Acute myocardial infarction due to coronary artery embolism in atrial fibrillation in Takotsubo cardiomyopathy patient: A case report

Muhammad Hanif[1], Mostafa Vasigh[2], Muhammad Malik[2], Vikash Jaiswal[3], Abhigan Shrestha[4], and Hami Kozman[2]

[1]SUNY The State University of New York
[2]SUNY Upstate Medical University
[3]AMA School of Medicine
[4]M Abdur Rahim Medical Medical College Hospital

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Introduction:

Myocardial infarction with nonobstructive coronary artery disease (MINOCA) is a group of conditions that share common characteristics and is characterized by the absence of [?]50% stenosis of coronary arteries and without any evidence of atherosclerotic plaque rupture [1]. A study conducted by Waller et al. reported that 4% to 7% of all patients diagnosed with AMI, do not have underlying atherosclerotic coronary disease on autopsy or angiography [2].

Coronary artery embolism is a rare and important non-atherosclerotic cause of acute myocardial infarction (AMI), and the first case of AMI secondary to coronary embolism was reported in 1856 by Rudolf Virchow and was classified initially as a precipitating factor for type-II MI, now recognized as one of the cause of MINOCA [3]. Coronary embolism (CE) is more frequently reported in infective endocarditis patients and it mainly involves the left main coronary artery system due to flow characteristics and aortic morphology [4, 5]. CE may also originate from mural thrombus within the left-sided cardiac chambers, but it has rarely been reported in the literature [6]. Most cases of coronary embolism in the literature have been reported secondary to infective endocarditis, valvular heart diseases, and atrial fibrillation [7-9]. Here, we are reporting a case of a 74-year-old female, who had a chronic history of atrial fibrillation on anticoagulation with questionable compliance and was admitted with sepsis and takotasubo cardiomyopathy and later developed AMI secondary to coronary embolism to the left anterior descending artery.

Case History/examination:

A 74-year-old female with a past medical history of atrial fibrillation on rivaroxaban with questionable compliance, type 2 diabetes, multiple cerebrovascular accidents, and suspected paranoid schizophrenia, was brought to the ED with altered mental status.

On examination, her pulse was 114/min, irregularly irregular, blood pressure of 123/109 mmHg, respiratory rate of 24/min, afebrile, and was maintaining oxygen saturation of 95% at room air.

Methods:

Baseline investigations showed WBC of 14.9 and urinalysis suggestive of UTI. Her Troponin level and proBNP were reported high (troponin 56 and proBNP of 16, 484). Electrocardiography (EKG) revealed atrial fibrillation with rapid ventricular response, low voltage QRS, poor R wave progression, and non-specific T wave

abnormalities. Transthoracic echocardiography reported a mildly dilated left ventricle and new severely reduced systolic left ventricular ejection fraction (LVEF 24%) with moderate diffuse hypokinesia, akinetic left ventricular apex, and severely hypokinetic inferolateroseptal wall, features suggestive of Takotsubo cardiomyopathy (Figure 1). There were no LA or LV clots detected on this echocardiogram, but this study was done without contrast. CT head reported negative for acute intracranial abnormality and the patient was started on IV antibiotics for treatment of sepsis in the setting of a urinary tract infection.

Cardiology was consulted for the newly depressed EF who recommended starting a statin, beta-blockers, and angiotensin-converting enzyme inhibitor (ACEI) if tolerable and a pharmacologic nuclear stress test to evaluate cardiomyopathy further.

The next day, the rapid response team (RRT) was called for hypotension of 66/38 mmHg and chest pain. EKG revealed atrial fibrillation with rapid ventricular response, right bundle branch pattern (RBBB), and ST elevations in the anterolateral leads suggestive of acute ST-elevation myocardial infarction (Figure no. 2).

The patient was started on inotropic support and was emergently transferred to the cardiac catheterization laboratory. On coronary catheterization, after placement of a balloon pump for hemodynamic support, a proximal LAD lesion with 100% occlusion and heavy thrombus burden was discovered (Figure 3). Initially, thrombus aspiration using a Pronto Extraction Catheter was attempted which was unsuccessful, but after POBA (percutaneous old balloon angioplasty) of the proximal LAD flow was restored. Post-injection revealed TIMI 3 flow in the LAD with no further underlying LAD disease, but a new 100% occlusion of the proximal LCx was now detected which likely occurred due to clot embolization during PCI to LAD (Figure 4). Again, after a trial of unsuccessful thrombectomy using the Pronto Extraction Catheter, POBA followed by deployment of a DES to the pLCX was performed without any further complications.

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A repeat transthoracic echocardiogram after PCI revealed an improvement in LVEF from 24% to 45% EF. The patient was eventually weaned off the balloon pump and pressor support after 4 days and was started on beta blockers and angiotensin-converting enzyme inhibitors (ACEI) and clopidogrel along with rivaroxaban and was discharged home after a week with outpatient follow-up at the cardiology clinic.

Conclusion and results:

Coronary artery embolism is a rare and fatal cause of acute myocardial infarction and has rarely been reported secondary to atrial fibrillation in takotsubo cardiomyopathy patients. Early diagnosis and treatment play a vital role in the prevention of short-term as well as long-term post-myocardial infarction complications. Physicians and cardiologists should have a low threshold for coronary artery embolism when encountering shock patients with atrial fibrillation, as early management prevents hazard complications.

Discussion:

According to angiographic and autopsy data, various causes of coronary artery embolism have been reported in the literature [10]. Different studies reported various causes as the most common cause of coronary artery embolism. A study conducted by Charles et al. reported bacterial endocarditis as the common cause of coronary embolism, while Prizel et al. reported valvular heart diseases and cardiomyopathy as the common risk factors for causing coronary embolism [11, 12]. However, a recent study conducted by Popovic et al. reported atrial fibrillation as the most common cause of coronary artery embolism, followed by dilated cardiomyopathy and bacterial endocarditis respectively [13]. Similarly, Shibata et al. conducted a study on new-onset MI between 2001 to 2013 and reported that 2.9% of AMIs were due to coronary embolism, with the most prevalent cause reported was atrial fibrillation, followed by cardiomyopathy and valvular heart disease [10]. Very rarely, paradoxical embolism causing acute myocardial infarction has been reported in the literature [14].

Atrial fibrillation patients are at increased risk of left atrium thrombus formation, particularly in the left atrial

appendage, through Virchow's triad of blood stasis, endothelial damage, and hypercoagulable state. These patients benefit from anticoagulation and require life-long anticoagulation to prevent thrombus formation and associated complications. In our case, a patient was admitted with a chronic medical history of atrial fibrillation on anticoagulation with questionable compliance and was admitted with sepsis and takotasubo cardiomyopathy, and later complicated by AMI secondary to coronary embolism to the LAD. Multiple factors played their role in developing coronary embolism in our patients, with the most common being atrial fibrillation with questionable compliance with anticoagulation, followed by takotsubo cardiomyopathy. Coronary artery embolism secondary to takotsubo cardiomyopathy has been reported in the literature [15], and the typical apical ballooning form of takotsubo cardiomyopathy seems to be more vulnerable to thrombus formation, which then leads to various thromboembolic complications [16].

Author contribution:

Conceptualization: Muhammad Hanif

All authors contributed equally. Supervision: Hani Kozman

Declaration of patient consent

Written informed consent was obtained from the patient for the publication of this case report and

accompanying images. A copy of the written consent is available for review by

the Editor-in-Chief of this journal on request.

Data availability: All the data are available in the manuscript.

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There are no conflicts of interest.

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Figure legends:

Figure No. 1: Two chambers echocardiography showing akinetic left ventricular apex (blue arrow) and overall diffuse moderate hypokinesia, features of takotsubo cardiomyopathy.

Figure no. 2: EKG showing right bundle branch block, ST-elevation changes in the

anterolateral leads with reciprocal changes in inferior leads.

Figure 3: PCI showing proximal LAD lesion with 100% occlusion (blue arrow).

Figure no. 4: PCI showing 100% occlusion of the proximal left circumflex artery (blue arrow)

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