

RISPERIDONE INDUCED CONVULSIONS SECONDARY TO HYPONATREMIA

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Summary

A patient suffering from chronic schizophrenia was treated with tablet risperidone 3mg/day. Patient developed convulsions during the treatment. Serum electrolytes showed severe hyponatremia. Since all other investigations were normal and there was no recurrence after discontinuing the drug, we attribute this episode of convulsions to hyponatremia induced by risperidone.

Key words: Risperidone, hyponatremia, convulsions

Running head: Risperidone induced convulsions

Introduction

Antipsychotic agents are the cornerstone of treatment for schizophrenia, of which the atypical antipsychotics promise enhanced efficacy and safety. Risperidone is one of the widely used and better tolerated atypical antipsychotic. Risperidone, a potent D2 antagonist shows less incidence of extrapyramidal symptoms compared to haloperidol^[1] The common adverse effects with risperidone are insomnia, anxiety and headache. Other adverse effects are cerebrovascular accidents, tachycardia, weight gain, increased liver enzymes, decreased neutrophils and dose dependent increase in prolactin levels. Dyspepsia, nausea, vomiting, sexual dysfunction, dizziness, orthostatic hypotension have been reported less commonly. Rare adverse effects encountered are seizures, hyponatremia, neuroleptic malignant syndrome and tardive dyskinesia.^[2] Here we are reporting a case of risperidone induced convulsions secondary to hyponatremia.

Case report

A thirty four year old male diagnosed of chronic schizophrenia according to ICD-10 was on chlorpromazine for the past 5 years. The patient was non compliant and was taking irregular treatment. On admission patient was aggressive with irrelevant speech and was abusing people. On physical examination he had severe pallor. Investigations revealed that his hemoglobin was 3.9g/dl, blood counts were within normal limits, liver function tests and renal function tests were normal. Peripheral smear showed microcytic hypochromic anemia. Blood transfusion was given followed by intravenous iron sucrose.

Risperidone 1mg was started to control psychotic symptoms. Parenteral iron was given for 5 days till hemoglobin reached 7 g /dl. Later the patient was switched to oral iron therapy. The dose of risperidone was gradually increased to 3mg / day. After receiving 3mg/day of risperidone for two days, patient had an episode of convulsions and vomiting (10th day of admission) for which he was given injection Lorazepam 1 mg intravenously. Patient had no previous history of seizures. Serum electrolytes revealed sodium 111mEq/l, potassium 3.9mEq/l and random blood sugar was 104mg/dl. Renal function tests were within normal limits. Urine output and BP were normal suggesting euvolemic status. Computed tomography and electroencephalography did not reveal any abnormality. All medications were stopped. Hyponatremia was corrected by administering 3% saline, followed by normal saline. Serum sodium gradually increased to 135mEq /liters. Risperidone was replaced by tablet Haloperidol 5 mg once a day and tab Trihexyphenidyl 2mg. Patient is maintained on Haloperidol, Trihexyphenidyl and oral iron for the past two months and there is no episode of convulsions. However, his psychotic symptoms are not so well controlled as compared with Risperidone.

Discussion

In the present case, the patient who was on Risperidone 3mg had an episode of convulsions. This can be most probably attributed to hyponatremia since there was no previous history of seizures, his computed tomography and electroencephalography was normal and no recurrent episode occurred after hyponatremia was corrected.

Hyponatremia, the most common electrolyte abnormality, is defined as serum sodium concentration of less than 130 mEq/L. Mild hyponatremia (130–135 mEq/L) is usually asymptomatic. Nausea and malaise can occur when the plasma sodium is below 125–130 mEq/L. Headache, lethargy, and disorientation are seen with plasma sodium levels of 115–120 mEq/L. The most serious symptoms of severe and rapidly developing hyponatremia are seizures, coma, permanent brain damage, respiratory arrest, brainstem herniation, and death^[3].

The potential causes of hyponatremia are CNS disorders, neoplasms with ectopic hormone production, pulmonary disease, endocrine disease and drugs.^[4] In the above case, we can attribute hyponatremia to be drug induced because the patient did not have any other systemic disorder. Psychogenic cause of hyponatremia can be ruled out as this patient did not have any history of polydipsia.

The patient was on oral iron and risperidone at the time of this episode. Since iron does not cause hyponatremia, we can attribute it to risperidone. As per the causality assessment of suspected adverse drug reaction, this reaction can be categorized as probable or likely adverse drug reaction.^[5] One earlier case of convulsions due to hyponatremia induced by risperidone has been reported.^[6] It is assumed that the mechanism of hyponatremia is similar to other psychotropic medications, in that, it is secondary to the syndrome of inappropriate antidiuretic hormone (SIADH).^[6]

Acute severe hyponatremia is a life threatening event that demands prompt and aggressive treatment hence periodic monitoring of serum electrolytes becomes mandatory in patients on risperidone. Physicians should be cautious of this side effect while prescribing risperidone.

Hyponatremia induced by psychogenic polydipsia is a common feature of schizophrenia. Antipsychotics like risperidone should be used cautiously since risperidone can cause hyponatremia per se. Reports suggest that risperidone is less epileptogenic as compared to clozapine, olanzapine and quetiapine,^[7] but our case report puts forth that seizures can occur with risperidone if serum electrolytes are not monitored regularly.

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