

AMISULPIRIDE INDUCED DYSMEGALOPSIA: DIAGNOSTIC DILEMMA**Dr. Prerna Kukreti***

¹MBBS MD(Psychiatry), IDMLHR (International Diploma In Mental Health Law & Human Rights) Assistant Professor, Department of Psychiatry, Hamdard Institute of Medical Science & Research (HIMSR), Jamia Hamdard.

***Correspondence for Author: Dr. Prerna Kukreti**

MBBS MD(Psychiatry), IDMLHR (International Diploma In Mental Health Law & Human Rights) Assistant Professor, Department of Psychiatry, Hamdard Institute of Medical Science & Research (HIMSR), Jamia Hamdard, India.

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ABSTRACT

Dysmegalopsia is a disorder of perception characterized by distorted perception of spatial intensity. It is usually seen in central nervous system lesions, especially temporal lobe, but also can be seen in disorders of accommodation or in few drug poisonings. We report a rare case of transient dysmegalopsia induced by a psychotropic amisulpiride; with complete resolution on discontinuation of medicine. Mental health professionals need to be cautious for such reversible ophthalmological side effects of psychotropics, which often are disregarded as part of psychopathology or illness in persons with psychotic disorders.

KEYWORDS: Dysmegalopsia, Amisulpiride, ophthalmological side effects.**INTRODUCTION**

Dysmegalopsia is a sensory distortion in which objects are perceived distorted in shape. It can result from disorders of temporal or parietal lobe or retinal diseases or disorders of accommodation and convergence. Occasionally it can also be seen in atropine poisoning or hypoxia and rapid acceleration in high altitude pilots.^[1] We hereby report a rare case of transient dysmegalopsia induced by a psychotropic, amisulpiride.

CASE REPORT

A young adult male, 32 years of age presented to psychiatry OPD of a tertiary care mental health facility with subacute onset continuous illness of past 4 years duration with nil precipitating factor, characterized by delusion of persecution and auditory hallucination third person type. Diagnosis of paranoid schizophrenia was made. Following eighth months of treatment with risperidone 8mg, patient showed near complete improvement in positive symptoms and score on Positive and negative symptom scale (PANSS) decreased from baseline 45 to 37. However, patient developed persistent negative symptoms; alogia, avolition, anhedonia and asociality. Risperidone was cross tapered to Amisulpiride. After one year of being on 400mg amisulpiride, patient showed marked improvement in negative symptoms and PANSS Score came down to 30. However, after 18months of amisulpiride usage, patient started to report features suggestive of dysmegalopsia. There was no apparent history of seizure, prolong work requiring near vision, headache, local pain or redness of

eyes, fever and blunt trauma to eye or head. No corroborative history of use of other medications or drug of abuse was apparent. Physical examination or lobar function tests did not reveal any abnormality. Local ophthalmological examination was unremarkable; visual acuity was 6/6 bilaterally and had no features of convergence insufficiency on examination. Patient's Visual evoked potential, intraocular pressure and MRI brain was normal.

Attempts at dose reduction and adding anticholinergics lead to partial improvement in symptoms. However, on stopping drug, over next three months, symptoms of dysmegalopsia disappeared completely but negative symptoms increased. Patient showed poor response to Aripiprazole, risperidone and activity scheduling. In view of poor response to medicines, family members did not come for follow up for next few months. After nearly nine months family again reported back and informed of self medication with Amisulpiride 500 mg which lead to improvement in negative symptoms but there was reappearance of dysmegalopsia after two months of usage. Amisulpiride was again tapered off with complete resolution of ophthalmologic symptoms over next six months. Patient was continued on olanzapine 35 mg and reported partial improvement and continued to maintain well with PANSS Score 34 with no perceptual distortions.

DISCUSSION

Antipsychotics during the initial days earned more defame for the acute extrapyramidal side effects,

however the turn of century diverted attention towards metabolic side effects. But the yet unseen and unnoticed are the ocular side effects. Antipsychotics can lead to potential ocular adverse effects. In fact literature reports eye to be the second most common organ affected by drug toxicity after liver.^[2] Eye being a small neural mass with extensive high vascularity and high metabolic rate is more vulnerable to drug toxicities.

Antipsychotics have been reported to cause oculogyric crisis, corneal pigmentation, retinopathy and less commonly cataract.^[3] But not substantial evidence exists of it being associated with distortions of visual spatial intensity.

In the index case, by the de novo A → B → A design, temporally amisulpiride proved to be the agent responsible for transient dysmegalopsia. In literature so far three case reports for aripiprazole^[4,5] and only single report for amisulpiride^[6] induced transient myopia has been described, but no case of dysmegalopsia has been reported. Possible hypothesis to support the preposition in current case are:

1. Antipsychotic induced change in refractive power of the eye or sustained accommodation:

Postel et al. (1996) suggested that psychotropics can bring the change by:^[1] sustained ciliary spasm, causing excessive accommodation^[2] increased refractive power of lens due to water imbibition; and ^[3] allergic ciliochoroidal effusion leading to block, by displacing forward the ciliary body-lens-iris complex, leading to macropsia.^[7,8]

2. Dystonia of intralaminar midline thalamic complex

Schiff et al. (1999) proposed that intralaminar-midline thalamic complex (ILN) have a unique projection pattern to basal ganglia, cortex and brainstem. They gate the parallel cortico-striatopallidal-thalamocortical loops related with ocular, motor, autonomic and cognitive functions. Thus, antipsychotic causing dystonia of the ILN can be responsible for effect on peripheral ocular movements and distorted perception of image.^[9,10]

CONCLUSION

Dysmegalopsia may be a benign but troublesome side effect to experience. Further functional imaging studies can be really helpful in discerning the exact mechanism responsible. It may often be missed as being disregarded as part of psychopathology by psychiatrists and ophthalmologist in persons with severe mental illness. Due to disabling personal experiences, it can be an important cause of non compliance with medications. There is need of mental health professionals to be cautious for it as a putative side effect and bring necessary changes in psychotropic regime. Also as a routine in practice, there is need to screen clients for potential ocular side effects also besides asking for acute and long term extrapyramidal or metabolic side effects.

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