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RISPERIDONE INDUCED TARDIVE DYSKINESIA - A CASE REPORT

O.V. VASUDEVAN, DENZIL A. PINTO & P.S. GOPINATH

ABSTRACT

Risperidone is a serotonin - dopamine antagonist which has got less propensity to cause tardive dyskinesia than conventional antipsychotics. There have been few reports of tardive dyskinesia induced by risperidone. This is a report of a case of risperidone induced tardive dyskinesia. A 56 year old female with a 6 months history of paranoid schizophrenia, developed bucco-oro-masticatory abnormal involuntary movements after receiving risperidone 8 mg/day for about 1 year. She had some of the risk factors for the development of tardive dyskinesia like age, sex, anti-cholinergic drugs and earlier emergence of neuroleptic-induced parkinsonism. Clinicians must be aware of the possibility of the patients developing tardive dyskinesia when they are given the supposedly safe neuroleptic risperidone.

Key words: Tardive dyskinesia, risperidone, atypical antipsychotics.

Tardive dyskinesia (TD) is a well recognized adverse effect associated with the long-term use of neuroleptic therapy, characterized by involuntary, choreiform, athetoid or rhythmic movements of the tongue jaw and / or extremities. Because it can be disfiguring, persistent and even permanent, it has been a major limiting factor in the use of antipsychotic medications (Daniel et al, 2000). The overall prevalence of neuroleptic - induced TD in individuals who have received long term neuroleptic treatment ranges from 20% - 30% (APA, 1994). The most significant and consistently documented risk factors for the development of TD is increasing age of the patient (Jeste & Wyatt, 1982;Kane & Smith, 1982). Women have been found to be at a greater risk for severe TD, especially the geriatric populations (Kennedy et al., 1971). It was hoped that the emerging generation of putative atypical antipsychotics such as, risperidone, olanzapine, sertindole, quetiapine and ziprasidone will have a reduced liability to produce TD. Herein, we report a case of TD induced by risperidone.

CASE REPORT

This patient, 56 year old female, was brought with 6 months duration of bizarre delusions of persecution, thought withdrawal and broadcast, somatic hallucination and second and third person auditory hallucinations, with no evidence of any substance abuse or organic pathology and no contributory past or family histories and the patient never received treatment with any neuroleptics prior to this. The patient was admitted and a diagnosis of paranoid schizophrenia was made (DCR-10) (WHO, 1993). She was started on risperidone, from 1 mg/day initially and at the time of discharge in 2 weeks, the dosage was increased to 8 mg/day. During the course of her stay in the hospital, she

developed neuroleptic - induced Parkinsonism and for which, 2 mg/day of Trihexypenidyl (THP) was added. In about 11 months of time, her delusions more or less disappeared,but occasional auditory hallucinations were still persisting. So, risperidone was reduced to 6 mg/day along with 2 mg/day of THP. After about 1 ½ years, there was no active psychopathology, so risperidone was further reduced to 4 mg/day with 1 mg/day of THP. During her next follow up, after a month, we noticed a typical choreiform and athetoid bucco-oro-masticatory movements in her, characterized by pouting, puckering and smacking of lips and chewing movements of the lower jaw. There were no abnormal movements in the tongue, trunk or extremities. The patient was not aware of and did not present subjective complaints of the abnormal movements. Her son reported that these movements were transiently reduced by voluntary movements of the jaw and absent during sleep. So, risperidone was stopped and olanzapine 7.5 mg/day was started. During the next visit, after a month, there was no change in the movements and the patient is continuing on olanzapine.

DISCUSSION

Risperidone is a benzisoxizole derivative, the second serotonin-dopamine antagonist, after clozapine, to be introduced in India. The incidence of TD in patient treated with risperidone for atleast 1 year has been estimated at around 0.3% (Brecher, 1996; Ayd & Pies,1997).

In our patient, she had both the significant and consistently documented risk factors for the development of TD - increasing age (>55 years) and female sex. Several investigators suggested that, one risk factor for developing TD is earlier emergence of other extrapyramidal symptoms (Owens, 1994). This patient had neuroleptic - induced Parkinsonism during admission. There is a hypothesis that anticholinergic medications also contribute to the development of TD (Kane, 1995). Our patient was treated with THP after she developed neuroleptic - induced Parkinsonism. However, there is no clear evidence for this hypothesis.

There have been isolated case reports of patients developing TD, when they were treated with a dosage of risperidone, 6 mg/day or higher. Unlike previous, this one clearly demonstrates the cause-and-effect relationship of risperidone and TD in a patient whose clinical course was well documented. But a single case can only serve to stimulate interest in further study. We want the clinicians to be aware of the possibility of patients developing TD when they are given the supposedly safe neuroleptic risperidone.

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O.V. VASUDEVAN, M.B.B.S. ,Junior Resident, DENZIL A.PINTO, M.D., Assistant Professor, P.S.GOPINATH, M.D., Emeritus Professor, Department of Psychiatry, Fr. Muller's Medical College, Mangalore - 575 002*

Correspondence