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INDICATOR OF DYSCIRCULATORY ENCEPHALOPATHY IN HYPOTHYROIDISM

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Abstract - In the scientific literature there are works devoted to the study of certain aspects of hemodynamics in hypothyroidism, but there is a lack of information about the features of cerebral circulation in conditions of deficiency of thyroid hormones. The aim of our study was to identify discirculatory encephalopathy and factors affecting its development in various forms of hypothyroidism.

Keywords: Hypothyroidism, encephalopathy, hemodynamics, hypertension, syndrome Gofmana, Hashimoto disease.

INTRODUCTION

Patients with hypothyroidism in Uzbekistan is constantly growing. The greatest prevalence of hypothyroidism is observed in the age group of postmenopausal women, older than 50 years [1]. The clinical manifestation of endothelial dysfunction in patients with hypothyroidism is dyscirculatory encephalopathy (DE), a cerebral pathology that develops as a result of metabolic and vascular disorders [6, 7].

The study included 60 women aged 50-54 with a history of clinical and subclinical hypothyroidism. For all nosological units accompanied by hypothyroidism syndrome, an endocrinologist was required to identify neurological disorders. Thyroid status (TSH, SV. T4, SV. T3) was evaluated twice, at 6-month intervals. The determination of antibodies to TPO was mandatory in the study of the thyroid gland, since an increase in antibodies in patients to thyroperoxidase indicated a lesion of the Central nervous system [8]. Hypothyroidism in the main group of patients was caused by autoimmune thyroiditis. In order to detect hypertensive encephalopathy in all subjects, a blood PRESSURE study was conducted, and women with blood PRESSURE were included in the study 125+4,0 / 70+10 in addition to the above, the neck vessels were examined by duplex scanning on the GeneralElectricVivid3.0 device (GeneralElectricHealthcare, USA).

Depending on the results obtained, all women included in the study were divided into 2 groups: group 1-30 patients with subcompensated hypothyroidism (SG) (TSH>4.5 med/l, SV.T4 within the reference values) and group 2 - 30 patients with decompensated hypothyroidism (TSH is higher, and SV. T4 is lower than normal). The exclusion criteria were thyroid hyperfunction, a history of cancer, stroke, or heart attack. The studied women had subjective or objective weak cognitive impairments: decreased memory, attention and intelligence, lethargy, apathy, drowsiness, the degree of these disorders depended on the severity of the decrease in thyroid function. These symptoms were preceded by neuromuscular manifestations such as Hoffmann's syndrome, hypothyroid myopathy. These States were emotionally colored: against the background of shortness of breath, anxiety, unmotivated fears, and vital longing appeared. The difference between these States and panic attacks was their duration and stereotype. It should also be emphasized that the administration of novopassit did not reduce the frequency and duration of seizures, but the appointment of thyroid hormone replacement therapy helped to reduce neurological symptoms.

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A 50-year-old man was admitted to drowsiness and confusion after three General attacks, each lasting 3-4 minutes, over two days. For the past four months, he has been acting strangely, putting things in their places and developing hand tremors. Two months prior to his current admission, he was hospitalized elsewhere with a generalized tonic-clonic attack and disinhibited behavior and was discharged for phenytoin. After that, he became withdrawn and stopped talking. At admission, he was slightly burned out, and it was noted that he had bilateral xanthelasma (cholesterol 7.5 mmol / l). He was conscious, but sleepy and disoriented in time, place, and person. He experienced hallucinations and delusions and displayed disinhibited behavior. Tremors were visible in all four limbs. Muscle tone, strength, and reflexes were normal. There was no sensory or cerebellar dysfunction, and there were no signs of meningeal irritation. The number of white cells was 13.2 × 10 9/ L and the profile of the electrolyte and liver were normal, with the exception of increased gamma-glutamyltransferase (296 u / l); the blood alcohol level was zero, and phenytoin was in the therapeutic range of 15.2 mg / l.the electroencephalogram (EEG) showed a General slowdown corresponding to encephalopathy. The CT scan of the head was normal, and the MRI showed a high signal in the right frontal lobe. The cerebrospinal fluid had an increased protein of 1.67 g / l, but there was no pleocytosis; the polymerase chain reaction to the herpes simplex virus was negative, as were the bacterial cultures. He had biochemical evidence of moderate hypothyroidism with TSH of 17 med / l (normal 0.35-5.0) and FT4 16.2 mmol / l (11-25). Titers of peroxidase antibodies against the thyroid gland were very high - 2699 IU / ml (0-75). Tests for rheumatoid factor, anti-nuclear antibodies, and all initial tests for common causes of metabolic encephalopathy were thus negative. The diagnosis of Hashimoto's encephalitis was based on the clinical picture, high antithyroid antibody titers, thyroid disorders, and elevated CSF proteins. After 4 days of treatment with oral prednisone 60 mg a day, he was oriented and behaved much more normally. When retested, TSH rose to 85 med / l, FT4 dropped to 3.2 mmol / l. It was started on thyroxine. Before his discharge, his score on the mini-mental health exam was 29/30, and his EEG was within normal limits.

The first case of Hashimoto's encephalopathy was reported in 1966. 1 in the few reported cases since then, the average age at admission was 41. This condition has been reported in children and is dominated by women (3.6: 1 ratio). 2

The diagnosis is difficult to make, since the underlying thyroid disease is often subclinical, and the symptoms mimic other neurological conditions.(10) Encephalopathy tends to be acute with a confused state, focal or generalized seizures, and episodes resembling a stroke. 3 Other manifestations -dysarthria, hallucinations, stupor, headaches and myoclonus. 2 the Diagnosis is confirmed by the detection of elevated antithyroid antibodies (antithyroglobulin, antithyroid peroxidase, anti-TSH receptor, anti-cytoplasmic).(15.6) the Main EEG disorders are generalized deceleration, frontal rhythmic deceleration, and three-phase waves. Cerebrospinal fluid protein is higher than normal in 75% of patients, and oligoclonal bands may be observed. 2MRI scans are normal in most patients, and reported abnormalities include generalized cerebral atrophy and reversible subcortical signal abnormalities. Single-photon emission computed tomography showed multiple areas of hypoperfusion in several cases.

Hashimoto's encephalopathy is believed to be caused by autoimmune vasculitis, and this concept is supported by the identification of the autoantigen α-enolase.Antibodies against α-enolase are associated with other forms of autoimmune vascular diseases, including systemic lupus erythematosus. Some workers object to the term Hashimoto's encephalopathy, arguing that thyroid autoantibodies may simply be a marker of other autoantibodies, not yet identified, that cause cerebral vasculitis. In the absence of evidence of a causal relationship between thyroid autoantibodies and encephalopathy, a more appropriate name may be encephalopathy associated with autoimmune thyroiditis.(12.13)Steroids seem to be an effective treatment in these conditions, although no official trials have been conducted. Other immunosuppressants have been reported to be effective. Some patients with hypothyroidism have fully responded to levothyroxine.

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Conclusions.

A decrease in blood flow in patients with clinical hypothyroidism suggested more pronounced atherosclerosis and improved diagnosis to detect atherosclerotic plaques. The appearance of connections between thyroid hormones and indicators of the common carotid artery, especially TSH and SV. T3, were associated with changes in metabolism, which also led to candothelial dysfunction and further encephalopathy. This is also indicated by the level of antibodies to thyroperoxidase.

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