

New onset postural orthostatic tachycardia syndrome following ablation of AV node reentrant tachycardia

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Abstract

Background Autonomic dysfunction presenting as inappropriate sinus tachycardia has been reported to occur following slow pathway ablation for atrioventricular node tachycardia. We report on a series of patients who developed new onset postural orthostatic tachycardia syndrome (POTS) following successful radiofrequency ablation of atrioventricular nodal reentrant tachycardia (AVNRT).

Methods The study was a retrospective analysis that was approved by our Institutional Review Board. Patients were identified from those seen at our Syncope and Autonomic Disorders Clinic. A total of six patients were identified who were previously healthy except for supraventricular tachycardia. Each was found to have AVNRT during electrophysiology study and each underwent successful radiofrequency modification of the slow atrioventricular nodal pathway. Following ablation each patient developed the new onset of symptoms of orthostatic intolerance consistent with POTS.

Results After an initial symptom-free period (3–6 weeks) post ablation each patient began to experience symptoms of orthostatic intolerance. All six patients began to experience progressive severe fatigue. Orthostatic tachycardia was

reported by five patients, syncope by three patients, and presyncope by all six patients. Each patient reported the occurrence of symptom while upright that were relieved by becoming supine. Each patient had experienced symptoms for greater than 6 months prior to being seen at our center. Three patients reported such severe symptoms of orthostatic tachycardia that they underwent repeat electrophysiology study; however, none had evidence of AVNRT. Each patient demonstrated a POTS response within the first 10 min of upright tilt with reproduction of their clinical symptoms that had occurred post ablation.

Conclusion POTS may be a complication of radiofrequency ablation of AVNRT.

Keywords AV node reentrant tachycardia · Postural tachycardia syndrome · Radiofrequency ablation

1 Introduction

Radiofrequency catheter ablation of a slow conducting pathway has become a standard modality for the treatment of atrioventricular nodal reentrant tachycardia (AVNRT; [1–3]). There have been reports of inappropriate sinus tachycardia (IST) occurring as a complication of successful radiofrequency catheter ablation of both, AVNRT [4–6] and following ablation of accessory atrioventricular reciprocating tachycardia pathways as well [7]. More recently, IST has been reported to occur following cryoablation of AVNRT [8]. The mechanism by which this occurs is not well understood nor has the frequency of this complication been elaborated [8]. Here, we report on a series of patients who developed new onset postural orthostatic tachycardia syndrome (POTS) following successful radiofrequency ablation of AVNRT.

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2 Methods

The study was a retrospective analysis that was approved by our Institutional Review Board. Patients were identified from among those referred to our Syncope and Autonomic Disorders Clinic. A total of six patients were identified who were previously quite healthy except for supraventricular tachycardia. Each was found to have AVNRT during electrophysiology study and each underwent successful radiofrequency modification of the slow AV nodal pathway. Following ablation each patient developed new onset symptoms of orthostatic intolerance consistent with POTS. Following radiofrequency ablation of each of these patients, six patients developed signs and symptoms of POTS. Each of these patients had their radiofrequency ablation performed at an outside institution. Data was collected from patient interviews, physical examinations, and careful review of the records of the procedures and other aspects of their medical records. In addition to the aforementioned evaluations, each patient underwent a head-up tilt table test. While the tilt table testing protocol employed has been described elsewhere [9–11], briefly it consisted of a passive head upright tilt on a table with a foot board meant for weight bearing at an angle of 70° for a period of 30 min during which time heart rate and blood pressure were continuously monitored. All tests were performed after an overnight fast.

We defined POTS as a chronic state of orthostatic intolerance and palpitations of greater than 6 months duration, associated with a reproducible heart rate increase of at least 30 beats per minute (or a peak heart rate of 120 beats per minute or more) observed during the first 10 min of upright tilt table testing. The condition had to occur in the absence of other chronic debilitating conditions. Commonly reported symptoms in patients suffering from POTS include exercise intolerance, fatigue, palpitations, lightheadedness, cognitive impairment, syncope, and near syncope.

Each of the ablative procedures was performed outside our institution. Records showed that each patient underwent a standard electrophysiology study during which programmed electrical stimulation induced typical AV nodal reentrant tachycardia (average rate 200 beats per minute) that matched their clinical arrhythmias. Records also showed that an average of 6±4 radiofrequency applications were delivered to the slow pathway with a power level of 40±10 watts each for a duration of 60 s. No complications were reported after any of the procedures.

3 Results

Six patients, all women, aged (25±5), were identified for inclusion in the study. The clinical features of the group are summarized in Table 1.

Table 1 Baseline clinical characteristics of the study patients (N=6)

Age	25±5
Sex (Females)	6 (100%)
Symptoms prior to ablation	
Palpitations	6 (100%)
Symptoms of orthostatic intolerance	
Orthostatic palpitations	5 (84%)
Dizziness	5 (84%)
Inability to concentrate	5 (84%)
Syncope	3 (50%)
Presyncope	6(100%)
Fatigue	6 (100%)
Chest pain	1 (16%)
Comorbid conditions	
HTN	1 (16%)
Migraine	1 (16%)

3.1 Symptoms of orthostatic intolerance

Post ablation each patient reported a 3–6-week asymptomatic period. Following this each first patient began to experience symptoms of severe progressive fatigue. Shortly thereafter all six patients reported having sensation of presyncope. These patients reported having experienced full syncopal events and five patients reported severe orthostatic tachycardia. Each patient reported the onset of symptoms while upright which were relieved by lying supine. Holter monitors were performed on each patient that showed sinus tachycardia of rates averaging 120 beats per minute while upright which returned to normal levels (60–70 beats per minute) while supine. Each of the patients reported a progressive decline in functional ability for a minimum of 6 months prior to referral. Three patients underwent repeat electrophysiological studies; however, none of these found evidence of recurrent AVNRT. Echocardiography was normal in all six patients, as were supine resting electrocardiograms. All six patients were so debilitated by their symptoms that they either lost their employment or had to discontinue educational pursuits.

3.2 Responses to Head Up Tilt Test

The mean duration of symptoms prior to referral to our center was 14±6 months. After a complete review of prior records, patients underwent a complete history and physical examination followed by HUTT. Each patient demonstrated a marked tachycardia response both during standing and during HUTT. The mean heart rate increase (both with standing and HUTT) was 43±10 beats per minute. All six patients reported that the symptoms provoked during HUTT reproduced their clinical symptoms. No additional autonomic testing was performed.

3.3 Treatment

The treatment protocols employed were based on our previous experiences with orthostatic disorders and are described in detail elsewhere [9, 10, 12, 13]. Briefly, a sequence of therapies was employed that included education to avoid predisposing factors, an increase salt and fluid consumption, physical counter maneuvers, and aerobic and resistance training. If these proved ineffective, pharmacotherapy was initiated in a sequence generally consisting of beta-blockers, central sympatholytics, fludrocortisone, midodrine, selective serotonin reuptake inhibitors, either alone or in combination. If the aforementioned agents were ineffective or poorly tolerated, second line agents like pyridostigmine, octreotide, and erythropoietin were employed.

All six patients reported a significant reduction in symptoms and increase in functional status once a satisfactory treatment regimen has been elaborated. The specific symptoms, which improved the most in all six patients, were orthostatic palpitations and tachycardia, presyncope, and syncope. Three patients continued to complain of severe fatigue, which failed to respond to the aforementioned medications. These three patients later reported significant improvement in their fatigue following treatment with Modafinil [14].

4 Discussion

POTS is a form of orthostatic intolerance associated with a significant increase in heart rate while upright that resolves with recumbency. POTS is perhaps better thought of as a physiologic state (analogous to the state referred to as heart failure) which can be brought on by a number of different alterations in the body's normal homeostatic mechanisms. As such POTS appears to be a heterogeneous group of disorders with similar clinical characteristics [15–19]. One group of POTS patients appear to have an autoimmune basis, with onset of symptoms following an acute febrile illness, associated with antibodies to the post-synaptic acetylcholine receptors of the autonomic nerves [15]. Another group has high circulating levels of norepinephrine that occurs as a result of a genetic disturbance in the function of the norepinephrine reuptake proteins of adrenergic nerves. Yet another group appears to be in a chronic state of “idiopathic hypovolemia” [20, 21]. In a recently published study, it was reported that POTS might be a manifestation of autonomic cardiac neuropathy [22].

However despite these observations there are many POTS patients where the etiology of their condition remains unclear, resulting from pathophysiologies that have yet to be elucidated. A number of different stressors have

been observed to bring on symptoms of POTS. While many patients describe an antecedent febrile illness (presumed to be viral), there are also reports of pregnancy [23, 24], traumatic brain injury [25], electrocution [26], and lightning injury [27], all appearing to cause onset of POTS.

We report on a series of six patients who were quite healthy except for episodic supraventricular tachycardia, who developed debilitating POTS after radiofrequency ablation of the slow AV nodal pathway. Review of the ablation reports showed that they were uncomplicated procedures that eliminated their AVNRT. While it could be argued that the ablation and the onset of POTS were unrelated, the temporal relationship between these events in a group of previously asymptomatic patients would suggest otherwise. The mechanism by which radiofrequency ablation could result in POTS is unclear. Inappropriate sinus tachycardia has also been reported to occur after ablation [4–8]. Here it has been suggested that damage to the vagal fibers supplying the sinoatrial and AV nodal area occurs, resulting in a disruption of normal autonomic regulation. Similar mechanisms may be responsible for POTS post ablation. Another potential explanation might involve an inflammatory stress caused by ablation that leads to a hyperadrenergic state and/or partial autonomic neuropathy. It is also possible that these patients had preexisting POTS that was made worse by ablation; however, the complete absence of symptoms prior to the procedure would make this seem less likely.

The frequency at which POTS may occur after ablation is unclear, and this group of patients represents only a small percentage of those referred to our institution each year. However this could principally reflect a lack of recognition of the disorder. None of the six patients reported herein had been referred by the physician that had performed the radiofrequency ablation itself (four were self referred and two were referred by their primary care physicians). Indeed in each case the physician who performed the ablation dismissed the patients' complaints as being psychiatric in nature and unrelated to the procedure itself. It is hoped that this report will provoke closer observation of patients who develop similar symptoms after ablative procedures, thereby facilitating a better understanding of the frequency and pathogenesis of POTS post ablation while at the same time providing prompter diagnosis and management of this potentially debilitating condition.

5 Conclusion

POTS may occur as a complication of radiofrequency ablation of AVNRT. Recognition of the condition may allow for better understanding of its incidence and cause and facilitate both diagnosis and management.

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