CASE REPORT

Ondansetron as a Cause of Transient Blindness: A Case Report

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Abstract

Aim and background: To study transient blindness caused by ondansetron, which is a very rare complication. Ondansetron is a selective 5-hydroxytryptamine receptor (5-HT-3) antagonist. It is a widely used antiemetic and has superior efficacy and safety compared with other antiemetics. However, transient blindness is a very rare complication of both oral and intravenous ondansetron.

Case description: We present a case of a 76-year-old female with ondansetron-induced transient blindness. The cause of transient blindness was established after ruling out other causes with the help of neurological and ophthalmological examination and relevant investigations.

Conclusion and clinical significance: Physicians should be aware of the potential for a rare complication of blindness due to ondansetron, which might confound with other causes of blindness in critical care and perioperatively.

Keywords: Blindness, Case report, Ondansetron, Transient.

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Introduction

Ondansetron, a selective 5-hydroxytryptamine receptor (5-HT-3) antagonist, is a commonly used antiemetic. It is used both for prophylaxis and treatment of nausea and vomiting, especially in perioperative settings and during chemotherapy.[1,2] Its use is also common in intensive care units for drug-induced nausea and vomiting. It has superior efficacy and safety compared to other antiemetics.[2] Though generally safe, it has a number of side effects—common ones include headache, dizziness, musculoskeletal pain, drowsiness,[1,2] or sedation[1] and shivering. The rare ones include chills, loss of vision, delirium,[2] QT prolongation, constipation, and serotonin syndrome.

We present a case report of a 76-year-old female who developed transient loss of vision during her stay in the intensive care unit for cough with expectoration, recurrent hiccups, and respiratory distress.

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Case Description

A 76-year-old female was evaluated for a preanesthetic checkup for bilateral inguinal hernia surgery. She had a history of hypertension, treated with tablet amlong 10 mg, a history of glaucoma under treatment with already lost left eye vision, and a history of chronic obstructive pulmonary disease under treatment. There was no other significant previous history. The patient was found to have dyspnea on exertion, cough with expectoration, hiccups (under treatment with baclofen 25 mg for 10 days), and a slight fall in saturation with slight nausea. She had been admitted to the high dependency unit (HDU) for 1 day. In view of her medical condition, her surgery was deferred, and she was kept admitted in the HDU for further management.

Her blood pressure was 130/80 mm Hg, heart rate was 78/minute, and SpO_2 was 89% on room air, increased to 97% on O_2 by face mask at 2 L/minute. Chest bilateral air entry was reduced at the bases with no added sounds. Cardiovascular examination was normal, and she was hard of hearing. In view of her elderly age, hypertension, and fall in saturation, a cardiology opinion was taken. Both electrocardiogram and echocardiography were normal. The patient was on injection ceftriaxone 1 gm IV twice a day, injection pantoprazole 40 mg IV twice a day, injection ondansetron 4 mg thrice a day, tablet amlodipine 10 mg twice a day, and tablet baclofen 25 mg twice a day.

The patient was improving with the treatment and was mobilized out of bed on the 3rd day of admission. However, on the 4th day of admission, the patient suddenly complained of a loss of vision. Her vitals were normal, and no other abnormality was detected. The patient had been administered intravenous ondansetron 4 mg 10 minutes prior to the onset of blindness. The patient experienced vertigo and sudden blackout, with everything appearing greenish.

A neurology consultation was sought. The neurological examination was normal, other than the blindness. However, the patient was agitated and restless. A provisional diagnosis of encephalopathy/acute psychosis was made. Color Doppler of carotid and vertebral vessels was normal. The electroencephalogram was also normal, as was the noncontrast magnetic resonance imaging (MRI) brain. Other laboratory investigations, such as serum electrolytes, serum calcium, albumin, magnesium, and thyroid stimulating hormone (TSH), were normal.

An ophthalmological consultation was also sought. Her left eye had absolute glaucoma with complete loss of vision for many years, and MRI revealed a left eye posterior staphyloma. The right eye examination revealed a normal anterior chamber. There was pseudophakia, and the patient had already undergone trabecular surgery. The pupil was irregular with sluggish reaction. The optic disk revealed some pallor. However, there was no vitreous hemorrhage or degenerative changes in the retina. Intraocular pressure was 17 mm.

Considering the chances of ondansetron-induced blindness, administration of ondansetron was stopped immediately following the development of blindness. The vision returned to baseline within 20 hours, and a diagnosis of ondansetron-induced transient blindness was made.

Discussion

Ondansetron is a widely used antiemetic for prophylaxis and treatment of nausea and vomiting in a number of situations, including perioperative, chemotherapy, radiotherapy, and intensive care units. It is metabolized in the liver and excreted in the kidneys and has a half-life of 5–7 hours.[2]

Blindness during admission to intensive care units can be due to a number of causes. These include hypotension, intracranial hemorrhage or ischemia, optic neuropathy, prone positioning causing pressure on the globe, acute glaucoma or exacerbation of chronic glaucoma, and retinal artery and vein occlusion.

Our patient had no hypotensive episodes and had not been placed in a prone position. Hence, pressure on the globe and hypotensive changes were ruled out as causes of blindness.

The patient had undergone extensive neurological examination, including MRI of the brain and Doppler examination of carotid vessels. Hence, transient ischemic attacks, intracranial hemorrhage, and intracranial ischemia were ruled out as causes. The patient also underwent extensive ophthalmologic evaluation, and hence, acute exacerbation of glaucoma, retinal artery, and vein occlusion were also ruled out as causes of sudden loss of vision.

Encephalopathy/acute psychosis was also ruled out since the patient returned to baseline in <24 hours.

Thus, having ruled out the other causes, blindness induced by ondansetron was a diagnosis of exclusion. The temporal association with the last dose given, as well as rapid recovery, supported the diagnosis.

Transient blindness following large doses of ondansetron given as a bolus (32 mg over 15 minutes) has been reported in patients undergoing chemotherapy.[3] Transient blindness was also reported by Cherian and Maguire in a 36-year-old patient due to postoperative use of ondansetron.[1] Barus et al. also reported blindness after intravenous or oral administration of ondansetron.[4]

The mechanism by which ondansetron causes blindness is unknown.[1,2] The 5-HT3A subgroup receptor modulates the signaling pathway of the retina. Ondansetron does act on these receptors.[1,2] Babi et al. reported a case of posterior reversible encephalopathy syndrome (PRES) complicated by intracerebral hemorrhage and refractory intracranial hypertension as a complication of ondansetron.[5] They had postulated that prolonged antagonism of 5-HT3 receptors (from consistent to massive administration of ondansetron) at the micro- and macrovascular levels might be toxic to vascular endothelium, causing capillary leakage and disruption of the blood-brain barrier, resulting in vasogenic cerebral edema.[5]

Conclusion

Physicians should be aware of the potential for a rare complication of blindness due to ondansetron, which might confound with other causes of blindness in critical care and perioperatively. Additionally, the administration of unnecessary medications should be avoided to prevent such iatrogenic incidents.

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