Long Standing Sinus Arrest Following Percutaneous Coronary Intervention of Proximal Right Coronary Artery

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Sinus node dysfunction following percutaneous coronary intervention (PCI) of the right coronary artery (RCA) due to sinus node artery (SNA) occlusion has been previously reported [1]. Restoration of sinus rhythm was achieved after a few days in most cases [2], and reports of long standing sinus arrest are rare [3,4]. We report a case of symptomatic long standing sinus arrest following PCI of proximal RCA.

PATIENT DESCRIPTION

A 62-year-old woman with hypertension, diabetes mellitus, dyslipidemia, and a history of smoking presented to the emergency department (ED) after one week of chest pain. The initial workup found significant elevation of troponin T levels, with Q waves in the inferior leads and ST segment depressions in leads I–AVL. The patient underwent coronary angiography and the following was revealed: 95% narrowing of the proximal RCA [Figure 1A], 85% narrowing of the proximal left anterior descending (LAD) artery, and 80% narrowing of the first marginal coronary arteries.

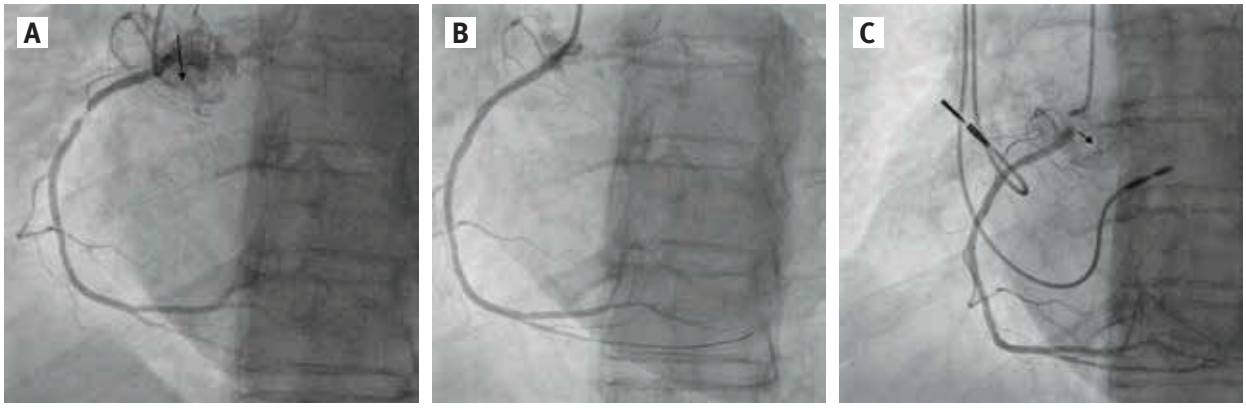
A drug-eluting stent was successfully placed in the proximal RCA. Immediately after stent deployment, junctional escape rhythm with ventricular rate of 40 beats per minute was noticed followed by symptomatic hypotension. Right coronary angiography after stenting showed SNA occlusion [Figure 1B]. Atropine administration had no effect in recovering sinus node function. Temporary ventricular pacing was initiated and the patient hemodynamically improved. Sinus rhythm was not restored after one week, and as the patient experienced symptomatic hypotension without pacing, a permanent dual chamber pacemaker was implanted.

The patient underwent elective stenting of the LAD coronary artery one month later. Right coronary angiography showed the patent stented proximal RCA and spontaneous recanalization of SNA [Figure 1C], but the sinus rhythm was not present, and the patient's heart was paced at a rate of 70 beats per minute to avoid symptomatic hypotension. Sinus rhythm was 76 beats per minute 6 months after RCA stenting and the patient was asymptomatic.

COMMENT

Side-branch occlusion of SNA is not rare. It has been reported in 14 of 80 patients

Figure 1. Right coronary angiography (RCA)
[A] Showing 95% narrowing of proximal RCA. The arrow shows the sinus node artery arising from the proximal segment of the RCA, before the intervention; **[B]** Right coronary angiography after stenting of the proximal RCA showing the occlusion of the sinus node artery immediately after the proximal RCA stenting; **[C]** Right coronary angiography a month after RCA stenting showing (arrow) spontaneous recanalization of the sinus node artery



who underwent stenting of proximal RCA (17.5%) and sinus dysfunction developed in 4 of these 14 patients (28.6%) [2]. Restoration recovery generally is seen within a few days [1,2], but in a few cases, it is prolonged [3,4]. However the appropriate management of these patients is not well established. In patients with sinus node disease, the ACC/AHA/HRS guidelines [5] recommend permanent pacing only when symptoms are clearly attributed to bradycardia. Pacing is not indicated in asymptomatic patients or when bradycardia is due to reversible causes.

Although SNA dysfunction following PCI is reversible, we do not know when sinus function will be restored and how long to wait before implanting a permanent pacemaker. It has been reported that in most cases of sinus dysfunction after PCI, sinus rhythm restoration was seen within 3 days after SNA occlusion. In our patient, sinus rhythm was not present until one month later. In the report by Abe et al. [3], this was seen 2 weeks after SNA occlusion.

CONCLUSIONS

We presented a case were SND persisted after SNA recanalization and surprisingly resolved at some point between 1 month to 6 months after SNA occlusion.

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Capsule

Pitching cGAMP as a vaccine strategy

One strategy to address the variable effectiveness of many influenza vaccines is to induce antiviral resident memory T cells, which can mediate cross-protection against multiple substrains (heterosubtypic immunity). Unfortunately, such vaccines typically use attenuated active viruses, which may be unsafe for certain populations. **Wang** et al. reported a vaccine using an inactivated virus that effectively induced heterosubtypic immunity in both mice and. They co-administered the virus with 2',3'-cyclic guanosine monophosphate-adenosine monophosphate (cGAMP), a potent activator of the innate immune system, encapsulated in pulmonary surfactant-biomimetic liposomes. This adjuvant was taken up by alveolar epithelial cells, whose activation resulted in effective antiviral T cell and humoral immune responses without accompanying immunopathology.

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Capsule

Tobacco smoking and somatic mutations in human bronchial epithelium

Tobacco smoking causes lung cancer, a process that is driven by more than 60 carcinogens in cigarette smoke that directly damage and mutate DNA. The profound effects of tobacco on the genome of lung cancer cells are well-documented, but equivalent data for normal bronchial cells are lacking. **Yoshida** et al. sequenced whole genomes of 632 colonies derived from single bronchial epithelial cells across 16 subjects. Tobacco smoking was the major influence on mutational burden, typically adding from 1,000 to 10,000 mutations per cell; massively increasing the variance both within and between subjects; and generating several distinct mutational signatures of substitutions and of insertions and deletions. A population of cells in individuals with a history of smoking had mutational burdens that were equivalent to those expected for people who had never smoked: these cells had less damage from tobacco-specific mutational processes, were fourfold more frequent in ex-smokers than current smokers and had considerably longer telomeres than their more-mutated counterparts. Driver mutations increased in frequency with age, affecting 4–14% of cells in middle-aged subjects who had never smoked. In current smokers, at least 25% of cells carried driver mutations and 0–6% of cells had two or even three drivers. Thus, tobacco smoking increases mutational burden, cell-to-cell heterogeneity and driver mutations, but quitting promotes replenishment of the bronchial epithelium from mitotically quiescent cells that have avoided tobacco mutagenesis.

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