

Seven Cases of Frontal Tumour with Psychiatric Presentation

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INTRODUCTION

In this article seven cases of frontal brain tumour, all of which were meningiomas, are reviewed and observation of their psychiatric presentation given. For various reasons frontal meningiomas are found more frequently in mental hospitals than in neurological centres (Hunter *et al.*, 1968).

Symptomatology may be (a) Neurological (b) Psychological general and specific causally related to the tumour, its site and type, pressure on various local structures or a general rise of intracranial pressure, (c) Psychological but more directly correlated with patterns of stress reaction due to impairment of function. These patterns are related to the premorbid personality type and to the patient's learned reaction to stress.

CASE HISTORIES

Case 1

This was a case of progressive dementia of presenile type, initially presenting as reactive depression due to marital discord, and developing neurological signs four years afterwards.

A 43-year-old Czechoslovakian man was referred in 1962 and admitted as a case of reactive depression with numerous other symptoms, such as headache and blackouts which were thought to be due to head injury sustained two years previously. He recovered sufficiently well to be discharged.

In 1966 he was readmitted in a state of apathy, with self neglect and inability to work, and also presented frontal headache, vomiting and bitemporal hemianopia. His thought content was not depressive, but his speed of thinking was reduced, associated with amnesia for recent events and rapid mood swings from euphoria to depression.

Radiologically he was found to have a right predominantly vascular cribriform plate meningioma, which was confirmed by excision. Twenty-seven months after operation he was improved generally, but had anosmia and persistent field defect, and was accident prone and still somewhat extrapunitive and paranoid. The change in personality was essentially an increase in his inability to cope with life in

England. In other words it was in the same direction as his previous personality in some respects. Irrevocable marital breakdown had occurred, he was just about functioning at his work, but although the outcome was overall a successful one he was still somewhat demented and euphoric.

Case 2

A woman of 56 was referred to a neurologist and then to a neurosurgeon with euphoria for three to four months, headaches and vomiting, loss of concentration, amnesia and impaired vision. On examination she was dull and facetious; no other information was available.

Radiology revealed a large olfactory groove meningioma which pushed backwards on both frontal lobes. This was excised, weighing 70 grammes. Follow-up after six years showed that her visual acuity was J1 right and left. She still had bilateral anosmia; no other information was available about her.

This case illustrates that in cases of basal tumour euphoria and facetiousness can co-exist with dullness (Hunter *et al.* 1968).

Case 3

A 49-year-old woman was admitted with a four-week history of right frontal headache, two weeks of inability to work, retardation and apathy, having retired to bed. She was immediately referred to a neurosurgeon. She showed disorientation in time, slowing of thought and mistakes in simple arithmetic. Her pupils were unequal and there was papilloedema, left hemiplegia and extensor plantar responses. A left carotid arteriogram and air and myodil ventriculography by burr hole revealed a temporo-parietal mass. After the patient died a meningioma was found at the inner one third of the right sphenoidal ridge.

This case illustrates that tumours at this site do not always cause excitement. The clinical deterioration was rapid, and most of the 'psychiatric picture' was due to raised intracranial pressure.

Case 4

A 44-year-old railway guard was admitted with a six week history of collapsing at work. His legs gave way, but he did not lose consciousness. This

was at first thought to be related to disciplinary trouble at work, but he also had headaches and some loss of memory.

There had been episodes of fainting three years previously; one associated with incontinence of urine. He noted that vision in the left eye had deteriorated. Persistent headaches had been present for 18 months, worse for the previous one or two weeks and severe in the morning whilst lying flat. He had been unable to work for six weeks.

On examination, his visual acuity was reduced more on the left than the right. No anosmia was present. Only the upper temporal quadrant was intact in the left visual field. The fundus showed some swelling on the left and papilloedema on the right. All reflexes were brisk and the plantar responses extensor. Radiologically there was a large vascular tumour on the floor on the anterior fossa near the cribriform plate, encroaching on the sphenoidal ridge. This was removed at operation. Afterwards this patient did well, and in spite of having bilateral anosmia on examination he did not complain of it.

This case illustrates the importance of full investigation of fainting occurring at this age, and the significance of being unable to work as a symptom of cerebral tumour and of raised intracranial pressure. In view of his determination to do so when recovered, this was obviously not related to his premorbid personality.

Case 5

A married woman, aged 51 years, who worked as a chambermaid in a local hotel and lived in a basement flat, had an epileptic fit followed by unconsciousness for twelve hours. She had had two blackouts eight months previously, and had suffered headaches, visual disturbances associated with lassitude, thirst, feeling cold and loss of weight.

On admission she had a right hemiparesis, and was pointing to the right side of her head as a site of pain. She was confused and had amnesia and nominal aphasia. The right pupil was dilated and there was a right field defect and reduction of ocular movements on the right. The fundi showed bilateral papilloedema. Arteriography revealed a tumour in the right frontal region. A cribriform plate meningioma arising from the right orbital roof was excised. In the immediate postoperative period, euphoria, ideas of grandeur, papilloedema, a raised temperature and photophobia were noted.

Later she suffered recurrent fainting attacks, lassitude, thirst, excessive sensitivity to cold and loss of weight; these symptoms were considered to be due to pituitary damage and treated with replacement steroids and thyroxine.

One could presume that there was some difficulty in her relationships with people generally, and particularly with her husband and her daughters, possibly related to her tumour, brain damage or premorbid personality. However she kept working.

Case 6

A man of 62, married, who was a retired musician and until just previously had owned a music shop inherited from his father, was admitted with a history of loss of visual acuity, worse on the left than the right for 18 months, and of dizziness and frontal headaches for three years. There had been no loss of consciousness. He had, however, had periods of drowsiness, apathy and euphoria.

At the age of 22 he had had a car accident but sustained no head injury. His premorbid personality had always been irascible, forgetful, erratic and somewhat euphoric, with a vivid imagination but was restricted by his parents. On examination he had complete anosmia, a right homonymous hemianopia and a constriction of the fields with bilateral optic atrophy. Acuity was greatly reduced in both eyes, worse on the left than the right. There was a possible left facial weakness and left Babinski response. Radiology revealed a probable suprasellar tumour, which proved to be a meningioma in the tuberculum sellae, both edges of the sphenoidal ridge and a centimetre anterior to each. The tumour was removed as completely as possible, but dissection was tedious in places.

At follow-up there was some amnesia and euphoria and there was also emotional lability, though this had been present before his illness. WAIS testing was possible only verbally and was recorded as 112, but I think it would have been higher than this before the onset of his illness. He claimed that his concentration, general health, vision, hearing and smell and the 'dermatitis' on his leg had improved since the operation. He had a loss of social judgement and insight; he admitted to a persistent lack of energy; he over-rated his various intellectual activities, but admitted a lack of libido. There was depression and mood swing. His speech was hesitant and there was disorientation in time (he tended to connect distant and recent events). Visual deterioration made him more nervous than usual when his wife drove the car.

In summary, this man had some euphoria in relation to a complex premorbid personality, with some mood swing to depression and evidence of mild to moderate dementia, lack of insight, confabulation, perseveration and a lack of social judgement. His personality change was essentially an exaggeration of his premorbid state.

Case 7

A man of 58 presented with a year's history of reported extravagance, boastfulness, excessive drinking, planning unrealistically, several changes in his job and marital discord. He had previously held a senior responsible post in a shoe factory and it appeared that some personality deterioration had taken place. Initially it was difficult to determine how far this was a functional state relating to the marriage and to his mother-in-law's presence at his home. He had been impotent for ten years and this had been a source of disharmony. In his family two relatives had committed suicide.

Two weeks after initial presentation he was admitted with bulimia and hypersomnia associated with wandering in the streets. He had a happy, confident brisk manner, believing he was rich, which was belied by his self-neglect and a sixpence in his pocket. He had gross lack of insight and lack of time sense. Physical examination revealed bilateral anosmia, brisk tendon reflexes, greater on the right than on the left, and extensor plantar responses; a left sided early papilloedema and reduced visual acuity. X-ray showed frontal calcification in the sphenoidal region. EEG showed a high voltage slow waves at 3 c/sec. WAIS was unhelpful. Brain scan revealed increased uptake in the lower frontal region. Left carotid arteriography revealed an olfactory groove meningioma confronted at operation with local hyperostosis. (c.f. Morgagni's syndrome—Hunter *et al.*, 1968).

After operation he had a persistent field defect in the left eye, except the lower nasal quadrant, behaved in a disinhibited way sexually, and also showed paranoid features, aggression, confabulation and poor insight. He was also noted to have vague tangential and irrelevant thoughts with some periods of nihilism and disorientation. He gradually improved but when seen two years after operation he still had the visual defect and anosmia and a rather brisk paranoid attitude with a tendency to over-evaluate his position. He was living with his wife and mother-in-law and was still impotent. His drive and time sense had returned. He was fully orientated, but lacked insight and made elementary mistakes in the WAIS. He had an inappropriate optimism about the cause of his illness, and his comment on 'mind over matter' implied that he believed it was a mental illness. His belief that his sight would improve implied that he thought this was functional too. At first sight he appeared to have made an excellent recovery, and his general smart appearance and demeanour presented a façade which hid some persistent defects.

This case illustrates the classical change from a

respected and reasonable to an extravagant, drinking, socially declining person. It is noteworthy that a continuous swing from euphoria to apathy and *vice versa* occurred both before and after operation.

TABLE I

| Cases | 1 | 2 | 3 | 4 | 5 | 6 | 7 | Range or totals |
|------------------------------------|----|----|-----------------------|----|----|----|----|--------------------|
| Age (years) | 43 | 56 | 49 | 44 | 51 | 62 | 58 | 43-62 |
| Personality change from premorbid | | | | | | | | |
| + = exaggerated | | | | | | | | 2 5 |
| - = opposite | | | | | | | | + - |
| Length history (months) | 48 | 4 | 1 | 36 | 8 | 36 | 72 | 1-72 |
| Mental symptoms | + | + | + | + | + | + | + | 7 |
| Mania | + | + | - | - | - | + | + | 4 |
| Depression | + | + | + | - | - | + | + | 5 |
| Mood swing | + | + | - | - | - | + | + | 4 |
| Work inability | + | - | + | + | + | + | + | 6 |
| Anergy | + | - | + | + | + | + | - | 5 |
| Amnesia | + | + | + | + | + | + | + | 7 |
| Personality deterioration | + | + | + | + | + | + | + | 7 |
| Physical symptoms | + | + | + | + | + | + | + | 7 |
| Headache | + | + | + | + | + | + | - | 6 |
| Motor activity | | | | | | | | |
| Increase + | + | - | - | - | + | - | + | 3 4 |
| Decrease - | | | | | | | | + - |
| Thought | | | | | | | | |
| Slowed - | - | - | - | - | - | + | + | 2 5 |
| Speeded + | | | | | | | | + - |
| Disorientation | + | + | + | + | + | + | + | 7 |
| Bilateral anosmia | + | + | - | + | + | + | + | 6 |
| Changes in acuity | + | + | - | + | + | + | + | 6 |
| Changes in fields | + | + | - | + | + | + | - | 5 |
| Changes in fundi | + | + | + | + | + | + | + | 7 |
| Changes in pupils | + | + | + | + | + | + | - | 6 |
| Differential diagnosis: | | | | | | | | |
| Presenile dementia | + | - | + | - | + | + | + | 5 |
| Space-occupying lesion | + | + | + | + | + | + | + | 7 |
| Radiology aid to diagnosis | + | + | + | + | + | + | + | 7 |
| Treatment: | | | | | | | | |
| Craniotomy | + | + | Burr-hole | + | + | + | + | C 6 B.H. 1 |
| Removal portion frontal lobe | + | + | - | + | + | + | + | 6 |
| Origin basal meningioma | + | + | + | + | + | + | + | 7 |
| Extension | + | + | - | + | + | + | + | 7 |
| Intracranial localized pressure | + | + | Raised General I.C.P. | + | + | + | + | Local 6 Gen-eral 1 |
| Adherence | + | + | - | + | + | + | + | 6 |
| Persistent postoperative symptoms: | | | | | | | | |
| Physical | + | + | Died | + | + | + | + | 6 |
| Mental | + | ? | Died | - | + | + | + | 4 |
| Return of drive | + | - | - | + | + | + | + | 5 |
| Work ability | + | + | - | + | + | - | + | 5 |

DISCUSSION

Selecki (1965) describes mental symptoms of various brain tumours and reminds the reader of chronic subdural haematomas in patients, such as alcoholics or epileptics in institutions, which might be considered in the differential diagnosis of dementia. He indicates that some symptoms and signs could have localizing value along the callosal axis. Fluctuations of symptoms would not rule out neoplasia. He describes frontal lobe tumours as presenting in the following order of frequency of symptoms: impairment of memory, inability to concentrate, depression, loss of interest, loss of planning ability, epilepsy and headaches. Loss of insight, fatuous euphoria, delusions, dysgraphia and dysphasia were also encountered. No papilloedema was found in this group, although raised intracranial pressure in the later stages of the disease was demonstrated in all cases. He stresses a high incidence of tumours in hospitalized psychiatric patients unrecognized during life, averaging 55 per cent of all such tumours, and a high incidence of bilateral and unilateral deep seated frontal tumours. He makes the following points:

(1) Benign (meningiomas) are more frequently encountered in autopsy material in mental hospitals (24.4–46.4 per cent) than in non-mental hospitals (6–18 per cent), that is of all brain tumours discovered (Patton and Sheppard (1956). (Dorothy Russell in her work with Rubinstein *The Pathology of Tumours of the Nervous System* gives the overall incidence of all brain tumours in general hospitals as about 1 per cent.)

(2) Meningiomas are four times as common in 'psychiatric' patients as in general hospital patients.

(3) Meningiomas are the most common tumours producing mental symptoms.

(4) Meningiomas frequently produce only psychiatric symptoms, and patients therefore tend to gravitate to psychiatric hospitals rather than to general or neurological units.

This particularly applies to the frontal region. Some had received psychiatric treatment for as long as 1–13 years, and in Hunter's series, incidentally, it was even longer than this in one case. Selecki's cases come mainly

from acute psychiatric wards where the rapid progression of malignant tumours enabled a correct early diagnosis to be made. However, benign excisable tumours can be found in the long-term patients in chronic wards, where vomiting and pain are reduced by phenothiazines, and this delays diagnosis. The incidence of organic mental reactions in tumours was highest in the frontal region where a 'leucotomy-like' state might be produced of heightened pain-threshold and lethargy.

Selecki, and Hunter *et al.* reiterate Morse's comments on 'lack of tumour consciousness in mental hospitals'. It is true that one often sees only what one is looking for. Sometimes prognosis of cerebral tumour is not necessarily equated with a poor outlook, as might be assumed.

From this small review the following points can be made:

(1) The age range of the patients presented was 43–62 years.

(2) Only in Case 1 was there a definite history of head injury.

(3) The change from normality occurred in the opposite direction to the premorbid personality in five cases and was an exaggeration of it in two.

(4) The length of history varied from four weeks to six years.

(5) Mental symptoms of some kind were present in every case. Depression was present in five out of seven cases and apathy in five. Inability to work was present in six and lack of energy in five. All cases showed recent impairment of memory and orientation and personality change. Four showed euphoria at times. Five showed depression at times and four had rapid mood swing. Thoughts were slowed in the five depressed cases and speeded in two euphoric ones.

(6) The seven patients had physical symptoms which lasted from four weeks to six years. Headache and impaired vision were present in six cases. General motor activity was altered in seven cases. Bilateral anosmia was present in six cases. Changes in fundi in seven, acuity in six, fields in five and pupil changes in six.

(7) The differential diagnosis of presenile dementia was considered in five cases, cerebral

tumour in seven. General Paresis was also considered in four cases and excluded in all.

(8) The most useful methods of diagnosis are:

(a) a reliable and full psychiatric and neurological history and examination.

(b) a plain skull X-ray carefully scrutinized by a competent radiologist, showing possible hyperostosis, pressure change in the sella turcica, erosions of the sphenoidal ridges and enlargement of one or other of the foramina spinosa.

(c) arteriograph to help further to localize and type the mass, reported on competently by a neurologically orientated radiologist.

In the four cases where EEG was used it contributed to the localization of the tumour.

(9) Craniotomy was performed in six cases, burr hole in one and removal of portions of the frontal lobes in six.

(10) The tumours were basal frontal meningiomas in all seven cases, extension was present in six cases and pressure on the local structures and adherence in six.

(11) Immediate improvement followed excision in the six cases who survived; long-term improvement varied with social and other factors, influenced by the operation or the preoperative state, such as visual field defect. The six cases had persistent physical symptoms.

(12) Of six survivors, four had known persistent mental symptoms, one probably had, and in one case it was not known.

(13) Return of drive and ability to work were important social results of the operation.

SUMMARY

A review of seven cases of frontal tumour presenting with psychiatric and other symptoms is given, with particular emphasis on presenting

personality change, work ability, amnesia, disorientation and alteration in motor activity.

History, examination and radiology, including angiography, were most helpful in diagnosis. The seven tumours were basal, frequently extending and adherent and pressing on local structures. Persistent disabilities, both mental and physical, are noted after operation, and subsequent improvement is marked by return of drive and ability to work.

In the diagnosis and treatment of these patients, the general practitioner, social worker, mental welfare officer, neurologist, psychiatrist, neurosurgeon and their ancillary departments, EEG, pathology and radiology all play vital roles. In the hope of earlier diagnosis, and in view of this persistent postoperative symptoms, there is need for closer co-operation between departments.

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REFERENCES

- HUNTER, R., BLACKWOOD, W., and BULL, J. (1968). 'Three cases of frontal meningiomas presenting psychiatrically.' *British Medical Journal*, *iii*, 9-16.
- PATTON, R. B., and SHEPPARD, J. A. (1956). 'Intracranial tumours found at autopsy in mental patients'. *American Journal of Psychiatry*, *113*, 319.
- SELECKI, B. R. (1965). 'Intracranial space-occupying lesions among patients admitted to mental hospitals'. *Medical Journal of Australia*. *i*, 383-90.

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