

Metabolic Complications with Aripiprazole

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Abstract

Second generation antipsychotics are widely used in the treatment of psychotic disorders, and has been linked to development of metabolic syndrome. Aripiprazole is a atypical antipsychotic with a novel mechanism of action and has been generally found to be least linked with the development of metabolic complications, such as obesity , diabetes and hyperlipidemia. We present a case report of a patient on aripiprazole who developed obesity and diabetes mellitus after initiation of aripiprazole (German J Psychiatry 2010; 13: 49-50).

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Introduction

Second-generation (atypical) antipsychotic medications are of great benefit to a wide variety of people with psychiatric disorders, especially patients with schizophrenia. However, one constellation of adverse effects is an increased risk of obesity, diabetes, and metabolic syndrome. Available evidence suggests that clozapine and olanzapine have a higher propensity to induce diabetes and metabolic syndrome compared with other atypical antipsychotic drugs, risperidone and quetiapine. Despite more limited available data, amisulpride, aripiprazole and ziprasidone showed less likelihood of precipitating diabetes. Interestingly, reversibility of drug-related diabetes has been reported with aripiprazole (Scheen et al. 2007, De Hert M et al. 2007).

Aripiprazole has been marketed with emphasis on its favourable metabolic profile with little or no elevating blood sugars or weight gain. (L'Italien et al. 2007). The incidence of metabolic syndrome in patients on aripiprazole was 5.3% as compared to 14.3% in patients on placebo. (L'Italien et al. 2007)

There are only a few case reports linking aripiprazole with elevating blood sugars and causing weight gain. (Makzouhmi et al. 2008, Reddymasu et al. 2008)

We present a patient who developed diabetes after starting aripiprazole as well as gained weight, adding to the existing literature about aripiprazole and metabolic complications.

Case Report

Mr X is a 40 year old single male of Caucasian background. His first presentation to psychiatric services was in 2001. He was seen in the outpatient clinic and was diagnosed to have a moderate depressive episode. He was commenced on citalopram, following which he recovered and there was no further contact with psychiatric services till 2008.

His second episode was in March 2008, when he presented with a manic episode, which required inpatient admission. He was started on risperidone; the dose was increased to 4 mg daily and was discharged following improvement in his mental state. He was followed up in the outpatient clinic. The dose of risperidone was gradually reduced and stopped as he started developing depressive symptoms as well as extra pyramidal symptoms after 1 month. He was commenced on mirtazapine and quetiapine as a mood stabilizer as well as sodium valproate.

As his symptoms continued to worsen, he was readmitted in April 2008 and stayed in hospital for 45 days. Mr X was diagnosed to have bipolar affective disorder, current episode severe depression with psychotic symptoms. Mirtazapine and sodium valproate was stopped and he was commenced on duloxetine 60 mg mane. Quetiapine was also tapered and stopped due to no improvement in mental state. He was commenced on aripiprazole in middle of April 2008.

Baseline blood tests were done before commencing aripiprazole which included fasting blood sugar and serum lipids which were within normal limits. Liver function tests were also normal. All other blood parameters including thyroid functions were normal. His baseline weight was 105 kg before commencing aripiprazole. His body mass index was 32.5 in April 2008.

He had a third admission in October 2008 with features suggestive of a psychotic depressive episode; the diagnosis was then changed to schizoaffective disorder. He remained on 15 mg of aripiprazole, and duloxetine was changed to venlafaxine. His liver function tests were noted to be abnormal with elevated GGT. His blood sugar and serum lipids were normal.

He was then seen in the outpatient clinic in December 2008, when his fasting blood sugar was quite high (18 mmol/l), with elevated gamma glutamyl transferase, elevated liver enzymes and alkaline phosphatase. His life style was sedentary with minimal physical activity. Fasting blood sugars were repeated and it was still elevated following which he was commenced on oral antidiabetic medication in December 2008 and was advised dietary modifications. His weight also increased from 105 kg to 123 kg after 8 months of being on Aripiprazole and Venlafaxine. In view of him putting on excessive weight and developing diabetes 8 months after starting aripiprazole, an attempt was made to change over from aripiprazole to amisulpride but the change resulted in a relapse of psychotic symptoms. He was then recommenced on aripiprazole.

There was no family history of diabetes or previous history of diabetes before commencing medication.

He remains on venlafaxine 225 mg mane and aripiprazole 15 mg mane as well as oral anti diabetic medication at this point of time and continues to be followed up by the community mental health team.

Discussion

This case report illustrates that aripiprazole can also contribute to weight gain and diabetes, adding to the existing limited literature.

This patient put on 18 kg of weight in 8 months and developed diabetes after being on 8 months of aripiprazole. There was no family history of diabetes, his baseline blood sugars were normal. He also developed elevated liver enzymes on aripiprazole.

Risperidone and quetiapine did not elevate his blood sugar, but he did not stay on it for more than 1 month.

Limitations are inherent, this is only a case report, there are multiple confounding factors such as lifestyle and predisposing factors, but this adds on to the limited literature linking aripiprazole with metabolic complications raising awareness to screen for these factors. (Makzhoumi et al. 2008)

One needs to be aware about screening all patients on antipsychotics for early diagnosis and treatment of metabolic complications therefore minimizing the risk of cardiovascular disease at a later stage.

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