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Original Research Paper

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A RARE CASE REPORT OF AMISULPRIDE INDUCED URINARY RETENTION

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ABSTRACT Amisulpride is an atypical antipsychotic with the preferential action on D2/D3 receptors. Its common adverse effects are extrapyramidal symptoms, insomnia, hyperkinesia, anxiety, weight gain, agitation, hyperprolactinemia. We have witnessed a adverse effect of urinary retention induced by amisulpride at minimal dosage and would like to present the same.

KEYWORDS : Urinary retention, Amisulpride

INTRODUCTION

Amisulpride is an atypical antipsychotic, however it lacks 5HT2A antagonism, atypicality may be due to its preferential action on limbic postsynaptic D2/D3 receptors at the low dose and preferential blockage of presynaptic D2/D3 receptors at high dose. Adverse effects are extrapyramidal symptoms, insomnia, hyperkinesia, anxiety, weight gain, agitation, hyperprolactinemia(1,2). Here, we have encountered a rare side effect of urinary retention associated with amisulpride therapy. There are case reports of urinary incontinence associated with amisulpride. However, only two case reports of urinary retention induced by amisulpride were available as per our search and would like to report the same.

CASE REPORT

Mrs k, 56 years old, hindu, married, belonging to middle socioeconomic status, graduate, housewife, diagnosed as paranoid schizophrenia with total duration of illness for 32 years. Patient had failed adequate trials with olanzapine, risperidone and quetiapine, so patient was treated as a case of treatment resistant schizophrenia and started on clozapine. Patient developed myoclonic jerks with 100 mg of clozapine so clozapine trial was stopped , later the patient developed seizure with 50mg of clozapine on retrial. So patient was started on low dose of paliperidone and uptritrated to 9mg. Initially patient was maintaining well for almost 15 years but then had a breakthrough episode of psychosis despite good drug compliance. So the dose of paliperidone was further increased to maximum dose of 12 mg and as the symptoms persisted , oral haloperidol was started and cross-titrated to 15 mg over a period of 6 week following which patient had more than 70% improvement in PANSS score . Patient maintained well for 4 years and then had exacerbation of symptoms. On examination, thought broadcasting, delusion of control and persecution was present.

So patient was given 8 sessions of ECT and showed improvement in symptoms. Patient was started on 50 mg of amisulpride and was increased to 400mg within a week. Though the patient showed significant reduction in symptoms score in PANSS , she developed severe low abdominal pain within a week of reaching 400mg of amilsulpride, due to retention of urine with distension of bladder. So the dose was reduced to 200 mg but still the symptoms persisted . So Amisulpride was stopped and the patient condition improved. After a period of two weeks , patient was restarted with 25mg of Amilsulpride and was increased to 50 mg within a period of week. Patient complained of difficulty in passing urine and this was confirmed with the ultrasonography which confirmed more than 200ml of residual urine even after voiding. And on stopping the drug the symptoms improved dramatically. So the patient was continued with 12 mg of paliperidone and 15 mg of haloperidol with maintenance ECT every two week once. Patient maintained well with the above medication.

DISCUSSION

The action of micturition depends on the coordinated actions of detrusor and sphincter musculature in the lower urinary tract. In the storage phase, sympathetic nerves causes relaxation of the detrusor by releasing noradrenaline and by binding to beta 3 adrenergic receptors. Pudental nerve causes contraction of the skeletal muscle tissue of the external sphincter by releasing acetylcholine. When it is safe to void, acetylcholine released in response to the parasympathetic signals from the pelvic nerve stimulates muscarinic receptors to cause contraction of the smooth muscle fibers in the detrusor(3). As amisulpride has no affinity for cholinergic receptors, we speculate that the adverse effect would be due to the dopaminergic system . An animal study results suggest that in conscious rats, D1 receptors tonically inhibit the micturition reflex and that D2 receptors are involved in facilitation of the micturition reflex(4).Reduction in the bladder capacity and detrusor overactivity has been associated with the central acute D2 receptor stimulation, suggesting that the blockage of D2 caused by higher dose of amisulpride may be a factor in urinary retention(5), but in our case , it was found that amisulpride caused urinary retention at a low dose itself. It shows that the dopaminergic system has a role in causing urinary retention. As the mechanism of action of amisulpride in causing urinary retention was not well known, so further studies are required and clinicans should be aware of this adverse effects while prescribing amisulpride.

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