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TRIFLUOPERAZINE INDUCED DYSTONIA-A RARE CASE REPORT

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ABSTRACT

Dystonia is a disorder characterized by involuntary muscle contractions that cause slow repetitive movements or abnormal postures which are painful. Drug-induced dystonic movements are caused by blockade of dopamine D2 receptors which are strongly associated with the efficacy of antipsychotics. Here we present a case of dystonia developed due to the use of typical antipsychotics. The first approach for treating acute dystonic reaction is reduction of the dosage or withdrawal of antipsychotics. Treatment strategies may also include switching from a First Generation Antipsychotics to a Second Generation Antipsychotics. Anticholinergic agents, dopamine agonists and benzodiazepines may often reduce the intensity of the acute dystonic reaction.

KEY WORDS: Dystonia, Antipsychotics, Anticholinergics.

INTRODUCTION

Dystonia is a disorder characterized by involuntary muscle contractions that cause slow repetitive movements or abnormal postures which are painful.[1] Dystonia is a symptom rather than a specific disease. It can be classified according to bodily distribution as focal, segmental, multifocal, hemi-dystonia or generalized. The etiological classification of dystonias are: primary (including genetic and idiopathic forms), secondary. The third form of classification relates to age of onset: child hood (early onset), adolescence and adulthood (late

onset).[2,4] Dystonias develop due to rapid increase in the dose of the antipsychotic medication, or of reducing the medication used to treat acute extrapyramidal symptoms (EPS). The prevalence varies widely from 2 % to 90 %.[3]

The differential rates of dystonia can be explained by the receptor blocking ratio between dopamine and acetylcholine in the basal ganglia. The higher the ratio of dopamine-acetylcholine antagonism, higher the risk of acute dystonia. When a patient is treated with high potency antipsychotic drugs, a low starting dose is recommended because this reduces the risk of acute dystonia compared with a standard dose. Atypical antipsychotic drugs such as olanzapine, sertindole, quetiapine, and a low dose of risperidone (<4 mg) are associated with a low incidence of acute dystonia while Clozapine is the only known atypical antipsychotic drug that does not induce acute dystonia but the mechanism is unclear. Anticholinergic properties may reduce the risk of acute dystonia .The balance between serotonin and dopamine blockade also play a role in reducing risk of dystonia. Olanzapine also reduces the risk of dystonia.[3]

Drug-induced dystonic movements are caused by blockade of dopamine, D2 receptors which are strongly associated with the efficacy of antipsychotics and is at least partially responsible for the movement disorders like those in acute dystonia. As First Generation Antipsychotics exert their therapeutic action primarily by blocking D2 receptors in the central nervous system have a high risk of inducing such side effects. While Second Generation Antipsychotics are effective against psychosis and at therapeutic doses rarely cause EPS including acute dystonic reaction.[5]

CASE REPORT

A 37 year old female was admitted with complaints of dystonia in a neuropsychiatric hospital. She was a case of unspecified psychosis with symptoms of fearfulness, auditory hallucinations and mood changes on Trifluoperazine 10 mg, Trihexyphenidyl 2 mg at the age of 36. After 1 month of therapy the dose of drug was titrated to 15 mg Trifluoperazine, Trihexyphenidyl 4 mg as the patient was unresponsive to the treatment. The patient developed involuntary movements within 4 months of therapy. Since they were not aware that the disorder was due to the drug she has been taking, therapy was continued. Within 6 months, the patient was presented with significant worsening of dystonic symptoms associated with chorea and elevated serum creatinine phosphokinase. The patient's symptoms

were kept stable with combination therapy that included Clonazepam (6 mg a day), Biperiden (6 mg a day) and Baclofen (60 mg a day).

DISCUSSION

Dystonia appears within 4 days of initiation of treatment with antipsychotic drugs or after a large increase in the dose in 95% of cases. Though it may appear in all muscle groups it is observed mainly in the head and neck area.[3] Antipsychotic-induced dystonia is a subtype of tardive dyskinesia.[8]

Here the patient developed lip puckering, persistent torticollis, pain, axial dystonia and unstable gait after one year of treatment with Trifluoperazine. The drug used for the treatment belongs to first generation antipsychotics which has high potential to cause dystonia.[3] Enough literatures are not available to support the case but literatures strongly provide evidence that typical antipsychotics causes dystonia. Since no other drugs except trifluoperazine was used to treat the patient health care provider came to conclusion that this is trifluoperazine induced dystonia. In this case, the patient's family members failed to recognize the importance of the dystonic symptoms, so it took long time before her dystonic symptoms were first diagnosed and treated.

Lehan et al, proposed First Generation Antipsychotics are associated with the greatest complications. The gradual increase in the use of Second Generation Antipsychotics in Japan, reduced the incidence of antipsychotic-induced acute dystonic reaction.[5]

The first step in treatment of dyskinesia is to change the current antipsychotic agent to another atypical antipsychotic agents like Clozapine, Quetiapine, Aripiprazole and if the dystonic symptoms are severe, high dose anticholinergic agents should be considered as it would deplete the excess of presynaptic dopamine; but this approach should not be followed if it is the symptoms of tardive dyskinesia as anticholinergics can worsen these indications. There has been a report of Tardive Dyskinesia successfully treated by Tetrabenazine.[8] Devendra Mishra et al., says that there are literatures favoring the use of intravenous agents for deep sedation, done with increasing doses of intravenous infusion of midazolam (30-100 µg/kg/hour), to which propofol (0.5-2.0 mg/kg/hr) may be added. The intense muscle activity of the patients leads to the metabolic complications such as rhabdomyolysis gradually results in renal failure. Bulbar and respiratory complications may occur which require tracheal intubation, hyper-pyrexia, muscle exhaustion, pain and dehydration. Therefore patients should be routinely managed in intensive care settings. Intrathecal baclofen is also

recommended, although the literature does not provide clear cut evidence for its efficacy. Second-line approaches, for those with progressive disorders, involve electrical deep brain stimulation of globus pallidus internus or bilateral pallidotomy. Clonazepam has also been used for Satus Dystonia management. There are also reports about the efficacy of benzodiazepines such as diazepam, clonazepam or lorazepam. For Tardive Dyskinesia with only localized or mild symptoms, botulinum toxin A can be considered.[7]

CONCLUSION

Dystonia is a common adverse effect of antipsychotic medication which disturbs the daily activities of the patient. The early recognition and adoption of treatment strategies accordingly may help in hasty recovery of the patient. Therefore the use of second generation antipsychotics are recommended along with close monitoring to avoid the complications. It is also advised to bring awareness among the family members about the adverse effects of the medicines taken by the patients to prevent all medical complications.

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