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A rare cause of acute ST-elevation myocardial infarction: case report of native aortic valve thrombosis

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Background

One to 13% of all patients with the clinical diagnosis of an acute coronary syndrome (ACS) show no evidence of significant obstructive coronary artery disease on angiography. Less common causes should be considered in those situations. A very rare cause of ACS is native aortic valve thrombosis.

Case summary

A 69-year-old previously healthy woman presented with acute chest pain. The electrocardiogram showed an anterolateral ST-elevation myocardial infarction (STEMI). She was immediately transferred for primary percutaneous coronary intervention. Shortly after arriving in hospital her condition deteriorated, with development of cardiogenic shock necessitating cardiopulmonary resuscitation. A coronary angiogram was performed during resuscitation that did not reveal any obstructive coronary artery disease. Echocardiography showed no pericardial effusion, no significant left-sided valve pathology, no signs of an aortic dissection or pulmonary embolism. She died of cardiogenic shock of unknown cause. Permission for autopsy was obtained. Pathologic examination revealed a large anterolateral myocardial infarction caused by a mass attached to the bottom of the left coronary cusp of the native aortic valve, which was large enough to occlude the ostium of the left main coronary artery. Microscopic analysis showed a thrombus of unknown origin. The aortic valve itself showed no signs of pathology.

Discussion

An ST-elevation myocardial infarction due to native aortic valve thrombosis is a rare condition, especially when there are no significant valvular abnormalities. This case demonstrates that thrombosis can develop in an apparently healthy middle-aged woman without any history of thrombotic disease.

Keywords

MINOCA • Case report • ST-elevation myocardial infarction • Native aortic valve thrombosis

Learning points

- Native aortic valve thrombosis without any valvular abnormalities is a rare condition.
- Native aortic valve thrombosis can cause serious complications in case of coronary ostium obstruction or systemic embolization.
- Native aortic valve thrombosis can be missed with coronary angiography or transthoracic echocardiography; cardiac magnetic resonance or computed tomography may be of additional value.

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Introduction

The majority of patients with an acute coronary syndrome (ACS) has evidence of a coronary stenosis and thrombus. Nevertheless, there is a variety of other conditions that can cause an ACS without significant obstructive coronary artery disease on angiography. This report describes a case of a large thrombus of a native aortic valve presenting as an ST-elevation myocardial infarction (STEMI).

Timeline

Admission to catheterization laboratory	Presented with typical acute chest pain and an ECG registration with acute anterolateral ST-elevation myocardial infarction
15 min after admission	Need for cardiopulmonary resuscitation (CPR) Coronary angiogram during CPR did not show evidence of obstructive coronary artery disease A transthoracic echocardiogram excluded pericardial effusion, valve disease, and direct or indirect signs of pulmonary embolism or aortic dissection
1.5 h after admission	Patient died of cardiogenic shock of unknown origin
1 day after death	Autopsy showed a large anterolateral myocardial infarction due to occlusion of the ostium of the left main coronary artery by a thrombus of a native aortic valve

Case presentation

A 69-year-old Caucasian female woke up with acute typical chest pain. Her previous history was negative for cardiovascular disease or cardiovascular risk factors. An electrocardiogram (ECG) was performed upon arrival of the ambulance (*Figure 1*). This showed an acute anterolateral ST-myocardial infarction and she was immediately transferred for primary percutaneous coronary intervention. Her vitals and cardiopulmonary exam were normal at admission (heart rate 91 b.p.m., blood pressure 133/97 mmHg, and oxygen saturation of 96% on room air). Shortly following arrival in the catheterization laboratory, her clinical condition rapidly deteriorated. She developed pulmonary oedema as a result of a cardiogenic shock and required intermittent cardiopulmonary resuscitation (CPR) due to pulseless electrical activity. A coronary angiogram was performed while performing CPR with a chest compression system (Lund University Cardiac Assist System, LUCAS), which showed no evidence of obstructive coronary artery disease. Subsequently, a transthoracic echocardiogram was performed. Despite the poor image quality due to the critical situation, pericardial effusion and significant left-sided valve disease could be excluded as a possible cause of the cardiogenic shock. There were no direct or indirect signs of pulmonary embolism or aortic dissection. Due to further deterioration with no detectable treatable cause of the condition, the team of health professionals decided to discontinue CPR. She died of cardiogenic shock of unknown origin. As the cause of her death was unclear, permission for autopsy was obtained. Autopsy found a mobile mass of 2.5×2 cm attached to the bottom of the left coronary cusp of the native aortic valve, which was large enough to occlude the ostium of the left main coronary artery (*Figures 2 and 3*). The pathologic examination of the myocardium revealed an acute anterolateral infarction (≥ 4 h duration; *Figure 4*). Histology showed a thrombus of unknown origin (*Figure 5*). Further investigation of the aortic valve and of the heart revealed no significant abnormalities.

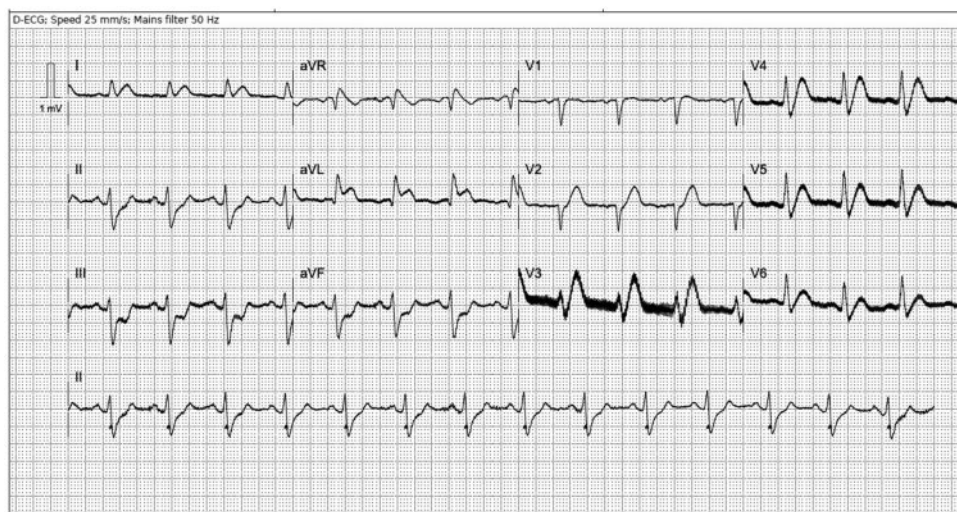


Figure 1 Electrocardiogram showing anterolateral ST-elevation myocardial infarction.

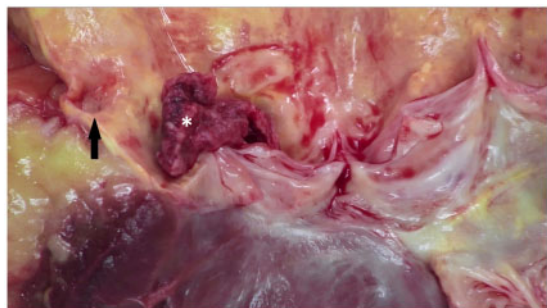


Figure 2 The heart cut through the aortic valve. Thrombus (*) is attached to the bottom of the left coronary cusp, which is partially mobile and obstructing the ostium of the left coronary artery (black arrow). No other valvular pathology was observed.

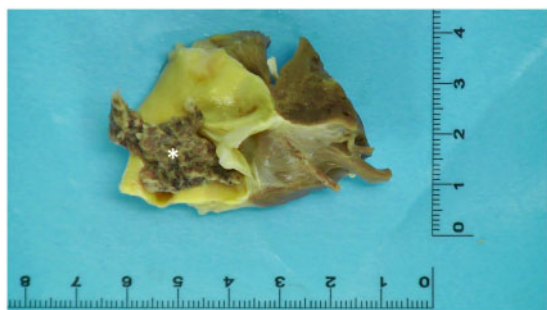


Figure 3 The thrombus (*) after formalin fixation. Scale bar in centimetres.

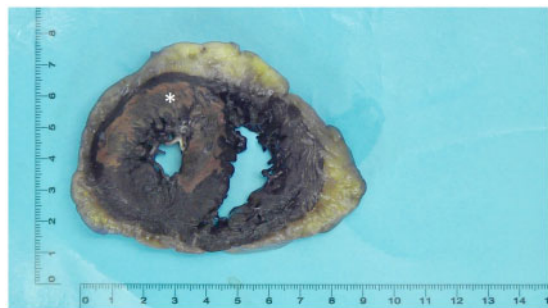


Figure 4 Transversal slice of the myocardium reveals discolouration of lactate dehydrogenase in the areas supplied by the left coronary artery (*). Scale bar in centimetres.

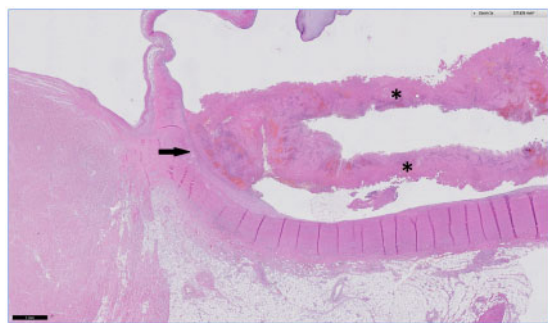


Figure 5 Histology of the thrombus (*) showed a base attached to the bottom of the cusp (black arrow). The thrombus consisted of fibrin, thrombocytes, lymphocytes, and neutrophils which are partially deteriorated. This could date the thrombus at no more than a few days old. No microorganisms could be detected.

Discussion

In this case, an idiopathic native aortic valve thrombosis was the unusual cause of STEMI. Despite the fact that the majority of STEMI patients has evidence of a coronary stenosis and thrombosis, some patients present with STEMI in the absence of obstructive coronary artery disease on angiography. This type of myocardial infarction is called 'myocardial infarction with non-obstructive coronary arteries' (MINOCA).^{1,2} MINOCA is a heterogeneous entity with a prevalence of 1–13% in all patients with the clinical diagnosis of an ACS.³ There are several differential diagnoses for MINOCA, which may arise from both coronary and non-coronary causes. Possible aetiologies of MINOCA are coronary artery spasm, type 2 myocardial infarction (due to supply/demand mismatch), coronary dissection, myocarditis, Takotsubo cardiomyopathy, pulmonary embolism, and (coronary) thromboembolism.⁴ The latter may cause an ACS in the setting of predisposing hypercoagulable conditions like prosthetic heart valves, atrial fibrillation, dilated cardiomyopathy with apical thrombus, rheumatic heart disease with mitral stenosis, infective endocarditis, and atrial myxoma. As in our case, thrombus formation apparently can also occur in the absence of all above factors.

As previously mentioned, valvular heart disease is a predisposing hypercoagulable condition, with an increased risk for thromboembolic events. Emboli may arise from prosthetic valves (mainly mechanical prostheses), endocarditis, valve tumours (e.g. papillary fibroelastoma), iatrogenic valve injury, and congenital or degenerative valvular diseases. Valve thrombosis in the absence of any valvular disorder, as in our case, is a rare condition.

In the present literature, a small number of case reports describe that a thrombotic disorder may predispose for thrombus formation on the aortic valve. Protein S and C deficiency, antiphospholipid syndrome, hypereosinophilic syndrome, and polycythaemia have been described to cause thrombosis of the native aortic valve.^{5–9} Nevertheless, no detectable thrombotic disorder is observed in most cases, or the coagulation state is unknown.¹⁰ Thrombophilia screening in patients with MINOCA reported a 14% prevalence of inherited thrombotic disorders.³ Because of the fatal course in our case, we had no possibility to perform blood testing. However, patient history and family history were negative for thrombotic

disorders and autopsy revealed no thrombus formation or embolization elsewhere.

Besides MINOCA, patients with native aortic valve thrombosis can also present with systemic embolization.⁶ Nagata et al.¹¹ reported a case of native aortic valve thrombosis presenting as a STEMI together with arterial embolization in the left leg.

Our case demonstrates that it is important to identify the underlying cause of MINOCA, since it may require specific therapies. Failure to identify the underlying cause may result in inadequate and inappropriate therapy. Computed tomography or cardiac magnetic resonance imaging (CMR) may be useful to differentiate between many of the different causes of MINOCA. Therefore, CMR is recommended by the ESC guidelines to identify the aetiology in the above clinical setting.^{1,4} In our case, due to critical patient condition, applying additional diagnostic tests beyond coronary angiography and echocardiography was impossible.

Lead author biography



Gerrie Beekman – van Solkema was born in 1986 in Zwolle, the Netherlands. She received her medical training at the University of Groningen. In 2017 she completed her residency in Internal Medicine at the Martini Hospital in Groningen. Currently she is following her residency in Cardiology at the University Medical Center Groningen.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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