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Venous thrombosis via pulmonary arteriovenous malformation causing acute myocardial infarction in a relatively young female patient

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SUMMARY

Pulmonary arteriovenous malformation (PAVM) is a probable cause of thromboembolic diseases such as acute myocardial infarction (MI); however, few cases have been reported. A woman in her early 40s developed acute-onset chest pain; an ECG showed ST-elevated MI. Emergency catheter angiography showed that the culprit lesion was a thrombus that was treated successfully with aspiration. She had a history of deep venous thrombosis and CT revealed PAVM. It was likely that the venous thrombus had moved into the coronary artery through the PAVM. Catheter embolisation of the PAVM was performed and she did not experience any other cardiac event until 6 months after embolisation.

BACKGROUND

Pulmonary arteriovenous malformation (PAVM) is a rare vascular anomaly where abnormal anastomoses of the pulmonary arteries and veins occur.[1] Most patients with PAVM are asymptomatic and remain undiagnosed until adulthood; however, PAVM has a direct right-to-left shunt and sometimes results in several severe comorbidities, such as chronic hypoxemia, rupture of the PAVM, central nervous system disorders and thromboembolic disease.[1] Here, we report the case of a female patient who developed acute myocardial infarction (MI) with venous thrombosis via PAVM, in which a favourable outcome was obtained with catheter intervention.

CASE PRESENTATION

A woman in her early 40s with a history of deep vein thrombosis (DVT) and pulmonary embolism 2 years prior had experienced acute chest pain and dyspnoea while walking in the evening. The patient was transported to our hospital by ambulance. Her systolic blood pressure reading was 110–120 mm Hg, and her routine medications were rivaroxaban 15 mg once daily (maximum tolerated dose in Japan) for her medical history of DVT and pulmonary embolism, and a contraceptive pill. She had smoked 15–20 cigarettes per day for 22 years. She had no remarkable family history or other medical history, including coronary risks.

INVESTIGATIONS

Initially, she reported strong chest pain with cold sweat, restlessness and peripheral coldness. Her vital signs revealed cardiogenic shock, with a blood pressure of 84/52 mm Hg, heart rate of 62 beats per minute, O_2 saturation of 98% on room air and body temperature of 36.2°C. An ECG showed sinus rhythm and ST-segment elevation in leads II, III and aVF with reciprocal changes in aVL and V1-4 (figure 1). Echocardiography revealed severe hypokinesis of the inferior wall. Thus, the patient was considered to have acute ST-elevated MI (STEMI).

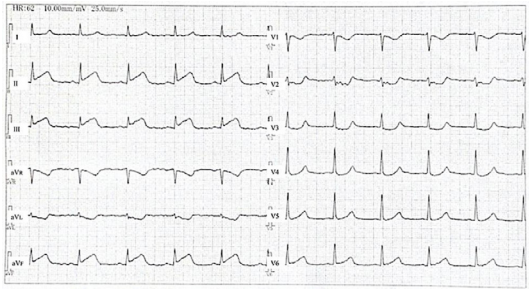

Figure 1 Electrocardiography on arrival. An ECG revealed sinus and ST-segment elevation in leads II, III and aVF with the reciprocal change of aVL and V1-4 on arrival in the hospital.

DIFFERENTIAL DIAGNOSIS

Possible causes of ST-elevation with acute chest pain include MI, pericarditis, myocarditis, takotsubo-cardiomyopathy and pulmonary embolism.

In pericarditis, diffuse concave ST-elevation and pericardial effusion are common. Myocarditis also provides diffuse ST-elevation and is often preceded by fever and gastrointestinal symptoms. ST-elevation is rare in pulmonary embolism; however, severe desaturation, sinus tachycardia in electrocardiography and right heart strain in echocardiography are usually obtained. Takotsubo-cardiomyopathy shows takotsubo-like wall motion with ST-elevation

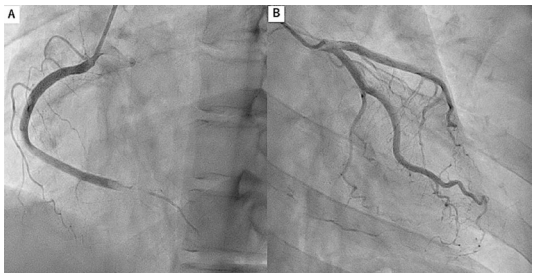

Figure 2 Emergent coronary angiography on arrival. Coronary angiography revealed (A) occlusion of the distal right coronary artery and (B) no other obstructive lesions in the left coronary arteries.

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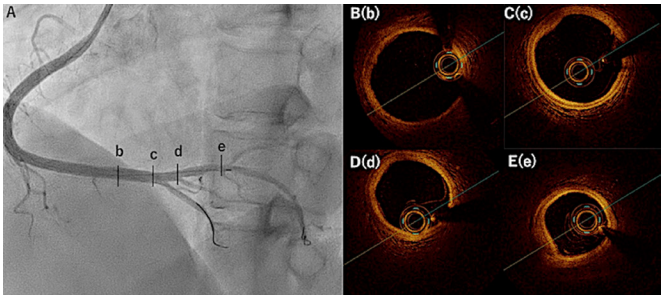

Figure 3 Coronary angiography and optical coherence tomography (OCT) after the aspiration. (A) Coronary angiography and (B–E) OCT findings showed no obstructive lesion after aspiration. (B–E) OCT revealed no plaque rupture, arteriosclerosis or erosion in the vascular lumen.

in echocardiography unless coronary obstruction has occurred. In this case, chest pain and dyspnoea were the main symptoms without fever and worsening oxygenation. The ECG and echocardiogram suggested ischaemia of the inferior wall. These findings were consistent with an acute inferior MI. Additionally, we had to rule out takotsubo-cardiomyopathy using coronary angiography for further investigation.

TREATMENT

Emergency coronary angiography showed occlusion of the distal right coronary artery (RCA) and no other obstructive lesions (figure 2). Aspiration was repeated on the culprit lesion of the RCA and blood clots were aspirated. The blood flow gradually improved and optical coherence tomography (OCT) was performed. OCT detected no plaque rupture, arteriosclerosis or erosion of the vascular lumen (figure 3). Additionally, the procedure was completed without stent use.

The patient's postoperative course was uneventful. Blood tests revealed no other abnormalities even during thrombophilia screening for Protein C and S antigens, antithrombin III activity and antiphospholipid antibodies (including anticardiolipin antibody and lupus anticoagulant). Contrast-enhanced CT showed a right PAVM (figure 4). Although DVT and pulmonary embolism were not detected on contrast CT at this time, her history of DVT and PAVM, smoking history and intake of contraceptive pills suggested that the thrombus had occluded the coronary artery through the PAVM from the venous system. We then performed percutaneous PAVM embolisation using the coiling technique and the PAVM was successfully blocked (figure 5).

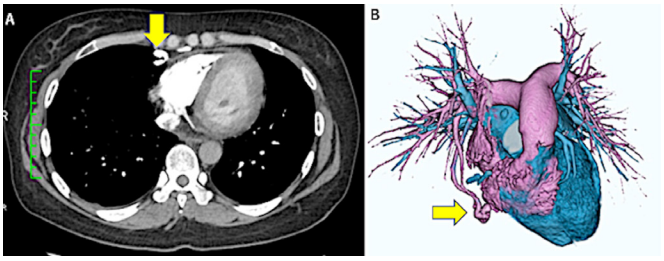

Figure 4 The right pulmonary arteriovenous malformation (PAVM) in enhanced CT. (A) Contrast CT of axial view and (B) 3D reconstruction revealed the right PAVM.

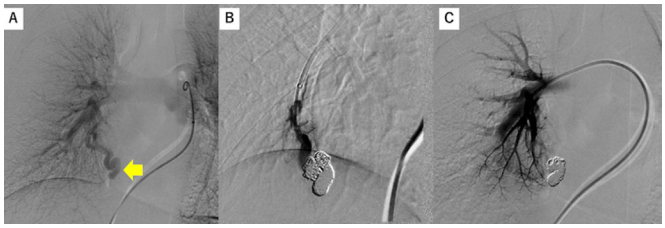

Figure 5 Pulmonary angiography and percutaneous pulmonary arteriovenous malformation (PAVM) embolisation. (A) Pulmonary angiography revealed a right PAVM and (B) percutaneous embolisation of PAVM was performed via the pulmonary artery. (C) Final angiography showed no significant shunt flow.

OUTCOME AND FOLLOW-UP

The patient's dyspnoea symptoms reduced after occlusion of the PAVM and she did not experience another event until the last follow-up, 6 months postoperatively.

DISCUSSION

This is a case of STEMI in a woman with PAVM and a history of venous thromboembolism (VTE). PAVM provides a direct right-to-left shunt, allowing the passage of paradoxical thrombotic embolisation. Such embolisation commonly presents as stroke or brain abscess; treatment of PAVMs is considered the standard of care to prevent these events.[1 2] However, few paradoxical coronary artery embolisation cases via PAVM are reported.[3 4] Here, we report a rare case of MI with PAVM that was treated only with aspiration and anticoagulation based on OCT findings.

According to a previous report, the mortality of untreated patients with PAVM was 11% after 6 years from diagnosis.[5] In addition, PAVM is often associated with hereditary haemorrhagic telangiectasia (HHT). PAVM is present in 15%–50% of people with HHT and has been associated with life-threatening complications.[6] Although, we did not perform a genetic test in the present case because there were no symptoms, arteriovenous malformation and family history to suspect HHT. However, we would have considered the genetic test if she had any symptoms of HHT.

Embolisation using a catheter is the major treatment for PAVM. It is a safe and less invasive intervention with a high success rate of 98.7% and a low major complication rate of approximately 1%.[2 7] British Thoracic Society's clinical statement on PAVM also indicates that all patients with radiographically visible PAVM should be considered to receive embolisation treatment even for asymptomatic patients because PAVM treatment reduces the risk for paradoxical emboli and improves oxygenation, other physiological parameters, symptoms exacerbated by right-to-left shunting and haemorrhage.[7] Moreover, the Cochrane Intervention Review of PAVM shows that based on observational studies, all PAVMs, irrespective of size, amenable to embolisation should be treated.[8] We should consider PAVM embolisation when a PAVM is detected even if the patients are asymptomatic.

In the present case, the limitation is that DVT and pulmonary embolisation were not detected, and it is not possible to prove whether a venous thrombus caused the actual coronary artery occlusion or not. However, considering that the patient, a relatively young woman with little coronary risk, was at high risk for venous thrombosis due to a history of DVT and pill therapy and had PAVM, the cause of MI was highly likely venous thrombosis.

We encountered a rare case of a relatively young woman who developed acute MI from venous thrombosis via PAVM and was successfully managed with PAVM occlusion. PAVM might cause

MI; thus, we might consider the possibility of PAVM if a patient with MI has a thrombotic lesion and no other significant coronary risk factors.

Patient's perspective

One day, I suddenly became distressed without any warning and was carried to the hospital by ambulance. I had experienced a pulmonary embolism before, but the pain this time was completely different from the last time. I appreciate the doctors and paramedical workers for treating me quickly. After the treatment, I have not experienced any further symptoms. I have heard that both myocardial infarction and pulmonary embolism may result in a fatal condition; hence, I intend to use this opportunity to review my lifestyle and prevent a recurrence.

Learning points

- Pulmonary arteriovenous malformation (PAVM) might cause various complications, including cerebrovascular and cardiovascular diseases.
- Catheter embolisation of PAVM should be considered to prevent thromboembolic comorbidities of PAVM, even in asymptomatic patients.

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