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Thyroid dysfunction in children with trisomy 21: when subclinical hypothyroidism should be treated

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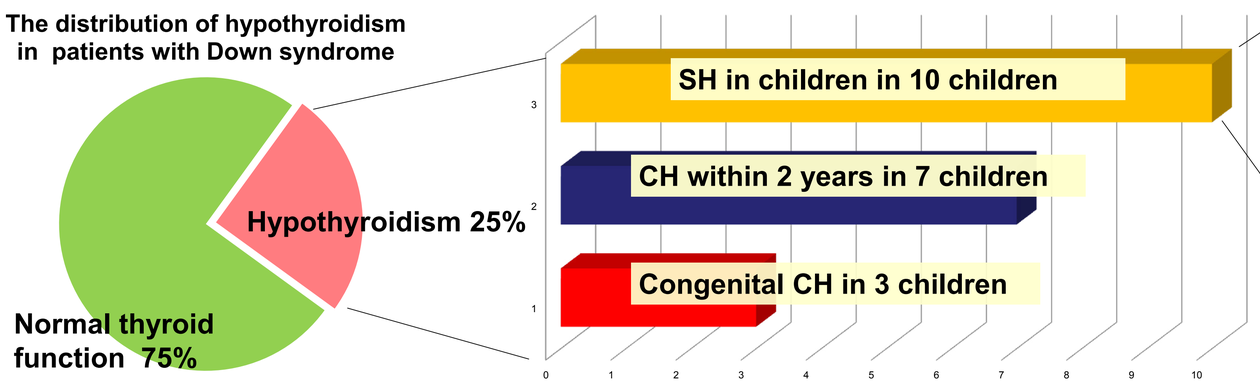
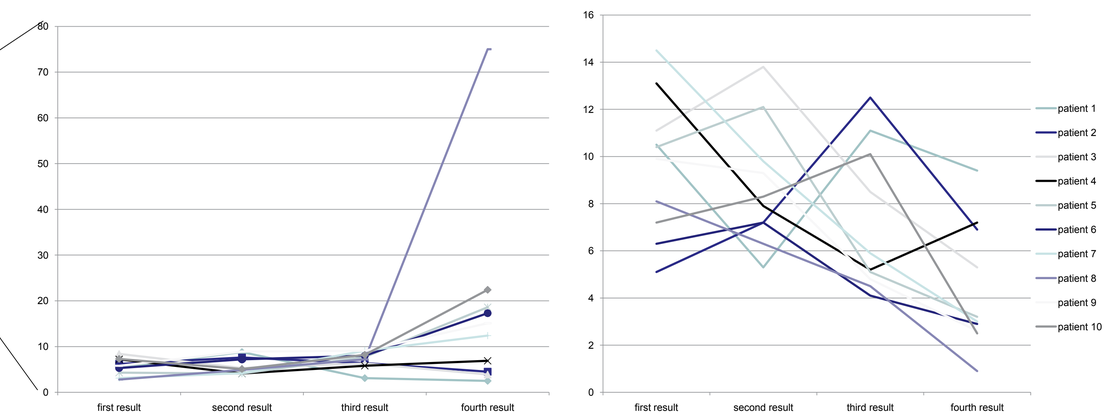
Background

Thyroid dysfunction is well-established feature in children with Down syndrome (DS). There are several reasons for both clinical (CH) or subclinical (SH) hypothyroidism in these children- thyroid dysgenesis and dyshormonogenesis early in life, thyroid insensitivity to TSH; or autoimmune disease during school age.

Materials and methods

Thyroid function from 80 children with DS was evaluated during the first 14 years of life. The cohort was divided according age and type of thyroid dysfunction. TSH and T4 were taken in all at neonatal age, and than consequently repeated: in patients with normal results yearly; in those with elevated TSH and those children with therapy in every three months. Ultrasonographic evaluation was made in most of the children. Various developmental tests according age were made in all; re-evaluation was made in the group of SH or when starting the therapy with l-thyroxine.





Follow-up of the levels of TSH (first chart) and T4 (second chart) in 10 patients with subclinical hypothyroidism in four consecutive visits. Patients 1-4 had normal thyroid function at the last point, while the remaining patients (5-10) developed clinical hypothyroidism.

Results

Impaired thyroid function was found in 25% of patients, from which 3 (3,7%) had congenital hypothyroidism detected on neonatal screening, the remaining 7 (8,7%) developed clinical hypothyroidism within the first 2 years. Subclinical hypothyroidism was noticed in 10 (12,5%) respectively. There was dysgenetic thyroid (hypoplastic, ectopic or unilobulated) in 30% of hypothyroid children. The children with SH were followed up for at least 3 years, of which 4 had improved thyroid function, and in 6 elevation of TSH and decrease of FT4 occur. In these children the therapy with l-thyroxine was given. In latter group developmental tests worsen at least 6 months before elevation of TSH; after establishing the therapy they showed better developmental skills. According age, there were two peaks of onset of hypothyroidism- first in 2nd year -mostly children with thyroid dysgenesis, and the second around 13th year of age, after the onset of the puberty. Thyroid antibodies were negative in majority of cases. Only one child had hyperthyroidism due to autoimmune process.

Conclusions

Thyroid dysgenesis seems to have major role in developing clinical hypothyroidism in children with DS at birth or early childhood. Autoimmune process is unlikely cause for thyroid dysfunction in DS children as stated in the literature.
Frequent follow up of thyroid parameters, ultrasonographic finding and developmental tests are needed in order to begin with therapy on-time.

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