

Case Report

Child with recurrent meningitis: Encephalocele causing anterior cranial fossa defect with CSF rhinorrhoea

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Abstract

Recurrent meningitis in children is potentially life threatening, causing long term morbidities and psychological trauma to the patient through the repeated hospital admission and multiple invasive investigations.

Immune deficiency and bacterial migration along congenital or acquired pathways connecting CSF pathways to external surfaces are the two important aetiologies which should be taken into consideration¹.

We report a case of a boy who presented with recurrent meningitis due to defect in the anterior cranial fossa ethmoidal air cells.

(Key Words : CSF rhinorrhoea, recurrent meningitis)

Introduction

The quest for evidence of CSF leakage in a young child with recurrent meningitis should start with thorough history including previous head trauma, procedures involving ear, sinuses, and recurrent sinusitis or otitis media. Complete physical examination including other anatomical abnormalities, autoscopy and audiological evaluation for sensorineural hearing loss. (3)

The bacterial specificity could leave significant clue of the aetiology: Pneumococcal or haemophilus suggest cranial dural defect, E-coli or other gram negative bacilli suggest spinal dural defects and meningococcal suggest immunologic deficiency albeit it rarely cause recurrent meningitis without recurrent infection in non-CNS sites. (1)

Case report

A ten year old boy was presented with second episode of meningoencephalitis, manifested as high spike fever for 4 days, severe fronto-temporal headache, vomiting and altered behaviour for 1 day.

He is the second child of non-consanguineous parents, delivered by elective caesarion section at term for pregnancy induced hypertension

and gestational diabetes. antenatal or postnatal complications. Neuro development was appropriate for age and he received all the immunizations according to Expanded Program of Immunization of Sri Lanka. He didn't have any history of recurrent infections except one episode of meningoencephalitis at his seven years of age.

At 10 years he presented with fever, recurrent convulsion and drowsiness. CSF studies suggestive of pyogenic meningitis and CECT brain showed 2×1.8cm cystic lesion in the anterior fossa suggestive of arachidonic cyst.

First episode was complicated with left side foot drop, noticed from day 12 of illness and it necessitated further brain imaging. It was managed as post meningoencephalitic UMN foot drop probably due to precentral gyral infarction and it resolved completely with physiotherapy.

Evaluation of second episode revealed the history of intermittent watery nasal discharge from his early childhood suggestive of CSF rhinorrhoea. However the last episode of CSF leakage was about 3 weeks before the illness and he was in remission of these symptoms throughout the hospital stay. He has neither head injuries nor recurrent ear infections in the past.

Clinical examination was normal without any facial dysmorphism or neurological weakness

CECT brain showed a well margined 1.9cm × 1cm size lesion in the anterior cranial fossa with few rim calcification suggestive of dermoid cyst in anterior cranial fossa associated possible CSF leak. *Streptococcal pneumoniae* was isolated from both the blood and CSF.

He was treated with IV antibiotic according to the antibiotic sensitivity and fever settled on 15th day of illness.

Discussion with neurology, radiology and ENT teams suggested the possibility of deficient anterior cranial fossa due to erosive cyst which need further

evaluation with MRI, CSF cisternogram, long term prophylaxis and repair of anterior cranial fossa defect. He was transferred to LRH where MRI brain was taken and it showed anterior cranial fossa cyst with defect in ethmoid air cell with evidence of CSF leak.

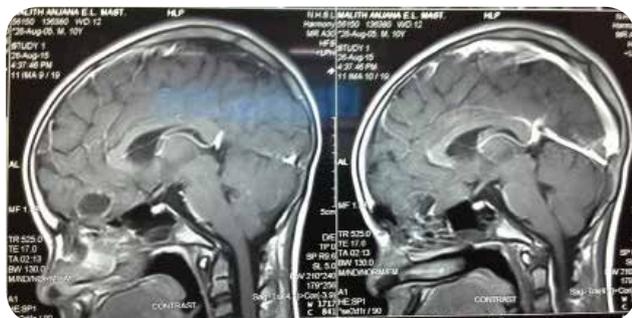


Figure 1; MRI brain showing cystic lesion communicating with ethmoidal air cells with subdural empyema in anterior cranial fossa.

He was given with 23 valent polysaccharide pneumococcal vaccine, Hib vaccine and started with oral penicillin prophylaxis.

He was referred to ENT surgeon at LRH and undergone endonasal endoscopic evaluation. It showed small encephalocele (0.5cm × 1cm) in the cribriform between the septum and right middle turbinate. He has undergone repair of CSF leakage by dural grafting with reduction of encephalocele size.

He is symptom free now and on regular follow up.

Discussion

A second episode of meningitis considered as a recurrence if it is resulting from a different bacterial pathogen than the first or resulting from the same organism but occurs more than three weeks after completion of therapy. (1)

Etiological studies on recurrent meningitis reveals a congenital CSF fistula in 55% cases, traumatic surgical fistula in 17% and immune deficiency in 21%. (2)

In our patient, the absence of other systemic involvement and history of watery nasal discharge makes the diagnosis conspicuous. However in most patients with recurrent meningitis, mild CSF

leakage often escapes the patients' attention as it is indistinguishable from nasal discharge and often intermittent. (1)

Traditional testing for glucose content of nasal discharge or middle ear fluids should be replaced by testing for $\beta 2$ transferrin or β trace protein⁴. In our patient we were unable to check that as he was free of nasal discharge from admission. Although CT scan may demonstrate a bony defect and suggest site of CSF leakage, the additional technique of CT cisternography is currently most reliable. (5)

The endoscopic endonasal repair of CSF fistula has become a routine approach. It is both safe and effective with a success rate of 98%. (6)

It is important to immunise the patient to prevent recurrences in this child. Unconjugated pneumococcal vaccine confers capsular specific immunity against 23 sero types in immunologically competent individuals and is the current international recommendation to prevent invasive pneumococcal infection in children more than 5 years of age⁴. However it is thought that organism bypass the circulating serum antibodies by direct invasion of the meninges from nasopharynx. (7)

Life-long benzathine penicillin prophylaxis at three weekly intervals to achieve higher drug levels is recommended to prevent invasive infection. (8)

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