Infantile Tuberculous Meningitis Complicated by West Syndrome

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ABSTRACT

Tuberculous meningitis rarely causes refractory epilepsy and is a rare cause of West syndrome. We describe three infants, who had tuberculous meningitis complicated by stroke and paradoxical tuberculoma, and, who later developed West syndrome.

KEYWORDS: Chronic meningitis, infantile spasm, meningeal tuberculosis, paradoxical tuberculoma, refractory epilepsy

INTRODUCTION

West syndrome is catastrophic epilepsy of infancy and childhood characterized by a triad of epileptic spasms, hypsarrythmia, and developmental retardation.[1] Tuberculous meningitis (TBM) is a rare cause of West syndrome.[2,3] Timely diagnosis and early initiation of treatment are crucial to morbidity and mortality in both TBM and West syndrome. We describe three infants with TBM and West syndrome.

CASE DESCRIPTIONS

Out of 289 children with TBM in our institute from January 2011 to August 2015, three children developed West syndrome. The diagnosis of TBM was based on the clinical symptoms suggestive of TBM (fever >5 days with or without headache, vomiting, focal deficits and/or encephalopathy, suggestive neuroradiology for TBM, suggestive cerebrospinal fluid, and/or evidence of extracranial tuberculosis). The children with West syndrome were treated with corticosteroids and followed up for outcome assessment [Table 1]. All children had basal exudates and hydrocephalus at diagnosis. Cerebrospinal fluid analysis of all three children was consistent with TBM. However, acid-fast bacilli were not isolated in any children. These children developed epileptic spasms after 3–14 months of TBM diagnosis. All three children had developmental delay and epilepsy on follow-up.

DISCUSSION

These cases highlight that epileptic spasms can complicate the course of TBM. The latency of epileptic spasms varies from 4 to 24 months following the neurological insult.[4] This latency depends on various factors such as severity of brain injury and age at insult. The initial insult results in pathological processes such as gliosis and abnormal synaptic reorganization. Infantile TBM is frequently complicated by progressive hydrocephalus and secondary neuronal injury, progressive meningovasculitis leading to infarcts, and paradoxical tuberculoma as in index cases, which contribute to epileptogenesis in TBM.[5]

There are only few reports of epileptic spasms in TBM. A 10-month-old girl with focal infantile spasm due to underlying cerebral tuberculoma has been described.[2] Zorn-Olexa et al. [3] described a child with TBM (with tuberculoma) who initially presented with refractory status epilepticus and subsequently refractory infantile spasm. The etiology was multifactorial with infarcts, tuberculomas, and paradoxical tuberculomas. Watanabe et al. [6] described a 10-month-old infant with TBM who presented with status epilepticus and developed spasms 3 months later. Thus, these cases highlight the multifactorial origin wherein stroke, porencephaly, and paradoxical tuberculoma contribute to refractory epilepsy in TBM.

Refractory epilepsy is an uncommon complication with TBM. In a series, 10% of children had epilepsy, 75% of seizures in TBM were controlled with only one antiepileptic drug, and 25% required two antiepileptic

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How to cite this article: Dhawan SR, Sahu JK, Sankhyan N, Vyas S, Singhi PD. Infantile tuberculous meningitis complicated by West syndrome. J Pediatr Neurosci 2018;13:237-40.
Epileptic spasms, as in our report, may be a marker for poorer developmental outcomes and possibly, for refractory epilepsy in children recovering from TBM.

**CONCLUSION**

West syndrome can rarely be seen in children recovering from TBM. Its occurrence may be a marker for poorer developmental outcomes and possibly, for refractory epilepsy in these children.

**Acknowledgements**

Author contribution: SD, JS, NS, and PDS were involved in patient management and preparation of the draft. SV interpreted radiological data and approved the manuscript. PDS did the critical review and would act as the corresponding author/guarantor.
Figure 1: (A) Contrast-enhanced computed-tomography scan showing communicating hydrocephalous and basal meningeal enhancement (black arrows). (B and C) Gadolinium contrast-enhanced magnetic resonance imaging (6 months after starting therapy) showing gross hydrocephalous (right > left). The right cortical mantle in right frontoparietal region (white arrow) is grossly thinned out with ventricle nearly abutting the calvaria. Multiple ring-enhancing lesions are seen in the right sylvian fissure suggestive of tuberculoma (curved arrow).

Figure 2: (A) Computed-tomography scan showing mild hydrocephalous and exudates in right sylvian fissure and basal cisterns (arrow). These exudates (arrow) and hydrocephalous worsened after 1 month of therapy (B). (C) Gadolinium contrast-enhanced magnetic resonance imaging (MRI) showing multiple conglomerate ring-enhancing lesions in basal cisterns (arrow) and bilateral sylvian fissure (right > left). (D) MRI T2-weighted image showing hyperintensity in right lentiform nucleus (arrow) suggestive of chronic infarct. These areas were hypointense in corresponding T1-weighted images (not shown). These tuberculomas reduced significantly (E) and were calcified (E and F) after 3 years of therapy.
Financial support and sponsorship
Nil.

Conflicts of interest
The authors have no conflicts of interest to disclose with regard to this article.

Ethical approval
An informed consent form was signed by the parents of the patients to approve the use of patient information or material for scientific purposes. The patient’s identity has not been disclosed anywhere in the article, and the article does not contain any identifiable images.

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