Case Report

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Recurrent Ventricular Tachycardia in Sheehan's Syndrome

Sheehan Sendromunda Tekrarlayıcı Ventriküler Taşikardi

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Abstract

Sheehan's syndrome is one of the common causes of hypopituitarism in developing countries. Electrocardiographic (ECG) abnormalities in Sheehan's syndrome are not well documented. However, in hypopituitarism due to other causes, ECG findings include low-voltage QRS complex, ST segment depression, T-wave inversion and prolonged QT interval. We hereby describe a 45-year-old female who presented with a history of recurrent syncope for last three years. Electrocardiography revealed ventricular tachycardia, which reverted back with hormone replacement therapy. Since it is a common problem in our community, clinicians should consider Sheehan's syndrome as an etiology of metabolic disturbances leading to ventricular tachycardia in women. *Turk Jem 2015; 19: 28-30*

Key words: Sheehan's syndrome, ventricular tachycardia, hypoglycemia

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Özet

Sheehan sendromu gelişmekte olan ülkelerdeki en sık hipopitüitarizm nedenlerinden birisidir. Sheehan sendromundaki elektrokardiyografik (EKG) anormallikler net bir şekilde gösterilmemiştir. Ancak, diğer nedenlere bağlı hipopitüitarizmde EKG bulgusu olarak düşük voltaj QRS kompleksi, ST depresyonu, T inversiyonu ve uzamış QT intervali saptanmıştır. Burada son 3 yılda tekrarlayıcı senkop öyküsü olan 45 yaşında kadın hasta sunulmaktadır. EKG de saptanan ventriküler taşikardi, hormon replasmanı ile düzelmiştir. Toplumda sık görülmesi nedeni ile klinisyenler, Sheehan sendromunu metabolik bozukluklara ve bunlara bağlı da ventirküler taşikardi gelişimine neden olan etiyolojik faktörlerden biri olarak değerlendirmelidir. *Turk Jem 2015; 19: 28-30*

Anahtar kelimeler: Sheehan sendromu, ventriküler taşikardi, post partum hemoraji

Çıkar Çatışması: Yazarlar bu makale ile ilgili olarak herhangi bir çıkar çatışması bildirmemiştir.

Introduction

Sheehan's syndrome (SS) is postpartum hypopituitarism, usually secondary to postpartum hemorrhage (PPH) and shock (1). This syndrome is rare in industrialized nations, but it is still a major threat to women's health in developing countries including India (2). SS presents with combined anterior pituitary hormone deficiency, although selective preservation of anterior pituitary function has been documented (3). SS presenting as ventricular tachycardia is rare with few reports in the literature (4,5). We here present a case of SS who presented to the emergency department with syncope. Electrocardiogram (ECG) revealed ventricular tachycardia, which did not recur after the patient was started on hormone replacement therapy.

Case Report

A 40-year-old female presented to the accident and emergency department of our hospital with sudden onset of dizziness and palpations for 6 hours. There was no history of vomiting, loose motion or any drug intake. On examination, the patient had a pulse of 100 beats/minute, and low-volume and irregular beats. Blood pressure was not recordable. General physical examination revealed features of hypothyroidism and absent axillary and public hair. In view of these findings, SS was suspected which is not rare in our region (2). She had delivered her last child 11 years ago at a local hospital. The delivery was complicated by PPH which required transfusion of at least 3 units of blood. After the last delivery, she did not lactate and did not resume menstrual cycles. The patient gave a history of similar complaints

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of sudden onset of dizziness and palpations for the last more than two years. Laboratory investigations revealed normocytic normochromic anemia, normal liver function tests (LFT), and kidney function tests (KFT). Random blood glucose was 62 mg/dL, serum sodium - 137 mEq/L, potassium - 2.4 mEq/L, and chest X-ray was normal. ECG revealed ventricular tachycardia (Figure 1). Echocardiography revealed ejection fraction (EF) of 54% and the rest was normal. Biochemical investigations revealed central hypothyroidism, secondary adrenal insufficiency, hypogonadism, and growth hormone and prolactin deficiency suggestive of panhypopituitarism Magnetic resonance imaging (MRI) of the pituitary revealed an empty sella (Figure 2). The patient was instituted 10% dextrose and hydrocortisone 100 mg intravenously. She was planned for cardioversion but she spontaneously reverted to normal sinus rhythm. The patient was then shifted to the medical intensive care unit (MICU) for observation, where 24-hour Holter monitoring did not reveal any rhythm disturbance. She was discharged on thyroxine 75 µg/day and prednisolone 7.5 mgs/day as a replacement therapy for anterior-pituitary hormone deficiencies. The patient is doing well with no history of syncope for more than two years of follow-up.

Discussion

SS is caused by massive PPH at the time of delivery which causes destruction of anterior pituitary cells (1). SS classically presents with a typical history of PPH, lactation failure, no menstrual periods or infrequent menstruation and signs/symptoms of hypothyroidism and adrenal insufficiency. In most cases, clinical picture unfolds slowly over a period of years and acute presentation is quite rare (6). Partial preservation of one or more anterior pituitary functions is also documented (3). Most of patients present to doctors with signs and symptoms of non-specific aches and pains, often attributed to anemia and come to medical attention only when they go to adrenal crisis or myxedema coma triggered by extreme stressors (7).

Cardiac abnormalities in patients with SS have rarely been described partly because of rarity of the disorder. Hypopituitarism resulting both in adrenal insufficiency and hypothyroidism has been reported to cause cardiac abnormalities which reverse with achievement of eumetabolic state (8). Electro cardiac abnormalities in SS are not well documented baring few scattered case reports (4,5). Hypoglycemia, hypokalemia and hypomagnesemia are common abnormalities in such patients (8). Our patient had blood glucose level of 62 mg/dL and his serum potassium level was 2.5 mEq/L. Regrettably, serum magnesium was not measured in our patient. Two mechanisms explain the arrhythmogenic potential of hypoglycemia: longer action potential duration and raised myoplasmic calcium ion concentration; both can convert quiescent cell into spontaneous action having abnormal pacemaker activity. Longer action potential fails to repolarize the ventricular cells and subsequently generate recurrent spontaneous action potentials. Such abnormal rhythm propagating to normal myocardium can result in ventricular tachycardia (9). Another pro arrhythmic mechanism during hypoglycemia is catecholamine surge. During hypoglycemia, the adrenal medulla secretes catecholamine which might convert already susceptible ventricular muscle into abnormal rhythms (10). Hypokalemia can also cause prolonged ventricular repolarization and pacemaker malfunction. The perpetuation of ventricular repolarization in hypokalemic setting is caused by inhibition of outward potassium currents. Aberrant pacemaker activity is credited by increased slope of diastolic depolarization in Purkinje fibers, as well as delayed afterdepolarizations caused by Ca2+ overload secondary to inhibition of Na+--K+ pump and stimulation of the reverse mode of the Na+--Ca2+ exchange (11). Adrenal insufficiency and hypothyroidism have been reported to result in ventricular tachycardia and, with replacement of deficient hormones, the timing of reversal has been reported to be immediate or delayed (12,13,14). The improvement or reversal of cardiac arrhythmias depends probably on whether predominant manifestation is hypocortisol state which reverses immediately or it is hypothyroid state where it takes many weeks. Immediate reversal is because of correction of hypocortisol state. Glucocorticoid replacement immediately corrects hypoglycemia and hypokalemia, both predisposing to ventricular arrhythmias. Our patient presented

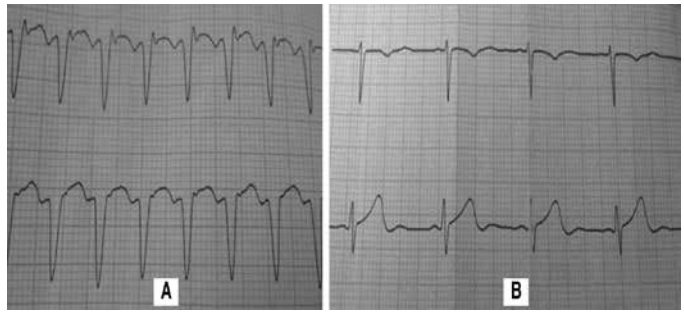

Figure 1. a) Electrocardiogram before treatment showing ventricular tachycardia; b) after treatment showing normal sinus rhythm

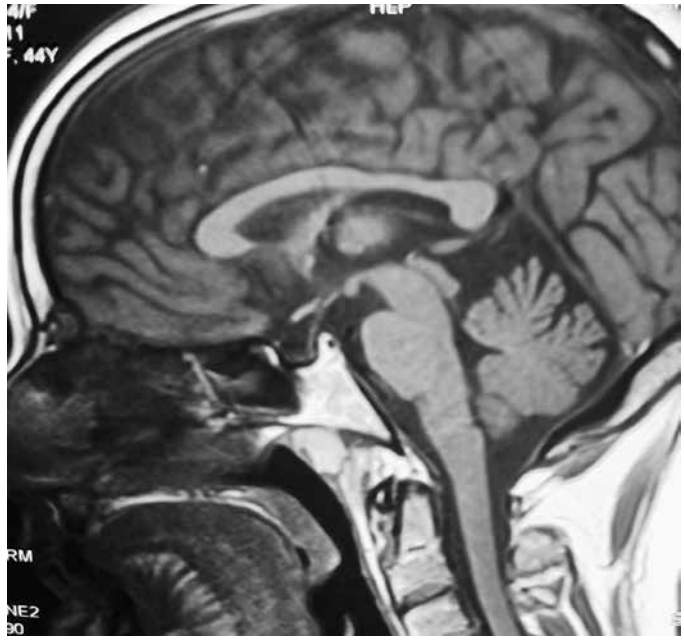

Figure 2. T1 weighted magnetic resonance image (sagittal view) showing pituitary gland filled with cerebrospinal fluid indicative of empty sella

with ventricular tachycardia which immediately got corrected with correction of hypocortisol state. Rhythm disturbances are reported both in patients with hypothyroidism and hypocortisol state. Wang et al. reported a young woman with SS who developed episodes of ventricular tachycardia which reversed immediately after instituting hydrocortisone (5). SS has multiple proarrythmic factors, including hypoglycemia, catecholamine surge and hypothyroidism. Therefore SS may be considered as one of the causes of such disturbances in areas where the disease is common.

Conclusion

Because SS is one of the common causes of hypopituitarism in developing countries, ECG abnormalities and their cause need to be studied. In view of seriousness of ventricular tachycardia, the clinicians should keep in mind that metabolic disturbances leading to ventricular tachycardia could be due to SS in areas where it is quite common. Moreover, we suggest that replacement therapy should be started as soon as possible because these electrocardiac abnormalities revert back without any cardiac intervention.

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