Case Report

Hyperthyroidism and Sick Sinus Syndrome, a Rare but Challenging Association: A Study of Three Cases

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Abstract

Hyperthyroidism is usually associated with sinus tachycardia or supraventricular tachyarrhythmias, but rarely with dysfunction of the sinus node or other conduction disturbances. Evidence of bradyarrhythmia in patients with hyperthyroidism is clinically relevant, but the fact that several drugs with negative chronotropic effects (beta-blockers and calcium channel antagonists) are frequently used in the management of these patients must be taken into account. In the presence of sick sinus syndrome (SSS) or other conductance disturbances, therapy with agents that delay the activity of sinus node or atrioventricular conduction can lead to extreme bradycardia with syncope. In this paper, aspects of diagnosis and therapy in three patients with SSS and hyperthyroidism, admitted in the Clinic of Endocrinology or/and Cardiology of the County Hospital, Timisoara, have been presented.

Key Messages: This study presents particularities of diagnosis and evolution of sick sinus syndrome in three patients with hyperthyroidism, the therapeutic implications of this association, and the best management of these patients.

Keywords: *Arrhythmias, bradycardia, hyperthyroidism, sick sinus syndrome*

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Introduction

Rhythm disturbances are frequently encountered in hyperthyroidism, the most common being sinus tachycardia, but atrial fibrillation (AF) is also frequently diagnosed.[1] Conduction disturbances, such as sick sinus syndrome (SSS)[2] and atrioventricular (AV) block,[3] are seldom seen in patients with thyrotoxicosis. Pathophysiological mechanisms are controversial. Few cases are mentioned in the literature and we found about three cases with SSS and hyperthyroidism.[4-8]

Case History

Case 1

A 48-year-old woman was admitted in February 2008 complaining of palpitations, diaphoresis, weight loss (15 kg), and insomnia. The clinical examination revealed tremor of the hands, exophthalmia, a small goiter, and heart rate of 62 b/min. Laboratory tests showed low thyroid-stimulating hormone (TSH) = 0.0001 μUI/mL (0.46-4.68), free thyroxine (FT_4) = 39 pmol/L (10.0-28.2), and free triiodothyronine (FT_3) = 21.04 pmol/L (4.28-8.1). Thyroid ultrasonography evidenced a small hypoechoic, hypervascularized, diffuse goiter, and total thyroid volume (TTV) of 21.12 mL. Electrocardiogram (ECG) and echocardiography were normal. The diagnosis was severe thyrotoxicosis due to Graves´ disease. Therapy with 30 mg antithyroid drug methimazole and 50 mg metoprolol succinate was started with good clinical evolution. After 3 days of therapy, the patient claimed dizziness and on the clinical examination, sinus bradycardia, 44 b/min, was noted. On Holter-ECG monitoring, the episodes of extreme sinus bradycardia, 32 b/min with five sinusal pauses of 2400–2680 msec. were detected [Figure 1a]. The beta-blocker therapy was stopped, heart rate rose to 50 b/min; but a new Holter-ECG monitoring revealed bradycardia 36 b/min and sinusal pauses. An (AAI)

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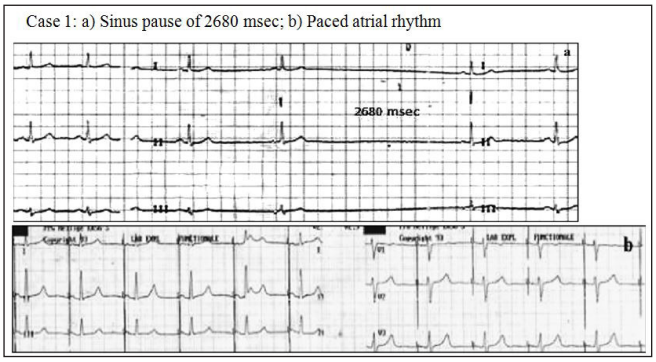

Figure 1: Case 1: (a) Sinus pause of 2680 msec and (b) stimulated atrial rhythm.

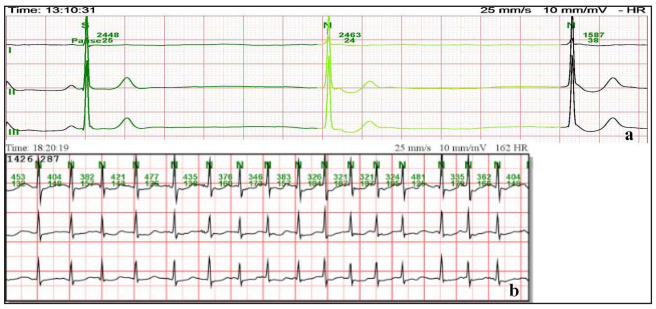

Figure 2: Case 2: Holter monitoring: (a) Sinus bradycardia and (b) paroxysmal atrial fibrillation.

atrial inhibited pacemaker was inserted with good clinical evolution [Figure 1b].

During treatment with methimazole, the patient presented several relapses of hyperthyroidism, requiring surgical treatment. At a control in September 2014, hypothyroidism was detected and therapy with L-thyroxine 50 μg daily was started.

Case 2

A 63-year-old woman was admitted in December 2012 complaining of palpitations, diaphoresis, restlessness, and weight loss (7 kg). Pathological findings during the clinical examination showed a nodular goiter and episodes of tachyarrhythmias. Thyroid ultrasonography revealed multinodular goiter (TTV = 43 mL) and the hormonal data were TSH = 0.10 μUI/mL, with normal FT_4 and FT_3. The thyroid scintigraphy with Tc99m, evidenced "hot nodules." ECG revealed sinus rhythm, 60 b/min; echocardiography was normal. Because of dizziness and bradycardia, Holter-ECG monitoring was performed (minimum heart rate 28 b/min, with five episodes of paroxysmal AF and isolated premature ventricular beats (PVBs) [Figure 2a and Figure 2b].

Surgical treatment of the goiter was recommended, but the patient refused it. Treatment with methimazole 15 mg/day, bisoprolol 1.25 mg/day, (in order to prevent arrhythmias), and rivaroxaban 20 mg/day was commenced, with good clinical response.

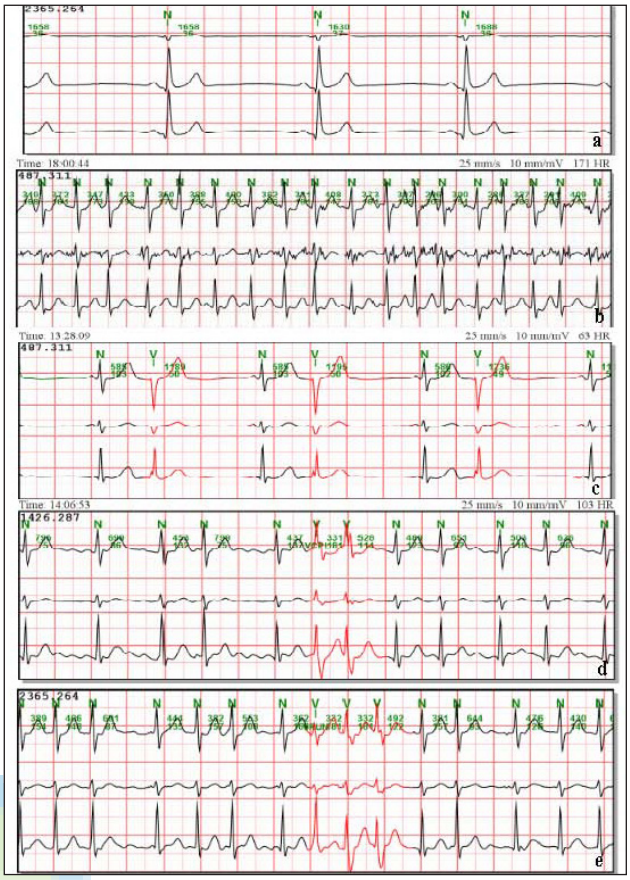

Figure 3: Case 3: (a) Sinus bradycardia, (b) paroxysmal atrial fibrillation, (c) bigeminated premature ventricular beat, (d) doublets, and (e) triplets.

In February 2013, the patient was seen in euthyroid state with similar thyroid-ultrasonographic and echocardiographic findings and sinus rhythm 62 b/min on ECG. The patient continued the former treatment with reduction of the dose of methimazole to 5 mg/day. No indication for a pacemaker implantation was established.

Case 3

A 66-year-old woman, followed up with Graves´ disease since 2008 with several moderate relapses, under chronic treatment with methimazole 2.5 mg/day, was admitted in October 2013 for palpitations, weight loss, insomnia, and a small goiter. The thyroid ultrasonography showed a hypoechoic and hypervascularized goiter with TTV = 26 mL. Values of TSH were 0.015 μUI/mL and of FT_4 = 20.3 pmol/L and FT_3 = 7.8 pmol/L. The physical examination and the ECG demonstrated paroxysmal AF. The echocardiography was normal. On Holter-ECG monitoring, there was evidence of sinus bradycardia (minimum 37 b/min), nine episodes of AF, [Figure 3a and 3b], as well as multiple PVB (bigeminated, doublets, and triplets), [Figure 3c, 3d and 3e]. Methimazole was increased to 15 mg/day, with gradual reduction. In order to treat arrhythmias, metoprolol succinate 50 mg/day was associated and anticoagulation with acenocoumarol

2 mg/day was started. After normalization of TSH value (0.32 μUI/mL), her evolution was good. The episodes of arrhythmias were fewer and shorter and there was no indication for pacemaker implantation.

Discussion

It is known that thyroid hormones exert a positive chronotropic effect on the heart, but their excess modifies the regulation of ion transporters and alters the action potential generated in the cardiac pacemaker cells. These mechanisms explain the development of tachyarrhythmias in hyperthyroidism.[1]

There are controversial opinions regarding the pathogenesis of conduction disturbances in hyperthyroidism: some authors suggest that the autonomic nervous system would act by reciprocal excitation and exacerbate a latent hypervagotonia;[1] whereas others suggest the possibility of an autoimmune response causing inflammation, followed by fibrosis of the cardiac conduction pathways.[1,2] Another hypothesis is that of a direct toxic effect of thyroxine in excess of the cardiac conduction system,[3] inducing SSS, sinoatrial block, or AV block.[7-9]

Because most of the reported cases with SSS or/and AV block have been observed among patients with Graves' disease, it has been suggested that the same autoimmune pathological process that affects the thyroid could influence the conducting system.[10]

Two of the cases presented in this paper had Graves' disease and one had multinodular goiter. However, the most consistent clinical observation appears to be the resolution of conduction delays as thyroid hormone levels decrease. This possibly implicates a direct role of thyroid hormones or hyperthyroidism-induced hypervagotonia.[3]

In two cases, bradycardia was evident during thyrotoxicosis, patient 2 had subclinical hyperthyroidism in the moment of the diagnosis of SSS, an aspect that was less described in the literature.[7] Yet, the question remains if there is not a concomitance between hyperthyroidism and SSS, but the improvement of symptoms under antithyroid therapy supports the hypothesis of an association between these two dysfunctions.[6]

Only the first patient needed a pacemaker, the other two received drug therapy and remained under observation. Neither of them developed syncope, requiring transitory or permanent pacing.[7] A problem was the treatment of arrhythmias, but their frequency and severity reduced in parallel with the improvement of the thyroid disease,[6] so there was no need for other antiarrhythmic agents.

Conclusions

Hyperthyroidism and SSS is a rare association, identified mostly in patients with Graves' disease, even in subclinical stage, raising therapeutic problems in the presence of concomitant tachyarrhythmias. The evolution is good in most cases, after the normalization of thyroid hormones, seldom requiring implantation of a pacemaker.

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Conflicts of interest

There are no conflicts of interest.

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