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Letter to the editor

Tardive dyskinesia following brief exposure to risperidone—a case study

Keywords: Risperidone; Tardive dyskinesia; Antipsychotics

1. Introduction

Risperidone is a second generation antipsychotic with a low incidence of tardive dyskinesia (TD) compared to conventional antipsychotics [1,2]. Cases of unequivocal risperidone-induced TD have been described after long-term exposure [3] or, in one case, after only 16 weeks [4]. We report a case of TD restricted to the extremities in a neuroleptic naive patient after less than 4 weeks of exposure to oral risperidone.

2. Case report

A previously well 19-year-old white male presented with a 4 week history of psychosis (subsequently diagnosed as paranoid schizophrenia) manifested by an abrupt onset of persecutory delusions, beliefs that he could read peoples' thoughts at a distance, wanting to communicate a dead man's thoughts, circumstantiality, sudden vocalizations of "dirty–dirty–dirty" and auditory hallucinations. There was a history of marijuana use over many years and consistent intake (three times per week) over the previous 3 years. There was a past history of occasional amphetamine use but none for 6 months. He was a light cigarette smoker (three cigarettes per day). There was no family history of neurological disorder. Physical examination and neurological assessment were normal. Brain CT showed mild ventricular asymmetry with the right ventricle more prominent than the left. Toxic screen was negative for amphetamines, benzodiazepines, cocaine, opiates and PCP. He was given a single dose of risperidone 2 mg with lorazepam 4 mg and diphenhydramine 50 mg; thereafter, he received only risperidone (1.5 mg increasing to 2.5 mg/day). Twenty-six days after starting risperidone, he displayed intermittent flexion and extension movements of the ankles while sitting. The movements persisted over the ensuing days. Nineteen days later, piano-playing movements of the hands in addition to the foot movements were observed. The parents also noted the movements, which were enhanced by anxiety. At this point, the psychosis had resolved. Risperidone was discontinued and replaced with olanzapine 5 mg bid. When seen 16 days later, no movement disorder was detected.

3. Discussion

The definition of TD includes an onset after 3 months of starting neuroleptics. This is, however, arbitrary [5]. An accepted qualifier for the diagnosis of TD applicable in the present case is "with less than 3 months' neuroleptic exposure" [5]. Disappearance of TD may have resulted from an antidyskinetic effect of olanzapine [6] or a masking effect.

Involuntary movements have been described in patients with schizophrenia before antipsychotic exposure [7] but none was noted by the family or when first examined. The movements observed are commonly seen in TD. Risk factors such as older age, female gender, diabetes mellitus, affective disorder, preceding Parkinsonism [7] or heavy nicotine exposure [8] were not present. Certain cannabinoids are known to affect dopaminergic function [9] so that it is possible this was a factor contributing to the unusual sensitivity for the development of TD in our patient. TD following brief exposure (4 weeks) to conventional antipsychotics has previously been reported [10]. The present report indicates that TD may also result from brief exposure to risperidone.

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