

Journal of Biological Science

IMIPRAMINE INDUCED EXTRAPYRAMIDAL SYMPTOMS

Dr Amitabh Saha MD, Dr Bikram Duttta MD, Dr SK Saini MD, MD, Dr Amit Kumar, MD

Abstract

Extra pyramidal szymptoms are common side effets een with antipsychotics , however its association with tricyclics is rare but seen in clinical practice. A patient with Moderate depressive episode was managed with tricycyclic antidepressants. Patient developed acute dystonia on the third day and there were no additional medications he was prescribed. Tricyclic antidepressant (TCA)-related extra pyramidal side effects have been reported but these side effects are not well known. The prevalence and reporting of these side effects is low, despite widespread use of these medications. In some cases, it has also been seen to disappear even though the same dose of TCA is continued, and they do not seem to be a drug class reaction. It is also possible that

Keywords: tricyclic antidepressants, acute dystonia, Extrapyramidal symptoms, antiparkinsonian agents, propranolol, akathisia

Introduction

Extra pyramidal szymptoms are common side effets een with antipsychotics, however its association with tricyclics is rare but seen in clinical practice. Extrapyramidal symptoms (EPS), also known as extrapyramidal side effects (EPSE), are drug induced movement disorders include acute and symptoms that tardive symptoms. These include dystonia (continuous spasms muscle contractions), akathisia(motor and restlessness), parkinsonism (characteristic symptoms such as rigidity, bradykinesia, and tremor), and tardive dyskinesia(irregular, jerky movements).^[2] Medications are often discontinued due to inefficacy or intolerable side effects such as extrapyramidal symptoms.^[3]

Tricyclic antidepressants are commonly used drugs for depression. Even though the SSRIs have been more routinely prescribed in current times, the use of Tricyclic antidepressants continues as efficacy wise it is comparable to any other class of antidepressants. TCA induced EPS is not seen commonly and hence is never a limiting factor in its use ^[1].

Since it is difficult to measure extrapyramidal symptoms, rating scales are commonly used to assess the severity of movement disorders. The Simpson-Angus Scale (SAS), Barnes Akathisia Rating Scale (BARS), Abnormal Involuntary Movement Scale (AIMS), and Extrapyramidal Symptom Rating Scale (ESRS) are rating scales frequently used for such assessment and are not weighted for diagnostic purposes. ^[4]

EPS would include the following <u>Acute dystonic reactions</u>: muscular spasms of neck, jaw, back, extremities, eyes, throat, and tongue; highest risk in young men ^[5].<u>Akathisia</u>: A feeling of internal motor restlessness that can present as tension, nervousness, or anxiety ^[6].drug-induced <u>parkinsonism</u> (rigidity, bradykinesia, tremor, masked facies, shuffling gait, stooped posture, sialorrhoea, and seborrhoea; greater risk in the elderly). ^[6]. <u>Tardive dyskinesia</u>: involuntary muscle movements in the lower face and distal extremities; this is a chronic condition associated with long term use of antipsychotics



1

Journal of Biological Science

A review of the medical literature reveals a substantial number of cases with similar clinical characteristics associated with the tricyclic antidepressants, monoamine oxidase inhibitors, and selective serotonin reuptake inhibitors (SSRIs). Although the data are not sufficient to make definitive pharmacoepidemiologic conclusions, the available number of case reports suggests the SSRIs may be more common offenders in producing these adverse drug effects than the TCA. The exact mechanism is elusive but likely involves complex interactions of dopamine, serotonin, and norepinephrine between cortical structures and the basal ganglia. The final common pathway for production of EPS seems to be the indirect modulation of dopaminergic function.

Predictors of patients at risk for antidepressant-induced EPS are not established, but a greater awareness of the potential for these drug side effects to occur may increase their recognition and decrease antidepressant-induced morbidity

Case Report

A 31-year-old male presented with features of remaining aloof, sad, having prominent guilt ideas and entertaining thoughts of causing self-harm for 3 month duration. The symptoms had developed insidiously when his wife learnt about his extramarital affair with a known relative. After his wife left him with their two kids, the feelings of low mood became more intense and he started to entertain suicidal thoughts. He had reduced sleep and was unable to concentrate at work. He was a clerk by trade and had difficulty in coping up with the pressures of the job entasked to him. His mood started to worsen and he had frequent awakenings in his sleep. He felt tired and would miss his family more so when he would return home to find it empty.

He was noted to be remaining increasingly aloof and withdrawn with poor self hygiene. He was noticed by his senior colleagues to be self absorbed and matter was brought to the notice of the superiors who after interviewing suggested psychiatric evaluation. He was admitted as psychiatric inpatient to a tertiary care hospital

Mental state evaluation did reveal depressed affect with prominent depressive cognitions, reduced biodrives, pessimism and negative ruminations. He was subjected to routine investigations and psychometric tests. BDI revealed scores of 18, Rorschach test revealed impulsivity, poor ego strength, no paranoid traits but low productivity. Personality inventory showed him to be irritable, with impulsive traits, and a cyclothymic mood.

He was started on tab Imipramine 25 mg HS, which after few days was increased to 50 mg per day. On the 6 th day he was noted to have acute dystonic movements involving the face, upper limb with acute discomfort and pain at having to maintain an awkward posture. He showed abnormal facial grimacing and jaw movements. He was given inj Phenargan 25 mg IM stat with which after 20 minutes the dystonic movements subsided and he felt relief. He denied consuming any other medications. His prescribed medications were stopped and he was seen drug free for a few days with no evidence of



Journal of Biological Science

recurrence of dystonia at any stage. He was started on Tab Venlafaxine 37.5 mg HS which was very gradually built up to 75 mg HS. He was given benefit of Tab Lorazepam 2 mg BD and subjected to regular counseling sessions.

After 5 weeks patients sleep and appetite gradually showed improvement and there was no evidence of pessimistic thoughts and he had no prominent depressive cognitons. He felt optimistic about life and looked for means to do well at job front and enter into possible couple therapy with his spouse who also agreed.

Discussion

As seen in this case, the symptoms were markedly present on the background of a presumptive stress. The case was all the more interesting for the rarity of the condition with which he presented later while he was an inpatient. The diagnosis is always determined after ruling out organic components ^[6] or other psychogenic diagnosis. ^[6] Thus, the diagnostic process consisted of precise medical history, thorough physical examination and serial mental state examinations. It is also noteworthy to reveal that individual developed Tricyclic antidepressant induced acute dystonia (6) which is known in literatures to be a probable side effect of the antidepressant given when he was not on any other offending psychotropic drug.

Hence it is note worthy that treating psychiatrist be aware of TCA induced EPS and take cautionary steps thereafter to mitigate patient distress.

References

- 1. Madhusoodanan S, Alexeenko L, Sanders R, Brenner R. Extrapyramidal symptoms associated with antidepressants—A review of the literature and an analysis of spontaneous reports. Annals of Clinical Psychiatry 2010;22(3):148-156
- 2. Foster AR, Lancaster NP. Disturbance of motor function during treatment with imipramine. Br Med J.1959;2:1452-1453
- 3. Dekret JJ, Maany I, Ramsey TA, et al. A case of oral dyskinesia Psychiatry. associated with imipramine treatment. Am J 1977;134:1297-1298.

 \uparrow

- 4. Sandyk R. Persistent akathisia associated with early dyskinesia. Postgrad Med J. 1984;60:916.10. Koller WC, Musa MN. Amitriptyline-induced Neurology. abnormal movements. 1985;35:1086.
- 5. Sedivec V, Valenová Z, Paceltová L. Persistent extra pyramidal oral dyskinesias following treatment with thymoleptics. Act Nerv Super (Praha). 1970;12:67-68.
- 6. Zubenko GS, Cohen BM, Lipinski JF Jr. Antidepressant-related akathisia. J Clin Psychopharmacol



Journal of Biological Science

Principal author

Dr Amitabh Saha MD , Psychiatry Assistant Professor Dept of Psychiatry Armed Forces Medical College Pune India E mail-sahaing@gmail.com

