ORIGINAL RESEARCH

Evaluation of CRP level in patients with meningitis

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ABSTRACT

Background: Meningitis is defined as an infection that mostly affects the subarachnoid space and is linked to an inflammatory process in the central nervous system that results in seizures, elevated intracranial pressure, and diminished or absent awareness. **Materials & Methods:**98 patients of meningitis of both genderswere diagnosed using the Careggi score (BM-CASCO) and Thwaites diagnostic grading. Routine CSF and blood tests were performed on the patients. **Results:** Out of 98 patients, males were 50 and females were 48. The etiology of meningitis was cryptococcal in 5, scrub typhus in 5, paraneoplastic in 16, bacterial in 32, tubercular in 26, and viral in 14 cases. The common clinical findings in patients were fever seen in 167, headache in 52, and loss of consciousness in 43, vomiting in 30, and seizures in 19 patients. The difference was significant ($P< 0.05$). The mean CRP level in cryptococcal was 0.35 mg/dl, in scrub typhus was 0.27, in paraneoplastic was 0.23, in bacterial meningitis was 3, in tubercular was 0.75, and in viral was 0.31. The difference was significant ($P< 0.05$). **Conclusion:** In contrast to other types of meningitis, the tubercular variety exhibited the highest level of CRP.

Keywords: Meningitis, C-reactive protein, paraneoplastic

INTRODUCTION

Meningitis is defined as an infection that mostly affects the subarachnoid space and is linked to an inflammatory process in the central nervous system that results in seizures, elevated intracranial pressure, and diminished or absent awareness.[1] Globally, meningitis is a major source of illness and mortality. In the absence of proper care, up to 70% of cases will result in death, and 1 in 5 meningitis survivors may experience long-term consequences such as hearing loss or any other neurological impairment. Before the effectiveness of antimicrobial therapy, damage to the central nervous system determines the result and survival of the nervous system.[2]

If bacterial meningitis is not treated promptly and aggressively, it can result in death in both wealthy and developing nations. If left untreated, the death rate is close to 100%; however, even with the most advanced pediatric critical care and antibiotics available today, the disease's fatality rate is only about 5% to 10%.[3] Globally, 20% of survivors get neurological aftereffects from meningitis after being discharged from the hospital. The largest risk of long-term, incapacitating secondary outcomes was found in low-income nations, which also had the highest rates of bacterial meningitis. Meningitis' aetiological diagnosis presents a diagnostic conundrum.[4] Under certain circumstances, the biochemical examination of the cerebrospinal fluid (CSF) and the cellular reaction overlap. Levels of CSF C-reactive protein (CRP) may offer a quick and easy way to diagnose meningitis specifically. Acute phase reactant C-reactive protein (CRP) belongs to the "Pentraxin" family. Hepatocytes alone create CRP within six hours after an acute inflammatory stimulation, and it is released in serum or fluids linked to affected organs.[5]The present study was conducted to assess CRP level in patients with meningitis.

MATERIALS & METHODS

The present study comprised 98 patients of meningitis of both genders. All agreed to participate with their written consent.

Data about patients such as name, age, gender, etc. was recorded. Patients were diagnosed using the Careggi score (BM-CASCO) and Thwaites diagnostic grading. Routine CSF and blood tests were performed on the patients. The CSF was sent for biochemical

(protein, sugar, lactate, ADA, and CRP) and microbiological (cytology, gram's stain, culture and sensitivity, TB-PCR, Z-N stain) examination. Using the Beckman Coulter CRP - latex reagent kit on its fully automated apparatus AU 480, CSF CRP levels were calculated.P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 98		
Gender	**Male**	**Female**
Number	50	48

Table I shows that out of 98 patients, males were 50 and females were 48.

Table II Assessment of parameters

Parameters	Variables	Number	P value
Etiology	Cryptococcal	5	0.05
	Scrub typhus	5	
	Paraneoplastic	16	
	Bacterial	32	
	Tubercular	26	
	Viral	14	
Findings	Fever	67	0.92
	Headache	52	
	Loss of consciousness	43	
	Vomiting	30	
	Seizures	19	

Table II, graph I show that the etiology of meningitis was cryptococcal in 5, scrub typhus in 5, paraneoplastic in 16, bacterial in 32, tubercular in 26, and viralin 14 cases.The common clinical findings in patients were fever seen in 167, headache in 52, and loss of consciousness in 43, vomiting in 30, and seizures in 19 patients. The difference was significant (P< 0.05).

Graph I Assessment of parameters

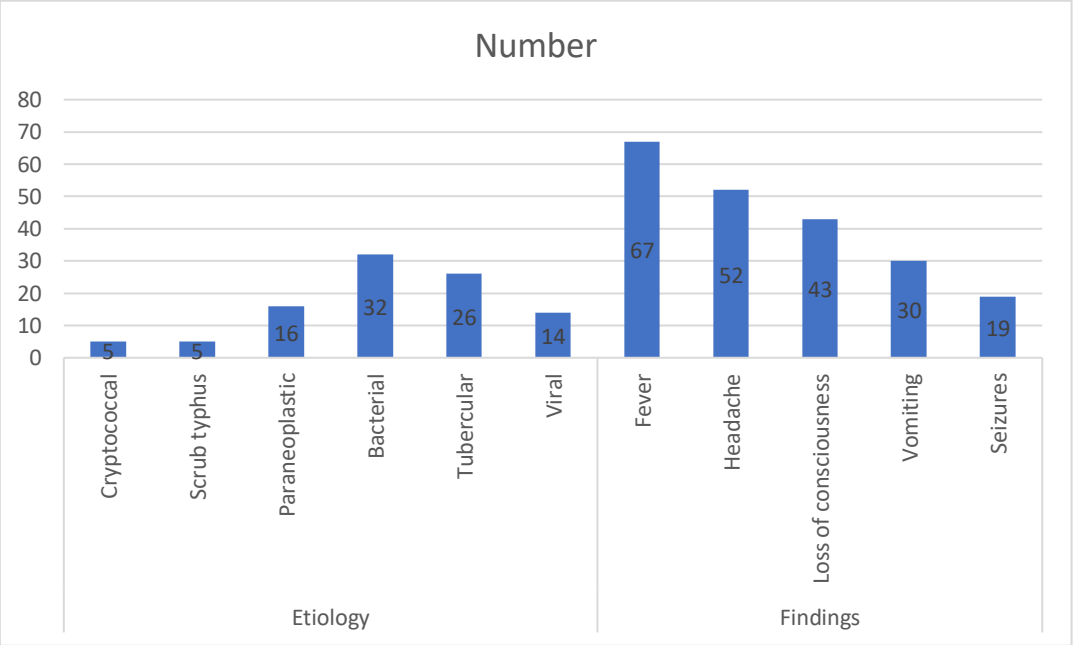

Table III Measurement of CRP level

Type	Mean (mg/dl)	P value
Cryptococcal	0.35	0.05
Scrub typhus	0.27	
Paraneoplastic	0.23	
Bacterial	3.0	

Tubercular	0.75	
Viral	0.31	

Table III, graph I show that the mean CRP level in cryptococcal was 0.35 mg/dl, in scrub typhus was 0.27, in paraneoplastic,was 0.23, in bacterial meningitis was 3, in tubercular was 0.75, and in viral was 0.31. The difference was significant (P< 0.05).

DISCUSSION

Meningitis claims the lives of about 52,000 children in India annually, making up 2% of all pediatric fatalities. A prompt diagnosis and efficient management are essential for success. The largest risk of long-term, incapacitating secondary outcomes was found in low-income nations, which also had the highest rates of bacterial meningitis. The majority of the observed outcomes may have been prevented with immunization against meningococcal, pneumococcal, and Hib. The extensive use of antibiotics has further confused the clinical and laboratory picture of meningitis, making it more difficult to diagnose bacterial meningitis and to develop a suitable treatment plan using standard microbiological techniques. Passive diffusion over inflamed meninges raises CSF CRP levels in meningitis. Typically, plasma contains minimal amounts of CRP (less than 0.5).[6]

We found that out of 98 patients, males were 50 and females were 48. Negrini et al[7] assessed the characteristics of the CSF differential in aseptic versus bacterial meningitis. One hundred fifty-eight cases of meningitis were reviewed: 138 were aseptic and 20 were bacterial. The patients ranged in age from 30 days to 18 years; 61% were male. Fifty-seven percent of cases of aseptic meningitis had a PMN predominance. The percentage of PMNs in the CSF in patients with aseptic meningitis was not statistically different for patients who had a lumbar puncture performed either within or beyond 24 hours of the onset of symptoms. Fifty-one percent of the 53 patients with aseptic meningitis and duration of illness >24 hours had a PMN predominance. The ability of a PMN predominance to differentiate between aseptic and bacterial meningitis was assessed. The sensitivity of a PMN predominance for aseptic meningitis is 57% whereas the specificity is 10%. The positive predictive value of a PMN predominance for aseptic disease is 81% but the negative predictive value is 3%. Alternative definitions of PMN predominance from 60% to 90% were not useful as a clinical indicator of bacterial disease.

We found that the etiology of meningitis was cryptococcal in 5, scrub typhus in 5, paraneoplastic in 16, bacterial in 32, tubercular in 26, and viral in 14 cases. The common clinical findings in patients were fever seen in 167, headache in 52, and loss of consciousness in 43, vomiting in 30, and seizures in 19 patients. Nigrovic et al[8] found that among 3295 patients with CSF pleocytosis, 121 (3.7%) had bacterial meningitis and 3174 (96.3%) had aseptic meningitis. Of the 1714 patients categorized as very low risk for bacterial meningitis by the Bacterial Meningitis Score, only 2 had bacterial meningitis (sensitivity, 98.3%; 95% CI, 94.2%-99.8%; negative predictive value, 99.9%), and both were younger than 2 months old. A total of 2518 patients (80%) with aseptic meningitis were hospitalized.

We found that the mean CRP level in cryptococcal was 0.35 mg/dl, in scrub typhus was 0.27, in paraneoplastic was 0.23, in bacterial meningitis was 3, in tubercular was 0.75, and in viral was 0.31. Chinchankar N et al[9]estimatedthe frequency of acute bacterial meningitis (ABM) in early childhood. 54 children (1.5% of all admissions) satisfied the criteria of ABM in early childhood; 78% were below one year and 52% were under the age of six months. The chief presentation was high fever, refusal of feeds, altered sensorium and seizures. Meningeal signs were present in only 26%. CSF C-reactive protein was positive in 41%, gram stain was positive in 67% LAT in 78% and cultures grew causative organisms in 50% of the cases. The final etiological diagnosis (as per LAT and/or cultures) were streptococcus pneumoniae in 39% Hemophilus influenzae type B 26% and others in 35%. The others included one case of Neisseria meningitis and 10 who were LAT negative and culture sterile. 39% of patients developed acute neurological complications during the hospital course. 31% of children with ABM died in hospital or at home soon after discharge. Six were lost to follow-up. Of the 31 children, available for long-term follow-up (1-3 years), 14 (45%) had no sequelae. The remaining had significant neurodevelopmental handicaps ranging from isolated hearing loss to severe mental retardation with multiple disabilities.

Bhat et al[10] studied the clinical profile of pyogenic meningitis in 256 cases. The male-to-female ratio was 1.46: 1 and 83.6% of the patients were less than 3 years of age. Fever, altered sensorium, refusal to feed, convulsions, and vomiting were the common presenting symptoms. Six (2.3%) of them had subnormal temperature and the sensorium was normal in 12.5% of cases. Cerebrospinal fluid was clear in 9.4%, cell count was less than 100/cumm in 8.2%, sugar was more than 40 mg% in 24.2% and protein was less than 50 mg% in 12.5% of cases. Diplococcus pneumoniae was the commonest causative agent. The overall mortality was 30.5%. It is concluded that young children suffering from this disease may not have the typical features and one should have the highest suspicion to make an early diagnosis so that the morbidity and mortality from this condition can be reduced.

Amarilyo G et al[11] in their study a total of 108 patients with suspected meningitis were enrolled. Meningitis was diagnosed in 58 patients (53.7%; 6 bacterial and

52 aseptic). Sensitivity and specificity were 76% and 53% for headache (among the verbal patients) and 71% and 62% for vomiting, respectively. Photophobia was highly specific (88%) but had low sensitivity (28%). Clinical examination revealed nuchal rigidity (in patients without open fontanel) in 32 (65%) of the patients with meningitis and in 10 (33%) of the patients without meningitis. Brudzinski and Kernig signs were present in 51% and 27% of the patients with meningitis, respectively, and had relatively high positive predictive values (81% and 77%, respectively). Bulging fontanel in patients with open fontanel was present in 50% of the patients with meningitis but had a positive predictive value of only 38%.

CONCLUSION

Authors found thatin contrast to other types of meningitis, the tubercular variety exhibited the highest level of CRP.

REFERENCES

1. Bassani DG, Kumar R, Awasthi S, Morris SK, Paul VK, Shet A, et al. Causes of neonatal and child mortality in India: a nationally representative mortality survey. Lancet 2010;376:1853-60.
2. Lee BE, Davies HD. Aseptic meningitis. Curr Opin Infect Dis 2007;20: 272-7.
3. Curtis S, Stobart K, Vandermeer B, Simel DL, Klassen T. Clinical features suggestive of meningitis in children: a systematic review of prospective data. Pediatrics2010;126:952-60.
4. Sunbul M, Atilla A, Esen S et al. Thwaites' diagnostic scoring and the prediction of tuberculous meningitis. Med Princ and Pract 2005; 14: 151-4.
5. de Onis M, Blossner M. WHO global database on child growth and malnutrition. WHO/NUT/97.4: World Health Organization, Department of Nutrition for Health and Development, 1997.
6. Weber MW, Herman J, Jaffar S, Usen S, Oparaugo A, Omosigho C, Adegbola RA, Greenwood BM, Mulholland EK. Clinical predictors of bacterial meningitis in infants and young children in The Gambia. Tropical Medicine & International Health. 2002 Sep;7(9):722-31.
7. Negrini B, Kelleher KJ, Wald ER. Cerebrospinal fluid findings in aseptic versus bacterial meningitis. Pediatrics2000;105:316-9.
8. Nigrovic L.E.Kuppermann N.Macias C.G.Cannavino C.R.Moro-Sutherland D.M.Schremmer R.D.et al.Clinical prediction rule for identifying children with cerebrospinal fluid pleocytosis at very low risk of bacterial meningitis.JAMA. 2007; 297: 52-60.
9. Chinchankar N, Mane M, Bhave S, Bapat S, Bavdekar A, Pandit A, et al. Diagnosis and outcome of acute bacterial meningitis in early childhood. Indian Pediatr2002;39:914-21.
10. B V Bhat, I C Verma, R K Puri, S Srinivasan, P Nalini. A profile of pyogenic meningitis in children. J Indian Med Assoc1991 Aug;89(8):224-7.
11. Amarilyo G, Alper A, Ben-Tov A, Grisaru-Soen G. Diagnostic accuracy of clinical symptoms and signs in children with meningitis. Pediatric Emergency Care. 2011;27(3):196–199.