Children's goiter

Definition

enlargement of the thyroid gland above the norm for the given age (in an ultrasound examination, the thyroid volume is greater than 2 standard deviations).[1]

Classification

By function

- eufunctional goiter,
- *hypofunctional* goiter,
  - bradycardia, weight gain, growth retardation, failure to thrive, dry skin and hair, facial flushing, eyelid swelling, drowsiness, fatigue, inefficiency, constipation, pseudopubertas praecox.
- *hyperfunctional* goitre,
  - tachycardia, weight loss, failure to thrive, warm, sweaty skin, heat intolerance, exophthalmos, diarrhea.[1]

⚠

By character

1. **diffuse goiter**:
   - lack of iodineu ("iodopenic goiter" - does not occur in the Czech Republic)
   - congenital disorder of hormone synthesis (dyshormonogenesis)
   - autoimmune inflammation (Hashimoto's lymphocytic thyroiditis, Graves-Based thyroiditis)
2. **multinodular goiter:**
   - sometimes occurs in AI inflammations of the SŽ
3. **localized node**:
   - thyroid carcinoma (most commonly differentiated papillary or medullary C-cell carcinoma - familial).[1]
   - solitary nodule in $^{3}/_{4}$ benign - cystic mass

Diffuse parenchymatous goiter

- right hyperplasia of the thyroid gland caused by chronic hyperstimulation, especially in chronic iodine deficiency (intake < 40 µg/day) and in morbus Basedow.

Goiter neonatorum

- etiology: insufficient intake of iodine during pregnancy; transplacental transfer of strimogenic substances (PAS, resorcinol); treatment of pregnant thyrostatics; transmission of TRAK antibodies in a mother with Basedow's disease;
- intrauterine lack of thyroid hormones → increased secretion of TSH → goiter;
- visible enlargement of the thyroid gland → stridor, difficulty breathing.

Juvenile euthyroid goiter

- iodine deficiency or familial iodine utilization disorder;
- development of goiter during puberty, more often in girls;
- eufunctional goiter, homogeneously enlarged → necrosis, cysts, nodules;
- thyroid hormones at the lower limit of normal, TSH normal, normal TRH test, thyroid antibodies negative;
- optimization of iodine supply (200 µg/day) if the goiter is refractory to iodine – suppression of TSH with thyroxine.[2]

Disease

Congenital hypothyroidism

- most common congenital endocrine disease (prevalence 1:4000);
- thyroid hormones play a key role in brain development, especially up to 8 months of age (a little less then up to 3 years of age);
- without replacement treatment, irreversible brain damage occurs – at the time of clinical diagnosis, the brain is already irreversibly damaged;
- since 1985, nationwide newborn screening has been introduced - determination of the TSH level;
- etiopathogenesis: **dysgenesis of the thyroid gland** (agenesis, aplasia, hypoplasia, hemithyroid, cystic malformation, ectopia) or *dyshormonogenesis* (disorder of any level of synthesis or secretion of hormones), or

rare **isolated congenital central hypothyroidism** (congenital TSH defect - cannot be detected by newborn screening);
- clinical picture without treatment: protracted neonatal icterus, failure to thrive, delayed growth rate and bone maturation – late closure of fontanels, delayed eruption of milk dentition, macroglossia, muscle hypotonia, omphalocele, constipation, hoarse cry;
- neonatal goiter or normal-sized thyroid gland;
- laboratory findings: ↑TSH, ↓fT_4; (for the central form ↓TSH and fT_4);
- therapy: lifelong L-thyroxine replacement therapy (started as soon as possible).[3]

Autoimmune thyroid disease

- the most common acquired thyropathy in children and adolescents; more often in girls;
- mostly **lymphocytic (Hashimoto's) thyroiditis'**;
- often associated with other autoimmune diseases (diabetes mellirus type 1, celiac disease) and with chromosomal aberrations (Down's syndrome, Turner's syndrome);
- soft diffuse goiter; USG: diffuse inhomogeneous texture (“pepper and salt”); histology: lymphocytic infiltration of the gland;
- etiopathogenesis: autoantibodies against thyroid peroxidase (anti-TPO) and against human thyroglobulin (anti-hTG);
- clinical picture: first phase of euthyroidism, then phase of permanent hypothyroidism; there may also be transient hyperthyroidism ("hashitoxicosis");
- without treatment: growth retardation, dyslipidemia, obesity, impaired school performance, anemia, dry and rough skin, premature pseudopuberty or delayed puberty, myxedema, constipation, bradycardia;
- laboratory findings: ↑TSH, ↓fT_4;
- therapy: lifelong L-thyroxine replacement therapy.[3]

Neonatal hyperthyroidism (thyrotoxicosis)

- a rare disorder that can endanger the life of a newborn;
- etiopathogenesis: transplacental transfer of maternal antibodies against the TSH receptor in maternal thyrotoxicosis of the Graves-Basedow type;
- clinical picture: hyperthyroidism from the fetal period – IUGR, tachycardia, acceleration of bone maturation, goiter, exophthalmos, risk of metabolic disruption and heart failure;
- laboratory finding: ↑fT_4;
- therapy: antithyroid treatment until the maternal antibodies disappear, i.e. in a decreasing dose for 2-3 months.[3]

Graves-Basedow thyrotoxicosis

- most common cause of hyperthyroidism in children; especially in adolescent girls;
- etiopathogenesis: autoantibodies against the TSH receptor (TRAb, rTSH-ab), which have a stimulating effect on the thyroid gland;
- clinical picture: hyperkinetic circulation with tachycardia and systolic hypertension with increased pressure amplitude, weight loss, impaired school performance, irritability, nervousness, mild hand tremors, diarrhea, sweating, in 60% orbitopathy with exophthalmos - occurs by the proliferation of retrobulbar connective tissue due to autoimmune stimulation;
- in 75% goiter – heavily perfused, warm, palpable vortex;
- laboratory findings: ↓TSH, ↑fT_4;
- therapy: thyrostatics (methimazole, carbimazole, propithiouracil), in case of repeated relapses total thyroidectomy and lifelong replacement therapy with L-thyroxine.[3]

Iodopenic goiter

- our natural diet is low in iodine → iodization of table salt since the 1950s;
- iodine deficiency → reduced production of thyroid hormones → ↑TSH → iodopenic goiter;
- endemic cretinism - eradicated.[3]

Investigation

Clinical examination

- palpation examination of the LV (between the jugular fossa and the beginning of the trachea)
- WHO criteria:
  - GRADE 0: the thyroid gland is not palpable or palpable
  - GRADE 1: thyroid gland is palpable but not visible in normal neck position
  - GRADE 2: thyroid gland is palpable and visible in normal head position[4]

Laboratory examination

- **TSH**
  - immeasurably low – hyperthyroidism
  - slightly elevated – subclinical hypothyroidism

    - significant increase (tens of mIU/l) – hypothyroidism,
- **fT4**
    - significantly increased – hyperthyroidism
    - significantly reduced – hypothyroidism
- **antibodies**
    - anti-TPO (thyroid peroxidase) – elevated values indicate autoimmune thyroiditis
    - anti-hTG (human thyroglobulin)
    - TRAK/TRAb (TSH receptor stimulating antibodies) – Graves-Basedow disease

Ultrasound examination of the spine

- we evaluate the size, echotexture of the gland and look for focal changes.[1]

FNAC under USG control

- fine needle aspiration biopsy for subsequent cytological examination

Differential diagnosis of goiter in children

- medial and lateral neck cysts,
- lymphangioma,
- hemangioma,
- thyroiditis,
- thyroid adenoma,
- thyroid carcinoma.[2]

Therapy

- prevention of endemic goiter: fortification of salt with iodine
- iodine substitution
- at volume +80ml: surgical thyroidectomy or radioablation

Complications

- risk of oppression of surrounding structures: dyspnoea, dysphagia, superior vena cava syndrome

Links

Related Articles

- Thyroid disease: Hypothyroidism • Hyperthyroidism
- Examination for thyroid gland diseases • Examination of thyroid gland function
- Symptomatic mental disorders in endocrinopathies

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