A benign course of sinus node artery occlusion after stenting at proximal right coronary artery

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To the Editor: The sinus node artery (SNA) can be occluded during the percutaneous coronary intervention (PCI), which produces bradycardia. Previous studies showed a high incidence of SNA occlusion (SNO). Improvements in stent design and anti-thrombotic strategies have decreased branch occlusion. Here, we investigated the results of this procedure in our center. The incidence of SNO was 7.0%. The percentage of diameter stenosis of the SNA, SNA originating at a diseased right coronary artery (RCA), and larger RCA/SNA diameter ratio were independent predictors of SNO. Bradycardia occurred in SNO, but only one temporary pacemaker was implanted. SNO and the consequent bradycardia were not rare during PCI for severe RCA in current practice, yet they led to a benign clinical course with few temporary pacemakers. Permanent pacemaker implantation seems to be unnecessary.

The SNA, usually arising from the RCA, can be occluded during PCI and produce various arrhythmias, including sinus arrest, junctional escape rhythm, sinoatrial blocks, and supraventricular tachycardia.[1,2] Side branch (SB) occlusion was seen in approximately 20% of patients in early studies, which demonstrated a relatively benign clinical course of SNO.[1] Advancements in stent technology and anti-thrombotic strategies seek to improve clinical outcomes.[3] However, the incidence, risk factors, and clinical results of SNO have not been systematically analyzed in Chinese people. Here, we report the results in our center.

The study was approved by the institutional ethics committee, and informed consent was obtained. PCI and perioperative management were performed according to the guidelines. Between January 2015 and December 2019, 460 consecutive patients presenting an SNA origin from the RCA and >90% diameter stenosis of the proximal RCA were enrolled in this study. The occlusion group included patients with final thrombolysis in myocardial infarction (TIMI) grades 0–1 of SNA, while the others (TIMI grades 2–3) were allocated into the control group.

Clinical characteristics and coronary procedures were recorded. Coronary angiograms were reviewed by two interventional cardiologists. The catheter tip was calibrated as a reference. The luminal diameters and percentage of stenosis were measured using quantitative coronary angiography analysis with automated edge-detection algorithms (CASS-5.2, Pie Medical, Maastricht, the Netherlands). The coronary artery flow was evaluated using the TIMI score, and a TIMI grade 3 SNA was recorded in all patients pre-operatively. The bifurcation angle was defined as the angle between the distal RCA and the SNA at its origin using the angiographic projection with the widest separation of the two branches.

Data were presented as mean ± standard deviation or frequency (percentage). Continuous variables were analyzed using Student's t test, while categorical data were analyzed using the χ^2 test. A multivariable logistic regression model was performed, adjusting for the covariates that were statistically significant at univariable analysis (variable entered multivariate analysis if $P < 0.20$), to identify independent predictors of SNO. A P value < 0.05 was considered to be statistically significant. SPSS (IBM Corp, New York, NY, USA; SPSS Statistics, 22 version) was used.

The incidence of SNO was 7.0% (32/460) during PCI for the proximal RCA. The occlusion group had a higher proportion of smokers (100.0% [32/32] *vs.* 47.2% [202/428] $P < 0.001$) and lower left ventricular ejection fraction (57.9% ± 4.1% *vs.* 59.8% ± 8.9%, $P = 0.04$). Other clinical characteristics showed no significant differences between groups.

Patients in the occlusion group presented more total occlusion, more often severe stenosis of the SNA, a larger

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RCA/SNA diameter ratio, a larger bifurcation angle, more SNA originating at a diseased RCA, and more stents covering the SNA ostium Supplementary Table 1, http://links.lww.com/CM9/A754. Otherwise, the stenting procedure showed no significant differences between groups.

Cigarette smoking, left ventricular ejection fraction, total RCA occlusion, acute occlusion, SNA opening disease, percent diameter stenosis of the SNA, the RCA/SNA diameter ratio, bifurcation angle, SNA originating at a diseased RCA, and stent covering the SNA ostium were included in the logistic analysis. Initial percent diameter stenosis of the SNA, SNA originating at a diseased RCA, and a larger RCA/SNA diameter ratio were independent predictors of SNO in the multivariate analysis. However, none of the risk factors related to coronary artery disease or other angiographic characteristics reached statistical significance.

When the SNA was occluded, more patients suffered new-onset bradycardia (4 [12.5%] *vs.* 0, $P < 0.001$). Of them, three patients recovered with atropine treatment. Sinus bradycardia of 45 beats/min and hypotension of 85/50 mmHg occurred in the other patient. With treatment with atropine, tirofiban, and guidewire and balloon intervention, the blood flow of the SNA was partially recovered, with a heart rate of 52 beats/min and blood pressure of 100/60 mmHg. Tirofiban was continuously intravenously administered for 48 h, but the patient suffered transient cardiac arrest for over 5 s without discomfort. A temporary pacemaker was implanted, and a wandering pacemaker occurred 3 days after the index procedure. The temporary pacemaker was removed upon recovery of normal sinus node rhythm 9 days later. Repeated coronary angiography showed TIMI grade 3 flow in the SNA. Peri-operative cardiac biomarkers and heart rate showed no differences between groups.

The major findings of this study are as follows: (1) The incidence of SNO was 7.0% during PCI for severe RCA. (2) Initial diameter stenosis of the SNA, SNA originating at a diseased RCA, and larger RCA/SNA diameter ratio were independent predictors of SNO. (3) SNO resulted in transient bradycardia and a benign clinical course in the hospital.

The incidence and clinical course of SNO have been reported in a small study, in which approximately 20% of patients treated with stents presented SNO.[1] The technological evolution of scaffolds with thinner struts and a decreased number of hoop connectors theoretically improved SB access given the decreased overall scaffold profile.[3] In fact, recent registries showed an occlusion rate of SB of approximately 7% to 8%,[4,5] which was consistent with our results. Another probable reason for the reduced SB occlusion rate is the advancement of antithrombotic drugs.[6] As reported by Seo *et al*,[6] less clopidogrel use was an independent predictor for SB failure. Pre-operative loading of clopidogrel or ticagrelor in daily practice may reduce microthrombosis formation at the SNA ostium. In addition, the use of glycoprotein IIb/IIIa inhibitors could reduce the SB occlusion rate. Glycoprotein IIb/IIIa inhibitors can improve and restore thrombotic impaired flow, as in our representative patient. In addition, SNA usually originates perpendicularly from RCA-like septal branches. In clinical practice, septal branches are rarely occluded during PCI procedures, which may be attributed to the small, if any, carina and plaque shift.

We found that severe SNA ostium diameter stenosis, SNA originating at a diseased RCA, and the RCA/SNA diameter ratio were independent predictors of SNO. Diameter stenosis of the SNA was predictive for SNO, concordant with previous studies.[2,4] Severe stenosis of the SB, reflecting a heavy plaque burden at the SB ostium, has been a predictor for SB compromise after single-stent implantation.[7] An SNA origin at a diseased RCA means there is a true bifurcation lesion, in which case PCI can predispose to plaque shift and carina shift as causal mechanisms of SB occlusion.[6] Displaced stent struts and the "snow plow" effect would deteriorate the SB stenosis, especially for true bifurcation lesions. In addition, we found that the risk of SNO was associated with a larger RCA/SNA diameter ratio, which is similar to earlier results.[7] A larger RCA/SNA diameter ratio indicates a relatively larger plaque burden in the SNA; thus, RCA stenting may predispose the SNA ostium to deterioration and even occlusion.

In this study, SNO caused transient bradycardia without pacemaker implantation. Kotoku *et al*[1] reported that sinus arrest with junctional escape rhythm developed in 28.6% (4/14) of patients with SNO. The junctional escape rhythm disappeared within 3 days, and only one temporary ventricular pacemaker was implanted. In a prospective study concerning the electrophysiological effects of atrial artery occlusion, more frequent intra-atrial conduction delay and atrial arrhythmias occurred in atrial artery occlusion, but no pacemaker was needed.[2] However, the frequency of atrial arrhythmias at the 1-year follow-up was not associated with procedural-related atrial branch occlusion.[4] Pacemakers were implanted in a few patients for severe bradycardia caused by SNO.[1] In these patients, sinus rhythm usually recovered within a couple of weeks, and the reported restoration duration was up to 6 months. Likewise, sinus rhythm recovered within 9 days in our patients. Thus, we suggest delaying permanent pacemaker implantation in patients with SNO caused by PCI. The low incidence of sinus arrest in SNO patients was attributed to the sinus node's hyposensitivity to ischemia and the abundant atrial arterial anastomosis. In addition, the frequent patency of spontaneous reperfusion was due to the negative remodeling of plaque at the SB ostium, the resolution of SB spasms, or local lytic processes of microthrombosis, which also limited the long-term effect of SB occlusion.

Several limitations need to be stated about this retrospective non-randomized study. First, the retrospective design of the study might have given it a selection bias. Second, the lack of follow-up angiography could not exclude transient coronary spasm or confirm spontaneous reperfusion. However, a second angiogram is not recommended in clinical practice, as there are no clinical guidelines for SB occlusion. Third, the use of various stent types prevented

us from exploring any possible association between a particular stent model and the occurrence of SNO. Finally, the follow-up was too short, although in-hospital outcomes were benign.

SNO and the resultant bradycardia were not rare during PCI for severe RCA in our current practice, yet they did lead to a benign clinical course with few temporary pacemakers. Permanent pacemaker implantation seems to be unnecessary.

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Conflicts of interest

None.

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