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Sick sinus syndrome secondary to primary hyperparathyroidism

Primer hiperparatiroidiye sekonder gelişen hasta sinüs sendromu

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SUMMARY

The effects of hypercalcemia on the heart and the resulting alternations of the electrocardiogram have been well established. However, there are only limited number of reports in the literature on primary hyperparathyroidism leading to clinically significant arrhythmias. We present a patient who was diagnosed with symptomatic sick sinus syndrome in the setting of moderate hypercalcemia secondary to primary hyperparathyroidism in this case report. After the surgical operation for primary hyperparathyroidism, serum calcium level returned to normal range and patient's complaints and arrhytmic findings recovered. The patient was asymptomatic for the following 13 years.

Keywords: Sick sinus syndrome, primary hyperparathyroidism, hypercalcemia

ÖZET

Hiperkalseminin kalp üzerine olan etkileri ve sonucunda gelişen elektrokardiyografik değişiklikler iyi bilinmektedir. Fakat literatürde, primer hiperparatiroidiye bağlı ciddi aritmi gelişimi ile ilgili sınırlı sayıda bildiri bulunmaktadır. Bu olguda, primer hiperparatirodiye sekonder gelilen orta dereceli hiperkalsemi varlığında, semptomatik hasta sinüs sendromu gelişen bir hasta sunulmuştur. Primer hiperparatiroidiye yönelik yapılan cerrahi operasyon sonrası, serum kalsiyum düzeyi normal sınırlara döndü ve hastanın şikayetleri ve aritmik bulguları düzeldi. Takip eden 13 yıl boyunca hasta asemptomatik kaldı.

Anahtar sözcükler: Hasta sinüs sendromu, primer hiperparatiroidi, hiperkalsemi

INTRODUCTION

Typical electrocardiographic changes of hypercalcemia, including hypercalcemia of hyperparathroidism, include prolongation of the P-R interval, shortenning of Q-T interval, and broad flattening of the apex of the T wave, bradycardia[1, 2]. There are only few reports in the literature on hyperparathroidism-associated hypercalcemia leading to clinically significant bradyarrhythmias[3-6].

However, the underlying pathophysiology of this process is still not fully clarified. Similarly, a recent study failed to demonstrate any effect of moderate hypercalcemia on cardiac conduction[7].

We present a case with moderate hypercalcemia secondary to primary hyperparathroidism, exhibiting electrocardiagraphic signs of sick sinus syndrome, including sinus bradycardia, sinus pause with slow ventricular rates, and bouts of sinus tachycardia.

CASE REPORT

A 50-year-old woman was referred to Ankara University, Department of Cardiology, Heart Center Hospital on July 2005, from another institution for an electrophysiologic study due to alternating cardiac arrhythmias and complaint of presyncope

of 2 months duration. The patient stated that she had these presyncopal episodes two or three times a day while she was walking in her house without any antecedent chest pain, dyspnea or palpitations. She described the feeling as "pumping of her heart", and suffered from dizziness, generalized weakness and constipation. She denied any chest pain, dyspnea, or orthopnea. She had no history of nephrolithiasis, hematuria, bone pain or fractures, osteoporosis and hypertension.

At admission, the patient was using aspirin and sertraline.She denied prior lithium and digoxine use or using any other illicit drugs. Family history was noncontributory. Physical examination findings included a heart rate of 45 bpm and blood pressure of 100/60 mmHg. The neck was supple, and the thyroid was not enlarged but a firm, round shaped mass was palpable at the left-upper portion of thyroid. All other system examinations were unremarkable.

Electrocardiography (ECG) showed sinus bradycardia with a rate of 55 bpm and QTc was 0.365 seconds (Figure 1). The 24-hour ambulatory holter monitoring showed episodes of sinus bradycardia, sinus pauses with slow ventricular rates and bouts of sinus tachycardia during day and night recordings (Figure 2). A two-dimensional (2D) echocardiography revealed normal systolic ejection fraction with no structural abnormalities. The laboratory examination showed elevated serum calcium level of 13.2 mg/dL (normal range 8.8-10.6 mg/dL) and elevated serum parathyroid hormone (PTH) level of 318 pg/mL (normal range 9.5-75 pg/mL). Corrected serum calcium level was 13.44 mg/dL (albumin level was 3.7 g/dL). Blood urea nitrogen, creatinine, potassium, magnesium levels were in normal range. Serum TSH, free T3 and T4 concentrations were in normal range. Cranial computed tomography revealed no pathology. Urinary system ultrasonography was normal. Thyroid ultrasonography revealed a 22 mm x 18 mm sized nodule consistent with parathyroid adenoma. Femoral and spine mineral densities were in normal range at bone mineral densitometry examination. Intravenous fluids and loop diuretic infusion were given during hospital stay. There was no need for temporary pacemaker implantation during follow-up. A sestamibi scan of the neck was suggestive of a parathyroid adenoma and the patient underwent parathyroidectomy. After the surgical operation for primary hyperparathyroidism, serum calcium level returned to normal range and patient's complaints and arrhytmic findings recovered. The patient was asymptomatic for the following 13 years.

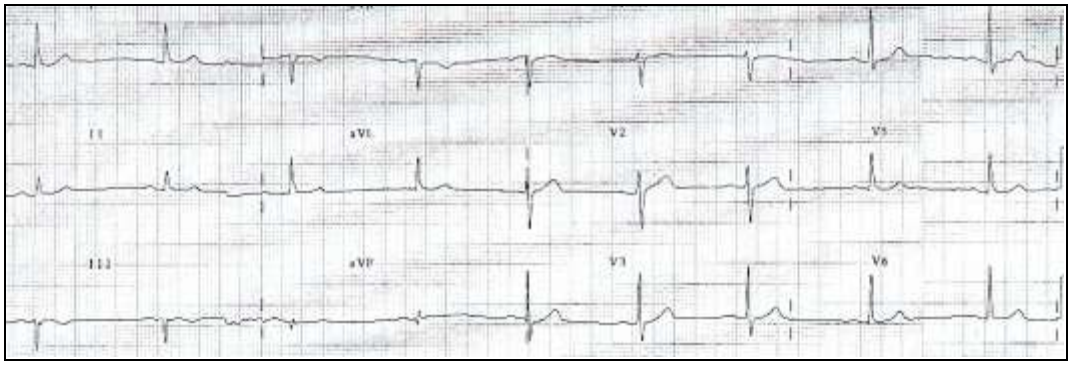

Figure 1: Sinus bradycardia (55 bpm) with QTc 0,365 sec.

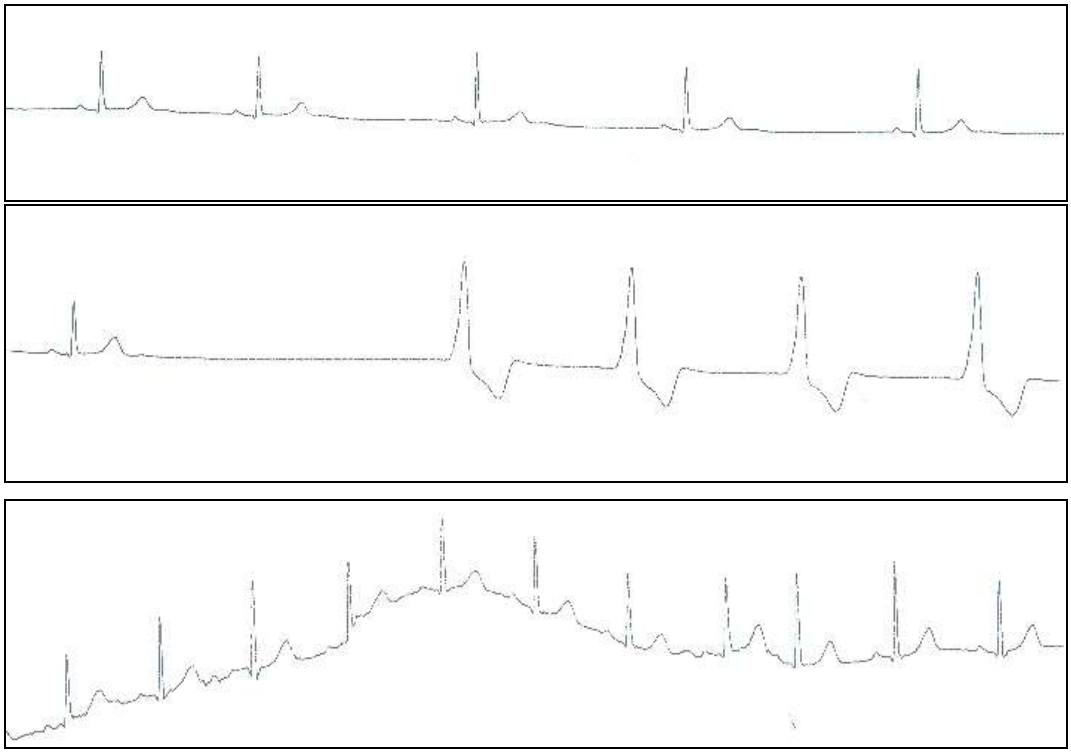

Figure 2: Sinus bradycardia with a rate of 34 bpm (top panel). Sinus pause with slow ventricular escapes (middle panel). Sinus tachycardia with a rate of 114 bpm (bottom panel).

DISCUSSION

The patient was diagnosed with sick sinus syndrome and referred to our hospital for an electrophysiologic study and further evaluation for pacemaker implantation.But the underlying pathology was hypercalcemia secondary to hyperparathyroidism manifested by elevated serum PTH levels. The biologic effect of PTH is to increase the mobilization of calcium into the extracelular fluid from other tissues. The elevation of hormone usually leads to hypercalcemia and hypophosphatemia. Solitary adenomas are responsible for approximately 80% of the cases of hyperparathyroidism, with hyperplasia and carcinoma occuring less frequently[8].

Disordered parathyroid hormone secretion is associated with cardiac arrhythmias and hypertension. Although most of the effects of PTH on the heart are due most commonly to hypercalcemia, the direct effect of PTH may be deleterious as current findings showed that administration of exogeneous PTH resulted in necrosis of rat myocytes[9]. Typical electrocardiographic changes of hypercalcemia, including hypercalcemia of hyperparathroidism include prolongation of the P-R interval, shortening of Q-T interval, and broad flattening of the apex of the T wave [1]. But the major change is the shortening of Q-T interval caused by the effect of high serum calcium concentration on the plateau of the action potential of cardiac fibers. Varing degrees of atrioventricular blocks may occur, but complete heart block is rarer[4-7]. A thorough literature research revealed only two cases that showed hyperparathyroidism causing sinus node dysfunction which required pacemaker implantation[6]. However, in our case pacemaker implantation was not necessary.

The anatomic basis of sick sinus syndrome includes total or subtotal destruction of the sinus node, areas of nodal- atrial discontinuity, and inflammatory or degenerative changes in the nerves and ganglia surrounding the node. However, in the setting of hypercalcemia, sick sinus syndrome might be caused by three mechanisms: First, hypercalcemia may cause calcification of sinus node leading to dysfunction. Second, hypercalcemia may have an adverse effect on the action potential of the SA nodal cells[6]. Third, as shown in an animal study, hypercalcemia may increase

vagal efferent activity and thereby cause sinus slowing which can be reversed by atropin[10].

In conclusion; this case report describes a rare presentation of sick sinus syndrome secondary to hyperparathyroidism-induced moderate hypercalcemia. Potential underlying etiologies include calcification of SA node, and adverse effect of increased vagal activity on the action potential of SA nodal cells. However, further studies are needed to clarify the issues on pathogenesis.

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