Original Research Article

Selenoprotein-p as Biomarker of Selenium Status in Obese Children and Adolescents

Abstract

Background: The child and adolescent obesity have become a major public health problem.Selenoprotien p_1 (SEPP1) is widely acknowledged to be among the most delicate functional indicators of Se statusand it plays a role in the metabolism of Se and in anti-oxidative defense. So, we aimed to assess Selenoprotein-p level in obese children and adolescents.

Methods: This cross-sectional comparativework included 60 children: 30 obese children and the control group consisted of 30 children who were healthy and matched in terms of gender and age. A comprehensive taking of history and clinical assessment with anthropometric measurements were conducted on all children and adolescents.Basic investigations and serum selenoprotien-p1 level were performed.

Results: Serum selenoprotien-p_1 (SEPP1) levels were substantially decreased in obese children contrasted to controls.

Conclusions: Selenoprotien p_1 levels were substantially decreased in obese children and adolescent. Selenoprotien p_1 could be a sensitive functional biomarker of selenium status.

Keywords: Obesity, Obese Children, Selenium status,Selenoprotien p_1

Background:

Obesity is a worldwide epidemic that is not restricted to adults. Unfortunately, child and adolescents' obesity had become a major public health issue[1]. It is a complex interaction among biological, developmental, behavioral, genetic, and environmental factors [2].

Obesity is strongly associated with multiple co-morbidities as insulin resistance (IR), dyslipidemia, cardiovascular disease, diabetes mellitus, hypertension, osteoarthritis, sleep apnea, and stroke [3].

Selenium, denoted by the chemical symbol (Se), is a crucial trace element that was first identified by the Swedish chemist Jons Jacob Berzelius in 1817[4]. Ithas antioxidant properties and protective role against oxidative damages [5]. The primary mechanisms by which Se exerts its physiological effects are through the actions of selenoproteins, which are proteins that contain Se in its natural state of selenocysteine. Seleno-proteins, including glutathione peroxidases and seleno-protein-p, have been first identified as antioxidants that facilitate the catalytic breakdown of organic hydroperoxides[6].

Selenoprotien p_1 (SEPP1) is widely regarded as among the most sensitive indicators of selenium levels. SEPP1 functions as a Se transporter to maintain antioxidative seleno-enzymes in multiple tissue, and it assumes an essential functionin the metabolism of Se and in anti-oxidative defense. So, a decrease in the expression of SEPP1 and seleno-proteins has been observed to give rise to a range of dysfunctions associated with oxidative stress[7].

Methods:

Aim: to assess the potential of Selenoprotein-p as a biomarker for evaluating Se levels in adolescents and kids with obesity.

Study design:

This cross-sectional Comparative work was performedbetween January 2022 to December 2022.

Study setting:

This work was performedinPediatrics Department, Tanta University Hospitals, Egypt.

Study participants:

Individuals in the work had been allocated into 2 groups: Group 1 consisted of 30 obese children according to definition of Centers for Disease Control and Prevention (CDC) with BMI $\geq 95^{th}$ percentile for age and sex, who were collected from clinical nutrition outpatient clinic and Group 2 consisted of A cohort of 30 children who were selected based on their health status, and matched for gender and age who constituted the control group.

Children with Syndromic obesity e.g.; Prader Willi, endocrinal causes of obesity such as Cushing's syndrome,history of drug intake as corticosteroids,history of chronic debilitating diseases, acute inflammatory processes, mental retardation, use of medication that affect; blood pressure, lipid profile, glucose level or causing weight gain and children under special diets or received any vitamins or dietary supplementations within the last 6 months before enrollment in the study; all were excluded from the work.All parents of the participants provided informed permission. The present work received ethical approval from the Ethical committee of the Faculty of Medicine at Tanta University, Egypt. Registration NO: (34888/9/21).

Study materials:

All children had been exposed to the follows:

A. Medical history review:

All participants provided a complete medical history, which included their demographic data, medical,Drug and Family histories, and age at diagnosis.

B. Physical examination:

The physical examination included a general condition assessment, system examination,measuring blood pressure. Anthropometric measures, such as height, weight, BMI, waist circumference (WC).The weight was recorded utilizing a Granzia digital weighing scale (PSTG-80, Italy), while the height was determined using a Germany anthropometer (Seca 216). The BMI was calculated as kg/ m^2.The National Centre for Health Statistics' matching growth charts were used to plot the weight, height, BMI measurements and to calculate Z-score**(Fryar et al., 2012)** and The circumference of the waist results have been displayed on a percentile curve for waist circumference. **(Sharma et al., 2015)**.

C. The following investigations were ordered:

i. **Initial investigations:** Full blood picture,laboratory tests conducted to assess the condition of the liver and kidneys, as well as the serum total lipid profile include measurements of [triglycerides, total cholesterol, HDL, and LDL].

ii. Selenoprotien-p using Enzyme Linked Immuno-Adsorbent Assay (ElISA) by DLdevelop ELISA kit.

Statistical analysis:

Data was collected using Microsoft Excel for Windows, and the statistical analysis was conducted employing SPSS v26 (IBM Inc., Armonk, NY, USA). Quantitative data was analyzed using the mean and standard deviation (SD), whilst qualitative data was presented using frequencies and percentages.

The statistical tests used for contrasting both groups included Fisher's exact test, the Chi-square test, the Mann-Whitney test, and/or the unpaired Student's t-test, as deemed appropriate. The statistical analysis used Spearman's rank-order correlation to examine the associations among numerical parameters. Statistical significance was set at $p < 0.05$.

Results:

This study included 60 children, 30 obese children and 30 healthy matched controls with no substantial variation among the two groups as regard to age, gender and residence.The percentage of female obese children was higher than that of male in our study. The demographic information of the groups under the studyand their anthropometric measurements are shown in **Table 1**.

The Obese children' weight, BMI, their z-scores and waist circumference were significantly higher than their controls, height (cm) didn't differ significantly between studied groups. However, the corresponding height-z scores had been substantially greater in children with obesitycontrasted to controls**.** Onset of obesity was higher in those aged below 5 years (56.7%) and obese children with class I obesity higher percentage (86.6%) than those with class II (6.66%) and class III (6.66%). Acanthosis nigricans was represented the higher percentage of clinical finding in obese children (73.35%)as shown in **Table 2**.

Serum triglycerides and total cholesterol had been substantially greater among obese children contrasted to controls;however, no substantial variation was existed in concentrations of HDL, LDL, CBC, Liver and renal function tests between both groups.In contrast to the control group, theSerum SEPP1 was substantially lower in obese children as summarized in **Table 3**.Also, asubstantial negative association was existed among SEPP1 and waist circumference in obese children.

Discussion

Child and adolescents'obesity has emerged as an important threat to public health;The modulation of inflammation and oxidative stress was hypothesized, while the micro-nutrient Se has been reported to possess qualities that are anti-inflammatory and anti-oxidative in nature[8].

SEPP1 is Se that contains protein,it is well recognized that this particular functional marker has significant importance in assessing selenium (Se) status. This marker primarily serves as a means of

transporting Se, ensuring its delivery to cells and tissues. Furthermore, it plays a crucial role in the metabolism of Se and serves as a key component of the antioxidative defense system[7].

In this regard we evaluate levels of circulation selenoprotienp_1 in obese children and adolescents .

As regard to gender in our study, about 66.7% (20) of obese children were female and 33.3% (10) males with Female: male ratio = 2:1. Similarly,Allam and Hamed et al.[9, 10]whileEl-Shafie et al.[11]and Salah et al.[12]studies showed that prevalence of obesity higher in boys than girls.

Numerous studies have emphasized the need of considering a familial background of obesity, DM, or hypertension when evaluating the health risks associated with obesity among children[13]**.** In the present work, a substantial variation was existed among participants and controls as regard to family history of obesity (p= <0.001*).

Regarding onset of obesity, our study revealed that obesity is higher prevalence among children less than 5 years,and this agreed withOsei Bonsu and Addo et al.[14]studythat showed higher prevalence of obese children below 5 years in Egypt, that may associated with over-nutrient**.**Our study also showed, higher rate of class I obesity between obese children than class II and III, and this agreed with Twig et al.[15]**.**

In terms ofto blood pressure, our work showed about 86.6% of obese children (n=26) were with normal BP, 10% of them pre hypertensive (90^{th} - <95^{th} centile for gender and age) (n=3) and 3.4% of them were hypertensive (95^{th} centile for gender and age) (n=1) and this agreed with Abd-Elghaffar et al.,[16]who discovered (8.9%) possess hypertension.

Our work revealed that no substantial variation was existed among obesity in children and controls in SBP and DBP (mmHg) and this agreed withMohamed et al.,[17]study. While Tanaka et al.[18]discovered that obesity in children substantiallygreater in diastolic and systolic blood pressure contrasted to their controls.

In our study, weight, BMI, and with their corresponding height z-scores, z-scores, waist circumference and waist circumference centile were substantially greater in children with obesity contrasted to controls ($p<0.001$). This result was in line withGajewska et al.,[19]and López-Peralta et al.,[20]thatdemonstrated significantly higher weight, height, BMI and BMI Z -scores in obese children than controls and they explained that the rapid weight gain in early childhood is often accompanied by an increase in height velocity and advanced bone age**.**

On other hand Mohammed et al.,[21] studiesagreed with our results as regard to weight, BMI and waist circumference, but as regard to height they found no significant difference between obese children and normal weight ($p=0.79$).

As we know acanthosis nigricans is pathognomonic for insulin resistance, we discovered that 22 out of 30 instances (73.33%) showed acanthosis nigricans and this agreed with Sudevan et al.,[22]who showed acanthosis nigricans had been observed more in children with obesity (61.5%). Also, Cardenas-Vargas et al.,[23]found that acanthosis nigricans was detected in (85.9%) of the children with obesity.

As regards CBC in our study, no significant difference of CBC datawas reported between cases and controls and our findings were consistent with Gajewska et al., [24]. However, Emam et al.[25] found that hemoglobin concentrations in children with obesity had been substantially decreased contrasted to were in healthy controls.

The liver seems to be significantly impacted by fat deposition in the presence of obesity. In our work, no substantial variation was existed among obese children and controls in ALT ($p=0.740$) and AST ($p=0.734$) and this agreed with Das et al., [26] and disagreed with the study of Mărginean et al., (2019) who revealed that liver enzymes concentrationshad been significant greater in children with obesity contrasted to control ($p<0.001$), ($p=0.03$).

Blood urea and serum creatinine were within normal range with no substantial variations among obese children and controls (p=0.141), (p=0.354) and this comes in line with the study of Maćkowiak-Lewandowicz et al.[27]. In contrary Khan et al.,[28] study revealed that blood urea, serum creatinine was substantially elevated in obese group as contrasted to controls i.e. ($p < 0.01$).

As regard to lipid profile, our study showed that total serum cholesterol and triglycrides (TG) levels had been substantially greater in obese children contrasted to controls (p=0.043), (p<0.001). while LDL and HDL didn't show substantial variation among both groups(p=0.154), (p=0.075) and this is agreed with Juliaty and Kurniasih**.**[29]who foundchildren with obesityhad substantially greater TG and cholesterol, in addition to decreased HDL contrasted to normal weight participants. While LDL concentrationshadn't been substantial various among groups. However, Maćkowiak-Lewandowicz et al.,[30]found that obese and control groups didn't vary in serum levels of total cholesterol and LDL. While substantially greater levels of HDL and TG ($p = 0.001$, $p = 0.001$).Ibrahim et al. [31]studyreported that no substantial variation in lipid profile among obese children and controls. While Mohammed et al.,[21]study found that LDL and serum TC had beensubstantially greater among children with obesityas contrasted to control. Nevertheless, HDL had beensubstantially lowered among children with obesity as contrasted to normal-weight one.

There is an elevated frequency of aberrant lipid levels among children and adolescents who are classified as overweight or obese. The prevailing form of dyslipidemia observed in these individuals is characterized by the coexistence of hypertriglyceridemia alongside decreased levels of HDL. This dyslipidemic profile is mostly attributed to underlying insulin resistance[32].

Our study revealed that selenoprotien-p_1 (SEPP1) is substantially decreased among children with obesity contrasted to controls (p<0.001) and this in line with Ko et al.[33] study who revealed that Selenoprotien-$p_1$concentrations had been decreased in obese children.Also, Sargeant et al.[34]study found that obese (2.81±0.3) had lower SEPP than normal weight (3.01±0.39). Gharipour et

al.[35]and Di Giuseppe et al.[36]studies reported that SEPP was inversely relation with BMI. While our results were disagreeing with Chen et al.[37]and El-Kafrawy et al.[38]studies who revealed that selenoprotein-p level higher in obese than their controls but there were performed on adult. Soares de Oliveira et al.,[39]found that Excessive adiposity might potentially manifest as a pro-oxidative and pro-inflammatory condition, resulting in an increased demand for selenium and leading to sustained alterations in Se levels over time. Also, our study demonstrated a statistically substantial negative association among serum level of selenoprotien p_1 and waist circumference in obese group and this is agreed with Ko et al.[33]as showenin Figure 1.

We faced some limitationsas the tiny number of subjects participated in our work that also, interfered with subgroup comparison in studied groups.

Conclusions:

Selenoprotien p_1 levels had been substantially decreased in children with obestyand adolescent contrasted to their controls.Selenoprotien p_1 could be a sensitive functional biomarker of selenium status.

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Table 1: The Demographic Data and Anthropometric Measurements of the Studied Groups.

		Controls (N = 30)	Obese Children (N = 30)	test statistic	p-value
Age of Enrolment (years)	**Mean ± SD.** **Min – Max**	10.07 ± 2.54 (6.00 to 15.00)	10.27 ± 2.59 (6.50 to 16.00)	t = 0.302	0.764
Sex	**Female**	14 (46.7%)	20 (66.7%)	X^2_{ChS} = 2.443	0.118
	Male	16 (53.3%)	10 (33.3%)		
Residence	**Rural**	18 (60.0%)	14 (46.7%)	X^2_{ChS} = 1.071	0.301
	Urban	12 (40.0%)	16 (53.3%)		
Family History	**Diabetes mellitus**	7 (23.3%)	12 (40.0%)	X^2_{ChS} = 1.926	0.165
	Hypertension	5 (16.7%)	11 (36.7%)	X^2_{ChS} = 3.068	0.080
	Obesity	1 (3.3%)	16 (53.3%)	X^2_{ChS} = 18.468	<0.001*
Weight (Kg)	**Mean ± SD** **Min – Max**	36.06 ± 12.04 (20.30 to 64.80)	61.04 ± 18.80 (35.20 to 110.60)	t = 6.130	<0.001*
Weight Z score (SD)	**Median [IQR]** **Min – Max**	0.43 [-0.37 to 0.97] (-1.52 to 1.45)	2.29 [1.94 to 2.54] (1.34 to 3.20)	Z = 6.640	<0.001*
Height (cm)	**Mean ± SD** **Min – Max**	138.28 ± 14.81 (117.50 to 169.70)	143.05 ± 14.34 (116.30 to 168.30)	t = 1.267	0.210
Height Z score (SD)	**Median [IQR]** **Min – Max**	-0.16 [-0.73 to 0.63] (-1.30 to 1.20)	0.40 [-0.33 to 1.20] (-1.90 to 1.80)	Z = 2.506	0.012*
BMI (Kg/m^2)	**Mean ± SD** **Min – Max**	18.41 ± 2.66 (13.40 to 23.40)	29.18 ± 5.38 (21.20 to 43.50)	t = 9.828	<0.001*
BMI Z score (SD)	**Median [IQR]** **Min – Max**	0.70 [-0.01 to 1.08] (-1.50 to 1.53)	2.30 [2.10 to 2.64] (2.08 to 3.13)	Z = 6.669	<0.001*
Waist circumference (cm)	**Mean ± SD** **Min – Max**	59.17 ± 4.47 48.00 - 67.00	87.57 ± 10.15 60.00 - 104.00	t =14.025	<0.001*

Waist circumference centile	**Median [IQR]** **Min – Max**	35.00 [25.00 -50.00] 5.00 – 60.00	96.00 [95.00 – 97.00] 75.00 – 99.00	Z = 6.69	<0.001*

IQR: interquartile range; Max: maximum; Min: minimum; SD: standard deviation; t: independent samples t-test; Z: Mann-Whitney test;FE: Fisher's exact test; NA: non-applicable; X^2_{ChS}: Pearson's Chi-square test for independence of observations;* significant at $p<0.05$

Table 2: Obesity Related Data

		N	%
Age of Onset	**< 5 years**	17	56.7%
	≥ 5 years	13	43.3%
Class of Obesity	**Class I**	26	86.6%
	Class II	2	6.66%
	Class III	2	6.66%
Clinical Finding	**Acanthosis Nigricans**	22	73.3%
	Abdominal Enlargement / Striae	3	10.0%
	Acne and Hirsutism	3	10.0%
Blood pressure	**Normal BP**	26	86.6%
	Pre-hypertension	3	10%
	Stage I hypertension	1	3.4%

N: number

Table 3: The laboratory investigations of the studied groups

		Control (N = 30)	Cases (N = 30)	test statistic	p-value
Hb (gm/dl)	Mean ± SD Min – Max	11.18 ± 0.96 9.50 to 13.00	11.35 ± 0.92 9.00 to 13.00	t = 0.671	0.505
PLT	Mean ± SD Min – Max	307.67 ± 98.29 153.00 to 478.00	311.57 ± 92.85 170.00 to 489.00	t = 0.158	0.875
TLC	Mean ± SD Min – Max	6.73 ± 2.08 3.00 to 10.30	7.79 ± 2.76 3.60 to 13.90	t = 1.682	0.098
AlT (U/L)	Mean ± SD Min – Max	24.67 ± 3.27 18.00 to 31.00	24.13 ± 8.13 14.00 to 61.00	t = 0.333	0.740
AST (U/L)	Mean ± SD Min – Max	22.33 ± 4.24 14.00 to 29.00	22.80 ± 6.17 12.00 to 46.00	t = 0.342	0.734
Urea (mg/dl)	Mean ± SD Min – Max	25.03 ± 4.53 17.00 - 36.00	23.47 ± 3.53 17.00 - 30.00	t = 1.494	0.141
Creat (mg/dl)	Mean ± SD Min – Max	0.58 ± 0.19 0.20 to 0.90	0.53 ± 0.20 0.20 to 0.90	t = 0.933	0.354
Total Cholesterol (mg/dl)	Mean ± SD Min – Max	148.53 ± 16.98 110.00 to 189.00	160.80 ± 27.54 103.00 to 221.00	t = 2.077	0.043*
HDL (mg/dl)	Mean ± SD Min – Max	45.03 ± 4.94 37.00 to 55.00	48.97 ± 10.70 35.00 to 65.00	t = 1.828	0.075
LDL (mg/dl)	Mean ± SD Min – Max	91.87 ± 12.26 70.00 to 114.00	99.13 ± 24.48 50.00 to 156.00	t = 1.452	0.154
Triglycerides (mg/dl)	Mean ± SD Min – Max	77.47 ± 18.40 43.00 to 110.00	104.77 ± 26.08 65.00 to 160.00	t = 4.685	<0.001*
SEPP (ng/dl)	Median [IQR] Min – Max	70.35 [65.89 to 77.42] 60.10 to 80.91	7.60 [5.82 to 13.99] 3.36 to 56.69	Z = 6.653	<0.001*

Z: Mann-Whitney test;Max: maximum; Min: minimum; SD: standard deviation; t: independent samples t-test; * significant at $p<0.05$.

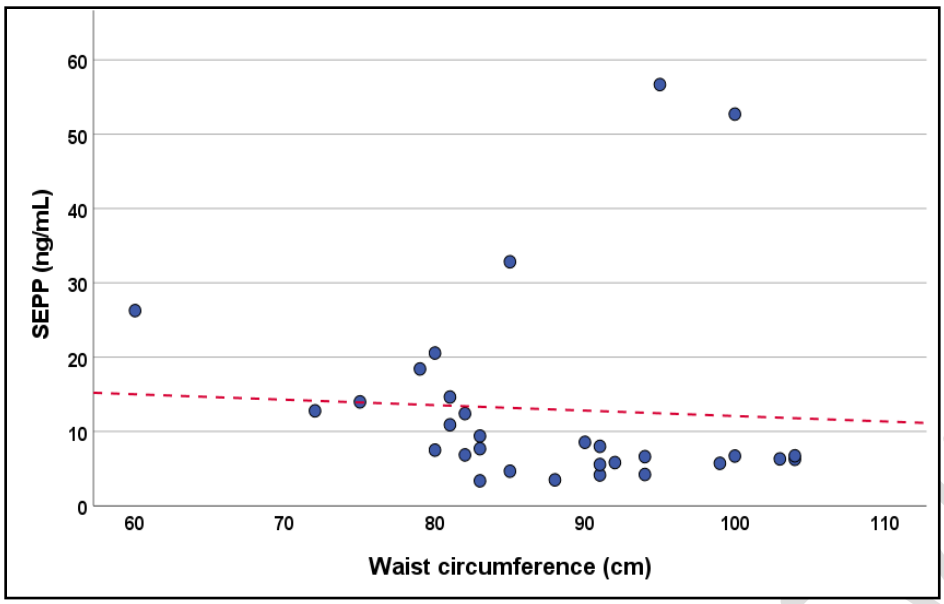

Figure 1: Correlation Between Selenoprotien P1 and Waist Circumference in Obese Group