



A Case Report on Concurrent Stroke and Myocardial Infarction

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Abstract

Concurrent myocardial infarction and acute cerebral infarction is a rare and poorly studied phenomenon that presents a challenge to treat as both conditions are life threatening with narrow therapeutic windows. We present the case of a 70 year old female who presented with symptoms concerning for stroke. However, an electrocardiogram revealed she was also having an acute myocardial infarction. The decision was made to treat the stroke with intravenous tissue plasminogen activator. Unfortunately, the patient ultimately decompensated and died. There are many proposed etiologies of this phenomenon including cardiac thrombi leading to concurrent acute myocardial infarction and cerebral infarction, a primary myocardial infarction leading to a cerebral infarction, and a primary cerebral infarction leading to an acute myocardial infarction. Treatment options include simultaneous mechanical thrombectomy and percutaneous coronary intervention in a cardiac catheterization laboratory, or treating with the intravenous tissue plasminogen activator dose for a cerebral infarction and then potentially also proceeding to percutaneous coronary intervention. Ultimately, the management of this situation will depend on the patient's specific situation including the type of stroke, the extent of irreversible tissue damage, and the hospital's available resources. A randomized controlled study is difficult because of the rare occurrence of both presentations and a systematic review of the available literature may provide physicians with better insight as to how to approach a simultaneous acute myocardial infarction and acute cerebral infarction.

Keywords

Myocardial Infarction, Cerebral Infarction, Stroke, ST Elevation Myocardial Infarction

Abbreviations

Myocardial Infarction (MI), Percutaneous Coronary Intervention (PCI), Tissue Plasminogen Activator (tPA), Electrocardiogram (ECG), Computerized Tomography (CT), Intravenous (IV), Cardio-Cerebral Infarction (CCI), Left Ventricular (LV), Neurogenic Stunning Myocardium (NSM), High-Sensitivity (HS), American Heart Association/American Stroke Association (AHA/ASA)

Introduction

Cardiovascular and cerebrovascular disease are major causes of death and disability globally. The simultaneous presentation of an acute myocardial infarction (MI) and acute cerebral infarction is a rare phenomenon with an incidence of 0.009% [1] and therefore has not been well studied. Both are life-threatening conditions with narrow therapeutic windows. The dilemma in the foregoing clinical situations is determining which condition should receive initial therapy. If the patient is taken to the catheterization lab for evaluation and revascularization to treat the MI first, this will delay the management of the stroke and possibly place the patient outside of the window of thrombolysis. Conversely, if the patient undergoes mechanical thrombectomy for stroke, delaying the treatment of the MI, the latter event may progress to further myocardial damage, arrhythmias, heart failure, or shock. Additionally, dual antiplatelet therapy following percutaneous coronary intervention (PCI) for the treatment of MI would further increase the risk of cerebral hemorrhage after administration of tissue plasminogen activator (tPA) for the stroke. We describe a 70 year old female patient who presented with both an acute MI and acute cerebral infarction.

Case Summary

A 70 year old woman presented to the emergency department with slurred speech, generalized weakness, and confusion. No other symptoms were noted. Baseline vital signs were stable. Telestroke was consulted and the National Institutes of Health Stroke Scale (NIHSS) score was 2. Electrocardiogram (ECG) showed ST-segment elevation in leads V2-V6. Computerized tomography (CT) scan of the head revealed no evidence of hemorrhage or other cerebral pathology. Troponin was elevated above 6,000. The diagnosis was stroke and acute anterior MI. Cardiology was consulted. There was concern regarding which condition to treat first. Vital signs remained stable. Ultimately, intravenous (IV) tPA was initiated and speech improved. Unfortunately the patient decompensated and died despite receiving resuscitative measures.

Discussion

Pathogenesis:

There are multiple mechanisms that may explain the simultaneous presentation of cardio-cerebral infarction (CCI). Three processes have been proposed: (1) concurrent CCI, (2) MI leading to cerebral infarction, and (3) cerebral infarction leading to MI.

1. Cerebral and myocardial infarction may occur simultaneously in cases in which intracardiac thrombi may form with subsequent emboli dislodged to both coronary and cerebral arteries simultaneously, as confirmed by pathologic studies of retrieved thrombi in one report [2]. The Framingham Study showed that atrial fibrillation is associated with stroke by causing stasis of the blood which leads to thrombus formation which can embolize to the brain [3]. Coronary artery embolism is considered an important nonatherosclerotic cause of MI, with a reported prevalence of 2.9% in acute MIs with atrial fibrillation considered as the most common cause [4]. Intracardiac thrombogenesis may occur from left ventricular (LV) failure and subsequent left atrial dilation leading to atrial fibrillation, or from LV dysfunction producing LV segmental akinesia and dyskinesia [2,5-9]. Right ventricular thrombus or deep vein thrombosis may also give rise to paradoxical emboli through a right-to-left intracardiac shunt such as a patent foramen ovale [10,11]. Other causes of coronary embolism include infective endocarditis, valvular heart disease, iatrogenic causes such as cardiac catheterization, and cardiomyopathy [4,12-15]. Another possible mechanism of CCI is thrombogenesis secondary to coronary vasospasm. Passage of vasoactive chemicals, including serotonin in patients with carcinoid heart disease may be transmitted through a patent foramen ovale inducing coronary vasospasm resulting in a prothrombotic state and cerebrovascular injury [10,16,17]. Another differential diagnosis to consider as a cause of CCI is type A aortic dissection, which may lead to proximal extension to the coronary ostia and distal extension to the carotid or vertebral and basilar arteries [18-20]. Extension of the dissection to the coronary ostia can cause MI, and extension to the carotid or vertebral and basilar

arteries may lead to acute cerebral infarction. The International Registry of Acute Aortic Dissection showed that the incidence of either MI or cerebrovascular accident found concurrently with Type A dissection was 4.8% and 6.1%, respectively [21].

2. Acute cardiac events may result in subsequent cerebrovascular events. A right ventricular infarct complicated by severe hypotension may lead to acute cerebral infarction by reduction in cerebral perfusion [18,22-26]. MI complicated by cardiogenic shock is managed with revascularization and percutaneous mechanical circulatory support. Cardiogenic shock due to MI and clinical management are both associated with an increased risk of stroke and higher in-hospital mortality [27]. The presence of significant carotid artery disease with impaired cerebral autoregulation distal to the occlusion may be unable to adequately compensate an acute drop in blood pressure, such as in cardiogenic shock. The presence of high-grade carotid stenosis in cardiac surgery with cardiopulmonary bypass has been shown to increase risk of stroke in the perioperative period and likely associated with exhausted autoregulatory reserve [28].
3. Central autonomic dysregulation of cardiac activity from acute cerebral infarction may also lead to concurrent MI. Neurogenic myocardial injury, such as neurogenic stunning myocardium (NSM), describes a neurological event, such as cerebral infarction, complicated by autonomic neural dysregulation with cardiac manifestations [29]. Intracardiac neuronal release of catecholamines may alter cardiac function and result in myocardial cell damage [30]. Histopathological studies have shown a difference in patients with thrombotic MI compared to MI secondary to NSM. Coagulation necrosis is the pathologic hallmark of thrombotic MI [31]. MI secondary catecholamine-induced cardiac injury, in contrast, occurs as transient myofibrillar degeneration or myocytolysis surrounding epicardial nerves in the subendocardium [32,33].

A stroke in the left insular cortex, supplied by perforating branches of the middle cerebral artery,

increases the risk of adverse cardiac outcomes, including MI, compared to acute cerebral infarction in other cortical regions [34,35]. The insular cortex has been implicated in NSM or in the autonomic modulation of cardiac activity [32,34,36,37]. Patients with lesions in the insular cortex have a higher rise in norepinephrine and are associated with increased frequency of arrhythmias [38].

Management:

Evaluation Considerations:

Since MI may occur concurrently or, more frequently, as a complication of acute cerebral infarction, current guidelines by the American Heart Association (AHA) recommend a cardiovascular examination comprising baseline ECG and cardiac troponin assessment in all patients presenting with acute cerebral infarction [39]. Cardiac examination focuses on identifying concurrent MI, valvular abnormalities, or irregular rhythms as a cause of a cardioembolic event [39]. Aortic dissection is a rare event but is crucial to detect as a cause of CCI, since thrombolytic therapy may worsen outcomes [19,40,41]. Predictors of aortic dissection may include upper extremity pulse discrepancy, widened mediastinum, and direct evidence by CT angiography, transesophageal echocardiogram, or carotid ultrasonography [20,21,41,42].

Patients with acute cerebral infarction commonly present with an increase in serum cardiac troponins [43-47]. Sustained increases in cardiac troponin levels or ECG changes in patients presenting with acute cerebral infarction may be due to brain-heart axis dysregulation or other chronic comorbidities. A study measuring serial high-sensitivity (HS) cardiac troponin in patients with acute ischemic stroke found that 60% of the patients with elevated HS cardiac troponin I had sustained elevations and the remainder experienced a dynamic change suggesting that the elevated cardiac troponin levels in patients with acute cerebral infarction may not always be due to an acute MI [44]. Similarly, electrocardiographic changes in acute cerebral infarction include QT-segment prolongation, ST-segment depression, T wave abnormalities, and prominent U waves [46]. These ECG changes have been found on initial presentation for acute cerebral

infarction in one study while another reported these ECG changes to be new [45-47]. Although elevations in HS troponin and abnormalities on ECG in patients with ischemic stroke may not indicate concurrent MI or result from acute cerebral infarction, they may also indicate nonischemic cardiac injury and both of these findings are associated with a worse prognosis [35,48-50]. Cardiovascular risk assessment may be useful to tailor management according to the patient's clinical profile.

Treatment Considerations:

The American Heart Association/American Stroke Association (AHA/ASA) 2018 guidelines recommend (Class I) mechanical thrombectomy for patients with large vessel occlusions [39] in acute cerebral infarction but most hospitals do not have the capability to perform this procedure. However, if the institution has a cardiac catheterization laboratory, it is possible for interventional cardiologists to work closely with neurologists to perform the mechanical thrombectomy [51]. One study demonstrated restoration of blood flow in 80% of patients who underwent mechanical thrombectomy by interventional cardiologists for acute cerebral infarction [51]. This may be a setting of opportunity for neurologists and cardiologists to explore a team effort in a cardiac catheterization laboratory to perform a mechanical thrombectomy and PCI for CCI.

tPA is the standard agent of thrombolysis for stroke and is a therapeutic agent for MI as well [39]. IV-tPA alone has been considered for treatment of CCI with favorable outcomes in cases cited in literature [52]. One consideration is the difference in dosage of tPA for treating acute cerebral infarction and MI. For MI, AHA guidelines recommend a bolus of 15 mg, then an infusion of 0.75 mg/kg (not exceeding 50 mg) over a 30 minute period, then 0.5 mg/kg (not exceeding 35 mg) over the following 60 minutes [53]. The total dose should not exceed 100 mg. The AHA/ASA 2018 guidelines recommend 0.9 mg/kg over 60 minutes for acute cerebral infarction, with 10% of the dose given during the first minute [39]. The total tPA dose should not exceed 90 mg. Administering a higher dose than recommended in acute cerebral infarction carries the risk of cerebral hemorrhagic transformation. The

AHA/ASA 2018 Guidelines for the Early Management of Patients with Acute Ischemic Stroke recommends (Class II) administering the dose for acute cerebral infarction and then proceeding to PCI. Treating with IV-tPA does not reduce the benefit of PCI [54,55].

Conclusion

The patient with an acute myocardial infarction presenting with acute cerebral infarction is a challenging situation in the emergency department as it is unknown if myocardial infarction or ischemic stroke should be treated first. The approach to management will depend on the type of stroke, extent of tissue affected, and the resources available at the hospital. Treatment must be individualized for each patient. For large cerebral vessel occlusions, mechanical thrombectomy and PCI in the same cardiac catheterization laboratory may be a solution. Another possible approach is administering the stroke-appropriate dose of tPA and then performing PCI. IV-tPA alone for simultaneous treatment may be considered as well, although the optimal dose is unknown. This dilemma in choosing which to treat first presents an opportunity for research. Future histopathologic studies may elucidate the cause of concurrent ischemia and provide insight into how early intervention may best prevent the rapid succession of the other. Because of the rarity of CCI, it is difficult to design a study to determine the best course of management. A systematic review of the case reports and case studies may provide physicians with better insight as to how to approach this challenge.

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Conflict of Interests

All authors have read and approved the final version of the manuscript. The authors have no conflicts of

interest to declare.

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