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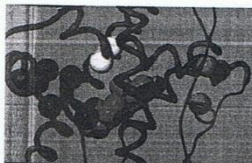
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P1004

Thyroid function during long-term GH treatment in children affected by idiopathic GH deficiency

A. Ciresi, V. Guarnotta, C. Perrone, A. Galluzzo & C. Giordano

¹Dipartimento Biomedico di Medicina Interna e Specialistica (Di.Bi.Mi.S), University of Palermo, Palermo, Italy.

Alterations in the thyroid axis have been reported following GH therapy both in adults and children with GH deficiency (GHD), even if the clinical significance of these changes, in small and short-term studies, remains uncertain. To evaluate the impact of GH replacement on thyroid status in a large selected cohort of prepubertal children with idiopathic GHD during a 3-years follow-up. Study outcome considered FT3, FT4 and TSH modifications (delta) during therapy and their correlations with IGF-1, biochemical and auxological data. Data of 105 children (82 M, 23 F; mean age 11.13) were retrospectively analysed. At diagnosis, the areas under the curve of GH (AUCGH) were calculated during GHRH-Arg test and ITT. At baseline and yearly up to 36 months during GH treatment, we measured height-velocity, BMI, IGF-1, FT3, FT4, TSH. A significant FT3 delta ($P < 0.001$) was documented in 89/105 (84.7%) patients at 12 months after starting GH treatment, without any further change at 24 and 36 months. No significant FT4 and TSH delta were observed during the follow-up. Grouping all patients according to FT3 delta at 12 months in those with lower (No 80, 76%) or greater value than 75 percentile (No 25, 24%), the latter showed significantly lower AUCGH during GHRH-Arg ($P = 0.018$) and ITT ($P = 0.023$). These children also showed lower FT3 levels at baseline ($P < 0.001$), without difference in FT4 and TSH. No significant differences in auxological parameters were detected between the two groups. In GHD children, GH treatment is associated with a significant increase in FT3 levels in the first 12 months. However, the lack of significant effects of these changes in relation to the auxological parameters does not suggest the routinely monitoring of thyroid function in initially euthyroid patients.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research project.

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Table 1

	baseline	12 months	24 months	36 months
Height (s.d.)	-2.11 ± 0.89	-1.73 ± 0.75	-1.44 ± 0.76	-1.03 ± 0.85
IGF-1 (µg/l)	98.83 ± 47.94	314.68 ± 193.25	330.30 ± 172.95	349.90 ± 145.88
FT3 (pg/ml)	3.10 ± 0.94	4.13 ± 0.53	4.12 ± 0.53	4.21 ± 0.53
FT4 (ng/dl)	1.25 ± 0.24	1.25 ± 0.28	1.19 ± 0.25	1.19 ± 0.23
TSH (µU/ml)	1.98 ± 0.84	1.92 ± 0.97	1.95 ± 1.03	1.51 ± 0.54
Δ FT3	-	1.02 ± 1.08*	0.02 ± 0.73	0.07 ± 0.44
Δ FT4	-	0.01 ± 0.04	0.06 ± 0.01	0.01 ± 0.02
Δ TSH	-	0.06 ± 0.13	0.03 ± 0.06	0.44 ± 0.59
Δ IGF-1	-	217.6 ± 181.5*	26.2 ± 153.3*	44.3 ± 98.5*

* $P < 0.005$.

P1005

Variations of IGF1 in GH deficiency and Algerian health children

N. Fedala, F. Chentli, A. Haddam & L. Griene

BEO Hospital, Algiers, Algeria.

IGF1 represent an important key player in several physiologic process, so it can be implicated in different pathologies among delays of growth. A comparative study was realized comparing a group of healthy Algerian children of normal size (n : 266) and a group presenting a GH deficiency (GHD n : 107) to a group of healthy westerners children of normal size (Serie of ROSENFELD).

The results are as follows

Before the age of 04 years, there is a great overlapping in the results of IGF1 between children who suffer from GHD and those of the comparative group.

After this age, results of IGF1 are significantly low in GHD subjects, compared to the Algerian reference group.

The values of IGF1 found in the Algerian healthy subjects are significantly lower than those observed in the western healthy children. So this parameter must be carefully interpreted when investigating a delay of growth in Algerian and must take into account the clinical and biological context of the patient. It is necessary to establish Algerian standards for IGF1. The introduction of the IGFBP3 not yet available would be more interesting in our context.

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P1006

Association Turner's syndrome and GH deficiency

I. Oueslati, I. Hadj Ali, K. Khiari, N. Mchirgui & N. Ben Abdallah

Charles Nicolle Hospital, Tunis, Tunisia.

One of the most clinical characteristic of Turner's syndrome is the final short stature.

In order to cure this handicap, several teams were interested to treat these patients by the GH.

We report two cases of Turner syndrome associated with GH deficiency.

In the first, a 17 years old girl, having a delayed growth lower than -4 s.d. associated with delayed puberty and dysmorphic syndrome. A karyotype made confirmed the diagnosis of Turner's syndrome. In front of the short stature lower than that usually found in the Turner syndrome, GH was measured during insulin induced hypoglycaemia test and L-dopa test. The two tests revealed a GH deficiency.

The second observation is about a 21 years old girl having a delayed growth between -2 and -3 s.d. associated with delayed puberty and dysmorphic syndrome.

The karyotype finds a chromosomal formula with 45×0 confirming the Turner's syndrome.

GH was measured under stimulation test, showing a GH deficiency.

The dosage of the GH in Turner's syndrome appears interesting and must be of current practice especially when the short stature is lower than that usually found in Turner syndrome.

Declaration of interest

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Male Reproduction

P1007

The homologous hormones lutropin and choriogonadotropin are interacting differently with the LH/CG receptorP. Grzesik¹, A. Teichmann¹, A. Kreuchwig¹, J. Furkert¹, C. Rutz¹,B. Wiesner¹, R. Schüle¹, J. Gromoll² & G. Krause¹¹Berlin, Germany; ²Reproduktionsmedizin und Andrologie, Münster, Germany.

Activation of the human LH/CG receptor (LH/CGR) by lutropin (LH) and choriogonadotropin (CG) is essential in the human reproduction. Deletion of the Exon10 (LH/CGR-delExon10) resulting in a lack of 27 amino acids within the extracellular hinge-region of LH/CGR causes Leydig cell hypoplasia type II. To clarify why this deletion impairs LH but not CG action, we investigated the molecular determinants of LH/CGR activation elucidating the different behaviour of both hormones.

It is reported that the LH/CGR exist as oligomers in a unitary signalling unit when activated with CG, since individual binding- and signalling-defective mutants can be rescued by combining both. We performed a similar strategy including also LH/CGR-delExon10 as signalling defective mutant. However, in contrast to CG,

