Case Report

Sudden Unilateral Loss of Vision in a Young Girl: A Case Report

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Abstract: A casual association seems to exist between the onset of intracranial hypertension (IH) and growth hormone (GH) therapy. Children receiving GH treatment, if they complain of headaches or visual difficulties, should undergo detailed ophthalmological evaluation. Herein, we report a young girl who was receiving GH treatment for idiopathic short stature, and due to the adverse effects of GH, she developed intracranial hypertension, as a result of which, she developed unilateral blindness.

Keywords: Growth hormone, Short stature, Elevated intracranial pressure, Papilledema, Blindness, Brain tumors.

INTRODUCTION

Intracranial hypertension (IH) is a condition defined as elevated intracranial pressure in the absence of clinical, laboratorial, or radiological evidence of infection, vascular malformation in the brain, hydrocephalus, or brain tumors. Intracranial hypertension can be classified as primary or secondary, with the secondary type being more common, accounting for 53-77% of children [1, 2]. Growth hormone (GH) has been approved by FDA to be used in children for various disorders, including short stature. Treatment with GH in children is considered safe, and adverse effects are not very common. However, GH can affect the eyes by two main mechanisms: myopia and elevation of intracranial pressure. Intracranial hypertension is rare, occurring in about 1/1000 children treated with GH. It is a serious condition, because it can lead to papilledema, and eventually, vision loss. Symptomatic elevated intracranial pressure was first described by the FDA in 1993 [3, 4].

Here, we report the case of a young girl who developed intracranial hypertension, resulting in unilateral blindness, while receiving GH treatment.

CASE PRESENTATION

A 12-year-old girl presented to our paediatric emergency department with a three-day history of sudden vision loss in her left eye, accompanied by a moderate to severe headache, and vomiting for the past two weeks. These symptoms were not associated with others such as fever, eye pain, redness, seizures, or encephalopathy. She has been receiving GH treatment for idiopathic short stature for the last one year.

She was immediately evaluated by an ophthalmologist, who reported visual acuity 20/18 in the right eye and 20/300 in the left eye. Intraocular pressure was normal in both eyes. The movements of both eyes were also normal in all directions without any discomfort, or pain. Both pupils were reactive to light equally and depth of the anterior chamber of the eye was also clear. The fundoscopic examination revealed round transparent vitreous cavities in both eyes, bilateral grade 3+ papilledema, and absent vessel pulsations with tortuous retinal vessels.

10 mL of CSF was drained following a lumbar puncture. Elevated CSF pressure of 38 cmH2O was revealed, which decreased to 24 cmH2O after the procedure. The MRI of the brain was reported as normal, without ventriculomegaly, no haemorrhage, and no SOL. Axial T1 contrast and Axial T2 FLAIR-weighted images showed widening of optic nerve sheath, and hyperintense signals bilaterally (Figs. **1**, **2**). Therefore, a diagnosis of growth hormone-induced intracranial hypertension was made, and treatment with acetazolamide (30 mg/kg/day) was initiated. After six weeks of treatment, she was re-evaluated by an ophthalmologist. Her visual acuity improved to 20/20 in the right eye, and 20/18 in the left eye, and the papilledema resolved completely.

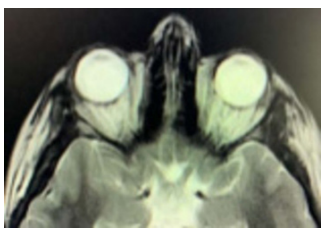

Fig. 1. Axial T2 FLAIR.

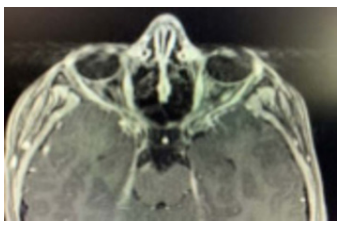

Fig. 2. Axial T1 with Contrast.

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DISCUSSION

One of the adverse effects of GH is elevation of intracranial pressure, although the exact mechanism is not well understood. One hypothesis suggests that the physiological antidiuretic effect of GH leads to the retention of sodium and water, which results in augmentation of blood volume, along with a reduction in CSF absorption by the arachnoid villi. Another hypothesis proposes that GH crosses the blood-brain barrier, leading to increased levels of GH and insulin-like growth factor 1, which ultimately results in increased CSF production [5, 6].

The first report of GH induced intracranial hypertension was published in 1992 by Horm Res. Later in 1995, Malozowski described 22 paediatric patients, identified by the FDA database, who had been treated with GH and developed intracranial hypertension [7].

In another case reported in 1997 from Saudi Arabia, that a patient with hypopituitarism was treated with GH and developed intracranial hypertension [8].

We also report a young girl presenting with symptoms of unilateral vision loss, headache, and vomiting, similar to those in already published reports. She was being treated with GH for idiopathic short stature for the last one year, and she developed clinical symptoms of intracranial hypertension. Her clinical symptoms, along with the CSF opening pressure and radiological findings, were consistent with a diagnosis of intracranial hypertension. The frequency of adverse effects of GH treatment on vision varies; while they may go unnoticed in some patients, in infrequent cases, they can lead to blindness. Headache and vomiting are commonly associated with intracranial hypertension [9, 10].

Our patient improved after six weeks of treatment with acetazolamide and discontinuation of GH therapy. Intracranial hypertension as an adverse effect of GH treatment can occur either early or late in the course of therapy and may affect vision. So, any patient receiving GH treatment must also undergo regular ophthalmological examinations to facilitate the prompt diagnosis of intracranial hypertension, which can result in visual complications. Early intervention is essential to prevent blindness.

CONCLUSION

Treatment with GH can result in serious complications of vision due to intracranial hypertension. Intracranial hypertension, whether primary or secondary, is no longer reported as a rare disorder. It is of paramount importance to have a high index of suspicion of intracranial hypertension in children receiving GH, who present with visual problems, headache and vomiting. These patients immediately undergo ophthalmological evaluation and treatment for intracranial hypertension to prevent permanent vision loss.

CONFLICT OF INTEREST

Declared none.

ACKNOWLEDGEMENTS

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