Cellular enviroment

Health & Medicine, Nursing

Cellular Environment due: Pathophysiological explanation of cellular swelling during the early stages of

cell injury

Cell injury is the domino effect of stressing cells severely, exceeding their capacity for physiological adaptation. This may take place when the stimulus is excessive. Stimulus includes genetic (gene defects), nutritional (iron, vitamins), immune (autoimmunity), endocrine, physical agents, chemical agents, infective, and ischemia (hypoxia). According to Brashers (2006), Cell injury also is a consequence of exposing the cells the cells to inherently damaging agents, or when the cells experience intrinsic abnormalities. Adaptation capacity and sensitivity to various types of injury range in accordance to the cell type, duration, and severity (p. 16-17).

According to McCance & Huether (2010), cellular swelling is the initial sign of cell injury; triggered by direct damage to the cell membrane, hypoxia, or damage to the electrolyte-pumping enzymes. The most common cause of cellular injury is hypoxia, which disrupts the cell’s capability of maintaining fluid homeostasis due to deficiency in oxygen. The main cellular mechanisms of cell injury include loss of calcium homeostasis, ATP depletion, oxidative stress, damage to mitochondria, and increases permeability of membranes. These adaptive mechanisms thus fail to maintain normal homeostasis, leading to a fall in oxidative phosphorylation. This triggers the depletion of cellular ATP and the swelling of mitochondria. What follows next is the failure of membrane calcium pumps, protein synthesis, NaK ATPase pump, and generation of reactive oxygen metabolites. This leads to an influx of Na+ and water, efflux of K+, entry of free calcium into the cytoplasm, and a drop in

intracellular pH. This results to accumulation of water into the cell triggered by failure of membrane NaK ATPase pump, and cellular swelling due to swelling of endoplasmic reticulum (p. 54-92).

Cellular swelling during the early stages is a reversible lesion, which is non-lethal; however, it becomes reversible if hypoxia persists for some time depending on the oxygen demands of the cell. An irreversible injury leads to cell death, mainly via apoptosis or necrosis.

Pathophysiological explanation of aging as it pertains to cellular structure

Aging is a gradual deterioration of a mature organism leading to irreversible structural changes as time passes by. According to Brashers (2006), cellular changes facets of aging include hypertrophy, and the weakened capability to undergo mitosis. Cellular function is impaired by the deposition of lipids (lipid peroxidation products), programmed changes in gene expression, damage from free radicals and advanced glycation end products. These intertwined processes induce apoptosis. Every cell is planned for a definite number of cell divisions, at the ending of the specified time, proliferation stops. The cell enters a quiescent state, which it goes through cell death via the procedure of apoptosis. Apoptosis is regulated cell death, a process responsible for worn out cells, induced by oxygen free radicals. Apoptosis is characterized by membrane blebbing, cell shrinkage, and chromatin condensation (p. 9, 309-313).

Concerning cellular structure, mitochondria plays an essential role in the aging process, as they are the main source of reactive oxygen species and free radicals; and, in addition, acts as the chief target for reactive oxygen species and free radical damage within the cell. The free radical theory of

aging studied by Harman et al (1952) revealed that oxygen free radicals are the cause of cellular damage that leads to aging, and age related pathologies. Linnane et al (1989) later polished up this theory in the mitochondrial theory of aging; where the buildup of somatic mutations in mitochondrial DNA was regarded as the chief contributor to the process of aging. It is evident that during aging, there is a decline in mitochondrial respiratory function, an elevation in reactive oxygen species production, a rise in the resulting damage to cellular constituents from increased reactive oxygen species production, enhanced mutations to mitochondrial DNA, and enhanced apoptosis (McCance & Huether, 2010). Thus, other than acting as energy producers for the cell, the mitochondria are key mediators in the initiation and regulation of apoptotic signaling (p. 91-93).

Concept map demonstrating the pathophysiology of hypoxia and cellular injury

Enzymes responsible for maintain osmotic balance of the cell lack function; as their ability has been disrupted.

Hypoxia continues for sometime

Severe swelling due to prolonged ischemia

References

Apoptosis p. 9, 309-313 and cell injury p. 16-17 in Brashers, V. L. (2006). Clinical applications of pathophysiology: An evidence-based approach. St. Louis (MO: Mosby/Elsevier.

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