Post-traumatic malignant glioma. Report of a case

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Only a few cases reported in the literature fulfill the currently established criteria for accepting the traumatic origin of some intracranial tumors. A case of post-traumatic glioma is presented. Several years after sustaining a commotio left parietal trauma, our patient developed symptoms of intracranial tumor. Neuroimaging (CT and MRI) showed a large neoplasia in the left tempo-parietal-occipital region, and stereotactic biopsy revealed a mixed glioma in continuity with the scar resulting from the trauma.

Key Words: Brain tumor — Head injury — Oligodendroglioma.

Introduction

The role of head trauma in stimulating neoplastic growth is debated. A trauma may act as a co-carcinogenic factor in evoking cerebral tumoral growth, although very rarely. Zulch (1953) and Manueilidis (1972) elaborated some definite criteria for the acceptance of a causal relationship between head trauma and brain tumors, but only a few of the cases reported in the literature fulfill them. We here describe a case of a mixed glioma arising on the site of a previous head injury.

Case report

Nine years before admission, a 47-year-old Caucasian man was hit by a metal pole and suffered a severe left parietal trauma with contusion of the scalp. He lost consciousness for several hours, complained of headaches for some weeks, but eventually made a complete recovery.

Five years later, he had a first generalized convulsive seizure with a focal motor onset, starting with convulsions of the right extremities; after a further two years, he had a second similar seizure.

Upon admission to our department, neurological examination showed mild right hemiparesis prevalent in the lower limb, minimal right hemisensory loss and right homonimus hemianopsia. Aphasia, dyslexia and dysgraphia were also present. X-ray films of the skull disclosed a left parietal mottled calcification. A CT scan showed a non-enhancing mass lesion, with large calcified areas surrounded by a brain edema, in the left parietal-occipital region (Fig. 1A). MRI study confirmed the presence of a large intra-axial tumor with heterogeneous signal modifications extending to within the splenium of the corpus callosum. The lesion involved the tempo-occipital cortex, the subcortical and deep white matter as well as the posterior aspect of the capsules (Fig. 1B).

The diagnostic procedure was a stereotactic needle biopsy of the lesion; an open procedure with a biopsy of the meninges, cortex and white matter would have been preferable, but this was avoided in view of the probable involvement of the eloquent cortex.

The patient was judged inoperable because of the extent of the lesion. He received radiotherapy with partial benefit (65 Gy of interstitial brachytherapy followed by 40 Gy of external beam irradiation).

Materials and methods

The stereotactic biopsy was performed using a Talairach stereotactic instrument along two orthogonal tracks in order to explore the tumor as well as the surrounding brain tissues, with specimens of 5-10 mm being serially taken. The samples, consisting of small cylinders 5-10 mm in length and 1 mm in width, were fixed in 10% formalin, embedded in paraffin, cut into 4-micron sections and finally stained.

Results

Histopathological examination of the outermost samples revealed a mixed glioma. The samples obtained from the central region of the tumor had neoplastic cells with typical gemistocytic astrocytoma features (Fig. 2A), isolated oligodendrogliomatose elements, with round dark nuclei and clear perinuclear cytoplasm, were often observed (Fig. 2B).

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Fig. 1. Neuroimaging findings. 1a. CT scan. Non-enhancing mass lesion in the left temporo-parieto-occipital region. 1b. MRI study. Large lesion with heterogeneous signal modifications involving the temporo-occipital cortex, the subcortical and deep white matter.

Fig. 2. Histopathologic findings of the samples obtained by stereotactic biopsy. 2a. Typical gemistocytic astrocytoma adjacent to the scar area (H.E., 250X). 2b. Oligodendrogliomatose elements in the vicinity of the scar area (H.E., 125X). 2c, 2d. Scar tissue: loss of neurons, marked astroglial reaction, focal calcium deposits (2c: H.E. 25X; 2d: 62.5X).