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General Medicine

BILATERAL ABDUCENS NERVE PALSY DUE TO IDIOPATHIC INTRACRANIAL HYPERTENSION AS AN INITIAL MANIFESTATION OF SYSTEMIC LUPUS ERYTHEMATOSUS

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KEYWORDS :

INTRODUCTION

Idiopathic intracranial hypertension (IIH) is characterized by symptoms of elevated intracranial pressure, elevated cerebrospinal fluid (CSF) pressure, normal CSF content, and normal neuroimaging studies. Although IIH generally emerges in overweight women of childbearing age, it is known that IIH is also induced by medication, infection, or medical disorders such as endocrinological disease, vitamin deficiency, and autoimmune diseases. We herein present a case of IIH as the onset of systemic lupus erythematosus (SLE) with bilateral abducens nerve palsy without headache or nausea.

MATERIALS AND METHODS

We report a rare case of Bilateral Abducens Nerve Palsy due to Idiopathic Intracranial Hypertension as an Initial Manifestation of Systemic Lupus Erythematosus

Case Study

A 14-year-old girl was admitted to a local hospital with a high fever that had lasted for a few days in September 2023. She had been treated for asthma and allergic rhinitis, and she had not suffered from any asthma attacks for several years. At the first visit, the patient was tentatively diagnosed with a viral infection, and her fever subsided with palliative care. However, the subsequent laboratory data revealed leukopenia, proteinuria, microscopic hematuria, positive anti-nuclear antibodies (ANA) and an increased serum level of anti-double-stranded DNA (dsDNA) antibodies. Ten days after thisadmission, the patient became aware of diplopia and was referred to our hospital for a further examination for ophthalmological abnormalities. She was not receiving any medications, such as corticosteroids or antibiotics, or any supplements, including vitamin A, which are known to be risk factors for IIH. She did not complain of either headache or nausea. Upon changing hospitals, an examination of her vital signs demonstrated no abnormalities except for slight diastolic hypertension (125/92 mmHg). Her height was 155.7 cm, her body weight was 54.3 kg and her body mass index was 22.4 kg/m2, indicating that she was not obese. She had a malar rash and palmar erythema, and her palpebral conjunctiva was anaemic. She was alert with no signs of meningeal irritation. On neurological examination, the ab duction of both eyes was insufficient (left; 3/5, right 4/5) and became exacerbated while she was looking to the left side. Her pupils were symmetrical and reactive to light. She had no other cranial neuropathy, no motor or sensory neuron disorders, and no pathological reflexes. Ophthalmological examinations demonstrated papilledema during both distal and proximal sight, indicative of bilateral incomplete abducens nerve palsy with papilledema.

Investigations

Routine blood investigations were normal. The laboratory findings showed pancytopenia with a high titre of anti-dsDNA anti bodies and hypocomplementemia. The anti-cardiolipin anti bodies were slightly positive, whereas lupus anticoagulant and anti-β2GPI antibodies were negative. The prothrombin time (11.6 sec) and activated partial thromboplastin time (27.5 sec) were normal. The D- dimer level was slightly elevated (2.5>0.9 μg/mL); however, there were no signs of thrombotic or haemorrhagic activity. A urinalysis revealed proteinuria and haematuria with active cellular casts. The viral specific antibody profile revealed no evidence of active viral infection. Brain magnetic resonance imaging (MRI), MR angiography and plain computed tomography (CT) showed no findings of ischemia, thrombosis or vasculitis, while a CSF analysis revealed an extremely high opening pressure (320 mmH2O) with normal CSF indices.

DISCUSSION

IIH is defined by the modified Dandy criteria as follows: symptoms and signs of increased intracranial pressure; no localized neurologic signs, except for unilateral or bilateral 6th nerve palsies; increased CSF opening pressure >200mm H2O, but normal CSF composition; no evidence of hydrocephalus, mass, structural ,or vascular lesions on imaging; and no other cause of increased intracranial pressure(ICP) identified. Corticosteroids and immunosuppressive agents were effective for the IIH in the present case. Most of the IIH in SLE cases can be treated using high-dose corticosteroids followed by immunosuppressants, which differs from IIH cases in general ,which are treated using acetazolamide or mannitol and weight loss.

CONCLUSION

In conclusion, we experienced a case of SLE with bilateral abducens nerve palsies due to IIH. The presence of IIH in SLE patients is rare, but this potential occurrence should not be overlooked.

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