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Case Report

Idiopathic Intracranial Hypertension: Neuropsychiatric Systemic Lupus Erythematosus or Gonadotropin-releasing hormone agonist side effect?

Abstract

A 31-year-old systemic lupus erythematotus (SLE) patient presented with headache and blurring of vision. Prior to this, she received 2 doses of monthly triptorelin for endometriosis. On examination, she had bilateral sixth nerve paresis. The diagnosis of idiopthic intracranial hypertension (IIH) was confirmed by an increased intracranial pressure and normal neuroimaging studies of the brain. After releasing the cerebrospinal pressure and cessation of triptorelin, the clinical symptoms resolved without further treatment. It is important to identify the drugs causing IIH rather than attribute to neuropsychiatric SLE to prevent unnecessary treatment.

Introduction

Idiopathic intracranial hypertension (IIH) is one of the rare presentation of neuropsychiatric systemic lupus erythematosus (SLE) and may present with unremitting headache [1]. IIH also known as pseudotumor cerebri or benign intracranial hypertension, can be presentations of neuropsychiatric SLE or an adverse events of drugs such as Gonadotropin-releasing hormone (GnRH) analogues.

Here, we report a SLE patient who developed IIH after treated with triptorelin, a synthetic GnRH analogue for endometriosis.

Case Report

A 31 year old lady presented with headache and blurring of vision for 3 days. She had double visions on lateral view of both eyes. There was no fever, joint pain, rashes, oral ulcers or dyspnoea. Two years earlier, she was diagnosed to have endometriosis after suffering from dysmenorrhea for more than 5 years. After failure of hormone pills, she was started on monthly triptorelin and had her second injection 2 weeks before this presentations.

Upon examination, she was not obese with blood pressure 110/75 mm Hg and temperature of 37 ^{o}C. Extraocular movements demonstrated bilateral sixth nerve paresis and fundoscopy didn't reveal papilledema. The other physical examinations were otherwise normal.

Laboratory examinations on admission were unremarkable. MRI (Magnetic resonance Imaging) brain done showed no intracranial mass or ventricular dilatation. The MR venogram disclosed no evidence of dural venous thrombosis.

Lumbar puncture was urgently performed which showed an opening pressure of 25 cm H_2O with patient lying on the side. Cerebrospinal fluid (CSF) was drained and analysis was normal. The closing pressure was 19 cm H_2O.Based on these findings, the patient was diagnosed with IIH and triptorelin was discontinued. One day after the lumbar puncture, her headache improved tremendously without any further treatment. A week later, she was seen in outpatient clinic, both her diplopia and headache resolved completely.

Discussion

Diagnosis of IIH is based on the modified Dandy criteria [2]. This patient fulfilled all these criteria which include symptoms and signs of intracranial pressure, no neurological examination except VI nerve palsy, normal neurodiagnostic studies, normal CSF analysis, increased CSF pressure and no abnormality in consciousness level.

IIH was first reported in SLE patient by Bettman el al in 1968 [3]. Since then it had been recognized as a syndrome of neuropsychiatric SLE and one of the causes of headache in SLE. The pathogenetic mechanism is not clear. Probable mechanisms include immune-mediated injury within the archnoid villi

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leading to decrease CSF absorption and/ or thrombotic obliteration of cerebral arteriolar and venous systems due to a hypercoagulable state [4-6]. IIH is an uncommon presentation of neuropsychiatric SLE but with favourable outcome [7]. High dose oral corticosteroids remains the main stay for treatment of IIH in patients with SLE while intravenous mannitol or acetazolamide can be used to reduce intracranial tension [8].

GnRH analogues was reported rarely to cause IIH in adult patient's [9,10]. This patient was given 2 doses of monthly triptorelin 3.75mg for endometriosis before she developed headache and diplopia. In view of this, we attributed the cause for IIH was GnRH analogues rather than presentations of neuropsychiatric SLE. The treatment for both is rather different. For GnRH analogues induced IIH, omitting the culprit drugs and normalizing the CSF pressure, clinical symptoms will improve as clearly seen in this case [11]. To our knowledge, this is the first GnRH analogues induced IIH in adult SLE patients reported.

Conclusion

In SLE patients, it is important to identify the potential drugs that might cause IIH rather than attribute it as neuropsychiatric SLE presentations as this would prevent patient from getting unnecessary treatment such as high dose steroids.

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