A rare case of glioblastoma multiforme

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Abstract

A male, 39 years, enters rapidly in coma, is admitted in the Neurological Hospital Brasov and deceases after 4 hrs in hospital. At necropsy, macroscopic appearances of the cut surface showing unique tumour mass in all brain from frontal lobe to cerebellum, with extensive cortical invasion. The tumor has a variable coloration, a yellowish central necrosis and cystis containing turbid fluid. Histopathological aspect is extremely variable with regional heterogeneity: striking cellular pleomorphism with a lack of cell processes, anaplastic glial cells, and some multinucleated giant cells, large necrotic areas with pseudopalisading of surrounding tumor cells and microvascular proliferation, as glomeruloid tufts.

Keywords: glioblastoma multiforme, diagnosis.

Introduction

The glioblastoma is the least differentiated, most malignant neoplasm of astrocitic origin which typically affects adults [1, 3] and is preferentially located in the cerebral hemispheres.

Glioblastomas frequently develop from low-grade or anaplastic astrocytomas ("secondary glioblastoma"), but could also arising *de novo* after a short clinical history (less than 3 months) without evidence of a less malignant precursor lesion ("primary glioblastoma").

In the WHO classification [8], the term glioblastoma is used with the understanding that this is synonymous with glioblastoma multiforme.

The introduction of GFAP immunohistochemistry confirmed it's origin from differentiated astrocytes or from precursor cells committed to astrocytic differentiation that are present in the adult nervous system.

The molecular genetic studies have demonstrated that loss of portion of chromosomes 10 and 17 and amplification of the epidermal growth factor receptor (EGFR) gene are the most frequent genetic alterations in glioblastoma [4-6]. The main objective of this paper is to present a rare case of glioblastoma, especially by macroscopical appearance.

Material and methods

A 39 year man entered rapidly in coma and was admitted in the Neurological Hospital from Braşov where he died 4 hours after admission.

The brain was obtained from necropsy, 10 hours after death and was fixed in a 10% formalin solution for 3 weeks.

After fixation, sagital sections of 2 cm thickness were made through the brain and 15 specimens from different parts of the tumour were processed into paraffin blocks using standard histological procedures.

Sections of 5 μm were stained with Haematoxylin-Eosin, van Gieson's Tricrom, Periodic Acid Schiff, Gömöri's method for reticulin and Orcein for elastin. GFAP expression was determinate by immuno-histochemistry.

The light-microscopically examination was made at Nikon microscope.

Results and discussions

Macroscopically, with exception of cerebral cortex, the tumour exists in the whole brain, from the frontal lobe to cerebellum and extends into basal ganglia (Figures 1-4).

The tumour is poorly defined and the cut surface of the sagital sections are showing a variable coloration with peripheral grayish aspect and yellowish necrosis in center that occupy 80% of the total tumour mass. There are present some macroscopic cysts that contain a turbid liquefied necrotic tumour tissue.

The histopathology of this tumour is extremely variable. In correspondence with macroscopical aspect, there is a prominent necrosis [2].

The tumour has pleomorphic cells as lipidized (Figure 5), granular and multinucleated giant cells (Figures 6 and 7).

Cellular pleomorphism includes the formation of small undifferentiated, fusiform, round or pleomorphic cells and differentiated neoplastic astrocysts with a lack of cell processes.

There are also lipidized cells with a foamy citoplasm and granular cells. Multinucleated giant cells are typical for glioblastoma multiforme but are few in comparison with giant-cell glioblastoma.

In addition, there are often areas with fusiform cells (Figure 8).

The presence of spindle cells does not justify the diagnosis of gliosarcoma [7].

Invasive spread is particularly fast along myelinated pathways (corpus callosum, internal capsule *et al.*) and as an adaptation of tumour cells to these pathways, they often acquire fusiform shape.

We observed that some lesions show a high degree

of cellular and nuclear pleomorphism and other are highly cellular but rather monotonous, with variable degree of GFAP expression.

There are proliferating blood vessels (Figure 9) and perivascular lymphocytes (Figure 10).

Conclusions

We consider that this tumor is primary glioblastoma multiforme arising de novo after a short clinical history.

Microscopic aspects are specific for glioblastoma multiforme but macroscopical appearances are rare in our cases.

References

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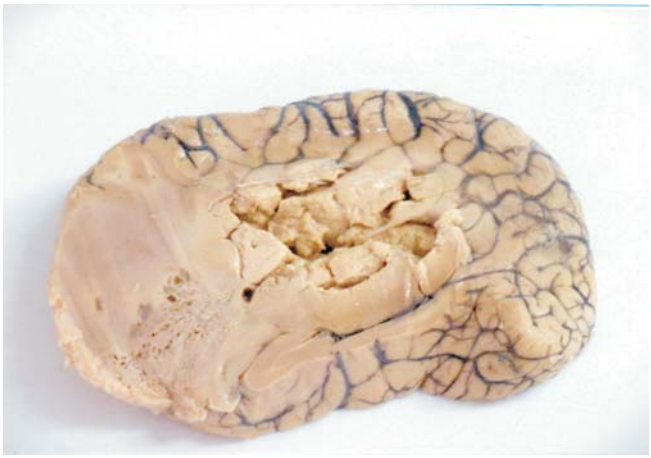

Figure 1 – ***Sagital section of the right cerebral hemisphere***

Figure 2 – ***Sagital section of the left cerebral hemisphere***

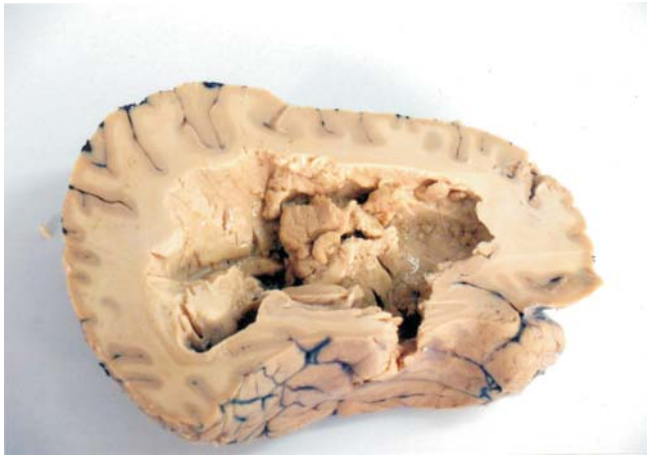

Figure 3 – ***Sagital section of the right cerebral hemisphere***

Figure 4 – ***Sagital medial section of the right cerebral hemisphere***

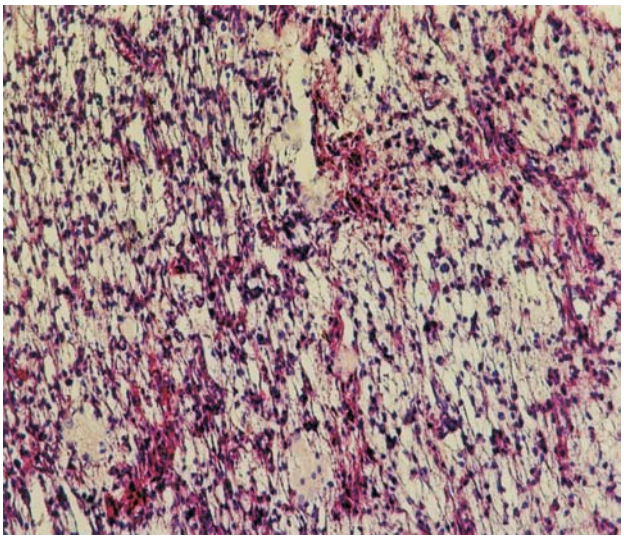

Figure 5 – ***Glioblastoma multiforme: area with lipidized cells (HE, ob. ×10)***

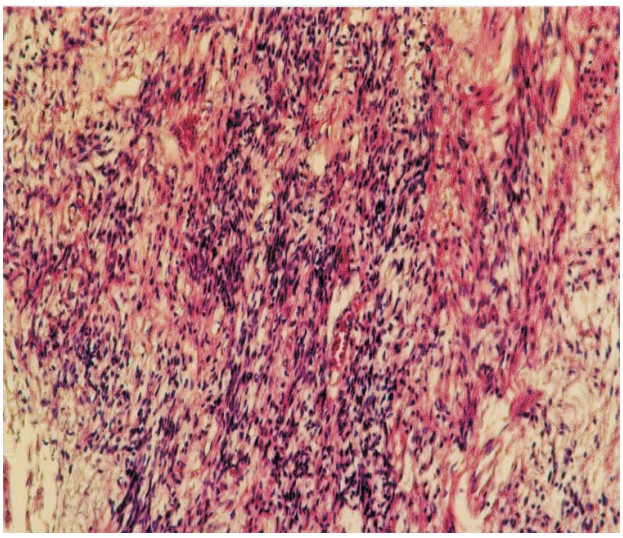

Figure 6 – ***Glioblastoma multiforme: area with pleomorphic cells (HE, ob. ×10)***

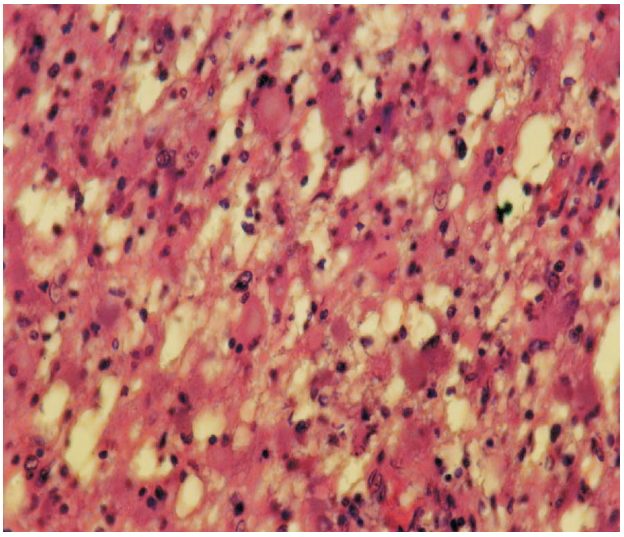

Figure 7 – ***Glioblastoma multiforme: area with multinucleated giant cells (HE, ob. ×20)***

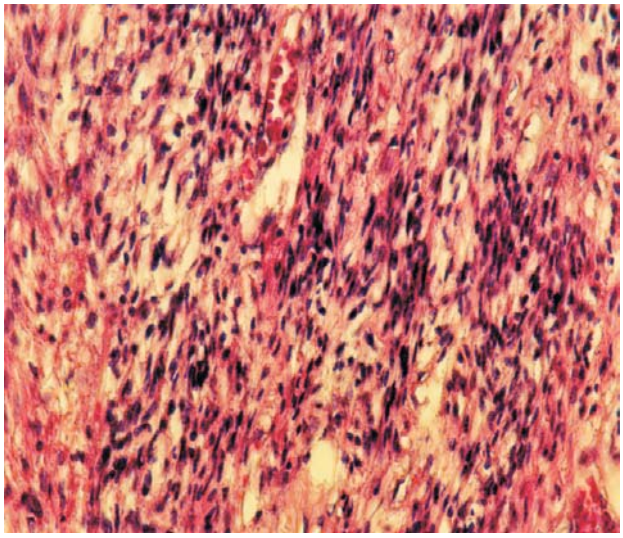

Figure 8 – ***Glioblastoma multiforme: area with fusiforme cells (HE, ob. ×20)***

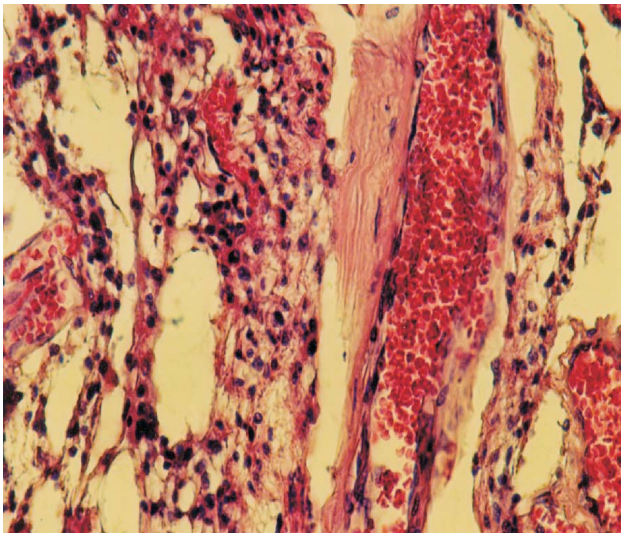

Figure 9 – ***Glioblastoma multiforme: area with proliferative blood vessels (HE, ob. ×20)***

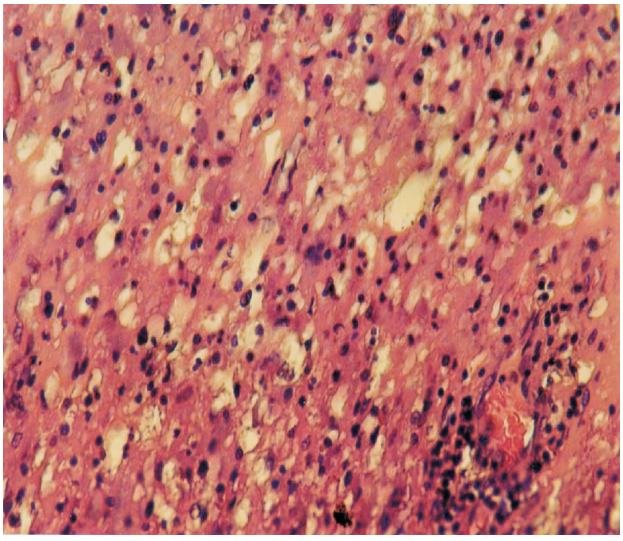

Figure 10 – ***Glioblastoma multiforme: area with perivascular lymfocytes (HE, ob. ×20)***