Infective Endocarditis Presenting as Recurrent Multifocal Ischemic Stroke in a Young Adult

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ABSTRACT:

Infective endocarditis (IE) is an uncommon but diagnostically challenging cause of cardioembolic stroke in young adults, particularly when neurological manifestations dominate the clinical presentation and no audible cardiac murmur is apparent.

Keywords: infective endocarditis; cardioembolic stroke; young adult; chronic haemodialysis; Corynebacterium; Duke criteria; multidrug resistance; echocardiography.

Introduction

Stroke in young adults (aged 18–50 years) is an increasingly recognised clinical challenge encompassing diverse aetiologies, including cardioembolism, arterial dissection, and prothrombotic disorders [1]. Infective endocarditis (IE), while uncommon in this age group, is a potentially fatal yet frequently overlooked cause of recurrent cardioembolic stroke. Neurological complications occur in approximately 25–70% of IE cases, with ischaemic stroke constituting the most frequent manifestation [2]. When recurrent stroke dominates the clinical picture in the absence of other classic features — such as an audible murmur — the cardiac source may be substantially delayed in recognition [3].

The management of IE complicated by recent major stroke presents an additional therapeutic dilemma. Current guidelines advocate early surgical intervention for IE with large vegetations and recurrent embolism [4]; however, cardiac surgery requiring cardiopulmonary bypass carries a substantially elevated risk of haemorrhagic transformation in the setting of acute or recent ischaemic stroke, creating a clinically important gap between guideline recommendations and realworld feasibility [5].

We report the case of a 37-year-old man receiving chronic HD who presented with recurrent multifocal embolic stroke as the primary manifestation of definite IE caused by MDR Corynebacterium amycolatum/striatum. The case is notable for the absence of murmur at presentation, the critical role of chronic HD and vascular access as a predisposing condition linking bacteraemia to valvular seeding, the rigorous application of the modified Duke Criteria in this context, and the fatal convergence of clinical constraints that precluded definitive surgical intervention.

2.Case Description

A 37-year-old man with type 2 diabetes mellitus, hypertension, chronic kidney disease on regular HD since May 2024, and a prior ischaemic stroke in June 2024 (with residual right-sided weakness) presented following sudden loss of consciousness while eating breakfast on 27 December 2024. His family reported that he had been conversing normally immediately before the event, without antecedent chest pain, palpitations, or new focal neurological symptoms. His regular medications included amlodipine, candesartan, glimepiride, metformin, allopurinol, and simvastatin.

On admission, the patient was confused and restless with a Glasgow Coma Scale score of 9 (E2V2M5). Vital signs showed fever (38.1°C), tachycardia (126 bpm), tachypnoea (22–24 breaths/min), and normotension (119/87 mmHg), with an oxygen saturation of 99% on a 12 L/min simple face mask. Notably, physical examination revealed no audible cardiac murmur. Conjunctival pallor, subcostal retractions, and bilateral coarse crepitations were present; there was no peripheral oedema, cyanosis, or jugular venous distension.

Laboratory investigations demonstrated severe anaemia (haemoglobin 6 g/dL), leukocytosis (12,540/μL), hypoalbuminaemia (2.91 g/dL), mild hyponatraemia (sodium 134 mmol/L), uraemia (blood urea nitrogen 59 mg/dL), and elevated serum creatinine (3.5 mg/dL). Chest radiography showed cardiomegaly with increased bronchovascular markings in the left hemithorax. Electrocardiography demonstrated sinus tachycardia with features of left ventricular hypertrophy.

Brain CT Findings

Brain CT demonstrated multifocal embolic ischaemic lesions in the left temporo-occipital, thalamic, and caudate regions, involving both the middle cerebral artery (MCA) and MCA/posterior cerebral artery (PCA) territories — a distribution strongly indicative of a cardioembolic source. Background chronic ischaemic changes were present bilaterally in the centrum semiovale, with an encephalomalacic cyst in the right occipital lobe attributable to the prior stroke. The multifocal embolic pattern in a young adult immediately prompted a dedicated cardiac evaluation.

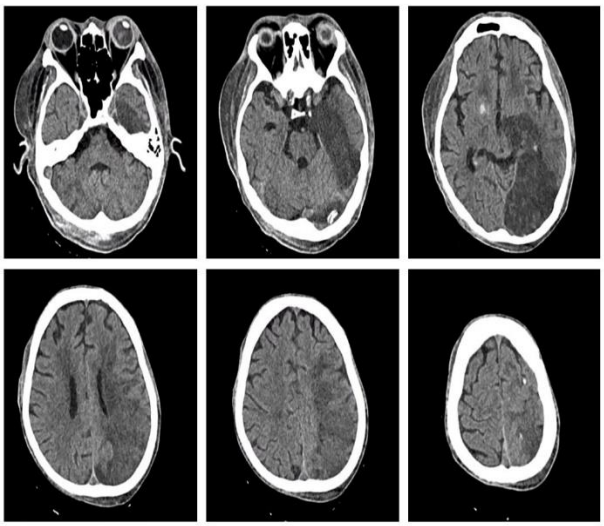

Figure 1. Brain CT demonstrating multifocal embolic ischaemic lesions in the left temporo-occipital, thalamic, and caudate regions (MCA and MCA/PCA territories), superimposed on chronic ischaemic changes in the bilateral centrum semiovale and an encephalomalacic cyst in the right occipital lobe.

Transthoracic Echocardiography Findings

TTE (Figure 2) revealed a 19 × 12 mm hyperechoic oscillating mass on the posterior mitral leaflet with Carpentier type II prolapse, producing moderate mitral regurgitation. Left ventricular systolic function was preserved (ejection fraction approximately 56%), with severe diastolic dysfunction (grade III) and left

atrial dilatation. Right ventricular function was impaired (TAPSE 1.5 cm), with elevated mean pulmonary artery pressure (32 mmHg), pulmonary capillary wedge pressure (18 mmHg), and systemic vascular resistance (1534 dynes·sec/cm^5).

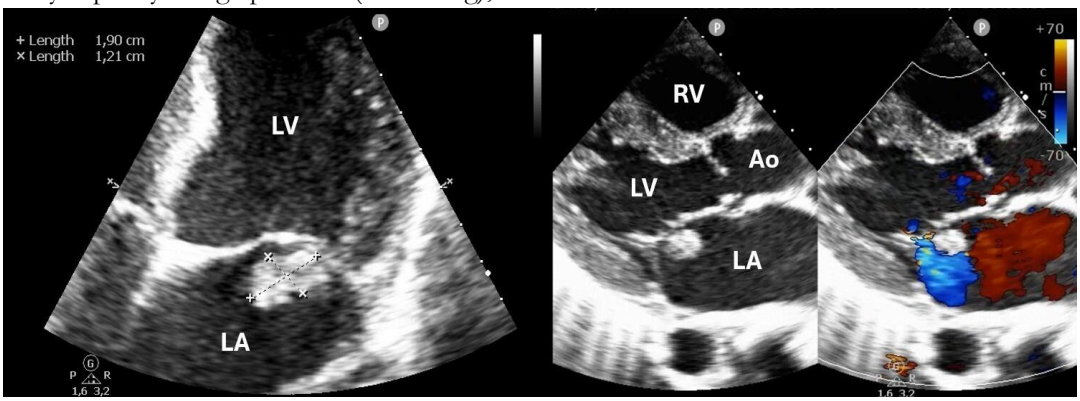

Figure 2. Transthoracic echocardiography (parasternal long-axis view) demonstrating a 19 × 12 mm hyperechoic oscillating vegetation on the posterior mitral leaflet (Carpentier type II prolapse) with moderate mitral regurgitation.

Blood cultures obtained prior to antibiotic initiation grew gram-positive bacilli identified as MDR *Corynebacterium amycolatum/striatum*. Susceptibility testing demonstrated resistance to vancomycin, ciprofloxacin, moxifloxacin, penicillin G, tetracycline, and clindamycin, with sensitivity exclusively to linezolid. Anaerobic culture was negative.

The clinical timeline from initiation of HD to the terminal hospitalisation is summarised in Table 1.

Table 1. Clinical Timeline

Timeline	Clinical Event
May 2024	Initiated regular haemodialysis (HD) for end-stage chronic kidney disease; repeated vascular access manipulation began, creating a sustained risk of healthcare-associated bacteraemia
June 2024	First ischaemic stroke; managed conservatively; residual right-sided weakness; no cardiac evaluation performed at that time
June–December 2024	Continued regular HD (3×/week via vascular access); presumed period of subclinical, recurrent bacteraemia with progressive endocardial seeding
27 Dec 2024 (Day 0 – Admission)	Sudden loss of consciousness while eating; GCS 9 (E2V2M5); fever 38.1°C; HR 126 bpm; no audible cardiac murmur; emergency hospitalisation
Day 0 – Brain CT	Multifocal embolic ischaemic lesions: left temporo-occipital, thalamic, caudate nuclei (MCA + MCA/PCA territories); chronic bilateral centrum semiovale lesions; encephalomalacic cyst right occipital lobe — cardioembolic source strongly suspected
Day 0–1 – Investigations	Hb 6 g/dL; WBC 12,540/μL; albumin 2.91 g/dL; Na 134 mmol/L; BUN 59 mg/dL; Cr 3.5 mg/dL; CXR: cardiomegaly + bronchovascular congestion left hemithorax; ECG: sinus tachycardia + LVH

Day 1 – TTE (Figure 2)	19 × 12 mm vegetation, posterior mitral leaflet, Carpentier type II prolapse, moderate MR; LVEF 56%; diastolic dysfunction grade III; TAPSE 1.5 cm; mPAP 32 mmHg; PCWP 18 mmHg; SVR 1534 dynes·sec/cm^5
Day 1–2 – Blood cultures	MDR *Corynebacterium amycolatum/striatum*; resistant to vancomycin, ciprofloxacin, moxifloxacin, penicillin G, tetracycline, clindamycin; sensitive only to linezolid
Day 1–2 – IE diagnosis (Duke Criteria)	DEFINITE IE: 2 Major (echocardiographic endocardial involvement + persistent MDR bacteraemia) + 2 Minor (HD/vascular access predisposition + fever ≥38°C). Empirical antibiotics commenced (ceftriaxone + ampicillin-sulbactam + gentamicin); PRBC transfusion planned; antiplatelet therapy deferred; antihypertensives withheld
Subsequent days	TEE not feasible (rapid clinical deterioration); cardiothoracic surgery consulted and declined — recent major stroke, ongoing sepsis, haemodynamic instability, multiple comorbidities; linezolid not available for empirical use
Final days	Progressive haemodynamic instability; multi-organ dysfunction; death during hospitalisation

Empirical antibiotic therapy was commenced with intravenous ceftriaxone, ampicillin-sulbactam, and gentamicin, pending culture and susceptibility results. Antihypertensive agents were withheld in the acute phase; packed red blood cell transfusion was planned to a target haemoglobin of at least 10 g/dL; and antiplatelet therapy was deferred in view of the risk of haemorrhagic transformation. Additional supportive measures included atorvastatin, paracetamol, vitamin B supplementation (B1, B6, B12), supplemental oxygen, and fluid restriction.

Transoesophageal echocardiography was planned for further characterisation of the vegetation and assessment of perivalvular extension, but could not be performed owing to rapid clinical deterioration. Cardiothoracic surgical consultation was obtained for consideration of mitral valve repair or replacement; however, operative intervention was deemed prohibitively high risk in the context of recent major stroke, uncontrolled infection, haemodynamic instability, and multiple comorbidities. Despite broad-spectrum antibiotics and multidisciplinary supportive care, the patient developed progressive haemodynamic instability and died during hospitalisation.

3. Results

Empirical intravenous therapy with ceftriaxone, ampicillinsulbactam, and gentamicin was commenced pending culture results. Susceptibility testing subsequently identified linezolid as the sole active agent given the extensive MDR profile. Transoesophageal echocardiography and cardiac surgery were not feasible owing to rapid clinical deterioration, recent major stroke, and multiple comorbidities. Despite multidisciplinary supportive care, the patient died during hospitalisation from progressive haemodynamic instability.

4. Discussion

This case illustrates four clinically distinct and interconnected features that collectively highlight the diagnostic and therapeutic complexity of IE presenting as recurrent embolic stroke in a young adult: (1) multifocal embolic stroke as the dominant and initial manifestation of IE in the absence of murmur; (2) chronic HD and vascular access as the key predisposing pathway linking healthcare-associated bacteraemia to *Corynebacterium* endocarditis; (3) the rigorous application of the modified Duke Criteria in this setting; and (4) the management deadlock arising from the convergence of high-risk IE and recent major ischaemic stroke.

Recurrent Embolic Stroke Without Murmur as the Presenting Feature of IE. Ischaemic

stroke is the neurological presentation of IE in approximately 3–10% of cases, and the absence of murmur — as observed in our patient — substantially delays recognition of the underlying cardiac source [2,6]. In a young adult without conventional atherosclerotic risk factors, a multifocal embolic pattern on brain imaging crossing multiple vascular territories should raise immediate suspicion for a structural cardiac source, most notably IE. Vegetation size and mitral valve location are the strongest echocardiographic predictors of embolism: vegetations exceeding 10 mm are associated with a more than tenfold increase in embolic risk, and mitral valve vegetations carry a higher rate of cerebral embolism than aortic valve lesions [6,7]. The 19 × 12 mm vegetation in this patient placed him at extreme embolic risk and explains the recurrent pattern of cerebral ischaemia.

Chronic Haemodialysis and Vascular Access as the Source of *Corynebacterium* Endocarditis. Chronic HD is a well-established independent risk factor for IE, primarily through the sustained bacteraemia risk associated with repeated vascular access manipulation — whether via arteriovenous fistulae or central venous catheters [7]. In this patient, seven months of thrice-weekly HD sessions preceded the terminal presentation, providing repeated opportunity for haematogenous seeding of a structurally compromised mitral valve. Corynebacterium amycolatum and Corynebacterium striatum are increasingly recognised causes of IE in HD-dependent patients, attributable to their capacity for biofilm formation on intravascular access devices [8]. Their isolation from blood cultures in a patient receiving chronic HD should therefore be regarded as a clinically significant finding rather than a contaminant. Importantly, the MDR profile — encompassing resistance to vancomycin, the empirical agent of choice in HD-associated bacteraemia — reflects the selective antimicrobial pressure of prolonged healthcare exposure and posed a critical barrier to effective treatment.

Application of the Modified Duke Criteria: Definite IE on Two Major and Two Minor Criteria. A structured application of the modified Duke Criteria [4,11] in this case establishes definite IE on firm evidential grounds. The patient satisfied **two major criteria**: (1) **Microbiological evidence** — persistently positive blood cultures with Corynebacterium amycolatum/striatum, an organism causally linked to healthcare-associated IE in the HD setting; and (2) **Evidence of endocardial involvement** — TTE demonstration of an oscillating intracardiac mass (19 × 12 mm) on the posterior mitral leaflet, consistent with vegetation. **Two minor criteria** were also fulfilled: (a) **predisposing condition** — chronic HD with vascular access, a well-recognised risk factor for IE; and (b) **fever** ≥38°C on presentation. Under the modified Duke framework, two major criteria alone are sufficient for definite IE classification, irrespective of whether TEE has been performed, provided TTE findings unambiguously demonstrate a vegetation [4,11]. In this case, the large vegetation size rendered TTE diagnostic. The absence of TEE is acknowledged as a limitation; nonetheless, it does not invalidate the definite IE classification, and the clinical context rendered TEE unfeasible.

The Management Deadlock: When Guidelines Cannot Be Followed. The 2023 ESC Guidelines for IE recommend early surgical intervention — ideally within 24–72 hours — for vegetations exceeding 10 mm with recurrent embolism, or in the presence of haemodynamic compromise [4]. This recommendation, however, is substantially constrained by recent major ischaemic stroke, as cardiopulmonary bypass with systemic anticoagulation markedly increases the risk of haemorrhagic transformation, particularly within two weeks of a large infarct [5,12]. In our patient, the simultaneous presence of recent extensive stroke, uncontrolled MDR sepsis, progressive haemodynamic deterioration, and severe comorbidity rendered cardiac surgery prohibitively dangerous. The patient thus occupied a position in which a guideline-level

indication for surgery existed but could not be safely executed — a recognised but unresolved management deadlock in the IE literature [5]. The absence of linezolid — the sole active antimicrobial agent — during the empirical treatment period further compromised the adequacy of medical management.

5. Conclusion

This case describes an uncommon but instructive presentation of definite IE as recurrent multifocal embolic stroke in a young adult without an audible murmur. Chronic HD constituted the key predisposing factor, establishing a direct pathophysiological link between vascular access–associated bacteraemia and MDR Corynebacterium endocarditis. In young adults presenting with a multifocal embolic stroke pattern, particularly those receiving chronic HD, a low threshold for TTE and blood cultures should be maintained regardless of murmur status. Systematic application of the modified Duke Criteria — explicitly classifying HD-related vascular access as a predisposing minor criterion alongside the major criteria of echocardiographic vegetation and persistent bacteraemia — enables early definite IE classification and informs management. The fatal outcome in this patient underscores how the compounding of MDR infection, large vegetation, recent major stroke, and multi-organ comorbidity can collectively foreclose both surgical and optimal medical options, highlighting an urgent need for evidence-based management algorithms in this high-risk scenario.

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