# Spontaneous Coronary Sinus Thrombosis in a Patient With Pulmonary Embolism and No Prior Cardiac Instrumentation

Islam A. Ibrahim, MD, Jamilah Alharbi, MD, Ibrahim Alsaadi, MD, and Ashraf M. Anwar, MD, PhD, *Jeddah, Mecca, Saudi Arabia; and Cairo, Egypt*

## INTRODUCTION

Coronary sinus (CS) thrombosis is an exceedingly rare condition in which a thrombus forms in the veins that drain blood from the heart.[1] Coronary sinus thrombosis can be classified as either iatrogenic or spontaneous, with iatrogenic CS thrombosis being more common. Most instances are attributable to right heart instrumentation (e.g., right heart catheterization, cardiac resynchronization treatment, Ebstein anomaly correction, ventriculoatrial shunting, and radiofrequency transcatheter ablation).[2,3] Unlike iatrogenic CS thrombosis, spontaneous CS thrombosis is extremely uncommon, with just a handful of examples recorded in the medical literature. Here we describe a case of spontaneous CS thrombosis in a man recently diagnosed with heart failure with reduced ejection fraction.

## CASE PRESENTATION

A 64-year-old man with controlled hypertension and a history of a cerebrovascular ischemic accident with residual right-sided weakness presented with the chief complaints of exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea for 3 months. The patient denied having any additional symptoms. The physical examination revealed an ill-appearing man who was tachypneic with a respiratory rate of 22 bpm, tachycardic with a pulse rate of 124 bpm, and moderately hypoxic with 89 percent $O_2$ saturation on ambient air and without hemodynamic instability as their blood pressure was 117/70 mm Hg. An assessment of the cardiovascular system indicated elevated jugular venous pressure, 3+ pitting edema in the lower extremities, an S3 gallop, and bilateral basal lung crackles. Electrocardiogram showed sinus tachycardia with QRS duration of 100 ms. The blood test findings were all normal except for an elevated B-type natriuretic peptide level of 4,200 ng/L. Chest x-ray indicated pulmonary edema. The patient was hospitalized for pulmonary edema due to heart failure. Despite adequate intravenous diuresis (furosemide boluses), the dyspnea did not improve. Therefore, the patient was assessed urgently for pulmonary embolism (PE) using contrast-enhanced computed tomography (CT) pulmonary angiography and cardiovascular CT (CCT) split bolus-tracking combined protocol. It demonstrated bilateral pulmonary thrombosis, right-sided pleural effusion, right atrial enlargement, and a round-shaped hypodense-filling defect in the right atrium (RA) and CS junction measuring 14 mm × 18 mm representing a CS thrombus (Figures 1 and 2). It also showed normal coronaries in electrocardiogram gated images. Two-dimensional transthoracic echocardiography (2D TTE) was requested to assess the structure and function of the heart. Transthoracic echocardiography revealed severely impaired left ventricular systolic function with an ejection fraction of 15% by the modified Simpson's method. The right ventricle (RV) was markedly dilated (basal diameter, 59 mm; midcavity diameter, 48 mm) with severely impaired systolic function (right ventricular [RV] fractional area change: 20.5%, RV free wall long strain: −6.2%), and no regional RV wall motion abnormality was noted (Figure 3, Video 1). There was a mild to moderate central jet of tricuspid regurgitation with an estimated RV systolic pressure of 52 mm Hg. No hemodynamically significant valve disease was detected. The RA was also dilated (indexed area of 27.1 $cm^2$). The CS

### VIDEO HIGHLIGHTS

**Video 1:** Two-dimensional TTE, parasternal long-axis right ventricular inflow view demonstrates a large, mobile thrombus in the CS adjacent to the Eustachian valve.

**Video 2:** Two-dimensional TTE, parasternal long-axis right ventricular inflow view without (*left*) and with color-flow Doppler, demonstrates a large, mobile thrombus in the CS, adjacent to the Eustachian valve and tricuspid regurgitation.

**Video 3:** Two-dimensional TTE, apical 4-chamber view demonstrates a severely dilated right heart with RV systolic dysfunction; also noted is the dilated left ventricle with severely reduced systolic function.

**Video 4:** Three-dimensional (live) TTE, parasternal long-axis right ventricular inflow view, volume-rendered display, demonstrates the mobile thrombus in the CS.

**Video 5:** Three-dimensional TTE, parasternal long-axis right ventricular inflow view with 3D volume-rendered (tissue-enhanced) display (*top*) and 2D orthogonal long-axis (*bottom left*) and short-axis (*bottom right*) displays, demonstrates a large, mobile thrombus in the CS.

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From the Adult Cardiology Department, King Fahd Armed Forces Hospital, Jeddah, Saudi Arabia (I.A.I., J.A., I.A., A.M.A.); and Faculty of Medicine, Cardiology Department, Al-Azhar University, Cairo, Egypt (A.M.A.).

Keywords: Coronary sinus, Thrombosis, Echocardiography, Anticoagulation

Correspondence: Islam A. Ibrahim, MD, Adult Cardiology Department, King Fahd Armed Forces Hospital, Al Kurnaysh Road, Al Andalus, 23311 Jeddah, Mecca, Saudi Arabia. (E-mail: *Islam.adnan@hotmail.com*).



2468-6441
https://doi.org/10.1016/j.case.2023.05.005

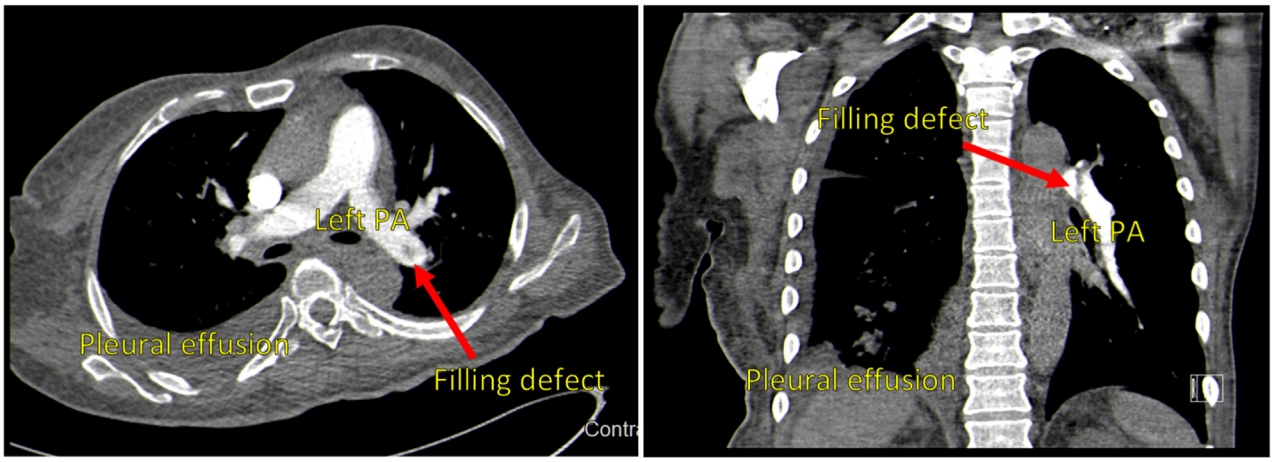


**Figure 1** Chest contrast-enhanced CT in axial and coronal displays demonstrates a filling defect (*arrow*) in the distal left main pulmonary artery (PA).

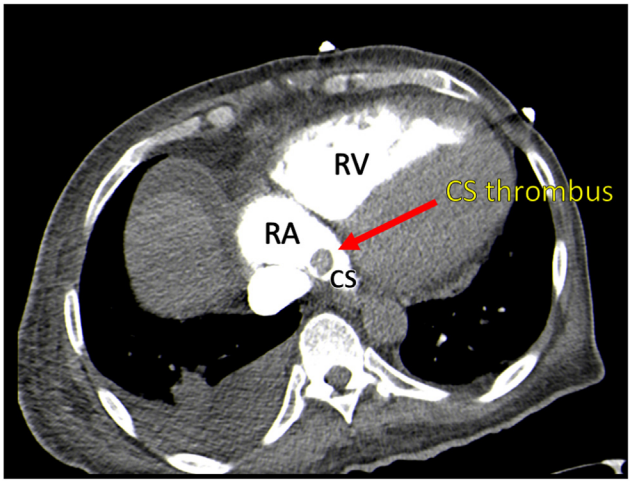


**Figure 2** Chest contrast-enhanced computed tomography at the level of the CS demonstrates a round-shaped hypodense-filling defect (*arrow*) in the RA and CS junction measuring 14 mm × 18 mm representing a CS thrombus. Also seen is a right-sided pleural effusion.

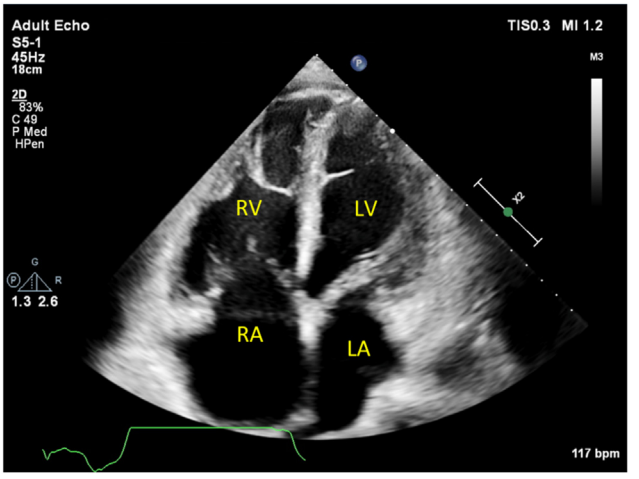


**Figure 3** Two-dimensional TTE, apical 4-chamber view, diastolic phase, demonstrates a dilated right heart. *LA*, Left atrium; *LV*, left ventricle.

was mildly dilated (diameter, 2.1 cm) with an echo dense mobile mass measuring 15 mm × 15 mm seen at its orifice highly suggestive of an acute thrombus. No other structure (e.g., valve, septum) was seen at the CS. The jet direction of the tricuspid regurgitation was away from the CS orifice (Figures 4 and 5, Videos 2 and 3). Three-dimensional TTE was performed for additional information. It showed the freely mobile mass with partial echolucency at its center. The mass attached to the posterior wall of the CS without stalk (Figures 6 and 7; Videos 4 and 5).

In consultation with the hematologists, anticoagulation with rivaroxaban was started (15 mg twice daily for 21 days, followed by 20 mg once daily) as therapy for both CS thrombosis and PE. Antifailure medications for nonischemic cardiomyopathy—heart failure with reduced ejection fraction—were started and titrated. Over the next 4 days of admission, the patient's condition improved enough for a safe discharge home in stable condition. One week later, the patient was seen in the heart failure clinic and reported mild dyspnea with moderate exercise on the New York Heart Association II scale, prompting an adjustment to the heart failure with reduced ejection fraction medications. The patient was then given follow-up appointments to maximize the medical therapy, followed by repeat TTE, and to undergo an assessment for implantable cardioverter-defibrillator implantation.

## DISCUSSION

The CS is responsible for the venous outflow of the myocardium. After receiving tributaries from the small, medium, oblique, and great cardiac veins, the CS traverses the coronary sulcus and empties into the RA between the entrance of the inferior vena cava and the septal leaflet of the tricuspid valve.[4] The CS is a therapeutically significant structure due to its suitability for delivery of several heart treatments. While CS thrombosis after cardiac instrumentation is well-documented, the clinical presentation, therapy, and prognosis of

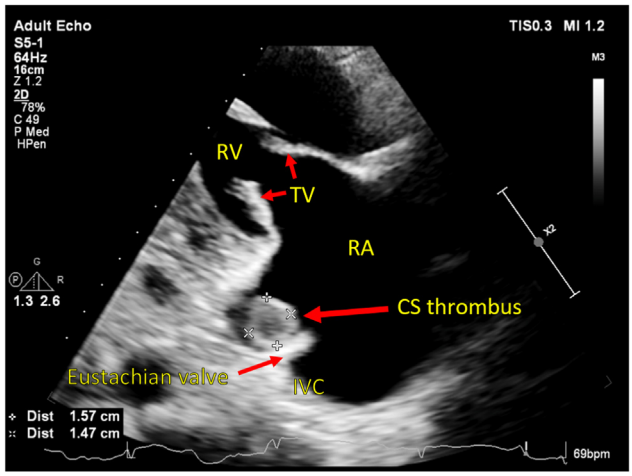


**Figure 4** Two-dimensional TTE, parasternal long-axis, right ventricular inflow view, diastolic phase, demonstrates a large thrombus in the CS (*arrow*), with imaging features suggesting this may be acute (hypoechoic center). *IVC*, Inferior vena cava; *TV*, tricuspid valve.

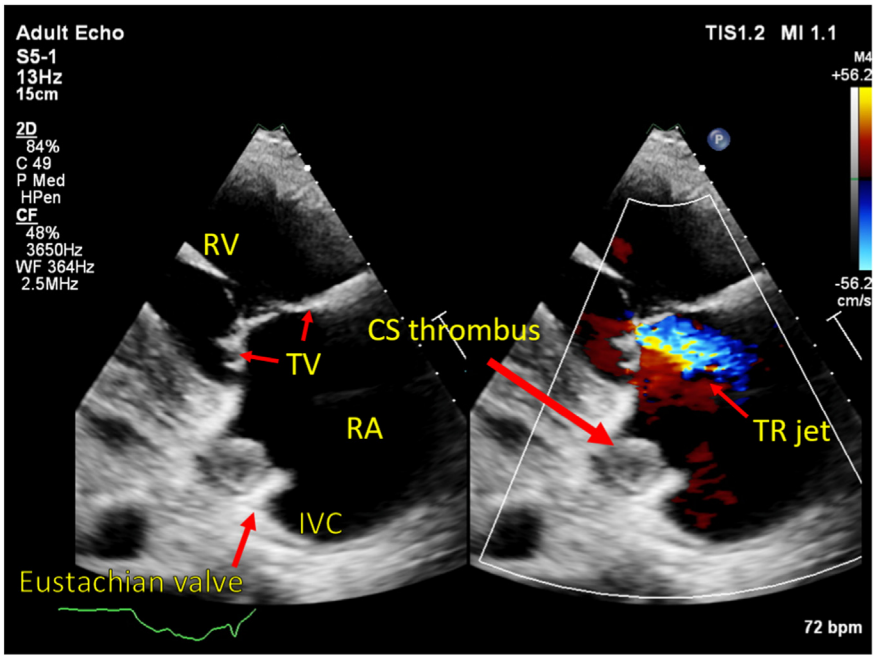


**Figure 5** Two-dimensional TTE, parasternal long-axis right ventricular inflow view, systolic phase without (*left*) and with (*right*) color-flow Doppler, demonstrates a large thrombus (*long arrow*) in the CS. The TV leaflets (*small arrows*), the Eustachian valve (*short arrow*), and TR (*arrow*) are also seen on the image. *TR*, tricuspid regurgitation; *TV*, tricuspid valve.

spontaneous CS thrombosis are not well studied, with just a handful of examples recorded in medical literature as summarized in Table 1.

One or more of Virchow's triad components (stasis, endothelial damage, and hypercoagulability) can contribute to CS thrombosis. In most cases of iatrogenic CS thrombosis, it is believed that endothelial injury is the primary cause of thrombus development.[14] In the case of spontaneous CS thrombosis, the pathogenesis is unclear, with published data showing different underlying pathological processes with a predominance of inflammatory processes.[5,8-11] In asymptomatic patients, CS thrombosis is often detected incidentally by contrast-enhanced CT scan or angiography performed to evaluate other diagnoses. A TTE may reveal a dilated RA with dilated CS. Infrequently, CS thrombus in symptomatic or severely ill patients can be detected by 2D TTE and 3D TTE.[14] Transthoracic echocardiography can be utilized to evaluate the CS in multiple windows. In the parasternal long axis, it appears as a mobile echolucency that narrows during diastole in sinus rhythm. It is located posterior to the left atrioventricular junction and anterior to the descending aorta, although its margins are sometimes ill-defined, and its caliber cannot be determined accurately. In the apical 2-chamber and 4-chamber views, M mode permits more precise measurements of both size and diameter.[15] Our patient's 2D TTE and 3D TTE were sufficient to diagnose CS thrombosis.

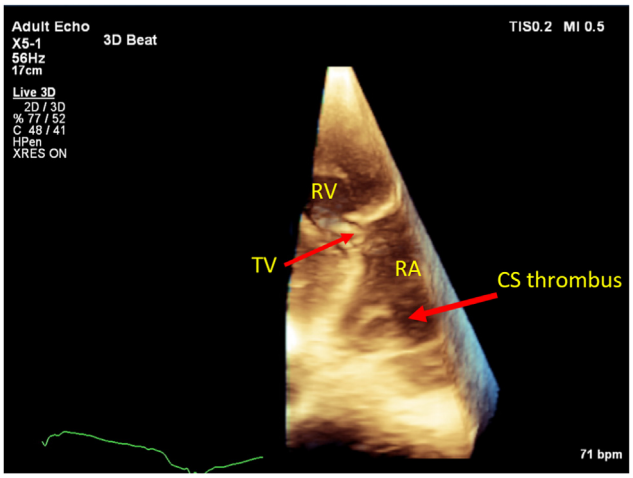


**Figure 6** Three-dimensional TTE, parasternal long-axis right ventricular inflow view, diastolic phase, demonstrates a large thrombus (*arrow*) in the CS with imaging features suggesting this may be acute (hypoechoic center). *TV*, Tricuspid valve.

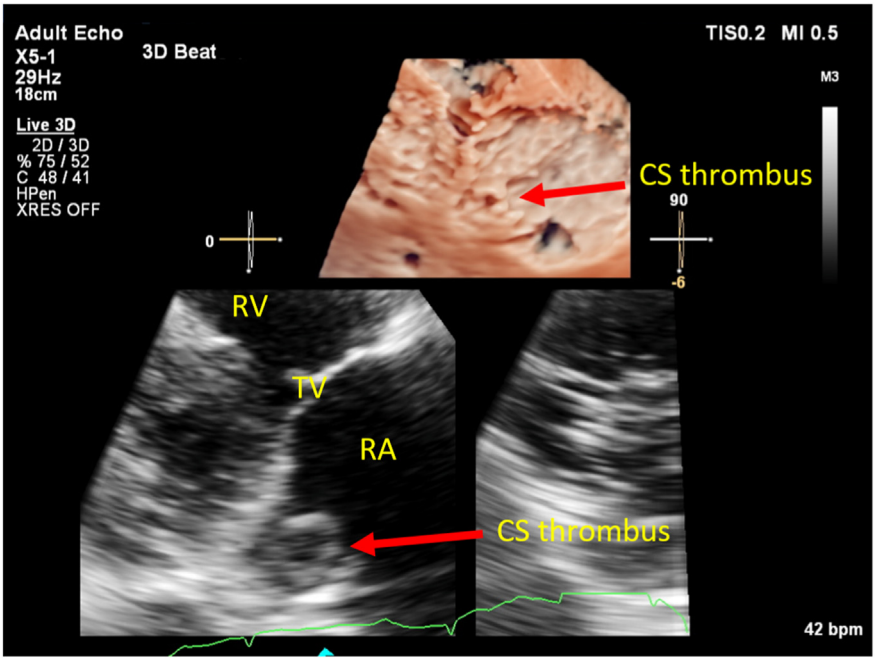


**Figure 7** Three-dimensional TTE, parasternal long-axis right ventricular inflow view, diastolic phase with 3D volume-rendered (tissue-enhanced) display (*top*) and 2D orthogonal long-axis (*bottom left*) and short-axis (*bottom right*) displays, demonstrates a large thrombus in the CS (*arrow*). *TV*, Tricuspid valve.

Transesophageal echocardiography (TEE) can more precisely visualize CS thrombus in the modified midesophageal 4-chamber view and the bicaval view than TTE. Due to the lack of definitive tissue characterization, its utility in distinguishing thrombus from tumor is limited. However, it permits precise evaluation of the size, location, and characteristics of atrial masses. In our case, TEE was not performed due to satisfactory image quality obtained by 2D and 3D TTE.[14] Cardiovascular CT, when combined with other modalities, can confirm the diagnosis and exclude other possible differential diagnoses such as tumors. It can detect CS thrombus, which in our case was determined to be a filling defect at the junction of the RA and CS. The use of cardiovascular magnetic resonance imaging to evaluate intracardiac thrombi is a developing indication. There are no data comparing the diagnostic utility of TTE, TEE, CCT, and cardiovascular magnetic resonance in CS thrombosis. The choice of imaging modalities should be based on the clinical presentation and the availability of multimodal imaging expertise.

The management of CS thrombosis is not established yet regarding drug selection, dose, and duration of therapy. The criteria for resolution of CS thrombosis are uncertain. Due to the association of CS

**Table 1** Summary of reported cases of CS thrombus in chronological order

| Clinical presentation | Age | Sex | Left ventricular function | Clinical diagnosis | Outcome | Case report |
|---|---|---|---|---|---|---|
| General fatigue | 83 years | Male | Not mentioned | Cardiac tamponade | Deceased | Kitazawa *et al.* 2009[5] |
| Exertional dyspnea | 50 years | Male | 20% | Nonischemic cardiomyopathy | Treated with warfarin for a month. | Güvenç *et al.* 2012[6] |
| Bloating | 23 years | Female | Not mentioned | Severe tricuspid regurgitation and congestive hepatopathy | Aspirated during surgical TVR. | Liu *et al.* 2014[7] |
| Fever, cough, and diarrhea | 3 months | Male | Not mentioned | Kawasaki disease coronary artery aneurysm | Treated with LMWH (IV) for 6 days. | Song *et al.* 2015[8] |
| Chest pain and worsening dyspnea | 76 years | Male | Not mentioned | Atrial fibrillation with rapid ventricular response | Treated with UFH (intravenous) and DOAC for 7 days. | Floria *et al.* 2016[9] |
| Abdominal pain, nausea, vomiting, and diarrhea | 27 years | Male | Not mentioned | Psoas muscle abscess secondary to Crohn's disease | Deceased. | Martin *et al.* 2017[10] |
| Acute abdominal pain, and nausea | 61 years | Female | 15%-20% | Congestive hepatitis secondary to decompensated heart failure | Treated with warfarin for 3 months. | Hart *et al.* 2017[11] |
| Acute encephalopathy and weight loss | 72 years | Male | Not mentioned | Persistent left superior vena cava and CS thrombosis | Treated with rivaroxaban. | Moey *et al.* 2018[12] |
| Worsening weakness | 64 years | Female | 60% | Hepatobiliary CA and bacteremia secondary to infected ascites | Patient opted for comfort care. | Mararaenko *et al.* 2021[1] |
| Dyspnea and chest pain | 67 years | Male | 60%-64% | Coronary artery disease with severe mitral regurgitation | Not mentioned. | Ojukwu *et al.* 2021[13] |

*CA*, Cervical adenocarcinoma; *LMWH*, low-molecular-weight heparin; *TVR*, tricuspid valve replacement; *UFH*, unfractionated heparin.

thrombus with PE, it is contemplated that blood stagnation is the underlying mechanism in addition to the absence of cardiac instrumentation. In conjunction with hematologists, we elected to prescribe the direct oral anticoagulant (DOAC) rivaroxaban as a treatment of both PE and CS thrombosis.

## CONCLUSION

A spontaneous CS thrombosis has a potential association with the occurrence of PE. Clinicians can detect and verify a CS thrombus using 2D TTE, either alone or in conjunction with 3D TTE. Due to its superior tissue characterization capabilities, CCT plays a crucial role in confirming a CS thrombosis diagnosis while also eliminating potential differential diagnoses. Although DOACs have demonstrated safety in treatment, further research is essential in determining the optimal anticoagulation regimen—the class, dosage, and length of treatment.

## ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

## CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

## FUNDING STATEMENT

The authors declare that this report did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

## DISCLOSURE STATEMENT

The authors report no conflict of interest.

## SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2023.05.005.

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