The Clot Strikes Thrice: Case Report of a Patient with 3 Concurrent Embolic events

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Abstract-

Purpose: Concurrent cerebral and systemic embolism are unusual occurrences. Acute myocardial infarction secondary to coronary embolism are particularly rare with only isolated reports in the literature.

Case Report: A 49-year old Chinese man presented with right posterior cerebral artery infarction to our hospital. He developed chest pain and ST-elevation on ECG the next morning. Urgent cardiac catheterization showed a right coronary artery as well as a right profunda femoris artery occlusion. The cause of these 3 concurrent emboli was a large mobile left ventricular thrombus seen on echocardiography. Urgent anticoagulation was initiated, and the patient returned home after 1 month of rehabilitation with no further ischemic events.

Conclusion: In selected patients with high embolic risks, urgent anticoagulation after acute ischemic stroke can be a possible treatment option.

Key Words: cardioembolic stroke, intracardiac thrombus, anticoagulation

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INTRODUCTION

Cardioembolic strokes are a common cause of ischemic strokes, accounting for 15-30%[(1,2)] of cerebral infarctions worldwide. The most frequently encountered cause is atrial fibrillation. Embolism from intracardiac thrombi is less common but well recognized. This report is of a patient with concurrent emboli to right posterior cerebral artery, the right coronary and the right profunda femoris arteries originating from an intracardiac thrombus. The patient was successfully treated with urgent anticoagulation.

CASE REPORT

A 49-year old man with a history of hypertension, dyslipidemia and non-ischemic cardiomyopathy presented with complaints of left-sided weakness and numbness upon waking up. Neurological examination revealed a left homonymous hemianopia, left hemiparesis with power 4+/5 on the MRC scale in both upper and lower limbs and reduced sensation on the left face, upper and lower limbs. Initial CT scan of the brain done in emergency did not reveal any acute lesions. The patient was treated for an acute ischemic stroke and given a loading dose of 300mg

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of clopidogrel. An MRI performed later on the same day confirmed the presence of a right posterior cerebral artery (PCA) territory infarction on diffusion weighted imaging and an abrupt cut-off of the right PCA on magnetic resonance angiography (Fig. 1).

The next morning, patient complained of a sudden onset of left-sided chest pain associated with dyspnea. The inferior myocardial infarction (MI) was not picked up immediately due to the existing left bundle branch block. However, a comparison with the baseline ECG done upon admission showed new elevation of the ST segments in leads II,II and AVF (Fig. 2).

The patient was sent for acute coronary intervention. Access was obtained through the right femoral artery. During angiography, a distal right coronary artery (RCA) occlusion was seen. After aspiration thrombectomy the underlying RCA was found to be free of atherosclerotic disease. (Fig. 3) The cause of the coronary occlusion was deemed to be embolic and coronary stenting was not performed. In addition, a right femoral angiogram performed during the procedure showed evidence of a thrombus occluding the profunda femoris that was clinically asymptomatic (Fig. 4). This was also thought to be embolic in nature.

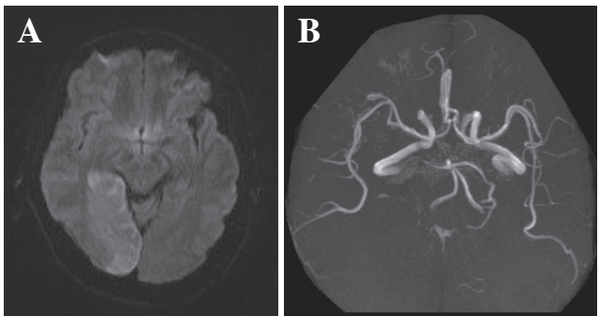

Figure 1. Magnetic Resonance Imaging of the brain performed on D1, 12 hours after admission (A) Diffusion weighted MRI showing a right posterior cerebral artery territory infarction (B) Magnetic resonance angiography showed abrupt cut-off in the right PCA.

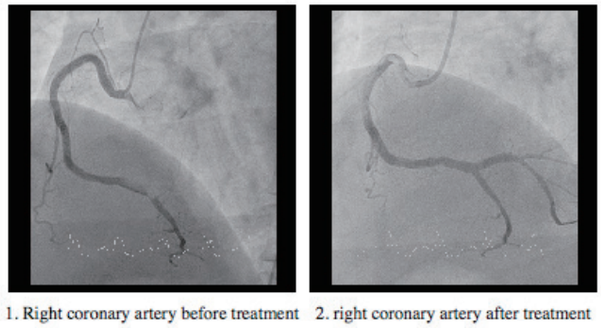

Figure 3. Coronary angiography on D2. (1) Pre-treatment angiogram with distal right coronary occlusion. (2) Post-aspiration angiogram showing normal underlying right coronary artery with no significant atherosclerotic disease.

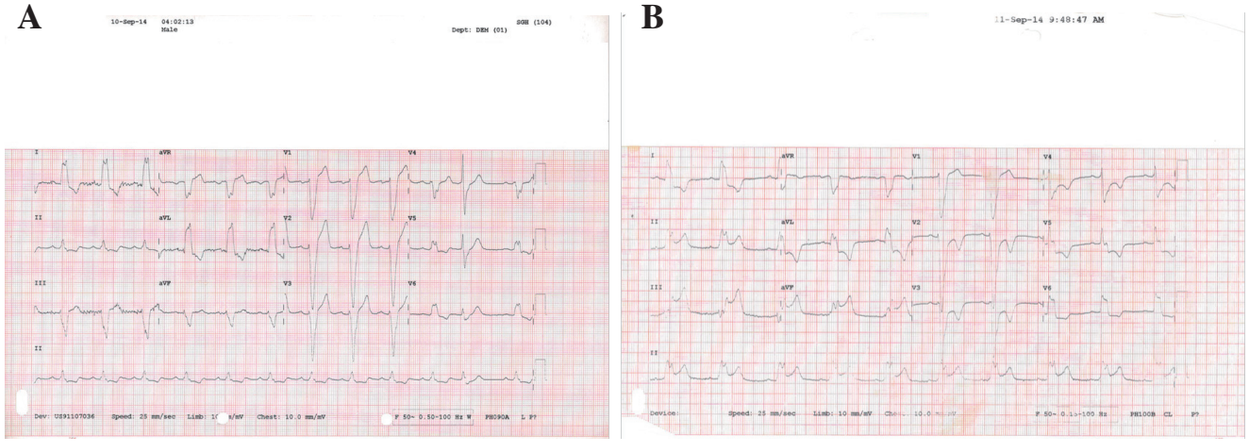

Figure 2. (A) ECG done on D1 of admission showing the pre-existing left bundle branch block (B) ECG on D2 of admission during complaints of chest pain with new ST- segment elevations in leads II, III and AVF

Post procedure, he was transferred to high dependency and started on intravenous heparin for acute treatment of acute MI. Activated partial thromboplastin time(APTT) was measured 6 hourly and titrated to 60 seconds. The source of these emboli was a large mobile left ventricular (LV) thrombus confirmed on transthoracic echocardiography performed the next day (Fig. 5).

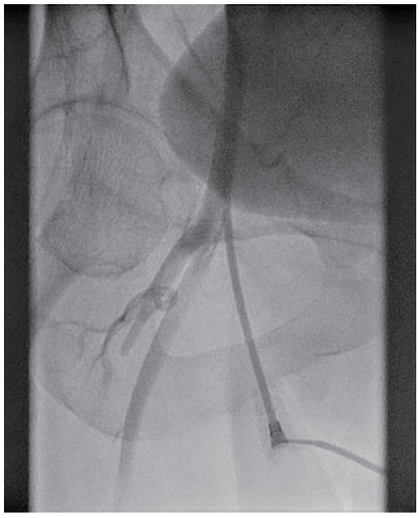

Figure 4. Angiogram of the right femoral artery performed during acute coronary intervention on D2 . "Cut-off" in the profunda femoris artery.

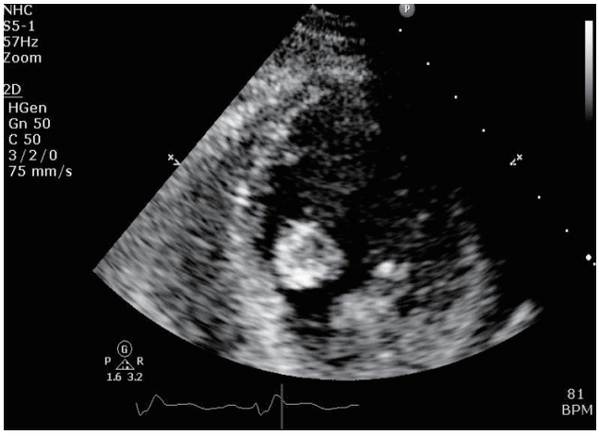

Figure 5. 2D echocardiogram performed on D3 (1 day after myocardial infarction). The hyperechoeic 0.9 x 3.2 cm left ventricular thrombus is seen near the bottom.

Markedly impaired left ventricular systolic function with a LV ejection fraction of 23% was noted. Blood stasis within the ventricle was deemed to be the cause of thrombus formation, no further tests for hypercoagulability was performed. Warfarin was initiated on D3 of admission and therapeutic INR of 2.22 was achieved on D7, upon which heparin was stopped.

The patient remained well for the rest of his hospital stay, there was no neurological deterioration to suggest hemorrhagic conversion and no further ischemic episodes. He was transferred out of high dependency on D6 of myocardial infarction. After 1 month of rehabilitation at our inpatient facility, he regained full functional independence and returned home.

DISCUSSION

Cardioembolism is a commonly encountered stroke etiology. However, myocardial infarctions as a result of emboli to the coronary arteries are much rarer[3]. The exact prevalence of this unknown, as published literature is confined mostly to case reports[4–7]. Concurrent cerebral and systemic embolism is also rare, registry-based studies have reported this in 2% of stroke patients[8]. Though there has been a previous report of concurrent cerebral and coronary embolism[9], this is the first case to our knowledge of a patient having 3 concurrent embolic events.

The unusual presentation of this patient entailed a unique set of problems for his management

There are no randomized trials for the treatment of intracardiac thrombi, and its optimal treatment remains to be elucidated. There are previous reports and series of documenting the successful treatment of patients with high-risk LV thrombi using surgical thrombectomy[10] and intravenous fibrinolysis[11,12]. These were however not considered for our patient due to the increased anesthetic risks posed by the recent stroke and the risk of hemorrhagic transformation.

In the absence of clear class I evidence, the American College of Chest Physicians recommend anticoagulation for patients with left ventricular (LV) thrombi in their 2012 guideline on Antithrombotic Therapy and Prevention of

Thrombosis[13]. This is mainly supported by observational studies which have showed reduced embolic risks for patients treated with warfarin[14,15].

However, routine use of urgent anticoagulation for acute ischemic stroke had largely fallen out of favor among neurologists. Clinical trials with heparin and heparinoids mainly showed either no benefit in reducing stroke recurrence or functional outcome or that the benefit was negated by the increase in hemorrhagic complications[16,17].

Sandercock in his metananlysis[18] of 23748 patients in 24 trials found acute anticoagulation to reduce the risk of early ischemic stroke recurrence (OR 0.76 95% CI 0.65-0.88) but this was offset by an increase in symptomatic intracranial hemorrhage (OR 2.55 95% CI 1.95-3.33). There was no net benefit in overall stroke reduction (OR 0.97 95% CI 0.85-1.11).

The possibility that certain high-risk patients may still benefit from acute anticoagulation was explored by Whiteley[19] in his metanalysis. Pooled individual patient data from the 5 largest trials of urgent anticoagulation in acute ischemic stroke was analysed according to predicted hemorrhagic and thrombotic risk. The study turned out to be negative, a specific group of "high-risk" patients who could potentially benefit from urgent anticoagulation was not found.

The optimal timing for initiating anticoagulation in post-stroke patients is also unclear as there are no prospective data. Given this, some clinicians wait for 2 weeks before starting anticoagulation. Others use a 1-3-6-12 rule, starting anticoagulation after 1 day for patients with transient ischemic attacks, 3 days for small infarcts, 6 days for moderate size strokes and 2 weeks or more for large infarcts[20].

The infarct in our patient is of at least moderate size. However, the presence of the large mobile thrombus and the 3 concurrent embolic events placed him at a very high risk for yet another embolic event. As such, after careful consideration and discussion with the patient, anticoagulation was initiated acutely (within 48h) using intravenous heparin, and the patient under close monitoring in high dependency. It was fortunate that coronary stenting was not required for our patient as this would have necessitated the use of antiplatelet agents which would further increase the hemorrhagic risk.

In summary, this report examined and discussed an extremely rare case of a patient with cardioembolic stroke secondary to an intracardiac thrombus with concurrent coronary and systemic embolism, whom we treated successfully with anticoagulation. Clinical trials and meta-analysis of ischemic stroke patients have failed to demonstrate unequivocal benefits from urgent anticoagulation. However, it should be noted that a selected subset of ischemic stroke patients may benefit from this form of therapy. Patients with mobile cardiac thrombi and recurrent embolism are very also rare and insufficiently represented in these studies. Additionally, the likelihood of a randomized trial specific for the treatment of these patients is also low given their small numbers. The successful treatment of this patient with urgent anticoagulation suggests that this could be a possible option for future patients of similarly high embolic risk.

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