

Case Report

Sertraline induced Psychosis

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Introduction

Antidepressants currently account for the largest proportion of psychopharmacologic drug prescribed in all psychiatric settings. Selective serotonin reuptake inhibitor (SSRIs) are the most commonly prescribed anti-depressants. Due to the widespread clinical use of SSRIs, pharmacologists have discovered that these drugs have actions at receptors other than the serotonin transporter.¹ Sertraline possess moderate affinity to block dopamine reuptake pump (dopamine transporter), which could increase dopamine.² This dopamine reuptake inhibitor property is highest among all SSRIS. According to Stahl¹ in his Essential Psychopharmacology series, sertraline could be considered a combined serotonin and dopamine reuptake inhibitor. Clinical significance of this increase in dopamine level by sertraline is under investigation. Four cases of sertraline associated psychosis have been reported till date.³ We are reporting a case of sertraline induced psychosis.

Case history

Mrs. A., 33-year-old Hindu married female presented to Psychiatry outpatient department (OPD) with complaints of anxiety, restlessness, worry, muscle tension, irritability, easy fatigability, palpitation, excessive sweating, shakiness of hands for last 2 years. There was no history of any substance use. There was no contributory past history or family history. Premorbidly she was fairly well adjusted. Her physical examination was within normal limits. No depressive symptoms, obsessions, delusions or perceptual disturbances were found in the mental status examination (MSE) and her cognitive functions were intact. Investigations including complete blood count, blood sugar, and thyroid functions were within normal

limits. On the basis of history and mental status examination (MSE) diagnosis of generalized anxiety disorder (F41.1) as per International Classification of Diseases-10th Edition criteria⁴ was made. She was treated on outpatient department (OPD) basis and was started on sertraline 50mg/day which was increased to 100 mg/day after 5 days along with clonazepam 1 mg/ day. On the next follow up after one month there was no significant reduction in her symptoms, so she was hospitalized. Her dose of sertraline was increased to 200 mg/day. After 15 days of treatment with 200 mg/day of sertraline, there was gradual improvement in her symptoms. But the patient developed additional symptoms like suspiciousness towards nearby patient in the ward, hearing voices of her relatives threatening to kill her. On mental status examination delusion of persecution and auditory hallucination of threatening variety were elicited. Due to this atypical presentation the diagnosis of the patient was again reviewed and it was confirmed to be generalized anxiety disorder. Dose of sertraline was decreased to 100mg/ day and olanzapine 5 mg/day was added. After 5days,her delusion and auditory hallucinations gradually increased. Then sertraline was stopped and dose of olanzapine was increased to 10mg/ day. After 15 days her delusion and auditory hallucinations resolved completely. She was again started on sertraline 100 mg/ day and dose of olanzapine was decreased to 5 mg/day. But after 7 days she again developed previous delusion and hallucinations. So sertraline was stopped, olanzapine was increased to 10 mg/day and escitalopram 10mg/ day was started. After one month her psychotic symptoms resolved completely and her previous anxiety symptoms also improved gradually. Olanzapine was gradually stopped over

next 15 days. She was discharged from the hospital with escitalopram 10 mg/day and clonazepam 1mg/day. On her next follow up visit after 1 month there were no psychotic symptoms and her anxiety symptoms were improved significantly. She is maintained well on 10 mg/day of escitalopram for last six months. During no time of her illness any manic or hypomanic symptom were reported or observed.

Discussion

In our patient the strength of association between sertraline use and emergence of psychotic symptoms is strengthened by factors such as the absence of a past history, family history or substance use, the fast remission of psychotic symptoms with a short course of antipsychotic and reemergence of symptoms on reintroduction of sertraline. Moreover, the patient did not develop any psychotic symptoms after sertraline was stopped, and she was maintained on escitalopram during the last six months. So in this case, a definite causal link between sertraline use and psychotic symptoms can be established by Naranjo adverse drug reaction probability criteria.⁵ In previous case reports³ of sertraline induced psychosis Naranjo adverse drug reaction probability criteria was not satisfied.

Previously antidepressants including the SNRI (serotonin and norepinephrine reuptake inhibitor), tricyclics and MAO inhibitors have been implicated in inducing or exacerbating psychosis in other psychiatric illnesses.⁶⁻⁸ Among SSRIs fluoxetine is associated psychosis in two published reports.^{9,10} In dopamine reuptake inhibition, sertraline is more potent compared to a number of common antidepressants (e.g. bupropion, venlafaxine, nefazodone, paroxetine, norfluoxetine, nortriptyline, desipramine). This dopamine reuptake inhibition might be the possible reason for psychosis in our patient. In case of venlafaxine dopamine reuptake inhibiting action is described in high doses (225-300 mg/day).¹ But no literature is available regarding relationship between dose of sertraline and its dopamine reuptake inhibiting action. Patients with factors for vulnerability to psychosis like past history of psychosis, family history of psychosis, substance abuse may be more susceptible to

emergence of psychosis with sertraline treatment. As the use of antidepressants continues to increase, vigilance is warranted in prescribing sertraline to individuals who may be susceptible to this form of behavioral change.

References

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