

ULTRASTRUCTURAL STUDY OF HUMAN PERITUMORAL CEREBRAL CORTEX AND WHITE MATTER BY TRANSMISSION ELECTRON MICROSCOPY.

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The cellular pathology of peritumoral cerebral tissue remains poorly defined (Goel et al.; 2003). To describe the ultrastructural cellular pathology of human cerebral peritumoral tissue and establish a possible link with the clinical features. Sixteen biopsies of human cerebral peritumoral tissue were conventionally processed for transmission electron microscopy. Clinical features and post-surgical treatment follow-up were analyzed. In both, neurons and macroglia processes exhibits edematous and hypoxic/ischemic cellular changes of variable severity (Fig. 1). Extracellular space dilatation was not observed. Necrotic zones in the white matter were observed. Dendrites and axons showed marked cytoskeletal fragmentation and disintegration (Fig. 2-4). Synaptic plasticity and synaptic degeneration were characterized (Fig. 5-6). Both, astrocytes and perivascular end-foot showed scarce or absent glycogen granules. Inter-endothelial junctions of capillaries exhibited elongate and curved shapes without expansion of paracellular space. Basal membrane showed reduplication and focal vacuolization. The ultrastructural changes were correlated with severity of clinical features and neurological deficit of patients under study. Ultrastructural pathology is compatible with the instauration of hypoxic/ischemic environment in the peritumoral cerebral tissue, with predominance of cellular edema type (Pennings et al.; 2003; Uematsu et al.; 2003). Diverse origin of specimen locations and tumor types represent significant factors to explain the differences between ours findings and those reported by others investigations (Castejón 2003; Castejón and Castejón 2004; Fiala et al., 2002; Semchenko et al., 1984; Spacek 1987). Ultrastructural pathologic alterations of neurons and their processes were possibly responsible for the clinical symptoms and neurological deficit origin. The findings of this study support the hypothesis that peritumoral cerebral edema is of cellular type due to oxygenation decrease in peritumoral tissue (Pennings et al.; 2003), and were linked with functional alterations, degenerative changes and with possibly neuronal death that finally cause neurological deficit.

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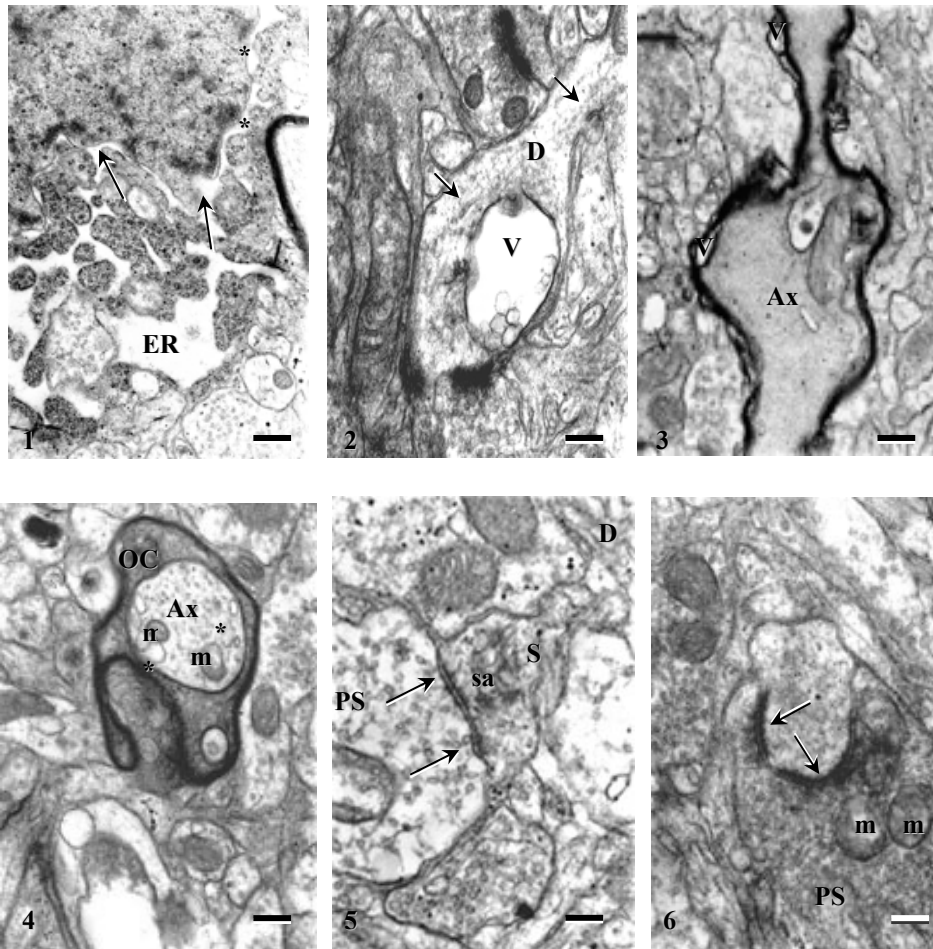


Fig. 1. Non-Pyramidal neuron with dilated peri-nuclear cistern (asterisks), focal rupture of nuclear membrane (arrows). Cytoplasm exhibits electron-dense feature, with multiples free-ribosomes, and endoplasmic-rough-reticula (ER) dilated severely. Bar: 0,125 μ m.

Fig. 2. Edematous dendritic process (D) with huge vacuole (V), few fragmented microtubules (arrows). Bar: 0,2 μ m.

Fig. 3. Beaded shape myelinated axon (Ax), with focal vacuolization (V) in myelin sheet and granular dense aspect of axoplasm. Bar: 0,3 μ m.

Fig. 4. Transversally sectioned edematous myelinated axon (Ax), shows two dense degenerated mitochondria (m) and, several endocytic vesicles (asterisks). Note the electron-dense oligodendroglial cytoplasm (OC). Bar: 0,2 μ m.

Fig. 5. Dendritic spine (S) that exhibit a degenerated spinous apparatus (sa). Note the perforated synapses (arrows) and edematous pre-synaptic ending (PS). D labels the dendritic shaft. Bar: 0,125 μ m.

Fig. 6. Convex and perforated synapse (arrows), the pre-synaptic ending (PS) exhibits two dense mitochondria (m) and numerous synaptic vesicles. Bar: 0,166 μ m.