Acute Myocardial Infarction Attributed to Coronary Artery Embolism in a Patient with Atrial Fibrillation Secondary to Thyrotoxicosis - An Underrecognized Entity: A Case Report and Literature Review

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ABSTRACT

Nonatherosclerotic causes of acute myocardial infarction (MI) are infrequent, with atrial fibrillation as the most common etiology in cases of embolic MI. This entity, however, along with other causes of coronary embolus remains underappreciated as a probable cause of acute coronary syndromes.[1] Our case delineates a rare presentation of STEMI due to cardioembolic origin secondary to atrial fibrillation in a patient with thyrotoxicosis, previously undiagnosed to have an abnormal cardiac rhythm.

Key words: Atrial fibrillation, coronary artery embolism, myocardial infarction, percutaneous coronary intervention, thyrotoxicosis

INTRODUCTION

ST‑segment elevation myocardial infarction (STEMI) of nonatherosclerotic etiology is uncommon and in a few cases, emboli to coronary arteries may be seen due to left ventricular thrombus, atrial fibrillation, septic embolic owing to infective endocarditis, tumors, or patent foramen ovale, resulting in paradoxical embolism.[2]

A few case reports have documented nonatherosclerotic causes of acute MI attributed coronary artery embolism.[2‑14] Our case elucidates the significance of early recognition of atrial fibrillation secondary to thyrotoxicosis as a cause of acute MI in a patient found to have a thrombus in the left atrial appendage, which has been previously reported as an alternative trigger site in the initiation of atrial fibrillation in some cases rather than the pulmonary veins.[15]

CASE PRESENTATION

A 52‑year‑old man, known to have hypertension and dyslipidemia for 5 years, experienced acute chest pain radiating to his left arm and shoulder associated with nausea and sweating. He did not have any prior history of similar symptoms or cardiac disease. The ambulance electrocardiogram showed atrial fibrillation with ST‑segment elevation in the inferior leads and reciprocal ST‑segment depression in lateral leads [Figure 1]. The diagnosis of ST‑segment elevation MI (STEMI) was confirmed, and he was loaded...
with 600 mg clopidogrel, 5000 units of IV heparin, and 300 mg of aspirin in the ambulance.

Subsequently, upon hospital arrival, he was transported directly to the catheterization laboratory for percutaneous coronary intervention (PCI). Focused physical examination physical examination revealed a height of 174 cm and a weight of 78 kg. He was afebrile with blood pressure of 171/109 mmHg and had irregularly irregular pulse rate of 105 beats/min. Auscultation of the heart and lungs revealed no abnormalities.

Coronary angiography (CAG), performed through the right radial artery, revealed a normal dominant right coronary artery, and an abrupt occlusion of the distal LAD denoted as 100% with TIMI 0 [Figure 2a]. The intervention was performed, using a 6F Launcher EBU3.5, and a Runthrough NS 180 cm. Plain Balloon was performed, using a Maverick 2.75 mm × 15 mm compliant balloon. The inflation pressure was 6 ATM for the duration of 60.0 s. Following the intervention, there was a 60% residual stenosis. There was TIMI 0 flow before the procedure and TIMI 2 flow following the procedure [Figure 2b].

At this point, the clinical suspicion of coronary artery embolism as the cause of the STEMI was high. The angiographic appearance in conjunction with atrial fibrillation was highly suggestive. Heparin was given intravenously during the procedure and continued after as well. A rate-control strategy was initiated for the atrial fibrillation since the duration of the abnormal rhythm was unclear. Transthoracic echocardiography showed an ejection fraction of 33%, and the left atrium was severely dilated [Figure 3].

During the same admission, transesophageal echocardiography revealed a definite thrombus in the left atrial appendage [Figure 4]. The patient was started on rivaroxaban 20 mg and clopidogrel 75 mg daily. Moreover, the laboratory investigations revealed a new diagnosis of hyperthyroidism with a TSH value of <0.01 mIU/L, FT4 of 34 pmol/L, and FT3 of 9 pmol/L.

In liaison with endocrinology assessment, carbimazole 20 mg once daily was initiated. Thereafter, he was discharged home in good clinical condition.

**DISCUSSION**

Coronary artery embolism is known as a rare etiology of acute MI, while the accurate diagnosis presents as a challenge for interventional cardiologists, it is imperative to emphasize the possibility of nonatherosclerotic cause of MI, especially in patients lacking evidence of atherosclerotic coronary disease on CAG. Atrial fibrillation is the most common underlying etiology in acute MI attributed to coronary embolism, followed by cardiomyopathy and valvular heart disease.\(^\text{16}\) Coronary embolism may be suspected based on the National Cerebral and Cardiovascular Center clinical criteria for the diagnosis of coronary artery embolism as proposed by Shibata et al.\(^\text{16}\)

As nonatherosclerotic etiology of acute MI remains challenging to diagnose in acute clinical setting, its exact

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prevalence remains unclear. One study reported the absence of atherosclerotic coronary disease on CAG or autopsy findings in 4%–7% of patients with acute MI,[17] while another study documented coronary artery embolic infarcts in 13% of patients upon autopsy.[18]

After direct current cardioversion of atrial fibrillation, the prevalence of thromboembolic events is 2.0%,[19] of which most thromboembolic events present within 3 days postcardioversion.[20] Patients with diabetes and heart failure have a greater prevalence of 9.8% of thromboembolic events after cardioversion of atrial fibrillation.[21]

In another retrospective study of patients with acute MI, the prevalence of coronary artery embolism was 2.9%, of which 73% were attributed to atrial fibrillation. Nonvalvular atrial fibrillation accounted for 58% of patients with AF-induced coronary artery embolism.[16]

The incidence of arterial embolism in patients with thyrotoxicosis and atrial fibrillation is alarming. In the study of patients with thyrotoxicosis and atrial fibrillation, Staffurth et al. reported 8% of patients (21 out of 262) with 26 episodes of arterial embolism and 11 patients with concurrent atrial fibrillation and hyperthyroidism had embolic events.[22] Yuen et al. identified 24% of thyrotoxic patients with systemic embolism in the hyperthyroid state.[23] Hurley et al. described arterial embolism in eight patients out of 381 cases of thyrotoxicosis.[24] Owing to the increasing incidence of thromboembolic complications in patients with thyrotoxic atrial fibrillation, the ACC/AHA recommends anticoagulation therapy in these cases.[25]

It must be noted that the thyroid status of the patients affects the anticoagulative effect of warfarin. Hyperthyroidism with elevated levels of thyroid hormone increases the anticoagulative effect while the patients on antithyroid agents such as methimazole or propylthiouracil may experience diminished anticoagulative effect. Therefore, these patients with thyrotoxic atrial fibrillation must be monitored closely to ensure INR within recommended range.[24]

Initial studies reported a higher prevalence of rheumatic valvular disease[18] and bacterial endocarditis[26] in patients with acute MI attributed to coronary artery embolism. The prevalence of rheumatic valvular heart disease and bacterial endocarditis as an underlying etiology of coronary artery embolism has decreased, likely due to the advancements in primary PCI and the use of thrombectomy devices over the past three decades.

Aging results in a significant increase in the prevalence of atrial fibrillation, approximately 4% in those in their seventies, and 10% in individuals older than 80 years of age.[27]

The 5-year recurrence rate for thromboembolism including coronary artery embolism after the primary event of coronary embolism was 8.7%, with 35 months of median time until recurrence.[16]

**CONCLUSION**

Our case emphasizes the importance of early recognition of acute MI due to coronary artery embolism in a patient with atrial fibrillation secondary to thyrotoxicosis. As these cases seldom appear in clinical practice, physicians must be vigilant to timely identify this underrecognized entity as a cause of acute MI due to coronary embolism, especially in patients without angiographic evidence of atherosclerotic involvement.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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