ANTICHOLINERGIC EXACERBATION OF PSYCHOTIC SYMPTOMS: A CASE REPORT

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ABSTRACT: A common practice in psychiatry when treating patients is the concurrent administration of anticholinergics along with antipsychotics, either to prevent or treat extrapyramidal syndrome reactions from occurring. However, most antipsychotics have inherent anticholinergic properties themselves. Therefore, this subtype of these patients have a higher than usual risk of developing anticholinergic side-effects, of which the central nervous manifestations can mimic psychosis, and may cloud judgement on patients’ progress towards their treatment. (JUMMEC 2003-2005; 8: 61-62)

KEYWORDS: Anticholinergics, anticholinergic toxicity, antipsychotics

Introduction

Since the introduction of Chlorpromazine in the 1950’s, antipsychotics have been the mainstay of treatment for psychosis. They have complex central nervous system actions with effects on numerous receptors such as dopaminergic, serotogenic muscarinic (cholinergic), alpha1-adrenergic and H1-histaminergic receptors, giving rise to numerous possible side-effects (1). A common practice when prescribing antipsychotics is the concurrent administration of anticholinergics, either to prevent or treat extrapyramidal syndrome (EPS) reactions from occurring. This addition of anticholinergics to a patient on antipsychotics, particularly those with significant inherent anticholinergic properties, would theoretically increase the risk of anticholinergic side-effects occurring. One of the side-effects that is rarely recognized or reported is exacerbation of the positive symptoms of schizophrenia, which if unrecognized, may cloud the physician’s judgement on the patient’s progress. This phenomenon of psychosis exacerbation, though replicated from several sources has failed to gain the recognition in clinical practice that its potential significance would justify (2). We present a case of a patient whose psychotic symptoms apparently worsened after he was commenced on anticholinergics, to illustrate this important point.

Case Report

A 22-year old single man was admitted to the psychiatric ward with aggressive and abnormal behaviour for one day. He believed that everyone was watching him, that the contents of television had significant importance to him, and admitted to hearing voices. He was physically aggressive towards his family members and destroyed things in the house. The only significant stressor elicited was that he had broken off with his girlfriend two months before, and had since established another relationship with another girl despite his father’s strong objections. Physical examination, lab investigations and a CT brain scan revealed no abnormal findings. He was started on Risperidone, an atypical antipsychotic at a dose of 2 mg twice a day after a provisional diagnosis of Brief Psychotic Disorder was made. His agitation and psychotic symptoms improved gradually over six days of treatment. He then developed severe dystonia, which was attributed on the antipsychotic he was on. The dystonia soon resolved after Benzhexol, an anticholinergic agent was added at a dose of 2 mg twice a day after a provisional diagnosis of Brief Psychotic Disorder was made. His agitation and psychotic symptoms improved gradually over six days of treatment. He then developed severe dystonia, which was attributed on the antipsychotic he was on. The dystonia soon resolved after Benzhexol, an anticholinergic agent was added at a dose of 2 mg three times a day. However, on the 8th day of admission, he became verbally abusive, agitated and aggressive again. Auditory hallucination reappeared, and at times he appeared confused and disinhibited. As repeat physical examination and lab investigations were normal, anticholinergic psychosis was suspected and Benzhexol was tapered down over three days. His condition then improved dramatically and he was discharged well on day 13 of admission.

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Discussion

The patient discussed above was on high doses of an anticholinergic, in addition to a high dose of an antipsychotic which by itself has anticholinergic properties, therefore increasing the risk of him developing anticholinergic side effects. Peripheral muscarinic receptors may result in dilated pupils, warm, dry and flushed skin, decreased secretions of the mouth, pharynx, nose, and bronchi; fever, tachycardia, hypertension, hyperreflexia, constipation and urinary retention. On the other hand, central nervous system manifestations may result in signs that mimic functional psychosis such as hallucinations, delusions, agitation, anxiety and paranoia (3). However, rarely in clinical practice do all of these signs manifest together. In milder forms, patients may only present with peripheral signs, and central signs may only be apparent in severe cases. However, it is important to note that some patients may present only with a few central signs, without any peripheral anticholinergic manifestations. It has been shown that addition of anticholinergic agents in acutely ill patients treated with antipsychotics can result in a small but definite exacerbation of positive (hallucinations and delusions) symptoms without any other anticholinergic signs (4).

However, this unique situation is rarely recognized in patients that are psychotic to begin with. Clinicians should be aware of this possibility, especially in psychotic patients who are not improving, or appear to be resistant despite adequate antipsychotic treatment. Failure to do so would lead to these patients being given increasing doses of antipsychotics to control the so-called escalating psychosis, and exposing them to potentially more side-effects, or being labeled as treatment resistant.

References