CASE REPORT
OLGU SUNUMU

Bradycardia following intake a single dose trazodone: case report

Tek doz trazodon kullanımı sonrası bradikardi: olgu sunumu

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SUMMARY

Trazodone is an antidepressent drug and it isn't get involved in other antidepressent drug such as tricyclic, tetracyclic agent. Trazodone is suggested to some event of insomnia–associated with antidepressent. When trazodone is taken over dose, cardiac side effect as conduction block, myocardial infarction, life-threatining arrythmia, bradycardia, hipotension and cardiac arrest can seem. Cardiac side events are uncommon and they occur dose-dependent. The patient who was taken trazodone, brought to emergency room following syncope by ambulance service. She has taken first dose of trazodone at last night. Sinüs bradycardia has established on her electrocardiography (ECG) and she hasn't any risk factor for bradycardia. In this case we aim of present a patient who had syncope that was induced by trazodone.

Keywords: bradycardia, sleep initiation and maintance disorder, trazodone

ÖZET

Trazodon trisiklik, tetrasiklik grubundan olmayan antidepresan bir ilaçtır. Trazodon, antidepresan ile ilişkili bazı uykusuzluk durumlarında önerilmektedir. Yüksek doz alındığında, iletim bloğu, miyokard enfarktüsü, hayatı tehdit eden aritmi, bradikardi, hipotansiyon ve kardiyak arrest gibi kardiyak yan etkiler ortaya çıkabilir. Kardiyak yan etkiler yaygın değildir ve doza bağımlıdır. Bazen doz aşımı olmadan da yan etkiler gözlenebilir. Bu vakada acil servise ambulansla senkop nedeniyle getirilen, etiyolojide ilk doz trazodon sonrası gelişen bradikardi ve senkop tespit edilen hastayı sunmayı amaçladık.

Anahtar kelimeler: bradikardi, trazodon, uyku bozukları

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INTRODUCTION

Trazodone is an antidepressent drug and it isn't get involved in other antidepressent drug such as tricyclic, tetracyclic agent. Particularly trazodone is recommended to some event that antidepressent –associated insomnia. If trazodone is taken over dose, cardiac side effect can seem. The side effect includes conduction block, myocardial infarction, life-threatining arrythmia, bradycardia, hipotension and cardiac arrest. But this events are uncommon and they occur dose-dependent. Sometimes the side effect can occur without over dose, especially trazodone. Some researches report of symptomatic bradycardia associated with single low dose of trazodone. In this case we aim of present a patient who had syncope that was induced by trazodone.

CASE

55-year old- woman was brought to emergency room following syncope by ambulance. The patient has complained chest pain, anxiety, dyspnea, insomnia for along time. She had been healty without systemic disease such as cardiovascular disease in past. In the recent days her complain's have increased and she had been examined by a cardiologist at the first. Although everything was normal about cardiyovascular system (ECG was normal and has sinus rhtym and normal rate), cardologist recommended to psychiatri clinic. Due to she has been examined by a psychiatrist and some recipe was given that include trazodone.

She took trazodone the first dose at last night. When she woke up, she felt sick. She lost consciousness and she was got brought to emergency department. Her was clear in emergency department. Glaskow Coma Scale was 15 and she was got monitorized. Her blood pressure 110/70 mmHg, blood sugar: 148 mg/dl, Sat O2: 99 %. There was sinus bradycardia on ECG recording and heart rate of 52 bpm, P-R and Q-T interval was normal. We ordered supportive treatment such as oxygen, intravascular salin infusion. The patient was kept under observation. Approximate a few hours later she felt better. Her heart rate increased and returned normal range. Trazodone treatment was stopped. We suggested to examine again her psychiatrist and change treatment.

The patient was informed about the case presentation without using the name and picture and the patient's consent was obtained.

DISCUSSION

Insomnia is the most common type of sleep disorder in all population (1). Insomnia is a subjective disorder and it manifests as prolonged sleep latency, sleep maintenance disorder, early awakenings, impaired total sleep time and decline in sleep quality; despite having an adequate opportunity to sleep, accompanied with distress and daytime dysfunction (2,3). Insomnia increases the risk of some illness such as hypertension, stroke, reduced body immunity and mental disorders (such as anxiety and depression) (4).

We use two main treatments for insomnia: psychotherapy and pharmacotherapy. Pharmacotherapy has the most important role in the treatment of insomnia. benzodiazepine drugs (BZDs) and nonbenzodiazepine compounds (non-BZDs) are often prescribed to treat insomnia. Antidepressants are often used the patient who is in depressed with insomnia (5,6). Trazodone, a second-generation triazolopyridine antidepressant, is well known as a sedative and hypnotic for its dose-dependent pharmacologic actions (7,8). It can block the serotonin transporter, inhibit the reuptake of 5-Hydroxytryptamine (5-HT), and function as an antidepressant. However, trazodone is more commonly prescribed as a hypnotic clinically for sleep disturbances including primary insomnia, and secondary insomnia, which often caused by co-morbid psychiatric disorders, physical diseases, or medication. Espacially (notably), the strongest indication of trazodone is insomnia with depression (8). A low dose of 25–150 mg is usually effective for treatment of sleep disturbances (2). Several case series and case reports have emerged suggesting electrophysiological abnormalities secondary to treatment with trazodone in both patients with premorbid cardiac co-morbidities and in those without it. These abnormalities ranged from prolonged PR interval, complete heart block, bradycardia, supra ventricular tachycardia, QT prolongation, ventricular tachycardia and torsades de pointes.

Trazodone is absorbed from the gastrointestinal tract and reaches peak plasma levels in about 1–2 h. It has a half-life of 5–9 h. The most common adverse effects associated with trazodone are sedation, orthostatic hypotension, dizziness, headache, and nausea. Because of its minimal anticholinergic effect, the incidence of cardiovascular side effects was less than that of other antidepressants. Some cardiovascular side effects which seemed to be dose-dependent have been reported in the scientific literature. However, reports of symptomatic bradycardia after trazodone

are rare. This case serves as a reminder to consider the possibility of developing bradycardia after a low dose of trazodone, even in young patients with no established cardiovascular disease. Patients at cardiac risk or elderly patients treated with trazodone are well advised to monitor the occurrence of cardiac side effects. Otherwise, when patients treated with trazodone complain of dizziness or chest discomfort at night, we should be aware of the possibility of bradycardia, in addition to the most common cardiovascular side effects with orthostatic hypotension.

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