IS MENINGIOMA GROWTH ASSOCIATED WITH HORMONAL REPLACEMENT USING SEX STEROIDS?

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INTRODUCTION

Hormone replacement therapy (HRT) is used for the relief of climacteric symptoms or as prophylaxis against the long term effects of oestrogen withdrawal. We report two patients who received sex steroids as hormonal replacement for a considerable period of time, and presented with symptomatic meningiomas.

CASE 1: A 47 year old female, was started on HRT for menopausal symptoms in August 1989. She was asymptomatic at first, but by July 1990 she developed progressive bifrontal headache, forgetfulness and unsteadiness of her gait. Neurological examination was unremarkable except for papilloedema in the right eye. The computed tomographic (CT) scan of the head showed a right sphenoid wing meningioma (Fig 1). The HRT was withdrawn and she underwent an uneventful complete excision of the meningioma on 9th January 1990.

CASE 2: A 12 year old female underwent aspiration of a cystic craniopharyngioma via a right frontal blurr hole in 1972 followed by radiotherapy. She made an excellent post operative recovery. However, she later developed panhypopituitaris and was treated with thyroxine, cortisone and sex steroid (oestrogens and progesterone) replacement from 1973. Check CT scans showed complete regression of the craniopharyngioma. At the age of 25, in 1985 she was treated for infertility with hormone manipulation using gonadotropin injections. In July 1987 she delivered quintuplets. In December 1987 she complained of progressive bifrontal headaches and deterioration in the vision of her left eye. A CT scan showed a moderate sized left frontal parasagittal meningioma (Fig 2). She underwent an uneventful excision of this tumour in May 1988 and made an excellent post-operative recovery.

Meningiomas are the commonest, benign, slow-growing tumors. They comprise 19% of all intracranial neoplasms, and are usually detected in the middle decades of life with a female to male ratio of 3:1. Considering their slow growth, the majority of meningiomas must arise during the period of maximal gonadal activity, at least in women.

Several studies have confirmed sex steroid hormone binding proteins in meningioma cells, and also the hormonal depandancy of cerebral meningiomas in vitro. The
documented association between mammary and/or genital cancer in the same patient has further raised the possibility that oestrogen and progesterone receptors might be present in these tumours. Bickerstaff et al have shown the presence of intra-cranial pressure symptoms in pregnancy or in the menstrual cycle from a meningioma that is clinically silent at other times. In case 1 it is possible that the HRT enhanced the growth rate of the lesion which was probably present for some years, thus producing symptoms. In case 2 it is tempting to postulate that the sex hormones actually induced the meningioma. Another possible etiological factor is radiotherapy which could have induced the second tumour. This has been reported previously. However, the radiotherapist felt that the meningioma had occurred outside the confines of the initial radiotherapy field.

Should we then adopt a more cautious approach in patients who require sex steroid replacements? Though we do not have conclusive evidence of the relationships between HRT and meningioma growth, we recommend that any patient on HRT who develops neurological signs or symptoms be investigated as early as possible since meningiomas can cause serious neurological disability. There is insufficient evidence to recommend any change in the current practice of using HRT, as the occurrence of these tumors may have been purely by chance. Currently, a major study is being undertaken to investigate the relationship of HRT and the occurrence of meningiomas at Royal Preston Hospital, Preston.

REFERENCES