Evaluation of the of Alpha2-Adrenergic Receptors Stimulation Effect on Prolactin Secretion, Based on the Result of the Test with Clonidine Used in the Diagnosis of Children with Short Stature

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INTRODUCTION

Prolactin (Prl) - secreting cells and growth hormone (GH) - secreting cells are derived from the common somatomammotropic cells. Prl secretion depends primarily on the inhibitory effects of dopamine and the stimulatory effects of TRH and estrogens. The effects of other factors, especially the stimulation of the adrenergic system, are not well recognized. It is known that presynaptic stimulation of the alpha2-adrenergic receptor results in suppression of noradrenaline secretion. It is presumed that at the same time, the secretion of Prl is inhibited, but this has not been fully explained. On the other hand, stimulation of the alpha2-receptor is widely used in the diagnosis of growth hormone deficiency (GHD) in children. Clonidine is the most commonly used factor. Clonidine, by stimulation of the presynaptic alpha2-adrenergic receptor, in addition to inhibiting the release of noradrenaline, simultaneously stimulates the secretion of somatoliberin (GHRH) from the hypothalamus, which in turn stimulates the synthesis of GH..

THE AIM

The aim of the study was to evaluate the effect of stimulating alpha2-adrenergic receptors after clonidine administration on Prl secretion, as well as to assess the differences in response to the abovementioned stimulation in groups of children with normal and decreased GH secretion (ISS and GHD groups).

MATERIAL AND METHODS

The serum concentrations of Prl and GH at individual time points during the 2-h stimulation test for GH secretion after oral clonidine administration at a dose of 0.15 mg/m2 were assessed. The test was carried out for diagnostic purposes in 49 children (mean age±SD: 9.55±3.48 yrs) with short stature (height <-2.0 SD). In 29 children ISS and in 20 - GHD were diagnosed.

RESULTS

In both GHD and ISS children, there was no significant reduction in Prl secretion after clonidine administration at any of the time points. Prl concentration decreased insignificantly at 30 minute after clonidine administration and then remained at the same level at subsequent time points. In both groups of children, no differences were found in the mean concentration of Prl at individual time points. There was no correlation between the concentration of Prl and GH during the test in GHD group, however, there was a significant positive correlation between the concentrations of both hormones at the 90. minute of the test in children with ISS.

CONCLUSIONS

Short-term adrenergic stimulation of alpha2-receptors (after oral clonidine administration) does not appear to affect Prl secretion in children with short stature in both the ISS and GHD groups.

Table 1. GH and Prl concentrations at indyvidaul time points during stimulation test after clonidine administration in ISS and GHD group.

	ISS group	GHD group
N=	29	20
Chronological age (years)	10.95±2.71	8.92±3.27
Height SDS	-2.26±0.95	-2.46±1.12
GH-0’ (ng/ml)	1.45±2.09	0.86±1.45
GH-30’(ng/ml)	2.86±2.96	1.23±1.80
GH-60’(ng/ml)	12.40±5.68	4.68±3.34
GH-90’(ng/ml)	10.79±4.11	4.52±1.96
GH-120’ (ng/ml)	6.21±3.16	2.66±1.61
PRL-0’ (ng/ml)	13.27±5.38	15.60±10.17
PRL-30’ (ng/ml)	9.68±4.11	10.69±5.96
PRL-60’ (ng/ml)	8.72±3.55	8.17±3.25
PRL-90’ (ng/ml)	10.09±5.24	8.04±3.42
PRL-120’ (ng/ml)	12.00±8.18	8.81±3.54

Figure 1. Correlation between Prl and GH concentration at 90’ of stimulation test after clonidine administration.