

DIAGNOSIS AND TREATMENT OF INAPPROPRIATE SINUS TACHYCARDIA SYNDROME IN A PATIENT SCHEDULED FOR SURGERY: CLINICAL CASE

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ABSTRACT

Background: Inappropriate Sinus Tachycardia is a rare syndrome in which increased cardiac frequency is not correlated with physiological requirements. The authors describe the clinical case of a woman whose pre-operative evaluation led to the diagnosis of Inappropriate Sinus Tachycardia.

Methods: The patient was a Caucasian woman, 28 years old, 165cm tall and weighing 55kg, who was scheduled for femoral hernia surgery. The pre-operative evaluation included a careful clinical exam and history, ECG, echocardiogram, Holter dynamic electrocardiography, and Tilt Test, which led to a diagnosis of Inappropriate Sinus Tachycardia. Heart rate lowering therapy was started with 5mg Ivabradine twice daily associated with 2.5mg bisoprolol, and local anesthesia was scheduled.

Results: During surgery cardiac frequency stayed under 120bpm, and no sedation of the patient was needed. The medical regimen continued in the post-operative period.

Conclusions: The careful pre-operative evaluation and subsequent heart rate lowering therapy resulted in an improvement of the clinical symptoms as well as better hemodynamic stability in the intra-operative period and a better clinical outcome.

Key words: Inappropriate sinus tachycardia; cardiac frequency; ECG; Ivabradin; bisoprolol.

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Introduction

Inappropriate Sinus Tachycardia (IST) is a syndrome characterized by increased cardiac frequency (CF) at rest (over 100bpm), which increases excessively even during mild physical effort, as well as a very slow return to base CF. The syndrome is often accompanied by a series of symptoms that include heart palpitation, easy fatigue, and sometimes even dizziness or syncope⁽¹⁾. When the long-term prognosis seems benign, no treatment may be necessary, but the conditions of surgical stress, excess of catecholamine or sympathetic activation may cause hemodynamically critical CF.

Materials and methods

In November 2013 the patient (female age 28, height 165cm, weight 55kg) came to our clinic for a pre-operative cardiology exam because she was

scheduled for femoral hernia surgery with a prosthetic plug. The ECG revealed sinus tachycardia at 120bpm, which increased to 140bpm when the patient became upright. The patient also reported asthenia and palpitation for the last 6-7 months. The history did not reveal any use of stimulant drugs, alcohol, tobacco, nor anxiety attacks. The patient was also afebrile and reported no algic pathology. In the clinical exam the patient had rhythmic tones with free pauses, physiological vascular murmur (VM), and an absence of peripheral edema. The lab exams excluded anemia and hyperthyroidism. The echocardiogram subsequently showed a conserved ejection fraction (EF), and normal segment kinetics. No mitral and/or aortic insufficiency nor pericardial loss was found. At the second visit the patient was evaluated again and we observed that the sinus tachycardia persisted.

A Holter dynamic ECG was then performed, which showed sinus rhythm with an average day-

time CF at rest over 100bpm, and an average 24h CF 90bpm, unexplainable on the basis of physiological mechanisms of demand or the usual conditions for increased CF. Hence, the diagnosis of suspected inappropriate sinus tachycardia. We then performed a Tilt Test to exclude Postural Orthostatic Tachycardia Syndrome (POTS). That exam revealed a sudden increase in CF when moving from reclined to upright posture, in the absence of any signs of orthostatic hypotension.

Once the IST diagnosis was confirmed and in preparation for the surgical operation the patient underwent heart rate lowering treatment with 5mg Ivabradin twice daily associated with 2.5mg bisoprolol, which reduced the base CF to 90bpm. The surgical operation was performed using local anesthesia. The skin and subcutaneous tissue were infiltrated using an anesthetic solution consisting of carbocaine 2% (10ml), buffered with sodium bicarbonate 8.4% (2ml) and physiological solution (8ml). The surgical incision followed with a less concentrated anesthetic solution consisting of carbocaine 2% (20ml), sodium bicarbonate 8.4% (6ml) and physiological solution (60ml).

Results

The surgical operation lasted 80 minutes, and the patient needed no sedation. Administration of both Ivabradin and bisoprolol continued in the post-operative period, during which CF did not exceed 120bpm.

Discussion

Differential diagnosis must consider appropriate sinus tachycardia, in which physiological and psychological factors may be recognized as the cause. Extra-systole tachycardia must also be considered, as well as atria tachycardia and sinus re-entry, and POTS⁽²⁾. The latter is diagnosed primarily through the Tilt Test⁽³⁾. IST is not postural as is POTS. In the latter there is a persistent increase in CF of over 30bpm or a frequency above 120bpm within 10min of moving from reclining to upright posture in the absence of orthostatic hypotension. In contrast, in IST the increase in CF is almost immediate⁽³⁾, as in our case.

We used low doses of beta-blockers associated with Ivabradin because of their synergic effect on CF⁽⁴⁾. While Ivabradin acts directly to modulate the IF current, beta-blockers reduces the increase in cAMP by indirectly modulating that

current⁽⁵⁾. Today the IF current is considered the principal determinant of PM activity of the SA node⁽⁶⁾. Beta-blockers improve the modulation of sympathetic activity which is normally emphasized during a surgical operation. The use of low doses of beta-blockers was motivated by their indirect action of blocking the calcium channel which can cause interference with cardiac contractility, while full doses of Ivabradin were used to better control the CF because in addition to the lack of systemic effects their effectiveness increases according to the increase in CF up to a maximum of 20-30%.

Conclusions

In general sinus tachycardia is an understandable clinical condition, but otherwise can be the result of IST. This symptomatic condition is difficult to explain since the disturbances are often correlated with increased automaticity of the SA node, an autonomic dysfunction between sympathetic and vagal activation or both. Pharmacological control of CF can guarantee better quality of life as well as better hemodynamic response in symptomatic subjects undergoing surgical treatment.

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