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INTERMEDIATE

CASE REPORT: CLINICAL CASE

Late Iatrogenic Coronary Sinus Hematoma During Cardiac Surgery

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ABSTRACT

Retrograde cardioplegia is commonly used in cardiac surgery to induce cardioplegic arrest. However, this method could be potentially associated with coronary sinus injuries, which can be fatal or extremely difficult to manage. This report describes the conservative management of an iatrogenic coronary sinus hematoma by daily transthoracic echocardiography and weekly computed tomography follow-up. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:649-654) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 63-year-old man was referred to our attention because of severe aortic regurgitation in the bicuspid aortic valve and ascending aortic aneurysm. He first presented to the emergency department because of worsening dyspnea. His arterial blood pressure was 130/50 mm Hg, his blood oxygen saturation was 96%, his pulse rate was 101 beats/min, and his body temperature was 36.8 °C. Physical examination revealed tachypnea, diffuse bilateral crackles throughout the lung fields; increased heart rate and regular rhythm, with decrescendo early diastolic blowing murmur. Echocardiography confirmed the diagnosis. Coronary angiography showed 90% stenosis of the proximal tract of the left anterior descending artery. The patient's medical history was not relevant.

LEARNING OBJECTIVES

- To be able to make a differential diagnosis in case of iatrogenic coronary sinus injury.
- To understand the main echocardiographic features of a self-limiting wall hematoma.
- To be able to set the best decision-making process.
- To consider not to treat the lesion, in selected cases, and to set a strict follow-up through advanced multimodality imaging techniques.

The patient underwent elective surgery. A single coronary artery bypass with left internal mammary artery to left anterior descending artery, aortic valve, and ascending aorta replacement was performed. Cardioplegic arrest was induced through antegrade cardioplegia delivery (Haematic) in the aortic root and retrograde through a coronary sinus (CS) cannula (LivaNova), monitoring a constant optimal perfusion pressure.

After weaning from cardiopulmonary bypass, after removal of heart cannulation, and during protamine infusion, transesophageal echocardiography (TEE)

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ABBREVIATIONS AND ACRONYMS

CS = coronary sinus

CSH = coronary sinus hematoma

CT = computed tomography

EWD = endocardial wall dissection

FU = follow-up

IAWH = intramural atrial wall hemorrhage

TEE = transesophageal echocardiography

TTE = transthoracic echocardiography

revealed an intramural lesion located on the inferior atrial wall, showing a progressive increase in size, reaching approximatively 30 mm × 20 mm (**Figure 1,** Video 1).

DIFFERENTIAL DIAGNOSIS

The intraoperative differential diagnoses included free wall rupture, endocardial wall dissection (EWD), intramural atrial wall hemorrhage (IAWH), and CS hematoma (CSH). Free wall rupture was excluded because of the absence of pericardial bleeding. EWD and IAWH were indeed the very first manifestations of the lesion, as clearly appreciable in **Figure 2A** (Video 2), where an anechogenic space was interposed between the endocardium and the muscle wall. No ruptures of the endocardial wall were reported, consistent with the absence of a color Doppler signal in the lumen of the lesion (**Figures 3A and 3B**). Later, the imaging allowed for better characterization, showing the lesion to have a typical hematoma-like echocardiographic pattern, namely, hyperechogenicity of the injured wall thickness, in the absence of blood flow replenishment. (**Figure 3,** Video 3).

INVESTIGATIONS

Intraoperative investigations were limited to real-time TEE and surgical inspection. Immediately after the procedure, CT showed a hypodense thickening of the lateral wall of the left atrium, in the arterial and venous phases (**Figure 4**), to confirm the diagnosis of CS hematoma. Afterwards, from postoperative day 1 until discharge, daily transthoracic echocardiography (TTE) was performed. The patient underwent CT again on postoperative days 6 and 30. We did not perform any magnetic resonance evaluation because of the firmness of the diagnosis provided by the first CT and because of the stability and progressive resolution of the lesion, as reported by daily TTE follow-up. Whether uncertainty about the diagnosis would have been still present, magnetic resonance could have been performed to obtain superior tissue characterization and image resolution, to discriminate between intramural dissection, CS thrombosis, and intramural hematoma.[1]

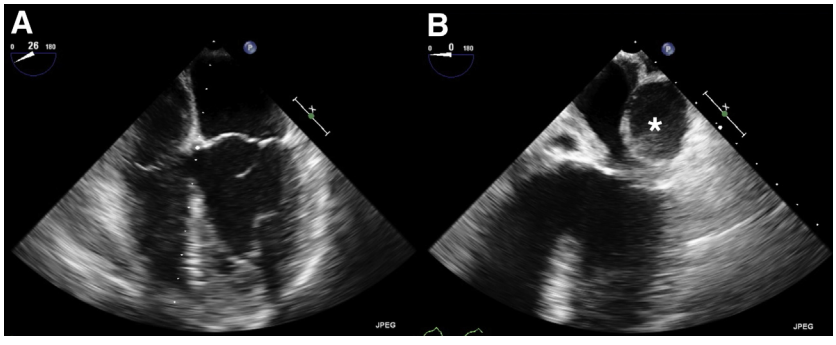
MANAGEMENT

The initial decision-making process, which considered whether to treat the lesion or leave it untreated, was soon encouraged by the absence of any echocardiographic modification of the filling pattern of the LA without any involvement of the mitral valve or pulmonary veins and the consequent hemodynamic stability of the patient. Therefore, a final wait-and-see strategy was undertaken

DISCUSSION

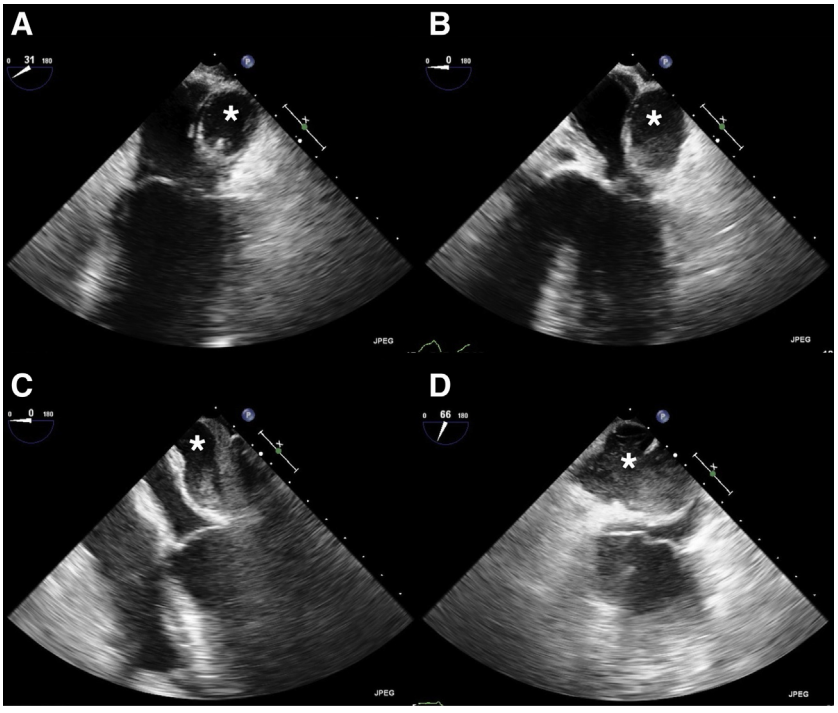
This report describes the conservative management of an iatrogenic coronary sinus hematoma, monitored by multimodality imaging techniques. The advantages of retrograde cardioplegia have been well documented,[2] but there have been several reports of complications caused by this technique.[3] CS injury with a retrograde-cardioplegia catheter is the major complication that can occur and can be recognized

FIGURE 1 Intraoperative Echo Imaging of the Lesion



(A) Before protamine administration, no sign of mural dissection or hematoma is evident. **(B)** After protamine administration, a bulging lesion is evident in the left atrial wall thickness **(asterisk)**.

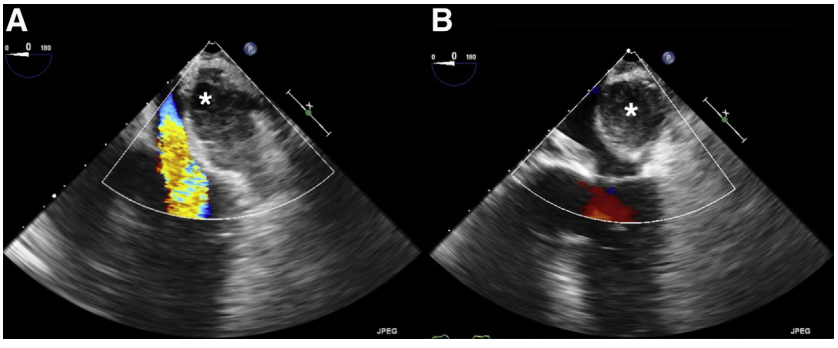
FIGURE 2 **Intraoperative Echo Evolution of the Coronary Sinus and Atrial Wall Hematoma**



The initial lesion **(A)** is clearly appreciable in the left atrial wall thickness and increases progressively in size **(B)** until it causes compression of the left atrial chamber **(C)** and almost complete obliteration **(D)**.

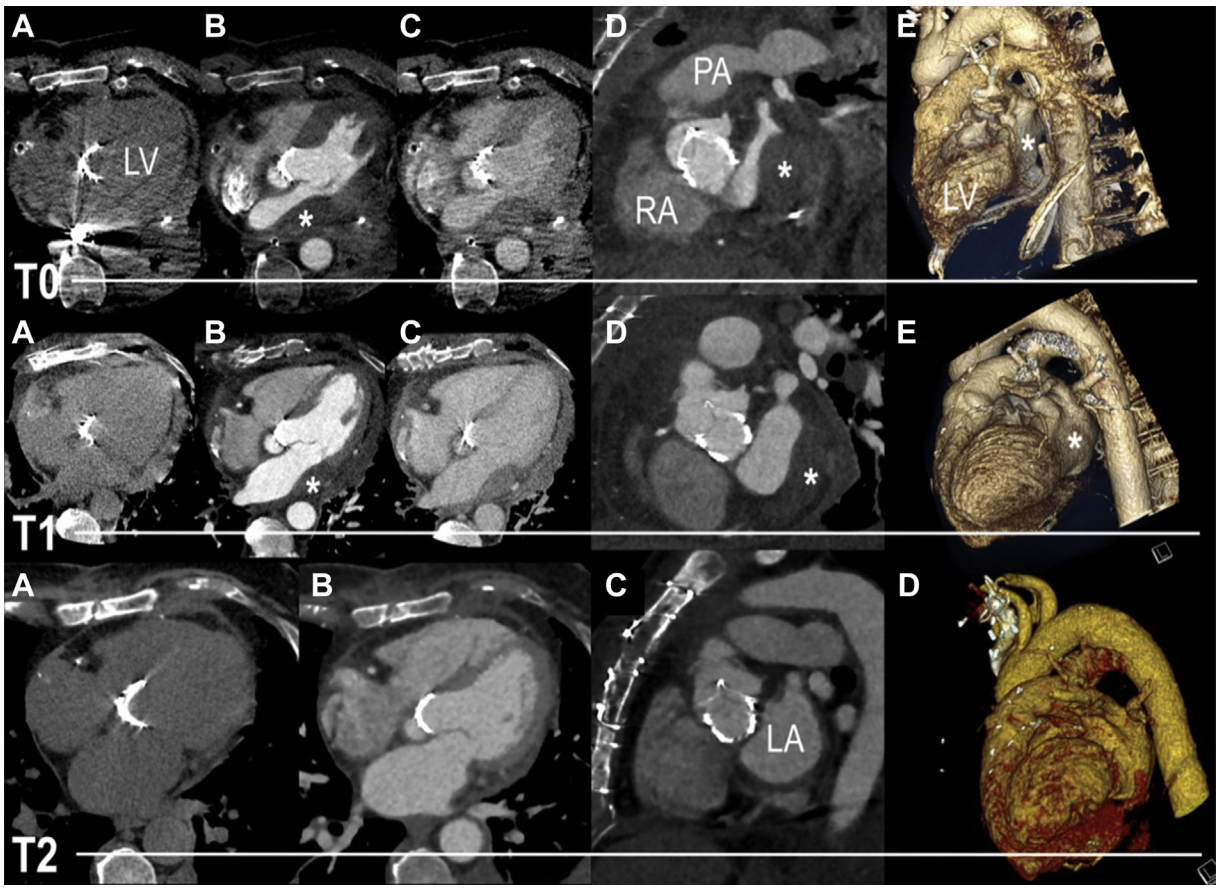
directly in case of external perforation or by the use of TEE in case of endocardiac hematoma formation. A blind insertion technique was used in this case; the right atrium was punctured, and the CS was cannulated with the self-inflatable balloon retrograde cannula. The correct positioning of the cannula was assessed by direct palpation, echocardiographic evaluation, and CS pressure monitoring.

FIGURE 3 **Echocardiographic Diastolic and Systolic Frames**



(A) Diastolic phase: in this frame, the lesion is excluded from transatrial blood flow, with no evidence of endocardial wall rupture or mitral stenosis. **(B)** Systolic phase: the lesion partially obliterates the left atrium, but it does not involve the mitral valve apparatus, causing no regurgitation.

FIGURE 4 Computed Tomography Evolution of the Atrial Hematoma

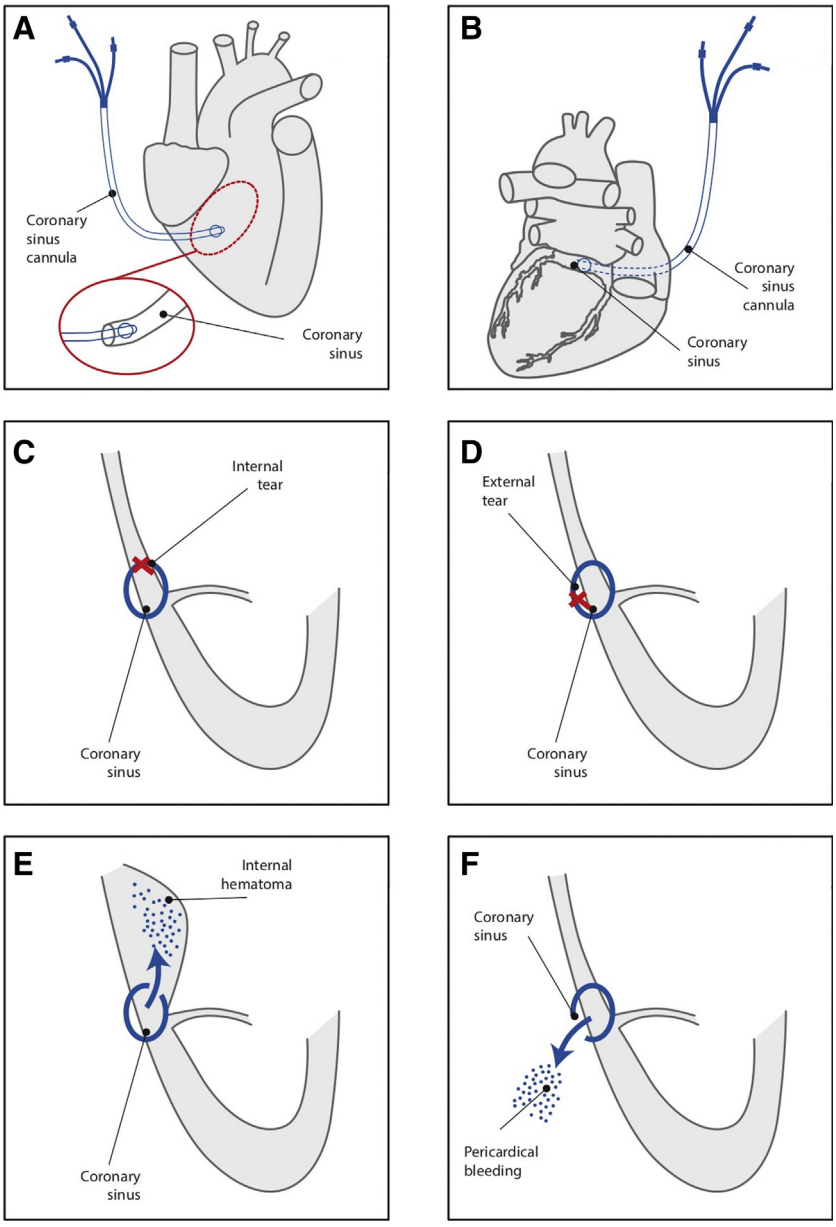


At time 0 (T0, **upper row**), computed tomography before contrast material was given **(A)** and in the arterial **(B)** and venous **(C)** phases clearly shows a hypodense thickening of the lateral wall of the left atrium (LA) **(asterisk, B)**; the hematoma significantly obliterates the LA cavity as displayed in a short-axis view **(asterisk, D)** and using 3D volume rendering **(asterisk, E)**. At time 1 (T1, **middle row**, 1 week) the hematoma is still present **(asterisk, B to E)** but significantly reduced in global volume and with much less mass effect on the lateral wall of the left ventricle (LV). At time 2 (T2, **lower row**, 6 weeks) the hematoma is completely reabsorbed in all images and projections. RA = right atrium.

Differently from previous reported cases,[2] we did not observe CSH occurrence immediately after the CS cannulation, but at the end of the procedure, after the CS cannula removal. This supports the hypothesis that the cannula provoked an iatrogenic laceration of the CS, located on the atrial wall face, and the expanding balloon had been occluding the tear, preventing the development of other injuries during the procedure. Once the cannula was removed, the blood flow provoked IAWH and subsequent EWD, followed by self-limiting CSH (Figure 5). Intrapericardial hemorrhage would have occurred if the laceration had been on the epicardial face of the CS (Figure 5).

A few cases of late intraoperative injuries (after cardiopulmonary bypass weaning) are described, as well as their postoperative management. Unlike CS external laceration, which is a clearly diagnosed condition resulting from massive pericardial bleeding and requires immediate intervention, in most cases CSH does not show up in a clear and well-defined pattern. Therefore, the decision-making process is initially influenced by the built-in uncertainty of the diagnosis, which is time-consuming and

FIGURE 5 **Coronary Sinus Injury: Consequences Based on the Tear Location**



(A, B) Schematic illustration of the coronary sinus cannulation site, from anterior and posterior views, respectively. **(C, D)** Schematic representation of the coronary sinus tear site, internally or externally, respectively. **(E)** An internal tear on the atrial wall of the coronary sinus provokes an intramural hemorrhage, potentially evolving in an atrial wall hematoma. **(F)** An external tear on the epicardial wall of the coronary sinus provokes pericardial bleeding.

has to take into account, first, the hemodynamic impact the hematoma provokes, by compression or tamponade of the cardiac chambers, and second, the extent of the lesion and the potential complications of the evolving process, ie, EWR. In the reported case, the hematoma progressively developed until it assumed a definite and stable pattern over time, which was thus encouraging for a non-further-evolving scenario. For this reason, the strategy to conservatively manage the lesion was adopted in the operating room and during the hospitalization.

The causes of CSH during surgery/transcatheter procedures have not been fully described. They can include tissue frailty, complications during the CS ostium cannulation, overpressuring, or balloon overdilation, and they could also overlap in the lesion pathogenesis.[4] In case of difficulties experienced during cannulation of the CS, and if repeated careful trials are attempted, direct vision CS cannulation should be considered an alternative strategy to minimize the risk of CS damage. However, avoiding the retrograde technique may be another option.[4]

FOLLOW-UP

After discharge, the patient underwent 1-week follow-up TTE, confirming the stability of the lesion. At the 1-month CT follow-up, complete resorption of the hematoma was reported (**Figure 4**). The patient was completely asymptomatic at the 30-day clinical visit.

CONCLUSIONS

CS injury is a possible complication of retrograde cardioplegia delivery. The best treatment for such rare and potentially lethal injuries is prevention. If the lesion consists of a nonobstructive endocardiac wall hemorrhage, prompt protamine infusion should be administered, followed by a strict multimodality imaging "wait-and-see" strategy.

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KEY WORDS cardiac surgery, cardio computed tomography, coronary sinus hematoma, retrograde cardioplegia

APPENDIX For supplemental videos, please see the online version of this paper.