Letter to the Editor

SPINAL SUBARACHNOID SPREAD OF PITUITARY ADENOMA

SIR,—Two of us (Ogilvy and Jakubowski, 1973) recently reported in this journal a highly unusual case of chromophobe pituitary adenoma which had given rise to a large, discrete, ‘seedling’ deposit in the parietal, parasagittal region (case I in the paper). We discussed the possible modes of spread, the problematic and sometimes pedantic classification of these tumours, and stressed the fact that the biological behaviour of pituitary adenomas does not necessarily run parallel to the tumour cytology.

A few months after publication of this paper the patient concerned died as a result of chronic respiratory and cardiovascular disease. His death occurred six years after removal of the original pituitary fossa tumour and two years after removal of the parietal parasagittal deposit.

At necropsy there was no evidence of recurrence in the parietal region but the pituitary fossa contained a small nodule of residual tumour which was invading the right cavernous sinus. Examination of the spinal cord revealed two circumscribed ‘seedling’ deposits within the subarachnoid space of sufficient size to give rise to spinal cord compression. The larger deposit was situated on the ventral aspect of the upper lumbar cord and measured 3 cm x 1.3 cm x 0.6 cm. The localization of this deposit in the subarachnoid space can clearly be seen in the Figure. The underlying cord was markedly indented but not infiltrated. The smaller deposit, 0.7 cm in diameter, was situated 2 cm distal to the main mass. This was surrounded by radiating blood vessels derived from the host meninges. Histologically, these spinal deposits were generally typical of chromophobe pituitary adenoma showing a fairly active growth rate, and were similar to the original pituitary tumour and the subsequently removed parietal deposit. At the lumbar level several small isolated clusters of tumour cells could be seen within the pia-arachnoid membrane. The general necropsy revealed an old myocardial infarct and evidence of chronic respiratory and urinary tract infection.

In the original paper we reported the fact that out of a total of 20 cases of pituitary adenoma in which the cerebrospinal fluid deposit had been examined for tumour cells, the only two cases having positive results subsequently proved to show intracranial dissemination of tumour. This fact, together with the clear localization of these spinal deposits within the subarachnoid space, emphasizes the importance of examination of the cerebrospinal fluid for tumour cells in all cases of pituitary adenoma.

We are, etc.,

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REFERENCE

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