Case Report

Late onset hydrocephalus in children with tuberculous meningitis

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Abstract

Hydrocephalus is a known complication of tuberculous meningitis (TBM). It is almost always present in patients who have had the disease for four to six weeks. However, hydrocephalus can also develop later in the disease course as seen in our 3 patients. All 3 patients had multi-drug resistant (MDR) tuberculosis (TB) and developed hydrocephalus after variable time after starting second line anti-tuberculous therapy (ATT). A 7 years old girl had hydrocephalus at onset of TBM and was shunted but the hydrocephalus increased in size after 6 months of being on second line ATT in spite of a patent ventricular peritoneal (VP) shunt. Hydrocephalus responded to oral acetazolamide. Other 2 patients, a 2 years old girl and 3½ years old boy developed hydrocephalus after being on treatment for 14 months. Both required insertion of VP shunt. Thus, in patients with MDR-TB, hydrocephalus may develop as late onset phenomenon and a neurological examination would be essential in each visit to the hospital.

Keywords: Hydrocephalus 1 year after anti-Koch's treatment, hydrocephalus in pediatric tuberculous meningitis patients, late onset hydrocephalus, multidrug-resistant tuberculosis, tuberculous meningitis in children

Introduction

A large number of tuberculous meningitis (TBM) patients develop hydrocephalus. It is almost always present in patients who have had the disease for 1–6 weeks.[1-3] However, hydrocephalus can also be a late manifestation. We present three children with multidrug-resistant (MDR) tuberculosis (TB) who developed hydrocephalus after 6–14 months of therapy.

Case Reports

Case 1

A 7-year-old girl was diagnosed to have TBM in February 2011. Cerebrospinal fluid (CSF) TB culture grew mycobacterium TB (MTB). She was started on antituberculous therapy (ATT) and underwent ventriculoperitoneal (VP) shunt surgery. In June 2011, her drug sensitivity testing showed resistance to isoniazid (H), rifampicin (R), ethambutol (E), streptomycin (S), ofloxacin (Ofx), ethionamide (Eto), and moxifloxacin (Mfx). She was shifted to clarithromycin (Clr), clofazimine (Cfz), para-aminosalicylic acid (PAS), linezolid (Lzd), cycloserine (Cys), amikacin (Amk), and pyrazinamide (Z). In January 2012, she presented with projectile vomiting for 10 days and early morning headache. Magnetic resonance imaging (MRI) brain showed increasing obstructive hydrocephalus with *in situ* VP shunt. She was started on acetazolamide to which she responded. She is currently asymptomatic and has completed 17 months on ATT.

Case 2

A 2-year-old female child was diagnosed as pulmonary TB in August 2011 with the presence of acid-fast *Bacilli* (AFB) on sputum smear. She was started on first-line ATT but was not compliant to therapy. In September 2011, she had a focal

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convulsion and left-sided hemiparesis. MRI brain showed multifocal acute to subacute infarcts due to TB vasculitis without hydrocephalus. Her CSF grew MTB which was resistant to HRES. In October 2011, she was shifted to second-line ATT consisting of Amk + Mfx + PAS + Cys + Cfz. In December 2012, she developed behavior changes with head banging and vomiting. Computed tomography brain showed dilated ventricles with periventricular ooze. She underwent a VP shunt. Her ATT was stopped in April 2013 as MRI brain was normal.

Case 3

A 3½-year-old male child was diagnosed in May 2011 to have left-sided pulmonary TB and his gastric lavage showed the presence of AFB on smear. He was started on first-line ATT. TB culture grew MTB resistant to HRZES, Mfx. He was started on Amk + Eto + PAS + Cys + Cfz in June 2011. In August 2012, he had headache and vomiting. MRI brain showed obstruction of 4th ventricle outflow suggestive of obstructive hydrocephalus with small right basal ganglia infarct. A VP shunt was done in the child. He is on regular follow-up and completed treatment after 18 months of second-line ATT.

Discussion

Hydrocephalus in TBM could be either communicating or obstructive.[4] The main cause is inflammatory exudates occupying the subarachnoid spaces or the ventricular pathways. In the earlier stages of the disease, the thick gelatinous exudates block the subarachnoid spaces in the base of the brain leading to communicating hydrocephalus. The exudates lead to a dense scarring of the subarachnoid spaces in the later stages of the disease again leading to communicating type of hydrocephalus. It can also result from the exudates blocking the arachnoid granulations which prevent the absorption of CSF. The inflammation of the choroid plexus and ependyma also leads to an overproduction of CSF in acute phase. Obstructive type develops when the fourth ventricular outlets are blocked by the exudates or leptomeningeal scar tissue or when there is obstruction of the aqueduct either due to a strangulation of the brain stem by exudates or by a subependymal tuberculoma.[5]

Incidence of hydrocephalus is very high in children. It has been reported in 87%–90% of children with TBM, whereas it is seen in about 12% of adults.[5,6] Hydrocephalus can occur as a presenting feature of the disease or might occur later in the clinical course.[7] However, majority of cases report early onset of hydrocephalus.[3] A retrospective study of 214 pediatric cases of TBM reported hydrocephalus as a presenting feature in 80% of the patients.[8] In our cases, all the three patients developed late onset hydrocephalus. Although the first patient had hydrocephalus initially, his hydrocephalus worsened later on during the disease despite a functioning shunt. All three had MDR TB. Late onset hydrocephalus has not been documented yet. There is no proved pathogenesis of hydrocephalus occurring as late as 1 year after the commencement of ATT. Since all three were MDR TBM cases, it can be hypothesized that the disease being resistant to first-line TB drugs, had not been completely cured, and was still progressing despite therapy.

Conclusion

Hydrocephalus can occur anytime during TBM irrespective of the commencement of drugs. Especially in the cases of MDR TBM, it can occur even after a year of ATT. Hence, there is a need for regular clinical follow-up to rule out the development of hydrocephalus, during the early or late course of the disease.

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Conflicts of interest

There are no conflicts of interest.

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