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Case Reports

Paradoxical Ischemic Stroke Following Streptokinase Fibrinolysis in STEMI: A Cardio-Cerebral Infarction Series from a Limited-Resource Setting

Wilbert Huang[1], Ryzufar Altarif Farsal[1], Harold J.M. Charlex[1], Hendrianus[1]

Abstract

Background: Ischemic stroke is a rare and paradoxical complication in patients with ST-segment Elevation Myocardial Infarction (STEMI) undergoing fibrinolysis. Definitive diagnosis is challenging in a resource-limited setting.

Case Illustration: We report two cases of STEMI treated with streptokinase fibrinolysis in a setting where Percutaneous Coronary Intervention (PCI) was unavailable. The first patient presented with an inferior myocardial infarction and developed neurological symptoms 4 hours after fibrinolysis, including acute focal neurological deficits. Brain Computed Tomography (CT) demonstrated an acute lacunar infarction in the left pons. The second patient presented with an anterolateral myocardial infarction and developed a seizure during fibrinolysis treatment, in which the treatment was immediately terminated. Brain CT revealed a lacunar infarct in the left hypothalamus and calcification of the right Middle Cerebral Artery (MCA). In both cases, the lacunar pattern of infarction raised suspicion of ischemic stroke due to embolization of microthrombi following fibrinolysis of coronary or intracardiac thrombi, with subsequent migration to the cerebral circulation. The first patient subsequently deteriorated and died due to catastrophic multisystem bleeding, whereas the second patient showed no progression of neurological deficits.

Conclusions: Ischemic stroke following fibrinolytic therapy in patients with STEMI is an uncommon but serious complication that requires high clinical vigilance. Early recognition and individualized multidisciplinary management are essential to minimize neurological sequelae and optimize overall outcomes.

[1]RSUD Natuna, Natuna, Riau Islands, Indonesia.

Correspondence:
Wilbert Huang,

RSUD Natuna, Natuna, Riau Islands, Indonesia.

Email: wilberthuang67@gmail.com

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Introduction

Acute Coronary Syndrome (ACS), encompassing ST-segment Elevation Myocardial Infarction (STEMI) and Non–ST-segment Elevation Acute Coronary Syndrome (NSTE-ACS), is a life-threatening condition resulting from acute occlusion of a culprit coronary artery. Current guidelines recommend immediate revascularization with Percutaneous Coronary Intervention (PCI) for patients with STEMI and for selected high-risk patients with NSTE-ACS. However, when timely PCI is unavailable or anticipated to be delayed beyond 120 minutes, an alternative reperfusion approach is the pharmaco-invasive strategy using fibrinolytic therapy.[1]

The efficacy of fibrinolysis is well established in current guidelines, with intracranial hemorrhage recognized as its most feared complication, consistent with its mechanism of action.[2] Nonetheless, paradoxical ischemic stroke following fibrinolytic therapy has also been reported in several case reports and series.[3–5] This phenomenon aligns with the cardio-cerebral infarction concept, in which myocardial and cerebral ischemia may share overlapping pathophysiological mechanisms.[6] In this case report, we describe two patients who presented to the emergency department within six months with a paradoxical complication of ischemic stroke following fibrinolytic therapy for STEMI, rather than the more commonly anticipated hemorrhagic stroke. Hemorrhagic stroke is a well-recognized complication of fibrinolytic therapy, whereas ischemic stroke in this setting is less frequently reported and discussed. Therefore, this case highlights an uncommon but clinically important complication that warrants greater awareness. In addition, this case reflects the challenges encountered in resource-limited settings, where determining the definitive etiology of such events may be constrained by limited diagnostic availability. Furthermore, the use of conventional fibrinolytic therapy (streptokinase), due to limited access to newer reperfusion agents, may influence both management decisions and clinical outcomes.

Case Illustration

Case I

The first case was a 68-year-old man who presented to the emergency department with typical anginal chest pain of 4-hour duration. He described a persistent retrosternal chest tightness without radiation, which did not improve with rest. His medical history was significant for percutaneous coronary intervention (PCI) performed in 2015; however, detailed procedural information was unavailable, and the patient was unable to recall specific details. He reported good adherence to his prescribed medications and regular follow-up with a cardiologist. Comorbid conditions included hypertension and dyslipidemia, both of which were reportedly well controlled on medical therapy.

On initial assessment, his vital signs revealed profound hypotension and bradycardia, with a blood pressure of 59/40 mmHg, heart rate of 45 beats per minute, respiratory rate of 22 breaths per minute, and oxygen saturation of 88% on room air. Despite hypotension and mild oxygen desaturation, the patient remained alert and communicative, without signs of respiratory distress. He denied dyspnea and had no respiratory symptoms suggestive of pulmonary pathology, such as cough. Physical examination demonstrated adequate peripheral perfusion, evidenced by warm extremities, strong peripheral pulses, and a capillary refill time of less than 2 seconds. Pulmonary auscultation was clear, with no crackles or rales, and cardiac examination revealed no audible murmurs or additional heart sounds.

A 12-lead Electrocardiogram (ECG) was obtained within 10 minutes of presentation and demonstrated ST-segment elevation in leads II, III, and aVF, with reciprocal ST-segment depression in all precordial leads, consistent with an inferior STEMI with total Atrioventricular (AV) block (Figure 1). Right-sided (V3R–V4R) and posterior (V7–V9) leads were not recorded at the time of initial evaluation. Laboratory investigations revealed leukocytosis (17.73 x 103/ ul), elevated liver enzymes (SGOT 73 U/L and SGPT 61 U/L), an increased Creatine Kinase–Myocardial Band (CK–MB) level of 46.3 U/L, and reduced renal function with an estimated glomerular filtration rate of 49 mL/min/1.73 m^2. Chest radiography demonstrated fibrotic infiltrates in the left upper lung field, suggestive of prior or active pulmonary tuberculosis.

Based on the initial findings, the working diagnosis was inferior STEMI with suspected concomitant pulmonary tuberculosis. The patient was promptly administered loading doses of aspirin (325 mg) and clopidogrel (400 mg). Hemodynamic support was initiated with dobutamine and norepinephrine, titrated to maintain a mean arterial pressure >65 mmHg. Supplemental oxygen therapy was also provided to correct the observed oxygen desaturation.

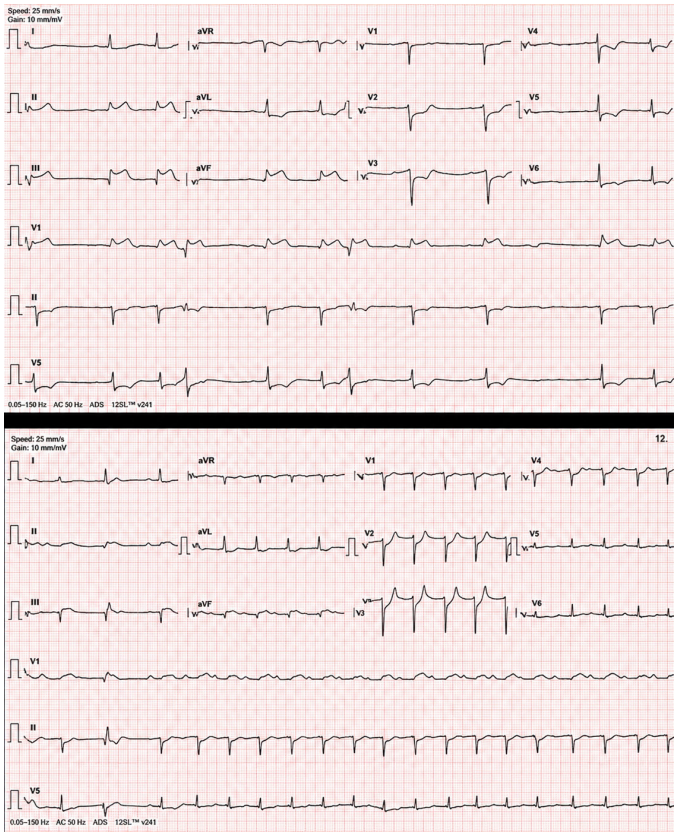


Figure 1. ECG findings showing ST segment elevation in inferior leads (above) and post-fibrinolytic ST segment resolution (below).

Given the limited-resource setting and lack of access to a PCI-capable center, the patient was managed with fibrinolytic therapy using streptokinase at a dose of 1.5 million units. A thorough evaluation for absolute and relative contraindications to fibrinolysis was conducted, and none were identified. Fibrinolysis was successful, as evidenced by greater than 50% resolution of ST-segment elevation on follow-up ECG (Figure 1), and the procedure was uneventful, with no clinical or laboratory evidence of bleeding complications. Adjunctive pharmacotherapy included fondaparinux 2.5 mg administered subcutaneously, bisoprolol 1.25 mg, and atorvastatin 40 mg.

Four hours after fibrinolysis, the patient acutely developed right facial droop, right-sided hemiparesis, and slurred speech while undergoing a routine dressing change in the emergency department. The neurological deficit had a sudden onset and was witnessed by medical staff. On focused neurological examination, the patient was alert but dysarthric, with central right facial weakness and decreased motor strength in the right upper and lower extremities, consistent with an acute brainstem or pontine syndrome. There were no signs of projectile vomiting or meningeal irritation. Given the recent administration of fibrinolytic and anticoagulant therapy, an intracranial hemorrhage was initially suspected as a potential complication.

An urgent non-contrast head Computed Tomography (CT) scan was conducted 25 minutes following stroke onset, which showed an acute lacunar infarction in the left pons without evidence of intracranial hemorrhage (Figure 2). These findings were consistent with an acute ischemic stroke involving the perforating branches of the basilar artery. A neurology consultation was obtained, and conservative management was recommended given the recent fibrinolytic exposure. Mannitol 125 mL every 6 hours was advised if the systolic blood pressure exceeded 110 mmHg. The patient was subsequently transferred to the Intensive Care Unit (ICU) for close neurological and hemodynamic monitoring.

During ICU observation, the patient did not exhibit progression of neurological deficits, deterioration of consciousness, or clinical signs of raised intracranial pressure. The neurological status remained stable, supporting the diagnosis of an acute ischemic lacunar stroke occurring as a rare complication following fibrinolytic therapy.

Seven hours after fibrinolysis, the patient acutely developed respiratory distress with oxygen



Figure 2. Head CT scan of the patient revealing a lacunar infarct in the left pons.

saturation declining from 95% to 83% despite 12 L/min oxygen via a non-rebreather mask, necessitating endotracheal intubation. During preoxygenation, he developed massive hemoptysis and hematemesis, consistent with alveolar and gastrointestinal bleeding, with an estimated blood loss of approximately 2000 mL and a hemoglobin decrease from 15.6 to 13.4 g/dL. Chest radiography demonstrated alveolar pulmonary edema. Hemostatic therapy with tranexamic acid, vitamin K, and a high-dose proton pump inhibitor was initiated, and red blood cell transfusion was planned. However, two hours later, the patient developed ventricular tachycardia and subsequently died.

Case II

The second case involved a 45-year-old man who presented to the emergency department with a 3-hour history of chest pain. The pain was typical of angina, described as retrosternal chest tightness radiating to the left arm and shoulder, and was not relieved by rest. The patient reported recurrent exertional chest discomfort over the preceding five years, which had progressively worsened but had never been medically evaluated. He was not receiving any regular medical therapy. His medical history was notable for long-standing, poorly controlled hypertension due to non-adherence to treatment. He denied a history of dyslipidemia, stroke, or a family history of cardiovascular disease. He reported heavy cigarette smoking since the age of 15 years. At presentation, the patient was not dyspneic and had no focal neurological deficits.

On initial physical examination, the patient was hypertensive with a blood pressure of 191/121 mmHg, heart rate of 78 beats/min, respiratory rate of 24 breaths/min, and oxygen saturation of 98% on room air. Cardiopulmonary examination was unremarkable, with vesicular breath sounds and no rhonchi or wheezing in both lung fields. Electrocardiography demonstrated ST-segment elevation in leads V2–V5, I, and aVL, ST-segment depression in lead III, and pathological Q waves in leads I and aVL. (Figure 3) Laboratory evaluation revealed elevated hemoglobin (16.5 g/dL) and hematocrit (50.4%), marked leukocytosis (50.4 × $10^3/\mu L$), and elevated CK–MB (49.3 U/L). The chest X-ray also showed no abnormalities.

A working diagnosis of hypertensive emergency complicated by anterolateral STEMI was established. Initial management for ACS included loading doses of aspirin 300 mg and clopidogrel 300 mg. Blood pressure was acutely managed with sublingual captopril 25 mg, reducing it to 162/103 mmHg, followed by maintenance therapy with candesartan 8 mg once daily and bisoprolol 2.5 mg once daily. The patient was subsequently deemed eligible for fibrinolytic therapy with streptokinase, as no absolute or relative contraindications were identified.

However, shortly after initiation of fibrinolysis, following administration of 18 mL (270,000 units) of streptokinase, the patient developed a generalized seizure lasting approximately two minutes. After a five-minute interval, a second seizure occurred, lasting one minute, followed by non-projectile vomiting. Fibrinolytic therapy was immediately discontinued, and the patient was transferred for an emergency non-contrast head CT scan to exclude

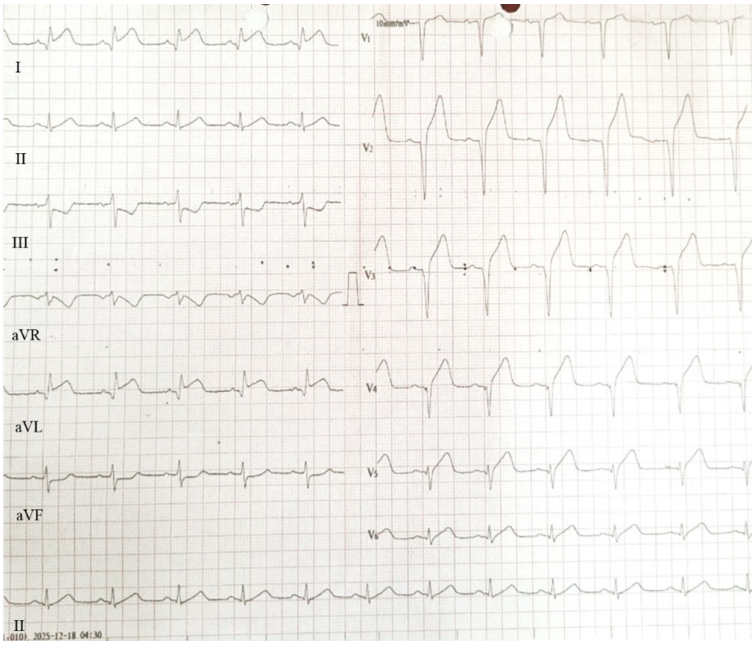


Figure 3. ECG findings of the patient.

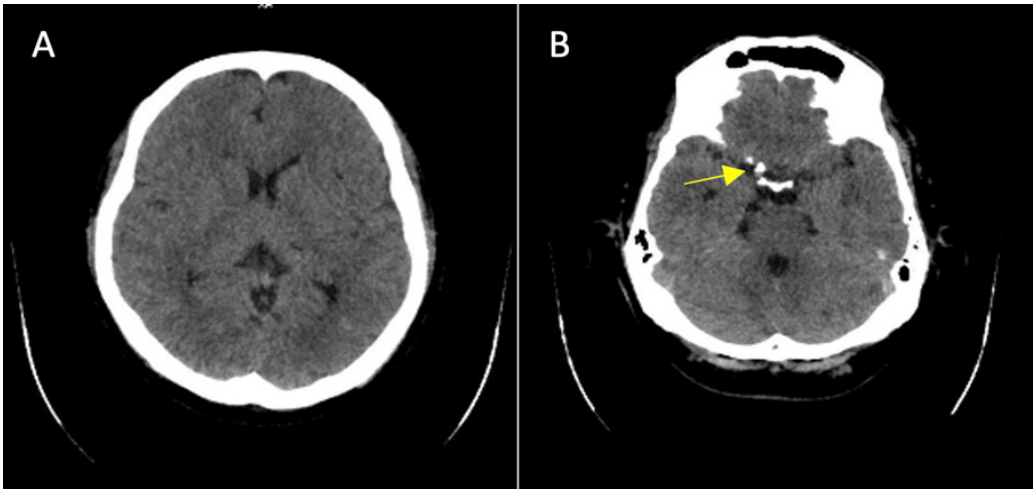


Figure 4. (a) Lacunar infarct in the left thalamus; (b) Calcification of the right MCA.

intracranial hemorrhage as a complication of fibrinolysis.

A non-contrast head CT scan was performed approximately 15 minutes after seizure onset, revealing a lacunar infarct in the left thalamus and calcification of the right MCA (Figure 4). Neurology consultation was obtained, and intravenous phenytoin 100 mg was administered as a bolus dose for seizure management, with concurrent discontinuation of streptokinase. The patient was subsequently transferred to the ICU for close neurological and hemodynamic monitoring. During the first 12 hours of ICU admission, the patient developed central left facial weakness and decreased motor strength in the left upper and lower extremities. The patient was treated with heparin 500 IU twice daily, aspirin 80 mg once daily, and phenytoin 100 mg three times daily for the remainder of the ICU stay.

One day after ICU management, the patient was clinically stable and transferred to the general ward. Medical therapy was continued, during which no progression of neurological deficits was observed, and the patient's chest pain completely resolved. The patient remained hemodynamically stable throughout the hospital course and was discharged on the fourth day of hospitalization. At discharge, the prescribed medications included aspirin 100 mg,

clopidogrel 75 mg, atorvastatin 40 mg, candesartan 8 mg, bisoprolol 2.5 mg once daily, and phenytoin 100 mg three times daily.

Discussion

In this case report, we describe a rare and paradoxical complication in patients with STEMI undergoing streptokinase fibrinolysis. Instead of developing the more well-recognized complication of intracranial hemorrhage, both patients subsequently experienced ischemic stroke with focal neurological deficits.

The concurrent occurrence of myocardial infarction and cerebral infarction, regardless of the sequence, is referred to as cardio-cerebral infarction. This entity has been reported to occur in approximately 0.34–0.5% of cases, although its true incidence remains uncertain due to the lack of uniform diagnostic criteria.[6] The simultaneous presence of cardiac and cerebral ischemia poses significant therapeutic challenges, as current guidelines for acute myocardial infarction and ischemic stroke recommend differing reperfusion strategies, including variations in fibrinolytic agent selection, dosing, and the use of anticoagulation.[7-8] Consequently, individualized management based on the severity and clinical dominance of each ischemic territory is essential. In our series, neither ischemic stroke case was eligible for mechanical thrombectomy due to the anatomical location of the occlusion. Consequently, management was directed toward the primary ischemic presentation, which in both cases involved the coronary circulation. As a result, both patients received similar treatment strategies focused on the acute coronary event.

Several pathophysiological mechanisms have been proposed to explain the occurrence of concurrent ischemic events, including atherogenic, neurogenic, cardiogenic, and inflammatory pathways.[6] In our case report, we postulate that ischemic stroke resulted from embolization of microthrombi following fibrinolysis of coronary or intracardiac thrombi, with subsequent migration to the cerebral circulation. This hypothesis is supported by the development of lacunar infarcts in both patients, consistent with occlusion of small-caliber cerebral vessels by microemboli. The temporal relationship in the first case—occurring shortly after patient movement during a dressing change—further supports a possible embolic mechanism. However, these findings will require a larger observational study to determine the pathophysiologic mechanism of lacunar infarcts caused by fibrinolysis-related microembolism.

However, echocardiographic evaluation was not performed in these cases, limiting our ability to confirm the presence of left ventricular thrombus. Left ventricular thrombus formation is most commonly observed in patients with large anterior or apical myocardial infarctions, reduced ejection fraction, and regional wall motion abnormalities leading to blood stasis.[9] In our reports, only the second patient had an anterolateral infarction, partially supporting this mechanism. Although atrial fibrillation is another recognized risk factor for cardioembolic events, neither patient demonstrated atrial fibrillation.

It is also important to consider the possibility of paradoxical coronary embolism, in which a thrombus originating from the venous system—most commonly from deep vein thrombosis—bypasses the pulmonary circulation through an intracardiac shunt, such as a patent foramen ovale, and enters the systemic circulation.[10] This mechanism may result in simultaneous or sequential embolic events, including STEMI and acute ischemic stroke. However, in our cases, neither patient had a prior history or clinical evidence suggestive of venous thromboembolic disease. Furthermore, a comprehensive echocardiographic evaluation to assess for intracardiac shunting was not performed, and therefore, the presence of a patent foramen ovale or other right-to-left shunt could not be confirmed or excluded.

An alternative mechanism may involve the heightened catecholamine surge following acute myocardial infarction, combined with an intense systemic inflammatory response—as evidenced by leukocytosis in our patients—which may destabilize pre-existing atherosclerotic plaques in the cerebral circulation, leading to acute ischemic stroke. Additionally, fibrinolytic therapy itself may paradoxically promote a prothrombotic state, probably through streptokinase-related inflammatory reactions, potentially mediated by free plasmin activation of contact factors, platelet activation, and acceleration of the coagulation cascade.[11] In our first case, we presented an even more dilemmatic condition where, post-fibrinolytic, the patient further developed catastrophic bleeding that further complicates the priority of managing the bleeding and thrombosis. In addition, with the current patient condition, further studies are needed to understand the individualized bleeding risk in patients undergoing fibrinolysis to prevent

such catastrophic bleeding events. In this scenario, it remains uncertain whether modification of anticoagulant or antiplatelet dosing would be warranted, given the limited available evidence to guide management in such complex presentations. In our case, the catastrophic event occurred abruptly, and in the absence of a specific reversal strategy, immediate control of the bleeding was not feasible.

In the second case, the incidence of seizure due to cerebral hypoperfusion following ventricular arrhythmia also could not be ruled out, and the short onset since the event and the CT scan could conceal the underlying pathologies. Unfortunately, a follow-up CT scan could not be performed because of financial limitations. Interestingly, although intracerebral hemorrhage as the cause of the seizure had been excluded by CT imaging—suggesting that fibrinolytic therapy could potentially be resumed under strict monitoring—this approach was not pursued in our case. Instead, a more conservative strategy was deliberately chosen to minimize any further risk.

Cardio-cerebral infarction is associated with increased morbidity and poorer prognosis. Although the mainstay therapies for myocardial and cerebral ischemia overlap, several aspects—particularly the choice, timing, and dosing of antithrombotic and anticoagulant therapy—require careful consideration, and currently no guidelines provide specific recommendations for the management of cardio-cerebral infarction. Clinicians should remain vigilant for paradoxical ischemic stroke following fibrinolytic therapy in STEMI patients, as new-onset neurological symptoms may be subtle and easily overlooked in the acute cardiac setting. Early neurological assessment and prompt neuroimaging are essential to differentiate ischemic from hemorrhagic events and to guide subsequent therapy. Additionally, the use of biomarkers such as high-sensitivity troponin and D-dimer can improve patient care by enabling earlier disease detection.[12] Improved identification of clinical, inflammatory, and procedural predictors of cardio-cerebral infarction would enable better risk–benefit stratification, closer post-fibrinolysis monitoring, and more individualized, safer treatment strategies. Importantly, the potential role of fibrinolytic therapy in precipitating cardio-cerebral infarction warrants particular attention, as paradoxical events have been reported in both directions—where fibrinolysis for acute myocardial infarction may be followed by ischemic stroke, and conversely, fibrinolysis administered for ischemic stroke may precipitate acute myocardial infarction, as described in several case reports.[13–16] In this case, we are also limited by the absence of magnetic resonance imaging as a gold standard for lacunar stroke diagnosis, and no autopsy was done to confirm the cause of death in the first case.

Conclusion

Ischemic stroke following fibrinolytic therapy in patients with STEMI can occur, and early recognition and individualized management are crucial given the lack of specific guideline recommendations.

List of Abbreviations

AV	Atrioventricular
ACS	Acute Coronary Syndrome
CK-MB	Creatine Kinase–Myocardial Band
CT	Computed Tomography
ECG	Electrocardiogram
ICU	Intensive Care Unit
MCA	Middle Cerebral Artery
NSTE-ACS	Non–ST-segment Elevation Acute Coronary Syndrome
PCI	Percutaneous Coronary Intervention
SGOT	Serum Glutamic Oxaloacetic Transaminase
SGPT	Serum Glutamic Pyruvic Transaminase
STEMI	ST-segment Elevation Myocardial Infarction

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

WH and RAF contributed to the conceptualization of the case report, patient management, data collection, literature review, and preparation of the original manuscript draft. HJC and H contributed to data interpretation, literature review, and critical revision of the manuscript. H contributed to clinical supervision, interpretation of the findings, and critical revision of the manuscript for important intellectual content. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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Generative AI and AI-Assisted Technologies in the Writing Process
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