

Sick sinus syndrome - a case report of paroxysmal supraventricular tachycardia due to atrioventricular node re-entry

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Abstract

Sick sinus syndrome encompasses a variety of EKG manifestations consisting of atrial bradyarrhythmias and tachyarrhythmias, alternating bradyarrhythmias and tachyarrhythmias as in tachycardia-bradycardia syndrome. Supraventricular tachyarrhythmias that can occur include atrial flutter, atrial fibrillation, atrial tachycardia and paroxysmal supraventricular tachycardia, although there is no direct causal relation between paroxysmal supraventricular tachycardia and sinus node disease. Atrioventricular node re-entry is a common cause of paroxysmal supraventricular tachycardia episodes. We present the case of a 70 year old female, hospitalized for atypical chest pain and dizziness when walking. The EKG on admission showed sinus bradyarrhythmia, anterior fascicular block, atrial and ventricular extrasystoles. During the hospitalization the patient presented an episode of palpitations, narrow complex tachycardia being registered on the EKG, with no response to the Valsalva maneuver or intravenous beta blocker. The tachyarrhythmia ceased spontaneously after one hour. 24 hour Holter EKG was performed and confirmed sinus node dysfunction. The electrophysiological study identified paroxysmal supraventricular tachycardia due to atrioventricular nodal re-entrant tachycardia, which was successfully treated by ablating the slow intranodal pathway. Therefore, in a case of sick sinus syndrome when the patient's symptoms cannot be attributed to the bradycardia, but to the tachyarrhythmic episodes, it is often most efficient to treat the patient's paroxysmal supraventricular tachycardia by radiofrequency ablation, rather than using cardiac pacing.

Keywords: *sick sinus syndrome, paroxysmal supraventricular tachycardia, atrioventricular nodal re-entrant tachycardia*

Introduction

Sick sinus syndrome is defined as an abnormality of the cardiac impulse formation

that can either be of an intrinsic or extrinsic cause, leading to the impairment of its pace making function. The condition is most common in the elderly and both sexes are affected approximately equally [1].

Sick sinus syndrome produces a variety of EKG manifestations consisting of atrial bradyarrhythmias, atrial tachyarrhythmias, and alternating bradyarrhythmias and tachyarrhythmias as in tachycardia-bradycardia syndrome [1]. Supraventricular tachyarrhythmias that can occur include atrial flutter, atrial fibrillation and atrial tachycardia,

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caused by the progression of atrial fibrosis in the right atrium, close to the sinus node area.

Another common form of supraventricular tachyarrhythmia is the paroxysmal supraventricular tachycardia (PSVT). PSVT comprises many types of arrhythmias, the most common ones being atrioventricular nodal re-entrant tachycardia (AVNRT) and atrioventricular re-entrant tachycardia (AVRT) [2]. AVNRT is the most frequent case of regular, paroxysmal supraventricular tachycardia and its mechanism is attributed to dual atrioventricular pathways physiology. Patients with AVNRT have two discrete functionally distinct pathways known as the slow pathway (slow conduction and short refractory period) and the fast pathway (fast conduction but prolonged refractory period) [3, 4]. Drug treatment or radiofrequency ablation (RFA) methods are used for the management of supraventricular tachycardia.

Case report

We hereby present the case of a 70 year-old female, who was admitted to our clinic for atypical chest pain and dizziness when walking.

Our patient was known with stage 3 essential hypertension with very high risk

(highest systolic BP registered 200 mmHg) from 2009, chronic heart failure NYHA class II and type 2 diabetes from 2011. The patient was undergoing treatment with Lercanidipine 10 mg, Isosorbide Mononitrate 40 mg twice a day, Candesartan 8 mg, Aspirin 75 mg, Atorvastatin 10 mg and Metformin 850 mg. The patient didn't drink alcohol and was a non-smoker with no history of stroke, upper gastrointestinal bleeding or syncope.

On admission, the patient presented with bradyarrhythmic heart sounds and premature heart beats, systolic murmur grade III /VI of maximal intensity in the third left intercostal space and aortic region, discrete swelling in the legs, BP = 160/70 mmHg, HR 56 bpm. The EKG showed sinus bradyarrhythmia, 45-60 bpm, QRS axis at -45 degrees, left anterior fascicular block, an atrial extrasystole with incomplete compensatory postextrasystolic pause, three isolated premature QRS complexes with a right bundle branch block morphology, with rsR' aspect in V1, but with normal QRS duration (0.08 sec), which could have been either junctional extrasystole with aberrant intraventricular conduction or ventricular extrasystole with the origin in the proximal portion of the interventricular septum, with compensatory pauses. In DII: PR = 0.20 sec, QRS=0.08 sec, QT = 0.40 sec (+ 2%) (Figure 1).

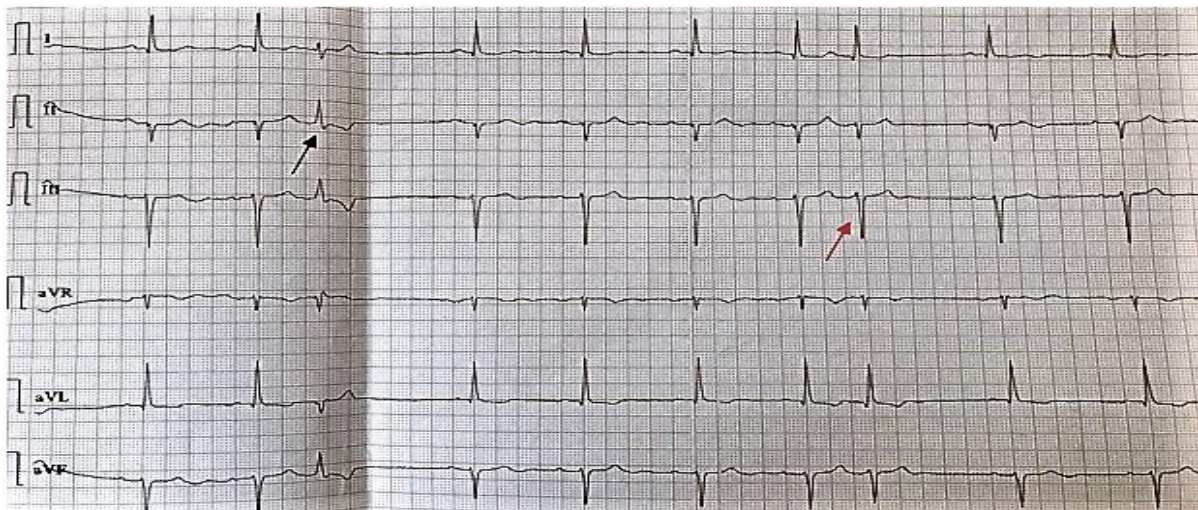


Fig. 1A. Limb leads - Admission EKG: Sinus bradyarrhythmia, 45-60 bpm, QRS axis at -45 degrees, left anterior fascicular block, a premature atrial contraction with incomplete compensatory postextrasystolic pause (red arrow) and one isolated premature ventricular contraction with RBBB morphology (black arrow), negative T waves in aVL

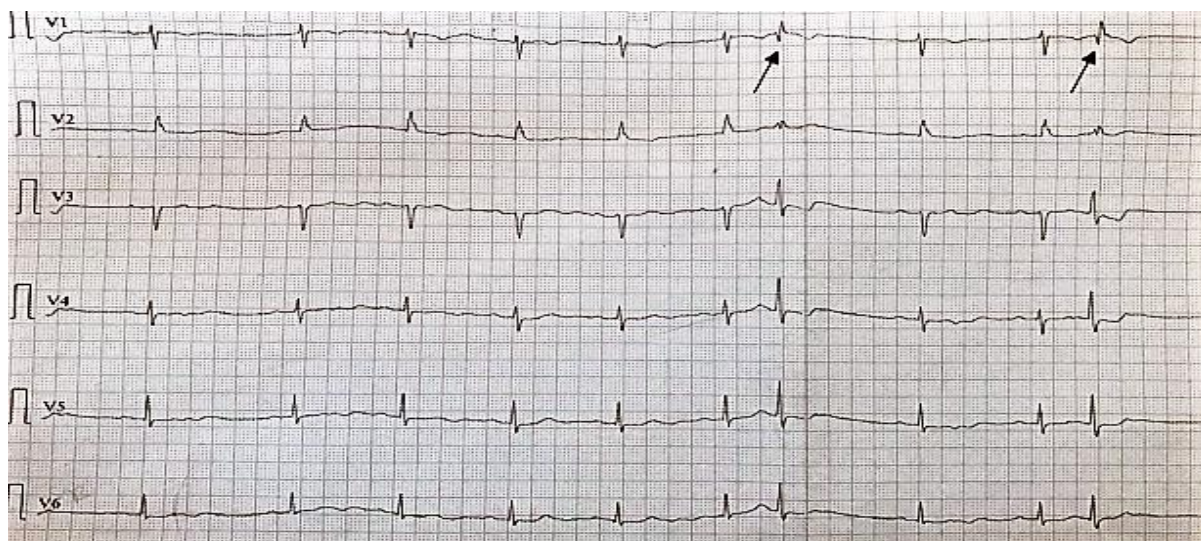


Fig. 1B. Chest leads: Admission EKG showing sinus bradycardia 45-60 bpm and two isolated premature ventricular contractions with RBBB morphology (black arrows) - rsR' aspect in V1

During hospitalization, the patient presented an episode of palpitations with fast and regular rhythm, registering PSVT on the EKG, with no response to intravenous beta-blocker or to the Valsalva manoeuvre (Figure 2).

Carotid sinus massage could not be performed due to the significant probability of

the existence of atherosclerotic plaques within the carotid arteries. Fortunately, the tachyarrhythmia ceased spontaneously after one hour, followed by sinus bradycardia 44-49/min with first degree atrioventricular block (PR interval = 0.24sec).

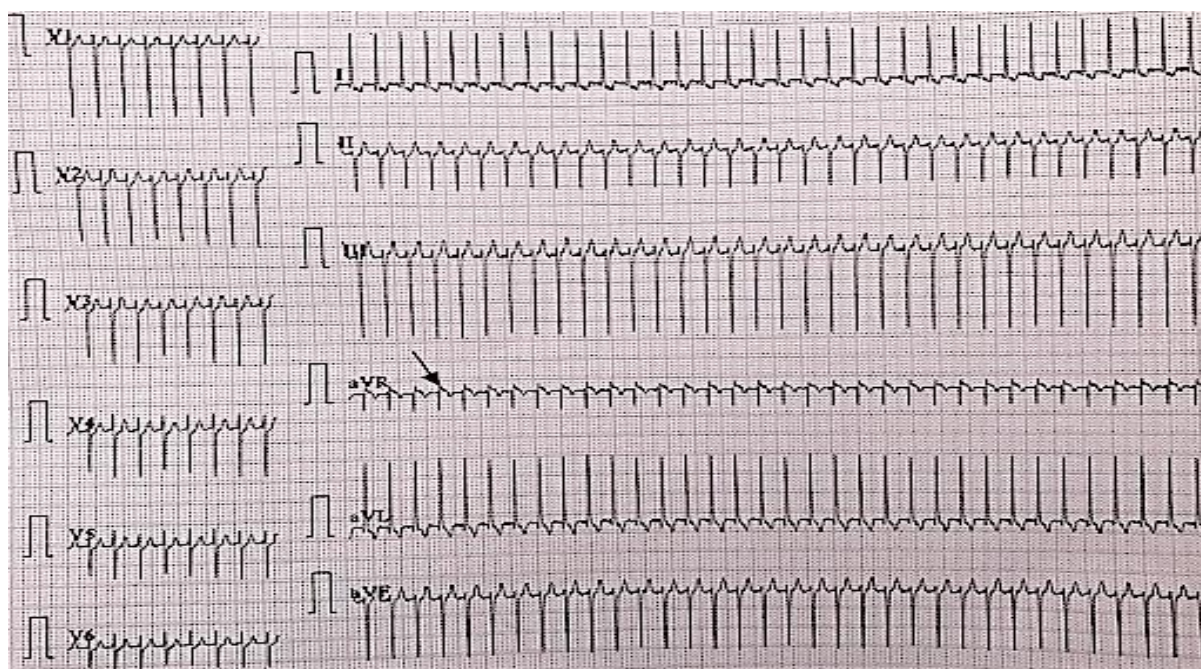


Fig. 2. Crisis EKG (10 mm/s): PSVT 120 bpm, QRS axis at -45 degrees, LAFB, pseudo r' aspect in lead aVR (arrow).

Echocardiography revealed mild concentric left ventricular hypertrophy, with normal global and segmental contractility (EF=50%), but with abnormal relaxation pattern; moderate left atrial dilation, mild dilation of the descending aorta, mild degenerative stenosis of the ascending aorta and mild aortic regurgitation with central regurgitation jet and modest calcification of the three aortic leaflets.

Holter EKG/24h confirmed the suspicion of sinus node dysfunction, identifying sinus rhythm (36-163 bpm) with an episode of PSVT (163 bpm) and 3932 premature polymorphic ventricular contractions without tendency to systemize.

The patient was subsequently addressed for invasive antiarrhythmic therapy. Electrophysiological study was performed starting with a local anaesthesia with 1% lidocaine, then the right femoral vein was punctured and the catheter used for the study was introduced and positioned on the side wall of the right atrium. Catheter ablation was positioned to record His potential (basal AH 88 ms, HV 42 ms).

After 1 mg atropine and atrial programmed stimulation, paroxysmal supraventricular tachycardia was triggered with a cycle length of 400 ms (150 / min). The mapping of the tricuspid annulus was realised, diagnosing slow-fast intranodal tachycardia because of

the sudden prolongation of the AH interval with 90 ms (from 120 ms to 210 ms) and the PR' interval of 60 ms at the septum level. Koch Triangle's elements were identified, then the slow intranodal path was located.

The radiofrequency current was used at the maximal range of 65W, 60 degrees (4 applications of 15 sec) with intact anterograde conduction during which slow-fast nodal rhythm was obtained, which limited the amount of radiofrequency current used. After this set of 4 applications, the stimulation protocol was resumed with no signs of inducibility of the paroxysmal supraventricular tachycardia and without nodal eco persistence beats.

After 15 minutes of waiting, atrial basal stimulation protocol was resumed and after 1 mg atropine the non-inducibility of tachycardia was demonstrated. Post ablation: AH 88 ms, HV 42 ms. Antegrade Wenckebach point at 460 ms (130 / min).

Therefore, the electrophysiological study showed that the patient had paroxysmal supraventricular tachycardia episodes due to atrioventricular nodal re-entrant tachycardia, which was successfully treated by ablating the slow intranodal pathway.

Post ablation the EKG objectified sinus bradycardia 45 / min, QRS axis at -45 degrees and a junctional extrasystole (Figure 3).

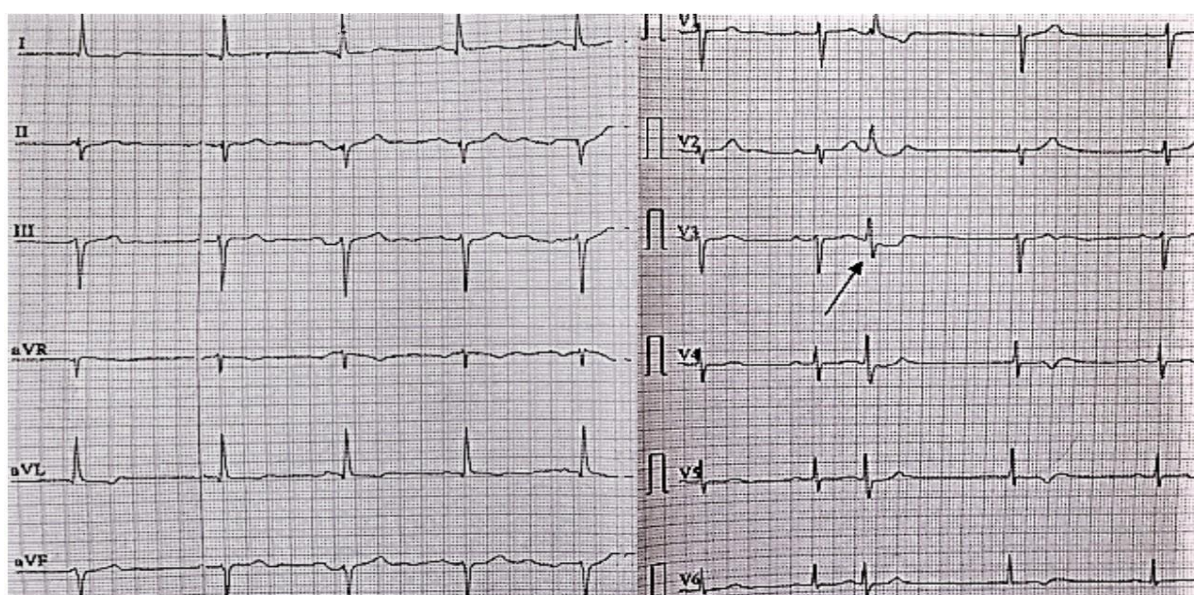


Fig. 3. Post ablation EKG: sinus bradycardia 45 bpm, QRS axis at -45 degrees, LAFB, a premature junctional contraction (arrow), negative T waves in aVL

Discussions

Patients with sick sinus syndrome are either asymptomatic or have symptoms that are nonspecific. Symptoms, which may have been present for months or years, can include syncope, palpitations and dizziness. The diagnosis requires documentation of sinus node dysfunction and correlation with the associated symptoms of sick sinus syndrome. The most frequent method of diagnosis is Holter monitoring [1].

Little is known about the incidence and the risk factors for the sick sinus syndrome. A new study published in 2016 [5] showed that essential hypertension is a strong risk factor for sick sinus syndrome. Our patient was known with stage 3 essential hypertension with very high risk.

PSVT is commonly diagnosed in patients with no structural abnormalities, between 12 and 45 years old [6]. Studies have proven that

PSVT due to AVNRT in older subjects is often related to an atrioventricular conduction abnormality, the longer AVN conduction intervals and refractory period explaining the late development of AVNRT [7]. This points out to a particularity of the presented case, taking into consideration the age of the patient (70 years old) and the length of the PR interval (200 ms), without receiving any drug associated with PR prolongation.

AVNRT is the most common form of PSVT whose diagnosis can be strongly suspected based on the surface electrocardiogram alone. Independent predictors of AVNRT diagnosis are the female sex, the age over 60 years and the classical EKG criteria: hidden P waves, pseudo-r' in V1, pseudo-s in DII, DIII, aVF. Recently, it has been shown that the presence of pseudo-r' in lead aVR is more accurate than the classical EKG findings [8], as shown in Figure 4.

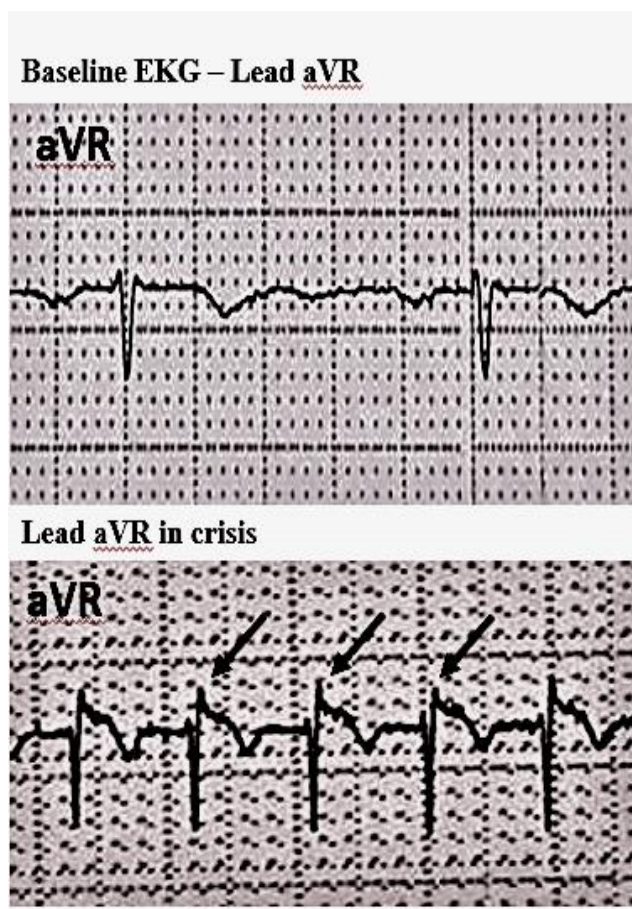


Fig. 4. During crisis, the presence of pseudo-r' wave in lead aVR is highly suggestive of AVNRT (black arrows)

Medical treatment of supraventricular tachycardia (SVT) often involves regular intake of drugs for several years and it might be challenging due to adverse effects and resistance to treatment. Due to this fact, more and more symptomatic patients with recurrent PSVT episodes are addressed for catheter ablation, with better results and toleration [9]. One study evaluating the effects of medical treatment on quality of life reported that quality of life worsened due to drug adverse effects and continuing tachycardia attacks in SVT patients, who were not treated by radiofrequency ablation but received long-term medical treatment [10]. On the contrary, catheter ablation decreases the general level of anxiety and improves the quality of life [2].

Patients with AVNRT and sinus node dysfunction exhibit characteristic electrophysiological alterations of both AV nodal pathways. Clinically, this results in significantly more frequent episodes of tachycardia. Slow pathway ablation appears to be safe and effective in these patients [11].

According to the 2013 ESC Guidelines recommendations on Cardiac Pacing and Cardiac Resynchronization Therapy, in sick sinus syndrome, pacing is indicated when symptoms can clearly be attributed to bradycardia (Class I, Level B) [12]. Thus, the clinical interest of this case is proven by the unusual source of synus node dysfunction. One would expect such symptoms to be attributed to bradycardia, implying the need for cardiac pacing. However, the tachyarrhythmic episodes were caused by an entirely different disease, therefore the preferred treatment option was an invasive slow pathway ablation.

Moreover, recent studies have proven that in patients with atrial fibrillation and sinus node dysfunction, some patients manifest sinus node dysfunction as a result of electric remodeling induced by periods of atrial fibrillation, while others develop progressive atrial structural remodeling that gives rise to both conditions together. Thus, the treatment strategy will vary according to the predominant disease phenotype. Ablation alone may improve symptoms associated with sinus node dysfunction in patients with atrial fibrillation, without the need of permanent cardiac pacing, but future randomized trials are needed to

clarify the epidemiology and optimal management of patients with sinus node dysfunction and atrial fibrillation [13, 14]. This could indicate towards new management strategies in AVNRT, in which slow pathway ablation has been a very effective treatment, with success rates of over 99% [15].

Conclusions

Patients with sinus node disease are generally old and frequently have a concomitant heart disease. In these situations, the demonstration of a clear cause-effect relationship between symptoms, which can be attributed to both the sinus node dysfunction and the PSVT, is often difficult to achieve. Moreover, cardiac pacing is not known to prolong survival in patients with sinus node dysfunction [12].

This case is particular because although PSVT is commonly diagnosed among young, healthy subjects, our patient is an elderly woman with important comorbidities - essential hypertension, a proven risk factor for sinus node dysfunction. Moreover, the surface ECG showed the presence of pseudo r' in lead aVR, a highly specific finding suggestive of AVNRT. Also, during the electrophysiological study, the patient exhibited prolongation of the anterograde Wenckebach cycle lengths (460 ms), with normal basal AH and HV intervals.

Therefore, in a case of sick sinus syndrome when the patient's symptoms cannot be attributed to the bradycardia, but to the tachyarrhythmic episodes, it is most efficient to treat the patient's PSVT by radiofrequency ablation, rather than using cardiac pacing or antiarrhythmic drugs.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of interest

The author(s) declare that they have no competing interests.

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