



Fig CT scan showing hypodense area in distribution of right anterior choroidal artery.

territory supplied by the anterior choroidal artery and its branches usually includes the posterior 2/3 of the posterior limb of the internal capsule, portions of the medial and intermediate segments of the globus pallidus, the lateral 1/2 of the lateral geniculate body and the proximal geniculocalcarine tract, the middle 1/3 of the basis pedunculus, the uncus, the amygdala, the anterior hippocampus, and the dentate fascia. In a given specimen, the area of supply may be slightly greater or slightly less.⁶⁻⁸ Total occlusion of this vessel has been reported to cause contralateral hemiplegia and hemianesthesia involving all sensory modalities but to varying degrees, hemianopia, and ipsilateral pupillary dilatation with sluggish pupillary light reaction.⁷ Occurrence on the non-dominant side may cause a syndrome of the minor hemisphere with anosognosia, visual neglect, constructional apraxia, and motor in-persistence. Occurrence on the dominant side has been reported to cause a mild aphasia with paraphasias and decreased fluency.⁹ The extent and location of lesions produced by occlusions of the anterior choroidal artery are variable owing to its anastomoses with the posterior choroidal artery, interpeduncular plexus, and posterior communicating artery. It is not an end artery.¹⁰ Cooper, in his report of ligation of this vessel for Parkinsonism in eight cases found no instance of contralateral hemiplegia nor hemianesthesia.¹¹ However, his

cases clearly had pre-existing disease in the region supplied by the anterior choroidal artery, and ligation of blood vessels is not physiologically equivalent to thrombosis.⁷ We believe this is a case of occlusion of the anterior choroidal artery because of CT confirmation of the predicted anatomic lesion.

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Absence of herpes simplex virus antigen in brain in encephalitis lethargica

Sir: Encephalitis lethargica was an acute encephalitic disease which occurred in Europe and America in epidemic proportions in the 2nd and 3rd decades of this century. A causal organism was not identified but the neuropathology of the condition as well as the epidemiological features were suggestive of a viral cause. Numerous attempts to isolate an infectious agent were undertaken, and some claims were made that a filterable agent had been isolated.¹ One such claim was made by Levaditi *et al*² who obtained an agent which was passed to rabbits and was found to have the properties of herpes simplex virus. As herpes simplex virus antigens can be identified in formalin-fixed, paraffin-embedded material we decided to examine stored material from a case of encephalitis lethargica with an antibody to herpes simplex virus using the immunoperoxidase technique.

The patient was a boy aged 17 years who had died in The London Hospital in 1920 after an illness of one week's duration consisting of headache, drowsiness and fever progressing to coma. At post-mortem examination (LH PM 15/20) there was swelling of the brain with widespread petechial haemorrhages. Stored paraffin-embedded blocks from the cerebrum and brain stem were recut; the sections showed intense inflammation and congestion in the midbrain with destruction of many neurons in the substantia nigra and periaqueductal grey matter. Unstained sections from this block were treated with an antibody to herpes simplex virus raised in rabbits (Dako) as previously described.³ No evidence of herpes simplex virus antigen was found.

We thought it worth placing this negative finding on record. As herpes simplex encephalitis is one of the commonest identifiable causes of sporadic encephalitis⁴ it would not be surprising if some cases of this disorder occurred and were investigated during the encephalitis outbreak. Although the distribution of damage in herpes simplex encephalitis differs from that of encephalitis lethargica the pattern of damage now recognised as typical for herpes simplex encephalitis had not been defined at that time and so it may well have been confused clinically and pathologically with encephalitis lethargica. As herpes simplex virus is only one of several viruses which retain some antigenicity after routine formalin fixation and paraffin

embedding it remains feasible that the virus that caused encephalitis lethargica (if virus it was) may yet be identified in material that has been stored since the time of the epidemic.

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Neurosarcoidosis presenting as major depression

Sir: Neurological complications of sarcoidosis occur in approximately 5% of all cases.¹ Rarely, dementia mimicking Alzheimer's disease may be the presenting or prominent manifestation of the illness.² Less commonly, prominent psychiatric manifestations in relative isolation can occur at the onset or during an exacerbation of the disease.^{2,3} We describe a patient who ultimately proved to have neurosarcoidosis and whose clinical presentation was indistinguishable from a major depressive episode.

A 57-year-old chronically dysphoric woman complained to her psychiatrist of deteriorating memory and concentration which forced her to give up her job as an office manager. Initial examination by her psychiatrist showed her to be oriented and coherent but tearful, with depressed mood and constricted affect. Her thought content included feelings of helplessness, hopelessness, anhedonia, and complaints of insomnia and anorexia causing a 10 pound (4.5 kg) weight loss. She named the past four presidents of the USA, spelled a four letter word forwards and backwards, and recalled

two of three objects after five minutes. There were no clear-cut cognitive deficits noted.

Outpatient treatment with imipramine was begun for major depression. This therapy was discontinued because of intolerable lethargy. The patient was then admitted to the inpatient psychiatric unit for electroconvulsive therapy. At that time her mental status examination was unchanged and the remainder of her neurological exam was reported as normal. After eight treatments (one bilateral and seven unilateral) she became severely confused with urinary incontinence and difficulty in walking. Neurological consultation was requested. On examination, she was disoriented to time, place, and could not identify her psychiatrist. There were no motor or sensory abnormalities other than a slightly wide-based, unsteady gait. An erythematous malar rash was noted. Investigation of the cause of her encephalopathy showed cerebrospinal fluid (CSF) to contain protein 160 mg/dl, glucose 40 mg/dl, 22 WBC/mm³ with 83% lymphocytes, 15% monocytes, 2% polymorphonuclear cells, negative VDRL, negative bacterial and fungal cultures, and no malignant cells. An electroencephalogram showed increased diffuse activity in the theta and delta frequencies. The CT scan showed decreased attenuation in the white matter adjacent to the lateral ventricles most prominently in the frontal region, and a cerebral angiogram was normal with no evidence of vasculopathy. Blood chemistry CBC, ANA, ESR, and thyroid function studies were all normal. The CSF findings from a second lumbar puncture were: protein 139 mg/dl, glucose 46 mg/dl, 21 WBC/mm³ with 83% lymphocytes, 14% monocytes, 3% polymorphonuclear cells, and no malignant cells. A skin biopsy taken in the area of the malar rash showed non-caseating granulomata. Kveim-Siltzbach antigen was injected and the biopsy several weeks later was positive for sarcoidosis. There was no definitive evidence of sarcoidosis on several chest radiographs.

The patient was treated with corticosteroids (prednisone 60 mg/day). Within three weeks of starting treatment her thinking was more lucid and her mood brighter. CSF findings at that time were: protein 68 mg/dl, glucose 70 mg/dl, 9 WBC/mm³ with 81% lymphocytes, 9% monocytes, 10% polymorphonuclear cells. A repeat CT scan was unchanged.

There are four reported cases in the literature of sarcoidosis in which a psychiatric syndrome was the presenting manifestation

or the major sign of an exacerbation.³ Gilmore *et al*⁴ reported a patient with known sarcoidosis thought to be in remission who had a depressive episode without other evidence of active sarcoidosis. The cerebrospinal fluid was suggestive of active disease. In Hook's⁵ report of nine cases of neurosarcoidosis, case number 2 was diagnosed as depression. Zerman⁶ described a mixed psychiatric-neurologic syndrome with features of Wernicke-Korsakoff psychosis as the initial manifestation of sarcoidosis. Suchenwirth and Dold⁷ described a man with a paranoid psychosis who was found to have sarcoidosis. His psychiatric symptoms responded to steroid medication.

Our patient initially had no clear evidence of active sarcoidosis. The clinical picture of tearfulness, constricted affect, anhedonia, and disturbance of sleep and appetite supported a diagnosis of a major depressive episode. Following ECT, the profound organic mental syndrome, gait disorder, and urinary incontinence indicated the organic basis of the disease and prompted the neurologic consultation. The diagnosis of neurosarcoidosis was based on the positive skin biopsy, positive Kveim test and cerebrospinal fluid formula of elevated protein, low glucose, and a predominantly lymphocytic pleocytosis. In contrast with Suchenwirth and Dold's⁵ case and other patients with dementia due to sarcoid,² our patient had a favorable clinical response to steroid medication with significant improvement of the psychiatric abnormalities.

The CT scan showed decreased attenuation of the periventricular white matter primarily in the frontal region. The relevance of this is not clear. CT findings in intracranial neurosarcoidosis are non-specific. They include normal scans, hydrocephalus, abnormal enhancement of the meninges (especially in the basal area), diffuse enhancement throughout the entire cerebrum and intraparenchymal mass lesions which often have a somewhat increased density compared to brain tissue and show enhancement on contrast studies.⁷⁻¹³ The association of sarcoidosis and progressive multifocal leukoencephalopathy is known.¹ In our patient this possibility cannot be entirely ruled out. However, it is unlikely because of the improvement in clinical status and CSF abnormalities with steroids. In addition, the usual CT findings of low density lesions of the central and convolutional white matter with scalloped lateral borders were not present.¹⁴