The interest of the case reported here is the association of an unusual late recurrence after radical hypophysectomy with early recovery of the hypothalamo-pituitary-adrenal axis and generalised corticotrophic hyperplasia on immune staining. Possible explanations for these observations are that the operative procedure was inadequate so that a partial rather than a radical procedure was performed, or that the small subgroup of patients with corticotrophic hyperplasia are more likely to have disease recurrence than those with a pituitary adenoma.

In our case the radical hypophysectomy was performed in the standard manner by an experienced surgeon who achieved good visualisation of the pituitary during the operation and remission was supported by subsequent biochemical investigations. Furthermore post-operative diabetes insipidus occurred and subsequent morphological and histological examination showed resection of an almost complete pituitary gland with generalised corticotrophic hyperplasia but no adenoma. Neither the recovery of the hypothalamo-pituitary-adrenal axis at 3 months or the absence of a need for replacement therapy necessarily imply an inadequate surgical procedure, since recovery of the axis by 3 months has been reported by others and has not predicted recurrence in patients followed up for 5–21 years. Thus, an alternative explanation for recurrence is that a generalised abnormality of corticotrophes facilitated regrowth of the cells remaining around the pituitary stalk. Such an abnormality could be due either to a primary defect of pituitary cells, or secondary to stimulation by CRF.

While radical hypophysectomy has proved an effective treatment for Cushing’s disease, this report questions whether patients with generalised corticotrophic hyperplasia may be expected to remain in remission. In this small but significant subgroup of Cushing’s disease, careful long-term follow-up is required to establish the efficacy of radical hypophysectomy.

Cysticercosis in the UK

Sir: Cysticercosis cellularis (infestation with the larval form of Taenia solium) in this country is rare. Previous reports from this region have been of adults acquiring the disease in other parts of the world and others have been from Eastern Europe where it has been prevalent. We report two cases of cerebral cysticercosis recently seen in this city.

A four year old British born Sikh child presented in December 1984 with a ten day history of focal seizures and post-ictal weakness of the left leg. She had visited India for a year at the age of two. Physical examination and routine laboratory investigations were normal. CT scan revealed a small ring enhancing lesion with surrounding oedema in the right parietal lobe close to the midline. The diagnostic possibilities were of abscess, tuberculosis or tumour. CT directed stereotactic localisation and excision was performed. Histology revealed a chronic abscess with a thick fibrous wall, scattered multinucleate giant cells, numerous chronic inflammatory cells and eosinophils and surrounding gliosis. The appearances were suggestive of cysticercosis and further sections fortuitously included one through the larva revealing its classical appearance with four suckers. Serological assays, which became available later, were felt to be diagnostic at a titre of 1:40 (tox acra serology was also positive). No specific therapy was instituted; she had remained well apart from a mild left sided weakness.

The second case is a 28 year old English schoolteacher who worked in Bhutan between August 1984 and May 1985. In August 1985 she had generalised convulsions and haematemesis during treatment for a tapeworm infestation. CT scan at that time was normal. In January 1986 she had a severe headache and tachypnoea for a week; these symptoms disappearing after treatment with Sanomigran. She had three further grand mal seizures and three partial seizures before presenting here in February 1986 when examination was normal. CT scan showed multiple cerebral enhancing lesions with surrounding oedema. Radiographs of skull, pelvis and thighs did not reveal abnormal calcification. Routine cerebrospinal fluid (CSF) analysis, including total protein estimation (0.30 g/l), was normal. Electrophoresis of concentrated CSF proteins indicated a normal gamma globulin percentage while isoelectric focussing of serum and CSF proteins revealed abnormal bands in the CSF alone. Stereotactic localisation and excision of a right frontal lesion was uneventful. Histology confirmed the diagnosis of cysticercus again showing a cysticercus with hooklets.

She was treated with Praziquantel 50 mg per kilogram per day for two weeks and phenytoin 300 mgm nocte. Apart from a fit she has remained well.

Infestation with the adult tapeworm is rare in the United Kingdom and is relatively asymptomatic. The larval form causes concern because of the cerebral lesions. The clinical diagnosis is based on the history, physical findings and the origin of the patient from an endemic area. Presentation is most common between twenty and fifty years of age but it may occur at any age, the interval from infection to symptom presentation ranging from several months to thirty years. Cerebral manifestations are reported to occur in 60–90% of cases.

The symptoms of nervous system involvement depend on the site and number of larvae and the host reaction. Cysts may be single or multiple and may be found within the brain parenchyma, the ventricular system or the subarachnoid space; there may be diffuse “racemose” meningobasal cyst formation. A generalised acute cysticercus encephalitis may present a difficult diagnostic problem and histological examination may be vital to differentiate between cysticercosis and the other conditions.
more commonly encountered viral encephalitides. Simple blood tests may reveal eosinophilia which only develops when the larva is dead. CSF examination may also demonstrate eosinophils or lymphocytes. Serological assay can be performed on serum or CSF: indirect haemagglutination is regarded as diagnostic although false positives are encountered and complement fixation in the CSF has almost the same diagnostic rate. Radioimmunoassaying has been developed recently.

Plain radiographs of the soft tissues and of the skull may show multiple small round calcified lesions and where the disease is endemic this is diagnostic. Cerebral lesions are not invariably calcified. Contrast ventriculography, cisternography and myelo- psychography may show solitary or multiple filling defects which may be mobile. CT will reveal the location of parenchymatous lesions with characteristic calcification or ring enhancement and variable surrounding oedema. Scanning after cisternal contrast will demonstrate basal or ventricular cysts but magnetic resonance imaging is better able to show ventricular cysts and the scolex within a cyst.

When the diagnosis has been made, treatment is symptomatic and aimed at the management of seizures and at the relief of local or generalised intracranial pressure. Undiagnosed mass lesions, especially if solitary, are best treated by resection. If excision is planned a complete resection is essential as the wall of the larva may reform a cyst. Definitive medical treatment with Praziquantel is now known to be effective and is also useful in disseminated central nervous system cysticercosis and the use of steroids is also advocated. Hydrocephalus when present can safely be treated by CSF diversion.

Cysticercosis is said to be the commonest parasitic infestation of the central nervous system. Worldwide reports of cerebral cysticercosis have increased recently, both from endemic areas and from population centres previously believed free from the disease. Only five cases of cerebral cysticercosis (and five of non-cerebral disease) have been reported to the Public Health Laboratory Service in this country since 1975, making cerebral cysticercosis rarer here than the travelling habits and multinational origins of the population would lead us to expect. There are, therefore, grounds to believe either that these figures do not reflect the true situation and that cysticercosis is more widespread or that we can expect an increase in this common international disorder. Should comparatively simple serological investigations fail to provide sufficient information then more sophisticated techniques such as stereotactic excision may be required.

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References


Purulent meningitis after percutaneous radiofrequency trigeminal rhizotomy

Sir: Since it was first described by Sweet in 1965,1 percutaneous radiofrequency trigeminal rhizotomy (RFTR) has been considered the procedure of choice for surgical treatment of most patients with trigeminal pain. The simplicity of the technique, the effectiveness, the low morbidity and almost non-existent mortality justify its wide use and acceptance.2 3 Although some complications occur, meningitis is seldom included among them. We describe two patients in whom purulent meningitis appeared after this treatment of idiopathic trigeminal neuralgia.

A 60 year old man suffered left-sided trigeminal pain involving the third division for 6 years. He had received carbamazepine, which had to be stopped because of gastric intolerance and development of glaucoma. Then, radiofrequency trigeminal rhizotomy was performed. Two days later the patient became febrile with nausea, vomiting, headache and mild alteration of consciousness. Meningeal signs were present at physical examination. Cerebrospinal fluid (CSF) contained 1,180 white blood cells/mm3 with 75% granulocytes, glucose 2 mg/dl and protein 2-2 g/l. No microorganisms were seen on Gram stain. Culture of CSF produced the growth of Gram positive cocci which were later identified as Streptococcus mutans. Blood cultures were sterile. Empiric therapy was changed to penicillin G, which was given for 14 days. The patient was discharged with no further complications. He remains asymptomatic after 6 months of follow-up.

A 66 year old man had received carbamazepine for 14 months for right trigeminal pain in the distribution of the third division. As the pain was not controlled with medical treatment, radiofrequency trigeminal rhizotomy was performed. Ten hours after the procedure the patient developed

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cardiac infarction. Some evidence suggests that this is to be explained by smokers' leu-
kocytosis, though other studies have shown an effect independent of smoking status. The mechanism is debated but leukocytes affect flow in the microcirculation, and influence platelet aggregation, and Light has suggested their proteinases might affect the development of atheroma.

To investigate the possibility that the leukocyte count predicts clinical events in the case of cerebrovascular disease, we have reviewed a group of patients with transient ischaemic attacks (TIAs) and related their leukocyte count at presentation to their subsequent course. The clinical outcome of some of these patients has been reported previously, many being from the study of strokes in young people.

The case records of 68 patients (56 males and 12 females of average age 48 years) presenting to one of us (JM) with a history of recent TIAs were reviewed. Patients with a recent completed stroke were excluded as a leukocytosis may be a response to a recent infarct or haemorrhage. Patients with polycythaemia rubra vera were also excluded. Note was taken of conventional risk factors, age, sex, blood pressure and smoking status. The white blood cell count recorded at their first clinic or hospital visit was extracted from the notes. The presence or absence of subsequent TIAs or strokes during a follow up period of an average of 5 years was also noted.

Twenty three patients had had further TIAs or strokes. The age, sex, blood pressure, smoking status, duration of follow up and leukocyte counts at presentation of those with and without further events is shown in the table. There was no difference in sex distribution, the prevalence of a blood pressure over 150/90 mm Hg, the proportion currently smoking or in the length of follow up achieved, between the two groups. Those with subsequent cerebrovascular events were slightly older (51.8 ± 6.9 years of 46.0 ± 9.5 years, t = 2.5, p < 0.02), and had higher leukocyte counts (9.74 ± 1.1 x 10^9/l vs 7.74 ± 2.5 x 10^9/l, t = 3.25, p < 0.002).

Fifty three per cent of patients with leukocyte count over 8.0 x 10^9/l had had further events whilst only 12.5% of those with a count of 8.0 x 10^9/l or less had (Chi Square 10.5, p < 0.002). Amongst the smokers there was still an apparent association between leukocyte count and risk. Thus the mean leukocyte count in smokers with subsequent events was 10.55 ± 1.9 x 10^9/l, that in smokers with no further TIAs or strokes was 8.05 ± 2.36 x 10^9/l (t = 3.72, p < 0.001). The chance of further events for a smoker with a leukocyte count over 8.0 x 10^9/l was 61% with a count of 8.0 x 10^9/l or less (n = 19), it was zero (Chi Square 15.5, p < 0.001).

This small retrospective study of young patients with early evidence of cerebrovascular disease presenting with one or more TIAs suggests that the leukocyte count in the peripheral blood may be predictive of the risk of further cerebrovascular events. A similar trend was obvious in a study from Hiroshima though these were asymptomatic patients not at such a high risk, as those with TIAs.

The data permit of no conclusion as to mechanism though the difference in apparent risk is clear within smokers as shown for myocardial infarction by Zalokar et al. It is possible that the degree of smokers leukocytosis is a reflection in some way of the biological impact of smoking in the individual. Other risk factors may be involved since leukocyte counts are higher in women on an oral contraceptive.

The evidence suggests that more investigation of the role of leukocytes in thrombosis, on blood rheology, and in atherogenesis is warranted.

We are grateful to Miss B Laatz for help with this study.

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Correction

Cysticercosis in the UK (J Neurol Neurosurg Psychiatry 1987;50:1050). The title of this letter should have been Cysticercosis in Birmingham and the authors C Shieff, ER Hitchcock, SP Valsangkar.