

Intracranial Structural Lesions in Young Epileptics: A Computed Tomographic Study

M. Kapoor
B. Talukdar
Veena Chowdhury
Vinod Puri
B. Rath

Intracranial structural lesions (ISL), lesions associated with morphological changes in the brain or the cranial cavity, are important cause of symptomatic epilepsy. Knowledge of such ISLs in the past were based on selective neuroradiologic investigations, observations at surgery, pathological examination of surgically resected specimens and autopsy findings. Modern neuroimaging techniques like computed tomographic scan (CT) and magnetic resonance imaging (MRI) scans have made the diagnosis of ISLs much easier. A good anatomic diagnosis of such lesions can be obtained through these techniques, although accurate pathologic diagnosis is not always possible. A study was done to find the pattern of ISLs in epileptic children aged 3 years or less using CT scan. This age group was selected as the incidence of developmental anomalies is relatively high in the younger age group and this might have a bearing on the frequency and type of ISLs associated with seizures in this age group.

From the Departments of Pediatrics, Radiodiagnosis and Neurology, Maulana Azad Medical College and Associated Lok Nayak and G.B. Pant Hospital, New Delhi 110 002.

Reprint requests: Dr. B. Talukdar, Professor of Pediatrics, Department of Pediatrics, Maulana Azad Medical College, New Delhi 110 002.

*Manuscript received: July 7, 1997;
Initial review completed: August 28 1997;
Revision accepted: January 2nd, 1998.*

Subjects and Methods

One hundred consecutive epileptic children aged between 1 month to 3 years admitted into our Pediatric wards during a period of 1 year were studied. Cases with at least two attacks of unprovoked seizures were included in the study. Febrile seizures and acute CNS insults were meticulously excluded. All the cases were subjected to CT scan of the head with and without contrast and also to electroencephalography (EEG). Selective investigations were done to find the possible cause of the ISLs.

Results

The age and sex distribution of the 100 cases are shown in *Table I*. The seizures, typed as per the International League Against Epilepsy (ILAE) classification(1), were generalized in 80 (80%) cases and partial in 20 (20%) cases. Twenty four (24%) cases had abnormal CT scans showing some ISL. The types of ISLs found in these abnormal scans are shown in *Table II*.

ISLs were found in 8/38 (21%) cases who were < 1 year of age and 16/62 (25.8%) cases who were between 1-3 years of age. These were also observed in 17/62 (27.4%) cases who had onset of seizure before 1 year of age and 7/38 (18.4%) cases who had onset of seizure between 1-3 years of age. ISLs were found in 14/20 (70%) cases of partial seizure and 10/80 (12.5%) cases of generalized seizure; 11 (55%) cases of partial seizures were of complex partial type and 69 (86.2%) cases of generalized seizures were of tonic and tonic-clonic types. Such ISLs were also found in 19/39 (48.7%) cases having abnormal neurologic findings which included cases with developmental retardation.

Primarily one type of lesion was seen in 18 of these scans, the rest of 6 scans showed a combination of lesions. The lesions were

TABLE I-Age and Sex Distribution.

Age groups	No. of cases (%)	Male (%)	Female (%)
1 mo - 1 yr	38 (38)	26 (26)	12 (12)
1 yr - 2 yrs	21 (21)	16 (16)	5 (5)
2 yrs - 3 yrs	41 (41)	33 (33)	8 (8)
Total	100	75	25

TABLE II-Details of ISLs in 24 Abnormal CT Scans.

Lesion	No.
Atrophy	9
Diffuse	6
Focal	3
Ring enhancing lesion	6
Single	5
Multiple	1
Dilated ventricle	6
Infarct	4
Aqueductal stenosis	3
Porencephaly	2
Calcification	2
Periventricular	1
Basal ganglia (bilateral)	1
Hydranencephaly	1
Dandy-Walker variant	1
Gyral enhancement	1
Prominent thalami	1
Basal ganglia hypodensity	1

Some CT scans had more than one abnormality.

focal or unihemispheric in 10 instances, these being ring enhancing lesions, atrophy, infarct and porencephaly. Seizures were partial in cases with focal atrophy (*Fig. 1*), ring enhancing lesions (*Fig. 2*), dilated ventricle (one case), infarct, porencephaly and gyral enhancement; these were generalized in cases with generalized atrophy, dilated ventricles, hydranencephaly, Dandy-Walker variant,



Fig. 1 CT showing atrophy (Right frontal) in a 17 months old child who had complex partial seizure

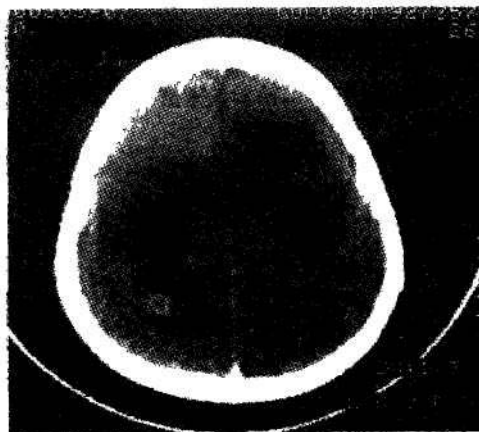


Fig. 2. CT showing ring enhancing lesion (left parieto-occipital) in a 3 year old child who had complex partial seizure.

basal ganglia calcification, periventricular clarification, prominent thalami and hypodense basal ganglia.

Investigations were useful in finding the probable cause of these ISLs in 8 (33.3%) instances. Five cases of single ring enhancing lesions, were suspected to be tuberculomas based on positive tuberculosis screen. All these cases had positive Mantoux test besides some other evidence of tuberculosis. The second child had positive FNAC of a cervical gland as well as positive tubercular serology (IgM-ELISA). The third child had hilar lymphadenopathy on X-ray chest. The fourth child had positive contact survey. In all these cases CSF was normal and ELISA for neurocysticercosis, was negative both in serum and CSF. Accurate etiologic diagnosis of such ring enhancing lesions are difficult at present in the absence of fool-proof laboratory methods and also routine stereotactic biopsy not being possible. In the present setting, therefore, cases of ring enhancing lesions in cranial CT with some evidence of tuberculosis in the body and at the same time without any serologic evidence or clear neuroimaging features (*i.e.*, Scolex) of neurocysticercosis can be considered as tuberculomas. One case of multiple ring enhancing lesions was suspected to be due to neurocysticercosis based on characteristic CT findings and positive serology (both CSF and blood). Serology for cytomegalovirus was positive in the infant with periventricular calcification and infantile spasms. In another infant with aqueductal stenosis serology was positive for toxoplasmosis. In the other 16 cases (66.6%), no cause could be found.

Discussion

The present study, suggests that a sizeable proportion of unselected epileptic infants and young children have associated

ISLs. A strictly comparable study restricted to this age group and including any seizure type is not available. There are, however, few studies which have included any seizure type like the present one, but the age group included was different which varied from neonate to adolescent(2-4). The incidence of ISL reported in these studies varied from 30-33%. The 24% incidence of ISL observed in the young epileptics in the present study suggests that the incidence of ISL associated with epilepsy is relatively higher in the young than the older children.

Bachman *et al.*(2) who studied 98 children aged 3 months to 20 years (mean age 11.0 years) including any type of seizure, reported ISL in 30% of cases. The type of ISLs observed by them were generalized atrophy (6 cases), focal atrophy (8 cases), hydrocephalus (5 cases), porencephaly (2 cases), recurrent tumor (2 cases) and post-operative changes (3 cases). Yang *et al.*(3) in a similar study of 256 cases (neonate to 18 years old) observed ISL in 33% cases and the important pattern of ISLs observed by them were generalized atrophy, focal atrophy, dilated ventricles, porencephalic cyst, tuberous sclerosis, agenesis of corpus callosum, hemiatrophy, calcification, tumor, periventricular leukomalacia, bleed, possible venous thrombosis, and possible degenerative brain disease. The findings of the present study also are similar to these studies so far the basic pattern of ISLs are concerned. There was however no case of tumor in the present study which is obviously due to the age factor (children upto the age of 3 years only were included in this study). Another deviant feature in this study was the ring enhancing lesions which were not seen in these studies.

The spectrum of ISLs associated with epilepsy could be diverse like static and developmental anomalies, sequelae of trauma, anoxia, infections and vascular

accidents and slow growing tumors(5,6). Metabolic disorders and degenerative brain diseases also can be associated with such lesions in CT(7). There are several cranial CT studies in children with seizure disorders aimed ultimately at finding associated ISLs. However, these have different primary objectives and methodologies like evaluation of specific seizure types, observations in relation to age at onset, inclusion of cases with wider age range and so on(8-13). The ISLs observed in these studies include atrophy (focal, diffuse, hemispheric), infarct, dilated ventricles, hydranencephaly, Dandy-Walker malformations and variants, aqueductal stenosis, porencephaly, gyral enhancement, tuberous sclerosis, hamartoma, phakoma and tumor with varying incidence. The type of ISLs in these studies also bear a close resemblance to the earlier mentioned reports. This indicates that the same type of ISLs can be associated with different types of seizures and be present in different age groups.

An interesting feature in the present study is the ring enhancing lesions in young epileptics. Ring enhancing lesions, however, have been reported in Indian children with seizures in CT studies done with specific objectives(14-17). Such lesions have become a common CT finding in cases of seizure disorders in the Indian subcontinent(18). Majority of these lesions, especially the single and small ones, are presently thought to be due to neurocysticercosis and some to be due to tuberculomas besides other possibilities, based on a few studies involving biopsy done in recent times(16,19). Irrespective of the cause, these ring enhancing lesions are usually slow growing and act as space occupying lesions and after healing often leave behind an epileptogenic scar.

It was difficult to implicate individual type of ISL to specific seizure type; same

type of lesion was found to be associated with both partial as well as generalized seizures. However, focality of the lesions was an important factor in causation of partial seizure. Earlier workers(2,3) had also observed a high incidence of partial seizure with focal lesions.

In consonance with earlier reports(2,3), parenchymal atrophy was the commonest of all the ISLs. Bachman *et al.*(2) observed atrophy in 13 cases and Yang *et al.*(3) in about 20 cases in their series. In several other related studies also atrophy occurred in relatively high frequency. Parenchymal atrophy could be focal, diffuse or involve one hemisphere or a lobe. This may occur alone or may occur in combination with other anomalies. These atrophic lesions appear to be an important cause of cerebral dysrhythmia and epilepsy.

Looking at the basic type of ISLs, majority of them (66.6%) in the present study were static and developmental in nature except eight cases (33.3%) of probable infective etiology (tuberculosis, neurocysticercosis, cytomegalovirus infection and toxoplasmosis). In the earlier studies(2,3) also, a high incidence of such lesions was reported although the etiology of the lesions was not evaluated. Similar observations were reported even in those studies done with different aims, objectives and methodologies. These findings suggest that intracranial static and developmental anomalies are the most important cause of epilepsy in children specially infants and young ones. Some such anomalies are presently detectable with CT and MRI and probably many more will become detectable in future with advancement of the imaging techniques.

REFERENCES

1. International League Against Epilepsy. Proposal for revised classification of

- epilepsies and epileptic syndromes. *Epilepsia* 1989; 30: 389-399.
2. Bachman DS, Hodges III FJ, Freeman JM. Computerized axial tomography in chronic seizure disorders of childhood. *Pediatrics* 1976; 58: 828-832.
 3. Yang PJ, Berger PE, Cohen ME, Duffner PK. Computed tomography and childhood seizure disorders. *Neurology* 1979; 29:1084-1088.
 4. Coe CJ, Lee YH. Organic disorders in children with epileptic seizures. *Acta Paediatr Jpn* 1989; 31: 267-272.
 5. Kim JH. Pathology of seizure disorders. *Neuroimaging Clin North Am* 1995; 5: 527-545.
 6. Farmer TW, Greenwood RS. Paroxysmal disorders. *In: Pediatric Neurology*, 3rd edn. Ed. Farmer TW. Philadelphia, Harper and Row, 1983; pp 205-264.
 7. Barkovich AJ. Toxic and metabolic brain disorders. *In: Pediatric Neuroimaging* 2nd edn. Ed. Barkovich AJ. New York, Raven Press, 1995; pp 55-105.
 8. Gastout H. Conclusions: Computerized transverse axial tomography in epilepsy. *Epilepsia* 1976; 17: 337-338.
 9. Lagenstein D, Kuhne D, Stanowsky HJ. Computerized cranial transverse axial tomography in 145 patients with primary and secondary generalized epilepsies: West syndrome, myoclonus static petitmal, absence epilepsy. *Neuropædiatrie* 1979; 10:15-28.
 10. Scollo-Lavizzari G, Balmer C. Electroencephalography and computerized transaxial tomography in patients with temporal lobe epilepsy. *Eur Neurol* 1980; 19: 33-38.
 11. Ladurner G, Fritsch G, Sager WD, Itiff LD, Lechner H. Computer tomography in children with epilepsy. *European Neurol* 1980; 19:180-184.
 12. Fritsch G, Ebner F, Schneider B. Computed tomography in partial epilepsy in childhood. *Eur Neurol* 1988; 28: 306-310.
 13. Minford AM, Forsythe WL. Computed tomographic findings in partial seizures. *Arch Dis Child* 1992; 67: 693-696.
 14. Kumar R, Kumar A, Kohli N, Pant MG, Bovil YC, Sharma B. Ring or disc-like enhancing lesions in partial epilepsy in India. *J Trop Pediatr* 1990; 36:131-134.
 15. Kalra V, Sethi A. Childhood neurocysticercosis: Epidemiology, diagnosis and course. *Acta Paediatr Jpn* 1992; 34: 365-370.
 16. Sethi PP, Wadia PS, Kiyawat DP, Inchaporia NR, Kothari SS, Singla SA, *et al.* Ring or disc enhancing lesions in epilepsy in India. *J Trop Med Hyg* 1994; 97: 347-353.
 17. Singhi P, Singhi S, Ramanathan RP, Bharti S. Neurocysticercosis in focal epilepsy in North India. *Epilepsia* 1995; 36: 156.
 18. Padma MV, Jain S, Maheswari MC. Intracranial space occupying lesions of infective origin. *In: Essentials of Pediatric Neurology*, 1st edn. Ed. Talukdar B. New Delhi, New Age International, 1997; pp 326-338.
 19. Rajasekhar V. Etiology and management of single small CT lesions in patients with seizures: Understanding a controversy. *Acta Neurol Scand* 1991; 84: 465-470.
-