nosis with the negative biopsy in this case, so that although the importance of biopsy of such lesions cannot be over-emphasised, misleading results can make it impossible to proceed further. This is the great disappointment, and a lesson in this unfortunate case.

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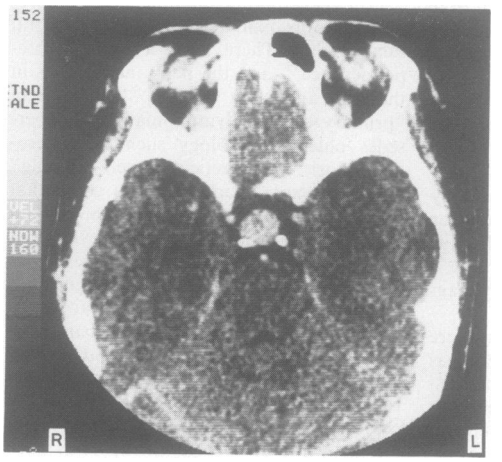
A pituitary tuberculoma

Sir: Tuberculomas within the central nervous system are unusual manifestations of tuberculosis. They constitute only 0·15% of intracranial tumours.[1] Pituitary tuberculomas, mimicking adenomas and causing disruption of endocrine function are very unusual.

A 37 year old Phillipino lady arrived in England in 1973. Four years later, in 1977, she had a cervical lymphadenectomy. Her Mantoux test at this time was positive, but no acid fast bacilli were seen in or cultured from the operative specimen. She was treated with three antituberculous agents for one month, followed by flucloxacillin and metronidazole. She remained well until January 1985 when, soon after a 1 month holiday in the Phillipines, she presented with a 3 day history of severe bitemporal headache. This was associated with vomiting, and diplopia on lateral gaze to the right. Her periods had been regular for 12 months, and there had been no change in libido. On examination, she had a right sixth nerve palsy, a right temporal hemianopia and a depressed right corneal reflex.

Initial biochemical investigations revealed a low T4 of 60 nmol/l (normal 70–160 nmol/l) with an inappropriately low TSH of 0·9 mu/l. A 9 am cortisol level was low at 79 nmol/l (normal 9 am 250–650 nmol/l). CSF examination revealed 5 lymphocytes/mm^3, protein 53 mg% and no acid fast bacilli were seen or cultured. A CT scan at this time showed a brightly enhancing lesion, which was lobulated and occupied the pituitary fossa, expanding into the suprasellar region (fig 1). Carotid and vertebral angiography showed the tumour to be avascular. Trans-sphenoidal exploration revealed a pale grey tumour of rubbery consistency, which was completely removed. The centre of the tumour appeared necrotic. Histology (Dr Robin Barnard) showed an active chronic inflammatory infiltrate with the formation of focal granulomata within pituitary tissue, compatible with the diagnosis of tuberculoma.

Ten days after operation she commenced treatment with rifampicin, isoniazid, ethambutol and pyrazinamide for 3 months, after which the ethambutol was stopped, the other agents continuing for a further 15 months. The patient made a good recovery from the operation, with resolution of her neurological deficit. Her pituitary function



Pre-operative enhanced CT scan showing a mass in the pituitary fossa.

also improved. Two weeks after operation her serum thyroxine rose to 72 nmol/l; basal TSH was minimally elevated at 6·5 mu/l and after 200 μg TRH, rose to 9·8 mu/l at 20 minutes. Basal prolactin was elevated at 38 μg/l. Her basal LH (4 u/l) and FSH (2 u/l) showed no response to 100 μg LHRH, and her plasma oestradiol was undetectable. During an insulin tolerance test her plasma glucose fell to 1·2 mmol/l and her basal morning plasma cortisol of 175 nmol/l rose to 420 nmol/l, a substantial response. During a water deprivation test her plasma osmolality rose to 303 mosm/kg, her urine rising to a maximum of 404 mosm/kg indicating diabetes insipidus. Two months after operation her serum thyroxine had risen to 85 nmol/l and TSH fallen to 1·6 mu/l. She had mild symptoms of diabetes insipidus and still had amenorrhoea with galactorrhoea. Her basal prolactin was elevated at 28 μg/l. Two months later her thirst and polyuria had improved, though she still had no periods and her prolactin remained elevated at 51 μg/l, whilst at 8 months she had no further symptoms attributable to diabetes insipidus although her galactorrhoea and amenorrhoea remained.

Isolated granulomatous lesions of the pituitary are rare and the differential diagnosis includes sarcoidosis, tuberculosis, syphilis and giant cell granuloma.[2] Although acid fast bacilli were not seen in this case, the patient's past history, results of investigation and improvement with anti-tuberculous treatment made a pituitary tuberculoma the likely diagnosis.

There have been few reports of pituitary tuberculoma in the past[3-5] although there have also been reports of giant cell granuloma of unknown origin[6-8] which might represent a solitary tuberculoma. Hassoun *et al*[8] claimed that the diagnosis of a pituitary granuloma is suggested by marked hypopituitarism, out of proportion to the size of the tumour. Clearly in our case, there was significant suprasellar enlargement, and the degree of hypopituitarism was not inconsistent with this.

Our patient presented as a neurosurgical emergency and although the diagnosis of tuberculoma was not considered preoperatively, the appropriate treatment was operative decompression of the optic chiasm. Post-operatively she still has some endocrine dysfunction as shown by the mild hyperprolactinaemia, gonadotrophin deficiency and mild diabetes insipidus. However, her hypothyroidism has improved, and although we do not have a pre-operative insulin stress test for comparison of dynamic pituitary adrenal function, morning cortisols have returned towards normal.

The potential importance of recognising a tuberculoma as a possible cause of a space occupying lesion within the pituitary fossa is that it may be treated conservatively with less hazard to the patient than operative intervention. Clearly, however, if the patient presents as a neurosurgical emergency, there may be no alternative to surgical decompression.

We thank Mr A Crockard who performed the trans-sphenoidal surgery on this patient and Dr RD Barnard for the pathological interpretation, and Professor WI McDonald for allowing us to report his patient.

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Protein C deficiency: a cause of amaurosis fugax?

Sir: We evaluated a 45 year old man who was admitted to hospital with a 2 day history of recurring left eye symptoms. He had experienced more than a dozen episodes of transient visual loss. He described "a shade coming down", sometimes over the entire left eye and sometimes over only the upper or lower half-field. His symptom lasted for 5 to 10 minutes. He had no history of migraine and there were no associated symptoms suggestive of migraine. A left Marcus-Gunn pupil was noted in the emergency room during an episode of amaurosis, but the neurological examination after admission was normal. Cardiac and vascular exams were normal except for chronic venous insufficiency in the legs. There was no evidence of acute deep venous thrombosis. Examination by a neuro-ophthalmologist revealed no abnormalities. The patient's medical history was unremarkable except for recurring thrombophlebitis in the legs. He had no history of hypertension, cardiac disease or diabetes mellitus. He smoked one-half pack of tobacco daily. He took no medication. His family history was noteworthy: both father and sister had a history of recurring thrombophlebitis.

Normal studies included: complete blood count, platelet count, biochemical survey, prothrombin time, partial thromboplastin time, platelet aggregation studies, serum antithrombin, erythrocyte sedimentation rate, sonoclot, fibrin split products and fibrin monomer. Cranial CT scan, electrocardiogram, echocardiogram and left carotid angiogram (including views of the ophthalmic artery) were normal. Protein C determination by immunological testing was abnormal at 46% (normal 70–180%). Testing of the patient's family disclosed similarly low values of protein C for five of 10 blood relatives. He was treated with coumadin and has been symptom-free in one year of follow-up.

Amaurosis fugax has been reported for more than 100 years. In 1952, Fisher [1] drew attention to the aetiological role of retinal emboli from the ipsilateral carotid. Others[2] have emphasised the heart as a source of emboli. Yet, in some series, 25–50% of the patients have neither carotid nor cardiac disease.[3,4] Reported additional mechanisms for amaurosis fugax include ocular disorders, vasculitis, platelet hyperaggregability, hypercalcaemia, myeloproliferative disorders, sickle cell disease, multiple myeloma, carotid artery trauma and dissection,