CASE REPORT

Thrombi in all four cardiac chambers in a patient with an old anteroseptal myocardial infarction and atrial flutter

Hanae Ida,¹ Kazuhito Hirata,² Takanori Takahashi,² Takanobu Hirose¹

¹Department of Internal Medicine, Okinawa Chubu Hospital, Uruma City, Okinawa, Japan

²Department of Cardiology, Okinawa Chubu Hospital, Uruma City, Okinawa, Japan

Correspondence to

Dr Hanae Ida,
hany.moo87@gmail.com

Accepted 2 November 2014

SUMMARY

Cases with thrombi in multiple cardiac chambers are rare. We report an extremely rare case in which thrombi were formed in all four cardiac chambers at the same time. A 55-year-old man presented with biventricular failure and liver dysfunction. A 12-lead ECG and an echocardiogram revealed an old anteroseptal myocardial infarction with aneurysm formation and atrial flutter. A transesophageal echocardiogram and a CT of the chest revealed thrombi in the right and left atrial appendage, right ventricular apex and left ventricular aneurysm. Pulmonary emboli were also identified. Stasis of the blood due to ventricular dysfunction and atrial flutter was considered to be responsible. The patient died of multiorgan failure due to circulatory failure.

BACKGROUND

Cases with thrombi in all four cardiac chambers are extremely rare. We experienced a case of quadrichamber thrombi in a patient with an old antero-septal myocardial infarction and atrial flutter. From the morphology and location of the thrombi, it was assumed that those thrombi were primarily formed in cardiac chambers due to blood stasis, not a floating thrombus migrated from lower extremities. This case illustrates the importance of blood stasis as the cause of thrombus formation in cardiac chambers.

CASE PRESENTATION

A 55-year-old man with no significant medical history presented to our hospital with general fatigue, shortness of breath, palpitation and loss of appetite of 12 months duration. One month prior to admission, his symptoms had turned worse and his daily activities gradually became so limited that he stayed in bed the whole day. Two weeks prior to admission, he started to notice further worsening of shortness of breath and leg oedema. Finally, he developed haemoptysis. On admission he looked sick and had unstable vital signs as follows: blood pressure of 94/57 mm Hg, heart rate of 124/min and respiratory rate of 18–20/min. His skin colour was yellow due to jaundice and his legs were oedematous. Auscultation of the heart revealed gallop with third heart sound. Auscultation of the lungs revealed crackles in the right lower lung field. Laboratory data were as follows: white cell count 14 100/ μ L, haemoglobin 14.5 g/dL, platelet 79 000/ μ L, blood urea nitrogen 54 mg/dL, creatinine 1.44 mg/dL, aspartate aminotransferase 102 IU/L,

alanine transaminase 129 IU/L, total bilirubin 22.8 mg/dL, direct bilirubin 16.2 mg/dL, lactate dehydrogenase 482 IU/L, alkaline phosphatase 261 IU/L, lactate 2.6 mmol/L and C reactive protein 9.28 mg/dL. A prothrombin time-international normalized ratio (PT-INR) was elevated to 1.81 and activated partial thromboplastin time was slightly elevated up to 39.8 s. A chest roentgenogram at ER showed cardiomegaly and infiltration in the right lower lobe. A 12-lead ECG showed common atrial flutter with 2:1 conduction, right bundle branch block, right axis deviation and Q waves in V1–V4 suggestive of an old antero-septal myocardial infarction (figure 1). Because of unstable vital signs and multiorgan failure, the patient was admitted to the intensive care unit. A transthoracic echocardiogram showed thin and calcified left ventricular septum and apex with a large, horseshoe-shaped mural thrombus in the apical half of the left ventricle. A transesophageal echocardiogram (TEE) confirmed a thrombus in the left atrial appendage and left ventricle (figure 2A, B, videos 1 and 2). There was severe systolic dysfunction in the left and right ventricle. An ejection fraction was estimated as 10% and moderate mitral and severe tricuspid regurgitation were noted. A contrast-enhanced CT of the chest revealed thrombi in all four cardiac chambers

Figure 1 A 12-lead ECG showing atrial flutter with mainly 2:1 conduction, right bundle branch block (RBBB), right axis deviation and Q waves in V1–V4, suggesting an old antero-septal myocardial infarction. RBBB and right axis deviation might be secondary to pulmonary hypertension, or bifascicular block associated with myocardial infarction. Arrows indicate flutter waves.

To cite: Ida H, Hirata K, Takahashi T, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-207231

Figure 2 (A) A four-chamber view of the transesophageal echocardiogram showing a horseshoe-shaped thrombus located at the apical half of the left ventricle as shown by smaller arrows (see [video 1](#)). (B) A mobile thrombus (larger arrow) was identified at the left atrial appendage. Smaller arrows indicate the thrombus in the left ventricle ([video 2](#)).

and bilateral pulmonary arteries, namely, right and left atrial appendage, right ventricular apex and the left ventricle ([figure 3](#)). There was no thrombus in the veins of the lower extremities, pelvis or inferior vena cava (IVC). An infiltration in the right lower lung fields appeared to be consistent with pulmonary infarction and the source of haemoptysis. The patient was diagnosed as biventricular heart failure secondary to an old anterior myocardial infarction and atrial flutter with rapid ventricular response, thrombi in all four cardiac chambers, a massive pulmonary embolism (possibly subacute or chronic) with pulmonary hypertension and congestive liver. Sepsis and vitamin B₁ deficiency were also suspected, and the patient was given broad spectrum antibiotics, thiamine and catecholamine. Low-dose intravenous heparin was used carefully, in order not to worsen bleeding tendency and haemoptysis. In spite of aggressive medical therapy, the patient's blood pressure remained as low as 90/60 mm Hg. Using a thrombolytic agent was deferred because the patient had haemoptysis. A catheter-based mechanical thrombectomy for pulmonary emboli was attempted as a last resort but it was not effective. Haematological examinations for thrombophilia, such as antiphospholipid antibody were negative and deficiencies of protein C or S were difficult to interpret because of severe liver dysfunction. He passed away on the ninth hospital day due to multiorgan failure.

OUTCOME AND FOLLOW-UP

In spite of aggressive treatment, including ventilator care, invasive haemodynamic monitoring, vasopressors, low-dose heparin and catheter-based thrombus aspiration from the right

pulmonary artery, the patient died on the ninth hospital day. An autopsy was declined by his family.

DISCUSSION

Torbicki *et al*¹ reported that the incidence of right heart thrombi detected with echocardiography in patients with pulmonary emboli was 3.7% (42 of 1113 patients) in the International Cooperative Pulmonary Embolism Registry. In a large autopsy series of pulmonary emboli, Vasil'tseva *et al*² reported that the incidence of right heart thrombi was 24% (157 out of 652) and almost a half of those cases had left heart thrombi also. However, premortem diagnosis of thrombi in all four cardiac chambers at the same time in patients with pulmonary emboli is extremely rare and we are aware of only a few case reports.^{3–5} Singh *et al*³ reported a case of quadri-chamber thrombi in alcoholic cardiomyopathy in normal sinus rhythm. They attributed the cause to a low flow state due to biventricular dysfunction. Galvez *et al* reported a case of renal cell carcinoma and inferior vena cava thrombus, who developed shock and died during surgery. Thrombi in all four cardiac chambers were identified by TEE.⁴ They considered that the thrombus had migrated from IVC and also migrated to the left heart via a patent foramen ovale. Lima and Soltesz⁵ reported a case of polycythaemia vera, who developed thrombi in all four cardiac chambers during coronary aorto-bypass surgery. A hypercoagulable state due to polycythaemia and transient cessation of antithrombotic medications were considered to be responsible.

Although the veins of the lower extremities are classic sources of pulmonary embolisms, thrombi in the chambers of the heart

Video 1 Horseshoe-shaped thrombi in the left ventricle.

Video 2 A mobile, ball-like thrombus in the left atrial appendage and thrombi in the left ventricle.

Figure 3 (A–D) Contrast-enhanced chest CT images showing the thrombi in all four cardiac chambers (thrombus in the right atrial appendage in A and B. Thrombus in the left atrial appendage in B. Thrombus in the right atrial apex in C, and thrombi in the left ventricle and right ventricular apex in D). Large arrow heads indicate pulmonary thromboemboli in the right and left inferior pulmonary arteries.

may be the potential source in some instances. The underlying mechanisms for intracardiac thrombus formation have been attributed to either blood stasis or haematological abnormalities causing hypercoagulability, including myocardial infarction, atrial fibrillation/flutter, dilated cardiomyopathic state, myocarditis, vasculitis, antiphospholipid syndrome, protein C or S deficiency and polycythaemia vera.^{2–8} Surgical procedures and underlying malignancy may act as precipitating factors.

Regarding the possible cause of thrombi in our patient, it was estimated that blood stasis due to an old anteroapical myocardial infarction (age-unknown) with aneurysm involving left as well as right ventricular apex resulted in the thrombi in the left and right ventricle.⁶ Thrombi in the left and right atrial appendage resulted from blood stasis due to atrial flutter, which occurred as a result of congestive heart failure secondary to poor ventricular function.^{7–8} In case of ‘floating’ thrombi which migrated from the deep vein thrombosis (DVT) in the lower extremities, thrombi transiently became entrapped in the right heart chambers or foramen ovale, resulting in thrombi in multiple chambers of the heart.⁹ The shape of those thrombi were usually worm-shaped and highly mobile.⁹ In our case, the shape of the right side thrombi was not worm-shaped and there was no evidence of DVT. In addition, the thrombi in the left and right atrium were located in the appendages where stasis of the

blood tends to occur in patients with atrial fibrillation or flutter. So those thrombi appeared to be formed primarily in the cardiac chambers. Subacute pulmonary emboli occurred as a terminal event leading to severe circulatory dysfunction and death.

Regarding the possibility of hypercoagulability, antiphospholipid antibodies were negative. Marked liver dysfunction, presumably due to congestion and low output, precluded the evaluation of proteins C and S. Moreover, there was mild thrombocytopenia due to liver dysfunction. Detailed evaluation for possible malignancy as a cause of thrombophilia was not possible because of the critical condition of the patient.

Contributors HI reported and organised this case. KH served as professional adviser and TT and TH were clinical advisers who provided treatment and cared for the patient.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- This was an extremely rare case in which thrombi were formed in all four cardiac chambers at the same time.
- An old anteroapical myocardial infarction, of which the history was not known, and atrial flutter were the possible underlying causes of thrombi in this particular patient.
- Blood stasis in the akinetic left and right ventricular apex was the pathophysiology for ventricular thrombi and blood stasis due to atrial flutter appeared to be responsible for the thrombi in the left and right atrial appendage.

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