CORTICAL BLINDNESS IN CHILDREN IN ENUGU, NIGERIA

EN ONWASIGWE *FMCOphthal*
Ophthalmology Department, University of Nigeria Teaching Hospital, Enugu

SUMMARY

Objective

- To analyse cortical blindness in Nigerian children.

Methods: The study covered a 5-year period with a minimum of 2 years follow-up. Diagnosis was made principally in blind eyes that were ophthalmoscopically normal.

Results: A total of 18 patients were selected with an upper limit of 16 years of age. Half of the patients studied presented by the 10th month of life. The mean age was 18.1 months. Meningitis with convulsion accounted for most of the cases (72.2%). Sixteen (88.9%) of the patients had visual return, with 10 (55.6%) occurring within the first 6 months.

Conclusion: Though the prognosis has been observed to be satisfactory, the need to create awareness amongst the populace on the need for early presentation at the hospital in order to avert cortical blindness as a complication from meningitis is important.

Key words: cortical blindness, ophthalmoscopy, meningitis, convulsion, visual return

INTRODUCTION

This study is an analysis of cortical blindness in the Nigerian environment. True cortical blindness implies a selective dysfunction of the visual occipital cortex. It is not easy to differentiate from cerebral blindness which however indicates a more extensive cerebral lesion.[1] It has been propounded that this lesion results from a cerebrovascular occlusive disorder which is caused by spasm involving the calcarine branches of the posterior cerebral artery. This leads to ischaemic changes and infarction.[1,2]

The principal sign is loss of vision with normal ophthalmoscopic details. Cortical blindness occurs in adults and also in children. In adults, studies by Mackenzie and Symonds[3] revealed that cerebrovascular accidents and cardiorespiratory arrest are the commonest causes. The sequence is usually that of thrombosis and embolization of the cerebral vessels.[3] In children, the incidence of cortical blindness from bilateral damage of the occipital lobes has been associated with meningitis and trauma.[4]

PATIENTS AND METHODS

This study was conducted in three centres in Enugu – the University of Nigeria Teaching Hospital (UNTH), Park Lane General Hospital and Ebran's Clinics. The study group consisted mainly of patients with a history of visual loss post convulsion referred to the eye clinics of the three centres over a period of 5 years. Patients' records were collected over the period, January 1995 – December 1999, and analysed with the EPI INFO software programme. The diagnoses corresponded with those made by the referring paediatricians. The ophthalmologist evaluated all the patients after they had been fully examined by the paediatrician and the referring doctor. They were healthy enough for examination under anaesthesia where indicated. The data was obtained partly from the report written by the referring paediatrician and the assessment by the ophthalmologist on seeing the patient. Most of the patients had visual assessment and further ocular examination under anaesthesia. Due to lack of facilities for full visual assessment in children, the assessment of most of the infants was limited to their ability to follow light. This was followed with fundoscopy and refraction under anaesthesia and the results were recorded. The patient's age, sex and duration of visual loss were noted. A minimum follow-up period of 2 years was required for inclusion in the study. For a patient to be diagnosed as a case of cortical blindness, no ocular abnormality would be detected in the presence of visual loss.

RESULTS

A total of 18 patients were seen, out of which 15 presented at the University of Nigeria Teaching Hospital, 2 at Ebran's Clinics and 1 at Park Lane General Hospital. Most cases of cortical blindness in children (50%) occurred before the age of 10 months (see table 1). The mean age was 18.1 months with a standard deviation of 14.7. A higher proportion of males (61.1%) were affected than females (38.9%), with a male to female ratio of 1.6 : 1. The youngest age of presentation

was 6 months and the highest was 4½ years. Table 2 indicates that meningitis was the most common cause of cortical blindness, with 13 patients (72.2%). Convulsion was the common denominator in all the cases. All the patients had a history of convulsion. Data was collected from January 1995 to December 1999. Sixteen patients showed signs of visual recovery. Definitive visual acuity recordings were not made, as all the patients were of pre-school age, i.e., less than 5 years of age, and therefore the use of the Snellen's chart test was not possible. Patients were considered to have made visual recovery if they reacted to stimulus, such as following light, reaching out to objects of regard, and navigational sight. Patients with cerebral palsy coupled with seizure did not show evidence of visual recovery. Ten of the patients (55.6%) showed evidence of visual recovery within the first 6 months. The rest of recovery occurred within 2 years of follow-up. The mean duration of visual recovery was 9.8 months.

Table 1. Age and sex distribution of 18 patients with cortical blindness

Age (months)	Sex		Total	Percent
	Male	Female		
0 - 10	3	6	9	50.0
11 - 20	1	1	2	11.1
21 - 30	4	-	4	22.1
31 - 40	1	-	1	5.6
41 - 50	1	-	1	5.6
51 - 60	1	-	1	5.6
Total	11	7	18	100.0

Mean age = 18.1 months; Standard deviation = 14.7
Male to female ratio = 1.6 : 1.

Table 2. Aetiological factors in cortical blindness in 18 children

Causes	Number	Percent
Meningitis	13	72.2
Cerebral palsy with convulsion	2	11.1
Febrile convulsion (with unclear provoking factor)	2	11.1
Seizure	1	5.6
Total	18	100.0

DISCUSSION

Cortical blindness is usually a consequence of selective visual cortical damage. It may be caused by conditions that provoke convulsion such as meningitis. Other conditions include head injury, complication of haemodialysis, cardiorespiratory arrest and carbon dioxide or nitrous oxide poisoning.[5-8] Cerebral hypoxia is a major factor in cortical blindness. It could be due to poor supply of oxygenated blood or difficulty in utilization.[5-6] Hypoxic states are noted for being selective in visual cortical damage. Laminae 3 and 4 of Brodmann that are usually damaged in hypoxia constitute a good part of the calcarine cortex which is the visual centre.[1,7]

In the presence of ophthalmoscopically normal eyes, patients usually present with non-responsiveness of the eyes to hazards, and particularly in children, they present with inability to follow light.

The incidence of cortical blindness was highest in the first 10 months of life (50.0%). This was always after episodes of convulsion. Visual acuity in children is often difficult to assess. Modalities for visual evaluation such as optokinetic stimulation and EEG response to photic stimulation are not commonly available. Visual response is therefore evaluated by the ability to follow light or objects of regard, ocular response (such as blinking) to hazards, and reaching out for objects. Convulsion has been noted as a common underlining factor in childhood cortical blindness, the commonest disease being meningitis. Table 2 shows that meningitis is responsible for cortical blindness in 72.2% of patients in the study group.

Visual return has commonly been noted in cases of cortical blindness.[1,8,9] Depending on severity, recovery may not start for weeks and may be partial or complete. Patients rarely end up with permanent blindness. Visual return has been observed to start peripherally, and then extending to the centre. This can be seen from recordings in the visual field charting.[1,7]

Though visual recovery is considered satisfactory, it is important that proper measures be taken for the prevention and management of key aetiological factors such as meningitis. This should also be considered in relationship with other neurological deficits such as hemiparesis, ataxia, hearing loss and impaired speech,[9] that may interfere at the early developmental stage of the child.

ACKNOWLEDGEMENT

I wish to express my gratitude to Dr. (Mrs.) C. N. Onwasigwe of the Department of Community Medicine, University of Nigeria Teaching Hospital, Enugu, for helping with the computer data analysis and word processing.

REFERENCES

1. Walsh FB. *Clinical Neuro-ophthalmology*. 3rd edition. William and Wilkins Company, Baltimore, 1969.

2. Singh BM, Morris LJ and Strobos RJ. Cortical blindness in puerperium. *JAMA* 1980; **243(11)**: 1134.
3. Symonds C and Mackenzie I. Bilateral loss of vision from cerebral infarction. *Brain* 1957; **80**: 415-454.
4. Bergman PS. Cerebral blindness. *Arch. Neurol.* 1957; **78**: 568-584.
5. Moel DI and Kwun YA. Cortical blindness as a complication of hemodialysis. *Journal of Pediatrics* 1978; **93(5)**: 890-891.
6. Drymalski WG. Cortical blindness. *Postgraduate Medicine* 1980; **67(4)**: 149-156.
7. Hoyt WF and Walsh FB. Cortical blindness with partial recovery following acute cerebral anoxia from cardiac arrest. *Archives of Ophthalmology* 1958; **60**: 1061-1068.
8. Weinberger HA, Woude RV and Maier HC. Prognosis of cortical blindness following cardiac arrest in children. *JAMA* 1962; **179(2)**: 134-137.
9. Ojinnaka NC and Iloeje SO. Neurological complications of childhood bacterial meningitis as seen in Enugu. *Nigerian Journal of Paediatrics* 1998; **25(2-4)**: 53-56.