INTRODUCTION

Spontaneous echocardiographic contrast (SEC) occurs because of abnormal blood flow velocity and predisposes to thromboembolism. Enlarged atrial size, mitral stenosis, and atrial arrhythmias are important risk factors associated with SEC. We report the case of a 20-year-old man diagnosed with constrictive pericarditis (CP) and significant SEC who was also found to have pulmonary embolism (PE).

CASE PRESENTATION

A 20-year-old Sudanese refugee was admitted with a 12-month history of progressive dyspnea, peripheral edema, and lethargy. Clinical examination revealed pronounced elevation in jugular venous pressure, hepatomegaly, and marked peripheral edema. Electrocardiography showed atrial flutter. Chest radiography revealed a calcified pericardium with minimal pulmonary congestion. He was previously healthy. Specifically, there was no history of tuberculosis.

D-dimer was elevated at 1.7 mg/L (normal range, <0.5 mg/L). Computed tomographic pulmonary angiography revealed extensive pericardial calcification and PE in the left upper and lower lobes (Figure 1).

Transthoracic echocardiography showed severely dilated left and right atria and a thickened calcified pericardium (Figure 2A, Video 1). Doppler findings were characteristic of CP. Interestingly, SEC was seen in all cardiac chambers (Figure 2B, Video 1). Similar finding was also appreciated on transesophageal echocardiography and was more pronounced in the right atrium and right ventricle (Figure 3, Videos 2-4). Bilateral lower limb Doppler ultrasonography did not demonstrate deep vein thrombosis as a potential source of PE. No underlying hypercoagulable state was identified.

Systemic anticoagulation was commenced, and the patient was referred for pericardiectomy. Histology revealed heavily calcified pericardium. Pericardial tissue cultures were negative for acid-fast bacilli, ruling out tuberculous CP, a common cause of CP in developing countries and an important diagnosis to consider in this patient.

DISCUSSION

SEC occurs because of low blood flow velocity and predisposes to clot formation and thromboembolism. Previous studies have established the relationship between left atrial SEC and systemic thromboembolism disease. Right atrial SEC has a much lower prevalence compared with left atrial SEC. In CP, increase in atrial size is commonly seen. However, significant enlargement is not common. The likely explanation for this finding in our patient is the lack of complete encasement of the heart, especially around the atria. Dilated atrium has been associated with SEC. In addition, Hjemdahl-Monsen et al. reported the case of a patient with CP with significant SEC in the inferior vena cava and right-sided cardiac chambers that resolved following pericardiectomy. In turn, the presence of SEC in the right atrium has been associated with increased risk for PE.

This is the first reported case of PE in a patient with CP. Dilated right atrium secondary to CP predisposed to SEC, a phenomenon associated with thromboembolism. This is the likely mechanism of PE in a patient with no other underlying hypercoagulable state.

Perhaps the presence of significant SEC in CP should prompt clinicians to assess for PE, especially when elevated D-dimer and significant dyspnea are detected. In our patient, systemic anticoagulation was commenced, and the patient underwent pericardiectomy. Unfortunately, this patient has not attended follow-up visits. It would be interesting to assess his echocardiographic findings for resolution of SEC following cardiac surgery.

CONCLUSION

SEC occurs because of blood stasis and is associated with an increased risk for thromboembolism. Previous studies have shown an association between left atrial SEC and systemic thromboembolism. Here, we report a case of PE in a young patient with CP and right atrial SEC and no underlying hypercoagulable condition. It is possible that right atrial dilatation secondary to CP led to SEC and subsequent right-sided thromboembolism.
Figure 1  Computed tomography showing (A) calcified pericardium (arrows) and (B) filling defects (arrows) consistent with PE.

Figure 2 Transthoracic echocardiography. (A) Parasternal long-axis view shows thickened pericardium (arrowhead). (B) Apical view shows SEC in all cardiac chambers.

Figure 3 Transesophageal echocardiography shows (A) SEC in bicaval view and (B) SEC and thickened pericardium (arrowheads) in four-chamber view. (C) Short-axis view with SEC in the right atrium and right ventricular outflow tract. (D) SEC seen in the left atrium and ventricle and right ventricular outflow tract in the long-axis view.
SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.case.2017.08.003.

REFERENCES

1. Hwang JJ, Ko FN, Li YH, Ma HM, Wu GJ, Chang H, et al. Clinical implication and factors related to left atrial spontaneous echo contrast in chronic non-valvular atrial fibrillation. Cardiology 1994;85:69-75.
2. Bashir M, Asher CR, Garcia MJ, Abdalla I, Jasper SE, Murray RD, et al. Right atrial spontaneous echo contrast and thrombi in atrial fibrillation: a transesophageal echocardiography study. J Am Soc Echocardiogr 2001;14:122-7.
3. Castello R, Pearson AC, Labovitz AJ. Prevalence and clinical implications of atrial spontaneous contrast in patients undergoing transesophageal echocardiography. Am J Cardio 1990;65:1149-53.
4. Black IW. Spontaneous echo contrast: where there’s smoke there’s fire. Echocardiography 2000;17:373-82.
5. Hjemdahl-Monsen CE, Daniels J, Kaufman D, Stern EH, Teichholz LE, Meltzer RS. Spontaneous contrast in the inferior vena cava in a patient with constrictive pericarditis. J Am Coll Cardiol 1984;4:165-7.
6. Yasuoka Y, Naito J, Hirooka K, Chin W, Miyatake K, Kusuoka H, et al. Right atrial spontaneous echo contrast indicates a high incidence of perfusion defects in pulmonary scintigraphy in patients with atrial fibrillation. Heart Vessels 2009;24:32-6.