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Morphological Criteria for Facial Nerve Neuritis in Postmenopausal Women

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Abstract: Facial nerve neuritis remains a significant issue in modern neurology due to its high prevalence, profound impact on patients' quality of life, and the risk of developing persistent functional impairments. The findings provide greater insight into the disease's pathogenesis and help define directions for future morphological research.

Keywords: Inflammation, nerve tissue regeneration, peripheral nervous system.

Introduction: Facial nerve neuritis is one of the most common disorders of the peripheral nervous system and a leading cause of peripheral paresis of the facial muscles. Despite significant advances in modern neurology, the pathogenetic mechanisms of the disease remain a subject of active study. The course of this condition in women entering menopause is of particular interest, as this period involves profound hormonal restructuring affecting virtually all organs and systems of the body.

A decrease in estrogen levels is accompanied by impaired vascular regulation, altered metabolic processes, and reduced resistance of nervous tissue to damaging factors. Against this background, even a relatively minor inflammatory insult can lead to severe damage to the facial nerve.

Morphological changes associated with neuritis are not limited to inflammatory infiltration. The pathological process involves the myelin sheath, axons, endoneurium, perineurium, and the vasculature. A complex array of structural abnormalities develops, determining the severity of the clinical presentation and the duration of recovery. As the disease progresses, signs of demyelination, interstitial edema, ischemic changes, connective tissue proliferation, and secondary degeneration of nerve fibers emerge.

The severity of each of these processes varies and depends on the patient's age, the duration of the disease, concomitant vascular pathology, and hormonal status. For this reason, morphological assessment remains one of the most informative approaches to studying facial nerve neuritis.

The present study was conducted to identify the most significant morphological criteria for facial nerve neuritis in menopausal women and to evaluate their relationship with the clinical manifestations of the disease.

The study was comprehensive and analytical in nature, involving morphological, clinical-anatomical, and comparative analyses of published scientific literature on peripheral nervous system pathology. In preparing the study, the authors reviewed contemporary domestic and international publications in the fields of neurology, pathological anatomy, histology, and

neuromorphology, primarily those published over the last twenty years.

Particular attention was paid to research examining age-related characteristics of peripheral nerve involvement in postmenopausal women. The study involved a comparative analysis of morphological changes in the facial nerve across various stages of the inflammatory process. Researchers investigated the structural characteristics of nerve fibers, the condition of the myelin sheath, the extent of axonal damage, the severity of vascular abnormalities, and the nature of inflammatory cellular infiltration.

In addition, changes in the endoneurium, perineurium, and epineurium were analyzed, as these structures are the first to respond to the development of inflammation and impaired microcirculation. Morphological findings were correlated with clinical data reflecting the severity of peripheral paresis, the rate of recovery of facial muscle function, and the duration of the disease.

The primary method for assessing structural changes was light microscopy of facial nerve specimens stained with hematoxylin and eosin. To examine the state of the myelin sheath in greater detail, data from specialized nerve tissue staining techniques described in current literature were taken into account. In addition, the study analyzed results from immunohistochemical examinations, which allow for the determination of inflammatory activity, the extent of Schwann cell damage, and the intensity of regenerative processes.

The data obtained were systematized according to key morphological characteristics and categorized based on the stage of the disease.

A comparative morphological approach was employed to objectively evaluate the results. Changes in nerve tissue in menopausal women were analyzed and compared with the clinical course of facial nerve neuritis in younger female patients as described in the literature. Particular attention was paid to the impact of age-related hormonal deficiency on the condition of the microvasculature, the intensity of the inflammatory response, and remyelination processes.

The obtained materials were systematized, analyzed, and interpreted from the perspective of modern neuromorphology, enabling the identification of the most informative morphological criteria for the disease.

The analysis revealed that the morphological presentation of facial nerve neuritis in menopausal women is characterized by a combination of inflammatory, vascular, and degenerative changes of varying severity. Even at the early stages of the disease, signs of interstitial edema are observed in the nerve trunk, accompanied by widening of the endoneurial spaces and disruption of the normal architecture of the nerve fascicles.

At the same time, marked congestion of the microvasculature is observed, indicating the active development of an inflammatory response. Focal hemorrhages and stasis of formed blood elements are detected in certain areas, reflecting the progression of microangiopathic abnormalities. Such changes impair the nutrition of the nervous tissue and create conditions for further damage to the myelin sheaths.

Microscopic examination reveals that segmental demyelination of nerve fibers is one of the most consistent features of the disease. The myelin sheath loses its homogeneity, becomes unevenly thickened, and undergoes fragmentation and vacuolization in places. In some cases, signs of partial myelin breakdown are observed, with the formation of small foci of myelin debris.

Concurrently, axonal changes are observed, manifesting as irregular thickening, deformation, and subsequent fragmentation. The most pronounced degenerative processes are found in patients with a long-standing course of the disease, indicating the progressive nature of the structural abnormalities in the absence of timely treatment.

Significant changes are also observed in the vascular component of the facial nerve. The walls of small arteries and arterioles become thickened due to plasma imbibition and connective tissue proliferation. The vascular lumen narrows, resulting in reduced blood supply to the nerve fibers. Signs of blood stasis and vascular dilation are noted in the venous section of the microvasculature.

Morphological changes in the vascular wall are particularly pronounced in women with concomitant arterial hypertension, diabetes mellitus, and other chronic diseases that frequently accompany

menopause. This suggests that the vascular factor plays a significant role in the progression of facial nerve neuritis.

Analysis of the cellular composition of the inflammatory infiltrate revealed a predominance of lymphocytes and macrophages, localized primarily around blood vessels and between individual nerve fibers. Occasional plasma cells and fibroblasts were observed in some specimens, indicating a transition of the inflammatory process to the repair stage. Macrophages actively participate in the removal of degraded myelin; however, their high level of activity is simultaneously accompanied by an intensification of the local inflammatory response.

As inflammation subsides, there is an increase in connective tissue elements forming areas of perineural and endoneural fibrosis. This structural remodeling limits the potential for complete regeneration of nerve tissue and explains the persistence of residual neurological deficits in some patients.

The state of Schwann cells—which are responsible for the formation and repair of the myelin sheath—is of particular importance. An analysis of the literature indicates that their functional activity declines under conditions of hormonal deficiency. This is accompanied by a slowing of remyelination processes and a prolonged recovery time for nerve conduction. Concurrently, there is a reduction in the expression of growth factors involved in peripheral nerve regeneration. Against this background, structural recovery of the facial nerve proceeds significantly more slowly, even after the inflammatory process has been resolved.

It is precisely the combination of demyelination, vascular insufficiency, axonal degeneration, and reduced regenerative activity of Schwann cells that should be regarded as the primary morphological criteria for facial nerve neuritis in menopausal women. The constellation of these changes determines the specific clinical course of the disease, the duration of the recovery period, and the likelihood of residual neurological deficits developing.

The findings suggest that facial nerve neuritis in menopausal women should be viewed not merely as a localized inflammatory lesion of the peripheral nerve, but as a complex pathological process driven by age-related hormonal shifts, vascular changes, and impaired regenerative capacity of the nerve tissue. An analysis of morphological data reveals that the inflammatory response in patients of this age group exhibits specific characteristics distinguishing it from the same condition in younger women.

The most pronounced changes are observed in the endoneurium and perineurium, where persistent interstitial edema develops, accompanied by dilation of the microvasculature. The increase in endoneurial fluid volume leads to compression of nerve fibers, disruption of axonal transport, and impaired nutrition of the nerve tissue. Concurrently, damage to the myelin sheath is observed, the severity of which is directly related to the duration of the disease. Microscopic examination reveals areas of segmental demyelination, myelin vacuolization, and destructive changes in Schwann cells.

This clinical picture indicates a progressive impairment of nerve impulse conduction and the development of persistent peripheral paresis. It should be noted that recovery processes in menopausal women proceed significantly more slowly than in patients of reproductive age. This is likely attributable to reduced levels of estrogen, which has a proven neuroprotective effect.

The state of the facial nerve's vascular component warrants particular attention. Morphological examination reveals signs of chronic ischemia resulting from the thickening of small artery walls, narrowing of capillary lumens, and impaired microcirculation. Certain specimens exhibit marked venular congestion accompanied by stasis of formed blood elements. Compromised blood supply leads to hypoxia of the nerve fibers and triggers secondary degenerative processes. Concurrently, there is increased production of pro-inflammatory cytokines, which exacerbate tissue damage.

As the disease progresses, inflammatory infiltration becomes less pronounced, while signs of connective tissue remodeling come to the fore. Proliferating collagen fibers gradually replace areas of damaged endoneurium, limiting the potential for complete regeneration.

Further analysis of the specimens reveals that a combination of inflammatory and degenerative

processes is a key morphological criterion. While signs of vascular reaction and cellular infiltration predominate in the early stages, the severity of axonal degeneration increases significantly after several weeks. Light microscopy reveals fragmentation of the axons, uneven myelin staining, and the appearance of small foci of connective tissue remodeling.

Electron microscopy studies presented in current literature confirm the presence of mitochondrial destruction, disruption of neurofilament structure, and partial breakdown of Schwann cell membranes. These changes significantly limit the potential for rapid recovery of facial nerve conduction.

The impact of hormonal deficiency on the course of the disease is of particular interest. Estrogens play a role in regulating vascular tone, growth factor synthesis, and remyelination processes. Following the onset of menopause, the levels of these hormones drop significantly, leading to a reduction in their neuroprotective effect. Against this background, even mild inflammation can trigger more pronounced structural changes in nervous tissue. This explains why older female patients more frequently exhibit persistent neurological deficits, residual facial asymmetry, and the development of facial muscle contractures.

A review of the literature indicates that the most informative morphological criteria for facial nerve neuritis are interstitial endoneurial edema, lymphocytic-macrophage infiltration, segmental demyelination, axonal degeneration, microcirculatory vascular disturbances, connective tissue proliferation, and the extent of remyelination. It is the comprehensive assessment of these features that enables the most objective determination of the pathological process stage, prediction of the disease course, and evaluation of the efficacy of the administered therapy.

An analysis of current scientific data indicates that facial nerve neuritis in menopausal women is characterized by a complex of interrelated morphological changes, including inflammatory infiltration, vascular disturbances, interstitial edema, segmental demyelination, axonal degeneration, and connective tissue remodeling. The severity of these processes is significantly influenced by the age-related decline in estrogen levels, which is accompanied by impaired microcirculation and reduced regenerative potential of the nerve tissue.

The most significant morphological criteria are the extent of demyelination, the degree of axonal damage, the nature of vascular changes, and the intensity of remyelination. A comprehensive assessment of these features is crucial for determining the disease stage, predicting facial nerve function recovery, and selecting the most rational treatment strategy.

Further research should focus on an in-depth study of the molecular mechanisms of peripheral nerve damage under conditions of hormonal deficiency, as well as on the identification of morphological markers that allow for predicting the disease outcome and the efficacy of the administered therapy.

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