May-August, 2012/Vol 32/Issue 2
doi: http://dx.doi.org/10.3126/jnps.v32i2.5534

Original Article

Ocular Manifestations of Meningitis in Children

Chaudhary M[1], Shah DN[2], Sharma PR[3]

[1]Dr. Meenu Chaudhary, MBBS, MD, B.P. Koirala lions Center for Ophthalmic Studies, [2]Prof. DN Shah, B.P.Koirala lions Center for Ophthalmic Studies, [3]Prof. Puspa Raj Sharma, Department of Paediatrics. All from the Tribhuvan University Teaching Hospital, Maharajgunj Kathmandu, Nepal.

Address for correspondence: Dr. Meenu Chaudhary, E-mail: drmeenu67@gmail.com

Abstract

Introduction: Meningitis is the most common central nervous system disease affecting children leading to focal neurological deficits and various oculovisual anomalies including blindness in children. The objective of this study was to evaluate the oculovisual anomalies occurring in Nepalese children suffering from different types of bacterial meningitis. **Materials and Methods**: A Prospective, study was undertaken for 18 months at B.P.Koirala Lion's Center for ophthalmic studies, TU Teaching Hospital to study the children suffering from bacterial meningitis admitted at Kanti Children's Hospital for ocular involvement. A through history, anterior and posterior segment ocular examination and investigations like blood, CSF and CT scan were done. **Results**: A total of 182 cases of bacterial meningitis were screened. Tubercular meningitis cases were 40 (21.97%) and Pyogenic were 142 (78.02%). Oculovisual anomalies were seen in 70 (38.46%) cases. The ocular abnormalities included pupillary changes (34.28%), Cranial Nerve Palsy (22.86%), Fundus changes (35.72%), Cortical Blindness (4.28%), Panophthalmitis and Proptosis (1.43%). Third nerve involvement was seen in 17.14% cases, sixth nerve in 4.29% cases, Papilledema in 11.43 % and Optic atrophy in 22.86 %. Risk factors included late presentation; hydrocephalous and increased CSF cell count and protein level. **Conclusion**: Oculovisual anomalies formed an important group of clinical manifestations of bacterial meningitis. Incidence of oculovisual anomalies was more frequently seen in Tubercular meningitis (55%).Children with early presentation and intervention had better prognosis. Hence, timely intervention and health education is important.

Key words: Bacterial Meningitis, Tubercular, Pyogenic, Oculovisual anomalies, Optic atrophy

Introduction

Meningitis is the most common central nervous system disease affecting children[1] leading to focal neurological deficits and various oculovisual anomalies including blindness in children. Commonly seen meningitis in children is of two types–Pyogenic and Tubercular. Both these types of meningitis can be associated with cranial neuropathies of II, III, IV, VI and VII cranial nerves due to focal or generalized inflammation[2]. Optic neuritis, optic atrophy and papilloedema are the most important neurological sequelae of meningitis, mainly, tubercular. Meningitis may also be associated with lid retraction, gaze paresis, squint, tonic deviation of eyes, nystagmus, pupillary abnormality in size and reaction, panophthalmitis and exposure keratitis. According to the WHO meeting on childhood blindness in 1990 there are approximately 1.5 million blind children in the world of which 90% live in the developing countries[3]. In Nepal the prevalence of childhood blindness (0-14yrs. age group) is 0.63/1000[4]. Tubercular meningitis (TBM) is a more dreaded form of meningitis than pyogenic meningitis. Modern medical management has increased the number of survivors who live with neurological deficits. Cortical blindness is a rare and generally unexplained complication of bacterial meningitis. Patients who have high CSF protein content showed more chances of development of primary optic atrophy. Choroidal tubercles and papilloedema were found to be signs of grave prognostic significance. Thus, meningitis is an important cause of mortality and morbidity in the form of neurological sequelae and remains a serious global health problem in spite of potent antibiotics and improved treatment modalities. This prospective hospital based study was therefore

Manuscript Received: 28th November 2011
Reviewed: 25th March 2012
Author Corrected: 9th April 2012
Accepted for Publication: 30th April 2012

undertaken to determine the correlation between meningitis and different ocular problems. This study was planned to evaluate the oculovisual anomalies occurring in Nepalese children suffering from different types of meningitis and to identify some of the risk factors leading to different ocular abnormalities. Identification of which may be helpful, if taken care of properly, in minimizing these anomalies and its sequelae.

Material and Methods

A Prospective, clinical observational study was carried out for 1.5 years on patients of meningitis aged 0 – 14yrs, admitted at Kanti Children's B.P.Koirala Lion's Center for ophthalmic studies, TU Teaching Hospital. A medical history of fever, convulsions, headache, unconsciousness, lethargy, refusal to feed, loss of appetite and weight, focal neurological signs, ocular problems and head injury were taken. Birth history of premature rupture of membranes, low birth weight, neonatal asphyxia, immunization history and history of contact with patient suffering from pulmonary tuberculosis was taken. Clinically diagnosed cases of meningitis proven by Cerebrospinal Fluid (CSF) examination were included after verbal consent from the parents.

Clinical examination included - State of consciousness, temperature, neck rigidity, Kerning's sign, Babinski's sign, bulging of anterior fontanelle and head circumference. Ocular examination included-Visual Acuity (according to the level of consciousness, age, intelligence and cooperation of the child), extra ocular movements, anterior segment and fundus evaluation under mydrasis. 1% Atropine eye ointment was used for pupillary dilatation. Examination under general anesthesia was carried out whenever felt necessary. Investigations included - CSF Analysis (Protein, Sugar, Cytological, and biochemical and culture sensitivity), Mantoux test, Chest x-ray, CT scan and USG of skull.

CSF Analysis: Diagnostic Criteria

- **Pyogenic**
  - Protein: >100mg/dl
  - Glucose: < 40mg/dl
  - Cytological: total count:>7500cell/cumm
  - DC: >80% PMN
- **Neonates**

  TC - >30cells/cumm
- **Tubercular**

  Protein - >100mg/dl

  Glucose- mildly reduced

  Cytological – total count – 50-500cells/cumm

  DC- 80% lymphocytes

  C/s – Mycobacterium tuberculosis
  Cobweb formation in CSF

Results

A total of 182 cases of Meningitis (Pyogenic + Tubercular) were included.

- Total no. of Tubercular Meningitis cases = 40(21.97%)
- Total no. of Pyogenic Meningitis cases = 142(78.02%)
- Oculovisual anomalies seen in 70 cases (38.46%).

Table 1: Incidence of Oculovisual Anomalies in meningitis cases.

Total	Tubercular	Pyogenic
38.46 %	12.09 %	26.37 %

Table 2: Pattern of different Ocular Abnormalities noted in meningitis cases:

Ocular Abnormalities	No. of cases	Percentage (%)
1. Pupillary Changes	24	34.28
2. Cranial Nerve palsy	16	22.86
3. Proptosis	1	1.43
4. Panophthalmitis	1	1.43
5. Fundus Changes	25	35.72
6. Cortical Blindness	3	4.28
Total no. of cases	**70**	**100**

Table 3: Involvement of different Cranial Nerves in the meningitis cases:

Type of Cranial Nerve Palsy	No. of cases	Percentage (%)
a. 3rd Nerve Palsy	12	17.14
b. 6th Nerve Palsy	3	4.29
c. 3rd+4th+6th Nerve Palsy	1	1.43

Table 4: Frequency Comparison of Ocular Abnormalities in Tubercular meningitis and Pyogenic meningitis:

	Tubercular Meningitis		Pyogenic Meningitis	
Ocular Changes	**No. of cases**	**Percentage (%)**	**No. of Cases**	**Percentage %**
Pupillary Changes	6	27.28 %	18	37.50 %
Cranial Nerve Palsy	9	40.91 %	7	14.59 %
Fundus Changes	5	22.72 %	20	41.67 %
Cortical Blindness	2	9.09 %	1	2.08 %

Table 5: Risk Factors for Potential Ocular Abnormalities in the Study Group

Risk Factor	No. of cases	Percentage (%)
1. Delayed Presentation	31	17.05
2. Increased Total cell count in CSF	34	18.68
3. CSF protein level >100.0 mg/dl	23	12.63
4. Hydrocephalous	22	12.08
a. Pyogenic	10	5.49
b. TBM	12	6.59
5. Subdural effusion	5	2.74
6. Head Injury	1	0.54
7. Protein-energy malnutrition (PEM)	1	0.54

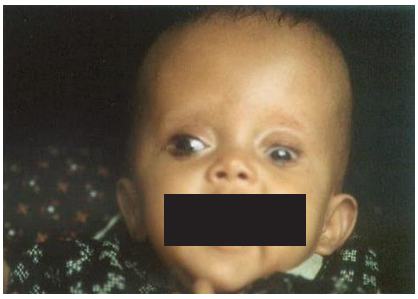

Fig 1: Hydrocephalous seen in a patient with meningitis (Sun –Set Sign)

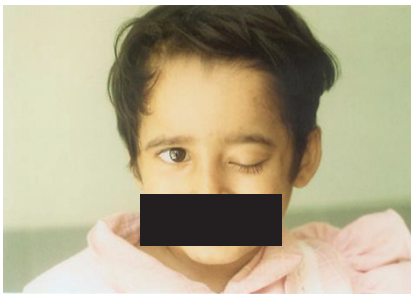

Fig 2: LE Ptosis - Complete Third nerve palsy seen in a child with meningitis

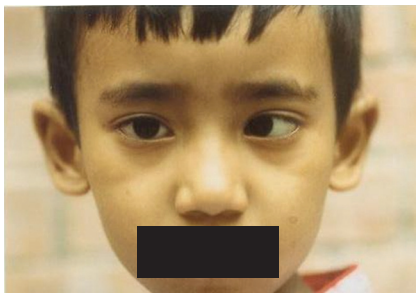

Fig 3: Bilateral sixth nerve palsy seen in a child with meningitis

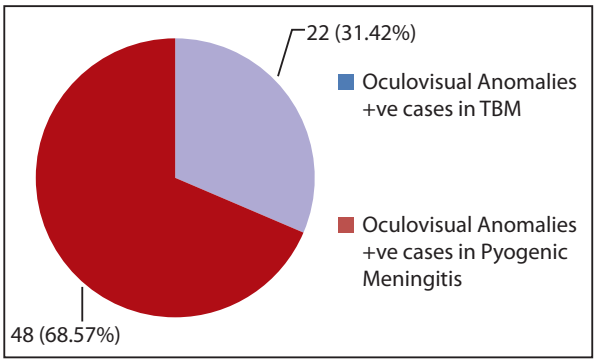

Fig 4: Number of cases with Oculovisual anomalies in different types of Meningitis

Discussion

This study showed that oculovisual anomalies (38.46%) formed an important group of clinical manifestations of Bacterial Meningitis. Common ocular abnormalities included pupillary changes (34.28%), cranial nerve paralysis (22.86%), fundus changes (35.72%), cortical blindness (4.28%), proptosis and panophthalmitis (1.43%) each. Majority of the cases of pyogenic meningitis were found in the age group of one to three years as also reported by other studies (Nelson)[1]. It is probably because of the fact, that immune mechanism and phagocytic functions are not fully matured in neonates and infants. Pyogenic meningitis patients usually present early to the hospital because of its acute onset and features like high fever, severe headache, vomiting, irritability and seizures.

A twelve year study done on bacterial meningitis[5] has reported that bacterial meningitis in children is associated with substantial excess risk of intellectual, cognitive and auditory impairment that persists into adolescence. They have reported 2% children with blindness following meningitis[5]. In the present study higher number of patients were seen with cranial nerve paralysis (22.86%) and fundus changes (35.72%) probably because this study was done on children when they presented in acute phase of the disease unlike the other studies where certain oculovisual anomalies like 3rd and 6th cranial nerve paralysis; Papilledema and retinal haemorrhage got resolved with time and

therefore not taken into account. Scott.L.Pomeroy et al[6] and Ireno Gomes etal [7] studies on Bacterial meningitis have reported cranial nerve deficit in 3% and 14.2 % of the children respectively. Hanna LS etal reported a fifteen year study on ocular complications in 4102 patients of meningitis where they reported pupillary changes in (82%), fundus changes in (5.2%) and cranial nerve paralysis in 23% patients[8].

In our study incidence of oculovisual anomalies was found to be 55% in tubercular meningitis compared to 33.80% in pyogenic meningitis. Ocular involvement in TBM in this study included pupillary changes (27.28%), cranial nerve palsy (40.91%), fundus changes (22.72%) and cortical blindness (9.09%). A study done on children with tubercular meningitis in Ireland by Alan J Mooney they have reported 72% patients with oculovisual anomalies[9]. Studies done by PA Lamba [10] etal and Shakuntala Saxena [11]et al. in India have also reported high incidence i.e. 72.9% and 89% of oculovisual anomalies in TBM. TBM seems to have produced higher incidence of ocular anomalies because of its subacute slowly progressive nature. The residual neurological deficits are produced due to intracranial vasculitis, infarction, cerebral edema and hydrocephalous which cause severe damage. Children with TBM present with headache, low grade fever, malaise and lassitude initially, and hence are treated only with antipyretics or a course of antibiotics. The patient's are brought to the hospital only after features like vomiting, seizures, severe or protracted headache, cranial neuropathies, stupor and coma develop. This delay in presentation and associated features like hydrocephalous and subdural effusion lead to more damage in TBM.

HG Desai etal [12] reported pupillary changes (62.5%), cranial nerve palsy (24.8%) and optic nerve affection (72.9%) in TBM. Shakuntala Saxena etal reported in TBM, cranial nerve involvement (38.2%) and fundus changes (61.8%) of cases[11].

In the present study Oculomotor nerve (III) was affected in 17.14% of cases as compared to 4.29% cases of abducent nerve paralysis. Hanna L.S[8] etal has also reported cranial nerve palsy in meningitis which included sixth cranial nerve in 16.5%; third and seventh nerve in 3.0% each and fourth and fifth nerve in 0.1% each. PA Lamba etal also reported that in TBM oculomotor nerve paralysis was more frequent than abducent nerve paralysis[10]. The Oculomotor nerve involvement in this study was complete (12.85%) or incomplete (4.29%). In 4.29% cases there was only ptosis whereas in (12.85%) cases both ptosis, limitation of extra ocular motility and pupillary involvement were noted. The Oculomotor nerve paresis was probably caused by infiltration or compression of the subarachnoid portion of the Oculomotor nerve and their vascular supply at the base of the brain by thick exudates as in TBM. The third cranial nerve is the nerve most frequently affected in temporal lobe herniation following raised intracranial pressure.

Pupillary changes were seen in 34.28% of cases and occurred more frequently in pyogenic meningitis (37.5%). Higher incidence of pupillary changes has also been reported by Hanna LS[8] (82%); Shakuntala Saxena [11] (53%) and PA Lamba[10] (48%).

Dilated and lack of response to light stimulation of the pupil presumably resulted from damage to the Oculomotor nuclei; one or both Oculomotor nerves or even the ciliary ganglion. The parasympathetic pupillary fibers located in the outermost portions of the nerve were compressed first by the extrinsic intracranial pressure, leading to parasympathetic paralysis which resulted in unopposed sympathetic innervation to the pupil and thus a dilated pupil. Relative afferent pupillary defect was also seen due to optic atrophy.

Panophthalmitis was seen in one case. Corneal ulceration following incomplete closure of the eyelids during coma and panophthalmitis has been reported by Hedges etal. Proptosis with total ophthalmoplegia was seen in one case in this study, which occurred due to cavernous sinus thrombosis, which was confirmed by MRI. This thrombosis thus led to occlusion, thereby reducing venous outflow from the orbit and thus producing compression on the 3rd; 4th and 6th nerves.

Fundus changes were found in 35.72% of cases in the present study. These included papilloedema (11.43%), optic atrophy (22.86%) and Retinal Haemorrhage (1.43%). Fundus changes have been reported in 5.2% of cases of meningitis by Hanna LS [8] etal. H.G. Desai etal has also reported optic nerve affection in 72.9% of cases[12]. VPS Tomar etal have also reported fundus changes in TBM which included Papillitis (56.4%); optic atrophy (29.1%); Papilledema (12.7%) and choroid tubercle in 1.8% cases[11].

In this study retinal haemorrhage was seen possibly due to raised intracranial pressure. Papilloedema occurred due to venous obstruction of the central retinal vein in the subarachnoid space. But Papilloedema was rather infrequent observation in both Tubercular (9.09%) and Pyogenic (12.5%) meningitis cases in comparison to other studies. This could probably be explained by the fact that infants had unfused cranial sutures which thus led to bulging of anterior fontanelle and hydrocephalous following increased intracranial pressure in infants up to 18 months. In the present study (52.19%) cases were below 18 months of age.

Optic atrophy seen in this study was in the form of pale optic disc as well as temporal pallor of optic disc. Blindness occurring secondary to optic atrophy (22.86 %) was also seen in this study. Optic atrophy occurred as post Papilledema and primary optic atrophy. Optic atrophy was either complete (total pallor of optic disc) or incomplete (temporal pallor of optic disc). Shakuntala Saxena has also reported 29.1% cases of optic [11]atrophy in TBM. PA Lamba etal has reported temporal pallor of disc (25%) and primary optic atrophy (14.3%) in the cases of TBM[10]. Optic atrophy was thus found to be a major cause of blindness in this study. Studies from west have also shown that optic atrophy accounts for more than a quarter of childhood blindness. Studies done in Blind schools in china[13] and Zaire[14] have also found that optic nerve affection (13.6%) and optic atrophy (16.3%) was one of the major causes of blindness in children. Studies in India and Sri-Lanka have also reported 4% and 1% cases respectively of preventable blindness in blind children due to meningitis[15].

Hydrocephalous is also one of the important risk factors leading to optic atrophy and finally permanent blindness in meningitis. Hydrocephalous was found to be either communicating (non-obstructive) or non-communicating (obstructive). But children who underwent ventriculoperitoneal shunt surgery for hydrocephalous had a better visual prognosis. Children with cortical blindness were seen in 4.28% of cases in the present study. These children had normal pupillary reflexes and normal fundus appearance on ophthalmoscopy. This complete bilateral, but usually temporary blindness occurs most probably from damage to superficial cortical veins during the course of the disease. Such type of blindness has been reported by Walsh and Hoyt in meningococcal meningitis. There are other reports by Lewis H Morgolis[16] etal and RD Ackroyd[17] where they have reported cortical blindness following meningitis and have found on CT scan examination that it was associated with atrophy of the occipital lobe.

In this study it was found that there were certain critical determinants which affected the visual prognosis. These included delayed presentation (more than 1 month) of the children (17.05%) to the hospital, hydrocephalous (12.08%), subdural effusion (2.74%), increased cell count (18.68%) and protein level >100.0 mg/dl (12.63%) in cerebrospinal fluid.

Children who presented late to the hospital were found to have more frequent oculovisual anomalies most probably due to the increased inflammation and damage that had already taken place before the patient came to the hospital. Similarly, children who had increased cell count and protein level >100.0 mg/dl in CSF showed more oculovisual anomalies because it signifies more severe infection leading to more severe inflammation and cerebral damage. P.A.Lamba in a study on TBM have also reported that patients who had high CSF protein (>75.0mg %) showed more chances of development of primary optic atrophy[10]. Children with hydrocephalous and Subdural effusion were also found to have poor visual outcome. Hydrocephalous was more common in TBM (6.59%) cases than Pyogenic meningitis cases (5.49%), as also reported by Walsh and Hoyt[18, 19].

The blindness may result from compression of the optic nerves by a distended third ventricle; from the effects of acute or chronic Papilledema, or from compression of the posterior cerebral arteries against the tentorium cerebelli. The blindness resulting from damage to the optic nerves, optic atrophy invariably developed, usually in about 4 weeks after loss of vision18. Thus in this study it was found that children who were brought early to the hospital; those not associated with high cell count and protein level in CSF and who did not develop hydrocephalous had good visual prognosis.

Thus from this study we can conclude that Bacterial meningitis is one of the most important diseases in children affecting the central nervous system. It produces different types of ocular abnormalities including optic atrophy which is an important cause of preventable blindness. The timely presentations, early and adequate medical and surgical intervention including V.P.Shunt are important to prevent the residual complications of meningitis.

Conclusion

Ocular abnormalities formed an important group of clinical manifestations of bacterial meningitis in children. The higher number of oculovisual anomalies seen in Tubercular meningitis cases may be explained by the fact that Tubercular meningitis is a subacute slowly progressive form of meningitis and the degree of damage to the blood vessels is greater since they are subjected to increased intra-cranial pressure and perivascular fibrosis for more prolonged periods of time. Delayed presentation, hydrocephalous and increased total cell count in CSF were found to be few of the critical determinants, which affected the visual outcome in meningitis patients. Children with hydrocephalous presented more frequently with cranial nerve paralysis, optic atrophy and cortical blindness. Children who underwent V-P-Shunt surgery for hydrocephalous were found to have good visual prognosis.

Thus modern medical management has increased the survival of children suffering from meningitis in

Nepal but with residual oculovisual anomalies. They occur in the form of strabismus caused by cranial nerve paralysis leading to amblyopia and optic atrophy, and thus leading to blindness. This has a social impact on the society. Hence, health education and timely intervention should be done to prevent morbidity and mortality due to meningitis and to minimize oculovisual anomalies in these children.

Acknowledgements: None
Funding: None
Conflict of Interest: None
Permission from IRB: Yes

References

1. Behrman, Kleigman. Jenson Text Book of Pediatrics Nelson. 1999: 751-757, 885-891.
2. Ghai OP, Kalra Veena. Text Book of Essential Pediatrics. 1996: 321-326.
3. Foster Allen, Gilbert Clare.Report of the WHO Meeting on the Prevention of Childhood Blindness. Geneva. WHO/PBL 1990. *Eye* 1992; 6: 173-176.
4. Brilliant E Girija, Pokhrel.RP, Grasse. C. Nicole. Epidemiology of Blindness in Nepal. Report of 1981 Nepal Blindness Survey. Seva Foundation, USA, 1988; 141-144, 322-323.
5. Grimwood Keith, Anderson Peter, Anderson Vicki, etal. Twelve year outcome following bacterial meningitis: further evidence for persisting effects. *Arch Dis child*. 2000; 83:111-116.
6. Pomeroy.L Scott, Holmes. J Sandra, Dodge.R Philip, etal. Seizures and other Neurological Sequelae of bacterial Meningitis in children. *N Engl J Med* 1990; 323: 1651-1654.
7. Gomes Ireno, Melo Ailton, Lucena Reeta, etal. Prognosis of Bacterial Meningitis in Children. *Arq Neurosiquiatr* 1996; 54 (3): 407 – 411.
8. Hanna L.S., Girgis NI, Ella Abu El, etal. Ocular Complications in Meningitis: "fifteen years study". *Metab Pediatr Systs Ophthalmol* 1988; 11(4): 160-2.
9. Mooney J. Alan. Some Ocular Sequelae of Tuberculous Meningitis. A preliminary Survey, 1953-1954. *Clin Neurophthalmol* 1954; 753-767.
10. Lamba P.A, Bhalla JS, Mullick.DN. Ocular Manifestations of Tuberculous Meningitis: A Clinico – Biochemical study. *JPOS* 1986; 23(3): 123-125.
11. Tomar VPS, Saxena Shakuntala. Ocular Lesions in Tuberculous Meningitis. *J. Indian Pediatr* 1970; 7(12): 662-664.
12. Desai HG, Anklesaria ED. Ophthalmological Aspects of Tuberculous Meningitis. *J. Ind Med Assoc* 1967; 49: 429-432.
13. Hornby Stella J, Xsiao, Foster A, etal. Childhood Blindness in China: Causes in 1131 Blind School Students in 18 Provinces. *Br J Ophthalmol* 1999; 83(8): 929-932.
14. Kayembe L. Common causes of blindness in Zaire. *Br J Ophthalmol* 1985; 69: 389- 391.
15. Taylor David. *Pediatr Ophthalmol* 1997: (10) 1.
16. Margolis Lewis H, Shaywits Bennett A, Rothman Stephen G. Cortical Blindness associated with occipital Atrophy: a complication of H.influenzae Meningitis. Developmental Medicine and child Neurology. 1978; (20): 490-492.
17. Ackroyod R.D. Cortical Blindness following Bacterial Meningitis: A case report with reassessment of Prognosis and Etiology. *Dev Med Child Neurol* 1984; (26): 227-230.
18. Walsh & *Hoyt. Clin Neurophthalmol* 1996: 2823-2825. (V).
19. Walsh & *Hoyt. Clin Neurophthalmol* 1996: 2823-2927, 2952-3059. (V)

How to cite this article ?
Chaudhary M, Shah DN, Sharma PR. Ocular Manifestations of Meningitis in Children. *J Nepal Paediatr Soc* 2012;32(2):136-141.