Etiology of Partial Epilepsy

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Epilepsy is an important public health problem in developing countries. Interaction of various genetic, environmental and physiological factors gives rise to epilepsy. The Commission of International League Against Epilepsy (ILAE) has suggested that seizures should be classified according to presence or absence of a presumed causative or precipitating insult(1). The etiological factors of epilepsy differ markedly in children as compared to adults. However, there is paucity of literature on etiology of epilepsy, particularly in children from developing countries. This study was carried out to determine the etiology of partial (localization related) epilepsy in children.

Subjects and Methods

The records of children attending the Epilepsy Clinic of Kalawati Saran Children's Hospital were analyzed. Of these,

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Manuscript received: April 17,1997; Initial review completed: June 4,1997; Revision accepted: July 18,1997 131 cases fulfilled the inclusion criteria: *(i)* Had two or more unprovoked parital motor seizures; (n) Had complete historical data and had undergone relevant investigations; and *(Hi)* Were attending follow up regularly for 6 months or more. Patients presenting with partial seizures during neonatal period or acute central nervous system (CNS) insult were not included.

All patients underwent clinical evaluation and routine investigations including blood counts, Mantoux test, chest X-ray. Computed tomography (CT) and electroncephalography (EEG) was advised in all cases soon after presentation. Cerebrospinal fluid examination was done in cases of inflammatory granuloma in whom clear distinction could not be made between tuberculosis (TB) and neurocysticerosis (NCC) using radiological criteria (lesion more than 20 mm and irregular outline)(2) and evidence of tuberculosis elsewhere in the body.

All patients were treated with first line antiepileptic drug, *i.e.*, phenytoin or carbamazepine. A second anticonvulsant was substituted if the highest dose of the first drug failed to achieve seizure control. Antitubercular therapy was given to patients with tuberculosis. Albendazole was given to few cases of active neurocysticercosis (NCC). In addition, decongestive therapy was given to patients with granuloma showing mass effect or midline shift.

Results

A total of 131 cases (72 males and 59 females) were studied. The mean age of presentation was 6.7 years (range 1-12 years); of these 64.19% cases were less than

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six years of age. The clinical and radiological features of the patients are shown in Table I. CT examination was carried out in 116 patients. Of 15 patients who did not undergo a CT, 10 were of benign partial epilepsy of childhood and rest 5 had documented evidence of previous CNS infection or perinatal insult. Inflammatory granuloma was the commonest CT abnormality seen in 64 cases, of these 7 had multiple granulomas. Parietal lobe was the commonest site of single granuloma seen in 68% cases followed by frontal (20.9%), occipital (9.4%) and temporal lobe (1.7%). Six of these were diagnosed as tuberculous using radiological criteria(2) and supportive evidence of tuberculosis elsewhere. Cerebrospinal fluid

TABLE I–Clinical and Radiological Features of Study Patients

Features	No.	%
Type of seizures		
Simple partial	67	51.2
Complex partial	59	45.0
Partial with secondary generalization	5	3.8
Risk factors of epilepsy		~
(i) Family history of epilepsy	13	9.9
(ii) Perinatal insult	13	9.9
(iii) Trauma	5	3.8
(iv) Previous CNS infection	18	13.6
– previous TBM	11	8.3
 previous pyomeningitis/ encephalitis 	7	5.3
(v) Previous H/O febrile seizures	3	2.2
CT scan findings (n=116)		
Normal	36	31.0
Granulomas	64	55.2
Atrophy	10	8.6
Infarcts	5	4.3
Serpiginous calcification	1	0.9

ELISA for NCC was done in 21 cases and was positive in 28.1%. Magnetic resonance imaging done in 2 cases showed presence of larval stage of cysticercosis.

EEG examination revealed abnormalities in form of focal slow waves or focal spike/spike and wave discharge in 66.7% cases and focal abnormality with generalization in 4.6% cases. EEG was normal in 28.7% cases. Based on history, clinical evaluation and findings of various investigations, 100 (76%) patients had symptomatic epilepsy and 31 (23%) were idiopathic (*Table II*). The idiopathic group included 10 cases of benign partial epilepsy of childhood.

At 6 month follow up 102 (77.9%) patients were seizure free, 26 (19.8%) had decreased frequency of seizures and 3 (2.2%) had no response to antiepileptic drugs.

Discussion

Partial epilepsy constitute a larger percentage of the epilepsy type seen in children. In developing countries partial epilepsy is reported to be more frequent in all

TABLE	II-Etiological	Profile of	Partial E	pilepsy
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Etiology	No.	%
I. Symptomatic Epilepsy	100	76.3
(i) Inflammatory ganulomas	64	
(ii) Previous CNS infection	18	
 previous TBM pyomeningitis/ 	11	
encephalitis	7	
(iii) Perinatal insult	12	
(iv) Trauma	5	
(v) Sturge Weber syndrome	1	
II. Idiopathic Epilepsy	31	23.7
(i) Benign Partial epilepsy of childhood	10	

ages due to higher incidence of symptomatic epilepsy in the tropics caused by cortical damage resulting from host of factors(3).

The etiology of epilepsy could be determined in all cases in the present study with a combination of history, examination and investigations carried out. Historical data revealed a high incidence of previous CNS infection in as many as 13.6% of cases. History of previous febrile seizures was seen in 2.2% cases. This is in contrast to the earlier observations(4) in which CNS infection was seen in only 4.6% of cases(4). They also demonstrated a higher incidence of perinatal insult 29.1% in contrast to 9.9% in present study. The differences in relative proportion is possibly because of higher incidence of infection in the study group and inadequate and at times unreliable data about perinatal period.

EEG correlated with CT scan in as many as 71.3% of cases. EEG is particularly useful to diagnose benign epilepsy of childhood in the group of partial epilepsy since they have an excellent prognosis. CT examination was carried out in 116 patients. CT abnormality was seen in 69.1% cases in the present study. Other studies from India which included both adults and children have also shown CT to be abnormal in 50-70% of cases(5,6). The commonest abnormality noted on contrast enhanced CT was an inflammatory granuloma seen as hypondense lesion surrounded by hyperdense lesion. In a previous study on focal epilepsy which included both children and adults, similar lesions were seen in 40.9% of children below 15 years of age(5). A study in which excision biopsy was done to evaluate the histology showed that 90% of these lesions are due to NCC(7). In the present study the differentiation between tuberculoma and NCC was made by the suggested radiological criteria(2), immunological tests of NCC on CSF and supportive evidencecof tuberculosis elsewhere in the body. Immunological tests such as enzyme linked immunosorbent assay in CSF for NCC was positive in less than 1/3rd cases tested. However, negative tests do not exclude diagnosis of NCC since ELISA is negative in inactive cases of neurocysticercosis(8). A follow up of at least 6 months in all these cases revealed the benign and self limiting nature of these single inflammatory granulomas. Magnetic resonance imaging offers better resolution to detect the scolex and often reveals more cyst than does CT(9). Being an expensive investigation and bearing in mind the benign course of most of these cases, it is presently reserved for cases in whom no clear distinction can be made on the basis of all investigations.

In the present study, 62.5% cases of partial epilepsy could be attributed to past or recent infection or infestation. This is in contrast to studies of developed countries where perinatal insult(4) and cortical developmental abnormalities(10) are the major cause of symptomatic epilepsy.

Poor sanitation may be the single most important social factor underlying the increased prevalence of NCC and subsequent epilepsy in tropical countries(11). The subjects of the present study were mainly from low socioeconomic status of the society in whom both bacterial infection as well as parasitic infestations are more likely. Besides the study was hospital based with an inherent selection bias towards more severe cases of epilepsy coming to hospital. Prospective studies are required to determine the extent of this problem in the community.

REFERENCES

1. Commission on Classification and Terminology of International League Against Epilepsy. Proposal for revised classification of epilepsies and epileptic syndromes. Epilepsia 1989; 30: 389-399.

- Rajshekar V, Haran RP, Prakash GS, Chandy MJ. Differentiating solitary small cysticercus granulomas and tuberculomas in patients with epilepsy. Clinical and computerized tomographic criteria. J Neurosurg 1993; 78: 402-407.
- Senanayake N, Roman GC. Epidemiology of epilepsy in developing countries. Bull WHO 1993; 71: 247-258.
- 4. Scarpa P, Carassini P. Partial epilepsy in childhood clinical and EEG study of 261 cases. Epilepsia 1982; 23: 333-341.
- Wadia RS, Makhale CN, Kelkar AV, Grant KB. Focal epilepsy in India with special reference to lesions showing ring and disc like enhancement on contrast computed tomography. J Neurol Neurosurg Psychiatry 1987; 50: 1298-1301.
- Sethi PK, Kumar BR, Mohan VS, Mohan V. Appearing and disappearing CT scan abnormalities and seizures. J Neurol Neurosurg Psychiatry 1985; 43: 866-869.

- Chandy MJ, Rajshekar V, Ghosh S, Prakash S, Joseph T, Abraham J, *et al.* Single small enhancing CT lesions in Indian patients with epilepsy. Clinical, radiological and pathological consideration. J Neurol Neurosurg Psychiatry 1991; 54: 702-705.
- 8. Zini D, Farrell VJR, Wadee AA. The relationship of antibody levels to clinical spectrum of human neurocysticerosis. J Neurol Neurosurg Psychiatry 1990; 53: 556-561.
- 9. Jabbari B, Gunderson CH, Wippold F, Citrin C, Sherman J, Bartoszek D, *et al.* Magnetic resonance imaging in partial complex epilepsy. Arch Neurol 1986; 43: 869-872.
- 10. Blume WT. Clinical profile of partial seizures beginning at less than 4 years of age. Epilepsia 1989; 30: 813-819.
- Commission on Tropical Diseases of International League Against Epilepsy. Relationship between epilepsy and tropical diseases. Epilepsia 1994; 35: 89-93.

Glucose-6-Phosphate Dehydrogenase Deficiency in Neonatal Hyperbilirubinemia in a South Indian Referral Hospital

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Glucose-6-phosphate dehydrogenase (G-6-PD) is essential to maintain stability of red blood cells(1). The inherited deficiency of this enzyme may manifest as congenital nonspherocytic hemolytic anemia, druginduced hemolytic anemia or hemolytic disease of the newborn.

G-6-PD deficiency is the most prevalent enzyme deficiency worldwide. Routine screening of children and adults in various parts of India indicates that the prevalence

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