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Idiopathic central precocious puberty – treatment criteria

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INTRODUCTION

Central precocious puberty is due to premature activation of the hypothalamo-pituitary-ovarian axis. In girls it is idiopathic in up to 95%. Children with clinical rapid progression are treated with prolonged activity GnRH agonist.

PURPOSE

Characterize cases of idiopathic central precocious puberty (ICPP) followed at our hospital and to compare the group treated with GnRH agonist (group B) with the group not treated (group A) at about 6-12 months of follow-up.

MATERIAL AND METHODS

Retrospective study including children ICPP diagnosed between January/2006 and December/2013, with a minimum of 6 months' follow-up. Data collected included age, auxologic data and Tanner stage, at admission and along follow-up; target family height (TFH), parental pubertal age, growth velocity (GV), hormonal levels, bone age, predicted adult height (PAH), and treatment. Children with ICPP with criteria for treatment at first visit were excluded. Statistical analysis with SPSS 21st (p<0,05).

RESULTS

DEMOGRAPHIC CHARACTERIZATION

ICPP without treatment criteria at first visit

N = 42
All female
Mean follow-up time: 11 months (3-13M)

N = 17
Slow progression
Group A

N = 25
Rapid progression*
Group B

LHRH
(Triptorelin 11,25 mg IM every 12 weeks)
started 12,8 months after the first visit

	Group A (N=17)	Group B (N=25)	p
Age (years)	7,7 ± 0,7	7,5 ± 0,9	NS
TFH (cm)	158,3 ± 1,3	159,3 ± 1,1	NS

Mothers' menarche age was under 10 years in:
12.5% in group A
32% in group B

* Rapid progression was defined as an advance of at least two Tanner stages (breast or pubic hair) in a 6 month's period

COMPARING GROUPS: SLOW VS RAPID PROGRESSION

N=42	Group A (N=17)	Group B (N=25)	p	Group A (N=17)	Group B (N=25)	p
	At first visit			At 12 months' follow-up		
Heigth sds	0,92 ± 1,1	0,93 ± 0,9	NS	1,08 ± 1,3	1,31 ± 1,0	NS
Growth velocity (cm/year)	No data	No data	-	6,8 ± 2,3	8,8 ± 1,9	0,004
Bone age (years)	9,9 ± 1,4	9,2 ± 1,7	NS	No data	10,5 ± 1,3	-
PAH (cm)	157,8 ± 9,1	158,7 ± 7,4	NS	No data	155,9 ± 6,6	-
IGF1 (ng/mL)	313 ± 105	328 ± 98	NS	331 ± 116	410 ± 125	0,05
IGF1 sds	1,6 ± 1,1	2,1 ± 1,3	NS	1,3 ± 1,1	2,5 ± 1,4	0,005
FSH (UI/mL)	1,9 ± 1,3	2,5 ± 1,5	NS	1,8 ± 1,1	3,3 ± 1,6	0,001
LH (mUI/mL)	0,4 ± 1,0	0,4 ± 0,6	NS	0,4 ± 1,1	0,8 ± 0,7	NS
Estradiol (pg/mL)	25 ± 9	33 ± 38	NS	30 ± 14	29 ± 15	NS

EVOLUTION ALONG TIME

N = 42	Group A (N=17)		p	Group B (N=25)		p
	First visit	12 months		First visit	12 months	
Heigth sds	0,92 ± 1,1	1,08 ± 1,3	NS	0,93 ± 0,9	1,31 ± 1,0	<0,0001
Bone age (years)	9,9 ± 1,6	No data	-	9,0 ± 1,6	10,5 ± 1,3	<0,0001
PAH (cm)	157,8 ± 9,1	No data	-	157,9 ± 7,7	155,9 ± 6,6	0,019
IGF1 (ng/mL)	293 ± 71	322 ± 113	NS	327 ± 98	410 ± 126	0,003
FSH (UI/mL)	1,88 ± 1,3	1,81 ± 1,1	NS	2,53 ± 1,5	3,43 ± 1,6	0,006
LH (mUI/mL)	0,35 ± 1,0	0,44 ± 1,1	NS	0,37 ± 0,6	0,78 ± 0,7	0,004

DISCUSSION

At first visit, some children have clinical signs of puberty development but have no criteria to initiate treatment.

At first visit, there were no significant differences between groups.

At about 12 months of follow-up, the group with rapid progression of puberty had significantly higher growth velocity, FSH, IGF1 and IGF1 sds.

The group with rapid progression of puberty, along follow-up time, had significantly difference in height-sds, LH, FSH, IGF1 and bone age (all increased) and decrease predicted adult height.

Data that support rapid progression of puberty are increased height-sds, increased IGF1 e bone age advance. It would be important to have ecografic evaluation along time.

CONCLUSION

Monitoring precocious puberty should include height, growth velocity, bone age, gonadotropic hormones and IGF1, along at least 6-12 months. It is the combination of clinical progression and the evaluation of these data, that allows the decision to treat these children, in order to avoid compromise their final height.

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