RISPERIDONE - INDUCED TARDIVE DYSKINESIA : CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

Risperidone is an atypical antipsychotic with broad spectrum of antipsychotic activity and lower potential for extrapyramidal side effects at therapeutic doses. This case report illustrates the development of tardive dyskinesia with therapeutic dose of risperidone in a paranoid schizophrenic patient who was not on any antipsychotic medication previously.

Key words: Risperidone, tardive dyskinesia, extrapyramidal side effects

Risperidone, a recently approved antipsychotic drug, is a benzisoxizole derivative that displays potent central antagonism of serotonin (especially 5HT₂) as well as dopamine (D_s) receptors. Because of its broad spectrum of pharmacologic properties, particularly its 5HT, antagonism, risperidone has been regarded as an atypical antipsychotic that shares with clozapine the potential for superior antipsychotic efficacy and reduced capacity for causing extrapyramidal side effects including tardive dyskinesia (Marder and Meibach, 1994; Jeste et al., 1999). There are reports that risperidone even supresses abnormal movements in patients with tardive dyskinesia (Chouinard, 1995). However there are isolated cases of tardive dyskinesia reported with risperidone treatment (Addington et al., 1995; Woener et al., 1996; Gwinn and Caviness, 1997; Silberbauer, 1989; Saran, 1998). A recent case report (Carroll et al., 1999). described an adolescent taking risperidone, 6 mg/day who had mild hand-dangling movements and tongue protrusion; these subsided with discontinuation of risperidone. Evidence from over 1100 patients, 503 of whom had taken risperidone for at least 1 year, suggest that the annual incidence of tardive dyskinesia in patients taking risperidone (7.6-9.4 mg/day) is 0.3%,

compared to an annual incidence of 5-10% in patients taking conventional neuroleptics (Gutierrez-Esteinou & Grebb, 1997). In view of the interesting nature of the subject, we describe a patient who develop extrapyramidal side effects that were followed by dyskinetic movements in association with risperidone treatment.

Mr. G, was a 48 year old, married, Christian male, with a 10-year history of persecutory delusions, referential delusion, delusion of infidelity and grandiose delusion of ability. Because of these problems, he had taken voluntary retirement from military service 5 years ago, but was not willing to consult a psychiatrist. Two years earlier, he had been forcefully admitted to our hospital with exacerbation of paranoid schizophrenia. There was no significant past or family history of psychiatric disturbances. Physical examination and routine investigations were within normal limits. He was a chain smoker and used to consume alcohol occasionally. During inpatient stay he had received trifluperazine (maximum dose, 20 mg/day) and trihexiphenidyl (4 mg/day). After discharge he was irregular on medication and relapsed. At this time, he was prescribed liquid risperidone, the dose of which was gradually increased to a maximum of 6 mg/ day over a period of 2 weeks. One week later, he

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came with severe drug induced parkinsonism for which trihexiphenidyl 4 mg/day was added. Within few days the pseudoparkinsonism subsided and trihexiphenidyl was stopped. Since then he was receiving only liquid risperidone and was maintained well with regular follow up once in every 2 months.

After about 8 months, he was noticed to have dyskinetic movements of both hands and a to-and-fro nodding movement of head which was prominent when he was lying on the bed. These movements were absent during sieep. Due to dyskinesia, the risperidone dose was reduced to 2 mg/day and vitamin E (400 mg/day) was added to the regimen. During the last 4 months, he continues to have the same type of dyskinetic movements without progression but the psychotic symptoms are well controlled.

Tardive dyskinesia is not a common side effect associated with risperidone treatment (Glazer, 2000). Along with many risk factors, a history of early extrapyramidal symptoms (EPS) represents a possible risk factor for the later development of tardive dyskinesia (Owens, 1994). As this patient has received risperidone as his main antipsychotic and as there was no dyskinetic movements in the past, the dyskinesia can be definitely considered as risperidoneinduced tardive dyskinesia. A pharmacological explanation could be that at a dose of 6 mg/day risperidone may lose the balanced 5HT_a/D_a blocking effect, resulting in more affinity for D, receptors, thereby producing comparable EPS with classical antipsychotics. As the same dose continues for long time, it can lead to supersensitivity of D, receptors in the nigrostriatal system producing tardive dyskinesia. Jeste et al. (1996) have reported that Asian patients require lower doses of most psychotropic indications than do Caucasian patients which may explain why certain patients in India respond to risperidone at doses that are as low as 1 mg/day and react unfavourably to the therapeutic dose range of 6-8 mg/day.

A single case we report is of heuristic value only. However, we believe that this case is unusually clear because of its antipsychotic drug

naive status, complete prospective medication data, systematic tardive dyskinesia monitoring, 8 months persistent dyskinesia, and dyskinesia free status preceding risperidone treatment. We are currently enrolling patients who are beginning treatment with risperidone to further examine the issue of tardive dyskinesia.

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