

PSEUDOTUMOR CEREBRI: A Case Report

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Abstract

Pseudotumour cerebri is a neurological disorder that is characterized by disorder of elevated intracranial pressure without any evidence of localizing signs or neurological deficits with normal intracranial structures. Diagnosis is primarily clinical and requires radiographic exclusion of an intracranial mass.

Keywords: Pseudotumor Cerebri, Intracranial pressure, Nalidixic Acid, Acetazolamide

We reported a case of Pseudotumour cerebri in four and half year male infant presented with acute history of low grade fever and deviation of eye towards medial side for two days. History revealed recurrent episodes of diarrhea, treated with nalidixic acid by local practitioner. Neurological examination was normal except the deviation of right eye towards the medial side. Diagnosis was established by MRI head which revealed pseudotumour cerebri as blood and CSF values are within normal limits. Infant was treated with acetazolamide and was followed. He improved with conservative management and convergent squint was resolved at the time of discharge

Introduction

Pseudotumor cerebri is a neurological disorder that is characterized by disorder of elevated intracranial pressure without any evidence of infection, vascular abnormality, space occupying lesion, hydrocephalous or alteration of consciousness^{1,2}.

The concept of raised intracranial pressure in the absence of space occupying lesion was first introduced by Nonne^{3,4} as Pseudotumor cerebri (PTC) and subsequently termed "benign intracranial hypertension" by Foley in 1955.⁵ The absence of a identifiable etiology for a clinical syndrome characterized by elevated intracranial pressure exists in nearly 90% of cases and this ambiguity has led to replacement of benign intracranial hypertension with idiopathic intracranial hypertension in light of vision loss resulting from this condition⁶.

Case report

A four months old male baby, born of nonconsanguinous marriage, was brought with the complaints of low grade fever and deviation of right eye towards medial side for two days. History revealed that child had six to eight episodes of foul smelling loose stools and was treated with nalidixic acid given in a dose on 300mg 8 hourly (900mg/day), as prescribed by a local practitioner. There was no associated rash, ear discharge, seizures, any trauma, refusal to feed or vomiting.

Birth history revealed that he was born to 27 years old mother by normal vaginal delivery with no history of birth asphyxia or hyperbilirubinemia. He is immunized according to the age and his milestones are normal.

On general physical examination, baby was conscious and playful but at times irritable. Patient was afebrile with heart rate 120/min, regular, good volume and respiratory rate 38/min regular, weighing 6.4 kg with anterior fontanel at level. Oxygen saturation was 96% on room air and all peripheral pulses were palpable. Respiratory examination was normal and abdominal examination did not reveal any abdominal mass or organomegaly. On examination central nervous examination was within normal limits except for the medial deviation of right eye resulting in convergent squint in right eye.

The child was admitted and his general condition improved with symptomatic treatment given. Hydration was maintained and breast feeding started subsequently.

Fundus examination was normal and there was no papilloedma.

Lumbar puncture was done to rule out meningitis. The CSF pressure was raised, cells were normal, four lymphocytes with glucose 56 mg/dl and proteins 36 mg/dl, Complete blood count was within normal limits.

It was presumed that offending agent was nalidixic acid so it was discontinued and symptomatic treatment was started. Infant was started with Zinc sulphate solution with ORS and was kept under observation for 48 hours with regular blood pressure monitoring. MRI brain was done on the day of admission and was normal. Taking into accounts the history, clinical findings and MRI brain, a diagnosis of pseudotumor cerebri was made. On day 2 of admission tab Acetazolamide was started. The infant responded to the treatment and convergent squint of right eye disappeared on 4th day of admission. The child was discharged with advice for continuing exclusive breast feeding for 6 months and to follow up. On follow up, ophthalmological examination showed no deficit.



Figure 1

Discussion

Pseudotumor cerebri is a condition of intracranial hypertension without localizing signs or neurological deficits except for papilloedema and normal CSF constituents with normal intracranial structure¹. The Pseudotumour cerebri may be primary (idiopathic intracranial hypertension) or arise from a detectable secondary cause.

There are many medications which can cause PTC and they are cyclosporine, minocycline, nalidixic acid, tamoxifen, tetracycline, hypervitaminosis A & D. Hormones such as recombinant human growth hormone, thyroxine (in children), leuprolin acetate, levonorgesterol implant, anabolic steroids and starting or withdrawal from chronic steroids can cause PTC⁷. The presenting symptoms of benign intracranial hypertension are known to vary with age as diagnosis in younger infant is challenging as they may only have irritability or apathy^{8,9}.

The infant in this case had history of nalidixic acid for the treatment of diarrhea and unilateral 6th nerve palsy of nontraumatic origin and not associated with papilloedema, as mentioned in similar studies¹⁰. The recommended dose of nalidixic acid is 55mg/kg in 3 divided doses in the treatment for diarrhea, if overdose occurs then this drug can result in pseudotumour cerebri¹¹ as in this case. The first step in the treatment of pseudotumour cerebri involves the discontinuation of possible offending medication or hormonal preparation used. Intracranial hypertension is managed medically and surgically. Therapy with acetazolamide was an effective first line method of treating raised ICT in children with pseudotumour cerebri¹². A lumbar puncture can also help to relieve CSF pressure in brain and prevent visual problems. In our case lumbar puncture was suggestive of raised intracranial hypertension and unilateral 6th nerve palsy of the child responded to acetazolamide therapy within 48hrs.

In some cases CSF shunting may be employed if medical treatment fails¹⁰. It is found that immediate intervention with no papilloedema in pseudotumour cerebri patients can prevent visual defects and can be monitored on follow up¹¹.

Conclusion

This case study stated that awareness of cause of pseudotumour cerebri is very important because prognosis of pseudotumour cerebri is excellent, if diagnosed early and correctly. It is a diagnosis of exclusion.

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