Letters

Depression caused by an intracranial meningioma relieved by leucotomy prior to diagnosis of the tumour

Sir: Frontal tumours sometimes present with symptoms which mimic psychiatric disease. It is not uncommon for diagnosis of the tumour to be delayed, especially if treatment of the psychological symptoms has been followed by improvement.

We report the case of a woman who presented with depression and focal epilepsy. Although an electroencephalogram showed a focal abnormality, she was diagnosed as suffering from idiopathic agitated depression and was treated with a bifrontal leucotomy. This led to a dramatic recovery from the depression but her epilepsy remained unchanged. Many years later, worsening of her epilepsy led to re-investigation and a large frontal meningioma which corresponded in position to the original EEG focus was diagnosed and removed. We have been unable to find any previous report of a patient where psychological symptoms attributable to an intracranial tumour had been treated successfully by leucotomy.

A 62-year-old housewife was referred by Dr LA Wilson in September 1982. She had been in good health until 1950 when she had received in-patient hospital treatment for depression. This responded to electroconvulsive treatment and she remained well until 1959 when she developed focal epileptic seizures characterised by aphasia and involuntary movements of the right arm and right side of the face. At the same time she developed a severe agitated depression which failed to respond to drug treatment. The depression was accompanied by an obsession that she had a brain tumour which was causing the fits! Because of the severity and intractability of the depression, which had led to several suicide attempts, she had been referred to a neurosurgical unit for consideration for a leucotomy. No neurological deficit could be found and a skull radiograph was normal. Although an EEG showed a left frontotemporal focus, bifrontal leucotomy was carried out.

After this operation her psychological symptoms were entirely relieved and she resumed a normal life at home. She was placed on phenobarbitone, 30 mg thrice daily and had occasional grand mal and focal fits. In 1981 she began having attacks of headache and vomiting and the frequency of her seizures increased to several times a day despite addition of phenytoin 300 mg/day to her anticonvulsant regime. She had no papilloedema and no abnormal neurological signs. Her mental state appeared normal apart from a very slight "oddness" of manner which had apparently been present since the leucotomy.

Skull radiographs were normal apart from old bifrontal trephine discs and multiple intracranial metal clips. CT scan showed a large well defined mass embedded in the convexity surface of the left frontal lobe. This enhanced uniformly with intravenous Conray and had the characteristics of a meningioma. The mass was associated with no shift of the intracranial contents and there appeared to be no oedema of the adjacent brain. There was atrophy of both frontal lobes which was thought to be related to the previous bifrontal leucotomy. The tracks caused by the leucotomy could be seen in the white matter of both frontal lobes. A 5 cm diameter spherical left frontoconvexity meningioma was completely removed. The tumour was rubbery in consistency with a well defined edge. Histological examination (Dr James McLaughlin) revealed a benign fibroblastic meningioma with numerous psammoma bodies. Recovery was uncomplicated. After operation her phenobarbitone was discontinued but she remains on phenytoin 300 mg/day. One year later she has no neurological deficit. She has had no epileptic fits since operation.

Although 23 years had passed between the leucotomy and the diagnosis of a meningioma, it seems a reasonable assumption that the meningioma had caused, or at least precipitated, the severe agitated depression for which the leucotomy was performed. The psychological symptoms accompanied the appearance of focal epilepsy and an electroencephalogram had shown a focus of abnormal activity corresponding to the location of the tumour. Inexplicably, the finding was ignored and leucotomy was carried out without any further investigation. Ironically, the patient's obsession that her fits were caused by a brain tumour was fully documented in the notes of the neurosurgical unit where the operation was carried out.

Meningiomas may be very slow growing and a history of symptoms for 23 years before diagnosis would not be unprecedented. A case of 42 years between the first symptom and diagnosis has been reported, and in one series of parasagittal meningiomas, 8% had histories of more than 10 years, extending to 37 years. In our patient, the fibroblastic nature of the tumour and the lack of oedematous reaction in the adjacent brain would accord with a very indolent course. Bifrontal atrophy caused by the leucotomy may have delayed the eventual appearance of symptoms of raised intracranial pressure, in the way that cerebral atrophy in elderly patients with cerebral tumours is believed to have this effect. It is even possible that the brief episode of depression which responded to electroconvulsive therapy 9 years before the leucotomy was the first symptom of the tumour. Prior to that time the patient had had no psychological disorder.

The psychiatric presentation of brain tumours is well documented. Intracranial tumours may give rise to symptoms simulating depression, anxiety states, hypomania and schizophrenia. Most often, slow-growing frontal meningiomas are responsible. Amongst cerebral tumours found in patients in psychiatric hospitals meningiomas are found twice as often as in the general population. In their early stages, premotor frontal tumours may not give rise to any abnormal neurological signs and with a slow-growing tumour gradual adaptation of the adjacent brain to deformation caused by the tumour may mean that development of symptoms of raised intracranial pressure may be long delayed. Most neurosurgeons have encountered patients with frontal meningiomas who have received prolonged psychiatric care before their tumours were diagnosed.

The leucotomy led to a dramatic improvement in the psychological symptoms although the epilepsy was unchanged. We have been unable to find a report of
any previous case where psychological symptoms caused by an unremoved cerebral tumour had been successfully treated by leucotomy but it is well-established that psychiatric symptoms caused by organic brain disease may at first respond well to other physical methods of treatment and this occurrence can be a pitfall for the unwary psychiatrist. We have experience of a further patient with a frontal menigioma whose initial depressive symptoms remitted for several months after electroconvulsive therapy.

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References


Kinesigenic foot dystonia as a presenting feature of Parkinson’s disease

Sir: Both Charcot1 and Gowers2 make brief reference to the occurrence of dystonic foot postures in patients with established Parkinson’s disease, but the symptom was disregarded until Duvoisin and colleagues reported that in-turning of the foot with associated dorsiflexion of the great toe could be aggravated by levodopa therapy.3 The occasional presentation of Parkinson’s disease with writer’s cramp is well known, but we would like to draw attention to a similar dystonic mode of onset in the foot as an early symptom, first described by Purves-Stewart in 1898.4

At the age of 42 years a marathon runner of international repute began to experience cramp-like discomforts in his right foot after running about 10 miles. This gradually became more severe and he began to notice a curling in of his toes with a tendency for his right foot to twist inwards; this would bring him to a halt, but after a rest period of a few minutes to half an hour he would be able to continue albeit for a shorter distance. Pain and discomfort became more incapacitating in the right foot and he underwent myelography and an abortive exploration of his right common peroneal nerve. Within a year of the onset of these symptoms he was forced to give up competitive running, finding that after only 5 minutes stiffness in the right popliteal region and splaying of his right foot would cause him to lose control and trip. A year later he noticed increasing difficulty with writing, micrographia was confirmed and shortly after this a mild rest tremor of the right hand was noted. Within a few months he found it almost impossible to write and typing became inaccurate. Even walking short distances on the level would cause his toes to cramp up for several minutes. Examination now revealed facies hypomimic with cogwheel rigidity of the right arm and a static Parkinsonian tremor. There was also unequivocal right-sided bradykinesia and a tendency after walking for his right foot to claw.

A woman who developed mild right-sided bradykinesia with micrographia and a postural tremor of the right hand at the age of 39 years, two years later found that after jogging for about half a mile she had to rest because of severe cramps and twisting spasms of her right foot causing her lower limb to buckle with pain. A short period of rest would enable her to continue and she found that running over pebbles would also temporarily relieve the discomfort. This patient subsequently developed severe early morning foot dystonia while receiving levodopa therapy.

In a third case, a man’s Parkinson’s disease presented at the age of 45 years with tremor of the left hand and within four years of the onset he was experiencing curling in of the toes of the left foot after walking fifty yards on the level. Rest and massage for two to ten minutes would enable him to continue for a further distance.

This effort induced phenomenon resembles the intermediate form of familial paroxysmal dystonic choreoathetosis5 and the dystonic seizures occasionally seen in multiple sclerosis.6 In preliminary studies on two additional Parkinsonian patients who were experiencing levodopa-induced early morning and end-of-dose dystonia,7 exercise on a bicycle has consistently induced shortlived dystonia of the affected foot even at times of peak dosage. Studies are in progress to determine the underlying pharmacological mechanism of this intriguing disability.

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References


Mild Reye’s syndrome in an adult

Sir: Since the description of Reye’s syndrome in 19631 several thousand cases have been notified in the USA alone.2 The small numbers of adult patients all had severe encephalopathies, often with fatal outcome.3-7

A 16-year-old male developed typical varicella having been exposed to the illness by his sister. Initial complaints were solely of rash and mild headache. On the fourth day of the illness he suddenly developed profuse vomiting, confusion and aggressive behaviour. He received no medication
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