Research Article

A Case Report on Risperidone Induced Diabetic Ketoacidosis

Bini Vincent[1]*, Aloysius James[1], Sreekrishnan T P[2], Gireesh Kumar[2], Meenu Vijayan[1]*
[1]Department of Pharmacy Practice, Amrita School of Pharmacy, Kochi, Amrita Vishwa Vidyapeetham, Amrita University, India.
[2]Department of Emergency Medicine, Amrita Institute of Medical Sciences, Kochi, Kerala, India.
***Corresponding author's E-mail:** meenuvijayan@aims.amrita.edu

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ABSTRACT

Risperidone is a second generation or atypical class of antipsychotic which is mainly used in the treatment of schizophrenia and bipolar disorder. Even though the mechanism of action is unknown, it has been proposed that the its therapeutic activity could be mediated through a combination of dopamine type 2 and serotonin type 2 receptor antagonism.[1] Its main ADR's include extrapyramidal symptoms, weight gain, hyperlipidemia.[2] Even though increased blood sugar is one of the main ADRs of Risperidone, Risperidone induced diabetes ketoacidosis are often less likely. Diabetic ketocidosis is a serious complication of diabetes that occurs when the body produces high level of blood acid called ketones.[3] Here in we report a rare case of risperidone induced diabetic ketoacidosis and how it was managed.

Keywords: Risperidone, Diabetic ketoacidosis, schizophrenia, extrapyramidal symptoms.

INTRODUCTION

Risperidone is a Second Generation (Atypical) antipsychotic, which is mainly indicated for the treatment of schizophrenia and bipolar disease. Apart from its main ADRs such as extra pyramidal side effects, weight gain, hyperlipidemia, QTC prolongation and hyperglycemia;[2] Diabetic ketoacidosis are seldom reported.[4] Diabetic ketoacidosis (DKA) is an acute, major, life-threatening complication of diabetes mellitus. This condition is a complex disordered metabolic state characterized by hyperglycemia, ketoacidosis, and ketonuria.[3] DKA is defined as an increase in the serum concentration of ketones greater than 5 mEq/L, a blood glucose level greater than 250 mg/dL (although it is usually much higher), and a blood (usually arterial) pH less than 7.3. Ketonemia and ketonuria are characteristic, as serum bicarbonate level of 18 mEq/L or less (less than 5 mEq/L is indicative of severe DKA).3 Mental status changes can be seen with mild-to-moderate DKA.[3] The following case report illustrates a rare clinical outcome as the result of risperidone therapy and its management.

CASE HISTORY

A 39-year-old male was admitted to the emergency department with vomitting, nausea, fatigue, weakness and abdominal pain. He had a history of bipolar/schizoaffective disorder but no history of diabetes mellitus. He was on Clozapine but changed to Risperidone 1.5mg BD one year back because of extrapyramidal side effects. On admission, there was signs of dehydration. Physical examination was otherwise normal. Routine laboratory tests showed leukocytosis that is WBC count was - 12.4 K/uL, Neutrophils- 87.3%, Lymphocytes – 7.4 %, Eosinophils – 0%; ketosis that is 3+; acidosis where Bicarbonate: 13.1 mmol/L and ABG was suggestive of metabolic acidosis; elevated blood sugars that is, his GRBS was found to be elevated that is 457 mg/dL. HB A1C was found to be 15.3 %, hyperosmolality where blood Osmolality was 327 mOsm/kg; Urine routine tests showed ketonuria where Urine Ketone was 3+ that is approximately 30mg/dl; and protienuria where Urine Protein was 2+ that is (2+ {++} means the proteinuria is at 100 - 200mg/dl) level. With a provisional diagnosis of DKA, Risperidone was withheld, then the sugar level was reduced significantly. Further glycemic control was acheived with Insulin. He was also given IV fluids. Psychiatry consultation was taken and his medications for BPAD were optimised. With the above measures, He improved symptomatically. Later he was prescribed with Clozapine again instead of Risperidone. The ADR was assessed using NARANJO probability assesment scale and found to be a probable ADR with score of 7.

DISCUSSION

Studies shows that patients taking atypical antipsychotics are prone for hyperglycemia.[1] The risk of developing diabetes mellitus or impaired glucose tolerance is 2-fold greater with atypical agents such as Olanzapine and Risperidone than with typical agents.[4] The exact mechanism underlying this toxic effect are yet unknown. Literatures shows that the lag period from initiation of atypical antipsychotics to development of hyperglycemia also varies.[5] Several factors increase the risk of diabetes and diabetic ketoacidosis induced by atypical antipsychotics, including- a diagnosis of schizophrenia, overweight, hypertension, family history of diabetes mellitus.etc.[5] While absence of these risk factors does not

preclude development of diabetic ketoacidosis or diabetes while a patient is receiving atypical antipsychotics, their presence should encourage clinicians to weigh the risk and benefits before initiating these drugs.[5]

In 1999, the results of a cross-sectional study revealed a possible association between typeII diabetes and antipsychotics.[6] Specifically, diabetes was diagnosed in 6% of those treated with Risperidone. A study by American Diabetes Association shows that Individuals taking an atypical antipsychotic drug, particularly younger patients under 40 years of age represent an under recognized group at high risk of type 2 diabetes.[4] A few cases reported in pubmed shows Risperidone induced diabetes mellitus and diabetes ketoacidosis where most of these patients where schizophrenic.[7, 6]

This case report illustrates that regular monitoring of body weight and blood sugar during the therapy is mandatory where a clinical pharmacist can play an important role.[6]

CONCLUSION

From this case report, we conclude that there are higher probabilities of occurring diabetes mellitus or diabetes ketoacidosis for patients taking long term therapy of atypical antipsychotics especially Risperidone which is reported here. It has been recommended that for such patients, baseline and 6-month monitoring of fasting plasma glucose levels, glycosylated haemoglobin levels at every three months should be monitored strictly. Switching from an atypical antipsychotic to another may improve glucose control in some individuals.

Table 1: Relevant Laboratory Investigations of the Case

Laboratory Investigations	Results (On Admission)	Normal Ranges
Ketones	3+ (Over 3.0 mmol/l)	<0.6 mmol/l
Bicarbonate	13.1 mEq/L	23 to 30 mEq/L
GRBS	457 mg/dL.	72 to 108 mg/dL
HB A1C	15.30%	4% - 5.6%
Blood osmolality	327 mosm/kg	275–295 mosm/kg
Urine ketone	3+ (30mg/dl)	Moderate DKA: 30 to 40 mg/dL
Urine protein	2+	0 to 20 mg/dL
PH	7.13	>7.3
PCo2	30	-2 to +2 mEq
Sodium	132	135 – 145 mEq/L
Potassium	3.8	3.5 – 5.1mEq/L
Chloride	101	96 to 106 mEq/L

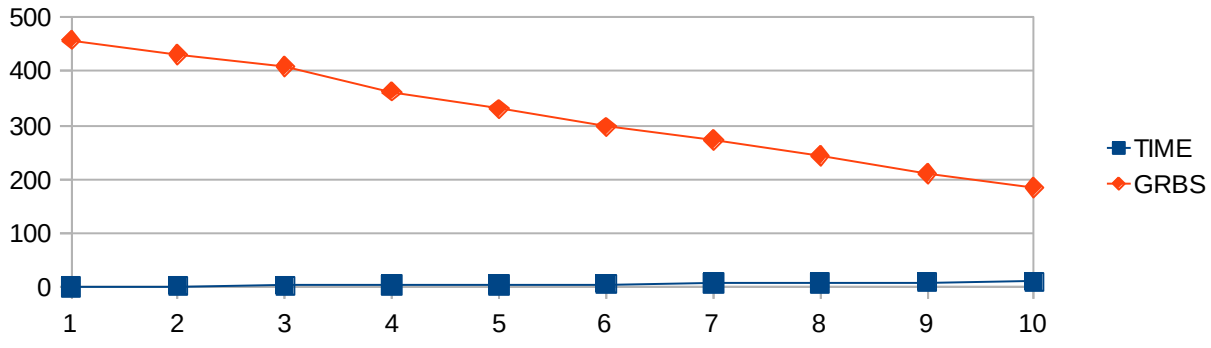

Figure 1: Reduction in GRBS values After Drug Discontinuation

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