EXPLORING THE RELATIONSHIP BETWEEN DRUG-INDUCED PARKINSONISM AND TARDIVE DYSKINESIAS

L. Giselle Aguilar, MD and William G Ondo, MD

Parkinson's Disease Center and Movement Disorders Clinic, Department of Neurology, Baylor College of Medicine, Houston, Texas

INTRODUCTION

Drug-induced parkinsonism (DIP) tends to have an insidious onset, often starting many months after treatment with dopamine receptor blocking drugs, while tardive dyskinesia (TD) may present concurrently with DIP or manifest as withdrawal emergent dyskinesia. Older patients and those of female gender tend to be more vulnerable to developing DIP and TD.

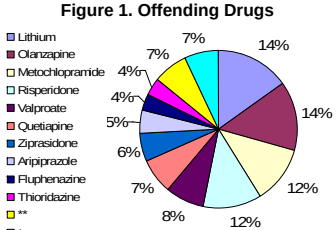
METHODS

We reviewed the medical records of 55 patients with initial diagnosis of DIP seen in our clinic who were subsequently revaluated in follow up.

RESULTS

The mean age at presentation was 63.2 years ± 12.8, and the mean duration of exposure to a potentially offending drug was 71.4 ± 101.5 months (range 2-276 months). The most frequent offending drugs were lithium, olanzapine, metoclopramide, risperidone and valproate (Figure 1). 21 patients were on more than one drug at the time of initial evaluation, and 26 patients had history of previous exposure to a neuroleptic, such as risperidone, haloperidol and thioridazine.

Figure 1. Offending Drugs



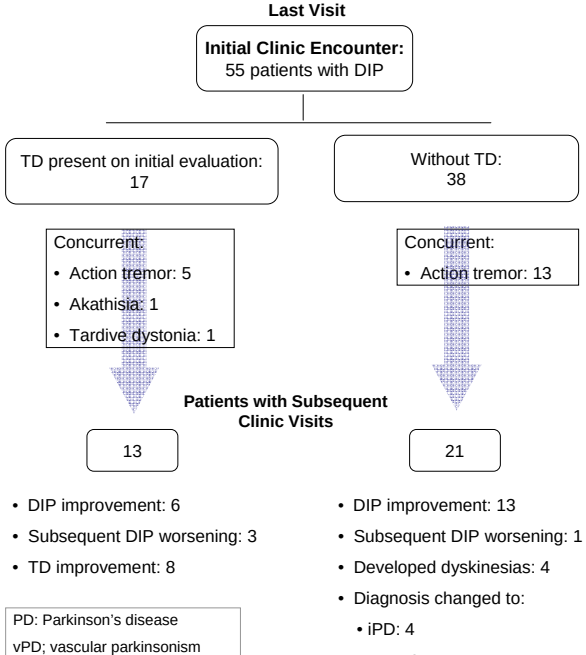
* 2.33% each: perphenazine, prochlorperazine, promethazine

** 1.67% each: thioridazine,loxapine, amoxapine, fluoxetine,trifluoperazine, estrogen receptor antagonist

The most frequent diagnoses for treatment with a potentially offending agent were bipolar disorder in 18 (32.7%), gastrointestinal symptoms in 11 (20%), depression in 10 (18.2%), and schizophrenia and psychosis in 8 (14.6%) patients. Three patients were under treatment for more than one diagnosis (e.g. GI symptoms and depression).

Concurrent with DIP, 17/55 patients had TD, 18/55 had tremor, and one each had tardive dystonia and tardive akathisia (figure 2).

Figure 2. Algorithm of Patient Diagnosis from Initial to Last Visit



PD: Parkinson's disease

vPD; vascular parkinsonism

34 patients returned for follow up, of which 4 had developed dyskinesia in the interval. Mean follow up at time of diagnosis of TD was 6.89 months with interval improvement of parkinsonian features. Of all patients seen in follow up, diagnosis of DIP was changed in 6 patients: to PD in 4 (12.12%), one of whom was subsequently diagnosed with levodopa-induced dyskinesias; and to vascular parkinsonism in 2 (6.06%).

Of the 13 patients who initially presented with tardive dyskinesia and DIP, none were later diagnosed with PD. Parkinsonism improvement was documented in 19 of the DIP patients, with subsequent worsening in four (tetrabenazine-related in 3 and paliperidone in 1).

CONCLUSIONS

DIP tends to improve once the offending agent, most frequently a neuroleptic agent, is discontinued. The presence of TD concurrent with DIP does not indicate poor prognosis and may suggest that these patients will not develop idiopathic PD.

REFERENCES

1. Jankovic J and Casabona J. Coexistent tardive dyksinesia and parkinsonism. Clin Neuropharmacol. 1987;10:511-21
2. Dean CE et al. Predictors of neuroleptic-induced dyskinesia and parkinsonism: the influence of measurement methods and definitions. J Clin Psychopharmacol. 2006;26:560-5
3. Kenney C et al. Metoclopramide, an Increasingly Recognized Cause of Tardive Dyskinesia. J Clin Pharmacol. 2008;48:379-84
4. Hadad PM and Dursun SM. Neurological complictions of psychiatric drugs: clinical features and management. Hum Psychopharmacol Clin. 2008;23:15-26