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Peripheral nervous system damage in hypothyroidism: current view on the problem (literature review)

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Abstract. *The article presents the pathogenesis of polyneuropathy symptoms in patients with hypothyroidism, whereby the approaches to the pathogenetic therapy are substantiated. The article represents the most important aspects of hypothyroid polyneuropathy treatment according to the latest international guidelines and perspective scientific approaches to optimize the medical care for patients with this pathology.*
Keywords: *hypothyroidism; polyneuropathy; hypothyroid polyneuropathy*

Nowadays the thyroid pathology is known to be one of the most widely spread ones in the structure of endocrine diseases [2, 15]. Recently the increase of occurrence of autoimmune thyropathies, mostly followed by the development of hypothyroidism has been observed in Ukraine and other countries. At the same time, the number of surgeries for nodular forms of goiter, tumors, etc., frequently resulting in hypothyroidism, growths as well. The results of epidemiological studies show, that the overall prevalence of manifested hypothyroidism in the population is 0.2–2 %, subclinical one — 7–10 % among women and 2.3 % among men, and morbidity rates are continuously rising every year [15]. There are more than 98 hundred people with this disease, officially registered in Ukraine at the beginning of 2016 [16].

At its early stages the disease is accompanied by a wide range of neurological syndromes, which often dominate in the clinical manifestation of the disease and involve practically all levels of the nervous system [7, 11]. However, despite a long history of detection of interconnections between the thyroid and neurological pathologies, the study of damaging mechanisms for the nervous system in hypothyroidism still remains a topical issue of contemporary neuroendocrinology.

Deficiency of thyroid hormones in the body leads to disorders of water and electrolyte balance, protein, lipid, carbohydrate metabolisms, causing morphological-functional and biochemical changes in various organs and systems [2, 15]. Hypothyroidism is accompanied by disturbed synthesis of neurotransmitters, increased levels of blood lipids, lowering the energy potential of the cells, the activation of free radical processes, reduction of synthesis of a nitric oxide and endothelial dysfunction, disturbance of blood microcirculation disorders, imbalance of proinflammatory cytokines and adipocytokines, etc. [18]. A dramatic inhibition of energy and anabolic processes, typical for hypothyroidism, promotes an organic damage of the nervous system [22].

Hypothyroid neurological disorders are various and numerous [14, 15]. Marked changes in the peripheral nervous system, typical for hypothyroidism, are implemented in the development of pseudomyotonic and pseudomyasthenic syndromes, radiculopathies, polyneuritis, tunnel neuropathies, and polyneuropathies as well. The latter are found in 18–72 % of patients with hypothyroidism [7, 11, 22], and symptoms of polyneuropathy can develop not only in manifested hypothyroidism, but also in subclinical one [30]. However, there is no consensus concerning the pathogenesis of hypothyroid polyneuropathy (PNP), correlation between the degree of its manifestation and hormonal status, state of the neuromuscular system during the compensation of the underlying disease. Thus, indicating a direct connection between the level of thyroid hormones, the degree of hypothyroidism compensation and polyneuropathy symptoms, some researchers believe that all clinical, electroneuromyographic and histopathological changes in patients with hypothyroid polyneuropathy are reversible in case an adequate replacement therapy is initiated [25, 28]. However, according to the other studies, clinical and pathomorphological signs of neuromuscular system disorders remain after the compensation of hypothyroidism [24, 27, 29].

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Degenerative, toxic, metabolic, ischemic and mechanical factors, leading to the changes of the connective tissue interstitium, myelin sheath and axial cylinder of the nerve are known to be underlying factors promoting the formation of PNP, particularly hypothyroid one. Hence, the division of polyneuropathy into *axonopathy*, related to the underlying primary damage of the axial cylinders of nerves, and *myelinopathy*, characterized by the disturbance of nerve conduction due to the myelin sheaths loss, is generally accepted. However, during the progression of the disease their combinations usually occur [17].

The development of the PNP in case of hypothyroidism is considered to be related to the mucinous infiltration of the perineurium resulted in nerves compression, as well as to disorders of oxidative processes due to the thyroid hormones insufficiency. Schwann's cells are primarily influenced by the metabolic disorders, and that leads to segmental demyelination. Thus, as morphological studies have demonstrated, glycogen and mucin deposits in Schwann's cells, bulbous thickening of the myelin sheath with mucinous inclusions, segmental demyelination, increased number of demyelinated fibers of a small diameter, reduced number of myelinated fibers of a large diameter are observed in the peripheral nerves [18, 22].

Clinically, hypothyroid PNP is manifested by the pain and paresthesias in the distal parts of the extremities, muscular weakness, seizures, polyneuritic impairment of sensitivity, decrease or loss of tendon reflexes [14, 17]. Such symptoms are amplified to complete immobilization while staying in a cold room or during winter. Severity of PNP clinical manifestation depends on the degree of involvement of motor, sensory and autonomic fibers into the pathological process.

Movement disorders are manifested as muscular weakness, located mainly in the distal areas, mostly in the extensor muscles, accompanied by hypo- or areflexia. In severe cases, patients are unable to stand or walk, hold objects in their hands.

Sensory changes are associated with positive (paresthesia, hyperpathies) and negative symptoms (loss of joint, muscle and tendon proprioception, leading to an imbalance when standing and walking, reduction of skin tactile and pain sensitivity).

Autonomic symptoms appear as sympathalgias, vasomotor, trophic and secretory disorders (burning pain, sweating changes, swelling of the distal areas of the limbs, abnormalities of their color and temperature, sores, muscular changes, nails deformation).

However, the diagnostics of motor, sensory and autonomic symptoms of PNP is generally impeded by multiform clinical manifestations of hypothyroidism and involvement of numerous tissues. Thus, the development of hypothyroid myopathy, characterized by permanent muscular weakness (more substantial in muscles of proximal areas of the limbs), prolonged muscular contraction and their relaxation period, by convulsions, muscular hypertrophy and hardening, myalgia, increased mechanical excitability of muscles during percussion, etc., is associated with the limitation and inhibition of movements, mistakenly considered as paresis or paralysis [1]. Moreover, the boundaries of loss and irritation symptoms, the type of sensory disorders, especially in mixed areas, vary in a wide range due to the variability of overlapping of innervation zones by adjacent nerves and variability of autonomous zones, as well as due to the double, triple innervation of certain muscles and skin areas.

In addition to complete neurological examination and selection of typical neurological symptoms a significant role in the differential diagnosis of the type of hypothyroid peripheral nervous system disorder is played by electroneuromyography (ENMG). During electromyographic examination the decrease in amplitude and slowing of the speed of an impulse transmission by sensory and motor nerves is recorded, the results of ENMG enable to differentiate of the damage of muscle and nerve fibers, detect the degree of nerve fibers' damage, differentiate between axonal (axonopathies) and demyelinating (myelopathy) PNP. Thereby, a slow speed of the transmission of an impulse along the nerve, increased distal latency period, change of F-response, blockage of the transmission and temporary dispersion are usually indicative of the myelin sheath damage, whereas the decrease of impulse level is a sign of axonal degeneration [19].

In doubtful cases nerve biopsy may be very helpful — histological changes in them are absent in case of the progressive muscular degeneration [14, 18].

Furthermore, neurological diagnosis is primarily polysyndromic: it is made according to the prevalence of clinical signs (sensory, motor, autonomic) and the distribution of lesions (symmetrical/asymmetrical, proximal/distal). These statements are important not only in terms of diagnostics, but also for adequate treatment and prognosis.

A particular algorithm to manage patients with hypothyroid PNP doesn't exist. Treatment and rehabilitation of patients with hypothyroidism should be based on hormone replacement therapy in doses, enabling to achieve and maintain euthyroidism [16]. On the other hand, considering those metabolic disorders developed under thyroid hormones insufficiency and causative of the central and peripheral nervous system damage, administration of additional medications normalizing the mentioned changes is substantiated. Therefore, in addition to the treatment of the underlying disease and achievement of hormonal status compensation, two main approaches may be suggested in the therapy of hypothyroid PNP: *pathogenetic therapy* (influence on the mechanisms of nerve fibers damage, stimulation of the regeneration of damaged nerve fibers) and *symptomatic*, targeted on the correction of the symptoms, first of all – on the pain management and patients' quality of life improvement.

Pathogenetic therapy is aimed at slowing of the neuropathy progression and correction of neuropathic deficiency. To achieve this, α-lipoic acid and group B vitamins are used [12, 26].

α-lipoic (thioctic) acid medications create the basis of pathogenetic treatment of PNP [4, 9, 12]. Accumulated in nerve fibers, they inactivate free radicals, block their generation, restore endogenous systems of antiradical protection, thereby providing a powerful antioxidant

effect, and restore a disturbed endoneural blood flow, normalizes the content of NO (a regulator of the vascular wall relaxation), improves endothelial function, reduces total cholesterol blood level, increases antiatherogenic lipoprotein fraction content [4]. Thioctic acid is a coenzyme to key enzymes of the Krebs cycle that explains its efficacy to optimize the energy metabolism of neurons. This action results in the improvement of nerve conduction by motor and sensory nerve fibers. Moreover, a positive effect of the medication on the liver cells is noticed — reduction of the severity of morphological manifestations of fatty liver and normalization of biochemical parameters [4, 9, 12].

A significant role in pharmacotherapy of PNP belongs to vitamin therapy. Group B vitamins improve the metabolism in the nervous tissue, metabolism of mediators, the transfer of excitation and, as a result, increase the rate of impulse transmission by nerve fibers, as well as implement a moderate analgesic effect, promote the processes of regeneration and remyelinization of nerve fibers [5, 6, 13].

As a result of phosphorylation processes thiamin (vitamin B_1) is converted in the body to cocarboxylase, which is a coenzyme of numerous enzymatic reactions and plays an important role in carbohydrate, protein and fat metabolism. Vitamin B_1 is involved in the synthesis of neurotransmitters that modulate the transmission of nerve impulses in the synapses, possesses anticholinesterase activity, hence stipulating neuromuscular conduction; it is one of the components of nucleic acid synthesis and stimulates the plastic and reparative processes in the nervous tissue [10].

Vitamin B_2 (riboflavin) is a catalyst for cell respiration, known to play an important role in redox processes of the nervous system, regulates the metabolism of carbohydrates, proteins, fats, potentiates the effect of pyridoxine and tryptophan, stimulates the regeneration of tissues.

Pyridoxine (vitamin B_6) reduces blood level of cholesterol and lipids, promotes the conversion of folic acid to its active form; it is a coenzyme in the metabolism of amino acids and proteins in the central nervous system cells, in the synthesis of biogenic amines, components of myelin sheath of neurons, neurotransmitters of the central and peripheral nervous system, thus providing a synaptic transmission.

Cyanocobalamin (vitamin B_{12}) provides hematopoietic, erythropoietic, anti-anemic, metabolic action, normalizes blood clotting processes, diversely influences liver function, including hematopoietic one, as well as digestive system, activates the metabolism of carbohydrates and fats, affects the synthesis of RNA, DNA. In addition, vitamin B_{12} suppresses the abnormal changes in case of degenerative atrophy of the nerve cells, causes resynthesis of myelin, creates the myelin sheath, and thereby provdes the restoration of normal nerve fiber structure and functions [5, 10].

Hence, the use of neurotropic group B vitamins can be considered as an important element of pathogenetic therapy of the PNP, promoting the regression of sensitivity disturbances, vegetative symptoms and pain syndrome. Since their simultaneous use is essential for the treatment efficacy, the use of combined B vitamins medications is practically expedient and simplifies patients' treatment significantly [5, 6, 13].

In addition to the mentioned drugs pathogenetic therapy of hypothyroid PNP is reasonably contributed by reparants (Actovegin, Solcoseryl) that demonstrate antioxidant, antyhypoxaemic, neurotrophic, neuroprotective action and are widely used in the rehabilitation of patients with various nervous system diseases of the vascular, atrophic, infectious, traumatic and other genesis [8, 20, 23].

In case of motor disorders anticholinesterase agents are used — neostigmine methylsulfate (proserine), ipidacrine (neiromidin), etc. These medications can restore and stimulate the neuromuscular transfer, restore impulse transmission by the peripheral nerves, enhance contractility of the smooth muscles, improve memory and learning ability through stimulation of nervous impulse transmission in the central nervous system as well, specifically moderately stimulating the CNS, provide analgesic effect due to the ability to block sodium permeability of the membranes.

The basis of symptomatic therapy of PNP is preferably related to the correction of the pain syndrome, programmed by the first-line drugs as anticonvulsants of a new generation — gabapentinoids (pregabalin, gabapentin) — soluble amino acids, chemically similar to the endogenous inhibitory neurotransmitter g-aminobutyric acid (GABA), involved in the transmission and modulation of the pain [3, 12].

Gabapentin has a number of biochemical properties that enable its influence on the pathogenesis of chronic neuropathic pain syndrome [3]:

— interaction with α2-δ2-subunits potential-dependent calcium channels, suppression of entry of Ca^{2+} ions into neurons inhibits excessive excitability of cell membranes, reduces sensitization of nociceptors;

— an increase of GABA synthesis stimulates the activity of glutamate decarboxylase, resulting in the enhancement of antinociceptive system activity;

— inhibition of the synthesis of glutamate (stimulating neurotransmitter with exitotoxicity) leads to a decrease in excitability structures nociceptive system and prevents neuronal death;

— modulation of the activity of NMDA (N-methyl-D-aspartate)-receptors affects the processes of «pain memory» formation.

Effective and safe for all types of spontaneous and stimulus-dependent neuropathic pain, gabapentin has flexible circuit of dose titration that provides highly individualized selection of therapy based on clinical characteristics of the patient and his pain syndrome [3].

Pregabalin is a modern anticonvulsant that has shown its efficacy regarding any type of neuropathic pain, fibromyalgia, seizures with a high analgesic activity and a positive impact on concomitant emotional-depression manifestations [21].

Treatment algorithm for neuropathic pain in PNP also includes tricyclic antidepressants (eg, amitriptyline), selective serotonin reuptake inhibitors (duloxetine,

venlafaxine), etc. [12], but their psychotropic side effects and cholinolytic action significantly limit their administration in patients with hypothyroidism.

Non-pharmacologic strategies for management of painful neuropathy include physiotherapeutic techniques (acupuncture, transcutaneuos electrical stimulation, high-wave external muscle stimulation, etc.).

Thus, neuromuscular disorders in case of thyropathies, including hypothyroidism, are known as polymorphic and still create significant diagnostic and therapeutic challenges. The processes of demyelination with secondary axonal damage, resulted from metabolic and bioenergetic disorders, initiated by thyroid hormones insufficiency, underlie the development of dysmetabolic polyneuropathy in hypothyroidism. Concerning this, early diagnosis, adequate replacement therapy and achievement of hormonal status compensation remain the major concerns to prevent and postpone the progression of hypothyroid polyneuropathy. However, pathophysiologically targeted therapy is a meaningful approach in management strategies for neuropathic changes, that amphasizes the importance of further study of pathogenesis, clinical and neurophysiological manifestations of hypothyroid polyneuropathy, particularly during the compensation period of the underlying endocrine disease, and develops algorithms for differential treatment.

Conflicts of interests. Authors declare the absence of any conflicts of interests that might be construed to influence the results or interpretation of their manuscript.

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Ураження периферичної нервової системи при гіпотиреозі: сучасний погляд на проблему (огляд літератури)

Резюме. У статті наведені дані про патогенез симптомів полінейропатії у хворих на гіпотиреоз, на підставі чого обґрунтовані шляхи патогенетичної терапії. Наведені найбільш важливі аспекти лікування полінейропатії на тлі гіпотиреозу згідно з новітніми міжнародними рекомендаціями, зазначені перспективні наукові напрямки для оптимізації надання допомоги пацієнтам із цією патологією.

Ключові слова: гіпотиреоз; полінейропатія; гіпотиреоїдна полінейропатія

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Поражение периферической нервной системы при гипотиреозе: современный взгляд на проблему (обзор литературы)

Резюме. В статье представлены данные относительно патогенеза симптомов полинейропатии у больных гипотиреозом, на основании чего обоснованы пути патогенетической терапии. Приведены наиболее важные аспекты лечения полинейропатии на фоне гипотиреоза согласно современным международным рекомендациям, указаны перспективные научные направления для оптимизации оказания помощи пациентам с этой патологией.

Ключевые слова: гипотиреоз; полинейропатия; гипотиреоидная полинейропатия