

Body Mass Index (BMI) in patients with Growth Hormone Deficiency (GHD) at diagnosis, one year and two years after treatment with Growth Hormone (GH)

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INTRODUCTION

GHD happens because the pituitary gland is unable to produce enough hormone for the body needs. It can be a full hormone deficiency or not. This syndrome is related to metabolism disturbance and pituitary disease. Among children GHD is usually idiopathic and not congenital. It is also related to accumulation of abdominal fat, reduction of insulin sensitivity and blockage of bone mineralization. Growth velocity is reduced in patients with GHD and this may result in an increase in Body Mass Index (BMI). Treatment performed with Growth Hormone (GH) while accelerating growth velocity, might reduce BMI.

OBJECTIVE

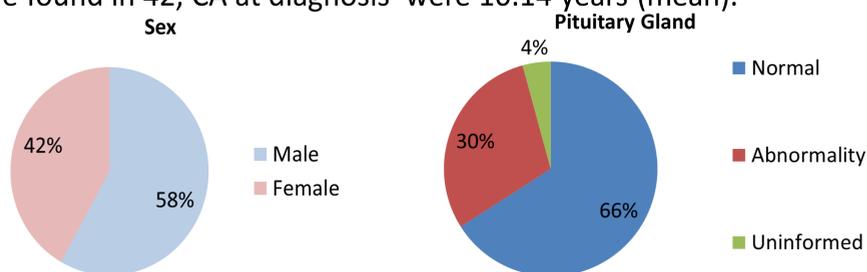
To evaluate BMI in patients with GHD at diagnosis, 1 year and 2 years after started treatment with GH and to compare if there is difference between the BMI of the patients with and without pituitary abnormalities.

METHODS

Were analyzed medical records of patients with GHD (sex, chronological age (CA) at diagnosis, height and weight to obtain BMI (BMI-SDS), 1 year and 2 years after started treatment of GH). Reports of MRI and/or CT scans of patients at age 3 to 16y among were analyzed since 1995 to 2016 at Pediatric Endocrinology Ambulatory at Regional University of Blumenau. BMI-SDS were classified: Accentuated lean: < -3 ; Lean: ≥ -3 to < -2 ; Eutrophy: ≥ -2 to $< +1$; risk overweight/overweight: $\geq +1$ to $< +2$; obesity: $\geq +2$. It was approved by the Ethics Committee.

RESULTS

141 patients were evaluated, 82 male. Pituitary abnormalities were found in 42, CA at diagnosis were 10.14 years (mean).



Findings of Pituitary morphology: pituitary hipoplasia (28), ectopic posterior pituitary (8), empty sell/complete agenesis(4), microadenoma(1), Rathke's cyst(1).

The accentuated lean group presented significant differences between BMI-SDS at diagnosis and the second year of treatment (-4.21 to -2.89, $p < 0.05$).

The lean group showed a significant difference between BMI-SDS at diagnosis and the first year of treatment (-2.4 to -1.94, $p < 0.05$) but not with the 2nd year of treatment(-1.84).

The patients with normal BMI-SDS at diagnosis remained normal at 2nd year of treatment.

In patients with overweight at diagnosis, the BMI-SDS decreased significantly in the 1st year (1.38 to 1.12, $p < 0.05$) and then it rose again (1.3) until 2nd year.

No differences were found in BMI-SDS when compared patients with and without pituitary abnormalities during 2 years of treatment.

Multiple hormonal deficiency were corrected when necessary.

BMI at diagnosis - classification	BMI at diagnosis	BMI on 1st year of treatment	BMI on 2nd year of treatment	P
Accentuated lean	(-4,21 + 0,79) ^a	(-3,61+0,42) ^{ab}	(-2,89+0,43) ^b	0,01832
Lean	(-2,4 + 0,26) ^a	(-1,94 + 0,58) ^b	(-1,84+0,7) ^b	0,00213
Eutrophy	(-0,43 + 0,71) ^a	(-0,46 + 0,75) ^a	(-0,4 + 0,48) ^a	0,09969
Overweight	(1,38 + 0,31) ^a	(1,12 + 0,54) ^b	(1,3 + 0,48) ^{ab}	0,02237
Obesity*	(2,25)	(2,16)	(2,32)	-

I: p-value of Friedman test. If $p < 0.05$, the differences are significative between the members. According to the multiple comparison test, differents letters means significative differences between the moments

II: * we could not perform the test because $n = 1$

CONCLUSION

BMI-SDS in patients with GHD after treatment, increased in patients with very low BMI, decreased in patients with high BMI and remained constant in eutrophic patients during treatment with GH. Although the gain in overweight BMI-SDS patients after the first year of treatment, should indicate necessity of feeding control during GH treatment in these patients. No differences were found in BMI-SDS when compared patients with and without pituitary abnormalities.

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