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Pulmonary embolism as a cause of unexplained sinus tachycardia after right ventricular myocardial infarction

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ABSTRACT We present the case of a patient who developed new-onset asymptomatic sinus tachycardia after undergoing treatment for a right ventricular myocardial infarction. Even after excluding heart failure, infection and bleeding, the sinus tachycardia persisted. Computed tomography pulmonary angiography showed multiple bilateral pulmonary emboli. The vital sign abnormality resolved after treatment with an anticoagulant. We postulate that the pulmonary emboli originated from thrombi that were formed in the infarcted and dysfunctional right ventricle. Pulmonary embolism is a very rare complication of right ventricular myocardial infarction, and patients usually present with pleuritic chest pain. Our case highlights that asymptomatic sinus tachycardia could be a presenting feature of pulmonary embolism after the occurrence of a right ventricular myocardial infarction. A high index of suspicion is warranted in order to detect this potentially lethal complication.

Keywords: arrhythmia, dysfunction, regurgitation, ventricle

INTRODUCTION

Sinus tachycardia is a common finding in patients presenting with myocardial infarction. Common causes include heart failure, bleeding and infection. Pulmonary embolism secondary to right ventricular infarction is a rare but potentially fatal complication. A high index of suspicion is needed to diagnose pulmonary embolism complicating myocardial infarction because many of its symptoms and signs overlap and are subtle.

CASE REPORT

A 42-year-old Indian man with a history of hypertension and hyperlipidaemia presented to our institution with acute chest pain. 12-lead electrocardiography showed inferior-posterior myocardial infarction involving the right ventricle. The patient was sent for emergency percutaneous coronary intervention, and a drug-eluting stent was implanted into the occluded proximal right coronary artery. Physical examination of the patient on admission was unremarkable, with a heart rate of 57 bpm and a blood pressure of 120/70 mmHg. The patient's oxygen saturation on room air was 98%.

Over the next few days, the patient was pain-free and afebrile. However, his recorded vital signs showed persistent sinus tachycardia (heart rate of 105–120 bpm) and hypoxaemia (90%–92% oxygen saturation on room air) that required oxygen supplementation. The patient's blood pressure was stable and he did not require inotropic support. There was no evidence of bleeding, and haemoglobin levels remained normal. Echocardiography showed a left ventricular ejection fraction of 40% (Fig. 1). There was dilatation and systolic dysfunction of the right ventricle, as well as moderate pulmonary hypertension (pulmonary artery systolic pressure of 61 mmHg). There was no evidence of a ventricular septal defect. As auscultation of the lungs revealed mild bibasal crepitations, the sinus tachycardia was subsequently attributed to mild heart failure. However, despite diuretic therapy, there was no improvement.

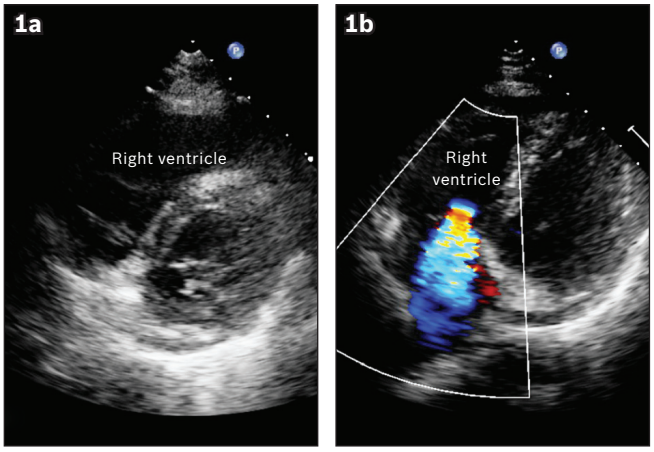

Fig. 1 Echocardiography images show (a) dilatation of the right ventricle and (b) severe tricuspid regurgitation.

In view of the persistent and unexplained sinus tachycardia, computed tomography pulmonary angiography was performed on Day 5 of admission. It showed multiple small bilateral pulmonary emboli, with prominence of the right heart chambers (Fig. 2). Doppler ultrasonographic examination of the patient's lower limbs showed no evidence of deep vein thrombosis. The patient was treated with low-molecular-weight heparin and warfarin. His tachycardia resolved and he was discharged on Day 10 of admission.

DISCUSSION

Our patient developed asymptomatic sinus tachycardia after admission for right ventricular myocardial infarction. Sinus

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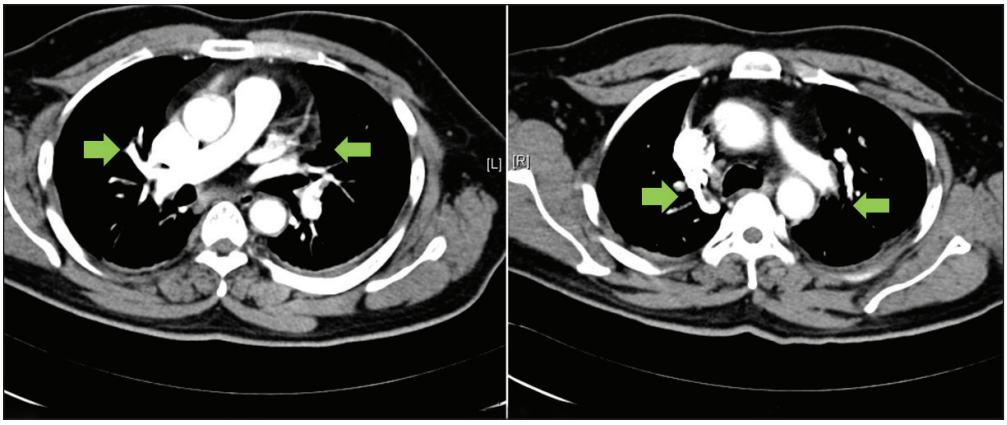

Fig. 2 CT pulmonary angiography images show multiple small bilateral pulmonary emboli (arrows).

tachycardia persisted in the patient even after common causes such as bleeding, infection and heart failure were excluded. Computed tomography pulmonary angiography revealed pulmonary embolism in the patient. However, pulmonary embolism is a very rare complication of right ventricular myocardial infarction,[1] and patients usually present with a new onset of pleuritic chest pain and/or dyspnoea.[1] Our case highlights that asymptomatic sinus tachycardia could be a presenting feature of pulmonary embolism, and that a high index of suspicion is warranted in order to detect this potentially lethal complication.

Sinus tachycardia is a common but nonspecific finding in patients with myocardial infarction. Heart failure, bleeding, pain, anxiety and concomitant infection are common attributing factors. There are many overlapping features between myocardial infarction and pulmonary embolism. While thromboembolism arising from a left ventricular infarct usually presents with distinct clinical syndromes such as stroke[2] or acute lower limb ischaemia,[3] clinical syndromes of thromboembolism from a right ventricular infarct leading to pulmonary embolism is often indefinite. Diagnosing pulmonary embolism complicating a right ventricular infarction could be challenging, and it is plausible that many cases are left unrecognised.

No large study has reported on the incidence of concomitant pulmonary embolism and right ventricular infarction because of the rarity of the association. The literature is unclear on the causal relationship between the two. In our case study, right ventricular infarction was likely to be the cause of pulmonary embolism rather than vice versa because an occlusive stenosis was detected on coronary angiography. Making the diagnosis of pulmonary embolism is crucial for improving patient outcomes. Early therapy is effective, but failure to treat can be fatal, with three-month mortality rates documented to be up to 20%.[4] Untreated pulmonary embolism can increase pulmonary vascular resistance and lead to haemodynamic compromise by impairing right ventricular function. The condition consequently diminishes left ventricular function by decreasing its preload. This can be particularly relevant in patients with right ventricular infarction. Right ventricular dilatation and dysfunction is a bad prognostic marker after pulmonary embolism. In contrast, the prognostic implication of pulmonary embolism complicating a right ventricular myocardial infarction is unclear. However, it is conceivable that the pulmonary embolism would further compromise left ventricular filling and worsen the prognosis of myocardial infarction.

Pathophysiologically, it is believed that impaired circulation in a dilated and poorly contractile, infarcted right ventricle predisposes a person to thrombi formation and embolisation.[1] The absence of deep vein thrombosis in our patient supports, but does not confirm, this hypothesis. Although no right ventricular thrombi were detected in our patient on echocardiography, it has been reported that unenhanced echocardiography could be insensitive to the detection of thrombi, and that contrast echocardiography or cardiac magnetic resonance imaging may be needed for detecting right ventricular thrombi.[5] In our patient, these modalities were not pursued, as the diagnosis of pulmonary embolism had already warranted anticoagulation therapy, and the presence or absence of right ventricular thrombi would not have altered our management.

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