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Case Report Open Access

# A Case of Lewy Body Dementia with Sensitivity to Risperidone Treated Successfully with Amisulpride

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A 78 year old retired businessman presented to our OPD with complains of behavioral abnormalities for past 1 year. He became irritable over trivial matters which was not the case before. Patient had fluctuating worsening of intellectual ability and executive functioning for past 6 months. He claimed to be seeing intruders in the house and left the house unannounced on two occasions. Patient developed resting tremor and slowness of movement for past 3 months. There were no other comorbidities in the patient. Mental State Examination revealed fearful affect, visual hallucinations. Neurological findings were cog wheel rigidity in upper limbs, unsteady gait and resting tremors. Mini Mental State Examination revealed a score of 16/30. Blood investigations were all within normal limit. A diagnosis of Lewy Body Dementia was made as per Revised criteria for the clinical diagnosis of probable and possible dementia with Lewy bodies (DLB) (2017) [1]. He was started on Risperidone 0.5 mg twice a day. Within a day patient became rigid, mute and was not responsive to commands. He was given intramuscular promethazine 1 ampoule. The rigidity reduced and patient became responsive. He was then started on Amisulpride 50 mg twice a day. Over next 4 days the hallucinations reduced. He was then placed on combination of Levodopa and Carbidopa (100 mg + 25 mg) twice a day. Parkinsonism features reduced after a week without worsening of psychosis and the patient was discharged on the same medications with addition of tablet Donepezil 11.5 mg.

There have been case reports of neuroleptic sensitivity to Risperidone in Lewy Body Dementia [2,3]. The pathophysiological mechanisms underlying neuroleptic sensitivity" in LBD seem to be a 60-70% reduction in dopaminergic neurons in the substantia nigra, together with a failure to upregulate post-synaptic dopamine receptors in the striatum either in response to the dopaminergic deficit or to D2 blocking drugs. Risperidone has high D2 receptor antagonism' and thus might be expected to provoke neuroleptic sensitivity reactions in LBD; the modifying effect of its 5HT receptor antagonism remains uncertain [4]. Amisulpride has less propensity to cause extrapyramidal symptoms among the second generation antipsychotics [5] so we used this in our case. Though it has been used for controlling behavioral symptoms in Alzheimer's Dementia [6], its use in the context of Lewy Body Dementia is rare. As well as in our case despite the patient having sensitivity to risperidone, the patient improved drastically to amisulpride. Again, more action on 5HT2A receptors compared to D2 receptors of amisulpride may explain the same [5]. Still the exact mechanism is still under scrutiny.

Thus, this is one of rare scenarios where amisulpride has been effective in a case of Lewy Body Dementia where patient had sensitivity to another agent of same class. Use of amisulpride in such cases should be encouraged.

**Ethical Concerns**: Due consent was taken from the patient regarding publication of the findings without revealing name.

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