



## Case Report

## Cardiogenic cerebral embolism due to sinus arrest associated with coronary intervention for the right coronary artery: A case report

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## ABSTRACT

Percutaneous coronary artery intervention (PCI) carries the risk of occlusion of the sinus node branch (SNB) which can lead to sinus arrest (SA). Generally, PCI-related SA recovers spontaneously, with a favorable clinical course. Herein, we describe a case of SNB occlusion after PCI for the right coronary artery which resulted in SA, subsequent left atrial appendage thrombus, and cardiogenic cerebral embolism (CE). Ultimately, the patient died due to cardiogenic CE. We report on the mechanism of intracardiac thrombus formation and discuss CE prevention strategies after PCI. Based on our experience, the possibility of adverse events due to PCI-induced SA must be considered, although PCI-induced SA is generally expected to resolve.

**<Learning objective:** Percutaneous coronary artery intervention (PCI) carries the risk of occlusion of the side branches, including the sinus node branch (SNB). The occlusion of the SNB can lead to sinus arrest (SA). Generally, PCI-related SA recovers spontaneously. However, it should be noted that persistent SA can result in thrombus formation and cerebral embolism.>

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## Introduction

In 50% of individuals, the sinus node branch (SNB) originates from the right coronary artery (RCA) [1]. The culprit lesion of acute coronary syndrome of the RCA is often rich in thrombi, which can lead to occlusion of the SNB during percutaneous coronary artery intervention (PCI). Occlusion of the SNB can cause sinus arrest (SA). The resulting SA generally recovers spontaneously, with a favorable clinical course. Herein, we report a patient in whom a fatal cardiogenic cerebral embolism (CE) developed after SA and left atrial appendage (LAA) thrombus formation after PCI for the RCA. We report on the mechanism of intracardiac thrombus formation associated with SNB occlusion and discuss prevention strategies.

## Case report

The patient's family provided consent for publication of this case report.

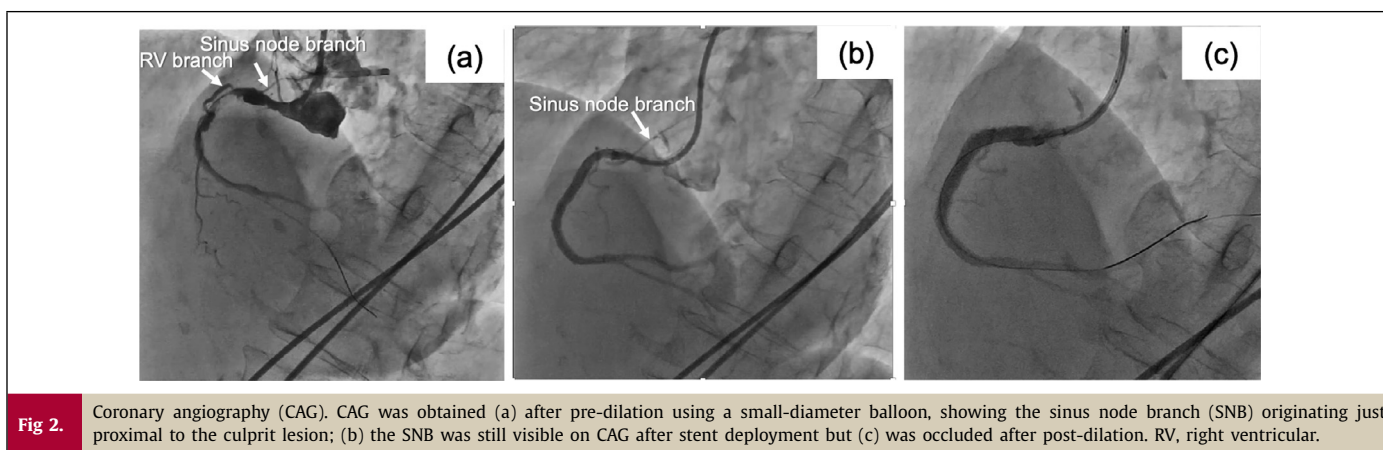
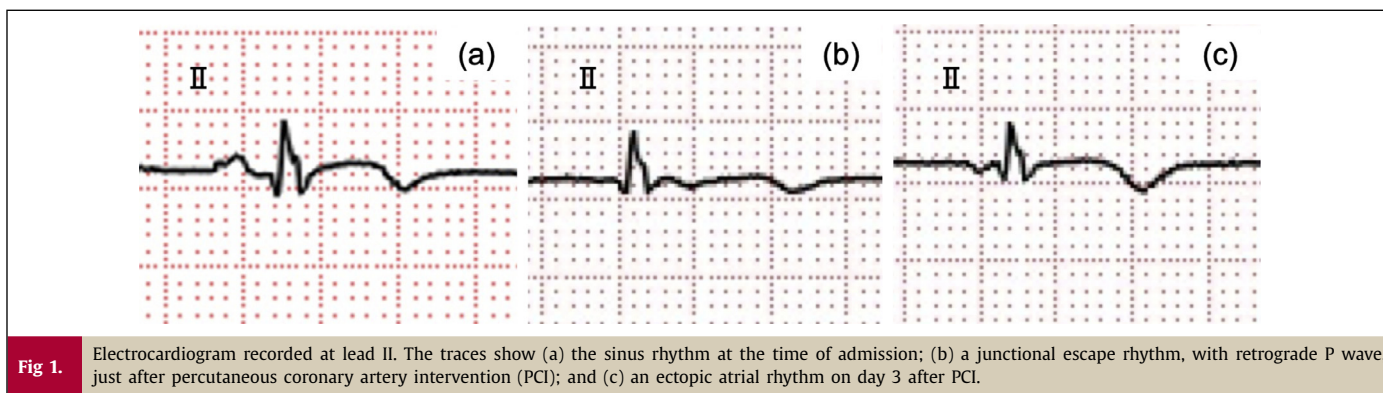
A 77-year-old female visited her doctor due to loss of appetite and general fatigue. She was admitted to the hospital because of

severe anemia (hemoglobin, 5.3 g/dl). On admission, electrocardiography (ECG) and transthoracic echocardiography (TTE) revealed findings of inferior myocardial infarction. She was also diagnosed with advanced rectal cancer by colonoscopy and transferred to our hospital for cancer surgery.

Her past medical history was unremarkable, including absence of paroxysmal atrial fibrillation (AF). On admission to our hospital, her vital signs were normal, with a blood pressure of 126/64 mmHg, heart rate of 78 beats per minute (bpm), and body temperature of 36.7 °C. On physical examination, a tumor was palpable in the lower abdomen. Laboratory results were as follows: hemoglobin level of 9.3 g/dl; hematocrit of 21%; creatine kinase level of 35 U/l; creatine kinase MB level of ≤ 5 U/l; cardiac troponin-T level of 0.055 ng/ml; N-terminal pro natriuretic peptide level of 6793 pg/ml; prothrombin time (PT) of 10.8 seconds; PT international normalized ratio of 0.87; active partial thromboplastin time of 37.3 seconds; fibrinogen of 424 mg/dl; and D-dimer of 2.3 μg/ml. Other general laboratory results were within normal limits. On chest radiography, her cardiothoracic ratio was 51%, without evidence of congestion or pleural effusion. ECG showed a sinus rhythm (heart rate, 70 bpm) and the II, III, and aVF leads revealed abnormal Q waves and negative T waves (Fig. 1a). On TTE, the left ventricular (LV) ejection fraction was 37%, with LV asyn-

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ergy in the inferior region. No significant valvular heart disease was observed. Coronary angiography revealed total occlusion of the proximal RCA. Dobutamine stress echocardiography, performed to assess regions of ischemia, confirmed induced ischemia in the inferior LV. Based on these findings, PCI was planned for the RCA before surgery for rectal cancer.

After passing a guide wire, with microcatheter support, pre-dilation was performed using a small-diameter balloon (Fig. 2a). Angiography and intravascular ultrasound (IVUS) revealed abundant organic thrombi at the culprit lesion, with the SNB originating just proximal to the culprit lesion. Two everolimus-eluting stents, with a distal protection device to prevent distal embolism (Fig. 2b), were deployed. After confirming insufficient stent expansion by IVUS, we proceeded with post-dilation. Immediately post-dilation, the patient developed bradycardia, with a heart rate of 31 bpm. The ECG revealed SA, with a junctional escape rhythm (Fig. 1b). Angiography confirmed occlusion of the SNB (Fig. 2c). At that point, we attempted to reinsert the guidewire into the SNB but the procedure failed. Therefore, we inserted a temporary pacemaker and proceeded to complete the procedure.

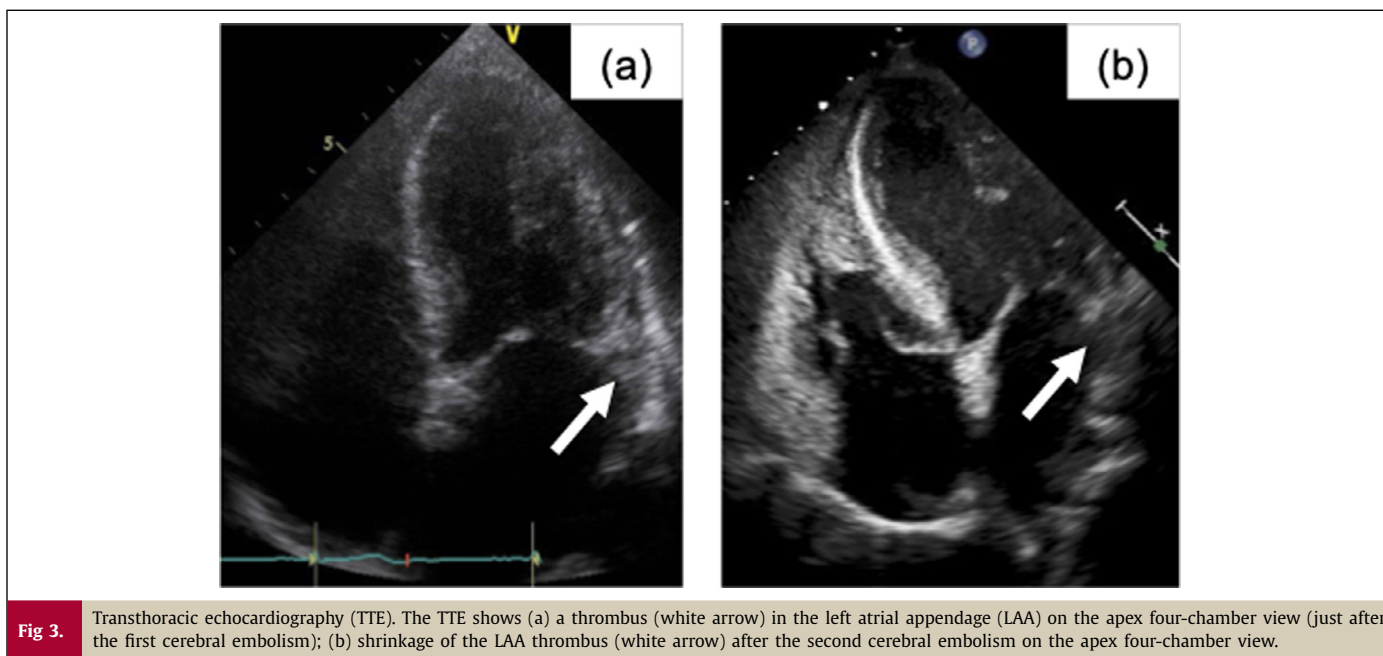
ECG findings on day 1 after PCI revealed a junctional escape rhythm with a retrograde P wave and heart rate of 37 bpm. Blood examination showed D-dimer of 2.5  $\mu\text{g/ml}$ . On day 3 after PCI, there was evidence of an ectopic atrial rhythm, with a heart rate of 50 bpm (Fig. 1c). On day 4 after PCI, the patient developed impaired consciousness, left conjugate deviation, and right paralysis. Brain magnetic resonance imaging revealed a cardiogenic CE causing occlusion of the left middle cerebral artery. Angiography and aspiration thrombectomy were performed immediately. TTE revealed an LAA thrombus (Fig. 3a) and anticoagulant therapy, using heparin, was immediately initiated. The patient experienced re-

peat CE and died of cerebral herniation eight days after the first CE.

## Discussion

The sinoatrial node is perfused by the SNB, which is reported to originate from the RCA (50%), circumflex branch (44%), or other branches (6%) [1]. Obstruction of the SNB during PCI is not an occasional occurrence. In a previous case series of 80 patients undergoing PCI for proximal RCA lesions, SNB occlusion occurred in 14 (17.5%) patients, four of which developed SA. Of the four patients who sustained SA, a normal sinus rhythm was recovered within three days after SNB occlusion [2]. Another report revealed that in most cases, late spontaneous reperfusion is generally achieved in most occluded side branches [3]. We do note a case report of atrial standstill causing CE in a pediatric case [4]. To our knowledge, this is the first report describing fatal LAA thrombus and CE developing as a complication of SA after PCI.

SA is associated with the disappearance of left atrium contraction. However, it is unclear how junctional escape rhythm, which leads to retrograde P waves, affects the hemodynamics of the left atrium, including the LAA. It should be noted that we did not perform transesophageal echocardiography after the first CE; however, TTE revealed a LAA thrombus which was not evident on preoperative contrast-enhanced computed tomography. As our patient had no prior history of AF, we considered that the LAA thrombus was caused by the SA, and restoration of atrial contraction with ectopic atrial rhythm caused scattering of thrombus. On TTE performed after the second CE, the LAA thrombus appeared to shrink, suggesting that it may have been scattered (Fig. 3b). Based on our experi-



rience with this case, we advocate for the necessity of preventing CE after PCI-induced SNB occlusion.

The most common method to prevent SNB obstruction is to use guidewire protection. However, guidewire protection is indicated when the perfusion area of the side branch is large. In our case, the perfusion area was small and, therefore, we did not insert the guidewire into the SNB. Based on our experience, however, we propose that guidewire protection for the SNB should be considered irrespective of the extent of the perfusion area for patients with thrombi or plaque-rich lesions assessed by angiography or intracoronary imaging.

Anticoagulant therapy may be the most feasible method to prevent thrombus formation. However, the addition of anticoagulants to dual antiplatelet therapy increases the risk of bleeding. Therefore, if SA persists for more than 24–48 h, using anticoagulants as initiation therapy should be considered. In this case, past medical history and family history did not suggest the presence of congenital prothrombotic disorders. Cancer-bearing status and depressed cardiac function due to myocardial infarction were considered as the factors of promoting thrombus formation. Especially in cancer-bearing patients, hypercoagulability and thrombosis are well known as Trousseau's syndrome [5]. In this case, blood examination on admission showed a slight increase in D-dimer and fibrinogen level, suggesting the existence of hypercoagulability. If there exist pathological conditions which are thought as prothrombotic status, more caution to prevent thrombosis should be considered. It may also be useful to evaluate the atrial wave of the transmitral flow, which represents atrial contraction, noting that this does not provide a direct measure of LAA blood flow. However, we did not evaluate transmitral flow in this case.

In conclusion, our case highlights the possibility of SA with a junctional escape rhythm due to obstruction of the SNB during PCI for the RCA. In our case, SA resulted in LAA thrombus formation and CE. Based on our experience, the possibility of adverse events

due to PCI-induced SA must be recognized and prevented, although PCI-induced SA is generally expected to resolve.

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#### Declaration of Competing Interest

None

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