OMCR

Oxford Medical Case Reports, 2018;6, 183–186

doi: 10.1093/omcr/omy019
Case Report

CASE REPORT

Giant isolated intracardiac thrombus presenting as acute heart failure secondary to right ventricular outflow tract obstruction in a patient with renal carcinoma

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Abstract

Right-sided intracardiac thrombi are potential causes of right ventricular (RV) failure, particularly when tricuspid or pulmonary obstruction occurs. In most cases, RV thrombus develops in patients with RV dysfunction and concomitant thrombosis in the systemic veins. However, RV thrombosis can rarely present as an isolated mass and despite preserved RV function, particularly in patients with thrombophilic states. In this report, we describe an unusual case of giant isolated RV thrombus presenting with acute RV failure secondary to dynamic RV outflow tract obstruction in a patient with renal carcinoma. Bedside echocardiography allowed a rapid assessment of the hemodynamic effects of the mass. The possibility of a thrombotic RV outflow obstruction should be considered in patients with acute RV failure, even in those with no evidence of thrombosis in the venous district. This may be particularly important in patients with prothrombotic states, where the effectiveness of routine thromboembolic prophylaxis could be reduced.

INTRODUCTION

Right-sided intracardiac thrombi are potential causes of right ventricular (RV) failure, particularly when tricuspid or pulmonary obstruction occurs [1]. In most cases, RV thrombus develops in patients with RV dysfunction and concomitant thrombosis in the systemic veins. However, RV thrombosis can rarely present as an isolated intracardiac mass and in the presence of preserved RV function, particularly in patients with thrombophilic states [2]. We describe an unusual case of giant isolated RV thrombus presenting with acute RV failure secondary to dynamic RV outflow tract obstruction in a patient with renal carcinoma.

CASE REPORT

A 51-year-old man presented to the Emergency Department complaining chest discomfort and dyspnea. His cardiovascular history was unremarkable except for smoking and emphysema.

Received: November 6, 2017. **Revised:** March 31, 2018. **Accepted:** April 19, 2018

Seven weeks earlier, because of persistent fever, cough and evidence of anemia, he had undergone chest–abdomen computed tomography (CT), with evidence of a solid mass (7 × 6.5 cm) in the left kidney. Brain CT scan, bone scintigraphy, colonoscopy and esophagogastroduodenoscopy had shown no significant findings. Two weeks before the current presentation, the patient had been submitted to left radical nephrectomy with regional lymphadenectomy. An echocardiogram performed at the time of the intervention had shown no significant abnormalities. The patient had been discharged 6 days after surgery, with the recommendation of continuing enoxaparin 4000 IU once daily, amoxicillin–clavulanic acid, lansoprazole, folic acid and antalgic treatment with oxycodone/naloxone and paracetamol/codeine.

On admission, the patient was in poor general conditions, his pulse rate was 135 beats/min, and blood pressure was 105/70 mmHg. The electrocardiogram showed sinus tachycardia. Blood examinations showed leucocytosis (23.29×10^3/μL, normal range: 4.40–10.10×10^3/μL), anemia (Hb 8.8 g/dL, normal range: 13.2–17.0 g/dL), thrombocytosis (734×10^3/μL, normal range: <150–400×10^3/μL), and increased C-reactive protein (46.3 mg/dL, normal range: 0–0.5 mg/dL). Blood gas analysis revealed hypoxemia–hypocapnia. Ultrasound examination showed no evidence of venous thrombosis. Echocardiography showed a large mass (56 × 33 mm) attached to the anterior wall of the RV outflow tract, characterized by irregular shape, inhomogeneous echogenicity, and no evidence of vascularization (Fig. 1, Movies 1–4). Doppler examination of RV outflow tract showed a peak subpulmonary gradient of 21 mmHg at rest. During forced expiration, the peak gradient increased up to 35 mmHg (Fig. 2, Movies 5), suggesting dynamic RV outflow obstruction. There were also right atrial and ventricular enlargement with preserved systolic function (TAPSE 24 mm, tricuspid s' 19 cm/s), severe tricuspid regurgitation with pulmonary hypertension (52 mmHg), and reduced inspiratory collapse of the inferior vena cava. CT pulmonary angiography (Fig. 3) confirmed the presence of a large mass obstructing the RV outflow tract, with irregular shape and no evidence of vascularization, suggesting RV thrombus. There were no emboli in the pulmonary vessels, but multiple pulmonary and hepatic metastases were observed. No thrombosis was found in the systemic veins. Because of the recent history of surgery and the oncological prognosis, thrombolysis or surgery were not indicated. The patient was hospitalized and therapy with full-dose enoxaparin, fluids, antibiotics and steroids was started. Ten days after admission, an echocardiogram showed no significant changes, except for a mild improvement in the degree of tricuspid regurgitation (moderate) and a slight reduction in mass dimensions (52 × 30 mm) and RV outflow tract gradient at rest (16 mmHg). The patient was discharged 1 week later on anticoagulation, but died 2 days thereafter.

DISCUSSION

RV thrombosis is a potentially life-threatening condition that can result from several underlying disorders, and that is usually associated with RV dysfunction [1]. The detection of an isolated RV thrombus is a relatively rare event in patients with

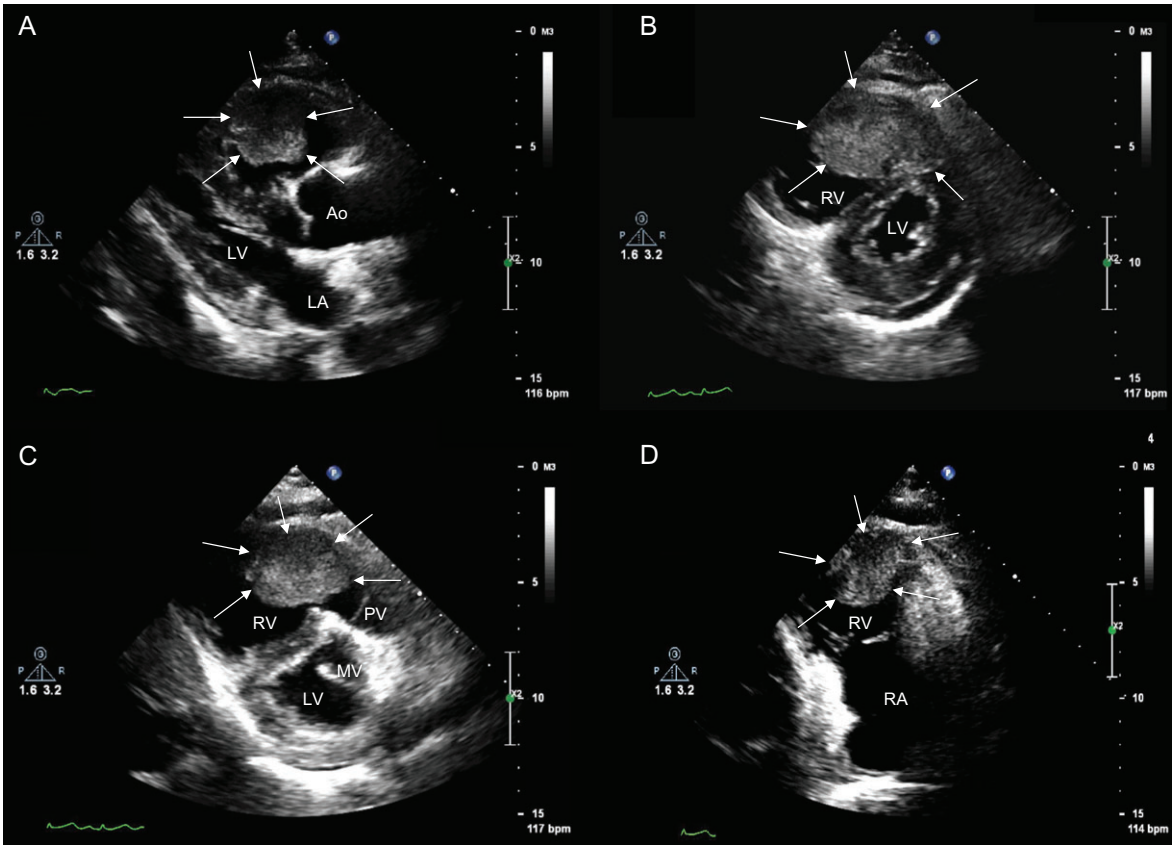

Figure 1: Echocardiographic images showing the right ventricular mass (white arrows) from the long-axis parasternal view (**A**), short-axis parasternal views (**B** and **C**), and parasternal right ventricular inflow view (**D**). AO= aortic root; LA=left atrium; LV=left ventricle; MV=mitral valve; RA=right atrium; RV=right ventricle

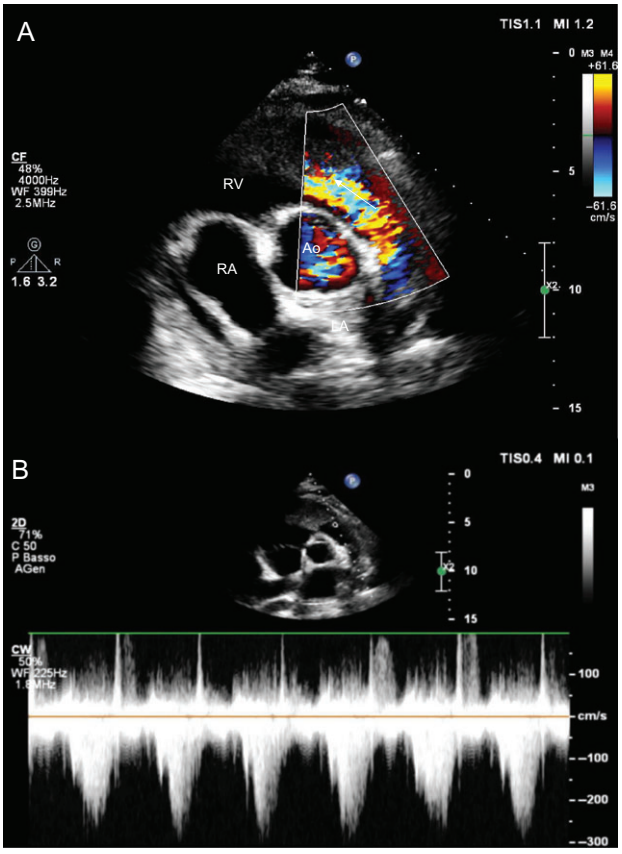

Figure 2: *Top panel*: Color Doppler image from the parasternal short-axis at the level of great vessels, showing aliasing in the right ventricular outflow tract. *Bottom panel*: pulsed Doppler image confirming increased velocity in the right ventricular outflow tract

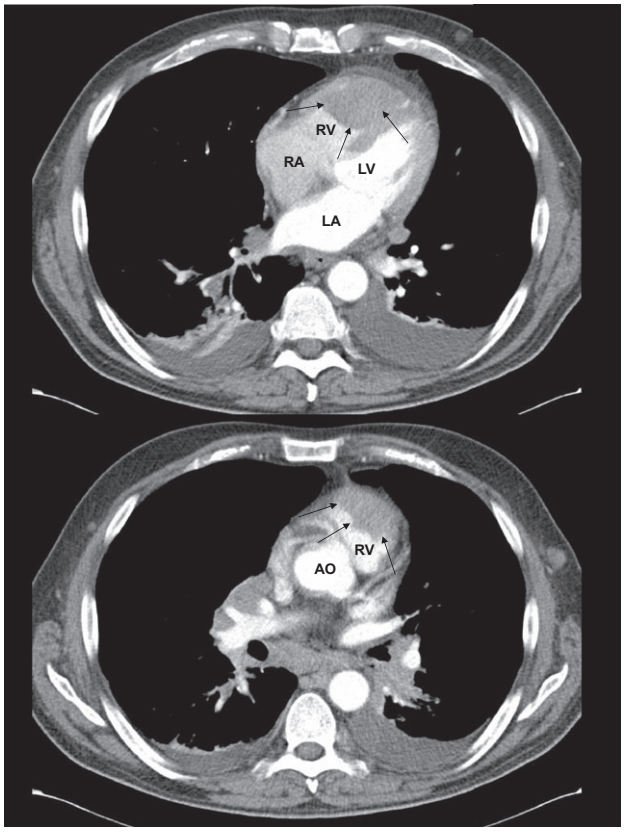

Figure 3: Computed tomography images showing the mass (black arrows) at the level of right ventricular inflow (top panel) and outflow (bottom panel). AO= aortic root; LA=left atrium; LV=left ventricle; RA=right atrium; RV=right ventricle

preserved RV systolic function, particularly when not associated with concomitant thrombosis in the systemic venous district. Isolated RV thrombi have been reported in the setting of RV myocardial infarction and arrhythmogenic RV dysplasia [3], Beçhet disease [4] and antiphospholipid antibody syndrome [5]. While neoplasms are associated with a thrombophilic state that increases the risk of cardiovascular events, isolated RV thrombi in cancer patients have been rarely described, in most cases resulting from extensive tumor-thrombus venous propagation [6].

This report describes a case of giant isolated RV thrombus presenting with acute RV failure secondary to RV outflow tract obstruction following left radical nephrectomy in a patient with renal carcinoma. Several atypical aspects should be pointed out in this case. First, the clinical presentation with acute heart failure secondary to dynamic RV outflow obstruction [7]. Second, the lack of thrombotic involvement of the systemic venous district, which in cancer patients commonly represents the pathway for thrombus propagation to the right ventricle. Third, the development of a large RV thrombus despite an apparently appropriate dose of enoxaparin, equal to that currently recommended by the 2014 ASCO guidelines for venous thromboembolism prophylaxis [8]. Lastly, the evidence of a rapid thrombus formation despite preserved RV systolic function, which highlights the importance of thrombophilic states in the pathophysiology of intracardiac thrombi [2]. On the other hand, although the echocardiographic and CT characteristics of the mass support its thrombotic nature, autopsy was not performed, so that no histological confirmation is available. Contrast echocardiography is a useful tool for the differential diagnosis of cardiac masses, and could have provided additional information about the avascularity of the mass [9]. It is also worthy of note that, for patients with no evidence of a concomitant acute cardiovascular event (e.g. myocardial infarction, pulmonary embolism), to date there are no specific guidelines for the management of intracardiac thrombi, and the relative efficacy of anticoagulation, thrombolysis or other approaches is still unclear [10]. Lastly, while the negativity of CT angiography makes the hypothesis of concomitant pulmonary embolism unlikely, this possibility cannot be completely excluded.

We believe that these issues could suggest clinical implications. The possibility of a RV outflow obstruction due to an isolated thrombus should be considered in patients with acute RV failure, even when there is no concomitant thrombosis in the systemic venous district. This may be particularly important in patients with neoplasms or other conditions associated with increased thromboembolic risk. Bedside echocardiography plays a fundamental role for prompt assessment in this context. Lastly, the effectiveness of standard prophylactic doses of enoxaparin might be reduced in patients with prothrombotic states.

SUPPLEMENTARY MATERIAL

Supplementary material is available at Oxford Medical Case Reports online.

ACKNOWLEDGEMENTS

None.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

FUNDING

There were no sources of funding for this article.

ETHICAL APPROVAL

Not needed for this type of article.

CONSENT

Written informed consent to publish this case was obtained.

GUARANTOR

All authors are the guarantors of this work.

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