Persistent Tachycardia
Diagnosis and Management

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AV=atrioventricular; PJRT=permanent form of junctional reciprocating tachycardia

CASE PRESENTATION

A 53-year-old woman came to the hospital in October 1991 with a 5-year history of tachycardia that had become more symptomatic during the preceding year. She was admitted to a hospital elsewhere for management. When given intravenously administered adenosine and metoprolol, the heart rate reportedly slowed. An echocardiogram disclosed mild to moderate reduction of global left ventricular systolic function with an estimated ejection fraction of 40%. She was discharged from the hospital on a regimen of orally administered quinidine gluconate and propranolol. However, she did not feel well on the regimen and continued to have frequent episodes of tachycardia. An electrophysiology study was performed elsewhere and, by report, showed “normal sinus function, normal atrioventricular function, and no evidence of an accessory pathway.” At that time, therapy with verapamil and digoxin was begun. The patient did not respond to that combination, and treatment with disopyramide was started. She continued having “sinus tachycardia” and was referred to our institution.

She had a persistent tachycardia at a rate of 120 to 130 beats per minute. Periodically, the heart rate accelerated to 200 beats per minute, and she described lightheadedness. On physical examination, her blood pressure was 108/76 mm Hg, and the resting heart rate was 135 beats per minute. No murmurs were heard. However, a soft “fourth heart sound” was detected. The lungs were clear. Results of the remainder of the physical examination were within normal limits.

An ECG showed supraventricular tachycardia at a rate of 130 beats per minute (Fig 1). An echocardiogram disclosed normal cardiac dimensions, but the left ventricle was hypokinetic. Results of the laboratory evaluation including CBC count, chemistry profile, electrolyte levels, and thyroid profile were within normal limits.

Disopyramide therapy was discontinued and propafenone treatment was begun. Although she subjectively felt better, tachycardia of 130 beats per minute persisted, and a more prominent diastolic gallop sound became audible. Nuclear ventriculography revealed an ejection fraction of 36%. A Holter monitor disclosed an average heart rate of 130 beats per minute with a rate varying from 90 to 190 over a 24-h monitoring period. Cardiac catheterization showed mild global hypokinesis of the left ventricle, and the coronary arteries were normal.

An electrophysiology study was repeated and disclosed an atrial tachycardia at a cycle length of 360 to 460 ms. Endocardial mapping identified a probable left atrial origin for the tachycardia. The tachycardia was not reset with atrial or ventricular premature beats, and there only was minimal suppression with rapid atrial pacing. Adenosine did not terminate the tachycardia, although the ventricular response was slowed transiently yielding an atrioventricular ratio of 2:1.

Questions for consultants:

1. What do you think is the primary underlying problem in this patient?

2. What is the relationship between reduced left ventricular function and the tachycardia? Which is the most likely primary abnormality?

3. How would you manage this patient? Please describe your preferences with regard to pharmacologic treatment as well as the various mechanical interventions.

![Figure 1. ECG demonstrating supraventricular tachycardia at 130 beats per minute.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21725/ on 06/26/2017)
With respect to question 1, the patient in this case has an incessant supraventricular tachycardia at a rate of about 130 beats per minute. A review of the 12-lead ECG shows a narrow complex tachycardia with a cycle length of 440 ms. The P wave can be seen preceding each QRS complex with a P-R interval of 160 ms. Thus, the P-R interval is longer than the P-R interval, and this may be characterized as a long R-P tachycardia. The P wave is upright in the inferior leads, but inverted in leads 1 and aVL, suggesting a left atrial source of atrial activation.

The differential diagnosis of the long R-P tachycardias includes atrial tachycardia, atrioventricular (AV) nodal reentry of the unusual variety (fast/slow), and AV reentrant tachycardia using a slowly conducting accessory pathway. The inferior and rightward P wave axis is not consistent with the concentric retrograde activation expected in the unusual variety of AV nodal reentry. Additionally, the response to adenosine with 2:1 AV block eliminates the ventricle as a component of the tachycardia circuit and rules out AV reentry using a concealed accessory pathway. Interestingly, this tachycardia did not terminate after adenosine administration. Intravenously administered adenosine is nearly 100% effective for the termination of AV nodal dependent arrhythmias. Although not widely appreciated, adenosine may also directly terminate atrial tachycardias, especially those arising in the right atrium. Thus, based on the analysis of the 12-lead ECG, as well as the response to adenosine, the most likely diagnosis is atrial tachycardia originating in the left atrium.

With respect to question 2, tachycardia-induced cardiomyopathy has been well described clinically and is likely the cause of left ventricular dysfunction in this patient. The myopathy usually occurs in adults and children after months to years of incessant tachycardia. Although not completely understood, the mechanism of tachycardia-induced myopathy has been hypothesized to be secondary to a decrease in myocardial cellular contractile proteins, an uncoupling of the beta receptor from subsequent intracellular components of the β-adrenergic system, and a depletion of intracellular high-energy phosphate stores. Additionally, a reduction in glycoside receptor density and myocardial adenosine triphosphatase activity have also been reported. The myopathy is often reversible with cessation of the tachycardia.

In regard to question 3, first-line therapy in the past has included antiarrhythmic agents to prevent and suppress atrial tachycardia. The most commonly used agents have included the type IA, IC, and class 3 agents. However, in many cases, atrial tachycardia is refractory to medical therapy. Additionally, antiarrhythmic drug therapy may be limited by side effects and long-term toxicities. Surgical ablative therapy has been described as an effective approach to patients with atrial tachycardia with excision of the active focus and cryoablation of the surrounding tissue. This approach, while highly successful, involves the risks of general anesthesia and a thoracotomy.

More recently, several reports have described the use of radiofrequency ablation for the treatment of atrial tachycardias. In this technique, radiofrequency energy is delivered through an ablation catheter positioned under fluoroscopic guidance in the atrium. Preliminary results indicate that the delivery of energy to a small area in the atrium, identified by electrical mapping, can eliminate the tachycardia with a high degree of success and low incidence of complications. Most series have described the use of this technique for right atrial tachycardias because the right atrium is more amenable to catheter placement. However, several recent case reports have described the use of a transseptal technique to access the left atrium for ablation. In this patient, who is refractory to medical therapy, I would recommend an attempt at curative radiofrequency ablation. If radiofrequency ablation is unsuccessful, then consideration should be given to His bundle ablation with implantation of a permanent dual-chamber pacing system to protect the ventricle from rapid atrial rates. Hopefully, left ventricular function will significantly improve within several months after the radiofrequency ablation.

References

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Comments by J. Marcus Wharton, MD*

In reference to question 1, the patient has a history of an incessant, or nearly incessant, atrial tachyarrhythmia. Possible diagnoses include inappropriate sinus tachycardia, the permanent form of junctional reciprocating tachycardia (PJRT), and an ectopic atrial tachycardia. Although she was initially reported to have “sinus tachycardia,” despite disopyramide therapy, the second electrophysiology study mapped the site of origin to the left atrium, effectively excluding inappropriate sinus tachycardia, assuming that only a single mechanism was responsible for her rhythm problems. PJRT is a nearly incessant form of AV reciprocating tachycardia due to the presence of a very slowly conducting accessory pathway. Although in a classic form the accessory pathways are located in the posteroseptal region, slowly conducting accessory pathways across the left-sided AV groove have been reported and would demonstrate earliest atrial activation during tachycardia at the site of accessory pathway insertion into the left atrium. However, the inability of a ventricular premature beat to reset (or terminate) the tachycardia and the development of 2:1 AV block after administration of adenosine during tachycardia eliminate the ventricles as a component of the reentrant circuit; thus, PJRT is ruled out. Thus, the patient data presented are most consistent with the diagnosis of an ectopic atrial tachycardia. The incessant (rather than paroxysmal) nature of the atrial tachycardia, the failure of atrial premature beats to reset the tachycardia, and the failure of rapid atrial pacing to terminate tachycardia are all consistent with an atrial tachycardia due to automatic, rather than reentrant or triggered, mechanism. We have recently presented data suggesting that most atrial tachycardias are terminated by adenosine. However, atrial tachycardias with a presumed automatic mechanism and left atrial origin are not likely to be terminated, as seen in this case. Thus, the most likely diagnosis is an automatic atrial tachycardia arising in the left atrium.

With respect to question 2, atrial tachycardias frequently arise in the setting of cardiomyopathic processes. However, if the tachycardia is incessant, or nearly so, the continued tachycardia may induce myocardial dysfunction, so-called tachycardia-induced cardiomyopathy. Since either situation may occur, it is clinically difficult to know with certainty which came first, the tachycardia or the cardiomyopathy. Nonetheless, it is important to treat the patient with the underlying assumption that the cardiomyopathy is secondary, because aggressive control of the arrhythmia may allow complete resolution of the myocardial dysfunction or, even if a preexisting cardiomyopathy was present, may prevent further deterioration in left ventricular function.

In regard to question 3, it is important, if possible, to cure the atrial tachycardia for reasons mentioned in the preceding paragraph. As seen in this case, automatic atrial tachycardias frequently are refractory to available medical strategies. Amiodarone possibly has the greatest efficacy, but there are few data to support this assumption, and the drug is associated with frequent toxicity. The new class 3 antiarrhythmic drug, sotalol, which is also a potent β-adrenergic blocking agent, also may be effective in some cases. However, radiofrequency current catheter ablation has evolved as the optimal form of treatment for atrial tachycardia, because catheter ablation is curative (rather than palliative), highly effective, safe, and not associated with side effects seen with drugs. Successful ablation can be achieved in greater than 90% of patients, even with left-sided atrial tachycardias. In this case, catheter ablation should be considered early in the course of the arrhythmia, given its incessant nature and the risk of tachycardia-induced left ventricular dysfunction. If atrial tachycardia ablation is not successful, serious consideration should be given to His bundle ablation for the same reason. Surgical ablation also is an option although certainly much less appealing than catheter ablation, given the morbidity and potential mortality associated with surgical ablation. In addition, automatic atrial tachycardias frequently terminate and remain noninducible during general anesthesia and open-chest procedures, which precludes intraoperative mapping to localize the site of origin and limits surgical success.

REFERENCES

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The electrophysiologic study described herein was consistent with incessant atrial tachycardia, most likely from a left atrial source. The precise site of origin, however, was never found, but its suspected location was the left atrium near the septum. Because of the anticipated technical difficulty in ablating the ectopic focus, medical therapy was first attempted. Propafenone therapy was started in increasing doses, peaking at 300 mg three times daily. Nevertheless, the patient continued having recurrences of rapid atrial tachycardia with one episode of secondary hypotension. In addition, she began having probable side effects from the medication, consisting of visual disturbances, dizziness, and generalized fatigue. Thus, it was decided to treat the disorder with radiofrequency ablation of the AV junction and implantation of a permanent rate-responsive ventricular pacemaker. This was accomplished without complications. Subsequent to that procedure, the patient became asymptomatic and has remained so for approximately 1 year, pursuing an active life of mail carrying and full physical activities. Follow-up echocardiogram performed 8 months after the procedure revealed a normal-sized left ventricle with normal wall motion with the exception of slight septal dyskinesis, compatible with ventricular pacing. Thus, the reduced left ventricular dysfunction was most likely secondary to the incessant tachycardia and was corrected by slowing the heart rate.

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