OLGU SUNUMU / CASE REPORT

Hipertiroidiye bağlı geri dönüşümlü birinci derece atriyoventriküler blok

Reversible first-degree atrioventricular block due to hyperthyroidism

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Özet– Hipertiroidi çoğunlukla taşiaritmilere neden olmaktadır. Hipertiroidiye bağlı geri dönüşümlü atriyoventriküler (AV) blok nadirdir. Bu yazıda, hipertiroidizmin neden olduğu ve antitiroid tedaviyle düzelen atriyoventriküler bloklu olgu sunuldu.

Summary– Hyperthyroidism often causes tachyarrhythmias. Reversible atrioventricular block caused by hyperthyroidism is a rare occurrence. In this paper a case of atrioventricular block due to hyperthyroidism that recovered after antithyroid drug treatment has been presented.

Abbreviations

AV Atrioventricular

EKG Electrocardiogram

Hyperthyroidism exerts important effects on cardiovascular system. In patients with hyperthroidism frequently atrial, and ventricular arrhytmias are frequently seen.In addition though rarely seen, most of the time antithyroid drug treatment may achieve sinus rhythm.

CASE PRESENTATION

A 58-year-old female patient presented to cardiology outpatient clinic with complaints of lassitude, and headache persisting for nearly two weeks The patient was followed up for approximately for 10 years with the diagnosis of hypertension, and she was regularly using ramipril at daily doses of 2.5 mg Onn her physical examination, her blood pressure was 124/75 mm Hg; her rhythmic pulse rate, 65 bpm, As an abnormal sign cardiac auascultation revealed only decrease in the severity of SI.. On electrocardiogram (EKG) heart rate was 75 bpm, and first-degree atrioventricular (AV) block was detected (PR interval, 275 ms) (Figure 1).

Transthoracic echocardiography, and telecardiography results were within normal limits. Biochemical parametres, C-reactive protein, sedimentation rate, blood counts were within normal limits Thyroid function test results were as follows: thyroid stimulating hormone (TSH) <0.005 IU/dL (normal range 0.4–4.5), free triiodothyronine 3 (T3): 9.44 pg/ml (normal range: 2.8–7.1) and free tetraiodothyronine 4 (T4): 36.41 ng/dL (normal range 12–22). Millimetric nodules were seen on tyhroid ultrasonograms. On 24-hour ambulatory Holter monitorization of the patient, first-degree atrioventricular block with average heart rate of 62 bpm, and maximum PR interval of 399 ms. Because of Holter and EKG findings, and complaints of dizziness of the patient electrophysiologic study was planned. After consent of the patient was obtained electrode catheter was inserted percutaneously, and advanced through femoral vein to obtain recordings from His bundle, and right ventricle. Basic measurements detected advanced first-degree AV block (AH interval 260 ms, HV: 46 ms and Wenchebach point 440 ms). With incremental stimulations of atrium, normal sinus, and AV node functions were noted.. AH interval (230 ms) did not normalize significantly after intravenous atropin administration (Figures 2a, and b). The symptoms of the patient suggested hyperthyroidism as the cause of AV block. Then antithyroid drug therapy was initiated, and on the seventh day of the therapy patient's sinus rhythm returned to normal, and her complaints disappeared. (Figure 3). During the first three months of the follow-up period arrhytmia was not observed.

Submitted on: 09. 06.2016 *Accepted for publication on:* 10.28.2016

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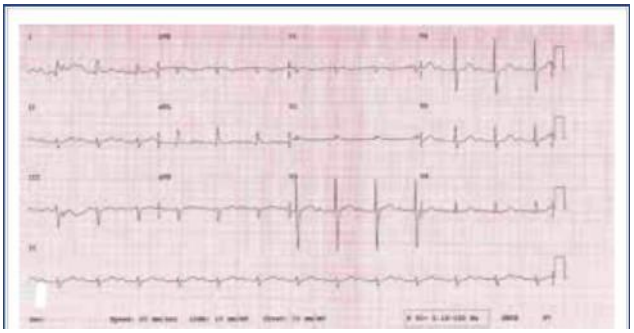

Figure 1. Admission electrocardiogram of the patient is consistent with 1. degree atrioventricular block.

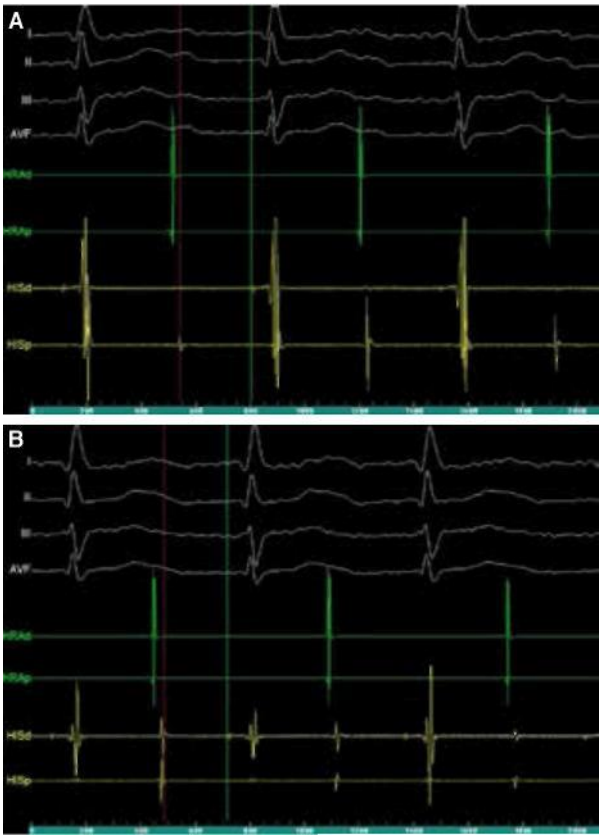

Figure 2. (A, B) baseline, and post-atropin AH recordings during electrophysiologic study

DISCUSSION

Thyroid dysfunction frequently effects cardiovascular system. Generally hyperthyroidism is associated with tachyarrhytmias, hypothyroidism with bradyarrhytmia. As reported in some studies, AV conduction disorders may also cause AV-conduction disorders.[1–3] However since it frequently induces tachyarrhytmias as an underlying pathology in patients with atrioventricular block, hyperthyroidism may be overlooked in differential diagnosis of these patients.

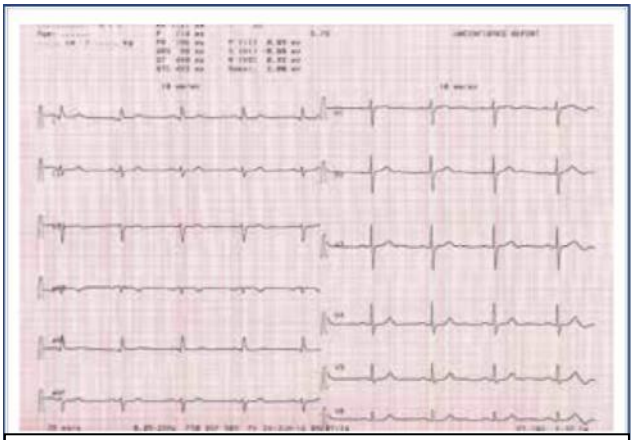

Figure 3. Electrocardiogram of the patient reveals sinus rhythm after treatment

During follow-up of 76 patients with hyperthyroidism, PR of >0.22 sec was reported in five patients.[4] Therefore not only in cases with advanced AV blocks, but also in patients with first-degree AV block hyperthyroidism sh be taken into consideration

Though mechanisms of the effects of hyperthyroidism on conduction system which result in bradycardia are not known for sure, some mechanisms have been suggested including for instance direct effects of thyroid hormones, autoimmune mechanisms, increase in vagal tonus as response to enhanced adrenergic tonus which predominates in hyperthyroidism .[1,5,6] However as we saw in our patient, conduction disorder detected on EPS was refractory to atropin which suggested weak effect of increased vagal tonus. Of course, progressive characteristic of the block may be another reason for lack of any post-atropin improvement in AH interval as demonstrated on EPS.

Bradyarrhytmias seen in patients with hyperthyroidism have been generally associated with acute infection, hypercalcemia or other concomitant pathologies as underlying structural heart disease .[7] However irrespective of all these etiologic factors, it has been conceived that atrioventricular block can occur secondary to the direct effects of autoimmune mechanisms on myocardium, and ion channels. Resolution of atrioventricular conduction disorder before thyroid hormone levels normalized after antithyroid drug therapy supports thie assertion. In our patient, AV conduction normalized before posttreatment decrease in thyroid hormones within physiologic range was observed. At the same time hyperthyroidism may also deteriorate underlying conduction disorder. AV blocks not primarily related to hyperthyroidism do not respond to antithyroid drug treatment, and most frequently the need for implantation of permanent pacemaker arises.[8]

Whereas, conduction disorders primarily due to hyperthyroidism, even at the level of complete AV block resolve within a short time following antithyroidal treatment.[9] Delay in the treatment of hypertthyroridism may induce progression of the block. Since our patient was symptomatic first-degree AV block, diagnosis was promptly made, and treatment was in initiated. Maybe thanks to appearance of symptoms, development of advanced AV may be precluded. However, beta-blockers are frequently prescribed in the treatment of hyperthyroidism which may lead to progression of AV conduction disorders. Therefore patients with hyperthroidism should be followed up with regular electrocardiographic examinations.

In conclusion, the diagnosis of hyperthyroidism should be considered in the differential diagnosis of the bradyarrhytmic patients, and this condition usually resolves with treatment.

Conflict of Interest: None declared.

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Anahtar sözcükler: Atriyoventriküler blok; baş dönmesi; hipertiroidi.

Keywords: Atrioventricular block; dizziness; hyperthyroidism.