Original Research Paper

Volume - 10 | Issue - 9 | September - 2020 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Paediatrics

DESCRIPTIVE STUDY ON ACUTE BACTERIAL MENINGITIS AMONG CHILDREN

Dr. Akshatha SP

Assistant professor, Department of Pediatrics, Adichunchanagiri Institute of Medical Sciences, B.G Nagar, Karnataka

ABSTRACT **Introduction:** Bacteria gain entry to the CSF through the choroid plexus of the lateral ventricles and the meninges and then circulate to the extracerebral CSF and subarachnoid space. Bacteria rapidly multiply because the CSF concentrations of complement and antibody are inadequate to contain bacterial proliferation. Chemotactic factors then incite a local inflammatory response characterized by polymorphonuclear cell infiltration. **Methodology:** The parents of children admitted to our institute who were fulfilling the inclusion criteria were informed about this study. Only those patients presenting as first complex febrile seizures are included in the study. Written informed consent from the parents of patients admitted in paediatric wards of Post Graduate Institute of Paediatric Sciences were taken. **Results:** Out of 125 subjects, 118 (94.4%) had a Negative CSF gram staining and 5.6% (n=2) had a positive CSF gram staining. Among 9 ABM cases, 7 (77.7%) had a positive CSF gram staining, among them 4 showed gram positive organism and 3 showed gram negative organism. None of the patients with no ABM had a positive CSF gram staining. **Conclusion:** This difference between CSF gram staining and ABM was statistically significant.

KEYWORDS : Acute Bacterial Meningitis, CSF, Polymorphonuclear Cell Infiltration

INTRODUCTION:

Bacterial meningitis most commonly results from hematogenous dissemination of microorganisms from a distant site of infection; bacteremia usually precedes meningitis or occurs concomitantly. Bacterial colonization of the nasopharynx with a potentially pathogenic microorganism is the usual source of the bacteremia. There may be prolonged carriage of the colonizing organisms without disease or, more likely, rapid invasion after recent colonization. Prior or concurrent viral upper respiratory tract infection may enhance the pathogenicity of bacteria producing meningitis.[1]

N. meningitidis and H. influenzae type b attach to mucosal epithelial cell receptors by pili. After attachment to epithelial cells, bacteria breach the mucosa and enter the circulation. N. meningitidis may be transported across the mucosal surface within a phagocytic vacuole after ingestion by the epithelial cell. Bacterial survival in the bloodstream is enhanced by large bacterial capsules that interfere with opsonic phagocytosis and are associated with increased virulence.[2]

Host-related developmental defects in bacterial opsonic phagocytosis also contribute to the bacteremia. In young, nonimmune hosts, the defect may be from an absence of preformed IgM or IgG anticapsular antibodies, whereas in immunodeficient patients, various deficiencies of components of the complement or properdin system may interfere with effective opsonic phagocytosis. Splenic dysfunction may also reduce opsonic phagocytosis by the reticuloendothelial system.[3]

Bacteria gain entry to the CSF through the choroid plexus of the lateral ventricles and the meninges and then circulate to the extracerebral CSF and subarachnoid space. Bacteria rapidly multiply because the CSF concentrations of complement and antibody are inadequate to contain bacterial proliferation. Chemotactic factors then incite a local inflammatory response characterized by polymorphonuclear cell infiltration.[4]

The presence of bacterial cell wall lipopolysaccharide (endotoxin) of Gram-negative bacteria (H. influenzae type b, N. meningitidis) and of pneumococcal cell wall components (teichoic acid, peptidoglycan) stimulates a marked inflammatory response, with local production of tumor necrosis factor, interleukin 1, prostaglandin E, and other inflammatory mediators. The subsequent inflammatory response is characterized by neutrophilic infiltration, increased vascular permeability, alterations of the blood–brain barrier, and vascular thrombosis.[5]

Meningitis-associated brain injury is not simply caused by viable bacteria but occurs as a consequence of the host reaction to the inflammatory cascade initiated by bacterial components. Rarely, meningitis may follow bacterial invasion from a contiguous focus of infection such as paranasal sinusitis, otitis media, mastoiditis, orbital cellulitis, or cranial or vertebral osteomyelitis or may occur after introduction of bacteria via penetrating cranial trauma, dermal sinus tracts, or meningomyeloceles.[6]

Methodology:

Study area: The study was conducted in the department of pediatric medicine, Postgraduate Institute of Pediatrics Sciences

Study population: The study includes the children between the ages of 6 months to 5 years admitted as first complex febrile seizure in the department of pediatric medicine.

Sample size: After applying the formula the minimum sample size is calculated to be equal to 62. Expecting the non response, the final sample size of 125 was taken for the study.

Exclusion criteria:

1. The patients who did not meet the criteria for CFS
2. The patients with prior history of simple/complex febrile seizures
3. Those patients who presented with unprovoked seizures (afebrile seizures)
4. The patients with any neurological abnormalities
5. The patients with a preceding history of trauma

Data Collection:

The parents of children admitted to our institute who were fulfilling the inclusion criteria were informed about this study. Only those patients presenting as first complex febrile seizures are included in the study. Written informed consent from the parents of patients admitted in paediatric wards of Post Graduate Institute of Paediatric Sciences were taken. After obtaining informed consent, demographic details, details of current illness like fever – type, duration and grading (High-grade fever was defined as the temperature >104°F), convulsions– type, duration and number of episodes, any prior hospitalization and medication, co-morbid diseases, past history of convulsions, birth and developmental history, immunization history and family history of febrile seizure was elicited. A thorough clinical examination was performed and observations were noted on the study Performa. Laboratory investigations including CSF analysis were done in all children. Lumbar puncture for CSF analysis was done after stabilizing the patient in less than 24 hours of admission. EEG was done in all patients after 2 weeks of febrile seizure to identify the nature of underlying cerebral pathology and predict the risk of future seizures. CT scan brain was done in patients who presented with febrile status epilepticus and having abnormal neurological examination before performing lumbar puncture.

Results:

Table 1: Distribution of High grade fever

High Grade Fever	ETIOLOGY				Total		P value
	ABM	%	NO ABM	%	Number	%	
Yes	9	100	9	7.7	18	14.4	0.0000001
No	0	0	107	92.3	107	85.6	
Total	9	100	116	100	125	100	

df-1,Fisher exact P value -0.0000001

Out of total 125 cases, 14.4% (n=18) suffered high grade fever while 85.5% (n=107) didn't suffer high grade fever. Whole cases of ABM were having high grade fever. This association of high grade fever with ABM was significant.

Table 2: Association between febrile status epilepticus and acute bacterial meningitis

Febile statu sepilepticus at the time of presentation	Etiology				Total		P value
	ABM	%	NO ABM	%	Number	%	
Yes	4	44.4	1	0.9	5	4	0.0001
No	5	55.6	115	99.1	120	96	
Total	9	100	116	100	125	100	

Out of total 125 study subjects,4%(n=5) presented with febrile status epilepticus while 96%(n=120) were normal. Among 9 ABM cases, 4 (44.4%) presented as febrile status epilepticus. This demonstrates a substantial association between febrile status epilepticus and ABM.

Table 3: Association between post ictal drowsiness and acute bacterial meningitis

Post-ictaldrowsiness	Etiology				Total		P value
	ABM	%	NO ABM	%	Number	%	
Yes	7	77.7	4	3.4	11	8.8	0.0001
No	2	22.3	112	96.6	114	91.2	
Total	9	100	116	100	125	100	

Out of 125 study subjects, 8.8% (n=11) had h/o post-ictal drowsiness and 91.2% (n=114) didn't have h/o post-ictal drowsiness. Among 9 ABM patients, 7 (77.7%) had h/o post-ictal drowsiness compared to 4 (3.4%) of patients with no bacterial meningitis. This association between post-ictal drowsiness and ABM was significant in our study.

Table 4: Association between CSF differential leucocyte count and ABM

CSF differential leucocyte count	Etiology				Total		P value
	ABM	%	NO ABM	%	Number	%	
Neutrophil predominate	9	100	0	0	9	69.2	0.000001
Lymphocyte predominate	0	0	4	100	4	30.8	
Total	9	100	4	100	13	100	

Out of 13study subjects with CSF pleocytosis , 69.2%(n=9) had predominate neutrophil count ,while 30.8%(n=4) had predominate lymphocyte count. All patients of ABM were having predominant neutrophil count in the CSF. This difference between CSF differential leukocyte count and ABM was found to be statistically significant.

Table 5: Distribution of CSF gram staining among study subjects

CSF gram staining	Etiology				Total		P value
	ABM	%	NO	%	Number	%	
Positive	7	77.7	0	0	7	5.6	0.0000001
Negative	2	22.3	116	100	118	94.4	
Total	9	100	116	100	125	100	

df-1,Fisher exact P value -.0000001

Out of 125 subjects, 118 (94.4%) had a Negative CSF gram staining and 5.6% (n=2) had a positive CSF gram staining. Among 9 ABM cases, 7 (77.7%) had a positive CSF gram staining, among them 4 showed gram positive organism and 3 showed gram negative organism. None of the patients with no ABM had a positive CSF gram staining. This difference between CSF gram staining and ABM was statistically significant.

DISCUSSION:

The onset of acute meningitis has 2 predominant patterns. The more dramatic and, less common presentation is sudden onset with rapidly progressive manifestations of shock, purpura, disseminated intravascular coagulation, and reduced levels of consciousness often progresses to coma or death within 24 hr.[7] More often, meningitis is preceded by several days of fever accompanied by upper respiratory tract or gastrointestinal symptoms, followed by nonspecific signs of CNS infection, such as increasing lethargy and irritability.

The signs and symptoms of meningitis are related to the nonspecific findings associated with a systemic infection and to manifestations of meningeal irritation. Nonspecific findings include fever, anorexia, poor feeding, and headache, symptoms of upper respiratory tract infection, myalgias, arthralgias, tachycardia, hypotension, and cutaneous signs, such as petechiae, purpura, or an erythematous macular rash.[8]

Meningeal irritation is manifested as nuchal rigidity, back pain, Kernig sign (flexion of the hip 90 degrees with subsequent pain with extension of the leg), and Brudzinski sign (involuntary flexion of the knees and hips after passive flexion of the neck while supine). In children, particularly in those younger than 12-18 months, Kernig and Brudzinski signs are not consistently present. Indeed fever, headache, and nuchal rigidity are present in only 40% of adults with bacterial meningitis.[9] Increased ICP is suggested by headache, emesis, bulging fontanel or diastasis (widening) of the sutures, oculomotor (anisocoria, ptosis) or abducens nerve paralysis, hypertension with bradycardia, apnea or hyperventilation, decorticate or decerebrate posturing, stupor, coma, or signs of herniation. Papilledema is uncommon in uncomplicated meningitis and presence of which shows a more chronic process, such as the presence of an intracranial abscess, subdural empyema, or occlusion of a dural venous sinus. Focal neurologic signs are usually due to vascular occlusion.

Cranial neuropathies of the ocular, oculomotor, abducens, facial, and auditory nerves may also happen as the result of focal inflammation. Overall, approximately 10-20% of children with bacterial meningitis have focal neurologic signs.[10]

Seizures (focal or generalized) caused by cerebritis, infarction, or electrolyte disturbances occur in 20-30% of patients with meningitis. Seizures that occur on presentation or within the 1st 4 days of onset are usually of no prognostic significance. Seizures that persist after the 4th day of illness and those that are difficult to treat may be associated with a poor prognosis.

Altered mental status are common among patients with meningitis and may be the consequence of increased ICP, cerebritis, or hypotension; manifests as irritability, lethargy, stupor, obtundation, and coma. Comatose patients have a poor prognosis.

Additional manifestations of meningitis include photophobia and tache cérébrale, which is elicited by stroking the skin with a blunt object and observing a raised red streak within 30-60 sec.

CONCLUSION:

Streptococcus Pneumoniae is the main causative agent of meningitis in our study with 3 out of 9 patients showed bacterial growth in the CSF.

REFERENCES:

1. Vestergaard M, Hviid A, Madsen KM, Wohlfahrt J, Thorsen P, Schendel D, et al. MMR vaccination and febrile seizures: evaluation of susceptible subgroups and long-term prognosis. JAMA. 2004 Jul 21;292(3):351–7.
2. Dubé CM, Brewster AL, Baram TZ. Febrile seizures: mechanisms and relationship to epilepsy. Brain Dev. 2009 May;31(5):366–71.
3. Shibasaki K, Suzuki M, Mizuno A, Tominaga M. Effects of body temperature on neural activity in the hippocampus: regulation of resting membrane potentials by transient receptor potential vanilloid 4. J Neurosci Off J Soc Neurosci. 2007 Feb 14;27(7):1566–75.
4. Cartmell T, Luheshi GN, Rothwell NJ. Brain sites of action of endogenous interleukin-1 in the febrile response to localized inflammation in the rat. J Physiol. 1999 Jul 15;518(Pt 2):585–94.
5. Cartmell T, Southgate T, Rees GS, Castro MG, Lowenstein PR, Luheshi GN. Interleukin-1 mediates a rapid inflammatory response after injection of adenoviral vectors into the brain. J Neurosci Off J Soc Neurosci. 1999 Feb 15;19(4):1517–23.
6. Ban E, Milon G, Prudhomme N, Fillion G, Haour F. Receptors for interleukin-1 (alpha and beta) in mouse brain: mapping and neuronal localization in hippocampus. Neuroscience. 1991;43(1):21–30.
7. Vezzani A, Granata T. Brain inflammation in epilepsy: experimental and clinical evidence. Epilepsia. 2005 Nov;46(11):1724–43.
8. Dubé C, Vezzani A, Behrens M, Bartfai T, Baram TZ. Interleukin-1beta contributes to the generation of experimental febrile seizures. Ann Neurol. 2005 Jan;57(1):152–5.
9. Aram JA, Lodge D. Epileptiform activity induced by alkalosis in rat neocortical slices: block by antagonists of N-methyl-D-aspartate. Neurosci Lett. 1987 Dec 29;83(3):345–50.
10. Bethune P, Gordon K, Dooley J, Camfield C, Camfield P. Which child will have a febrile seizure? Am J Dis Child 1960. 1993 Jan;147(1):35–9.