

LETTERS TO THE EDITOR

Intralaminar dural haematoma developing in the contralateral convexity after temporal lobectomy

A so called "subdural haematoma" actually develops in the dural border cell layer, which belongs to the dura; a true intralaminar dural haematoma to our knowledge has not been reported.

A 29 year old right handed man presented with a 14 year history of intractable epilepsy, which did not respond to major antiepileptic drugs. He usually felt an epigastric rising sense as an aura, then stared blankly, and showed oroalimentary automatism. Postictal confusion persisted for several hours. The frequency of attacks ranged from several times a month to several times a day, and he had been taking phenytoin, phenobarbitone, and vigabatrin. During childhood, he had had severe febrile seizures.

No neurological deficit was found on detailed physical examination. Brain MRI (1.5 T) disclosed bilateral hippocampal and diffuse cortical atrophy, and sharp waves were seen in the right anterior temporal region on interictal EEG. Prolonged video-EEG monitoring, however, showed sharp waves in the right temporal region and slowing in the left temporal region. Interictal single photon emission tomography (SPECT) did not show asymmetry between the two temporal lobes. Preoperative routine laboratory examination, including a coagulation study, showed no abnormality. Bilateral depth electrodes were inserted into both temporal lobes on 19 May 1995; postoperative CT showed no haematoma. After confirmation of right temporal onset, right temporal lobectomy and amygdalohippocampectomy were performed 10 days later. Postoperatively, the patient awoke fully and CT showed no unexpected findings. The following day, however, he became drowsy and CT showed an extra-axial haematoma in the contralateral convexity (fig 1). A diagnosis of epidural haematoma was made and he underwent emergency left frontoparietal craniotomy; The bone flap showed

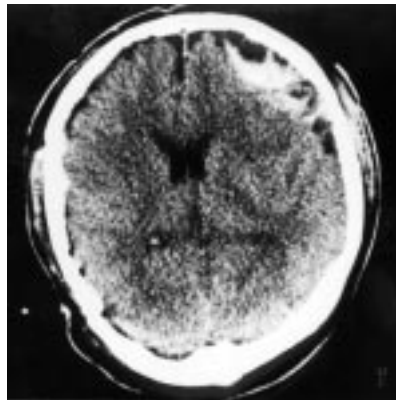


Figure 1 Computed tomogram obtained before the evacuation of intralaminar dural haematoma. Lobulated extra-axial haematoma is clearly visualised. The layering in the haematoma cavity, indicating an acute state, is not linear.

no penetration mark from the skeletal fixator used during the previous surgery. There was no haematoma in the epidural space. Incision of the visible dura showed blood clots, which were partially removed by suction; a yellow membrane, which also seemed to be dura, was noted at the bottom of the haematoma cavity. There was no active focus of bleeding. After removal of all the haematoma, it was apparent that this yellow membrane lined the entire cavity, and to expose the pial surface, this inner dura was incised. At the margin of the haematoma cavity, the covering and lining membrane was merged in a single layer, below which was the surface of the brain. Biopsy was performed at the margin, around which the two layered membranes merged to form a single layered dura. Because it adhered tightly, the underlying yellow membrane was difficult to dissect from the pial surface, and so was isolated and left in place. Concern about blood oozing from the under-surface of the covering membrane led to the removal of this membrane. Duraplasty was performed, using lyophilised cadaveric dura. The patient's postoperative course was uneventful, and he was discharged on the 7th postoperative day. Histological examination of the biopsy specimen showed separation of the dura and intralaminar dural haematoma (fig 2). There was no vascular malformation in the dura, but hippocampal sclerosis was found in the resected hippocampus. During two years of follow up, the patient remained seizure free without antiepileptic drugs.

Cranial dura mater is a composite structure of cranial periosteum and dura propria; the second is composed of fibroblasts and a large amount of extracellular collagen, and the innermost part of the dura is formed by the dural border cell layer.¹ The dura-arachnoid junction, identified as the subdural compartment (dural border layer), consists of avascular tissue with flake-like, relatively electron lucent cells stacked in several layers with narrow intercellular clefts.² The dural border

layer may be attached to the underlying arachnoid by an occasional cell junction. There is, however, no intervening space between the dural border cell layer and the arachnoid barrier cell layer that would correlate with what has been called the "subdural space." A survey of reports describing the morphology of the inner and outer capsule of so called subdural haematomas shows that dural border cells are found in both parts of the capsule.¹ These data support the view that what has been called a subdural haematoma is most often a lesion found within the layer formed by dural border cells.¹

The reason for the postoperative delayed development of intralaminar dural haematoma contralaterally is unclear. Postoperative epidural haematomas developing contralaterally after supratentorial craniotomy have been reported.^{3,4} The mechanism of this complication was reported to be unclear. In hydrocephalus, these massive epidural haematomas are probably caused by dura-skull detachment when the brain volume is strikingly reduced by a decompressive procedure. On rare occasions, pins from the head rest may detach the dura and cause epidural haematomas.³

In our case, however, there was neither hydrocephalus, nor penetration marks from the pins of the skeletal fixator. We used a negative pressure drainage system epidurally in this case, which might exert negative pressure intracranially. The inner surface of the dura and the pia-arachnoid were difficult to separate during surgery, indicating prior change at the dura-arachnoid junction. There is a possibility that the developing haematoma splits at the dural fibrous layer instead of at the dural border cell layer. To the best of our knowledge, this is the first report of a haematoma occurring between split dura propria.

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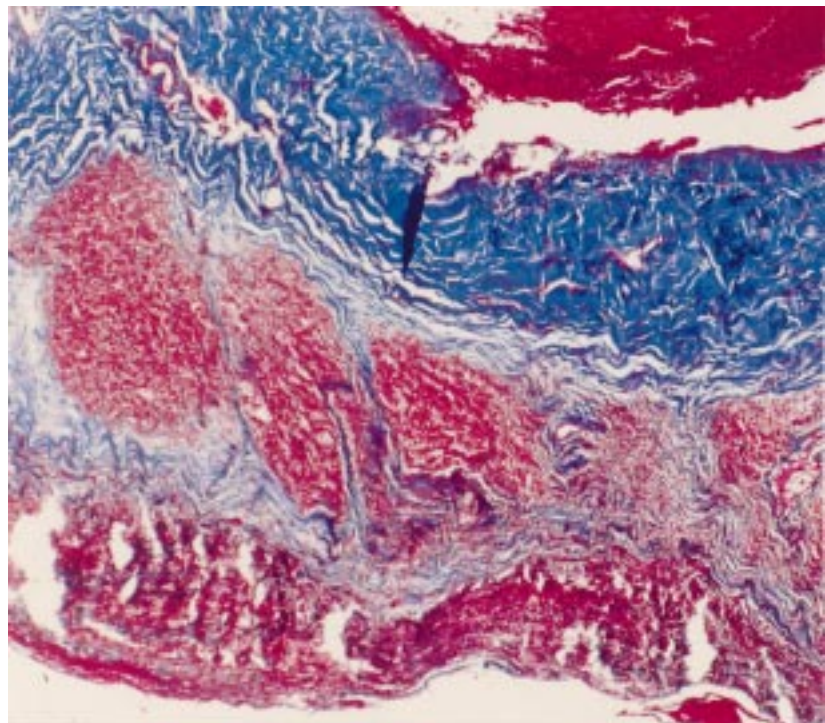


Figure 2 Photomicrograph shows separation of the dura propria and intervening haematoma. Masson's trichrome. Originally $\times 200$.

CHUN-KEE CHUNG
Department of Neurosurgery

YEON M KIM
JE G CHI

Department of Pathology, Seoul National University
College of Medicine, Seoul, Korea

Correspondence to: Dr Chun-Kee Chung, Department of Neurosurgery, Seoul National University College of Medicine, 28 Yongon-dong, Chongno-gu, Seoul, 110-744, Korea. Telephone 0082 2 760 3701; fax 0082 2 744 8459; email chungc@plaza.snu.ac.kr

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Carbamazepine hypersensitivity syndrome presenting as vasculitis of the CNS

Carbamazepine is a drug widely used in the treatment of partial and generalised tonic-clonic seizures, trigeminal neuralgia and other pain syndromes, affective disorders, and paroxysmal symptoms of multiple sclerosis. Common side effects are diplopia, dizziness, headache, nausea, and rash. Less common side effects include blood dyscrasias, toxic hepatitis, hyponatraemia as a consequence of inappropriate antidiuretic hormone secretion, orofacial dyskinesias, and cardiac arrhythmias.¹ Carbamazepine is also known to cause a severe systemic hypersensitivity reaction, known as carbamazepine hypersensitivity syndrome (CHS).² It consists of a triad of fever, lymphadenopathy, and rash; so called pseudolymphoma syndrome.² Other organs are often involved, most commonly the liver and, more rarely, lungs and kidneys.³ There are two cases of meningitis described as a complication of the carbamazepine therapy.^{4,5}

We describe a case of severe CHS with a typical pseudolymphoma picture and involvement of other organs, including the CNS. We suggest a possible pathogenetic mechanism for encephalopathy.

A 63 year old woman was started on carbamazepine (2×200 mg), as a seizure prophylaxis after a meningioma operation. About 3 weeks later she developed a flu-like illness. Two days afterwards, she developed a generalised rash and facial oedema, and fever appeared. She was admitted to hospital where physical examination disclosed a diffuse purpuric rash, oedema of the face and the tongue, pharyngitis, enlargement of lymph nodes, and a raised body temperature (39.5°C). Laboratory data showed leucocytosis (45.1), eosinophilia (42%), and high C reactive protein (98).

Liver enzymes were also raised. Additional tests disclosed a polyclonal increase in γ -globulin. Tests for Anti-DNA, anti-ENA, anti-ANA, and anti-ANCA antibodies were negative and complement C3 and C4 were normal. Chest radiography was normal, as well as lung function tests. Abdominal ultrasound investigation showed hepatosplenomegaly. Microbiological investigations failed to show any evidence of bacterial, fungal, viral,

or parasitic infections. Skin biopsy showed vasculitis of small vessels with perivascular infiltrates of lymphocytes, monocytes, and macrophages (figure). The findings were compatible with an allergic reaction.

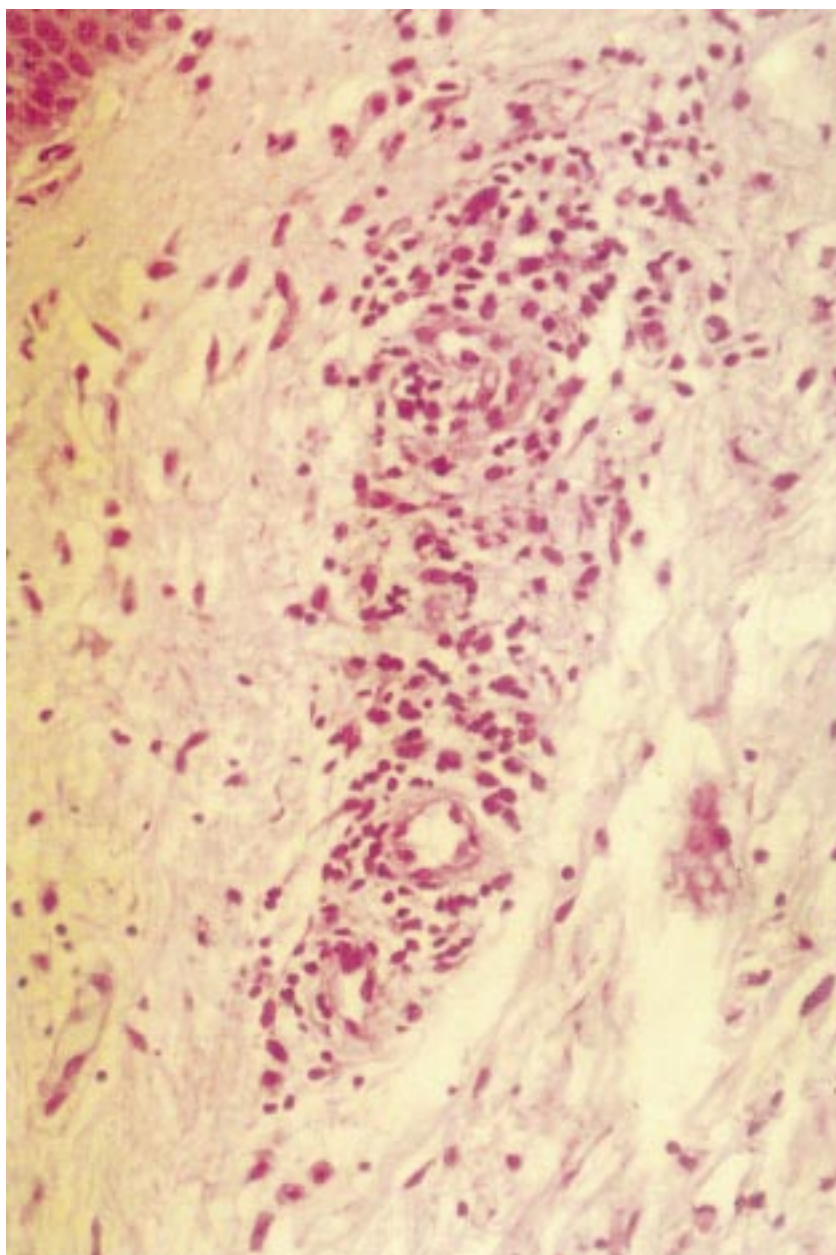
Carbamazepine was withdrawn and she was started on antibiotic treatment.

The patient's condition improved after the withdrawal of carbamazepine; the temperature fell, the lymph nodes regressed, the rash started to scale, and laboratory tests began to normalise. Antibiotic treatment was discontinued after a week.

Two weeks after admission and discontinuation of carbamazepine she became confused. Neurological examination disclosed cognitive deficits (mini mental state examination score 18), bilateral facial palsy of peripheral type, pyramidal (brisk reflexes, extensor plantar responses), extrapyramidal signs (rigidity, postural tremor), and ataxia. There was no neck stiffness and her temperature did not

rise again. A lumbar puncture disclosed 211 cells/mm³; more than 90% of them were lymphocytes. The concentration of protein in CSF was 1.29 mg/l and the concentration of sugar 2.85 mmol/l (40% of serum concentration). Concentrations of IgG, IgM, and IgA in CSF were raised. Oligoclonal bands were positive in serum and in CSF. Cultures for bacteria, fungi, and acid fast bacilli were negative, as were antibodies to mycoplasma, borrelia, and viruses. Serum and CSF VDRL and TPHA tests were negative. A T2 weighted MR image showed hyperintensive lesions in periventricular and white matter of the frontal and occipital lobes of both cerebral hemispheres. The lesions showed some opacification after injection of gadolinium contrast. There was no meningeal enhancement.

A few days after the deterioration in the patient's condition she started to improve again. The skin lesions remitted completely



Dermal vasculitis of small vessels with abundant perivascular infiltrate of lymphocytes, monocytes, and macrophages..