Case Report

A young female patient with recurrent paroxysms of inappropriate sinus tachycardia

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Abstract

Tachycardia is one of the most common presentations in medical practice. In most cases, it could be attributed to emotional problems, anemia, or thyroid disorders. A 16-year-old female visited primary care clinics with the complaints of palpitation for years and initially diagnosed as a case of anxiety or stress. Her condition worsened and became more symptomatic over the last few months for which investigations were done. A provisional diagnosis of pre-excitation syndrome, Lown-Ganong-Syndrome, was made. This was based on short PR interval in ECG and an attack of supraventricular tachycardia and another attack of brief atrial fibrillation in addition to the family history of her mother of having pre-excitation abnormality. She was sent to electrophysiological study where no concealed accessory pathway was confirmed, and the diagnosis of inappropriate sinus tachycardia was the final diagnosis and medical treatment was initiated.

Keywords: Electrophysiological study, inappropriate sinus tachycardia, pre-excitation syndrome, tachycardia

Introduction

Palpitations are a common problem in the primary care setting of which cardiac causes are the most concerning etiology and the sinus arrhythmia is the most reported. In addition, the pre-excitation syndrome via an accessory pathway as in Wolff-Parkinson-White (WPW) and Lown-Ganong-Levine (LGL) syndrome is not uncommon.[1] Inappropriate sinus tachycardia (IST) is an often-under-recognized problem with significant morbidity. There is a paucity of knowledge regarding its pathophysiology. IST is one of the rare presentations in general practice. Pellegrini CN *et al.*[2] identified 7 of 604 middle-aged subjects as meeting criteria for IST. The electrophysiological study (EPS) evolved rapidly over the recent years, in the diagnosis of arrhythmias.

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Case Report

A 16-year-old female was seen in primary care clinic with patient consent signed by her and the guardian, presents with 9-year history of palpitation of fast heartbeats of sudden onset and which used to stop abruptly. She was reassured that her racing heart is mostly due to stress and anxiety. For the last 3 months, her condition worsened as she started to have shortness of breath, giddiness, and fatigue. Most of these attacks were triggered with efforts like climbing stairs, and recently over the last few days, the attacks were triggered when she eats or drinks. She was not on any medication. Her past medical history was unremarkable.

Family history: her mother is a known case of WPW syndrome and she is stable for the last few years. Her father is known hypertensive with sustained ventricular bigeminy. She has 2 young healthy brothers of 6 and 9 years.

Vital signs: weight 51 kg, height 157 cm, and blood pressure of 112/64 mmHg. On examination: pulse 94 beats per minute (BPM) and regular, no signs of anemia or hyperthyroidism,

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cardiovascular: normal heart sounds and no added murmur; chest and abdomen were normal.

The following investigations were requested: CBC, coagulation profile biochemistry (blood sugar, renal, liver, bone, and lipid profiles), thyroid functions, and chest X-ray, all came to be within normal limits. ECG revealed regular sinus rhythm, short PR interval <3 mm, absent delta waves with normal QRS morphology [Figure 1].

The ECG findings raised the diagnosis of pre-excitation abnormality and the high possibility of LGL syndrome.

Cardiac echocardiogram came to be within normal findings.

Holter ECG monitor for 48 h was requested [Table 1].

Narrative summary of Holter ECG monitor

The average heart rate was 97 BPM; the minimum and maximum heart rates were 53 and 190 BPM, respectively. The patient rhythm included 2 min 57 s of bradycardia and 20 h 24 min 20 s of sinus tachycardia. The fastest one lasted 2 h 59 min 14 s with maximum heart rate of 190 BPM. The supraventricular ectopic activity consisted of 70 beats of which, 4 were in atrial couplets, 41 were late beats, and 25 were single premature atrial contractions (PACs). The patient had 1 episode of atrial fibrillation, with total time of 29 s. The episode of atrial fibrillation (AF) with the fastest ventricular response was 90 BPM and the slowest rate was 63 BPM.

She was admitted for EPS with 3-D mapping +/- ablation as a provisional diagnosis of LGL syndrome was the highest possibility. During admission, the Holter monitor documented an attack of supraventricular tachycardia of 200 BPM [Figure 2] during climbing the first flight of stairs.

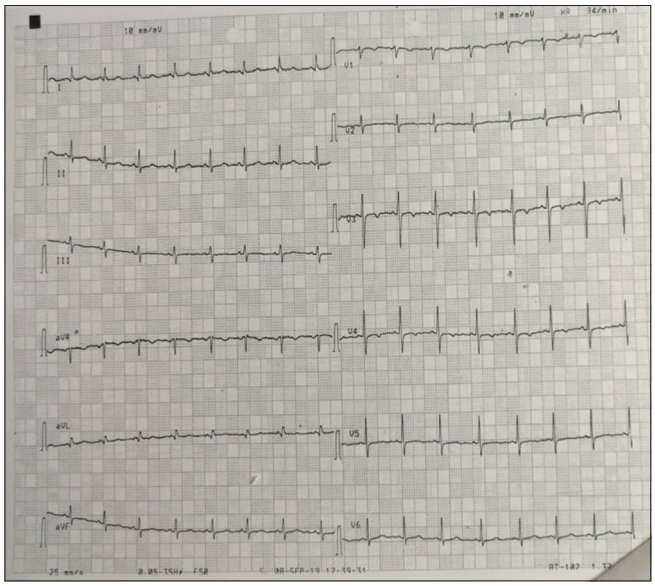

Figure 1: A 12-lead resting ECG showing short PR interval

EPS was done with 2 radiofrequency (RF) sheaths: coronary sinus (CS) catheter and RF ablation catheter. No evidence of dual atrioventricular node physiology. No evidence of a concealed accessory pathway. During catheter manipulation, atrial flutter was induced by 2:1 conduction where adenosine and digoxin were given. The flutter was followed by a brief run of AF, then converted spontaneously to sinus rhythm. Trials were made by pacing to induce any atrial tachyarrhythmia but were failed. Activation map was performed during tachycardia of 130–140 and it was clearly originating from usual sinoatrial (SA) node location. Based on this study and her history, our assessment was Inappropriate Sinus Tachycardia (IST), which necessitate medical treatment and LGL syndrome was excluded. The patient was put on bisoprolol 2.5 mg daily and advised to be seen after 3 days. When seen, she was fine with a heart rate of 92 BPM and blood

Table 1: 48-h Holter ECG monitor showing sinus tachycardia and one episode of atrial fibrillation

Heart Rate Data	Ventricular Ectopy
Total Beats: 280214 Beat	Total VE Beats: 0 (0.0%)
analyzed% 99.8	Triplets: 0
Min HR: 53 BPM at 06:27:10	Couplets: 0
Avg HR: 97 BPM	R on T: 0
Max HR: 190 BPM at 07:12:24	Bi/Trigeminy: 0
Heart Rate Variability	Supraventricular Ectopy
ASDNN 5: 48.4 ms	Total SVE Beats: 70 (0.0%)
SDANN 5 : 133.1 ms	Atrial Runs: 0
SDNN: 143.1 ms	Beats : 0
RMSSD: 38.4 ms	Longest : 0
QT Analysis	Fastest : 0 BPM
QT Min: QTc Min:	Atrial Pairs : 2 Events
QT Avg: QTc Avg:	Longest R-R : 1.3 s at 05:12:30
QT Max: QTc Max:	Single PAC's : 25
QTc Max >450 ms	Bi/Trigeminy : 0/0 Beats
ST Episode Analysis	Atrial Fibrillation
ch1ch2 ch3	A Fib Beats : 37 (0.0%)
Min ST Level - - -	Duration : 0.5 min
Min ST Level - - -	Events: 1
ST Episode - - -	

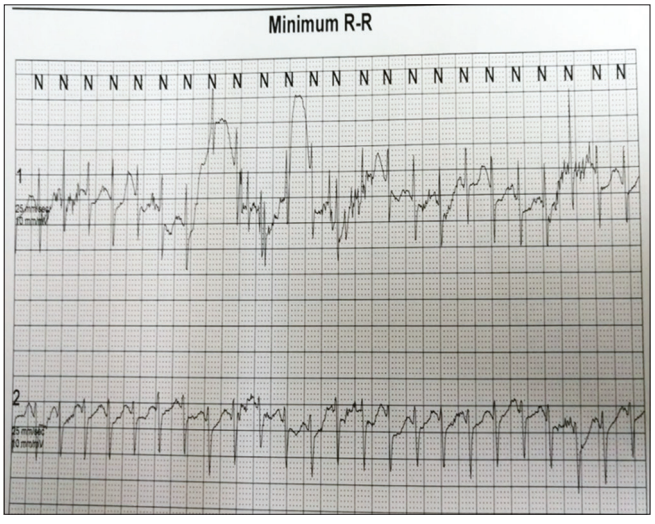

Figure 2: ECG strip recorded by Holter monitor showing supraventricular tachycardia

pressure of 108/62 mmHg. The dose was remained and advised to be seen after a week. In this visit, pulse was 78 BPM and blood pressure 106/58 mmHg and did not experience any paroxysms of tachycardia but trying to avoid more efforts. She was advised to go back to normal physical efforts gradually and regular follow-up.

Discussion

Inappropriate sinus tachycardia, also called chronic nonparoxysmal sinus tachycardia, is an unusual condition that occurs in individuals without apparent heart disease or other causes of sinus tachycardia, such as hyperthyroidism, anemia, or fever, and is generally considered a diagnosis of exclusion.[3]

The pathophysiologic mechanism of this syndrome is poorly understood and is thought to consist of intrinsic hyperactivity of the sinus node coupled with autonomic agitation modulated by neurohormonal influences.

IST is a syndrome characterized by unexpected attacks of fast sinus rates at rest, with minimal physical effort, or even both. The resting daytime sinus rates reach more than 100 BPM and average 24-h heart rates usually exceed 90 BPM. The condition could be triggered by caffeine or alcohol and anxiety conditions. IST could be manifested by a spectrum of symptoms including palpitations, shortness of breath, weakness, fatigue, dizziness, or near syncope.[4,5]

Most patients are young females, but the epidemiologic characteristics are uncertain. The episodes of tachycardia are noticed to come abruptly and can persist over months or even years. The prognosis generally is benign. A reason for a benign prognosis is that although IST patients have paroxysms of fast sinus tachycardia, the heart rate slows down during sleep and in various diurnal patterns.[6]

Managing patients with IST remains a challenge, because the syndrome itself is obscure and the complexity of its nature. In IST, there is no one therapy that can reduce heart rate and alleviate the symptoms effectively. Beta-blockers, non-dihydropyridine calcium channel blockers beside the use of exercise training to improve quality of life could be the solution in most cases.[7] It is important to distinguish IST from so-called appropriate sinus tachycardia and from postural orthostatic tachycardia syndrome, with which overlap may occur. In addition, we have to determine first if there is a trigger or an event that precipitated the symptoms because this may help to determine the longevity of the problem.

Usually, the treatment begins with modest doses of beta-blockers. Small studies and several case reports have shown the potential value of the drug ivabradine to treat IST.[8] Ivabradine can have a dramatic effect on slowing of heart rate. It can be efficient and alternative to beta-blockers but it is not available in Saudi Arabia.

Ivabradine may precipitate excess bradycardia, especially in combination with-blockers or calcium-channel blockers. The other side effect, which is rare, AF. The number of days of the use of ivabradine in dose of 5 mg twice daily, to cause one new case of AF is 208 days/year.[9] Ivabradine at a dose of 5.0 to 7.5 mg twice daily, if available, may be highly effective and should be considered in cases of IST. In our case, parents were advised if they can get the medicine, beta-blocker is preferred as it is highly selective on SA node to reduce its rate.

Radiofrequency catheter ablation can be attempted although ablation is performed very rarely and only after all other therapeutic options have been exhausted. The goal is to modify the sinus node without ablating it completely to avoid the need for permanent pacemaker implantation.[10] Finally, IST is a diagnosis of exclusion and is difficult to characterize symptomatic condition that represents a spectrum of disorders related to increased sinus node automaticity, disordered autonomic activation, or both.

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Conflicts of interest

There are no conflicts of interest.

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