

Significant prevalence of endemic goiter is an utterly urgent problem of modern thyroidology in Ukraine. The severity of this problem is caused by the fact that general state of health in the endemic goiter regions is in many ways worse than in regions with sporadic manifestations of the disease. However, as clinical experience shows, quite often in case of somatic disorders, changes of total and/or free fractions of thyroid hormones levels in the blood together with absence of organic lesions of the thyroid gland (TG) appear.

Changes in the concentrations of thyroid hormones are not usually accompanied by relevant clinical information and have direct dependence on the severity of main disease.

For description of this condition a variety of terms is used in the literature – «nonthyroidal illness syndrome», «pathological euthyroid syndrome», «euthyroid sick syndrome», «thyroid pseudodysfunction syndrome» «euthyroid patient syndrome» and some others. None of the terms fully reflects pathogenesis and clinical significance of this syndrome. The most appropriate to describe this phenomenon from the point of view of the essence of condition (somatogenically caused by the metabolism of thyroid hormones violation during the absence of thyroid pathology) is the term «thyroid pseudodysfunction syndrome» (TPDS).

Mechanisms related to violation deiodination of thyroxine ( $T_4$ ) in the liver, increase or decrease of binding of thyroid hormones with plasma proteins, increased utilization of triiodothyronine ( $T_3$ ) with tissues, deviations in secretion of thyroid stimulating hormone (TSH) are syndrome underlying basis. Due to modern existing approaches to the regulation of the major pathogenetic variants of TPDS, the following types are distinguished:

Type 1 – associated with  $T_3$  level decrease («low-  $T_3$  syndrome»);

Type 2 – associated with  $T_3$  and  $T_4$  levels decrease («low-  $T_4$  syndrome»);

Type 3 is characterized by high content of  $T_4$  and / or  $T_3$  («high  $T_4$  syndrome»);

Type 4 – isolated decrease of TSH («low TSH syndrome»);

Type 5 – isolated increase of TSH («high TSH syndrome»).

Most often in adolescents type 2 (TPDS with low  $T_4$ ) is characterized by early recorded changes related to the concentration of  $T_3$  («low  $T_3$  syndrome»). Hypothyroidism this age may manifest only with certain cognitive disorders, associated with memory and behavioral disorders. Often, especially in girls, panic attacks are observed.

There is a perception that hypothyroidism in adolescents may cause development of metabolic disorders and be a predictor of metabolic syndrome. It is known that reduction of thyroid function leads to hyperlipidemia. Another early effect of hypothyroidism change of general peripheral vascular resistance. Diastolic hypertension in case of hypothyroidism is common and is found in 15% of adolescents which is about 3 times as high as in euthyroid population. Sleep

apnea syndrome is often recorded in adolescents. It is combined with hypothyroidism in 10-20% of cases.

Hypothyroidism syndrome complicates the course and compensation of bronchial asthma, disorders of the gastrointestinal tract (dysfunction of the gallbladder, spasm of the sphincter of Oddi, chronic gastritis and gastroduodenitis, duodenal ulcer, chronic hepatitis). There is a certain connection between the severity of somatic disease and the probability and severity of hormonal abnormalities: the more severe is the background illness, the more massive changes of thyroid hormones levels are seen; substantial regression of nonthyroidal pathology shows notable regression of hormone levels variations.