PATHOPHYSIOLOGY- BP204T

UNIT : 1 Morphology of cell injury

CLASS:1

Atrophy:

When confronted with a decrease in work demands or adverse environmental conditions, most cells are able to revert to a smaller size and a lower and more efficient level of functioning that is compatible with survival. This decrease in cell size is called atrophy. Cells that are atrophied reduce their oxygen consumption and other cellular functions by decreasing the number and size of their organelles and other structures. When a sufficient number of cells are involved, the entire tissue or muscle atrophies. The general causes of atrophy are: decreased workload, loss of innervation, diminished blood supply (ischemia), inadequate nutrition, loss of endocrine stimulation and aging. Atrophy is a normal physiologic process of aging in some tissues which could be due to loss of endocrine stimulation or arteriosclerosis. For example, atrophy of lymphoid tissue in thymus and lymphatic nodes. Others stimuli are clearly pathologic. The decreased workload upon the skeletal muscles results in disuse atrophy. An extreme example of it may be seen in the muscles of extremities that have been encased in plaster casts. Because atrophy is adaptive and reversible, muscle size is restored after the cast is removed. Denervation atrophy occurs in the muscles of paralyzed limbs. Ischemic atrophy occurs when there is gradual diminishment of blood supply due to atherosclerosis; it may result in shrinkage of affected organ (e.g. atrophy of brain in cerebral atherosclerosis). In the case of inadequate nutrition or even starvation cells decrease their size and energy requirements as a means of survival. Loss of endocrine regulatory mechanisms results in reduced metabolic activity of the tissue and hence its atrophy. For example, in women, the loss of estrogen stimulation during menopause results in atrophic changes in the reproductive organs.

Hypertrophy

refers to an increase in the size of cells and, with such change, an increase in the size of the organ. It is usually seen in muscles because this type of tissue is not capable of mitotic activity. Hypertrophy can be caused by increased functional demand or by specific hormonal stimulation and may occur under both physiologic and pathologic conditions. The increase in muscle mass associated with exercise is an example of physiologic hypertrophy. Another example of physiologic hypertrophy affected by hormonal stimulation is enlarged size of the uterus in pregnancy. The cellular hypertrophy is stimulated by estrogen through smooth muscle estrogen receptors. Pathologic hypertrophy occurs as the result of disease conditions and may be adaptive or compensatory. Example of adaptive hypertrophy is the myocardial hypertrophy that results from valvular heart disease or hypertension. Compensatory hypertrophy is the enlargement of a remaining organ or tissue after a portion has been surgically removed or rendered inactive. For instance, if one kidney is removed, the remaining kidney enlarges to compensate for the loss

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Hyperplasia

is an increase in the number of cells of a tissue or organ. It occurs in tissues where cells are capable of mitotic division such as the epidermis, intestinal epithelium, and glandular tissue. Hyperplasia is a controlled response to an appropriate stimulus and ceases once the stimulus has been removed. The stimuli that induce hyperplasia may be physiologic or pathologic. There are two common types of physiologic hyperplasia: hormonal and compensatory. Breast and uterine enlargement during pregnancy are examples of a physiologic hyperplasia that results from estrogen stimulation. The regeneration of the liver that occurs after partial hepatectomy (i.e., partial removal of the liver) is an example of compensatory hyperplasia. Hyperplasia is also an important response of connective tissue in wound healing, during which proliferating fibroblasts and blood vessels contribute to wound repair. Most forms of pathologic hyperplasia are due to excessive hormonal stimulation or the effects of growth factors on target tissues. Excessive estrogen production can cause endometrial hyperplasia and abnormal menstrual bleeding.

Metaplasia

is a reversible change in which one adult cell type (epithelial or mesenchymal) is replaced by another adult cell type. Metaplasia is thought to involve the reprogramming of undifferentiated stem cells that are present in the tissue undergoing the metaplastic changes in response to chronic irritation and inflammation. Metaplasia is seen in the respiratory tract in the habitual cigarette smoker. The normal columnar ciliated epithelial cells of the trachea and bronchi are replaced by stratified squamous epithelial cells. Stones in the excretory ducts of the salivary glands, pancreas, or bile ducts may cause replacement of the normal secretory columnar epithelium by nonfunctioning stratified squamous epithelium. Squamous epithelium is more able to survive under circumstances in which the more fragile specialized epithelium most likely would have succumbed.

Dysplasia

is characterized by deranged cell growth of a specific tissue that results in cells that vary in size, shape, and organization. Minor degrees of dysplasia are associated with chronic irritation or

inflammation. Although dysplasia is abnormal, it is adaptive in that it is potentially reversible after the irritating cause has been removed. Dysplasia is strongly implicated as a precursor of cancer. In cancers of the respiratory tract and the uterine cervix, dysplastic changes have been found adjacent to the foci of cancerous transformation. However, dysplasia does not necessarily lead to cancer.

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