A case of metastasizing meningioma

A. GORDON and A. F. J. MALONEY

From the Neuropathology Laboratory, Department of Pathology, University of Edinburgh

Primary intracranial neoplasms rarely give rise to distant extracranial metastases despite the fact that many of them possess the other attributes of malignant neoplasms. Paradoxically it appears that extracranial metastases are more liable to originate from the less rather than the more malignant primary intracranial neoplasms (Russell and Rubinstein, 1963). Cushing and Eisenhardt (1938) reported only one example of metastasizing meningioma, while Kernohan and Sayre (1952) found none in their series of 794 meningiomas.

We describe in the following case report an intracranial meningioma which metastasized to the thyroid gland and lungs.

CASE HISTORY

A man aged 42 presented in December 1952 with a short history of intermittent motor and sensory disturbances in the right limbs and more recently headache, right homonymous hemianopia, and dysphasia. Clinical and angiographic examination indicated a left parietal lesion. In January 1963, through a left parieto-occipital craniotomy a tumour approximately $7 \times 5 \times 5$ cm. arising from the falx and embedded in the left parietal lobe was removed. A small extension through the falx to the right of the midline was left.

The tumour, received in two portions, was lobulated, partially encapsulated, firm, haemorrhagic in some areas and greyish white in others. It was highly cellular in most areas, composed of oval or spindle-shaped cells (Fig. 1). The cell boundaries were indistinct, the cytoplasm moderate in amount and sometimes clear. The cells were arranged in sheets traversed by numerous thin-walled vascular channels; small, ill-defined whorls were delineated by a reticulin network (Fig. 2). There were no psammoma bodies. Parts were composed of fibrous tissue containing a few strands of tumour cells. A few mitotic figures were present but pleomorphism was not conspicuous.

Post-operatively, radiotherapy (4,750 r) was given. The patient made an excellent recovery and was able to resume his normal work. He remained practically symptom free except for slight proprioceptive difficulties in the right foot until 1960 when over a period of four months this deficit became severe and extended to involve both lower limbs and to a less extent the upper limbs. Minor sensory epileptic manifestations were experienced in the right hand, and on one occasion there was a transient loss of consciousness. Examination indicated recurrence of the

tumour and in December 1960 a biparietal craniotomy was performed. The tumour, which lay between and embedded in the parietal lobes, arose from either side of the falx, on the right side forming a mass $5 \times 5 \times 3$ cm. and on the left $3 \times 2 \times 2$ cm. It was removed, apparently completely, together with its attachment to the falx and a portion of the superior longitudinal sinus.

The tumour had the gross appearance of a meningioma, being discrete with an irregularly lobulated smooth surface. The cut surface was granular, white and showed small foci of old haemorrhage. Microscopically it was identical with the original.

The patient again made a good recovery, hampered only by gross spatial disorientation of both legs. Despite this he was able to walk up to two miles and carry on his business. In the middle of 1962 the neurological symptoms gradually became worse. He now had frequent focal epileptic attacks affecting the left lower limb, hyper-tonus in both lower limbs, and bilateral extensor plantar reflexes. Investigation in September 1962 revealed, however, no evidence of raised intracranial pressure, his general condition was good, and he showed no intellectual deterioration. Swelling of the right lobe of the thyroid gland was noted. This had been present for five weeks. The swelling was firm and painless and had displaced the trachea to the left. No bruit was audible. As no further neurosurgical procedure was indicated and the thyroid enlargement was considered to be a separate problem he was referred to a general surgeon. In October 1962 the right lobe of the thyroid gland infiltrated by tumour was removed.

The tumour, which measured $4 \times 2.5 \times 2.5$ cm., was irregular in outline and infiltrated most of the lobe. It was pale and firm and showed some haemorrhage. Microscopically it closely resembled the intracranial tumours previously removed (Figs. 3 and 4). Surviving thyroid acini were seen within the tumour limits.

Gradually thereafter the patient's neurological condition deteriorated and he became confined to a wheel chair. There was still no evidence of raised intracranial pressure. A chest radiograph taken shortly before death in June 1963 revealed multiple metastases in both lungs. The necropsy was performed by Dr. T. G. Napier who kindly permitted us to examine the brain and relevant histological sections from various organs and to use his report.

SUMMARY OF THE NECROPSY REPORT

There was a large biparietal bony defect deep to which the meninges were thickened, particularly on the right side.

FIG. 1. Cerebral tumour: highly cellular with suggestion of whorling and containing numerous irregular vascular channels. Haematoxylin and eosin $\times 200$.

FIG. 2. Cerebral tumour: reticulin network about small groups of tumour cells. Slidder's $\times 200$.

FIG. 3. Thyroid tumour: acini encircled by tumour. Haematoxylin and eosin $\times 100$.

FIG. 4. Thyroid tumour: showing similarity to Fig. 1. Haematoxylin and eosin $\times 300$.

Attached to the inner surface of dura on the floor of the anterior fossa were several small tumour nodules up to 5 mm. in diameter which indented the frontal lobes. The brain was removed and fixed in formalin. The spinal cord and leptomeninges appeared normal.

The site of the right lobe of the thyroid gland was occupied by scar tissue; the left lobe was normal. Both lungs contained numerous rounded, white, friable tumour nodules which varied in size from 0.5 to 4 cm. in diameter. There was a terminal pneumonia. No further tumours were found elsewhere in the body and there were no relevant pathological changes in the alimentary, urogenital and cardiovascular systems, or the suprarenal glands.

Examination of the brain after fixation showed that residual fragments of falx and dura were firmly adherent to the parietal convexities. A portion of the superior longitudinal sinus had been surgically removed. The remainder was patent as far as the torcular.

The cerebral hemispheres were swollen and the gyri flattened, the right more than the left, and there was herniation of the medial surface of the right frontal lobe beneath the falx. Large areas of cortical scarring were seen on the supero-medial border of each parietal lobe extending laterally on to the convexities and anteriorly on to the medial surfaces of these lobes. There was no uncal or tonsillar herniation.

Serial coronal sectioning of the cerebrum revealed a large firm mass of tumour lying between the two cerebral hemispheres above the corpus callosum and adherent to the falx (Fig. 5). It involved the medial surface of each cerebral hemisphere, apparently by a process of indentation rather than infiltration. The tumour was firm and lobulated, and scattered throughout were tiny areas of cystic necrosis. It measured 12.5 cm. antero-posteriorly by, in the right hemisphere, 5 × 4 cm. and in the left, 3 × 4 cm. A separate nodule 3 cm. in diameter was embedded in the right superior parietal lobule immediately behind the main mass close to the operative site. The corpus callosum was attenuated and displaced downwards, compressing the bodies of the lateral ventricles and splaying the third ventricle. There was no calcarine infarction.

FIG. 5. Coronal section of cerebrum: tumour embedded in medial surfaces of cerebral hemispheres.

A small extension, 1 cm. diameter, from the main tumour was embedded in the superior surface of the right cerebellar hemisphere close to the mid brain.

HISTOLOGY

All sections of the main cerebral mass showed tumour (Fig. 6) which was identical to that removed from the brain in 1953 and 1960 and from the thyroid in 1962. Areas of necrosis were common but mitotic figures were rare. Its margins were sharply demarcated from the adjacent compressed and gliotic nervous tissue. In places a distinct layer of fibrous tissue was interposed.

The small nodules attached to the inner surface of dura in the floor of the anterior fossa and the tumours

FIG. 6. Section of intracranial tumour taken at necropsy. Haematoxylin and eosin × 200.

in the lungs (Fig. 7) all had a structure identical to one another and to the intracranial tumour. Those in the lung, however, were locally invasive.

The spinal cord showed slight loss of myelin from the lateral columns. No tumour was present in the meninges or cord.

No tumour was seen in the left lobe of the thyroid gland.

DISCUSSION

We submit that the intracranial tumour in this case is a meningioma by its appearance and mode of growth, while histologically its structure corresponds to that of the angioblastic variety. The thyroid and pulmonary nodules are identical with the intracranial tumour except for the slight evidence of invasive growth and we consider that they are

FIG. 7. Pulmonary metastasis in the wall of a small bronchus. Haematoxylin and eosin $\times 200$.

metastases. The alternative view that the primary tumour arose in either the thyroid gland or the lung and metastasized to the brain appears unlikely for the following reasons: the clinical presentation and evolution; the prolonged survival; the gross and microscopical appearances of the cerebral tumour; the late presentation of the thyroid tumour and its failure to recur after resection; the discrete, multiple lung tumours which looked like secondaries.

Local invasion by a meningioma is not uncommon. It is usually restricted to dura and venous sinuses, but may extend externally to reach the scalp or internally into the brain. Metastases, however, are exceptionally rare. Kruse (1960) reviewed cases before 1960 and found 20 acceptable examples and added two of his own. Single cases have also been recorded by Meredith and Belter (1959), Robertson (1960), Hukill and Lowman (1960), Noto and Gyorfi (1961), Arnould, Lepoire, Pierson, and Barrucand (1961), and Russell and Rubinstein (1963). This behaviour is not confined to one histological type of meningioma. The structure of metastasizing meningiomas ranges from the well-differentiated, psammomatous or arachnoidal type (e.g., Ringsted, 1958) to the anaplastic (e.g., Russell and Sachs, 1942) and includes the angioblastic variant (Hukill and Lowman, 1960).

The recorded sites of the metastases are usually lung and liver, suggestive of a haematogenous spread (Kruse, 1960) but rarely they occur in the cervical

lymph nodes following invasion of the scalp (Ringsted, 1958). The penetration of major venous sinuses by a meningioma is not uncommon whereas metastases are; the factors governing their appearance are unknown. The opening up of a channel of communication between cranial cavity and the exterior by operation is not essential (e.g., cases of Jurow, 1941; Vlachos and Prose, 1958).

Our patient survived for 10 years after the first operation; the average survival of patients with metastatic meningioma is about nine years with an extreme of 24 years (Ringsted, 1958). Residual or recurrent tumour is usually found within the skull but metastases have been reported in the liver up to 15 years after complete removal of the primary tumour (Russell and Sachs, 1942, case II).

SUMMARY

The rare occurrence of metastases from a meningioma cannot be correlated with its histological structure. In spite of such behaviour the average survival time is long. We record a case of angioblastic intracranial meningioma, treated successfully twice which ultimately metastasized to the thyroid and lungs. The thyroid nodule presented as an apparently separate diagnostic problem.

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