

Elevated liver enzymes after olanzapine use: a case report

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Received: 31 October 2015

Accepted: 18 December 2015

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ABSTRACT

Olanzapine is a second-generation antipsychotic used in bipolar mania and schizophrenia. We are reporting the case of a young female patient who developed elevated liver enzymes after olanzapine use. Patient's liver enzymes improved after the discontinuation of olanzapine making it a possible cause for reversible hepatotoxicity. We recommend physicians to check liver enzymes regularly if patient is on olanzapine.

Keywords: Olanzapine, Liver enzymes, Bipolar mania

INTRODUCTION

Olanzapine is one of the second generation antipsychotics that have D₂ receptor and serotonin 5HT₂ receptor affinity.¹ Our patient, who is a 27 year old female, was taking olanzapine for bipolar mania. Patient developed elevated liver enzymes while she was on olanzapine, however, the mechanism by which olanzapine causes liver injury is unknown.²

CASE REPORT

Patient is a 27 year old African American female, single, domiciled in a shelter, with past psychiatric history of bipolar disorder, no significant past medical history, and was brought to the emergency room because of anxiety. After stabilization, patient was transferred to the psychiatric emergency room since patient was loud and aggressive. Upon psychiatric interview, patient reported of being extremely anxious. Patient reported sleep disturbance but no change in appetite was noticed. Patient reported of easy distractibility, goal directed activity and more interest in pleasurable activities for a week. Patient reported history of multiple psychiatric hospitalizations in

the past and received Depakote, Risperdal, and Cogentin. Upon psychiatric evaluation, patient seemed to be aggressive, restless and agitated. Patient denied any current drug abuse. Patient seemed to have elevated mood with labile affect. Patient's speech seemed to be loud and pressured. Patient seemed to be grandiose with racing thoughts. Patient denied any signs of feeling depressed. Patient also denied any suicidal or homicidal ideations or plans. Patient denied any auditory or visual hallucinations. Patient was medicated in the emergency room because of threatening behaviour. After stabilization, patient was transferred to inpatient psychiatric unit.

Upon arrival on the unit, patient seemed to be loud, irritable, and was laughing inappropriately. Patient was started on Klonopin 3 mg/day, lithium 900 mg/day, olanzapine 30 mg/day, and zolpidem 10 mg/day.

On 5th day of admission, patient's symptoms seemed to be improving with less loud and pressured speech although she remained grandiose, delusional and preoccupied with inappropriate sexual thoughts. Patient was hypomanic with less frequent mood swings. Lithium was increased

to 1200 mg/day and the blood lithium level was within the therapeutic range (0.8 mEq/L).

On 10th day post admission, patient's blood work was repeated that revealed elevated liver enzymes (Alanine transaminase or ALT= 240, Aspartate transaminase or AST = 141, Alkaline phosphatase or ALP =133), although hepatitis panel and abdominal sonogram were unremarkable. Patient's medications needed to be adjusted because of elevated LFTs hence olanzapine was tapered off and eventually discontinued. Clonazepam was discontinued and patient was planned to be started on lurasidone (Latuda) 80 mg/day. Patient's repeated LFTs showed improvement except alkaline Phosphatase that remained elevated. According to the patient, she does exercise so may be alkaline Phosphatase was coming from the bones.

Patient showed improvement in the behaviour and she seemed to be calmer and cooperative hence was planned for discharge.

DISCUSSION

Olanzapine, a second generation antipsychotic, acts as a D₂ receptor antagonist.³ Some of the reported cases of side effects possibly due to olanzapine are myoclonus,⁴ tardive dystonia,⁵ and restless leg syndrome.⁶ Moreover, in 2007, three cases of pancreatitis followed by olanzapine were reported. Olanzapine got approved by FDA in 1996 and it does not cause agranulocytosis as a side effect.⁷

Literature review regarding the interaction of olanzapine with lithium revealed a reported case of delirium and EPS in 2005.⁸ Although literature review regarding interactions of olanzapine with clonazepam and zolpidem showed no significant data on PubMed.

Literature reviewed for the common side effects of Ambien, and Lithium but none of them cause hepatic enzyme derangement. Patient was also on clonazepam for anxiety and to the best of our knowledge only one case of clonazepam induced liver injury was reported in 1988,⁹ whereas, according to a study done in Turkey, asymptomatic increase in liver enzymes is commonly seen in patients who are on atypical antipsychotics,¹⁰ making it a likely cause of hepatotoxicity in our patient but unfortunately the mechanism involved is unknown.²

Considering alcohol abuse as a cause of liver enzymes derangement, patient was screened for substance abuse, particularly, alcohol abuse but she denied any current substance abuse including alcohol. Our patient showed improvement in the liver enzymes after discontinuation of olanzapine hence clinicians should be monitoring the liver enzymes regularly.

CONCLUSION

Liver injury in our patient was possibly as a result of olanzapine use since patient showed improvement after its discontinuation. Although it was reversible in our patient, periodic monitoring of liver enzymes if a patient is taking olanzapine is crucial, and we recommend switching patient to other antipsychotics if hepatotoxicity develops.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

1. Miyamoto S, Duncan GE, Marx CE, Lieberman JA. Treatments for schizophrenia: a critical review of pharmacology and mechanisms of action of antipsychotic drugs. Mol Psychiatry. 2005;10(1):79-104.
2. Manceaux P, Constant E, Zdanowicz N, Jacques D, Reynaert C. Management of marked liver enzyme increase during olanzapine treatment: a case report and review of the literature. Psychiatr Danub. 2011;23(1):S15-17.
3. Divac N, Prostran M, Jakovcevski I, Cerovac N. Second generation antipsychotics and extrapyramidal adverse effects. BioMed Res Int. 2014;2014. <http://dx.doi.org/10.1155/2014/656370>.
4. Tikka SK, Pratap A, Sinha VK. Dose-dependent Olanzapine-induced myolonus. Toxicol Int. 2014;21(3):335-6.
5. Sun Z, Wang X. Case report of refractory tardive dystonia induced by olanzapine. B Shanghai Arch Psychiatry. 2014;26(1):51-3.
6. Basu A, Kundu S, Khurana H. Olanzapine-induced restless leg syndrome: A case report and review of literature. Indian J Pharmacol. 2014;46:450-2.
7. Kerr TA, Jonnalagadda S, Prakash C, Azar R. Pancreatitis following olanzapine Therapy: A report of three cases. Case Rep Gastroenterol. 2007;1(1):15-20.
8. Tuglu C, Erdogan E, Abay E. Delirium and extrapyramidal symptoms due to a lithium-olanzapine combination therapy: A case report. J Korean Med Sci. 2005;20(4):691-4.
9. Olsson R, Zettergren L. Anticonvulsant-induced liver damage. Am J Gastroenterol. 1988;83(5):576-7.
10. Atasoy N, Erdogan A, Yalug I, Ozturk U, Konuk N, Atik L et al. A review of liver function tests during treatment with atypical antipsychotic drugs: a chart review study. Prog Neuropsychopharmacol Biol Psychiatry. 2007;31(6):1255-60.

Cite this article as: Ahmed S, Warraich S. Elevated liver enzymes after olanzapine use: a case report. Int J Basic Clin Pharmacol 2016;5:213-4.