Coincidental meningioma and glioma

Report of two cases

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The authors report two cases in each of which both a meningioma and a glioma were present. The anatomical and clinical presentations of double tumors are reviewed and their pathological significance considered. Some suggestions for the recognition and treatment of such tumors are offered.

KEY WORDS • glioma • meningioma • multiple tumors

The occurrence of multiple meningiomas or gliomas is a well recognized feature of the central form of neurofibromatosis, and either multiple meningiomas or multiple gliomas have also been recorded in patients without this predisposition. However, the occurrence in a single patient of a meningioma and glioma, each solitary, is observed less frequently. We wish to report two cases in both of which a single meningioma and glioma were present. The anatomical and clinical presentations, which differed in each patient, will be discussed and compared with previously reported cases. The potential pathological significance of such coincident lesions will be considered, and some observations offered on the management of such patients.

Case Reports

Case 1

A 56-year-old woman was admitted with a 3-week history of focal fits affecting the left fingers; for 1 week there had been progressive weakness of the left hand, as well as headache, incontinence, disturbance of gait, and urgency of micturition.

Examination. On examination the patient was drowsy and had a left hemiparesis, most pronounced in the face and arm: as the investigation proceeded, left hemisensory disturbance and papilledema also developed despite the use of dexamethasone. Plain skull films showing calcification and overlying hyperostosis in the right postfrontal parasagittal region indicated the presence of a meningioma. Electroencephalography and an isotope scan also suggested abnormalities in this region. Angiography (Fig. 1) showed leftward displacement of the distal right pericallosal artery; the posterior frontal branches of the anterior cerebral artery were also displaced away from the area of calcification. Depression of the Sylvian vessels suggested a substantial mass effect in the frontal lobe. There was no tumor circulation from either the internal or external carotid system. A computerized axial

Fig. 1. Left: Right carotid angiogram (Case 1), anteroposterior view, showing leftward displacement of pericallosal artery. Right: Right carotid angiogram (Case 1), lateral view, showing displacement of posterior frontal branches of anterior cerebral away from a small area of calcification, and depression of Sylvian vessels indicating a large frontal mass effect.

tomography (CAT) scan indicated an ill-defined area of increased density high in the right hemisphere near the midline. A meningioma was considered most likely from the radiological findings, although it was noted that the lesion could be intracerebral, and the clinical presentation seemed strongly in favor of the latter site.

First Operation. At operation, a typical calcified meningioma in the right frontoparietal parasagittal region was completely excised. The underlying brain appeared normal. According to our usual practice, neuromuscular blockade was reversed following hemostasis; very marked brain swelling developed, requiring a further period of hyperventilation. Although this development is sometimes observed following decompression of gliomas, the degree of swelling was considered unusual for a meningioma; the bone flap was removed temporarily.

Postoperatively, there was a complete left hemiplegia with some persistent tension in the wound, but after 8 days some movement in the leg returned. However, over the next 4 weeks the patient’s condition again deteriorated. An isotope scan indicated that the original uptake had now increased. A CAT scan showed increasing edema of the hemisphere; there was no evidence of tumor, although Conray enhancement was not attempted. A further angiogram indicated a large intracerebral, avascular mass.

Second Operation. At reexploration, swollen, diffusent cortex presented under tension, with typical glioblastoma at a depth of 3 cm. An internal decompression was carried out and the bone flap was now replaced. As anticipated the patient’s hemiplegia was unchanged postoperatively.

Histological Examination. The first tumor was composed mainly of elongated and crescentic cells with pale nuclei and delicate chromatin. These cells formed whorls, some with a central capillary, or interweaving bundles interspersed with bands of coarse collagen. The microscopic diagnosis was meningioma. The second tumor was a glioblastoma multiforme: most tumor cells had dark, hyperchromatic nuclei and indistinct cell margins, and mitotic figures were seen. The tumor contained areas of necrosis and hemorrhage, and the blood vessels displayed endothelial hyperplasia.

Case 2

This 53-year-old man complained of speech disturbance for 6 weeks and increasingly severe headache.

Examination. On examination, there was a moderate expressive dysphasia with some dyscalculia, bilateral papilledema, and a right homonymous, upper quadrantic field defect. There was a mild right hemiparesis, with some sensory inattention in the right upper
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limb. An isotope scan showed an ill-circumscribed zone of uptake in the left temporal region, and angiography indicated a large, superficial mass within the anterior and middle portions of the left temporal lobe, with early filling veins. The preoperative clinical and radiological diagnosis was glioblastoma.

Operation. At surgery, a glioma was demonstrated surfacing on the middle temporal gyrus, and a 6-cm lobectomy was performed. A meningioma lying on the inner one-third of the sphenoidal wing was also found and removed; it ran medially as far as the petroclinoid ligament and measured 3 × 1.5 cm. Postoperatively, the patient made an uneventful recovery, with resolution of papilledema and loss of headache. At the time of his return home his deficits were minimal.

Histological Examination. The lobectomy specimen contained a tumor composed of densely packed pleomorphic cells with numerous mitoses. It had considerable endothelial hyperplasia and extensive areas of necrosis, and appeared to be a glioblastoma multiforme (Grade IV). The second tumor was composed of cells with uniform oval nuclei and was divided into lobules by fibrous septa. It appeared to be a syncytial meningioma.

Discussion

The manner of presentation of coincident tumors, both in our own cases and in those of other authors, may be classified in three groups. First, a clear discrepancy may exist between clinical and radiological findings. Such a discrepancy apparently led Nagashima, et al., to a correct diagnosis of dual pathology, where the radiological finding of an intrinsic left frontal mass was felt to be incompatible with anosmia. Similarly, in our first case, the radiological diagnosis of meningioma was at variance with the 3-week history of focal fits and 5 days of progressive hemiparesis. It is possible that CAT with Conray enhancement may demonstrate two separate tumors if clinical and angiographic data are in conflict.

The second presentation of double tumors arises where an additional, asymptomatic, tumor is disclosed incidentally during routine surgery or autopsy, where the primary causal lesion is not in doubt. Our second case, an incidental meningioma, falls into this category; Alexander's patient may be similarly categorized.

Third, unexpected pre- and postoperative deterioration may occur, as in our first case, in which swelling of the tumor bed occurred to an unexpected degree prior to closure of the craniotomy. Likewise, during convalescence, the steady increase in swelling in the decompression (bone flap removed) over some weeks defied diagnosis until reexploration was undertaken. The second tumor reported by Feiring and Davidoff, a glioma, also presented with unexplained postoperative deterioration, and the glioma in the second case of Sackett, et al., was probably responsible for both preoperative brain swelling and papilledema, which persisted subsequently.

In an attempt to provide an explanation for the occurrence and pathogenesis of multiple tumors in a single patient, both Arieti and Myerson invoked the concept of a locally acting and nonselective variation in germ-cell plasm or in the local environment. However, as Alexander points out, such a locally acting factor is unlikely to be responsible for the development of tumors at some distance from one another, and in some cases, in opposite hemispheres. At a more fundamental level, we see no reason to depart from the views of Russell and Rubinstein, Alexander, and Sackett, et al., that two not uncommon tumors, each distributed randomly in the same population at risk, will occasionally occur together in the same patient. No factor other than this simple law of chance need be invoked to explain two tumors coincident in the same patient. On the same basis, the anatomical distribution of such tumors will vary randomly, with the majority being separate (our Case 2, and that reported by Alexander), while a few are adjacent (our Case 1, and that reported by Gass and van Wagenen).

In the recognition and management of patients with double tumors, the possibility of a dual pathology should first be considered where a discrepancy exists between clinical and angiographic data. Scanning with CAT may clarify the diagnosis. Where doubt remains, the intracranial exploration should be more comprehensive, and the lesion initially revealed and removed should not be
accepted as the sole, definitive diagnosis without careful examination and perhaps biopsy of the surrounding brain. Finally, unexplained intra- and postoperative deterioration may occasionally be due to a second neoplasm rather than to any of the better recognized postoperative complications of intracranial surgery.

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References


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