Evaluation of Clinical, Laboratory and Radiological Data of Obese and Non-Obese Girls Evaluated for Early Puberty

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INTRODUCTION and OBJECTIVES:

Although genetic factors are primarily responsible for the etiology of puberty, nutrition and environmental factors are also known to be effective. Obesity is associated with various metabolic complications and affects many aspects of pubertal development such as changes in pubertal hormones and onset of puberty. However, the relationships between these factors are still unclear. Some of studies have shown a correlation between early onset of puberty and increased prevalence of obesity in girls. It has been reported that overweight and obese children generally have advanced bone age with accelerated growth and sexual maturation. In addition, it has been reported that early puberty is associated with psychological problems, reproductive system cancers and increased risk of development of metabolic syndrome components in individuals' future lives.

In this study, we aimed to investigate whether there is a differences in terms of clinical, laboratory and radiological findings between obese and non-obese girls who presented with breast development and were examined for early puberty.

METHODS:

Fifty overweight and obese and fifty normal weight girls (between 3-8 years of age) who were admitted to the pediatric endocrine outpatient clinic for breast development and investigated for early puberty were included in the study.

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Cases with;

- Genetic or hormonal (hypothyroid, cushing, etc.) obesity,
- Chronic disease and drug use,
- Laboratory or radiologically diagnosed as central precocious puberty (CPP) due to pituitary or intracranial pathologies and peripheral precocious puberty due to liver, renal, thyroid, adrenal pathologies

were excluded from the study.

Chronological age (CA), body weight standard deviation score (SDS), height SDS, body mass index (BMI) SDS, bone age (BA), BA-CA difference (ΔBA-CA), basal and stimulated follicle stimulating hormone (FSH), luteinizing hormone (LH), estradiol (E2) and peak LH / FSH ratio were recorded. Uterine long diameter, uterine and ovarian volumes were evaluated by pelvic ultrasound.

LHRH test was performed in patients with;

- ΔBA-CA > 1 and/or
- Basal LH > 0,1 mIU/mL (ICMA), E2 > 12 pg/ml and/or
- Pubertal pelvic ultrasound findings (uterine volume> 2 ml / uterine long diameter > 34 mm, ovarian volüme > 2 cc.

CPP was diagnosed when the peak LH levels after gonadorelin stimulation was > 5.0 U/L.

RESULTS:

Height SDS and ΔBA-CA values of obese subjects were found to be statistically higher than normal weight controls (p = 0.001, p = 0.02 respectively).

Baseline FSH levels were higher in non-obese subjects than in obese subjects (p = 0.03).

There was no statistically difference between the two groups in LH-RH test responses. Eight (16%) of the obese and 13 (26%) of the non-obese subjects were diagnosed as CPP.

Clinical, Laboratory and Radiological Findings	Obese Group (N:50)	Control Group (N:50)	p
Chronological age (year)	7,2 (3,1-8)	7 (3,7-8)	0,8**
Weight SDS	2,2 ± 0,8	0,4 ± 0,6	0,000*
Height SDS	**1,4 ± 1,1**	**0,74 ± 0,9**	**0,001***
BMI SDS	1,9 ± 0,6	0,1 ± 0,7	0,000*
BMI percentiles	97 (85-99,9)	60 (8,9-84,7)	0,000**
Basal FSH (mIU/mL)	**1,58 (0,35-12)**	**2 (0,47-6,4)**	**0,03****
Basal LH (mIU/mL)	0,1 (0,1-1,8)	0,1 (0,1-0,6)	0,88**
Basal E2 (pg/mL)	5 (4,3-46,9)	5 (5-92)	0,67**
Bone age (year)	**8,5 (2,5-11)**	**7,8 (2,2-11)**	**0,05****
Uterine long diameter (mm)	31,6 ± 7,8	29 ± 6,4	0,1*
Uterine volume (ml)	7,5 (2,5-40,9)	7,3 (2,7-66,5)	0,96**
Right ovarian volume (ml)	1,3 (0,1-8,1)	1,4 (0,1-6,2)	0,85**
Left ovarian volume (ml)	1,3 (0,1-5,7)	1,3 (0,1-6,2)	0,95**
Stimulated peak LH level (mIU/mL)	2,94 (0,7-13,2)	2,95 (0,5-16,9)	0,35**
LH/FSH ratio	0,22 (0,08-1,4)	0,25 (0,03-1,5)	0,35**
ΔBA-CA	**1,34 (-1,75-3,2)**	**1 (-1,5-3,1)**	**0,02****

*Student T-test
**Mann-Whitney U test

LHRH Test Responses	Obese Group N(%)	Control Group N(%)	p
Pubertal	8 (%16)	13 (%26)	0,22
Prepubertal	42 (%84)	38 (%74)	

Chi-square test

CONCLUSION:

In our study, it was shown that height SDS and ΔBA-CA values were higher in obese subjects. Despite advanced bone age, rate of CPP diagnosis was low in the obese group.

In obese girls, underlying biological mechanisms such as compensatory hyperinsulinemia, insulin resistance, endocrine disruptors, and androgens may be contributing to bone age progression and pubertal characteristic changes. Consequently those can be misleading for CPP predicting.

Referances:

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