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Acute Ischemic Stroke with Acute Myocardial Infarction, Neurologist or Cardiologist First? A Rarely Reported Case

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ABSTRACT

A 51-year-old man presented to the hospital with right-sided paresis, reported within 1 hour of onset, and a National Institutes of Health Stroke Scale score of 4. Initial evaluation revealed elevated blood pressure, a heart rate of 120 bpm, and electrocardiography indicating sinus rhythm with anteroseptal wall infarction. Computed tomography confirmed cerebral infarction, particularly in the temporal lobe, and elevated Troponin I levels, suggesting cardiac involvement. The neurologist initiated intravenous Actilyse and Manitol, and the patient was admitted to the intensive care unit with a subsequent referral to a cardiologist. Cardiologist-approved interventions included Furosemide and Morphine due to emerging left chest discomfort. The patient experienced somnolence, leading to additional prescriptions of subcutaneous Diviti and oral Carvedilol. By the third day, the patient regained consciousness, and chest discomfort alleviated. Days four to five marked minimal chest discomfort, with improved right-sided muscle strength. Physiotherapy was initiated, and echocardiography revealed ischemic cardiomyopathy with left atrial and ventricular dilatation, anteroseptal and apical akinesia, mild mitral regurgitation, and a 23% ejection fraction. Thrombus in the left ventricular apex was identified. After day 8, the patient was discharged home.

KEYWORDS: Cardio-cerebral Infarction, AIS, AMI

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INTRODUCTION

Acute ischemic stroke is a critical neurological event characterized by the sudden interruption of blood flow to a specific region of the brain, resulting in the rapid onset of focal neurological deficits. A common etiology is the occlusion of cerebral arteries, often attributable to thrombotic or embolic events. The urgency in addressing acute ischemic stroke lies in the potential for irreversible damage to brain tissue and subsequent functional impairment. Time-sensitive interventions, such as the administration of tissue plasminogen activator (tPA), have demonstrated efficacy in restoring blood flow and minimizing neurological deficits when administered within a specific therapeutic window.[1]

In the realm of acute ischemic stroke management, endovascular procedures have emerged as a pivotal therapeutic option. Mechanical thrombectomy, in particular, involves the physical removal of obstructing clots from cerebral vessels, allowing for the rapid restoration of blood flow. Recent clinical trials, such as the HERMES collaboration, have demonstrated the superiority of endovascular therapy, particularly when combined with intravenous thrombolysis, in achieving better functional outcomes for eligible patients. This paradigm shift in stroke care underscores the significance of a comprehensive approach that combines both pharmacological and interventional strategies to optimize patient outcomes.[2]

Acute myocardial infarction (AMI), commonly known as a heart attack, is a life-threatening cardiovascular event resulting from the sudden obstruction of blood flow to a portion of the heart muscle. Ischemia and subsequent

necrosis occur due to the occlusion of a coronary artery, often caused by atherosclerotic plaque rupture or erosion. Timely reperfusion therapy is crucial to salvage viable myocardium and improve patient outcomes. Primary percutaneous coronary intervention (PCI), a catheter-based procedure to restore blood flow, is considered the gold standard for AMI management, offering superior results compared to fibrinolytic therapy, particularly when administered within the recommended time frames.[3]

Beyond the acute phase of myocardial infarction, secondary prevention strategies play a crucial role in reducing the risk of recurrent cardiovascular events. Medical therapies, including antiplatelet agents, beta-blockers, angiotensin-converting enzyme inhibitors, and statins, form the cornerstone of long-term management post-AMI. These medications target various pathways involved in the progression of atherosclerosis, hypertension, and ventricular remodeling, thereby mitigating the risk of subsequent cardiac events.[4]

Acute ischemic stroke (AIS) and acute myocardial infarction (AMI) are both life-threatening medical conditions, bringing lethal prognosis if not addressed immediately. One-third of patients with ischaemic stroke with no cardiac history have more than 50% coronary stenosis and 3% are at risk of developing MI within a year.[5] Cardio-cerebral infarction (CCI) term can be used to describe the simultaneous incident of AIS and AMI.[6] However, the approach to immediate management of CCI remains unclear.

CASE REPORT

A 51-year-old-man was brought to hospital complaining right-sided paresis without chest pain within 1 hour onset. The National Institutes of Health Stroke Scale scored 4. On initial evaluation, blood pressure was 140/90 mmHg, heart rate (HR) was 120 bpm with electrocardiography (ECG) showing : sinus rhythm, HR 120 bpm and anteroseptal wall infarction.

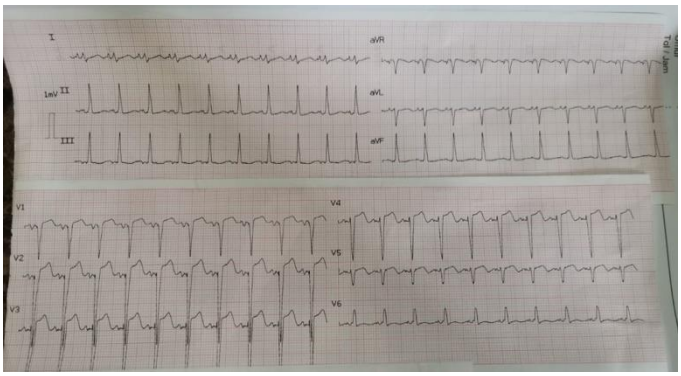

Figure 1. Initial ECG Showing Sinus Tachycardia with Anteroseptal Wall Infarction

CCI was confirmed by non-contrast head computed tomography (CT) scan, showing temporal lobe infarction , and rising Troponin I level in 2.97 ng/mL (refference value = <0.3 ng/mL) on blood test.

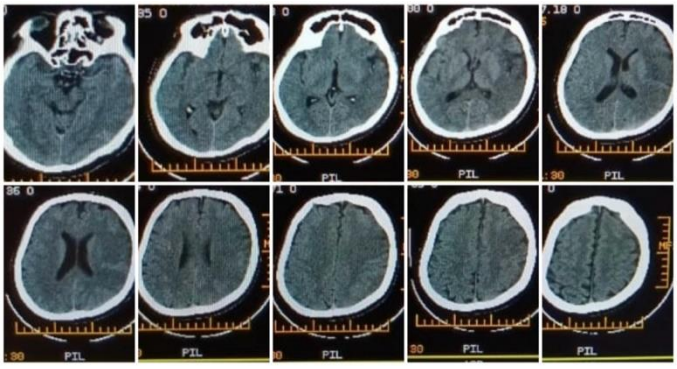

Figure 2. Initial Head Non-Contrast CT Scan Showed Temporal Lobe Infarction

Neurologist was contacted first due to the chief complain, ordering : intravenous Actilyse and Manitol; oral Aspilet, Clopidogrel and Atorvastatin; and intensive care unit admission with interspecialty refferal to cardiologist. Cardiologist accepted the therapy given by neurologist without additional advice. After 3 hours observaation, left chest discomfort started emerging, hence Furosemid and Morfin were administered by cardiologist. Patient dropped to somnolen state on second day admission leading to subcutaneous Diviti and oral Carvilol prescriptions by cardiologist. On third day patient regained his consciousness and chest discomfort were relieved. On day four-to-five, chest dicomfort was minimal, with right-sided muscle strength was almost 5 on both upper-andlower limbs. Physioteraphy was scheduled to train muscle strength and echocardiography was performed showing ischaemic cardiomyopathy : left atrium

(LA) and left ventricular (LV) dilatation; anteroseptal and apical akinesia; mild mitral regurgitation ; and 23% ejection fraction (EF). Thrombus was also found on left ventricular apical explaining why CCI could happen. After day 8, patient was allowed to be sent home

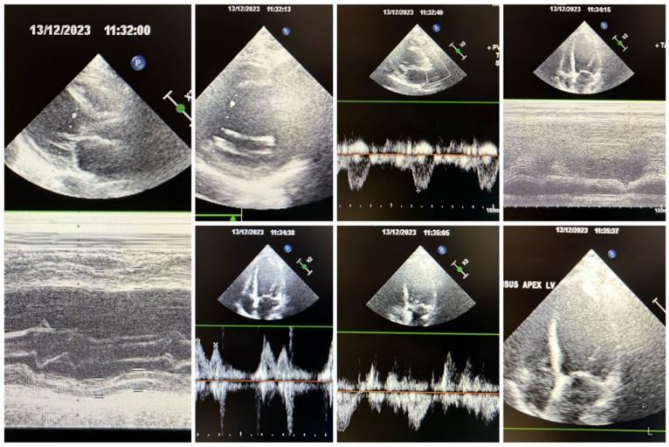

Figure 3. Echocardiography Performed on Fifth Day Observation Showing Left Ventricular Apical Thrombus; and Mild Mitral Regurgitation

DISCUSSION

Delirium emerges as a prevalent and serious complication in older patients following hospital admission, with stroke identified as a recognized predisposing factor. Despite its significance, the association between stroke and delirium remains inadequately explored, yielding conflicting results with prevalence estimates ranging widely. The aetiology of delirium post-stroke remains poorly understood, adding complexity to its management. The absence of a consensus on the optimal screening tool further complicates efforts to detect delirium in the post-stroke setting. Moreover, specific stroke types, such as intracerebral haemorrhage and total anterior circulation infarction (TACI), may be more prone to precipitating delirium. The intriguing association between delirium and specific brain lesions, such as those in the thalamus and caudate nucleus, as suggested by case reports, adds to the nuanced understanding of this complex phenomenon.[7]

Thrombus formation is a critical factor in the pathogenesis of both myocardial infarction (MI) and acute cerebral infarction (ACI), contributing to the interruption of blood flow in coronary arteries and cerebral vessels, respectively. In the context of MI, a thrombus, often initiated by the rupture of an atherosclerotic plaque, can obstruct the coronary arteries, leading to inadequate blood supply to the heart muscle. This deprivation of oxygen and nutrients results in myocardial cell death, manifesting as a heart attack. Similarly, in ACI, thrombus formation plays a pivotal role in the blockage of cerebral arteries, causing a sudden cessation of blood flow to specific regions of the brain.[8]

The timely administration of intravenous tissue plasminogen activator (IV tPA) has demonstrated effectiveness in the treatment of myocardial infarction when administered early after the onset of symptoms. While some recommendations caution against the use of IV tPA in acute stroke patients with a recent history of acute myocardial infarction (AMI), considering it a relative contraindication, it is emphasized that IV tPA continues to be the primary therapeutic approach for patients presenting with acute ischemic stroke (AIS) and coexisting AMI.[9]

Both unfractionated heparin and low molecular weight heparins (LMWHs), such as Enoxaparin, exert their anticoagulant effects by inhibiting both factor Xa and thrombin, critical components of the coagulation cascade. However, it is important to note that LMWHs, including Enoxaparin, exhibit a relatively lower impact on thrombin compared to unfractionated heparin. Fondaparinux, on the other hand, is more selective in its mechanism, specifically targeting factor Xa coagulation factors. This succinctly summarizes the distinct anticoagulant properties of these medications, providing clinicians with valuable information for tailoring anticoagulation strategies based on individual patient needs and clinical scenarios.[10]

The comparative analysis between Enoxaparin and Fondaparinux reveals a notable advantage for Fondaparinux in terms of safety, as it was associated with a significantly lower incidence of bleeding episodes when compared to Enoxaparin. Moreover, the observed reduction in severe bleeding on day 9 further supports the favorable bleeding profile of Fondaparinux.[11]

In patients with acute myocardial infarction, oral treatment with beta-blockers is recommended when they have reduced ejection fraction (≤40%) or heart failure unless contraindicated, as this patient has a reduced ejection fraction, beta-blocker was administered.[12]

CONCLUSION

In conclusion, the co-occurrence of acute myocardial infarction (AMI) and cerebral ischemic events poses a

complex medical challenge, as both conditions share common risk factors and underlying mechanisms, often leading to adverse outcomes. Thrombus formation, a critical factor in the pathophysiology of both AMI and cerebral ischemia, underscores the interconnected nature of these cardiovascular and cerebrovascular events. The simultaneous occurrence of AMI and cerebral ischemic events places individuals at a heightened risk for complications, necessitating a comprehensive and multidisciplinary approach to management. Strategies focused on early detection, prompt intervention, and aggressive risk factor modification are crucial in mitigating the impact of these dual pathologies. Collaborative efforts between cardiology and neurology specialties are essential for optimizing patient care, improving outcomes, and reducing the overall burden of cardiovascular and cerebrovascular diseases.

CONFLICT OF INTEREST AND AUTHOR CONTRIBUTIONS

I affirm that there are no conflicts of interest related to the submission of this manuscript. Each author has transparently disclosed any financial or personal associations with individuals or organizations that might potentially influence the research, analysis, or interpretation of the data presented in this case report. The collaborative efforts of all authors were integral to the conceptualization, design, analysis, and interpretation of the data for this case report.

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