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CASE REPORT

Solifenacin induced dyskinesia

Solifenazin'e bağlı diskinezi

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ABSTRACT

Overactive bladder syndrome is a chronic condition characterised by urgency, with or without associated urge incontinence. Solifenacin succinate is a once daily, bladder selective antimuscarinic available. The recommended dose is 5 mg once daily and can be increased to 10 mg once daily if 5 mg is well tolerated. We report here a case with dyskinesia emerged following the use of solifenacin, and disappeared in a few days with stopping the agent. *J Clin Exp Invest 2012; 3(4): 539-540*

Key words: Overactive bladder syndrome, solifenacin, dyskinesia

INTRODUCTION

Overactive bladder (OAB) syndrome is characterized by urinary frequency and urgency with or without urge incontinence, and often accompanied by nocturia. The prevalence of OAB increases with aging; it is a particularly common condition among the elderly, affecting at least 25% of people aged \geq 65 years.^{1,2}

Solifenacin is a competitive M3 receptor antagonist with a long half-life (45–68 hours). It is available in two dosage strengths namely a 5 or 10 mg once-daily tablet.^{3,4} A literature search about this drug revealed that the most common side effects are constipation, nausea, somnolence, abdominal pain, blurred vision, and urinary retention.

According to our knowledge, there was no data or article about solifenacin induced dyskinesia. This case report describes an unknown side effect of solifenacin in lower doses (5 mg).

CASE

84-year-old male applied to the Department of Neurology due to amnesia and urinary incontinence.

ÖZET

Aşırı aktif mesane inkontinans olsun ya da olmasın urgency ile ilişkili kronik bir durumdur. Solifenazin günde tek doz kullanılan selektif antimuskarinik bir ajandır. Günde 5 mg olarak başlanır, tolere edilebilirse 10 mg'a artırılabilir. Biz burada solifenazin kullanımı sonrası ortaya çıkan ve ilacın kesilmesini takiben birkaç gün içinde ortadan kaybolan diskinezili bir olguyu sunduk.

Anahtar kelimeler: Aşırı aktif mesane, solifenazin, diskinezi.

General condition was medium, patient was alert, cooperative, and orientated. There was no involuntary movement. Vital signs were stable and there were not any abnormalities in biochemical laboratory values. On head CT hydrocephalia seconder to parenchymal atrophy and periventriculer ischemic gliosis was seen. Patient was admitted to clinic for further examination in order to define the reason of amnesia. Patient was observed with a medical therapy, donapezil 5 mg/day. The patient discharged on fifth day with an arrangement in dosage of donapezil to 10mg, and solifenacin 5 mg / day was given due to his urinary incontinence. After a few days discharging from neurology clinic, patient applied to emergency service with a complaint of involuntary movements in the arms and legs. Patient relatives told that they observed involuntary movements a few hours after discharging from hospital and intension and frequency of these movements gradually increased. On physical examination in the ED, general condition was medium, vital signs biochemical values were normal. On neurological examination dyskinesia was seen more significant in lower limbs as myoclonus. On his medical history newly started drug solifenacin was reported. The patient was admitted again to neurology clinic from

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emergency service. Then thought to be a possible drug-related dyskinesia and the drug stopped. After quitting medication involuntary movements gradually decreased and completely disappeared in the second day.

DISCUSSION

We are presenting this case to describe solifenacin induced dyskinesia which was not reported on literature before. Solifenacin 5 and 10 mg once daily were efficacious and well tolerated in the treatment of these elderly subjects with OAB.¹ In our case adverse effect –dyskinesia- observed in lower doses.

Acetylcholinesterase inhibitors (e.g., donepezil, galantamine, physostigmine, rivastigmine, tacrine) may antagonize the effects of anticholinergic agents and other agents that rely partially on their anticholinergic activity for therapeutic effects (e.g., some antiparkinsonian and antiemetic/antivertigo agents; class IA antiarrhythmics).5 By inhibiting the metabolism of acetylcholine, more of the neurotransmitter may be available to compete at muscarinic receptors, the site of action of anticholinergic agents. Conversely, anticholinergic agents may negate the already small pharmacologic benefits of acetylcholinesterase inhibitors in the treatment of dementia. These agents may also adversely affect elderly patients in general. Clinically significant mental status changes associated with anticholinergic agents can range from mild cognitive impairment to delirium, and patients with Alzheimer's disease and other dementia are especially sensitive. In our case there were no sign about solifenacin worsening the effects on cognitive functions of donepezil. It may be due to use of solifenacin for a very short time.

Although seizures have been reported following abrupt discontinuation of anticholinergics during acetylcholinesterase inhibitor therapy,⁶ there were no sign without disappearing of involuntary movements in our case. It may also be due to discontinuation the drug after using for a short time.

This side effect that began after drug intake disappeared in a few days with stopping the agent. Because of this relation between drug and dyskinesia, it is thougt to be a side effect due to solifenacin so this is the first case reported, dyskinesia induced by solifenacin. In conclusion; dyskinesia may occur due to the use of solifenacin, and this side effect may disappeared in a few days with stopping the agent. Clinicians should be alert to the very side effects by solifenacin.

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