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1

GROWTH HORMONE (GH) TREATMENT OF CHILDREN WITH CHRONIC RENAL FAILURE (CRF) AFTER RENAL TRANSPLANTATION (Tx)

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Children with CRF grow poorly and, frequently, neither dialysis nor Tx improve growth. It has been proposed that these children have target organ resistance to the effects to GH/IGF-I which could be overcome by supraphysiological doses of GH. Our objective was to evaluate growth response and renal function after GH treatment (1 IU/Kg. week) in patients with CRF after Tx. Seven patients (5 prepubertal and 2 pubertal children) were studied: mean \pm SD chronological age (A) 12.2 \pm 3.25 years (y), bone A 8.47 \pm 3.94 y, height SDS -3.82 \pm 1.44 and post Tx time 1.99 \pm 2.16 y. All patients had normal serum T4 and T3 levels and GH responses to pharmacological tests (> 7 ng/ml). Mean GH treatment time was 7.0 \pm 3.46 months. Parameters of biological actions of GH (growth velocity: GV, serum levels of SHBG and IGF-I) and of renal function (Cr cl) were evaluated before and after treatment. Findings were as follows: a significant increment in GV (from 2.46 \pm 0.63 to 7.42 \pm 1.68 cm/y, $p < 0.001$, ANOVA), similar CrCl (96.2 \pm 12.9 and 100.6 \pm 25.8 ml/min.1.73m), a significant decrement in serum SHBG (from 75.9 \pm 32.4 to 55.5 \pm 31.5 nmol/L, $p < 0.01$) and a non significant increment in serum IGF-I (from 220 \pm 66.5 to 305 \pm 142.5 ng/ml). It is concluded that GH treatment at supraphysiological doses in CRF patients after TX does increase GV without changing functions parameters of the transplanted kidney. Serum SHBG is a biological response parameter to GH.

2

ENERGY EXPENDITURE AND DIENCEPHALIC SYNDROME IN INFANCY

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The diencephalic syndrome (DS) of infancy is one of the definable causes of failure to thrive. It produces a very characteristic syndrome of emaciation, in spite of apparently adequate food intake, and a hypermetabolic state with hyperactivity, hyperalertness, tachycardia, fever, excessive sweating and flushing of the skin.

We have measured the resting energy expenditure (REE) by indirect calorimetry in one male infant, 9 month old, during two consecutive hospital admissions. Both REE were 38 and 32% higher than normal by age or by body weight. Daily intakes were estimated by difference of food weights. Due to the continuous state of hypermetabolism, we admitted an increase of about 40% of REE. Therefore, daily energy expenditure were calculated in the order of 123 and 129 kcal/kg/day, practically similar or higher than the intake. We conclude that the failure to thrive of the DS may be consequent to the increased REE, associated to the insufficient intake to cover the total daily energy expenditure.

3

ZINC NUTRITION IN CHILEAN PREGNANT TEENAGERS OF LOW INCOME GROUPS.

Martin VB, Castillo CD, Gattás V, Castillo F, González M, Zerrazzi G, Alcázar ML.

Zinc deficiency may affect pregnancy outcome. In order to carry out longitudinal studies of zinc status in Chilean population of low income groups, we evaluated 124 pregnant teenagers at the beginning the prospective follow-up. They were 16.3 \pm 1.4 years old (12-18), beginning health controls at 12.6 \pm 3.7 weeks (7-20); nutritional status showed 28% of inadequate weight for height and gestational age. Dietary history records revealed daily intakes of: Zn 6.8 \pm 2.7 mg., or 0.81 mg/1000 Kcal (60% of RDA) with 47.5% of animal origin; vegetal fiber 5.3 \pm 2.8 g. Plasma zinc was 96.3 \pm 13.7 ug/dl (15/126 $<$ 80 ug/dl); Hb 13.0 \pm 1.3 (4/116 $<$ 11 g/dl).

We conclude that Chilean pregnant teenagers of low income groups present a higher prevalence of low zinc intake that those of other nutrients, not associate to low plasma zinc.

4

PREVENTION OF VITAMIN D DEFICIENCY IN USHUAIA.

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The Vitamin D deficiency enhanced the risk of rickets in children. Previous studies in Ushuaia (55°S) showed that the 47% of infant population have diminished levels of 25 hydroxy-Vitamin D (25(OH)D) (8 ng/ml) at the end of winter, being normal at end of summer. To prevent the vitamin D deficiency a single dose of 150,000 IU of Vitamin-D2 was given to a group of 79 children from Ushuaia with age 8.6 \pm 1.4 years (X \pm SD). Serum levels of calcium, phosphorous, alkaline phosphatase, 25(OH)D, Parathyroid Hormone (PTH) and the ratio of urine calcium/urine creatinine in a 2 hs. urine sample (UCa/UCreat) was measured at basal, 6 weeks and 5 months (end of winter) after an oral Vitamin D2 dose. The results are as follows:

	Ca (mg/dl)	P (mg/dl)	AP (KAU/dl)	25OHD (ng/ml)	PTH (pg/ml)	UCa/UCreat (mg/mg)
Basal	9.7 \pm 0.5	5.6 \pm 0.7	29 \pm 8	18.9 \pm 10.7	45.9 \pm 14.2	0.09 \pm 0.07
6 weeks*	9.6 \pm 0.6	5.6 \pm 0.6	28 \pm 7	26.7 \pm 13.8*	50.0 \pm 15.2**	0.09 \pm 0.06
5 months*	9.4 \pm 0.5***	5.4 \pm 0.6	30 \pm 7	17.2 \pm 9.2	50.9 \pm 14.1**	0.07 \pm 0.05**
	* post 150.000 IU Vit D2 ** $p < 0.006$ *** $p < 0.04$ **** $p < 0.02$					

After the 150,000 IU of Vit D2 administration the serum 25(OH)D levels at the end of winter were similar to those at the beginning of autumn, but significantly higher from those obtained in a previous study without Vit D (9.8 \pm 3.8 ng/ml) ($p < 0.001$). PTH levels were higher at the end of winter, but this increment was lower than without Vit. D. Both, serum calcium levels and the ratio UCa/UCreat were lower at 5 months after dose. Neither hypercalcemia nor hypercalciuria were observed. Conclusion: A single dose of 150,000 IU of Vit. D maintained appropriate levels of 250 HD without hypercalcemia nor hypercalciuria.