

## Vanishing CT lesions in epilepsy

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### Summary

Experience with computed tomography (CT) in patients with epilepsy has drawn attention to specific type of cerebral lesions widely prevalent in this part of the world. The lesions disappear spontaneously while patients receive only anticonvulsant therapy. The exact nature of these lesions is not yet understood. One-hundred and twenty-two such patients (M 77; F 45) were studied. Their age ranged from 3 to 57 (mean  $21.8 \pm 12.7$ ) years. Seizures were partial motor in 86 (70.5%), generalised tonic clonic in 31 (25.4%), complex partial in 4 and atonic in one case. Postictal hemiparesis was seen in 6 patients who completely recovered within 5 min to 7 days. One hundred patients underwent EEG examination. It was abnormal in 84 cases; 40 patients showed localisation which correlated with CT findings. Plain CT showed low attenuation lesions in all cases. Morphology on contrast enhancement was ring lesion in 50 (40.9%), disc lesion in 47 (38.5%), target lesion in 3 (2.5%), mixed lesion in 2 (0.8%), no enhancement in 18 (14.8%) and a speck with oedema in 2 (0.8%) cases. All lesions were cerebral, present at cortical-subcortical junction. The first follow-up scan was performed in 78 (63.9%) patients within 6–12 weeks. It showed complete disappearance of the lesion in 47 (60.3%) cases. Reduction in size of the lesion and surrounding oedema was observed in 24 (30.8%) cases. The follow-up scans in these 24 cases revealed disappearance of lesion in 11 cases and almost complete resolution of the lesion with a remaining speck in 13 cases. Cerebrospinal fluid (CSF) was normal in 95 out of 100 cases in which it was performed. All patients received only antiepileptic drugs except 5 cases with unresolving lesions and ancillary evidence of tuberculosis who were given additional antitubercular chemotherapy. The vanishing CT lesions are unlikely to be a postictal phenomenon since these are rarely encountered in patients with seizures from the Western countries. These appear to be inflammatory and self-limiting in nature although they may not be due to single aetiology.

### Introduction

Epileptic seizures may be idiopathic or associated with structural lesions of the brain due to congenital disorders, trauma, infections, vascular disorders and degenerative diseases. Those with structural lesions are best evaluated by computed tomography (CT). CT abnormalities in all these cases are usually persistent unless specific therapy is given. Extensive CT studies of epilepsy in the Western hemisphere have not revealed CT lesions which disappear spontaneously. In a series of 401 cases of epilepsy, CT was normal in 96% cases with primary generalised epilepsy and a cerebral lesion

was found in 61% cases of partial seizures with generalisation (Gastaut and Gastaut 1976). Similar findings have been reported in other series (Bordanoff et al. 1975; Gall et al. 1976; Scollo-Lavizzari et al. 1977 and Bauor et al. 1980). None of the CT lesions disappeared spontaneously in these series. Feinstein et al. (1990) have reported two cases of disappearing brain lesions in association with epilepsy and psychosis. A ring or disc lesion on contrast enhanced CT (CECT) is the commonest accompaniment of focal epilepsy reported from the Indian subcontinent. Many such patients are put on antituberculous therapy empirically. These are labelled as tuberculomas or some other inflammatory lesions (Wadia et al. 1987; Ahuja et al. 1989), as the follow-up scans show regression or disappearance of the lesions. However, we have observed clearing of such lesions on CT without any specific therapy in a large number of cases. This has raised many questions

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regarding the nature of these lesions and the management of such cases. We report our findings on focal CT lesions and their follow-up in a series of 122 cases with seizures.

### Patients and methods

One-hundred and twenty-two consecutive patients with different types of seizures who exhibited focal CT abnormality in the form of low attenuation, ring or disc enhancing lesion were included in the study. Patients with papilloedema or progressive neurological deficit were excluded. All patients were attending the Neurology Clinic of our Institute and most of them were admitted for detailed examination. Haemogram, ESR, blood biochemistry, EEG, chest X-ray and Mantoux test were routine investigations. CSF examination carried out in 100 cases included cell count, malignant cytology, protein estimation, globulin content, electrophoreses, gram stain and culture for pyogenic organisms, acid fast bacilli, fungal studies and cysticercus haemagglutination (CHA). All patients were treated with anticonvulsants, e.g. phenytoin, carbamazepine or phenobarbitone in appropriate dosage and were followed up at a 1–3 month interval. A second CT was performed within 6–12 weeks and was repeated at 3–6 months if the lesion persisted. Antiepileptic therapy was tapered off in those who remained seizure-free for 3 years and whose CT lesions had vanished.

### Results

There were 77 male and 45 female patients with an age range of 3–57 ( $21.8 \pm 12.7$ ) years and majority

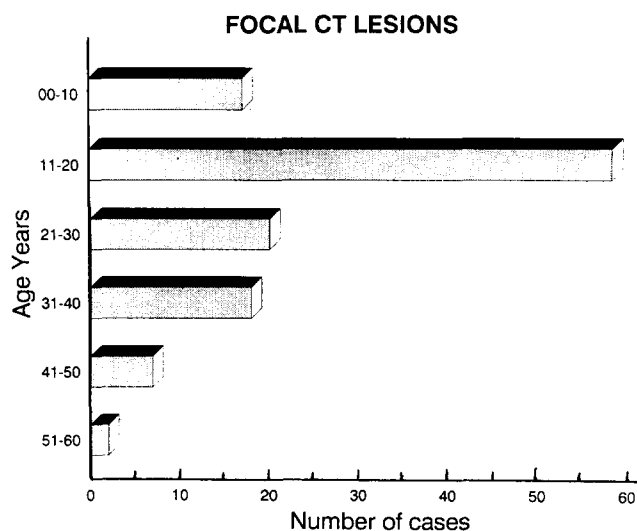


Fig. 1. Age distribution of patients with focal lesions on CT scan.

TABLE 1

TYPE OF SEIZURES AND POSTICTAL DEFICIT IN PATIENTS WITH CT LESIONS ( $n = 122$ )

Type of seizures	No.	%	Postictal deficit
Right partial motor	48	39.3	4
Left partial motor	38	31.1	2
Generalised tonic-clonic	31	25.4	Nil
Complex partial	4	3.3	Nil
Atonic	1	0.8	Nil

(78.6%) being in the 11–20 years age group (Fig. 1). All patients presented with seizures and none of them had any preceding illness in the form of fever, exanthema, features of raised intracranial pressure, history of birth anoxia, head injury, febrile convulsions, tuberculosis or any other infective disease. *Seizures:* Eighty-six patients (70.5%) presented with partial seizures including 20 (16.4%) cases of simple partial motor seizures and 62 (50.8%) patients with secondary generalisation. Partial sensorimotor seizures were seen in 3 patients and pure sensory seizure in one case. Thirty-one patients (25.4%) had generalised tonic-clonic seizures and one of them had presented with status epilepticus. Complex partial seizures were present in 4 cases and atonic seizures in one case (Table 1). The frequency of various type of seizures and postictal deficit in those patients in whom CT lesions vanished on repeat CT scan is given in Table 2. There was no case of absence seizures or myoclonic jerks.

*Neurological deficit and course.* Postictal hemiparesis was seen in 6 patients. It showed complete recovery in 5 min to 7 days. One patient aged 20 years with an occipital lesion had visual field defect. Fundus examination revealed a choroid tubercle in another 10-year-old female child. None of the patients had papilloedema. Most of the patients (95%) had one to two seizures before they were seen in the Neurology clinic and following therapy had no further seizures. The seizures were controlled with anticonvulsants in the rest of the patients within 3–6 months. Seizure control was excellent both in children and adults. None of the

TABLE 2

TYPE OF SEIZURES AND POSTICTAL DEFICIT IN PATIENTS IN WHOM CT LESIONS VANISHED ON REPEAT CT SCAN ( $n = 71$ )

Type of seizures	No.	%	Postictal deficit
Right partial motor	26	36.6	2
Left partial motor	21	29.6	1
Generalised tonic-clonic	20	28.2	Nil
Complex partial	3	4.2	Nil
Atonic	1	1.4	Nil

TABLE 3  
LOCATION OF FOCAL LESIONS ON HEAD CT SCAN ( $n = 122$ )

Site of lesion	No. of cases	%
Parietal	47	38.5
Frontal	37	30.3
Frontoparietal	17	13.9
Parieto-occipital	7	5.7
Occipital	9	7.4
Parasagittal	5	4.1

patients had subcutaneous nodules suggestive of cysticercosis or systemic evidence of vasculitis.

**CSF examination.** CSF was examined in 100 cases. It was normal in all except 5 cases. The abnormality included elevated protein (100 mg%) in all the 5 cases and lymphocytic pleocytosis (20 cells/mm<sup>3</sup>) in one case. The immunological and microbiological tests were normal in these 5 patients.

**EEG** One hundred patients underwent EEG examination. Forty showed localisation in the form of sharp waves, spike and wave, focal theta slowing or polymorphic delta slowing which correlated with CT findings. Interictal evidence of generalised seizure activity was seen in 44 records. EEG was normal in 16 cases.

**Chest X-ray and Mantoux test.** Chest X-ray was performed in all the cases. It did not reveal evidence of pulmonary tuberculosis in any patient. Mantoux test was done in 92 cases. It was strongly positive in 5 cases. All these patients had abnormal CSF and one of them had choroid tubercle.

**Other investigations.** Erythrocyte sedimentation rate was normal in all cases except 5 patients with abnormal CSF in whom it was elevated (30 mm/1st hour). Skull X-ray and CSF cysticercous serology was normal in all the cases.

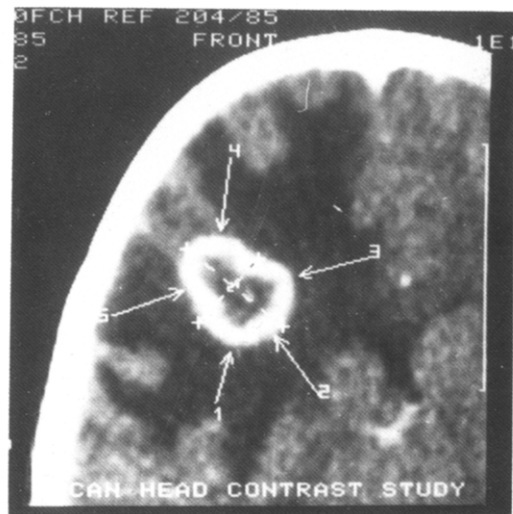


Fig. 3. Left frontal ring lesion in CECT near junction of grey and white matter and surrounding oedema in a 10-year-old girl with generalised tonic-clonic seizures.

**CT.** Plain and CECT were performed in all the patients mostly within 1–2 weeks following the last seizure (mean 12 days; range 1–60 days). Plain CT showed low attenuation lesion with ill-defined margins (Fig. 2i) in all except two cases who had a small speck of high density surrounded by an area of low attenuation. CECT revealed enhancing lesion within low density area, mainly at the junction of grey and white matter (Figs. 2j and 3). The lesions were situated in parietal lobe (38.5%), frontal lobe (30.3%), frontoparietal (13.9%), occipital/parieto-occipital (13.1%) and parasagittal regions (4.1%) in that order (Table 3). The location of CT lesions in patients in whom the focal lesions vanished on repeat CT scan is given in Table 4. Simultaneous occurrence of two lesions, one each in ipsilateral frontal and occipital lobes in two cases, and contralateral frontal and occipital lobes in one case

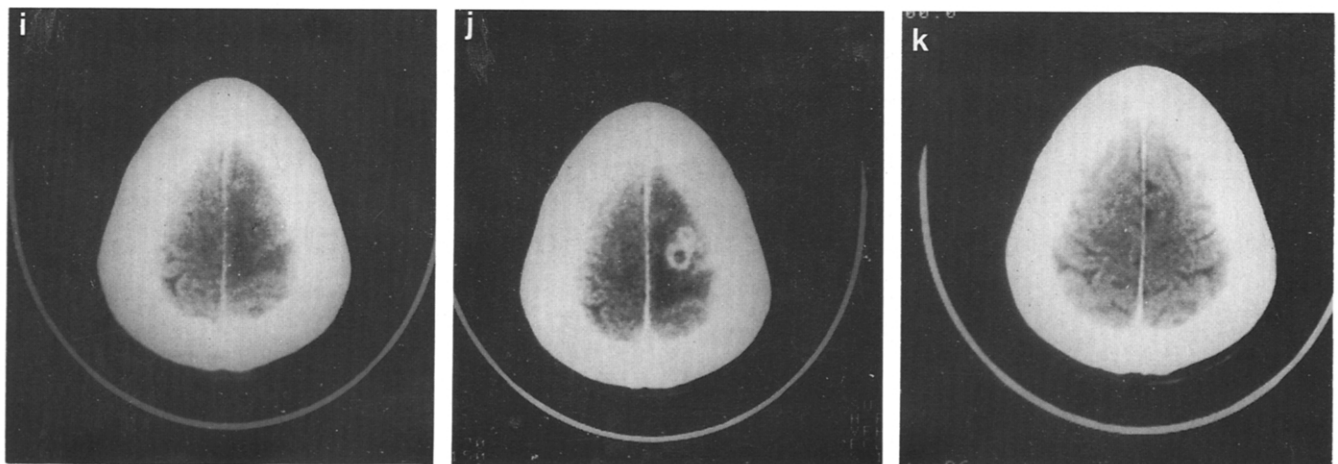


Fig. 2. CT scan of a 4-year-old male child with left partial motor seizures showing: (i) low attenuation area in right frontal region in plain scan; (j) a small ring lesion at the junction of grey and white matter on contrast enhancement and (k) complete disappearance of the lesion after 7 weeks.

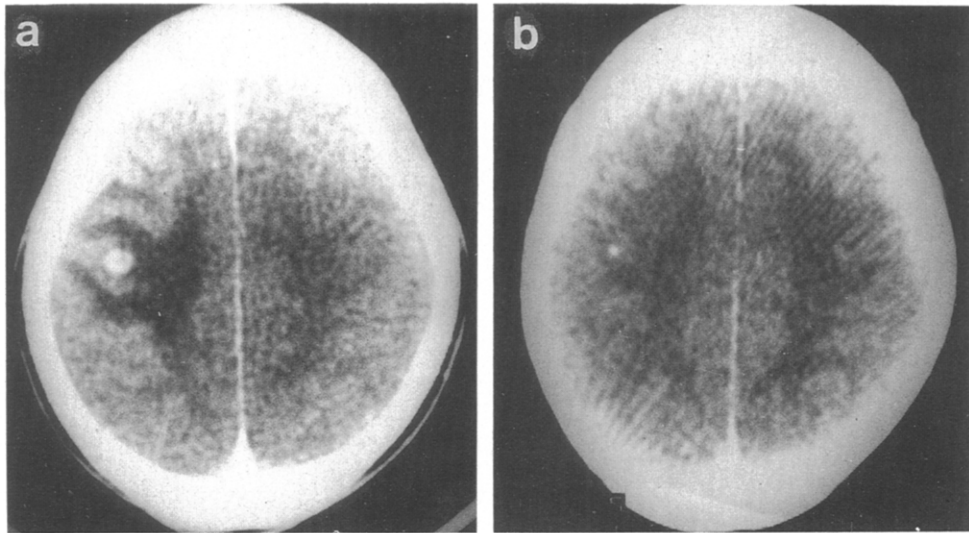


Fig. 4. (a) Small ring lesion in posterior frontal region in CECT of a 40-year-old male with right partial motor seizures involving face and tongue; (b) repeat CT 4 years later showing a small high attenuation speck in the same region. Patient was seizure-free during this period.

was also observed. Two of these patients presented with generalised seizures and one with right partial motor seizures. There was no lesion in the region of

basal ganglia, brainstem or cerebellum. There was no evidence of midline shift. Subarachnoid space, ventricular system and cisterns were normal.

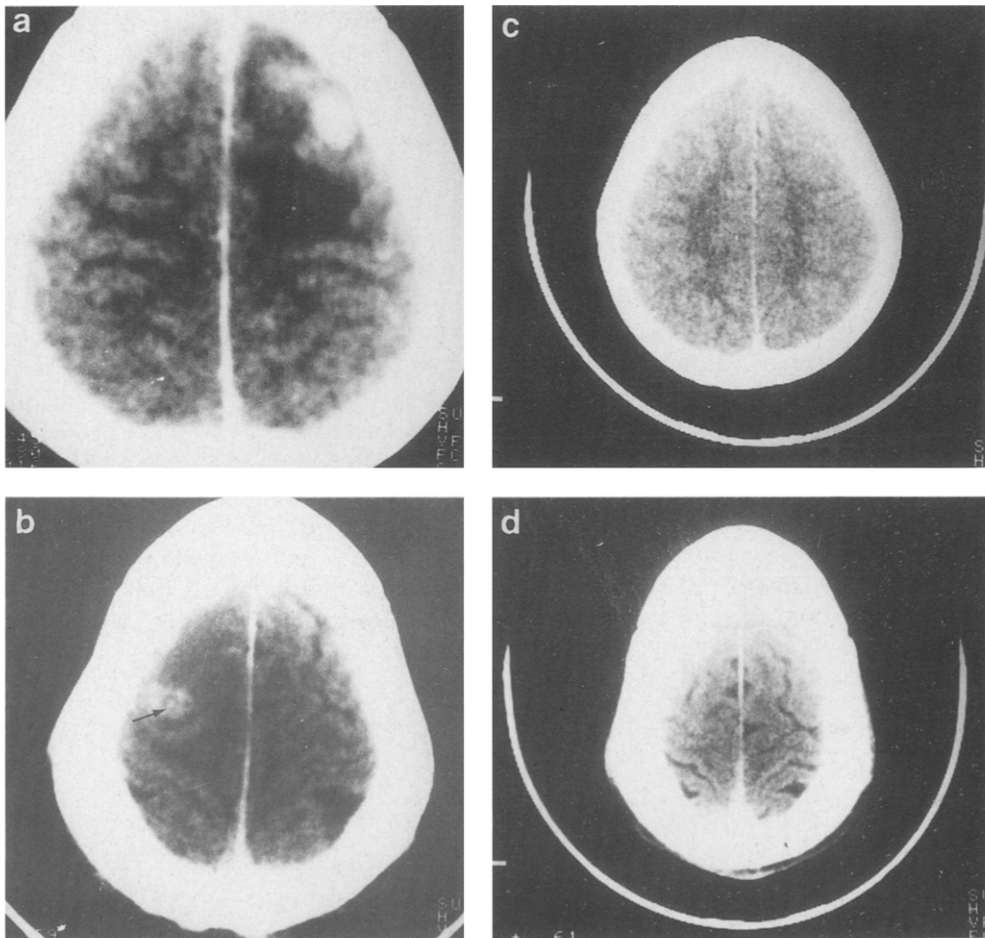


Fig. 5. CECT showing (a) disc and (b) target type of lesions in a 13-year-old female and a 55-year-old male respectively. Lesions completely disappeared on repeat CT after 8 weeks (c and d).

TABLE 4  
LOCATION OF CT LESIONS IN PATIENTS IN WHOM THE LESIONS VANISHED ON REPEAT CT SCAN (n = 71)

Site of lesion	No. of cases	%
Parietal	25	35.2
Frontal	20	28.2
Frontoparietal	10	14.1
Parieto-occipital	7	9.9
Occipital	7	9.9
Parasagittal	2	2.9

Table 5 shows the morphology of lesions on CECT. Ring or disc enhancing lesion was the commonest form (79.5% cases) (Figs. 2–5). Target lesion and mixed attenuation was observed in three and two cases respectively, and lesions remained unenhanced in 18 cases (Fig. 5).

The follow-up CT were available in 78 patients, usually within 6–12 weeks. The interval between initial and last follow-up CT was  $11.3 \pm 7.7$  weeks (range 6 weeks to 4 years). Findings on repeat CT, summarised in Table 6, were as follows: CT lesions completely disappeared in 47 (60.3%) patients (42 within 12 weeks) including two patients with two lesions. In two patients lesions had disappeared as early as 6 weeks and in one case after 10 months. All these patients showed good control of seizures with antiepileptic drugs. Reduction in size of lesion and surrounding oedema was observed in 24 (30.8%) cases. The follow-up scans revealed disappearance of lesions in 11 cases and almost complete resolution of the lesions with a remaining speck in 13 cases (Fig. 4). Thus the lesions disappeared in 71 out of 78 cases and there was no regression of lesion in 7 cases. Most patients received only antiepileptic drugs. A subgroup of patients who required antituberculous therapy is discussed later.

*Non-vanishing lesions*

Seven patients showed no regression of lesion in various forms on first follow-up CT scan. Five patients with unchanged or increasing lesions (disc, 2; ring, 3) at 3 months also had a positive Mantoux test and/or CSF abnormality and were started on standard antitubercu-

TABLE 5  
FOCAL CT LESIONS SEEN ON FIRST CT SCAN IN PATIENTS WITH SEIZURES (n = 122)

	No.	%
Plain CT		
Low attenuation	120	
High density speck with oedema	2	
Total	122	
Contrast enhanced CT		
Ring lesions	50	41.0
Disc lesions	47	38.5
Target lesions	3	2.5
Mixed lesions	2	1.6
No enhancement	20	16.3
Total	122	

lous treatment only on non-disappearance of the lesion. Follow-up revealed gradual reduction and disappearance of lesion in 3 out of the 5 cases (Fig. 6) and a residual speck in one case after 6 months and one year of repeat CT. One of these cases was lost to follow-up. In two cases (age 14 years and 10 years) the initial ring lesion was replaced by a small cystic lesion with central high attenuation (plain CT) resembling a cysticercus cellulosae cyst (Fig. 7). The last follow-up in these cases was done at 6 months and 3 years respectively.

*Treatment*

All patients received anticonvulsant therapy. Two patients complained of headache and the CT showed significant cerebral oedema. They were treated with oral glycerol for 2 weeks. Antituberculous therapy was instituted in 5 cases. Anticonvulsants were tapered off after a 3-year seizure-free period in 5 patients. None of them had recurrence of seizures in 9–12 months of further follow-up. The majority of other patients are seizure-free but have not completed the 3-year seizure-free period.

*Illustrative case histories*

*Case 1.* An 8-year-old male child presented with 3 episodes of generalised tonic-clonic seizures in 24 h. Neurological examination was normal. EEG showed

TABLE 6  
EVALUATION OF FOLLOW-UP CONTRAST ENHANCED CT SCANS IN PATIENTS WITH SEIZURES AND FOCAL CT ABNORMALITIES ON FIRST CT SCAN (n = 78)

	Ring lesion	Disc lesion	Target lesion	Mixed lesion	Unenhanced lesion	Total
Disappeared	19	20	1	2	5	47
Regressed	3	4	1	–	3	11
Speck	6	4	1	–	2	13
Persisted	3	2	2	–	–	7
Total	31	30	5	2	10	78

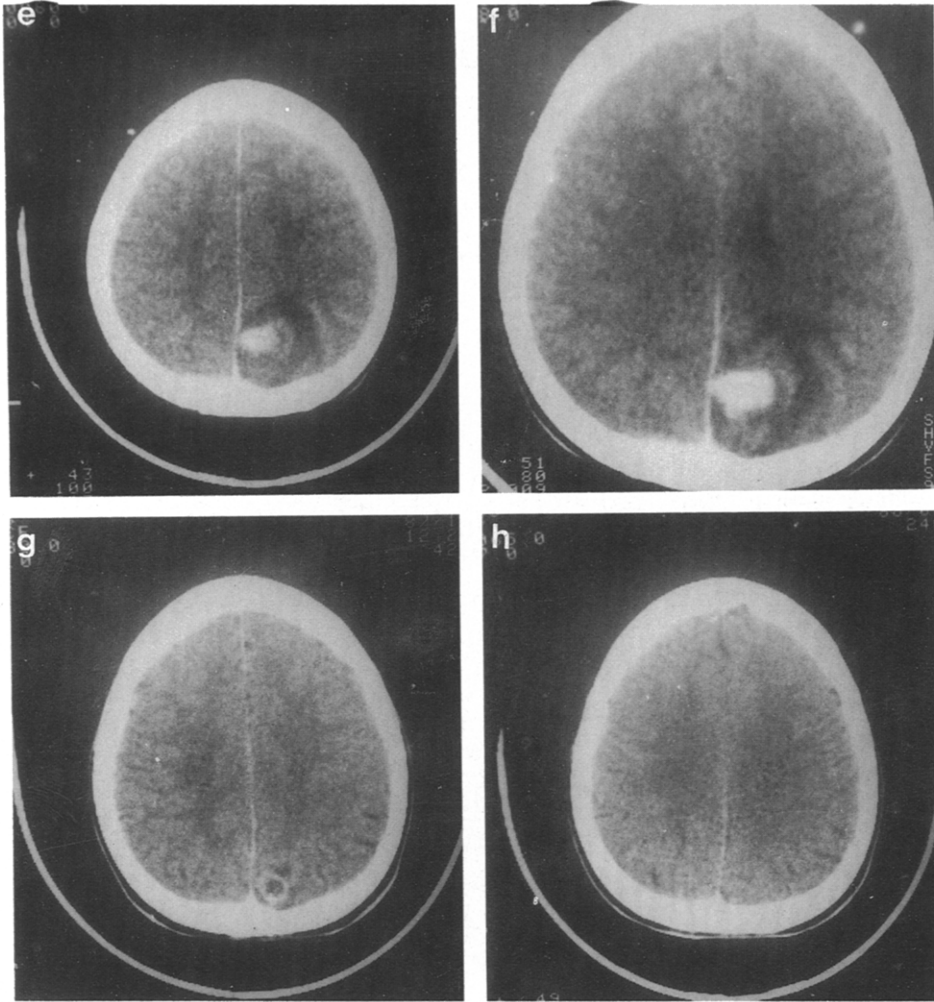


Fig. 6. Serial CECT in 10-year-old female with complex partial seizures showing (e) solid lesion in right occipital region, (f) persisting two months later, (g) gradual reduction in lesion on antituberculous treatment and (h) complete disappearance after one-and-a-half years.

interictal record of generalised seizure activity. CT was performed one week after the ictus and revealed right parieto-occipital ring lesion with surrounding oedema. CSF, chest X-ray and Mantoux test were normal. Re-

peat CT after 8 weeks was normal. Seizures are well-controlled on carbamazepine.

*Case 2.* A 17-year-old boy presented with right partial motor seizures and postictal right ataxic hemi-

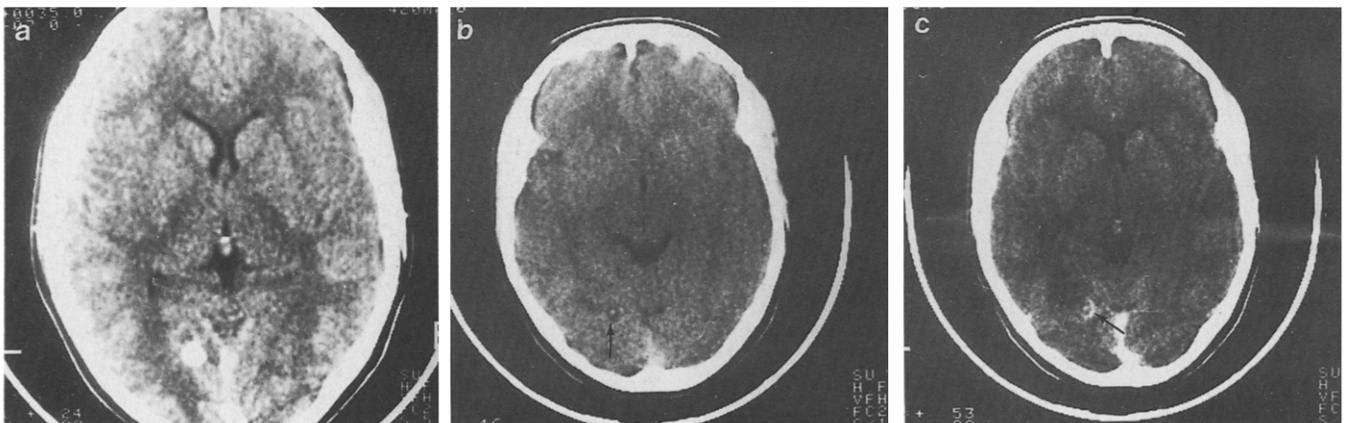


Fig. 7. (a) Disc lesion with irregular ring lesion anterior to it in CECT of a 14-year-old female with generalised tonic-clonic seizures; (b) plain and (c) CECT scans 6 months later showed small cystic lesion with central high attenuation and enhancing margins (arrow).

paresis. The neurologic deficit recovered in one week. CT showed a ring lesion in left parietal lobe. The lesion disappeared except for a speck of high attenuation on repeat CT scan. The patient is on anticonvulsants.

*Case 3.* A 29-year-old male presented with bifrontal, non-throbbing headache for 25 days. There was no history of seizures. Neurological examination was normal except for fundus findings. The optic cups were full, veins were dilated and disc margins were clear. CT scan showed two ring lesions in left frontal lobe with oedema (Fig. 8e, f). Montoux test was positive, chest X-ray was normal and cysticercosis serology was negative. The headache subsided with paracetamol. A follow-up examination after 3 weeks revealed normal fundus. It was decided to repeat the CT scan after 8–10 weeks. He was not given any specific therapy for the CT lesions. He presented with recurrent complex partial and generalised tonic-clonic seizures

10 weeks after the first CT scan. CT scan was repeated at this time and was normal (Fig. 8g). Seizures were controlled with phenytoin. MRI scan done 2 weeks later was also normal (Fig. 8h).

### Discussion

CT lesions with or without seizures which disappear without specific therapy have been reported (Vern and Butler 1983; Weingarten et al. 1983; Dillon et al. 1984; JayaKumar, 1985; Zegers De Beyl et al. 1985; Nakasu et al. 1985; Sethi et al. 1985). The numbers of patients in each study are small. CT lesions which are known to subside spontaneously include infarcts secondary to vasculitis, focal encephalitis, cysticercosis, demyelination and oedema due to cerebral trauma. Spontaneous regression of tuberculoma, glioma and lymphoma has also been reported, though such instances are very rare

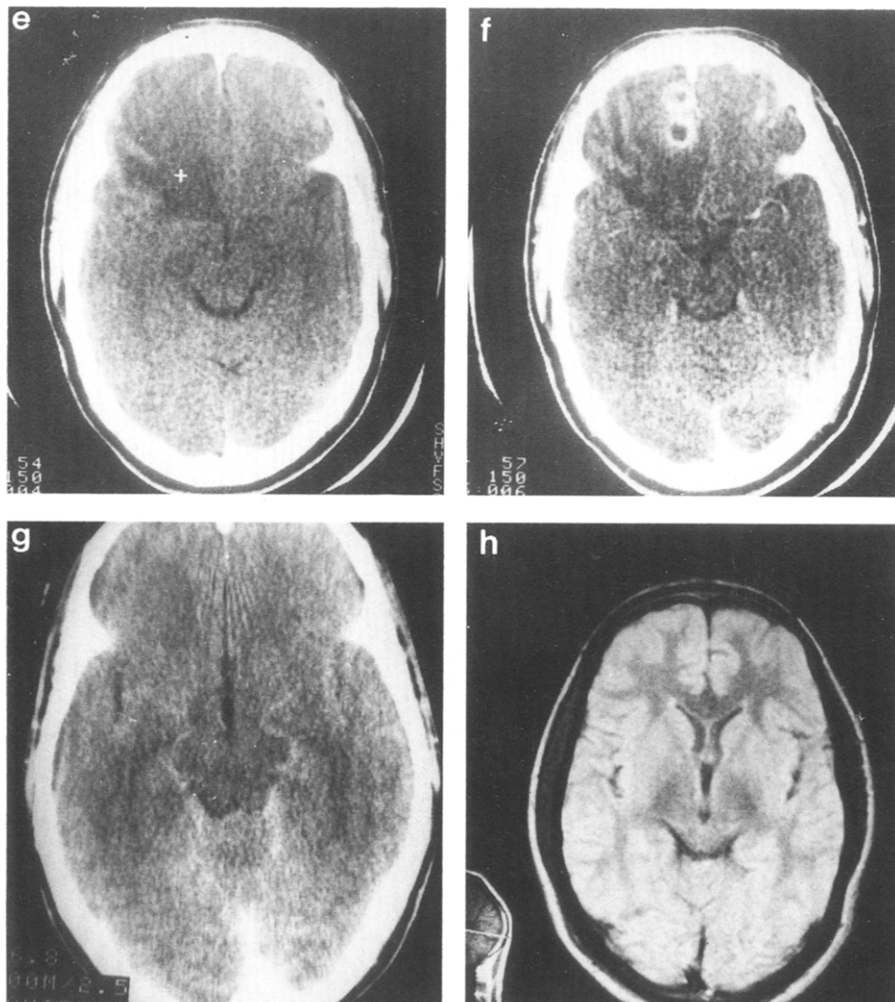


Fig. 8. (e) Plain CT scan showing left frontal lobe oedema; (f) CECT of the same showing two ring lesions with surrounding oedema in left frontal lobe; (g) CECT after 10 weeks showed complete disappearance of the lesions; (h) MRI of the same performed 12 weeks after the first scan was normal.

(Weingarten et al. 1983; Nakasu et al. 1985). In spite of extensive use of CT, there are very few reports of transient postictal CT lesions in the Western literature (Rumack and Guggenheim 1980; Vern and Butler 1983; Dillon et al. 1984). These authors are not sure about the nature of these lesions except Vern and Butler (1983) who believe that they could be due to vasculitis. However, such lesions are frequently seen in India. The patient is usually a child or young adult who presents with partial motor seizures with/without generalisation. There is no associated neurological deficit in most of the cases. Plain CT scan shows low attenuation area without any mass effect. Contrast enhancement usually brings out a ring or disc lesion. Other characteristics brought out by the present study are their location at the junction of grey and white matter, the common involvement of frontal and parietal lobes, the rare occurrence of multiple lesions and the absence of antecedent or concomitant systemic illness. The majority of these lesions disappear or show a residual speck within 8–12 weeks without any specific therapy other than anticonvulsants. Some may show gradual resolution over 6–12 months but seizure control is excellent.

There is controversy regarding the cause as well as the management of these lesions. The main point of discussion is whether these lesions are the cause or the result of seizures. We will discuss some of the known facts, our own postulations and management of these lesions. Various biochemical and physiological changes occur at the site of abnormal neuronal activity during or following seizures. These include changes in the neurons, glial cells, blood vessels and blood–brain barrier (BBB). Positron emission tomographic studies have revealed a hypermetabolic neuronal state and hyperperfusion in the region of epileptic focus during ictus (Theodore et al. 1983). This increased perfusion could be due to hypoxia, lactic acidosis and local cerebral dysautoregulation (Lee and Goldberg 1977; Engel et al. 1983). The enhancing lesion could be due to a break in the BBB. Such BBB breach has been demonstrated following a prolonged seizure in an experimental setting (Nitsch and Klatzo 1983). However, BBB breakdown has been demonstrated in prolonged seizure activity such as status epilepticus and such a breakdown occurs commonly in hypothalamus septum pallidum and cerebellum (Nitsch et al. 1984). None of the patients in the present series had status epilepticus. The lesions were seen in cerebral cortex rather than in the usual sites quoted for BBB after prolonged seizure activity. These facts do not support the postulation that these lesions are the result of BBB breakdown. Moreover, such lesions are commonly seen in the Indian subcontinent only. There are very few reports in the western literature where CT scan has been used for investigating epilepsy for the last two decades but such

vanishing lesions have not been reported. Had these lesions been an effect of the seizures, these would have been seen in the West as frequently as in India. Hence we believe that these lesions are the cause of epilepsy rather than its effect. In case 3, ring lesions were picked up on CT scan before the onset of seizures and disappeared subsequently without specific therapy. It proves our point beyond doubt that these lesions are not a postictal phenomenon.

Very few pathological studies are available in these cases. Zegers De Beyl et al. (1985) reported a patient who underwent stereotaxic biopsy under CT monitoring for the postictal lesion 3 weeks after the seizure. The biopsy revealed normal brain tissue. However, the biopsy was done when the lesion was disappearing. Chandy (1988) performed CT-guided stereotaxic biopsies on 6 such lesions which were reported as chronic non-specific inflammation.

Various CT lesions which can present with a similar CT picture are:

*Tuberculoma.* CT appearance of tuberculoma has been well described by various authors (Bhargva and Tandon 1980; Vangsarkar et al. 1986). This includes small ring, disc and target lesions, low attenuation with massive oedema and irregular coalesced masses. These can occur in any part of the brain at single or multiple sites. Most of these lesions are labelled tuberculomas on the basis of supportive findings such as history of fever, elevated ESR, positive tuberculin test, abnormal CSF findings and response to treatment with antituberculous drugs. The tuberculomas rarely disappear spontaneously. But serial CT scans of such patients have shown disappearance of lesions with antituberculous therapy.

The CT morphology of disappearing lesions is almost the same as that of tuberculomas. This is why most of the patients were put on antituberculous treatment even without ancillary evidence before this entity was clearly established in early days of the use of CT. Spontaneous disappearance, absence of recurrence and lack of ancillary evidence shows that these lesions are unlikely to be tuberculomas. A probable diagnosis of tuberculoma was made only in 5 cases of the present study who had abnormal CSF findings and positive Mantoux.

*Glioma.* Spontaneous regression of glioma has been reported by Nakasu et al. (1985) in sequential CT scans, but complete disappearance of the CT lesion has not been reported. Eventually all these cases showed a gradual increase in mass size. It is unlikely that any of our patients has a glioma since none of them exhibited increase in size in serial CT scans.

*Lymphoma.* The characteristic location of primary CNS lymphoma is near the meninges or subependymal. They are known to regress spontaneously (Weingarten et al. 1983). Primary CNS lymphoma is a very rare



entity and it is known for its recurrence. The postictal lesions in the present study are unlikely to be due to a malignant disease process.

*Vasculitis.* Vern and Butler (1983) have reported transient thalamic hypodensity in association with seizures. Vasculitis produces low attenuation areas and rarely presents with ring, disc or target lesions. None of our patients had systemic evidence of vasculitis. Occlusive vascular (ischaemic) disease also seems unlikely as the majority of patients belong to a younger age group.

*Demyelination.* It can also produce low attenuation areas which may disappear on follow-up CT examination. Lack of typical clinical picture, involvement of grey matter and normal CSF findings are against such a possibility.

*Cysticercosis.* CT findings of neurocysticercosis include low attenuation cysts, ring lesions, enhancing lesions surrounded by oedema and diffuse cerebral oedema (Minguetti and Ferriera 1983). The characteristic features of these lesions is multiplicity; single or isolated lesions are rare. CT lesions of cysticercosis are known to disappear spontaneously or with steroid therapy (Minguetti and Ferriera 1983; Mitchell and Crawford 1988). The authors have also observed a number of such cases in the last decade. The CT picture of the patients in the present study could be due to cysticercosis. But none of these patients had any evidence of cysticercosis in the form of subcutaneous nodules or positive CHA in CSF. Cystic change in the form of a small solitary cyst with central high attenuation in place of previous ring lesion occurred in two cases. Although multiple lesions were not present, the morphology of the cyst appears consistent with cysticercus cellulosae. It is possible that a proportion of spontaneously disappearing lesions may be due to cysticercus cellulosae. The prevalence of neurocysticercosis in the Indian subcontinent is high.

*Focal encephalitis.* Viral encephalitis produces a low attenuating lesion on CT scan; ring or disc enhancement is also known but it is very rare. Viral encephalitis may regress spontaneously. Absence of systemic symptoms and normal CSF are factors against such a possibility. However, a focal disease process may not produce these changes.

Acute nature of the lesion, its spontaneous disappearance or a residual high density speck without any specific therapy indicates the possibility of focal inflammation. There is no evidence of bacterial involvement and in the absence of viral or histopathological studies the exact nature of the lesion cannot be defined. Sethi et al. (1985) are of the opinion that these lesions may be due to focal encephalitis, or cryptic AVM. We disagree with Wadia et al. (1987) that these lesions are tuberculomas which tend to disappear after antituberculous therapy. The present study clearly shows that majority of these lesions disappear without

specific therapy. The first phase of the present clinical CT study was designed to establish management policy in epilepsy patients presenting with such lesions. In the second phase we are planning to do stereotaxic biopsy in some patients in spite of our reservations. Are we justified ethically to do large-scale stereotaxic biopsies on such patients when the lesions disappear spontaneously? Virological studies of CSF will of course be pursued. Chandy (1988) performed CT-guided stereotaxic biopsies on 6 such patients. In all the cases biopsies showed non-specific chronic inflammation. The time of these biopsies in relation to CT has not been specified.

#### *Non-vanishing CT lesions*

CT lesions associated with seizures which do not regress or disappear in 8–16 weeks should not be considered as postictal spontaneously vanishing lesions. In the present series, 5 such cases were further investigated and put on antituberculous therapy because of the ancillary evidence. All of them showed good response to antituberculous therapy. All these 5 cases had no definite clinical evidence of tuberculosis in spite of the fact that Mantoux was positive and CSF showed lymphocytic pleocytosis. It is well known that the incidence of positive Mantoux in the normal population is high in the Indian subcontinent because of BCG vaccination and/or subclinical tuberculous infection at an early age. With this in mind, these 5 cases were not started with antitubercular therapy in the very beginning and we waited the result of the repeat CT in view of our experience with vanishing lesions. However, these were the 5 cases in which lesion showed no regression at all. Therefore it is recommended that antitubercular therapy be started along with anticonvulsants only in those cases who show positive Mantoux and abnormal CSF or any other evidence of tuberculosis. Mere positivity of Mantoux test is not enough evidence for starting antitubercular therapy; close follow-up is necessary. Four patients with cystic or unchanged lesions are being followed-up. The lesions disappeared in two of these patients at 10–12 months follow-up. There was a complete disappearance of the lesion in one case and a residual speck in the second case. Non-vanishing CT lesions, if associated with raised intracranial tension, progressive neurological deficit or uncontrolled seizures on follow-up, should be admitted for specific medical and/or surgical therapy. None of our patients showed such worsening. We believe strongly that surgery should not be resorted to in such cases without the second CT scan in 8–12 weeks showing progression of the lesion.

#### *Management*

We recommend the following guidelines for management of such focal enhancing CT lesions in patients with seizures:

(i) Patients without raised intracranial tension, progressive neurological deficit, ancillary evidence of infection or malignancy should be treated with anticonvulsants only. CT should be repeated after 8–12 weeks. CT should be repeated any time if there is deterioration in the clinical condition of the patient. CSF should be screened by ELISA for tuberculosis or cysticercosis.

(ii) The patient only needs clinical follow-up if repeat CT shows regression or disappearance of lesion. The anticonvulsant therapy is completed, as is usual for epilepsy. In asymptomatic cases even though repeat CT at 6–8 weeks interval does not show any change in lesion, they should be followed-up clinically and CT should be repeated after 8–12 weeks until lesion disappears. In case the lesion does not regress, stereotaxic biopsy is recommended, even when the patient remains asymptomatic, lest a very slow growing tumour is missed.

(iii) Patients with ancillary evidence of tuberculosis such as positive Mantoux test and abnormal CSF with or without systemic evidence of tuberculosis should be treated with antituberculous therapy in addition to anticonvulsants. They should be followed-up with serial CT scans.

(iv) If the follow-up studies shows increase in size of the CT lesion, raised intracranial tension, fresh neurological deficit: such patients should be subjected to appropriate investigations and specific medical therapy and or surgical intervention.

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