

showed nuchal rigidity; three patients had uni- or bilateral abducens paresis. Three patients had an occipital fracture on skull radiograph. Besides the haematoma, CT showed mild hydrocephalus in three and slight frontotemporal contusion in two patients. Five patients were treated operatively with suboccipital craniotomy, immediately after the diagnosis was made. The patient who did not present until the second week was treated conservatively. All patients had good recovery: only one patient had a mild abducens paresis at discharge from the hospital.

Clinical diagnosis of an epidural haematoma in the posterior fossa is difficult because the symptoms and signs are usually not specific, especially in acute cases. The major sign is deterioration of consciousness: this was present in five of our patients but occurs with all types of traumatic haematoma. Although the classical lucid interval is reported to occur in only a minority of patients,<sup>1-3,5,6</sup> four of our patients had a lucid interval varying from one to 24 hours. Diagnosis is more easy when patients present more than a day after injury as in three of our cases: the signs may then be either of raised intracranial pressure or of a posterior fossa lesion (lower cranial nerve dysfunction, cerebellar signs, nuchal rigidity), or both. A haematoma in the posterior fossa was suspected in five of the reported patients because of deterioration of consciousness after occipital injury and in three patients because of either an abducens paresis or nuchal rigidity, or both. In all our patients who presented in the first week the haematoma was diagnosed by CT immediately after admission. Early recognition and diagnosis of a posterior fossa clot is possible if the possibility is considered in every patient with occipital injury. If such a patient develops neurological symptoms, especially deterioration of consciousness or deterioration of vital signs, CT should be undertaken, even if there is not a skull fracture. Before CT was available mortality was high because diagnosis was difficult; often, the extradural clot in the posterior fossa was not found until necropsy and many series included post-mortem cases,<sup>7,8</sup> CT enables prompt diagnosis and treatment and improved results: it may show the associated intracranial lesions which are important for prognosis. In the report of Brambilla *et al* two of the three patients who had died, had bilateral frontal lacerated-contusive areas and haemorrhages of the brainstem and basal ganglia.<sup>1</sup> Three of our patients had only mild hydrocephalus and two had slight frontotemporal contusion. The lack of mor-

tality and minimal morbidity in our patients reflects prompt diagnosis with CT, followed without delay by surgical evacuation of the haematoma and to the lack of serious associated intracranial lesions.

JELIS BOITEN

Department of Neurology,  
University Hospital Maastricht,  
PO Box 1918, 6201 BX Maastricht,  
The Netherlands

#### References

- 1 Brambilla G, Rainoldi F, Gipponi D, Paoletti P. Extradural haematoma of the posterior fossa: A report of eight cases and a review of the literature. *Acta Neurochirurg (Wein)* 1986;**80**:24-9.
- 2 Ammirati M, Tomita T. Posterior fossa epidural hematoma during childhood. *Neurosurgery* 1984;**14**:541-4.
- 3 Roda JM, Giménez D, Perez-Higueras A, Blázquez MG, Pérez-Alvarez M. Posterior fossa epidural hematomas: A review and synthesis. *Surg Neurol* 1983;**19**:419-24.
- 4 Zuccarello M, Pardatscher K, Andrioli GC, Fiore DL, Iavicoli R, Cervellini P. Epidural hematomas of the posterior cranial fossa. *Neurosurgery* 1981;**8**:434-7.
- 5 Stone JL, Schaffer L, Ramsey RG, Moody RA. Epidural hematomas of the posterior fossa. *Surg Neurol* 1979;**11**:419-24.
- 6 Neubauer US. Extradural haematoma of the posterior fossa. Twelve years experience with CT-scan. *Acta Neurochir (Wien)* 1987;**81**:105-11.
- 7 Hooper RS. Extradural haemorrhages of the posterior fossa. *Br J Surg* 1954;**42**:19-26.
- 8 Jamieson KG. Posterior fossa haematoma. In: Vinken PJ, Bruyn GW. Eds. *Handbook of Clinical Neurology. Vol 24. Injuries of the brain and skull. Part II.* Amsterdam: North-Holland publishing Company 1976:343-50.

Accepted 7 March 1989

#### Disappearing CT lesions in epilepsy: is tuberculosis or cysticercosis the cause?

Sir: Goulatia *et al*<sup>1</sup> reported a series of patients, with seizures, who showed CT lesions which resolved over a 6-12 week period, when seizures were controlled with anticonvulsant medication. These were labelled as "Disappearing CT lesions". Similar CT findings in epilepsy have been reported by others.<sup>2-4</sup> There has been considerable speculation regarding the nature of these lesions. The lesions cannot be solely attributed to the postictal state as they are

focal and take several weeks to resolve. Furthermore, such lesions are not common in patients with seizures. Since almost all cases are reported from India, an inflammatory aetiology is considered likely. Tandon and Bhargava<sup>5</sup> considered these to be tuberculomas. They treated their patients with antitubercular drugs and advanced the argument that since the lesions then resolved, their contention was confirmed. Other possibilities to be considered are cysticercosis,<sup>1</sup> focal encephalitis<sup>2</sup> and microabscess.<sup>4</sup> In the present study we investigated such patients for evidence of tuberculosis and cysticercosis by testing serum for antibodies against *M tuberculosis* and cysticercus using ELISA.

Thirty eight patients with seizures who on contrast enhanced CT of head done within 2 weeks of a seizure showed a single enhancing ring lesion or a hyperdense lesion (with or without surrounding hypodensity) and a complete or substantial resolution on CT scan 8-12 weeks later were studied. There were 16 male and 22 female patients. The age of the patients was from 7 to 65 years. Eighteen had generalised convulsions and 20 had partial motor seizures. None of the patients had neurological deficit on examination. Chest radiographs were obtained in all. Patients who had overt evidence of tuberculosis or subcutaneous nodules suggestive of cysticercosis were excluded. Also excluded were patients in whom the CT lesion remained unchanged after 8-12 weeks. Serum was tested for tubercular and cysticercus antibodies using ELISA. Antibodies to cysticercus and tubercle bacilli were measured by ELISA as described previously.<sup>6,7</sup> Serum ELISA was also done in healthy controls, patients with systemic tuberculosis, CNS tuberculosis, documented cerebral cysticercosis and epilepsy.

The results of the ELISA tests are shown in the table. Twelve out of 38 patients with "disappearing lesions" had serology positive for cysticercosis while two were positive for tuberculosis. None of the healthy controls showed a positive reaction for cysticercosis. Nineteen out of 22 proven patients with neurocysticercosis were positive for cysticercus antibodies while none showed evidence of tubercular antibodies.

The prevalence of epilepsy is not significantly different in different areas of the world. CT scan is frequently done to detect underlying structural lesion in the brain. For reasons which are not understood, the "disappearing lesions" have almost exclusively been reported from India. Because of this geographic feature, an infection or infestation has been the prime suspect. Tandon and Bhargava considered these to be tuber-

Table Serum antibodies to cysticercus and mycobacterial antigens

Group	Cysticercus antibodies		Mycobacterial antibodies	
	OD 492 nm (mean, 2 SD)	Positive/total	OD 492 nm (mean, 2 SD)	Positive/total
Control	0.01, 0.02	0/36	0.16, 0.12	3/56
Systemic active Tb	0.01, 0.03	0/26	1.3, 0.4	31/31
CNS Tb	0.03, 0.09	1/29	0.76, 0.5	13/18
Neurocysticercosis	0.67, 0.5	19/22	0.12, 0.09	0/32
Epilepsy	0.04, 0.11	2/39	0.18, 0.2	1/38
Disappearing CT lesions	0.46, 0.4	12/38	0.09, 0.1	2/38

Antimycobacterial and anticysticercus antibodies in sera were measured by ELISA as described earlier. The mean + 2 SD of control OD values in each case were taken as cut-off values to determine positivity rates.

culomas and treated these patients with antituberculous drugs. After this report, in India, most patients who presented with epilepsy and an enhancing ring or disc lesion have been treated for tuberculosis. Ahuja and Mohanta<sup>8</sup> in a prospective study of late onset epilepsy investigated 253 patients all of whom had CT. There were 23 patients with a space occupying lesion shown by CT. The lesion disappeared in 11 patients with antituberculous treatment. The authors concluded that these were tuberculomas. Goulatia *et al*<sup>1</sup> presented evidence that most of these lesions disappeared when treated with anticonvulsant drugs alone, thus casting doubt on the tuberculoma hypothesis. However, the nature of the underlying lesion remained a matter of speculation. Sethi *et al*<sup>2</sup> speculated that it could be focal encephalitis of obscure origin.

Since cysticercus is well known to produce epilepsy and a similar CT morphology in cysticercosis has been described, we investigated our patients for evidence of cysticercosis. Approximately one third of our patients showed evidence of antibodies against cysticercus in serum. Ghosh *et al*<sup>3</sup> (reported at the 38th Annual Conference of the Neurological Society of India, 1988) have performed stereotaxic biopsy in 14 such patients. Biopsy revealed evidence of parasitic granuloma in 10 patients of which six were definite cysticercus. None of the patients showed evidence of tuberculosis. It is our contention that the CT lesions could be produced by more than one underlying pathology and cysticercosis is an important cause. Our current hypothesis is that these patients have a "micro" lesion producing an epileptogenic focus and since the lesion is small it is not detected on CT scan in the interictal period. During a seizure there is a breakdown of the blood brain barrier (BBB) around the lesion which produces the enhancing lesion. As the BBB is repaired, the lesion regresses. We have seen five patients in

whom the lesion disappeared and reappeared at the same or different site after a flurry of seizures, to disappear again when the seizures were controlled. This observation would support the concept of breakdown in BBB. Though tuberculomas can have similar image morphology on the CT scan, it seems that it is not the cause of "disappearing lesions" in any significant number of patients.

GK AHUJA\*

M BEHARI\*

K PRASAD\*

RK GOULATIA†

BANSI L JAILKHANI\*

Departments of Neurology,\*

and Neuroradiology,†

All India Institute of Medical Sciences,  
New Delhi, India

Address for correspondence: Dr G K Ahuja, Professor of Neurology, Neuro-Sciences Centre, All India Institute of Medical Sciences, New Delhi 110029, India.

#### References

- Goulatia RK, Verma A, Mishra NK, Ahuja GK. Disappearing CT lesion in epilepsy. *Epilepsia* 1987;28:523-7.
- Sethi PK, Kumar BR, Madan VS, Mohan V. Appearing and disappearing CT scan abnormalities and seizures. *J Neurol Neurosurg Psychiatry* 1985;48:866-9.
- Jaykumar PN, Taly AB, Mohan PK. Transient computerised tomographic (CT) abnormalities following partial seizures. *Acta Neurol Scand* 1985;72:26-9.
- Bhatia S, Tandon PN. Solitary 'Microlesions' in CT: A clinical study and follow up. *Neurology (India)* 1988;36:139-49.
- Tandon PN, Bhargava S. CNS tuberculosis: lesions learnt from CT studies. *Neurology (India)* 1980;28:207-12.
- Chawla TC, Atreyi M, Jaiikhani BL. Immunohistochemical characterization of saline extracted antigens of mycobacterium tuberculosis H 37

Ra, *Int. Arch. Allerg Appl Immunol* 1986; 80:221-3.

7 Diwan AA, Coker-Vaum M, Brown P, *et al.* Enzyme linked immunosorbent assay (ELISA) for the detection of antibodies to cysticerci of *Tania Solium*. *Am J Trop Med Hyg* 1982;78:492-6.

8 Ahuja GK, Mohanta A. Late onset epilepsy. A prospective study. *Acta Neurol Scand* 1982; 66:216-26.

Accepted 3 March 1989

#### Exercise induced diplopia as a presentation of midline cerebral tumour

Sir: We report a patient who presented with exercise induced diplopia caused by a midline cerebral tumour. To our knowledge this presentation has not been previously reported for any neurological disease, including intracerebral malignancy.

A 22 year old white, male, telephone engineer presented to his general practitioner in April 1986 with a 10 day history of exercise induced diplopia. These symptoms had occurred on three occasions and lasted between 15 and 90 minutes. Twice diplopia was noticed while playing football and once while undergoing physical training. The symptoms subsided 5 minutes after cessation of exercise. He reported that the images were displaced vertically and were more divergent on upward gaze. When not exercising he had no diplopia, headache or other symptoms related to the nervous system. Examination in April 1986 revealed no ophthalmological or neurological abnormality. He was normotensive.

Between June and August 1986 he was investigated and during this period his episodes of diplopia became more frequent and occurred with lesser degrees of activity. He was observed undergoing vigorous exercise, when no abnormalities of extra-ocular muscle function were noted but the symptoms of diplopia were reproduced. CT of the head with contrast merely revealed some possible enlargement of the ventricular system which was not considered to be definitely abnormal. Lumber puncture revealed a pressure of 18 cm of cerebrospinal fluid with normal protein levels, immunoglobulin electrophoresis and cytology. Following these investigations no definite conclusions were reached but follow-up with a repeat of CT was arranged.

By December 1986 he had deteriorated and complained of diplopia occurring much of the time, a worsening of memory, headache, inco-ordination and drowsiness. On re-



## Disappearing CT lesions in epilepsy: is tuberculosis or cysticercosis the cause?

G K Ahuja, M Behari, K Prasad, R K Goulatia and B L Jaikhanī

*J Neurol Neurosurg Psychiatry* 1989 52: 915-916  
doi: 10.1136/jnp.52.7.915

---

Updated information and services can be found at:  
<http://jnp.bmj.com/content/52/7/915.citation>

---

### Email alerting service

*These include:*

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

---

### Notes

---

To request permissions go to:  
<http://group.bmj.com/group/rights-licensing/permissions>

To order reprints go to:  
<http://journals.bmj.com/cgi/reprintform>

To subscribe to BMJ go to:  
<http://group.bmj.com/subscribe/>