

CASE REPORT

Acute thrombosis of the sinus node artery: arrhythmological implications

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A 53 year old woman was referred for percutaneous coronary intervention because of a recent inferior myocardial infarction. During right coronary artery stent implantation, intermittent occlusion of the coronary side branch for the sinus node occurred, associated with intermittent sinus arrest and junctional escape rhythm. This led to speculation about the potential mechanisms for sinus node dysfunction. Degenerative fibrosis of nodal tissue is actually considered the most common cause of bradyarrhythmias. Yet, in everyday practice, no particular attention is usually paid to other potential pathogenic mechanisms such as coronary artery disease. This may be particularly true for elderly patients or patients with multiple risk factors. Thus, sinus node dysfunction may be an unrecognised marker of coronary artery disease.

Ischaemia of the sinus node (SN) is generally considered to be an uncommon pathogenic mechanism of SN dysfunction causing rhythm disturbances. Indeed, so far, direct evidence of an ischaemic insult to the SN has seldom been reported. We had the opportunity to document angiographically this phenomenon in a patient undergoing right coronary artery (RCA) stent implantation.

CASE REPORT

A 53 year old woman with hypertension and hypercholesterolaemia was referred to our institution because of a recent inferior myocardial infarction. Predischarge exercise testing had shown significant ST segment depression associated with

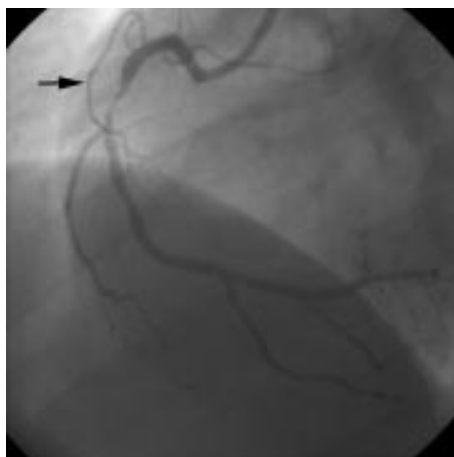


Figure 1 Basal right coronary angiogram showing a critical stenosis in the middle tract of the vessel. The arrow indicates the sinus node artery originating from the proximal tract of right coronary artery.



Figure 2 Right coronary angiogram after stent deployment. Total occlusion of the sinus node artery is evident. The arrow indicates haziness in the proximal tract of the right coronary artery.

angina at a low work load. Coronary angiography showed a critical stenosis in the middle tract of the RCA and a remarkable SN artery originating from the proximal tract of the vessel (fig 1).

The percutaneous procedure was performed according to standard technique. Immediately after direct stent implantation, a diffuse haziness appeared in the proximal tract of the RCA, close to the proximal edge of the stent, followed by total occlusion of the atrial side branch directed to the SN (fig 2). ECG showed junctional escape rhythm with P wave retroconduction at a heart rate of 50 beats/min, suggesting SN ischaemic arrest. Despite repeated boluses of intracoronary glyceryl trinitrate and intravenous atropine, sinus rhythm did not re-emerge. Intravenous abciximab at conventional doses was then administered. Repeated contrast injections showed slow progression of dye into the sinus branch. TIMI (thrombolysis in myocardial infarction) flow 2–3 in the SN artery was definitely achieved over 10 minutes. The patient remained completely asymptomatic. In the following 12 hours ECG monitoring showed sinus rhythm alternating with junctional escape rhythm.

The day after the procedure the patient was in stable sinus rhythm and a slight increase in cardiac markers (creatinine kinase MB 25 U/l and cardiac troponin I 2.14 ng/ml) was detected. Two days after the procedure she was discharged and at 7 and 30 days' follow up she was still asymptomatic and in stable sinus rhythm.

Abbreviations: CAD, coronary artery disease; RCA, right coronary artery; SN, sinus node; TIMI, thrombolysis in myocardial infarction

DISCUSSION

In the present clinical case the fortuitous intermittent occlusion of the coronary branch for the SN allowed us to speculate about the potential mechanisms of SN dysfunction.

The degenerative fibrosis of the SN and the atrioventricular node is actually considered to be the most common, if not the only, cause of sick sinus syndrome and atrioventricular block. Indeed, in most patients structural modifications account for an age related decrease of the intrinsic SN discharge rate.¹ The organisation of collagen in the SN gradually changes from coarse strands between clusters of nodal cells into a fine web of isolated fibres surrounding each nodal cell.² SN cells atrophy occurs and SN volume is reduced, with a qualitative, but not quantitative, change in the connective tissue.³

In everyday practice, no particular attention is usually paid to other potential pathogenic mechanisms such as coronary artery disease (CAD). Yet an ischaemic mechanism may well be the cause of SN dysfunction. Most older patients with symptomatic bradyarrhythmias have multiple risk factors for CAD. A recent study evaluated the incidence of coexistent CAD in Chinese people with symptomatic bradyarrhythmias and its relation to coronary risk factors.⁴ The incidence of angiographic CAD involving node related arteries was quite high (9%). Furthermore, in a postmortem angiographic study in patients with chronic SN dysfunction,⁵ CAD was relatively common, although no significant lesion obstructing blood flow to the SN was seen in the majority of patients. These studies suggest that among patients with sick sinus syndrome CAD is probably more frequent than believed and that SN ischaemia is an underestimated contributor to SN dysfunction.

SN dysfunction, in patients with inferior myocardial infarction and isolated proximal RCA disease, is related to a reduced flow to the SN artery.⁶ Clinical studies have shown the relation between ischaemia and SN dysfunction,⁷ and, accordingly, deliberate SN artery occlusion has been suggested as a potential treatment of chronic inappropriate sinus tachycardia.⁸

Our case provides further evidence for the ischaemic aetiology of SN dysfunction, emphasised by the intermittent occlusion of the SN branch associated with alternating sinus and escape rhythm. The cause of SN artery occlusion is uncertain in our patient. More than 50% of coronary side branches are at risk for iatrogenic occlusion during percutaneous coronary intervention, because they originate either from the lesion segment itself or from its immediate vicinity, so that they are subjected to temporary occlusion during balloon inflation. Periprocedural myocardial infarction occurred in 5–20% of patients in the pre-stent era. Coronary stenting can prevent abrupt vessel closure but side branch occlusion, especially with low reference diameter,⁹ and microembolisation are

still the most frequent factors responsible for myocardial damage after stent implantation.¹⁰ In our case, abciximab opened the SN artery and allowed for successful reperfusion and functional recovery of the SN. Thus, although an athero-embolic mechanism was probably involved, it is quite unlikely that atherosclerotic debris embolised into the SN artery, as the culprit lesion was fairly distal to the SN artery. Coronary spasm can be ruled out because high dose nitrates were ineffective in restoring flow in the SN artery. Plaque shift or direct scratching of the diseased endothelium during stent progression may have played a part in the pathogenesis of this event.

Conclusions

The present clinical case highlights the importance of considering an ischaemic aetiology of SN dysfunction when evaluating patients with symptomatic bradyarrhythmias. This may be particularly true for elderly patients or patients with multiple risk factors for CAD. The presence of SN dysfunction, indeed, may be an unrecognised marker of CAD.

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