Neurobiology

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Derangement of nerve cell membranes in traumatic human brain edema.

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In severe traumatic human brain injuries complicated with subdural hematoma the non-pyramidal neurons, reactive and hypertrophic astrocytes and hydropic oligodendrocytes showed plasma membrane fragmentation, enlargement of rough and smooth endoplasmic reticulum cisterns, extensive areas with detachment of membrane-bound associated ribosomes, and a marked reduction in the number of polysomes The plasma membrane forms endocytic and clathrin coated vesicles internalizing toward the cytoplasm. The nuclear envelope appears irregularly dilated. Areas of focal necrosis of plasma membrane, cytomembranes, outer nuclear membrane, smooth membranes of Golgi endoplasmic sacs, and limiting membrane of lysosomes are observed. At the level of neuropil large extracellular spaces are found with presence of proteinaceous edema fluid (Figures 1 and 2).The degenerated myelinated axons show invaginations of axolemmal membrane and formation of endocytic vesicles. The myelin membranes appear separated forming large intraperiod vacuoles. (Figure.3). [1]

Synaptic vesicle exocytosis at the synaptic active zones, and endocytosis at the non specialized regions of presynaptic ending limiting membrane are frequently observed at sensitized and degenerative synapses. Synaptic disassembly occurs featured by wide separation of pre- and post synaptic membranes and detachment of perisynaptic astrocytic glial ensheathment (Figure 4). [2]

Astrocytic gap junction displayed wide separation, disruption and disassembly. The endothelial cell luminal membrane of brain capillaries exhibits profound activity changes that characterize the increased cerebrovascular permeability, such as increased formation of micro- and macropinocytotic vesicles and clathrin-coated vesicles, deep invaginations and formation of incomplete transendothelial channels, and emission of pseudopods to form endothelial vacuoles.

The role of free radical and lipid peroxidation, disturbed energy metabolism, altered metabolic cascades, glutamate excitotoxicity, hemoglobin toxicity, protein aggregation, and presence of proteinaceous and non-proteinaceous extracellular edema fluid are discussed in relation with the derangement of nerve cells membranes.

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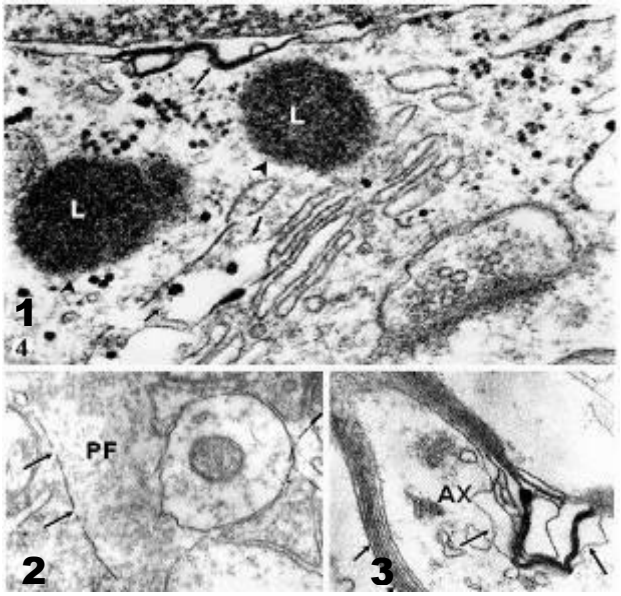

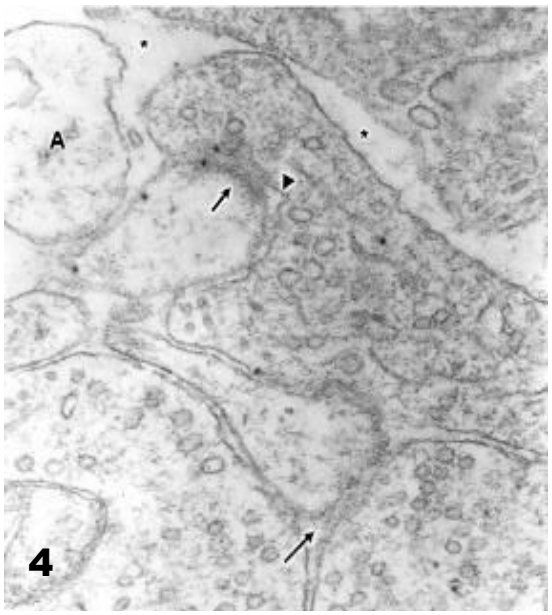

Figure 1. Brain trauma. Subdural hematoma. Right parietal cortex. Non-pyramidal neuron showing focal necrosis of nuclear envelope (long arrow) and of smooth Golgi complex membranes (short arrows). The lysosomes (L) show a discontinuous globular limiting membrane (arrowheads). X 75.000.

Figure 2. Severe frontal contusion. Left frontal cortex. Non-pyramidal neuron showing a high electron dense and fragmented plasma membrane (arrows) in contact with the proteinaceous edema fluid (PF) occupying the enlarged extracellular space. X 60.000.

Figure 3. Brain trauma. Right parieto-tempotal hematoma. Right parietal cortex. Severely edematous neuropil showing a degenerated myelinated axon (AX). The myelin sheath lamellar arrangement appears disrupted and forming intramyelinic vacuoles (long arrows). Note the formation of neighboring axolemmal endocytic vesicles, and the apparently normal and compact arrangement of a segment of myelin sheath at the opposite side (short arrows). X 60.000

Figure 4. Brain trauma. Subdural hematoma. Left parietal cortex. Severely edematous neuropil showing synaptic disassembly and disappearance of pre- and postsynaptic densities (long arrow) of an axospinodendritic contact. The short arrow labels another spine synapse exhibiting partial vestiges of the postsynaptic density, and irregular profile of presynaptic membrane (arrowhead). Note the expansion of extracellular space (asterisks), and the detachment of perisynaptic astrocytic cytoplasm (A). X. 60.000.