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Imipramine Induced Acute Dystonia : Case Report

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ABSTRACT

Imipramine is a drug that has been widely used in the management of enuresis. Here we report a case of a child started on imipramine for bed wetting and developed an acute dystonia. Though rare, this side effect of imipramine must be kept in mind and due precautions must be exercised when prescribing imipramine in vulnerable populations.

KEYWORDS : Imipramine, Dystonia

INTRODUCTION

Imipramine is a tricyclic antidepressant that acts via action on the serotonin and nor adrenaline systems. It has been used widely in the management of depression in adults and as a treatment of enuresis in children [1]. Dystonia though a rare side effect of imipramine has been described in the past in anecdotal case reports [2-3]. There have been many hypotheses proposed to explain the onset of dystonia and the most commonly accepted one is that of dopamine acetylcholine imbalance and- the intrinsic anticholinergic activity of tricyclic drugs [4]. Here we present a case of imipramine induced dystonia in a child who started on imipramine as a treatment for enuresis.

CASE REPORT

A 13 year old male student, studying in the 6th standard, elder among 2 siblings, presented to the out patient department of our hospital with chief complaints of academic difficulties and bed wetting. He was referred from the school for our assessment. His academic complaints included difficulties in writing, incomplete class note books, he couldn't catch up with the speed of the teacher. He would also have difficulties in understanding certain subjects, memorising facts and was particularly poor mathematics. The parents also informed about the child having primary nocturnal enuresis daily, once in the night. Patient never had wetting episodes in the day. He had never taken any treatment for the same. The patient was evaluated and had an IQ of 78. He was issued a Borderline IQ certificate (slow learner) and was told to pursue the open schooling system. For the nocturnal enuresis, the patient was prescribed 25mg of Imipramine at bedtime and behaviour therapy in the form of star charting and positive reinforcement along with intermittent awakening. He was on 25mg of tablet imipramine for over 6 months when suddenly developed an acute dystonia.

The patient woke up in the morning and it was observed that the patients neck was turned upwards, there was uprolling of the eyeballs, he was conscious and calling out to his mother as he couldn't bring his neck or his eyeballs downwards despite efforts. Family members rushed him to the emergency medicine department of our hospital where he was given an injection of Promethazine and his symptoms subsided in 5 minutes. The patient could recollect the entire incident which lasted 30minutes as all the events were in intact consciousness. Imipramine was the only medication that the patient was taking and the last dose was a night before the episode occurred. Imipramine was discontinued and behaviour therapy was continued alone. He was given Promethazine 12.5 mg orally at bed time for a week to avoid recurrence of a dystonia. The patient's EEG was done and was within normal limits. No subsequent episode of dystonia has been reported over the last month. Neuroimaging studies too revealed no abnormality. It was noteworthy that the patient had a history of febrile seizures at the age of 2 years and he was prescribed Sodium Valproate 250mg in syrup form daily. His MRI Barin at that age too were normal while the EEG when he was 2 years old had bilateral neuronal hyperexcitibility. He continued the medicine for 3 years (till the age of five), and it was then gradually tapered and stopped. Those were the only seizure episodes he ever had in his life.

DISCUSSION

It has been hypothesized that tricyclic antidepressants indirectly modulate dopaminergic neurotransmission though that it not their main area of action [5]. The tricyclic drugs increase serotenergic transmission in various areas of the brain and in turn modulate glutamtergic transmission and exert and indirect effect on D2 and D3 receptors [6]. Increased serotenergic transmission in the mid brain leads to decreased dopamine firing rate and this causes an indirect inhibition of the nigro-striatal dopaminergic system [7]. This is coupled with increased sensitivity in the dopamine receptors in striatal post synaptic areas that may results in various movement disorders including dystonia [8]. This sensitization takes a few months and hence dystonia or movement abnormalities may be seen usually a few months after starting tricyclic drugs rather than in a few weeks [9]. Stopping the drug reverses the processes and in turn causes the dystonia to disappear. The occurrence of dystonia with tricyclic drugs can also be genetic in nature due to intrinsic genetic susceptibility of the patient or may be due to a hyperexcitable brain which is sub clinical as in our case where an early childhood insult to the brain in the form of febrile seizures had occurred along with a low IQ that may lead to brain vulnerability [10]. Nevertheless clinicians need to exercise caution and must watch for the presence of movement abnormalities in patients on long term antidepressant therapy.

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