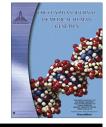


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ORIGINAL ARTICLE

Leptin, insulin like growth factor-1 and thyroid profile in a studied sample of Egyptian children with Down syndrome

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KEYWORDS

Leptin; IGF-I; TSH; FT4; Obesity;

Down syndrome

Abstract *Background:* Several mechanisms have been suggested for obesity in Down syndrome. *Aim of the study:* Assessment of serum levels of leptin, insulin like growth factor-I (IGF-I), thyroid stimulating hormone (TSH) and free thyroxin (FT4) in a prepubertal Egyptian sample of children with DS compared to their age and sex matched healthy controls and sibs of some of them.

Subjects and methods: A prospective case control study was conducted on 80 children, classified as follows: Groups I & II: enrolled 20 cases with DS for each, sibs were studied only for group I, Group III: 20 healthy siblings of group I, and Group IV: 20 healthy controls. Anthropometric measurements, serum leptin, IGF-1, TSH, and FT4 assessment using enzyme linked immuno-sorbent assay (ELISA) were carried out for all studied children.

Results: DS children whether with studied sibs or without studied sibs had significantly higher mean values of leptin levels compared to sibs of group I & IV (P = 0.0001 for all). Meanwhile, mean values of IGF-I showed statistically insignificant differences between all studied groups (p > 0.05 for all). Studied DS children whether with studied sibs or without studied sibs had significantly higher mean values of TSH levels compared to sibs of group I and controls (P = 0.0001 for all). Mean values of FT4 were significantly higher in enrolled DS without their studied sibs compared to sibs of group I (p = 0.01), while mean values of FT4 were significantly lower in sibs of group I compared to controls (p = 0.001).

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H.H. El Gebali et al.



Conclusion: Serum leptin levels were significantly higher in studied DS children compared to both studied sibs and healthy controls and they were also positively correlated with BMI in studied DS children and their sibs highlighting a possible role of body fat% and leptin values in the pathogenesis of obesity in DS children.

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1. Introduction

Down syndrome (DS) is the most common chromosomal anomaly among live-born infants, occurring at a frequency of one in 700 live births [1]. Individuals with DS are at increased risk for several endocrinological conditions, including hypothyroidism, growth retardation, diabetes mellitus, and obesity [2,3]. The reason for the increased risk of obesity in DS individuals is unclear, but several mechanisms have been suggested, including a decreased resting metabolic rate [4,5], and differences in physical activity patterns [6]. With the increase in the life expectancy of people with DS, obesity and related morbidity and mortality are emerging as important long term consequences [3]. Adipokines such as leptin have been implicated in the pathophysiology of obesity. Leptin is a hormone secreted by adipocytes, acting in the hypothalamus to suppress appetite and regulate body weight [7]. It is positively correlated with percentage of body fat; thus, it is postulated that obese individuals have some degree of leptin resistance [8]. It is unclear whether this mechanism is also at work in children with DS.

The current study was carried out in accordance to the code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans. This study was also done after taking consent of legal caregivers of enrolled patients and controls as well as the acceptance of the Ethics Committee of our University. It aimed at the assessment of serum levels of leptin, IGF-I (insulin like growth factor-I), and thyroid profile (TSH&FT4) in a sample of prepubertal Egyptian children with Down syndrome compared to their age and sex matched healthy controls and the sibs of some of them in order to investigate the hormonal mechanisms of obesity in DS children.

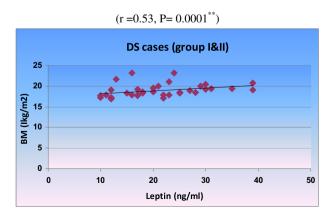


Figure 1 Statistically significant positive correlation recorded between serum leptin (ng/l) levels and BMI (kg/m²) of all studied DS cases (group I & II). $(r = 0.53, P = 0.0001^{**})$.

2. Subjects and methods

The present study was designed to be a prospective case control study that was conducted on 80 children. They were classified into four groups: Group I: included 20 cases with DS whose siblings were studied as well; 9 males (45%) and 11 females (55%). Their ages ranged from 3 to 10 years with a mean value of 6.4 \pm 2.3 years. Group II: included 20 cases with DS without studied siblings; 10 males (50%) and 10 females (50%). Their ages ranged from 3 to 11 years with a mean value of 7.1 ± 2.6 years. Children of both groups were recruited from the Special Needs Clinic, Ahmed Maher Teaching Hospital and Special Needs Unit, Institute of Postgraduate of Childhood Studies, Ain Shams University during the period of clinical part of the study from January 2011 to May 2011. Group III: included 20 healthy siblings of group I; 6 males (30%) and 14 females (70%). Their ages ranged from 5 to 11 years with a mean value of 9.06 \pm 1.7 years. Group IV: included 20 healthy control children; 10 males (50%) and 10 females (50%). Their ages ranged from 3.75 to 10.5 years with a mean value of 6.6 \pm 1.9 years. They were sibs of outpatients attending the Pediatrics Clinic, Ahmed Maher Teaching Hospital during the clinical period of study from January 2011 to May 2011.

Inclusion criterion for all studied children was a BMI between 5th to 95th percentile for age and sex. Exclusion criteria for DS cases (group I & II): Down syndrome associated with (1) cancer including leukemia, (2) congenital heart disease necessitating open heart surgery, (3) children with history of intestinal resection or (4) other chronic conditions affecting the growth or energy balance.

All children enrolled in the current study were subjected to the following: (I) full history taking: laying stress on age, sex, consanguinity, dietetic history, history of perinatal hazards,

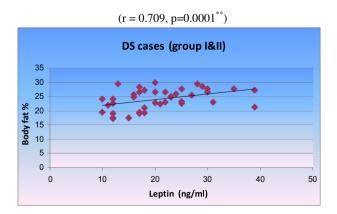


Figure 2 Statistically significant positive correlation recorded between serum leptin (ng/l) levels and body fat% of all DS cases (group I & II). $(r = 0.709, p = 0.0001^{**})$.

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