Case Report

Acute myocardial infarction secondary to patent foramen ovale

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Abstract

A 32-year-old female presented with severe chest pain after few episodes of vomiting day after 5 h skiing and few alcoholic beverages consistent with ST-segment elevation myocardial infarction. Coronary angiography showed multiple complete distal obstructions including LAD, OM1 and OM2. Recovery was uneventful until 3rd day in our clinic when subsequent chest pain and vomiting occurred. Repeated vomiting and chest pain and new documented ST-segment elevation on ECG with a history of 5 h long skiing, made us look for paradoxical embolism. This intention was directed by MINOCA algorithm as one of the causes of epicardial/microvascular obstruction is paradoxical shunting. This case depicts our diagnostic approach to patent foramen ovale.

Keywords: patent foramen ovale; STEMI; valsala maneuver.

Highlights

- To consider patent foramen ovale in young patients with no obvious cause of acutely presented symptoms and no classical risk factors for coronary artery disease as the potential cause of myocardial infarction.
- To understand the pathophysiology of the Valsalva maneuver and that vomiting can reverse the interatrial pressure gradient and create a temporary right-to-left shunt.
- To highlight the importance of diagnostic tests like TEE with Valsalva maneuver or bubble echocardiography.
A Patent Foramen Ovale (PFO) is caused by an inadequate fusion of the septum primum with the endocardial cushions leaving the foramen ovale open after birth. A PFO is found in approximately 25% of the population. In most cases, it causes no health issues, but in some, it has been recognized as a possible source of paradoxical embolism. The emboli tend to be transmitted down the correspondent vascular beds by the influence of centrifugal forces (i.e., primarily into cerebral arteries and posterior cranial vessels). A few studies and case reports are reporting paradoxical embolism of non-cerebral circulation causing limb ischemia, splanchic ischemia, and splenic or renal infarction [1]. This case report depicts an unusual association among several triggers of thrombus formation, vomiting-dependent shunting, and coronary artery embolism in a young patient.

Case presentation

A 32-year-old female patient with no significant medical history and without classical atherosclerosis factors, presented with new-onset severe, acute chest pain and few episodes of vomiting after 5 hours skiing and 3 alcoholic beverages the previous day, in a local regional hospital. The initial physical examination revealed blood pressure of 135/85 mmHg, pulse of 75 beats/min, no temperature, normal breath sounds on auscultation and no edema in her lower extremities. Initial 12-lead Electrocardiogram (ECG) showed ST-segment elevation in leads II, III, and aVF, and ST-segment depression in leads aVL, V1, and V2, consistent with MI. At this point, she received DAPT (Aspirin 300 mg; Clopidogrel 300 mg) and was referred to our University hospital for coronary angiography. Upon arrival, her vital signs were unchanged and an inferior myocardial infarction was ECG confirmed (Figure 1). HsTroponin and creatine kinase levels were elevated at 4.028 ng/ml and 3552 U/L, respectively. Transthoracic Echocardiogram (TTE) showed discrete hypokinesis of the apical inferior and apical posterior wall. Urgent coronary angiography showed dissection with complete distal LAD obstruction and complete occlusions in distal OM1 and OM2 as well (Figure 2). She was started on antiplatelet and anticoagulant therapy and repeated coronary angiography was indicated. The course of recovery was uneventful until 3rd day in our clinic when subsequent chest pain and vomiting occurred with documented ST elevation in diaphragmatic leads.

Although there is limited data on the association between vomiting and embolization of coronary arteries, this combination made us look for paradoxical embolism. The patient was sent to a more sensitive Transesophageal Echocardiogram (TEE). In the resting state, the intra-atrial septum seemed to be intact, but upon Valsalva maneuver, there was a significant right-to-left shunt, consistent with PFO. Meanwhile, a hypercoagulable state in her labs was confirmed with genetic testing that detected mutations for Factor V Leiden mutation and PAI-1 4G/5G polymorphism, which were the most likely underlying causes of thrombus formation, possibly accelerated by skiing. The patient underwent PFO closure 12 months after the initial presentation and she was asymptomatic on follow-ups. Control coronary angiography showed normal coronary arteries (Figure 3).

The entity of paradoxical embolism through PFO remained a diagnostic challenge. The no-guidelines land about patients with non-cerebral paradoxical embolization requires an individualized approach [2]. In our case, no obvious cause of acutely presented symptoms and the intention to find the source of the paradoxical embolism directed us to MINOCA algorithm, as one of the causes of epicardial/microvascular obstruction is paradoxical shunting. However, our patient did not have non-obstructed coronary arteries.

In all patients with an initial working diagnosis of MINOCA, it is recommended to follow a diagnostic algorithm to differentiate true MINOCA from alternative diagnoses. Non-invasive tests that can be used in search for PFO are TEE and bubble contrast echocardiography [3,4].

The high prevalence of PFO in echocardiographic studies makes a definitive diagnosis of paradoxical embolism challeng-
ing. Rather, three criteria have been proposed to make a presumptive diagnosis: (i) embolization that was not sourced from left heart but (ii) had originated from the venous system and was (iii) associated with abnormal communication between the venous and arterial circulations, in the form of atrial septal defect or PFO [5].

Approximately 6% of all STEMIIs do not have atherosclerosis detectable by coronary angiography or autopsy and in these cases coronary vasospasm, in situ thrombosis, coronary embolism, and coronary artery dissection are among the leading causes. Myocardial infarction secondary to coronary artery embolism is not infrequent, seen in about 10-13% autopsy series. However, Paradoxical Embolism (PDE) to the coronary tree is rare and is seen in about 5-10% of all PDE phenomena. An autopsy series of 1050 patients with myocardial infarctions by Prizel et al. did not find a single case of PDE among the 55 patients identified with coronary embolism [6].

The left atrium pressure is higher than the right atrium, which prevents passage by holding down the septum primum flap to septum secundum. Even if the flap is partially open, the blood flow will be from left to right. However, daily activities such as lifting, coughing, vomiting, and pushing which increase Intrathoracic (ITP) and intraabdominal pressure may reverse the interatrial pressure gradient, creating a temporary right-to-left shunt. One of the most effective methods is an extended and forced Valsalva maneuver [7].

In this case, the specific cause of the possible deep venous thrombosis is identified, however, there is no definite evidence that hypercoagulable states would increase the risk of embolic events in a patient with PFO [8]. That is why we assume that vomiting which occurred just before the chest pain leads to increased right atrial pressure with shunt opening and increase right-to-left blood flow.

**Declaration of interest:** The authors report no conflicts of interest.

**References**


