Case Reports

Swallowing-induced Paroxysmal Supraventricular Tachycardia

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SUMMARY

A 54-year-old man had palpitations on swallowing without any esophageal or heart disease. Electrophysiological findings, including an A wave in the high right atrial leads appearing prior to the A wave on His bundle electrogram, revealed that the arrhythmia was paroxysmal supraventricular tachycardia originating in an ectopic focus of the atrium with intraventricular aberration. Treatment with verapamil, 120 mg/day, reduced his symptoms in spite of an insignificant decrease in the arrhythmia observed with Holter dynamic electrocardiography.

Additional Indexing Words:

Neural reflex

CARDIAC arrhythmias accompanying swallowing are rare disorders, and bradyarrhythmias may be associated with syncopal attacks due to various degrees of atrioventricular (AV) block and AV nodal or sinus dysfunction. Much less information is known concerning the induction of tachyarrhythmias by swallowing. Deglutition tachycardia, first reported in 1926 by Sakai and Mori,¹⁾ has been seen in only 22 references. This report describes a patient presenting paroxysms of atrial tachycardia with intraventricular aberration induced by swallowing and vomiting.

CASE REPORT

A 54-year-old man was referred to our hospital in June, 1985 for evaluation of arrhythmia induced by swallowing and vomiting.

In 1984, he noted the onset of an irregular heart beat and short runs of rapid palpitations accompanied by dizziness and diaphoresis during meals upon swallowing liquid or solid foods. He had no history of hypertension or angina pectoris.

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On physical examination his blood pressure was 120/70 mmHg. Pulse rate was regular and 80 beats/min with a respiratory rate of 16/min. Neck veins were flat. There was no evidence of struma or lymphadenopathy. No abnormal precordial pulsation was evident and heart sounds were normal. The lungs were clear and the abdomen was not remarkable. The extremities were free of edema and all the peripheral pulses were normal. Neurological examination showed no pathological findings.

A 75 gm oral glucose tolerance test gave borderline results. Other routine laboratory studies including complete blood count, serum lipids, serum electrolytes, thyroid function test, urine vanillyl mandelic acid (VMA) were normal, as were chest roentgenograms.

Gastrointestinal work-up including upper gastrointestinal series and gallbladder series revealed no demonstrable abnormality.

Standard 12-lead electrocardiograms (Fig. 1A) revealed regular sinus rhythm at a rate of 80 beats/min. P waves were normal and the P-R interval measured 0.14 sec. QRS configurations and intervals and corrected Q-T intervals were normal. No ST-T change was observed. An echocardiogram was normal and the calculated left ventricular ejection fraction 0.78. A 24-hour Holter dynamic electrocardiographic record revealed numerous episodes of supraventricular tachycardia with intermittently wide QRS complexes. Of 107,012 beats monitored, 1,892 (1.8%) were atrial premature beats or brief paroxysms of supraventricular tachycardia. The majority occurred during the three major meals; the patient was almost free of tachycardia during the sleep period. Swallowing saliva produced either premature atrial contractions or paroxysmal atrial tachycardia. Each episode consisted of 2 to 12 beats at a rate ranging from 140 to 195 beats/min (Fig. 1B). An esophageal lead electrocardiogram revealed a P wave corresponding to each QRS complex, even to wide QRS complexes. Thus, we concluded that this arrhythmia was paroxysmal supraventricular tachycardia with intraventricular aberration.

Neither physical maneuvers including carotid massage, Valsalva test and Ashner's test, nor pharmacological studies with pilocarpine 0.7 mg i.m. and atropine sulfate 0.35 mg i.m. could induce or terminate the tachycardia. The intramuscular injection of 0.7 mg of adrenaline caused a paroxysm which lasted a shorter time than the arrhythmia induced by swallowing. Supraventricular tachycardia was consistently induced by inflating a balloon with 7 to 10 ml of air in the esophagus located between 28 to 35 cm from the incisors.

Cardiac catheterization was performed. Pressure studies of the right atrium, right ventricle and left ventricle were normal. Left ventricular function was normal and coronary arteriography showed no stenosis. Intracardiac electrograms, including His bundle electrogram (Fig. 2), revealed that A-H and

Fig. 1. Resting 12-lead electrocardiogram (A) showing no abnormality and that during swallowing (B) showing paroxysmal tachyarrhythmia including wide QRS complexes.

H-V intervals were normal during both normal sinus rhythm and the tachycardia on drinking water, and that the A-H interval during the tachycardia was almost the same as the interval while the patient was in sinus rhythm. These suggested the tachycardia arose in the atrial lead because the A wave in the high right atrium appeared prior to the A wave in the His bundle electrogram. The tachycardia was not induced by programmed extrastimulation and jumping phenomenon was not observed, suggesting no evidence of an accessory pathway between the atrial and ventricular walls.

Treatment with a combination of propranolol 60 mg/day and quinidine 0.8 gm/day failed to control the arrhythmia. Verapamil 120 mg/day reduced

Fig. 2. His bundle electrocardiogram showing that the A wave in the high right atrial lead appeared prior to the A wave in the His bundle electrocardiogram.

his symptoms in spite of an insignificant decrease in the arrhythmia observed with Holter dynamic electrocardiography.

DISCUSSION

Since the first report on swallowing-induced tachycardia by Sakai and Mori¹⁾ in 1926, only 24 cases have been published.^{1)-9),12)-20)} One case had significant esophageal disease²⁾ and another Wolf-Parkinson-White syndrome,³⁾ while the remaining 22 patients had no demonstrable heart or esophageal diseases. Except for 3, all of the cases, including ours, showed atrial tachycardia; 2 had ventricular tachycardia^{2),4)} and 1 atrial fibrillation.⁵⁾

Electrophysiologic basis of the dysarrhythmia:

The electrophysiologic origin of the tachycardia is unknown. Some⁵⁾⁻⁷⁾ suggest that an ectopic focus is operative, whereas others support a reentrant pathogenesis. In our case, an upright P wave in leads I, V₅, V₆ and the similar shape of the A wave in a high right atrial lead during tachycardia and during normal sinus rhythm suggested that the origin of the abnormal impulses was located around the sinus node or in the high right atrium. In the present studies, however, it is hard to confirm that the arrhythmia was caused through an intra-atrial reentrant mechanism or ectopic focus.

Mechanism of tachycardia (direct mechanical versus vagal stimulation):

The pathophysiology of the arrhythmia is obscure. Some authors⁵⁾⁻⁷⁾ suggested a direct mechanical interaction between the distended esophagus and the adjacent left atrium. They demonstrated the induction of the arrhythmia by inflation of a balloon in the esophagus only at the subcarinal

level. However, others⁸⁾ could not reproduce the arrhythmia by the procedure. Lindsay⁹⁾ proposed a vagal nerve-mediated neural reflex as the initiating mechanism. We also believe that in the present case the neural reflex plays a major role in inducing the tachycardia. This concept is supported by three lines of clinical evidence confirmed by electrophysiological and roentgenographic studies: (1) the arrhythmia was quite reproducible with swallowing, independent of the kind and volume of foods, drinks and saliva; (2) intracardiac electrogram elucidated earlier excitation of the high right atrium; (3) tachycardia was produced by the inflation of a balloon in the esophagus 28 to 35 cm from the incisors where the left atrium was not in direct contact. Furthermore, there is also some reason to believe that the afferent path of the reflex is the superior laryngeal nerve in some cases of swallowing-induced atrial tachycardia. Although the more common observation is that vagotonia suppresses supraventricular tachycardia, there are experimental and clinical data demonstrating that increases in vagal tone could induce tachycardias.^{10), 11)} Intramuscular injection of 0.35 mg atropine sulfate failed in our patient to abolish induction of the tachycardia. However, the dose of atropine was not sufficient to block the vagal reflex completely, and it is theorized that a higher dose of atropine could eliminate the tachycardia.

Therapy:

Circular esophageal myotomy was performed with some success in a case presented by Kalloor²⁾ in which incoordinate peristalsis of the esophagus induced ventricular tachycardia and atrial fibrillation. Nevertheless, almost all patients reported previously received medical treatment with good results. Various drugs have been proposed for the treatment of the arrhythmia and the most available agents are quinidine sulfate, procainamide, and recently, verapamil. In a case with WPW syndrome³⁾ arrhythmia was only controlled by amiodarone. In our case quinidine with propranolol was not effective. With verapamil his complaints disappeared, while Holter dynamic ECG revealed a minimum effect of the drug on the tachycardia. He has been in good health for more than 4 months.

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