CASE REPORT

A Case Report on Multiple Episodes of Deliberate Self-harm in a Patient with Amisulpride-associated Akathisia and Tardive Dyskinesia

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Abstract

Tardive dyskinesia is a movement disorder that causes involuntary, repetitive body movements and is commonly seen in patients who are on long-term treatment with antipsychotic medications. It occurs in 20–50% of patients taking APDs. Akathisia is a neuropsychiatric syndrome characterized by subjective and objective psychomotor restlessness whose severity can extend to self-harm and suicide.

Case description: Our index case, a 57 years old, Hindu married female, of lower socio-economic status, diagnosed as a case of schizophrenia for 2 years, and well maintained on Tab. Amisulpride 600 mg daily, came in July 2022, with complaints of pain and upper abdominal discomfort and distension of abdomen for which she was prescribed Levosulpiride 50 mg daily. On next visit, in October 2022, she was having dyskinetic movements of the lips and rhythmic protrusion of the tongue, along with rhythmic movement of the whole abdomen. Eventually, she was diagnosed as a case of Levosulpiride-induced Tardive Dyskinesia and Levosulpiride was stopped. Tetrabenazine 50 mg daily was started. Subsequently, she became very restless and complained of uneasiness of whole body. Due to severe uneasiness and restlessness, she attempted suicide three times in presence of her family members and thus was prevented. In March 2023, she was having dyskinetic movement of oral cavity and rhythmic involuntary movement of the abdomen and was restless and irritable. She was continuing Amisulpride 600 mg and Tetrabenazine 50 mg daily. But unfortunately, there was no symptomatic improvement. NARANJO assessment score was applied which showed probable drug reaction. Then, it was diagnosed as a case of Amisulpride-induced Tardive Dyskinesia with Akathisia. Amisulpride was also stopped and no antipsychotics were given. For Akathisia, Lorazepam 4 mg in divided doses and Propranolol 40 mg was added to Tetrabenazine 75 mg daily. Now, patient started improving, having no fresh complaints although presently not on antipsychotics.

Keywords: Akathesia, Amisulpride, Case report, Deliberate self-harm, Tardive dyskinesia.

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Introduction

Tardive dyskinesia is the movement disorder commonly seen in patients who are on long-term treatment with antipsychotic medications. It causes involuntary, repetitive body movements of the tongue, jaw and/or extremities which occurs in around 20–50% of patients taking antipsychotic drugs, but can vary with advanced age.[1,2] Akathisia constitutes a neuropsychiatric syndrome marked by both subjective and objective psychomotor restlessness that can progress into self-inflicted harm and suicidal attempts in severe cases.[3,4]

Case Description

Our index case, a 57 years old, Hindu married female, belonging to lower socio-economic status who was diagnosed as a case of schizophrenia since 2 years, and well maintained on Tab. Amisulpride 600 mg daily, came to our OPD, in July 2022, with complaints of abdominal pain and upper abdominal discomfort along with distension of abdomen. She was then prescribed Levosulpiride 50 mg daily. On her next visit, in October 2022, she was having dyskinetic movements of the lips and rhythmic protrusion of the tongue, along with rhythmic movement of the whole abdomen. Eventually, she was suspected as a case of Tardive Dyskinesia triggered upon by Levosulpiride and thus, Levosulpiride was stopped. Tetrabenazine 50 mg daily was started.

Subsequently, she became very restless and complained of uneasiness of whole body most of the time. Due to severe uneasiness and restlessness, she attempted suicide three times in the presence of her family members and thus was prevented. In March 2023, she was having dyskinetic movement of oral cavity and rhythmic involuntary movement of the abdomen and was restless and irritable. She was continuing with Amisulpride 600 mg and Tetrabenazine 50 mg daily during that time. But unfortunately, there was no symptomatic improvement. NARANJO assessment

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Table 1: As per NARANJO's drug reaction probability scale assessment[5]

Question	*Yes*	*No*	*Do not know*	*Score*
1. Are there previous *conclusive* reports on this reaction?	+1	0	0	1
2. Did the adverse event appear after the suspected drug was administered?	+2	−1	0	2
3. Did the adverse reaction improve when the drug was discontinued or a *specific* antagonist was administered?	+1	0	0	1
4. Did the adverse event reappear when the drug was re-administered?	+2	−1	0	0
5. Are there alternative causes (other than the drug) that could on their own have caused the reaction?	−1	+2	0	2
6. Did the reaction reappear when a placebo was given?	−1	+1	0	0
7. Was the drug detected in blood (or other fluids) in concentrations known to be toxic?	+1	0	0	0
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	+1	0	0	0
9. Did the patient have a similar reaction to the same or similar drugs in *any* previous exposure?	+1	0	0	0
10. Was the adverse event confirmed by any objective evidence?	+1	0	0	0
Total score				6

Table 2: NARANJO algorithm – ADR probability scale[5]

Total score	*Interpretation of scores*	
≥9	Definite	Clearly caused by the exposure
5–8	Probable	Likely to be caused by the exposure
1–4	Possible	May be related to the exposure
≤0	Doubtful	Clearly not related to the exposure

score was applied which showed a score of 6; implying probable drug reaction. Then, it was diagnosed as a case of Amisulpride associated tardive dyskinesia with Akathisia. Amisulpride was also stopped and no antipsychotics were given. For Akathisia, Lorazepam 4 mg in divided doses and Propranolol 40 mg was added and Tetrabenazine dose was increased to 75 mg daily. Now, patient started improving, having no fresh complaints although presently not on antipsychotics.

Results

The relevant data can be obtained by referencing Tables 1 and 2 provided here.

For this case, the total score is 6, which indicated probable drug reaction.

Final diagnosis is Amisulpride-associated tardive dyskinesia and Akathisia.

Discussion

Recent studies have revealed that atypical antipsychotic medications do not always result in akathisia, with composite rates ranging from 2.9 to 13.0%, even though they have been found to have lower risks of extra pyramidal side effects (EPS) than standard antipsychotic medications.[6]

The common mechanism of akathisia, according to Ferré et al., is thought to involve an increase in presynaptic transmission of dopamine in the ventral striatum and concurrent activation of the D1 receptor, which is found in the ventral GABAergic striatonigral neurons and forms heteromers with the D3 and adenosine A1 receptors.[7] Akathisia has also been linked to increased central norepinephrine activity and hyperserotonergic neurotransmission in addition to ventral striatal release of dopamine.[8,9]

According to Davis et al., the second-generation agent (SGA) amisulpride, a substituted benzamine, is an effective treatment for schizophrenia.[10] Preferentially blocking presynaptic dopamine autoreceptors at low doses, it blocks postsynaptic dopamine receptors at large doses in a manner akin to first-generation antipsychotics (FSAs).[11] Comparatively to the striatal regions, amisulpride shows a preferential affinity for dopamine D2/D3 receptors in the limbic regions. It has been suggested that amisulpride in particular exhibits unusual features, such as therapeutic benefit without motor side effects, due to the D2 receptors' quick dissociation from it.[12] According to Leucht et al., it has a dose-dependent EPS-causing potential that is comparable to that of conventional antipsychotics, olanzapine, and quetiapine.[13] Likely in our case, patient was on higher dose of amisulpride which makes the patient vulnerable for the EPS.

The levo-enantiomer molecule of sulpiride is called levosulpiride. It is a benzamide replacement that is indicated for use in treating a number of conditions, including dyspepsia, psychosis, somatoform disorders, nausea, and depression. In India and other parts of the world, fixed dose combination (FDC) products of levosulpiride with proton-pump inhibitors (PPIs) are being prescribed on a long-term basis for a variety of gastrointestinal diseases.[14] Levosulpiride inhibits D3 and D2 receptors that are present in the rat striatum or nucleus accumbens both pre- and postsynaptically. At low doses, the preferential binding of the presynaptic dopamine receptors reduces dopamine synthesis and release, whereas at higher doses, it results in antagonistic postsynaptic D2 receptors.[10] Levosulpiride frequently results in movement disorders brought on by medication, with the most common symptoms being levosulpiride-induced parkinsonism (LIP) and lower face dyskinesia.[15] And as our patient was on tab 600 amisulpride which makes her vulnerable and addition of Levosulpiride produced the EPS. Drug-induced dyskinesia is characterized by either orofacial dyskinesia or limb and trunk movements (antipsychotics/D2 antagonists). Orofacial dyskinesia causes specific movements in the mouth and face, such as protruding or twisting the tongue, smacking and pursuing the lips, puffing out the cheeks, chewing with the jaw, and grimacing with the face, while the limb and trunk movements include purposeless, jerky, choreiform movements, gait disturbances, lordosis, athetosis of the extremities, limb and axial dystonia, shoulder shrugging, and rotatory movements of the pelvis, which includes the presenting features of our patient being the lip-smacking movement, puffing of cheeks, and purposeless abdominal movements.[16]

Conclusion

Atypical antipsychotics, which are known to have a favorable side-effect profile including drug-associated movement disorder, are increasingly used because typical antipsychotics are frequently linked to drug-associated movement disorders.

In conclusion, we have presented a case of severe akathisia brought on by long-term amisulpride dose, which was successfully treated with Tetrabenazine in combination with propranolol and lorazepam. The current instance highlighted the possibility of akathisia and tardive dyskinesia after long-term amisulpride dose use. As a result, while taking amisulpride and other antipsychotic medications over an extended period of time, careful monitoring for akathisia and EPS is advised. Newer generations of atypical antipsychotics do, however, cause undesirable side effects, as in the case reported here.

As the severity of the side effects is as high as with deliberate attempts of self-harm, it is very much necessary to keep a close eye and follow up the patients regularly.

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