LIMITATIONS OF BASAL IGF-1 STATUS REFLECTING THE SEVERITY OF GROWTH HORMONE DEFICIENCY AND PREDICTING RESPONSE TO REPLACEMENT THERAPY. A RETROSPECTIVE COHORT STUDY IN A TERTIARY REFERRAL CENTRE IN SPAIN

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INTRODUCTION:

Growth hormone (GH) replacement in adults has demonstrated to improve body composition, inflammatory cardiovascular biomarkers and quality of life. However, this benefit is uneven, and factors that stratify treatment response are required.

It has not been described before if GH deficient patients with normal IGF-I get the same clinical benefit from replacement as individuals with low IGF-I.

OBJETIVE:

The aim of this study is to compare the response to GH replacement (GHR) between the subgroup with normal IGF-I and patients with low IGF-I.

MATERIALS & METHODS:

We analyzed retrospectively 34 GHD adults (mean age 40.4 yr; 16 females) from our centre who received GHR for at least two years (mean duration of treatment 7.4 years). Anthropometric parameters, bone mineral density (BMD) of the lumbar spine and hip, and the scores in quality of life questionnaires (AGHDA and Nottingham) were measured before starting treatment and at the end of the follow-up. Differences in these parameters were tested by Mann-Whitney U test.

RESULTS:

	BASELINE	FINAL	MEAN ±SD	р
IGF-1 (ng/dl)	65,96	184,35	-118,39±53,5	0,00
BMI (kg/m²)	34,63	30,33	4,3±15,68	0,113
T-score lumbar spine	-1,22	-1,24	0,01±0,8	0,934
T-score hip	-1,14	-1,09	-0,05±0,9	0,788
LDL (mg/dl)	134,03	123,42	10,61±42,83	0,178
Triglycerides (mg/dl)	112,09	131,47	-19,38±37,7	0,005
Fasting glucose (mg/dl)	87,4	94,03	-6,62±10,96	0,001
HbA1c %	5,33	5,53	-0,19±0,42	0,014
Nottingham score	12,66	8,86	3,79±7,39	0,010
AGHDA score	10,35	7,35	3,0±3,94	0,003
Lean body mass(Kg/%)	0,310	0,292	0,018±0,07	0,285
Fat mass (Kg/%)	0,12	0,212	-0,012±0,04	0,224

Nine patients had normal basal IGF1 levels. No significant differences were found in the baseline parameters between the subgroup with normal IGF-I and patients with low IGF-I. The AGHDA and Nottingham mean scores improved significantly at the end of follow-up [-3 (SD3.95) p=0.003 and -3.8 (SD7.4) p= 0.01 respectively] (table 1), but there were no differences between patients with normal or low IGF-I (p<0.109 and p<0.533) (table 2). No significant changes were observed in BMD or body composition.

	NORMAL BASAL IGF-1	LOW BASAL IGF-1	
	MEAN CHANGE±SD	MEAN CHANGE±SD	p
T-score lumbar spine	-0,16±1,10	0,03±0,72	0,8
T-score hip	-0,1±0,8	0,1±0,95	0,933
LDL (mg/dl)	-12,37±32,08	-13,13±45,16	0,836
Triglycerides (mg/dl)	14,12±43,57	20,48±37,24	0,918
Fasting glucose (mg/dl)	6,55±11,92	7,04±10,89	0,565
HbA1c %	0,12±0,55	0,25±0,34	0,504
Nottingham score	-1,71±6,99	-4,52±7,73	0,533
AGHDA score	-0,66±3,38	-4,0±3,84	0,109
Lean body mass (Kg/%)	-0,02±0,04	-0,01±0,08	0,736
Fat mass(Kg/%)	0,006±0,01	0,01±0,04	0,574

Table 2. Effects of GH replacement therapy between the groups with normal and low IGF-1

CONCLUSION:

Our data show that benefits of GH replacement are maintained across different scales of IGF-1 secretion. Thus, this study highlights the limitations of basal IGF-1 status in reflecting the physiopathology of GHD, indicating the severity of GHD and selecting candidates for replacement.

Table 1. Effects of GH replacement therapy