manifestation of previously undiagnosed moderate mitral stenosis with atrial fibrillation in a 40-year-old woman.

The urgency of addressing this diagnostic challenge is underscored by the potential for preventable recurrent stroke in younger patients, for whom the long-term consequences of neurological disability can be substantial. Secondary prevention strategies, particularly anticoagulation and appropriate management of the underlying valvular abnormality, depend on accurate identification of the stroke mechanism and characterization of the mitral valve disease (Martha et al., 2021; Liu et al., 2022). Current guidelines distinguish anticoagulation strategies according to the presence and etiology of clinically significant mitral stenosis; for example, vitamin K antagonist therapy remains recommended for atrial fibrillation associated with rheumatic mitral stenosis rather than direct oral anticoagulants. Decisions regarding percutaneous mitral balloon commissurotomy or surgical intervention likewise depend on disease severity, valve morphology, symptoms, and overall clinical context rather than etiology alone (Abu Rmilah et al., 2021; Guo et al., 2022). The difficulty of determining the etiology of MS when historical or morphological evidence is incomplete therefore represents an important diagnostic challenge in patients presenting with acute ischemic stroke.

The novelty of this case report lies in its deliberate diagnostic restraint regarding the etiology of mitral stenosis. Rather than assigning a rheumatic etiology in the absence of definitive morphological or historical evidence, the report emphasizes etiological classification based on demonstrable clinical and imaging findings. This approach is consistent with contemporary principles of diagnostic precision and evidence-based valvular heart disease assessment (Vahanian et al., 2021; Otto et al., 2021). Moreover, this report provides a clinically relevant illustration of the diagnostic reasoning required when a young patient presents with AF-associated ischemic stroke and echocardiographically confirmed MS, emphasizing systematic differential diagnosis and careful interpretation of imaging findings within the broader clinical context.

The purpose of this case report is to describe the clinical presentation, diagnostic evaluation, and multidisciplinary management of a 40-year-old woman who experienced cardioembolic ischemic stroke as the initial manifestation of previously undiagnosed moderate mitral stenosis with atrial fibrillation. It also highlights the diagnostic and therapeutic challenges associated with determining the etiology of MS when supporting historical or morphological evidence is incomplete. This report reinforces the importance of comprehensive cardiovascular assessment in younger patients with stroke, illustrates the systematic evaluation of competing stroke mechanisms, and emphasizes diagnostic precision in the classification of mitral stenosis when definite rheumatic features are absent. These considerations may improve

recognition of occult valvular heart disease as a potentially modifiable stroke risk factor and support more appropriate secondary prevention strategies.

METHOD

This case report was prepared in accordance with the CARE (CAse REport) guidelines. Data were obtained from the patient's medical records, including medical history, physical examination findings, laboratory results, electrocardiography, chest radiography, noncontrast head computed tomography, and transthoracic echocardiography performed at the referring hospital and tertiary referral center.

Clinical and diagnostic findings were reviewed by the treating neurology and cardiology teams, and the stroke subtype was classified according to the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) criteria.11 A narrative literature review was also conducted using PubMed/PMC, ScienceDirect, Frontiers, and StatPearls to contextualize the case within the existing evidence.

Written informed consent was obtained from the patient for publication of the case report and accompanying clinical images. All personally identifiable information was anonymized to maintain patient confidentiality.

RESULTS AND DISCUSSIONS

A 40-year-old woman was referred to a tertiary healthcare center for transthoracic echocardiographic evaluation after five days of hospitalization at a regional hospital with non-hemorrhagic stroke suspected secondary to atrial fibrillation and a concomitant urinary tract infection. On 4 April 2026, she had developed sudden slurred speech, inability to speak properly, and difficulty swallowing, with no prior documented history of cardiovascular disease, thyroid disorder, or previous stroke. On admission she was alert and oriented with stable hemodynamics: blood pressure 120/81 mmHg, heart rate 81 beats/minute, respiratory rate 20 breaths/minute, temperature 36.2°C, and oxygen saturation 99% on 4 L of supplemental oxygen. Peripheral perfusion was adequate, with warm extremities and a capillary refill time of less than 2 seconds. Neurological examination demonstrated central paresis of cranial nerves VII and XI. Pulmonary examination was normal, cardiac auscultation revealed normal heart sounds without murmur, peripheral pulses were palpable, and no peripheral edema was observed.

Electrocardiography showed an irregularly irregular rhythm, absence of distinct P waves in all leads, narrow QRS complexes, and a controlled ventricular response of 86 bpm—findings consistent with atrial fibrillation. Laboratory investigations, including complete blood count, urinalysis, electrolytes, and thyroid hormone profile, were all within normal limits, effectively excluding infectious, metabolic, and thyroid-related contributors to the arrhythmia. Transthoracic echocardiography revealed mild mitral regurgitation and moderate mitral stenosis with a mitral valve area of 1.44 cm^2, accompanied by left atrial dilatation. No intracardiac thrombus or vegetation was identified. Left ventricular systolic function was reduced (ejection fraction 44.4%) with anteroseptal hypokinesia, and right ventricular systolic function was likewise reduced (TAPSE 1.11 cm); left ventricular diastolic function could not be evaluated because of the atrial fibrillation. No left ventricular hypertrophy was found. A minimal pericardial effusion was also noted, measuring 0.739 cm laterally, 0.618 cm inferiorly,

and 0.586 cm posteriorly. Chest X-ray demonstrated cardiomegaly, and non-contrast head computed tomography revealed infarction involving the left frontal lobe and left corona radiata, together with additional lacunar infarctions in the midbrain and pons.

The patient's clinical course began on 4 April 2026 with the sudden onset of slurred speech, aphasia, and dysphagia. She was hospitalized at a district hospital from 6–11 April 2026 with a working diagnosis of non-hemorrhagic stroke, atrial fibrillation, and urinary tract infection, before being referred on 12 April 2026 for further echocardiographic evaluation at the tertiary center. Between 12–16 April 2026, her clinical condition stabilized, with gradual improvement in her speech difficulties, and she remained hemodynamically stable throughout this period. On 17 April 2026, she was discharged home for outpatient follow-up and routine monitoring. Throughout her hospitalization, the patient was jointly managed by cardiology and neurology specialists. Intravenous therapy consisted of Asering 500 cc every 12 hours, ceftriaxone 1 g every 12 hours, piracetam 3 g every 8 hours, citicoline 500 mg every 12 hours, mecobalamine 500 mcg every 12 hours, omeprazole 40 mg every 24 hours, and furosemide 20 mg every 12 hours. Oral therapy consisted of bisoprolol 1.25 mg twice daily, simvastatin 40 mg once daily, aspilet 80 mg once daily, clopidogrel 75 mg once daily, and spironolactone 25 mg once daily.

Several differential diagnoses were systematically considered using an etiologic approach to ischemic stroke based on the TOAST framework, which classifies ischemic stroke into cardioembolism, large artery atherosclerosis, small vessel occlusion, other determined etiology, and undetermined etiology, with a probable subtype requiring concordant clinical, neuroimaging, and diagnostic findings after competing mechanisms have been excluded.

The first and ultimately retained differential was cardioembolic ischemic stroke secondary to atrial fibrillation in the setting of moderate mitral stenosis. Theoretically, atrial fibrillation causes ineffective atrial contraction and promotes blood stasis, particularly within the left atrial appendage, thereby facilitating thrombus formation and systemic embolization; mitral stenosis further compounds this risk by increasing left atrial pressure, driving left atrial dilatation and atrial remodeling, which in turn perpetuates atrial fibrillation and heightens thromboembolic risk. This diagnosis was most strongly supported by the abrupt onset of focal neurological deficits, electrocardiographically confirmed atrial fibrillation, echocardiographically confirmed moderate mitral stenosis with a mitral valve area of 1.44 cm^2 and left atrial dilatation, and neuroimaging demonstrating cortical infarction in the left frontal lobe together with additional lesions. Notably, the absence of a visible intracardiac thrombus on transthoracic echocardiography did not refute a cardioembolic mechanism, since a thrombus may have already embolized prior to imaging or may be more reliably detected by transesophageal echocardiography. This diagnosis was therefore retained as the final working diagnosis; however, a rheumatic origin for the mitral stenosis was deliberately not assigned, as there was no documented history of rheumatic fever and no definite echocardiographic description of commissural fusion, leaflet thickening, or subvalvular involvement.

The second differential considered was large artery atherosclerotic ischemic stroke, which is typically supported by significant stenosis or occlusion—often exceeding 50%—of a relevant intracranial or extracranial artery, compatible vascular-territory imaging, vascular risk factors, carotid bruit, peripheral arterial disease, or recurrent transient ischemic symptoms within the same vascular territory. This diagnosis was not favored in the present case because

the patient was relatively young and had no documented history of hypertension, diabetes mellitus, dyslipidemia, carotid bruit, peripheral arterial disease, or significant vascular imaging abnormality, whereas atrial fibrillation with structural mitral valve disease and left atrial enlargement provided a more coherent embolic source; large artery atherosclerosis was therefore considered less likely as the primary etiology, although its definitive exclusion would require dedicated vascular imaging if clinically indicated.

The third differential was small vessel occlusion, or lacunar ischemic stroke, which typically produces lacunar infarcts within deep perforator territories and is classically associated with lacunar syndromes without cortical signs, commonly supported by hypertension and diabetes mellitus as risk factors, and in principle should occur in the absence of a major cardiac embolic source. Although the brain CT in this patient did show lacunar infarctions in the midbrain and pons, raising the possibility of concomitant small vessel disease, the patient also exhibited left frontal cortical involvement and aphasia, findings not adequately explained by a pure lacunar mechanism, while the presence of atrial fibrillation with moderate mitral stenosis and left atrial dilatation strongly supported a cardioembolic source instead. Lacunar disease was therefore judged to represent a concomitant imaging finding rather than the main cause of the index stroke.

The fourth differential was infective endocarditis with septic embolic stroke, a mechanism in which septic embolization—particularly from left-sided valvular vegetations—can cause ischemic stroke, with clinical suspicion typically heightened by fever, bacteremia, a new or changing cardiac murmur, peripheral stigmata, elevated inflammatory markers, and echocardiographic evidence of vegetation. This differential was substantially weakened in the present case by the absence of fever, absence of documented bacteremia or peripheral stigmata of infective endocarditis, entirely normal routine laboratory findings, and no vegetation identified on transthoracic echocardiography; infective endocarditis with septic embolization was therefore considered unlikely in the available clinical context.

The fifth differential was thromboembolic stroke due to isolated non-valvular atrial fibrillation, given that atrial fibrillation alone is a well-established cause of thromboembolic stroke, as irregular atrial activity increases the likelihood of atrial thrombus formation and subsequent embolization. Although atrial fibrillation was indeed present in this patient, the echocardiographic finding of moderate mitral stenosis with left atrial dilatation indicated an underlying structural valvular substrate; consequently, the mechanism was more accurately classified as cardioembolic stroke associated with atrial fibrillation in the setting of mitral stenosis, rather than isolated non-valvular atrial fibrillation, and this differential was not retained as the final diagnostic label.

The sixth and final differential comprised other determined etiologies relevant to ischemic stroke in young patients, including arterial dissection, vasculitis, or an underlying hypercoagulable state, all of which should be considered when common cardioembolic and atherosclerotic mechanisms are insufficient to explain a stroke occurring at a younger age. In this patient, the available history did not document neck trauma, neck pain, systemic inflammatory features, hematologic abnormalities, recurrent miscarriages, malignancy, or any other clinical clues suggestive of these less common etiologies. Because a plausible, high-probability cardiac embolic source had already been identified, these etiologies were not

considered the leading diagnosis, although further targeted testing would remain appropriate should recurrent events, atypical imaging findings, or new clinical data emerge in the future.

Taken together, the abrupt neurological presentation, the electrocardiographically confirmed atrial fibrillation, the echocardiographic demonstration of moderate mitral stenosis with left atrial dilatation, and the neuroimaging pattern of cortical and lacunar infarction converged most coherently on a single mechanism, allowing the competing differentials to be systematically excluded or deprioritized. The final working diagnosis was therefore cardioembolic ischemic stroke secondary to atrial fibrillation in the setting of previously undiagnosed moderate mitral stenosis, without a rheumatic label being assigned given the absence of supporting historical or morphological evidence.

This case highlights ischemic stroke as the initial manifestation of previously undiagnosed moderate mitral stenosis accompanied by atrial fibrillation in a relatively young woman without any previously established structural heart disease. It underscores the importance of comprehensive echocardiographic evaluation in young stroke patients presenting with atrial fibrillation, since such an evaluation may uncover a previously silent structural cardiac substrate that fundamentally changes both the understanding of the stroke mechanism and its subsequent long-term management. In addition, this report deliberately avoids assigning a rheumatic etiology to the mitral stenosis, precisely because no definitive morphological evidence—such as commissural fusion, leaflet thickening, or subvalvular involvement—or historical evidence of rheumatic fever was available to support such a label, illustrating the importance of diagnostic precision and appropriate restraint when the supporting evidence for a specific etiological subtype remains incomplete.

CONCLUSION

Previously undiagnosed moderate mitral stenosis with atrial fibrillation may initially manifest as a cardioembolic ischemic stroke, even in relatively young patients. Comprehensive cardiovascular evaluation, including echocardiography, is essential in patients presenting with ischemic stroke and newly diagnosed atrial fibrillation. Careful interpretation of echocardiographic findings is necessary to avoid unsupported assumptions regarding the etiology of mitral stenosis. Early multidisciplinary management may contribute to improved neurological and cardiovascular outcomes.

REFERENCE

Abd Elkareem, T. S., Ahmed, T. A., & Mohamed, L. A. (2023). Left atrial remodeling in patients with severe rheumatic mitral stenosis and sinus rhythm using two-dimensional and three-dimensional speckle tracking echocardiography. *Cardiology Research*, *14*(2), 142.

Abu Rmilah, A. A., Tahboub, M. A., Alkurashi, A. K., et al. (2021). Efficacy and safety of percutaneous mitral balloon valvotomy in patients with mitral stenosis: A systematic review and meta-analysis. *International Journal of Cardiology Heart & Vasculature, 33*, 100765. https://pmc.ncbi.nlm.nih.gov/articles/PMC8050729/

Autherith, M., Hauptmann, L., Palaparti, R., Bartko, P. E., & Nitsche, C. (2026). Myocardial remodeling in valvular heart disease: pathophysiology, multimodality imaging and treatment implications. *Heart Failure Reviews*, *31*(1), 89.

Campora, A., Lisi, M., Pastore, M. C., Mandoli, G. E., Ferrari Chen, Y. F., Pasquini, A., Rubboli, A., Henein, M. Y., & Cameli, M. (2024). Atrial fibrillation, atrial myopathy, and

thromboembolism: the additive value of echocardiography and possible new horizons for risk stratification. *Journal of Clinical Medicine*, *13*(13), 3921.

Cozza, E., Cito, V., Giumbini, G., Marsili, G., Barletta, M., Zannoni, J., Mainardi, C., Bedogni, F., & Tusa, M. (2026). Cardioembolic sources and stroke prevention: a systematic review. *Cardiovascular Ultrasound*, *24*(1), 8.

Giannini, C., Mazzola, M., Pugliese, N. R., & Petronio, A. S. (2022). Mitral valve stenosis in the current era: a changing landscape. *Journal of Cardiovascular Medicine*, *23*(11), 701–709.

Guo, G., Liang, S., Guan, Z., & Zhu, K. (2022). Effect of direct oral anticoagulants in patients with atrial fibrillation with mitral or aortic stenosis: A review. *Frontiers in Cardiovascular Medicine, 9*, 1070806. https://doi.org/10.3389/fcvm.2022.1070806

Ip, B., Au, L., Chan, A., Fan, F., Ip, V., Ma, S. H., Ma, K., Mok, V., Lau, A., & Leng, X. (2022). Evolving ischemic stroke subtypes in 15 years: a hospital-based observational study. *International Journal of Stroke*, *17*(4), 444–454.

Johansen, M. C., Chen, J., Schneider, A. L. C., Carlson, J., Haight, T., Lakshminarayan, K., Patole, S., Gottesman, R. F., Coresh, J., & Koton, S. (2023). Association between ischemic stroke subtype and stroke severity: the atherosclerosis risk in communities study. *Neurology*, *101*(9), e913–e921.

Kato, Y., Tsutsui, K., Nakano, S., Hayashi, T., & Suda, S. (2024). Cardioembolic stroke: past advancements, current challenges, and future directions. *International Journal of Molecular Sciences*, *25*(11), 5777.

Liu, B., Wang, Y., Liu, Y., Wu, P., & Li, T. (2022). Effects of percutaneous balloon mitral valvuloplasty in patients with mitral stenosis and atrial fibrillation: A systematic review and meta-analysis. *Acta Cardiologica, 77*(10), 890–899. https://doi.org/10.1080/00015385.2021.1989837

Manzo, R., Ilardi, F., Nappa, D., Mariani, A., Angellotti, D., Immobile Molaro, M., Sgherzi, G., Castiello, D. S., Simonetti, F., & Santoro, C. (2023). Echocardiographic evaluation of aortic stenosis: a comprehensive review. *Diagnostics*, *13*(15), 2527.

Martha, J. W., Pranata, R., Raffaelo, W. M., Wibowo, A., & Akbar, M. R. (2021). Direct acting oral anticoagulant vs warfarin in the prevention of thromboembolism in patients with non-valvular atrial fibrillation with valvular heart disease: A systematic review and meta-analysis. *Frontiers in Cardiovascular Medicine, 8*, 764356.

Molnar, A. A., Santa, A., Pasztor, D. T., & Merkely, B. (2023). Atrial cardiomyopathy in valvular heart disease: from molecular biology to clinical perspectives. *Cells*, *12*(13), 1796.

Myrmel, G., Waje-Andreassen, U., Pirinen, J., Sinisalo, J., Järvinen, V., Putaala, J., & Saeed, S. (2025). Multimodality cardiac imaging in ischemic stroke: Insights into the heart-brain interaction. *Cardiology*, *150*(5), 525–539.

Pala, B., Piscione, M., Gaudio, D., Gualtieri, P., Laudazi, M., Steffani, S., Garaci, F. G., Perrone, M. A., & Di Renzo, L. (2026). Mitral Stenosis in the Multimodality Imaging Era: Pitfalls, Stress Echocardiography, and Integrated Therapeutic Assessment. *Diagnostics*, *16*(14), 2285.

Perera, K. S., de Sa Boasquevisque, D., Rao-Melacini, P., Taylor, A., Cheng, A., Hankey, G. J., Lee, S., Fabregas, J. M., Ameriso, S. F., & Field, T. S. (2022). Evaluating rates of recurrent ischemic stroke among young adults with embolic stroke of undetermined source: the young ESUS longitudinal cohort study. *JAMA Neurology*, *79*(5), 450–458.

Rolando, M., Elmasry, N., Gobbi, F., Moreo, A., Ajmone Marsan, N., Carluccio, E., & Fortuni, F. (2025). Echocardiographic Assessment of Patients Undergoing Mitral Valve Repair. *Journal of Cardiovascular Development and Disease*, *12*(12), 498.

Rudiktyo, E., Teske, A. J., Yonas, E., Ambari, A. M., Cramer, M. J., Guglielmo, M., Semino,

T., Siswanto, B. B., Doevendans, P. A., & Soesanto, A. M. (2025). Upstream and downstream cardiovascular changes in rheumatic mitral stenosis: an update. *Journal of Clinical Medicine*, *14*(8), 2639.

Sadeghpour, A., & Behjati, M. (2026). Echocardiographic Assessment of Mitral Valve Disease. In *Case-Based Textbook of Echocardiography and Cardiac MRI* (pp. 263–283). Springer.

Silva, V. R., De Castro Faria, S. C., de Azevedo Figueiredo, F., Pantaleão, A. N., de Oliveira, M. A. R., & Nunes, M. C. P. (2024). Rheumatic mitral stenosis: Update in diagnosis and evaluation. *Current Treatment Options in Cardiovascular Medicine*, *26*(7), 207–220.