

*Case report*

# Neuroleptics and family history of Parkinson disease: case report

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## Introduction

We observed a 20-year-old female patient who presented with symptoms suggestive of psychosis and who developed severe extrapyramidal side-effects (EPSE) on low doses of atypical antipsychotic medications. The patient was found to have a strong family history of Parkinson disease.

We wonder whether this observation has any clinical significance for either our patient or others with psychosis who have a family history of Parkinson disease and concurrent administration of neuroleptics. There are many reports of EPSE with antipsychotics as well as reports of EPSE occurring more often in those susceptible to or having organic brain disease. Could a family history of Parkinson disease be a predictor for severe EPSE for a patient taking atypical antipsychotic medication, and as a consequence, a predictor for developing Parkinson disease?

## Observations

A 20-year-old Caucasian female presented to the Adult Psychiatric Outpatient Clinic with delusions regarding her bodily appearance and with depressive symptoms. She was diagnosed with psychotic depression and advised to commence fluoxetine hydrochloride 20 mg once daily and risperi-

done 1 mg twice a day on the first day increasing to 2 mg twice a day on the second day, to continue at 2 mg thereafter.

Two days later she presented to the Casualty Department with an acute dystonic reaction involving the head and neck muscles. Intramuscular procyclidine was administered with swift resolution of the adverse event. She was advised to stop the risperidone and seek another appointment at the psychiatric clinic. During the outpatient appointment she was advised to commence amisulpride 200 mg twice a day and procyclidine 5 mg as and when required. The fluoxetine hydrochloride prescription remained unchanged.

Unfortunately several weeks later, our patient's mental state necessitated hospital admission. On admission, she was noted to be severely parkinsonian with an expressionless face, tremor and cogwheel rigidity despite procyclidine 5 mg twice a day. The amisulpride was stopped. The fluoxetine hydrochloride, now at 40 mg once daily, was continued and over the next few days our patient was free of EPSE. Quetiapine was commenced slowly, building up to a dose of 200 mg twice a day. Fortunately she did not appear to experience EPSE. Symptomatic hypotension associated with the quetiapine, however, was a problem and thus limited us to using a maximum of 200 mg twice a day. Electroencephalogram

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and brain magnetic resonance imaging were unremarkable.

During her admission it was evident that the most likely diagnosis now was schizophrenia as there were delusions of thought interference, passivity phenomena, other bizarre delusions and marked negative symptoms. Clozapine therapy was considered but the patient was reluctant to switch to another neuroleptic.

It was during her admission that we discovered that both the patient's maternal grandmother and uncle had died in their fifties as a result of Parkinson disease.

## Discussion

There are many reports of EPSE with antipsychotic medication as well as frequent occurrence of EPSE among those susceptible to or having organic brain disease. For example, risk factors for tardive dyskinesia (TD) according to primary psychiatric diagnosis have been studied [1]. It was found that schizophrenic subjects constituted the largest absolute number of TD patients, although they were the least likely to develop TD in comparison with those with affective disorders or organic mental disorders [1]. In another study, 35% of the parkinsonian patients also had TD [2]. It was found that overt brain damage was not a significant predictor of an abnormal involuntary movements score (AIMS) and the difference between the neuroleptic-medicated and the neuroleptic-free groups on the AIMS scores was highly significant [2].

Quetiapine as an alternative to clozapine in the treatment of dopamimetic psychosis in patients with Parkinson disease has been compared through case histories and has been found to be a viable alternative to clozapine for these patients [3].

Risperidone treatment of drug-related psychosis in parkinsonism was found to be useful in the treatment of hallucinosis in a series of 39 patients with parkinsonism [4]. In the same study, 23 patients with Parkinson disease had either complete or near complete resolution of hallucinosis whereas an unsatisfactory response (6 patients) or worsening of parkinsonism (6 patients) was noted in 12 patients, only 6 of whom had Parkinson disease.

There have been reports of worsening of motor features of parkinsonism in patients receiving olanzapine [5]. The worsening was considered to be dramatic in 6 of 12 patients treated with olanzapine for drug-induced psychosis occurring in Parkinson disease.

In our case, the patient has not yet developed Parkinson disease, but appears to be susceptible to developing EPSE. An issue was raised during treatment with antipsychotics or selective serotonin-reuptake inhibitors as to whether a long preclinical or asymptomatic period may occur in Parkinson disease as well as the existence of many long-latency parkinsonian syndromes. This suggestion is supported by a similar observation of a preclinical asymptomatic period before the full onset of Parkinson disease [6]. In that report, it was concluded that individual sensitivity to drug-induced parkinsonian syndromes suggested a preclinical state.

We must point out, however, that selective serotonin-reuptake inhibitors are a known cause of EPSE lasting up to 6 weeks [7]. However, in our patient, on withdrawal of the atypical antipsychotic medication, EPSE ceased despite continuing fluoxetine treatment.

We are continuing follow-up assessments and quetiapine treatment. Our patient may possibly benefit from clozapine; however, she is reluctant to switch at present.

## Conclusion

In view of the limited research in this area, we hope that this brief report will stimulate

further discussion on the topic as well as encourage others to report similar observations.

## References

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