developed abnormal vision, 3 suffered eye discomfort and 1 had tired eyes. One 57-year-old patient experienced a photosensitive facial rash with conjunctival reaction after sitting in the sun following administration of a single tablet of minocycline. There have been a total of 512 recorded cutaneous reactions to date associated with minocycline, including 100 cases of rash, 25 cases of photosensitivity eruption, 45 cases of angioedema and 41 cases of bullous dermatoses. One patient was reported to have suffered an intense photosensitive facial reaction around the eyes and trunk to a degree that the irritation prevented her from leaving her house for the duration of the symptoms.

Whilst ocular effects of minocycline are rare, we would recommend that patients being started on such treatment be advised to seek ophthalmic advice if ocular symptoms occur. Although this represents the first documented case of minocycline-induced actinic keratoconjunctivitis it may be reported more frequently in the future with the increased use of minocycline.

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Sir,

Chiasmal apoplexy, an unusual complication of cerebral glioblastoma

The differential diagnosis of the rapid development of bilateral visual failure includes optic glioma.[1,2] and meningeal carcinomatosis.[3] We now describe a case of cerebral glioblastoma complicated by infarction of the optic chiasm.

Case report

A 70-year-old woman with no past medical history of note initially presented to the physicians with symptoms and signs consistent with a left hemispheric cerebrovascular accident. A right carotid bruit was present, her blood pressure was elevated (200/90 mmHg) and she was commenced on metoprolol and aspirin. At this time her visual functions were intact.

Ten weeks later, over a 12 h period she developed profound bilateral visual loss which was associated with an impairment of her short-term memory. A computed tomographic (CT) head scan was performed, the appearance of which was consistent with a cerebral glioblastoma arising in the left parietal lobe.

On examination the following day, she was normally oriented and had minimal weakness of the right arm and leg. There was no perception of light bilaterally, the pupils were equal and unresponsive, and the ocular movements were full. The remainder of the ocular examination was normal; in particular, the optic discs were neither swollen nor atrophic.

As the visual loss was bilateral, symmetrical and pupil-involving, the lesion was localised to the optic chiasm. The rapidity of the visual deterioration suggested the underlying cause was acute vascular insufficiency. The patient had no symptoms suggestive of giant cell arteritis and the ESR was 41 mm/h. The magnetic resonance (MR) image is shown in Fig. 1.

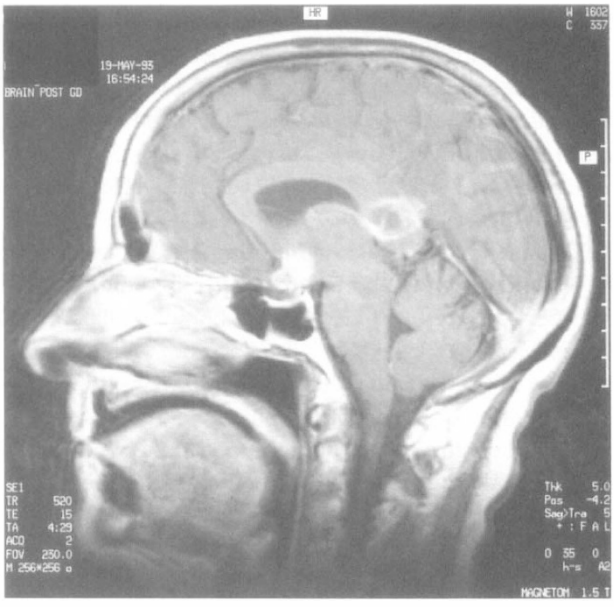

Fig. 1. *Sagittal, midline, T1-weighted, post-gadolinium MR scan demonstrating an enhancing cystic mass in the splenium of the corpus callosum. A separate enhancing focus arises from the floor of the third ventricle and assumes the configuration of the chiasmatic and infundibular recesses. Within this focus lies an area of low-density signal that is iso-intense with that of brain and is consistent with infarction of the optic chiasm. The pre-contrast images (not shown) demonstrate the presence of perichiasmal haemorrhage.*

A stereotactic biopsy from the left parietal lesion confirmed the tumour to be a glioblastoma multiforme (grade 4 astrocytoma). Two days after the biopsy the patient developed raised intracranial pressure and died. A request for a post-mortem was refused.

Comment

The visual pathways can be affected by gliomas arising either as a primary event (optic gliomas) or as a result of spread from a cerebral glioma, as in this case.

Optic chiasmal gliomas characteristically present within the first two decades of life with slowly progressive bilateral visual loss. Acute haemorrhage within the tumour can produce precipitous visual loss that can affect one or both eyes and which has been termed 'chiasmal apoplexy'.[1] Optic gliomas of adulthood present with progressive visual loss, neurological symptoms and death. The pattern of visual loss in those patients in whom the tumour originates intracranially is bilateral, simultaneous and is associated with normal-appearing or pale optic discs.[2]

Visual loss, which occurs in up to one-third of patients with meningeal carcinomatosis, usually progresses over the course of a few weeks; however, complete loss of vision may occur within 48 h.[3]

Visual symptoms are rarely one of the dominant clinical features of cerebral glioblastoma; even after radiotherapy and chemotherapy less than 10% of patients survive 2 years. Progressive bilateral visual loss from diffuse infiltration of the anterior visual pathways can be the presenting symptom in patients with gliomatosis cerebri.[4]

Given the extensive nature of the left parietal lobe tumour demonstrated on the CT and MR scans it seems unlikely that the optic chiasm was the primary site of origin of the glioblastoma. Whilst tumour continuity could not be confirmed radiologically, between 5% and 20% of all cerebral gliomas are multicentric. The cerebrospinal (CSF) fluid was not examined in our patient; however, meningeal gliomatosis occurs in up to 20% of supratentorial gliomas. As a rule, these metastases enlarge within the cavity of the ventricle and seldom penetrate deeply into subjacent tissue. It therefore seems unlikely that the perichiasmal glioma demonstrated in Fig. 1 represents evidence of leptomeningeal spread.

In summary, the rapidity of the visual loss experienced by our patient represents acute vascular insufficiency of the optic chiasm. This most likely reflects external compression of the chiasm and its vascular supply by acute haemorrhage occurring within the perichiasmal glioma.

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