

doi: 10.5455/2319-2003.ijbcp20131027

Case Report**Chlorpromazine induced ocular myasthenia gravis****Nitya S*, Deepa Kameswari P, Mangaiarkkarasi A, Jagadeesh A, Meher Ali R**

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ABSTRACT

Drug induced bilateral ptosis is a very rare adverse drug reaction. Here we report a case of ten year old male child with chlorpromazine induced bilateral ptosis due to ocular myasthenia.

Keywords: Chlorpromazine, Adverse drug reaction, Bilateral ptosis

INTRODUCTION

Chlorpromazine is a typical antipsychotic drug which has been commonly used for the treatment of schizophrenia, bipolar disorder, delusional disorder, paranoia and psychotic depression.¹ In addition, it is also used for other conditions like intractable hiccup, pruritus and preanaesthetic medication etc., It acts by blocking central dopaminergic (D₂) receptors and also blocks other receptors like muscarinic, cholinergic (M), adrenergic (α_1) and histaminergic (H₁) receptors.¹ Common adverse effects observed with chlorpromazine therapy are extrapyramidal, anticholinergic, endocrinal and cardiovascular effects. Very rare adverse effects like myasthenia gravis, systemic lupus and Horner's syndrome were also reported.^{2,3} Drug induced myasthenia gravis has been reported rarely with other central nervous system medications like Phenytoin, Trimethadione, Lithium and Trihexyphenidyl. We report a case of 10 year old male child who had developed reversible ocular myasthenia gravis after chlorpromazine therapy.

CASE REPORT

A 10 year old male child was hospitalized with complaints of emotional instability and weakness of both

upper and lower limbs which is more marked on the right side since seven days. He also had involuntary movements all over the body which disappeared during sleep. History of slurring of speech, difficulty in feeding was present. Scholastic performance was average. There was no significant past history of similar episodes. He was not a known case of acute rheumatic fever. There was no history of birth trauma, prematurity or significant systemic illness. On examination child was anemic, there were no neurocutaneous markers/ Kayser Fleischer rings/ markers for acute rheumatic fever. Cardiovascular system examination revealed pansystolic murmur. Tone of his right lower limb was diminished and signs suggestive of chorea (pronator sign, milkmaid grip) were observed. There was a marked elevation of ASO titre suggesting the evidence of previous streptococcal infection. Echocardiogram revealed mitral valve thickening. CT imaging of the brain was normal. Based on the above findings, patient was diagnosed as a case of acute rheumatic chorea and child was started on oral chlorpromazine 25mg thrice daily and oral penicillin V 250mg sixth hourly.⁴ Supportive measures such as folic acid, paracetamol, multivitamin syrup were given. However after a week of therapy, child developed bilateral ptosis as well there is worsening of dysarthria and muscle weakness. Diurnal variation i.e., worsening of

ptosis by the end of the day was also noted. There were no features of associated ocular abnormalities and congenital ptosis was ruled out by checking the old picture of the child which was obtained from his parents where the child's eyes were absolutely normal. Cogan's lid twitch sign was positive. Chlorpromazine induced ptosis was diagnosed and the offending drug was withdrawn and continued with other treatment. There was a complete recovery of ptosis within a week of cessation of the drug.

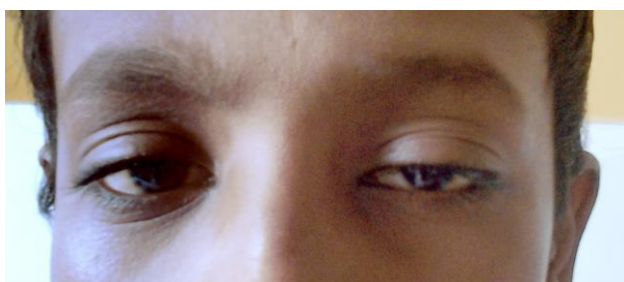


Figure 1: Patient with bilateral ptosis.

DISCUSSION

Ptosis is drooping or falling of the upper eyelid. It can be unilateral or bilateral. Causes of ptosis include myogenic, neurogenic, aponeurotic, mechanical, drug induced and pseudoptosis.⁵ Drugs causing ocular myasthenia includes antimicrobials like gentamicin, streptomycin, colistin, kanamycin and others like d-penicillamine, chloroquine, phenytoin, lithium etc.⁶ The pathogenesis of drug induced myasthenia is not well understood. However probable increase in serum antibodies to acetylcholine receptor could be attributed for the pathogenesis. Altered immunological reactivity with the population of B cell lymphocytes induced antibodies to acetylcholine receptors had been postulated in a previous study.⁶

In this case, child had bilateral ptosis with no other signs of myasthenia gravis. Hence diagnosis of chlorpromazine induced ocular myasthenia was made. The tests used to confirm the ocular myasthenia were tensilon test, electromyography studies, serological studies, receptor studies and muscle biopsy. However in our hospital, only tensilon test was done which was positive.

Naranjo ADR probability scaling was done and the score was 8, which indicated 'probable' causality reaction to chlorpromazine.⁷ The causality score were as follows: previous conclusion reports on this reaction(+1); adverse event appeared after chlorpromazine was administered(+2); this adverse effect improved within one week of discontinuation of drug(+2); this ptosis reappeared when the drug was re-administered(0);

alternative causes for the reaction(+2); reaction reappeared when placebo was given(0); drug detected in blood(0); reaction became severe when the dose was increased/less severe when the dose was decreased(0); similar reaction with previous exposure(0); ptosis confirmed by objective evidence(+1). So in this case, chlorpromazine induced ocular myasthenia appeared after short term administration and also the symptoms disappeared within a week of discontinuation.

CONCLUSION

Chlorpromazine, which is one of the most commonly prescribed drugs, had been reported to cause many adverse effects. However, to our best knowledge, this is a rare case of ocular myasthenia gravis in pediatric age which had not been reported so far. This is the first such case report from South India.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

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doi:10.5455/2319-2003.ijbcp20131027

Cite this article as: Nitya S, Deepa Kameswari P, Mangaiarkkarsi A, Jagadeesh A, Ali RM. Chlorpromazine induced ocular myasthenia gravis. *Int J Basic Clin Pharmacol* 2013;2:653-4.