



Paroxetine Induced Akathisia: Necessity for Rational Use

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Received: 2018-10-12, Revised: 2018-11-04, Accepted: 2018-11-08, Published: 2018-12-01

ARTICLE INFO

Article type:

Case Report

Keywords:

Paroxetine;

Akathisia;

Selective Serotonin Reuptake Inhibitor

ABSTRACT

Rare cases of akathisia have been reported with paroxetine in the literature. However, accurate diagnosis for early treatment is important since untreated akathisia can result in poor patient's compliance, treatment failure, aggressive or suicidal acts.

We present a case of a 25-year-old female patient, suffering from akathisia, who was treated with paroxetine and olanzapine due to hypomania and general anxiety disorder. Our finding shows that side effects like akathisia could be addressed by the timing of the peak level of paroxetine. Thus, shifting the time of paroxetine consumption to reach its peak level at the sleeping period could be a rational approach for many patients. More research is required to confirm that the observation of this case report was not accidental.

J Pharm Care 2018; 6(3-4): 79-80.

► Please cite this paper as:

Mousavi M, Ghaeli P, Fahimi F. Paroxetine Induced Akathisia: Necessity for Rational Use. J Pharm Care 2018; 6(3-4): 79-80.

Introduction

Paroxetine, a selective serotonin reuptake inhibitor (SSRI), is widely used in the treatment of many psychiatric disorders. This medication has FDA approval for major depressive disorder (MDD), panic disorder, social anxiety disorder, posttraumatic stress disorder (PTSD), obsessive-compulsive disorder (OCD) and premenstrual dysphoric disorder (PMDD). The antimuscarinic activity of paroxetine that differentiates it from other SSRIs is responsible for reduced gastrointestinal side effects of this medication. However, this medication is considered to be more sedating than other SSRIs (1).

Although, rare cases of akathisia have been reported with paroxetine, accurate diagnosis for early treatment is important due to the fact that untreated akathisia can result in poor patient's compliance, treatment failure, aggressive or suicidal acts (2).

We present a case of a 25-year-old female patient suffering from akathisia, while taking paroxetine and olanzapine for hypomania and general anxiety disorder.

The clinical pharmacist changed the timing of paroxetine dose to the evening. The patient symptoms were improved by this intervention. No dose adjustment, discontinuation of causative agent, β blocker or anticholinergic antiparkinsonian drug administration was required.

Case presentation

A 25-year-old female patient presented with a history of anxiety and severe agitation for 5 years. She has not been able to continue her social activity effectively since 5 years prior to this last psychiatric visit.

She was diagnosed with hypomania and general anxiety disorder for which she was treated with different psychotropic medications. However, no drug history was available. According to the patient, none of those medication has been effective. In 2017, she was put on paroxetine 20 mg and olanzapine 2.5 mg, each taken one pill at night for over 1 month and showed significant improvement in her symptoms.

With no known reasonable reason like sleep disturbances,

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the administration of paroxetine was changed from evening to the morning by her physician. Possibly to prevent insomnia that might be induced by paroxetine. During this period, she experienced restlessness in both feet during the day most of the time after taking paroxetine in the morning. She had to move her feet restlessly in the sitting position.

The patient was extensively examined for the accurate diagnosis. No other extrapyramidal symptoms were detected on examination. The Barnes Akathisia Scale[®] (3) confirmed moderate akathisia (score = 8).

Fortunately, the akathisia completely resolved when the morning administration of paroxetine was shifted to the afternoon. The Barnes Akathisia Scale[®] was zero after resolving of akathisia.

Discussion

A common type of extrapyramidal syndrome (EPS) is akathisia that is defined as a side effect consisting of personal feeling of restlessness with or without restless movements, generally in the legs or feet. Due to similarities between symptoms of agitation and anxiety with those of akathisia, this side effect can be misdiagnosed. Thus, it is important to get a precise medication history when patient is complaining of restlessness.

It is hypothesized that akathisia induced by paroxetine or other medication in SSRI antidepressant class is mediated via augmenting serotonin that inhibited dopamine cells in the ventral tegmental area (VTA) (4).

The present case report identifies akathisia induced by paroxetine that could be resolved by a simple intervention that was changing of the time of the administration from am to pm.

The diagnosis of akathisia was supported by symptoms and scored on the Barnes Akathisia Scale. Of note, akathisia rates were significantly reduced with paroxetine, despite continues use when its time of administration was changed from morning to evening. One explanation for this effect is that movement related side effects may be related to the peak concentration of the medication which can be minimized when the pick time is shifted to the time when the patient is lying down. The improvement could be corresponding to the peak level that occurs during sleep time.

Paroxetine undergoes a nonlinear metabolism, steady-state occurs within 8 days, mean terminal half-life of 18 hours. The pharmacokinetics support a once-daily dosing administration. The metabolism of paroxetine is largely dependent on the activity of CYP2D6. It should be noted that peak concentration of paroxetine occurs in 5-6 hours (1) and the patient experienced akathisia at the time that was

compatible with pick time.

Although in the presented case, her only other medication, olanzapine, is often caused akathisia, a rapid onset of symptoms and remission happened following paroxetine time change. Score with Naranjo scale (5) was 7 which shows a probable causality relationship. On the other hand, olanzapine, like other neuroleptics, induced akathisia accompanied by the other extrapyramidal symptoms, as mentioned before no other extrapyramidal symptoms were detected on examination.

Previous case reports of akathisia with paroxetine showed improvement within one week by discontinuation of paroxetine or starting prescriptions of propranolol and benzodiazepines (6-9).

In our presented case, akathisia relief was observed after 24 hours of paroxetine usage in the evening. During follow up, this patient was not complaining of insomnia induced by the paroxetine usage at night. Olanzapine is associated with somnolence and could help with the insomnia related to other medication. On the other hand, anticholinergic effect of paroxetine makes it more sedating compared with other SSRI's.

Our finding shows that side effects like akathisia could be addressed by the timing of the peak level of paroxetine. Thus shifting the peak time to the sleeping period could be a rational approach for many patients. More research is required to prove that the observation of this case report was not accidental.

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