# *Clinical Case Reports*

## Risperidone-induced anaemia

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**ABSTRACT**

**Risperidone is an atypical antipsychotic drug, which is used in schizophrenia and also to treat excitation and aggression in patients with delirium. Risperidone has a low risk of haematotoxicity because of its different chemical and pharmacological profile compared to other drugs such as clozapine. Haematological abnormalities have life-threatening complications, especially neutropenia, leucopenia and agranulocytosis, but their effect on erythrocytes in adults is less well known. We highlight the effect of risperidone on erythrocytes and the mechanism that leads to anaemia. To the best of our knowledge, this is the only report of 2 patients showing combinations of mechanisms leading to risperidone-induced anaemia.**

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### INTRODUCTION

Second-generation antipsychotics are considered to be safer compared to first-generation ones due to relatively fewer side-effects. Risperidone, one of the second-generation antipsychotics, is widely used in both adults and children to treat many psychiatric disorders. Although many adverse events due to risperidone are widely documented,[1] haematological side-effects and more specifically effects over erythrocytes in adults are not reported till date. The action of risperidone on iron stores is being reported, but its mechanism is least understood.[2] To the best of our knowledge, this is the first and the only case series showing combinations of mechanisms leading to risperidone-induced anaemia.

### THE CASES

*Case 1*

A 25-year-old man presented with complaints of easy fatiguability for 2 months. The patient was previously diagnosed with a psychotic disorder and was started on risperidone, which he took for about 12 years. The dose of the drug was increased 6 months ago. On examination, he had tachycardia, pallor and icterus. Systemic examination revealed mild splenomegaly, with other systems being unremarkable. On investigation, his haemoglobin was 6.1 g/dl with raised indices such as mean corpuscular volume and peripheral smear, suggestive of macrocytic anaemia. Liver function test revealed unconjugated hyperbilirubinaemia (total bilirubin 4.2 mg/dl, unconjugated bilirubin 2.9 mg/dl) with rise in liver enzymes. On further evaluation, serum vitamin B12 (1000 pg/ml) and folic acid (2.29 ng/ml) concentration was normal, but serum iron concentration was low (26 µg/dl) for which a battery of investigations such as stool for occult blood and upper gastrointestinal endoscopy and other haemolytic profile including direct and indirect Coombs test were negative for haemolysis, hence no cause could be found. G6PD enzyme levels were 13.6 U/g Hb (normal >6.7 U/g Hb). The patient was asked to withhold risperidone, and was started on a haematinic. His symptoms started to improve after a while. He was switched over to amisulpride (100 mg/day) and is on regular follow-up for the past 1 year, showing improvement in his haemogram profile and doing well.

*Case 2*

A 24-year-old man presented to the psychiatry outpatient department with complaints of vomiting, excessive sleep, decreased appetite and hearing of voices. His previous psychiatric history suggested that he was suffering from acute transient psychotic disorder for 6 months and was on risperidone. On examination, his vitals were stable, general examination revealed pallor and systemic examination was unremarkable. On investigation, haemoglobin was 6.8 g/dl, peripheral smear was suggestive of macrocytic anaemia with thrombocytopenia with haemolysis and liver function test showed unconjugated hyperbilirubinaemia (total bilirubin 3 mg/dl, unconjugated bilirubin 2.2 mg/dl). While we looked for the above phenomenon, no past and family history of any haemolytic disorder or any other history suggestive of haemolysis was found. On further evaluation, his serum iron concentration (74 µg/dl) was normal and G6PD enzyme levels were 11.4 U/g Hb (normal >6.7 U/g Hb). Haemolytic profile including direct and indirect Coombs test for haemolysis was done, which came out to be negative, while there was mildly increased osmotic fragility with reticulocytosis, suggestive of drug-induced haemolysis. With the above supportive investigations, risperidone was switched to amisulpride (100 mg/day) and the patient was started on a haematinic. He started to improve gradually after withdrawal of risperidone. The patient is on regular follow-up for the past 6 months and doing well.

### DISCUSSION

Risperidone is an atypical antipsychotic drug approved by the Food and Drug Administration. It is primarily indicated for schizophrenia, bipolar disorder and also used in many other psychiatric conditions such as developmental and disruptive disorders and personality disorders.

Risperidone has its action mainly on serotonergic and dopaminergic receptors. Its antagonistic action on $5HT_{2C}$

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receptor is linked to weight gain, whereas action on $5HT_{2A}$ receptor produces antipsychotic action and is also associated with lower risk of extrapyramidal symptoms. Risperidone has its antagonistic action mainly on D2 receptor family compared to D1 receptor family, leading to diminished dopaminergic activity and hence may cause akathisia, tremors, galactorrhoea, gynaecomastia and reduced bone mineral density. These side-effects are also associated with increased prolactin secretion. This drug also has minor action on alpha adrenergic and histamine (H1) receptors, which explains its sedative action and effects on blood pressure. Many other side-effects such as prolonged corrected QT interval (QTc), diabetes mellitus, hyperlipidaemia, arterial hypertension and posterior reversible cerebral oedema syndrome are also reported.[1]

In comparison to other antipsychotics, risperidone, due to its different chemical structure and pharmacological profile, is considered to have a low risk of haematotoxicity. Haematological adverse effects of psychotropic drugs including atypical antipsychotic medication affects mostly leucocytes[3] and thrombocytes,[4] whereas its effect on erythrocytes in adults is limited and not reported till date. The pathogenesis of risperidone-induced anaemia is not clear. This might be due to multiple mechanisms such as iron deficiency,[5] drug-induced haemolysis and bone marrow depression.

We have reported two patients with risperidone-induced anaemia. In our first patient, anaemia was due to iron deficiency and also some amount of haemolysis as he had unconjugated hyperbilirubinaemia and splenomegaly, which can be linked to the long-term use and escalation of dose of risperidone. Whereas in the second patient, it was due to drug-induced haemolysis and a short duration of use of risperidone. In both instances, the patients showed improvement on withdrawal of risperidone and supplementation with haematinics.

*Conflicts of interest.* None declared

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# Bilateral internal jugular vein ectasia: A rare cause of neck swelling

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## ABSTRACT

**Internal jugular vein ectasia or phlebectasia is a condition in which there is an isolated fusiform dilatation of the internal jugular vein. The patient usually presents with swelling in the neck, which aggravates in size while coughing or straining. This is a rare condition and is often mis-diagnosed. It can be diagnosed by proper history, clinical examination and imaging. We report a 5-year-old boy who had bilateral internal jugular vein ectasia aggravating in size while straining and coughing. Ultrasonography and computed tomography scan showed dilatation of internal jugular veins on both sides. Since the patient was asymptomatic and had no complications, he was advised regular follow-up. This rare benign condition should be kept in mind as a differential diagnosis of an expansile neck mass.**

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## INTRODUCTION

Ectasia or phlebectasia of the internal jugular vein (IJV) is rare and is often misdiagnosed and managed inappropriately. 'Phlebectasia' is a fusiform dilatation of the vein without tortuosity. The term phlebectasia differs from varix which denotes dilatation of the vein along with tortuosity. More than a hundred patients with phlebectasia are reported in the world literature.[1,2] We report a 5-year-old boy with bilateral IJV phlebectasia.

## THE CASE

A 5-year-old boy presented with a painless swelling on the right side of the neck which appeared on coughing and while straining. The patient had no history of trauma, previous neck surgery or pain. There was no history of dysphonia, dyspnoea or dysphagia. Family history was non-contributory. All developmental milestones were found to be appropriate for age.

On examination of the patient in a relaxed state, there was no obvious neck swelling. While the patient was performing Valsalva manoeuvre, the swelling appeared on the right side of the neck. It was fusiform, measuring 3×4 cm in size, palpable on the right anterior border of the sternocleidomastoid muscle extending to the anterior triangle of the neck and clavicle, was soft in

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