FOLLOW-UP in CHILDREN with NON-OBESE and NONAUTOIMMUNE SUBCLINICAL HYPOTHYROIDISM

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Introduction

Subclinical hypothyroidism is a form of thyroid dysfunction in which the TSH level is high, while serum total/free thyroxin (T4 /fT4) is within the normal reference range. In the majority of patients with subclinical hypothyroidism, there are no symptoms or findings of hypothyroidism while structural, clinical and biochemical evidences of hypothyroidism are present in some cases. The etiology of subclinical hypothyroidism is similar to that of overt hypothyroidism; temporary and persistent causes have been identified. Hashimoto thyroiditis is the most common cause of subclinical hypothyroidism, and therefore, most studies on subclinical hypothyroidism were conducted in patients with Hashimoto thyroiditis. The natural course in subclinical hypothyroidism is unclear. It has been suggested that the risk for progression to overt hypothyroidism is rather low in juvenile subclinical hypothyroidism, and that subclinical hypothyroidism is a benign, reversible process. Despite the lack of high-quality evidence, current data suggest that untreated subclinical hypothyroidism has no adverse effect on growth, development and neurocognitive functions. However, there is a need for high-quality, randomised-controlled trials to clarify issues regarding treatment and follow-up in children. This study aimed to identify clinical and laboratory characteristics of non-obese patients (aged 3-18 years) with non-autoimmune subclinical hypothyroidism (negative thyroid auto-antibodies) and to investigate the effects of subclinical hypothyroidism on anthropometric characteristics, blood pressure, glucose and lipid metabolism by evaluating the course of subclinical hypothyroidism during follow-up without treatment.

Patients and Methods

The study included patients aged 3-18 years (BMI<85th percentile) with negative thyroid autoantibodies (anti-TPO and anti-Tg), who were diagnosed with subclinical hypothyroidism. Patients with acute or chronic disorder, which may interact with thyroid function tests, and those with a history of medication, were excluded. Subclinical hypothyroidism was diagnosed when the TSH level was elevated, but serum-free thyroxin (fT4) and free triiodothyronine (fT3) were within the normal reference range. Patients with subclinical hypothyroidism were assessed at time of diagnosis, on month 3 and at year 1 during follow-up.

Results

At month 3, TSH elevation persisted in only 7 31.8%) patients, while TSH levels were normal in 15 (68.2%) of 22 cases. At year 1, TSH levels were normal in 14 cases (73.7%) and elevated in 5 cases (26.3%). Table 1 presents the clinical, laboratory and sonography findings at time of diagnosis and during follow-up in patients with subclinical hypothyroidism. There was no progression to overt hypothyroidism during 1-year follow-up and that subclinical hypothyroidism had no effect on height SDS, BMI SDS, blood pressure, glucose and lipid metabolism during follow-up without treatment in this group of patients.

Table 1: Clinical, laboratory and sonography findings at time of diagnosis and during follow-up in patients with subclinical hypothyroidism

	Diagnosis n=25	Third month n=22	First year n=19	p
Height SDS	0.71 ± 0.87	0.69 ± 0.74	0.68 ± 0.79	p=1.00
Weight SDS	0.63 ± 0.86	0.76 ± 0.85	0.83 ± 0.93	p=0.411
BMI	16.62 ± 2.09	16.65 ± 2.3	16.39 ± 2.27	p=0.864
BMI SDS	0.31 ± 0.77	0.41 ± 0.96	0.65 ± 1.11	p=0.021*
Bone age	8.89 ± 4.14		9.76 ± 3.94	P=0.609
BA/CA	0.92 ± 0.16		0.92 ± 0.16	p=0.609
Pulse rate (/min)	88.68 ± 12.98	89.73 ± 9.05	87.32 ± 9.4	p=0.396
Systolic blood pressure	98.6 ± 11.22	102.27 ± 9.35	104.58 ± 8.63	p=0.009†
Diastolic blood pressure	62 ± 9.89	62.05 ± 8.4	65.26 ± 7.72	p=0.492
TSH (mIU/ml)	.92 ± 0.92	4.77 ± 1.57	4.51 ± 1.79	p=0.000*
sT4 (ng/dl)	1.22 ± 0.12	1.19 ± 0.14	1.15 ± 0.13	p=0.067
sT3 (pg/ml)	4.22 ± 0.52	3.98 ± 0.39	3.88 ± 0.424	p=0.017†
Urine iyodine level (µg/l)	209.16 ± 138.88		114 ± 65.07	p=0.062
Urine iodine status	Adequate (n=11) (%45.8) Deficiency Mild (n=2) (%8.3) Moderate(n=1) (%4.16) Excess (n=10) (%41.6)		Adequate (n=7) (%36.8) Deficiency Mild (n=8) (%42.1) Moderate(n=2) (%10.5) Excess (n=2) (%10.5)	
Thyroid volume (ml)	3.91 ± 2.11 ml		3.37 ± 1.6	P=0.573
Hemoglobin (g/dl)	13.5 ± 1.09		13.24 ± 0.86	p=0.393
White blood cell (x10^3/µl)	6.97 ± 1.65		7.07 ± 2.26	p=0.809
Platelets (x10^3/µl)	322 ± 75.86		319 ± 97.42	p=0.243
Total cholesterol(mg/dl)	155.6 ± 23.04		160.52 ± 28.4	P=0.888
LDL cholesterol(mg/dl)	85.7 ± 22.13		92.76 ± 25.66	p=0.205
HDL cholesterol (mg/dl)	52.5 ± 10.85		51.66 ± 10.31	p=0.286
Triglycerides(mg/dl)	81.24 ± 46.92		80.84 ± 45.33	p=0.732
Fasting Glucose (mg/dl)	86.72 ± 8.2		88.74 ± 9.79	p=0.073
Fasting insulin(µIU/ml)	8.01 ± 2.99		8.84 ± 6.3	p=0.778
HOMA-IR	1.71 ± 0.68		1.91 ± 1.25	p=0.794
CRP (mg/L)	3.81 ± 2.53		5.27 ± 5.8	p=0.468
Sensitive CRP (mg/L)	0.08 ± 0.1		0.22 ± 0.21	p=0.035†

*p <0.05 (3. month - 1. year), † p <0.05 (Diagnosis-1. year)

Conclusion

In the limited number of follow-up studies on juvenile subclinical hypothyroidism, it was suggested that the risk for progression to overt hypothyroidism is rather low in subclinical hypothyroidism and that subclinical hypothyroidism is a benign, reversible process.

In our study, the normalisation of the TSH level in the majority of cases can be explained by the study population, which consisted of patients with mild subclinical hypothyroidism (TSH level of 5-10 mIU/L), lack of thyroid or extra-thyroidal disorders, negative thyroid auto-antibodies and absence of goitre, since it is known that high TSH levels at time of diagnosis, goitre and positive thyroid auto-antibodies are the main risk factors for progression to overt hypothyroidism.

We concluded that there was no progression to overt hypothyroidism during one-year follow-up and that subclinical hypothyroidism had no effect on height SDS, BMI SDS, blood pressure, glucose and lipid metabolism during follow-up without treatment.

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Thyroid

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