

# EVALUATION OF MUSCULOSKELETAL PAIN, SERUM VISFATIN, LEPTIN LEVEL AND BLOOD PRESSURE IN OBESE CHILD AND NON OBESE CHILD

By

**Hegazy Mogahed Altamimy\* ; Mahmoud Farag Salem\*\* ;  
Albakry Mohamed Tharwat Albakry\*\*\* and Ayman Alsaeed Sadek\*\*\*\***

Departments of Rheumatology\* , Clinical Pathology\*\* , Pediatric\*\*\* , Cardiology \*\*\*\* ,  
Al-Azhar Faculty of Medicine (Cairo and Damietta)

## ABSTRACT

**Background:** The increasing prevalence of obesity is becoming an important public health problem in childhood and presents numerous problems. Similarly to the risks of obesity in adulthood, childhood obesity is also a leading cause of pediatric hypertension associated with type 2 diabetes mellitus, and increases the risk of cardiovascular diseases.

**Objective :** This study was designed to compare the anthropometric measurements (height, weight, BMI, waist circumference) systolic and diastolic blood pressure, lipid profile (T.C, TG, HDL-C, LDL-C), fasting blood glucose, leptin level , visfatin level and correlation between musculoskeletal pain and weight, waist, BMI ,leptin an Visfatin level within obese children and non obese children.

**Patients and methods** The study included two groups. Group (1) thirty age and sex-matched non-obese children were included as the control group. Group (2) sixty healthy obese children aged 11–15 years. The case-control observational study was evaluated from December 2013 to December 2014 with informed consent. A careful history and physical examination included anthropometric measurements in all subjects. Body weight, height and waist circumference were measured, body mass index (BMI) was calculated (kg/m) as an index of overall adiposity. Children with BMI over their age- and sex-specific 95th percentile values were defined as obese children, and those with BMI <85 percentiles were considered non-obese . Joint site model was used in order to account for correlations between musculoskeletal pain and weight, waist, BMI, leptin an Visfatin level within obese children and non obese children.

**Results:** The obese children showed higher significant difference in fasting blood glucose, TG, LDL-C, T.C, diastolic and systolic blood pressure and they had lower HDL-C compared with non obese children. The study showed that no significant correlations between anthropometric indices (weight, height, BMI, and waist circumference), age ,systolic and diastolic blood pressure with visfatin in the obese group . There was no significant correlations were found between leptin, fasting blood sugar, total cholesterol, HDL-C and LDL-C with visfatin in the obese group , however positive correlation was found between visfatin and Triglycerides . There was no significant correlations between anthropometric indices , systolic, diastolic blood pressure and age with leptin in the obese group . There was no significant correlations were found between FBS, Total cholesterol, Triglycerides, HDL-C, and LDL-C with leptin in the obese group Significant correlations of hip pain, knee pain, feet pain and back pain with weight, waist and BMI were found .

**Conclusion:** Obesity has a significant impact on the health and well-being of these children and may contribute to ongoing health problems such as musculoskeletal pain and bone/joint dysfunction in later life.

**Keywords:** Musculoskeletal pain, leptin,visfatin, lipid profile, obese child and non-Obese.

## INTRODUCTION

Associated systems affected by obesity in childhood include the cardiovascular, metabolic, gastrointestinal, pulmonary, and skeletal as well as psychosocial functioning (*Daniels, 2009*). Although physical pain is common in obese adults, and is recognized as a comorbidity of obesity in adults (*Hitt et al., 2007*). It is typically not recognized as such within pediatric obesity. This is unfortunate because among other negative implications, pain can act as an independent barrier to physical activity (*Long et al., 2008*), a key intervention goal (*Janssen et al., 2005*). In our efforts to develop appropriate treatment interventions for pediatric obesity, it is critical that we understand the pain experiences of obese children.

Chronic nonspecific musculoskeletal pain in children and adolescents has been reported as a common occurrence. Children affected by chronic musculoskeletal pain have been found to have increased levels of anxiety and depression, as well as lower levels of activity. Recent reviews on general musculoskeletal pain identified the most frequent site of nonspecific musculoskeletal pain in children in the lower limbs, with some estimates suggesting that this affects 24% of children aged between 6 and 10 years of age (*Smith et al., 2014*).

Visfatin is an insulin-mimetic adipokine that was originally discovered in liver, skeletal muscle and bone marrow as a growth factor for B lymphocyte precursors. Circulating visfatin levels are closely correlated with fat accumulation,

Visfatin mRNA levels increase in the course of adipocyte differentiation, Visfatin synthesis is regulated by several factors including glucocorticoids, TNF, IL-6 and growth hormone. Recently, PBEF was identified as visfatin a novel adipokine - a protein mediator secreted by fat cells (high levels of expression in visceral fat cells (*Francisca et al., 2007*).

Leptin is also secreted by adipocytes and immune cells in response to increasing levels of systemic inflammation. The effects of leptin on immunity are complex and less well understood. Leptin secretion appears to potentiate the activity of both innate immune cells (neutrophils, monocytes/macrophages, natural killer cells) and adaptive immune cells (T-helper 1 cells). By potentiating the immune response and modulating cytokine secretion, leptin may promote effective resolution of the acute inflammatory response (*Danese et al., 2014*).

This study was designed to compare the anthropometric measurements (height, weight, BMI, waist circumference), systolic, diastolic blood pressure, lipid profile (T.C, TG, HDL-C, LDL-C), fasting blood glucose (FBS), leptin level, visfatin level and musculoskeletal pain between obese children with non obese children.

## PATIENTS AND METHODS

The present study contained two groups. Group (1) thirty age and sex-matched non-obese children and group (2) sixty healthy obese children aged 11–15 years. The case-control observational study was evaluated at Pediatric, Rheumatology, Cardiology, Clinical Pathology

Departments, Al-Azhar University Hospitals (Cairo and Damietta, Egypt) from December 2013 to December 2014. The study was approved by the Ethics Committee of our hospital. All patient parents gave their informed consent before the beginning of the study.

All obese children included in the study were diagnosed as having simple obesity by calculating BMI and cross BMI with age on growth chart, without additional diseases such as diabetes mellitus, hypertension and hypothyroidism. Children with secondary obesity were excluded from the study.

A careful history and physical examination including anthropometric measurements were obtained in all subjects. Body weight, height and waist circumference were measured. Body mass index (BMI) was calculated ( $\text{kg}/\text{m}^2$ ) as an index of overall adiposity. Height and weight were determined using precision stadiometers and scales to the nearest 0.1 cm and 0.1 kg respectively. The waist circumference was measured midway between the inferior margin of the last rib and the crest of the ileum in a horizontal plane. Circumferences were measured to the nearest 1 mm. Children with BMI over their age- and sex-specific 95th percentile values were defined as obese children, and those with BMI <85 percentiles were considered non-obese. Children with a BMI between the 85th and 95th percentiles were defined as overweight and excluded from the study. Systolic and diastolic (phase V) BP were taken two times 2 min apart with a random zero sphygmomanometer after the

subject had been sitting for at least 10 min. The average of the first and the second readings was recorded. Hypertension was defined as BP 140 and/or 90 mmHg or current antihypertensive drug treatment. Joint site model was used in order to account for correlations between musculoskeletal pain and weight, waist, BMI, leptin and Visfatin level within obese children and non obese children ( *Stovitz et al., 2008*). The level of Visfatin ( *Nusken et al., 2007*) and leptin ( *Considine et al., 1996*) were tested.

**Statistical analyses:** Data were analyzed by SPSS 18 (Social package of statistical sciences computer program). Data were checked for normal distribution and the results were expressed as mean  $\pm$  SD for normal distribution variable. Data that have be normal distribution, logarithm were taken for convert them to normal distribution such as visfatin. Difference in variables that were normally distributed were tested using independent t-test. Pearson's correlation analysis was performed to evaluate the relationships between metabolic, hormonal, anthropometric parameters and musculoskeletal pain. The significant difference were indicated if P value <0.05.

## RESULTS

The mean age of obese was  $12.7 \pm 0.7$  years which ranged from 11-14 years and non-obese children was  $12.6 \pm 0.6$  years which ranged from 12-14 years. The mean level of weight in obese was  $71 \pm 6.5$  kg which ranged from 55-86 kg, while control was  $31.8 \pm 5.2$  and ranged from

23-43 kg. There was a significant difference between them. The mean level of height (cm) in obese was  $157 \pm 5.6$  which ranged from 141-166, while control was  $142 \pm 19.5$  and ranged from 124-150. There was a significant difference between them. The mean level of BMI ( $\text{kg}/\text{m}^2$ ) of obese was  $28.9 \pm 2.6$  which ranged from 22.8-37.9, while control was  $15.8 \pm 1.6$  ranged from 13-19.5. There was a significant difference between them. The mean level of waist circumference (cm) in obese was  $64 \pm 4$  which ranged from 48-84, while control was  $46.6 \pm 2.4$  and ranged from 32-50. There was a significant difference between them. The

mean level of systolic blood pressure (mm Hg) in obese was  $107 \pm 8.7$  and ranged from 90-120, while control was  $97.6 \pm 6.7$  and ranged from 90-110, in which there was a significant difference between them. The mean level of diastolic blood pressure in obese was  $68.6 \pm 3.3$  mm Hg which ranged from 60-75, while control group was  $66.8 \pm 4$  and ranged from 60-70. There was a significant difference between them.

Our results revealed that the mean of an anthropometric indices and demographic data of obese children had higher significant difference than control healthy subjects (Table 1).

**Table (1):** Anthropometric indices in obese and control children (mean $\pm$  SD).

Parameters \ Groups	Control group (n = 30)	Obese group (n = 60)	p value
Age (years)	12.6 $\pm$ 0.6	12.7 $\pm$ 0.7	0.4
Weight (kg)	31.8 $\pm$ 5.2	71 $\pm$ 6.5	<0.001
Height (cm)	142 $\pm$ 19.5	157 $\pm$ 5.6	<0.001
BMI ( $\text{kg}/\text{m}^2$ )	15.8 $\pm$ 1.6	28.9 $\pm$ 2.6	<0.001
Waist circumference (cm)	46.6 $\pm$ 2.4	64 $\pm$ 4	<0.001
SBP (mmHg)	97.6 $\pm$ 6.7	107 $\pm$ 8.7	<0.001
DBP (mmHg)	66.8 $\pm$ 4	68.6 $\pm$ 3.3	0.04

Table (2) showed that the mean level of visfatin (ng/ml) of obese was  $1.9 \pm 1.8$ , while control was  $1 \pm 0.3$  with a significant difference between them. The mean level of Leptin (ng/ml) of obese

was  $13.6 \pm 8.4$  which ranged from 3.1-42.9, while control was  $4.5 \pm 1$  ranged from 3-7.5. There was a significant difference between them. The mean level of fasting blood glucose (mg/dl) in obese

was  $108 \pm 23.3$  which ranged from 84-125, while control was  $84 \pm 7.2$  which ranged from 75-100. There was a significant difference between them. The mean level of total cholesterol (mg/dl) in obese was  $145 \pm 48$  which ranged from 39-180, while control was  $105 \pm 12$  which ranged from 85-130. There was a significant difference between them. The mean level of triglycerides (mg/dl) of obese was  $104 \pm 57$  which ranged from 50-414, while control was  $76 \pm 16.9$  which ranged from 51-104. There was a significant difference

between them. The mean level of high-density lipoproteins cholesterol (mg/dl) in obese was  $35 \pm 7.4$  which ranged from 24-58, while control was  $49.8 \pm 6.7$  which ranged from 36-66. There was a significant difference between them. The mean level of low-density lipoproteins cholesterol (mg/dl) in obese was  $90 \pm 45$  which ranged from 32-356, while control was  $40 \pm 13.5$  which ranged from 10-70. There was a significant difference between them.

**Table (2):** Laboratory values in obese children and the control group(mean± SD).

Parameters	Control group (n = 30)	Obese group (n = 60)	p value
Visfatin (ng/ml)	$1 \pm 0.3$	$1.9 \pm 1.8$	0.01
Leptin (ng/ml)	$4.5 \pm 1$	$13.6 \pm 8.4$	<0.001
FBG (mg/dl)	$84 \pm 7.2$	$108 \pm 23.3$	<0.001
Total cholesterol (mg/dl)	$105 \pm 12$	$145 \pm 48$	<0.001
Triglycerides (mg/dl)	$76 \pm 16.9$	$104 \pm 57$	0.01
HDL-C (mg/dl)	$49.8 \pm 6.7$	$35 \pm 7.4$	<0.001
LDL-C (mg/dl)	$40 \pm 13.5$	$90 \pm 45$	<0.001

There were no significant correlations found between anthropometric indices (weight, height, BMI, and waist circumference), age, systolic and diastolic blood pressure, leptin, FBS, total cholesterol, HDL-C and LDL-C with visfatin in the obese group. However, positive correlation was found between

visfatin and Triglycerides. There were no significant correlations found between anthropometric indices (weight, height, BMI, and waist circumference), visfatin, systolic, diastolic blood pressure, age, FBS, total cholesterol, triglycerides, HDL-C, and LDL-C with leptin in the obese group (Table 3).

**Table (3): Pearson's correlation between age, systolic and diastolic blood pressures, anthropometric, FBS and lipid parameters with Visfatin and leptin in obese group.**

Parameters	Pearson Correlation	Visfatin	Leptin
	<b>SBP</b>	Pearson Correlation	.188
Sig. (2-tailed)		.159	.445
<b>DBP</b>	Pearson Correlation	.038	.238
	Sig. (2-tailed)	.777	.072
<b>Waist</b>	Pearson Correlation	-.156	.065
	Sig. (2-tailed)	.242	.630
<b>BMI</b>	Pearson Correlation	-.134	.101
	Sig. (2-tailed)	.317	.450
<b>Weight</b>	Pearson Correlation	-.116	.093
	Sig. (2-tailed)	.387	.488
<b>High</b>	Pearson Correlation	.013	.235
	Sig. (2-tailed)	.924	.075
<b>Age</b>	Pearson Correlation	-.050	.039
	Sig. (2-tailed)	.709	.771
<b>Leptin</b>	Pearson Correlation	.005	00
	Sig. (2-tailed)	.971	00
<b>LDL-C</b>	Pearson Correlation	-.086	-.025
	Sig. (2-tailed)	.520	.853
<b>HDL-C</b>	Pearson Correlation	.047	-.072
	Sig. (2-tailed)	.728	.591
<b>TG</b>	Pearson Correlation	.272*	.044
	Sig. (2-tailed)	.039	.740
<b>Cholesterol</b>	Pearson Correlation	.050	-.065
	Sig. (2-tailed)	.710	.630
<b>FBS</b>	Pearson Correlation	.093	.069
	Sig. (2-tailed)	.489	.604

\* Significant

In obese child, back pain was the most common complaint (43.3%), followed by hip pain(36.6%), knee pain (35%) and foot pain (31.6%) (Table 4).

**Table (4):** Numbers of hip pain, knee pain, feet pain and back pain in obese and non-obese child.

Parameters Groups	Back pain	Hip pain	Knee pain	Foot pain
<b>Obese child</b>	26 (43.3%)	22(36,6%)	21 (35%)	19 (31.6%)
<b>Non obese child</b>	7 (23.3%)	5 (16.6%)	6 (20 %)	5 (16.6%)

There were significant correlations between hip pain and knee pain with weight, waist and BMI (Table 5)

**Table (5):** Correlations between hip pain and knee pain with weight, waist and BMI.

Parameters Pain	Weight	BMI	Waist
Hip pain	0.001	0.000	0.001
Knee pain	0.000	0.000	0.003
Feet Pain	0.735	0.707	0.829
Back pain	0.261	0.357	0.091

There were no significant correlations between hip pain, knee pain, feet pain and back pain with leptin and visfatin (Table 6).

**Table (6):** Correlations between hip pain, knee pain, feet pain and back pain with leptin and visfatin.

Parameters Pain	Leptin	Visfatin
Hip pain	0.040	0.193
Knee pain	0.036	0.175
Feet Pain	0.254	0.877
Back pain	0.380	0.770

## DISCUSSION

Because visceral adipose tissue is considered an important source of visfatin, studies on visfatin alterations in children may be useful in understanding some complications of obesity. Obese children often display increased linear growth, the body heights of obese children in this study were found to be significantly higher than those of non-obese children this is in consistent with *Bouhour et al. (2007)*.

Our study revealed that there was significant difference in serum visfatin level between obese children and non obese children. This was in agreement with *Berndt et al. (2005)* who had demonstrated that serum visfatin level elevated in obese children. *Fukuhara et al. (2005)* suggested that visfatin secreted from visceral adipose tissue and serum visfatin level were found to be significantly correlated with visceral fat tissue in adults. The elevation of serum visfatin in obese adolescent may be attributed to the increase in adipose tissue mass. This may emphasis the role of adipose tissue as an important source of visfatin production (*Moschen et al., 2010*). Serum visfatin levels are significantly associated with obesity as defined by the modified Adult Treatment Panel III guidelines. Studies performed in a relatively large population of subject strong correlation between serum visfatin and obesity which is derived mainly from visceral but not subcutaneous fat (*Sandeep et al., 2007*).

No correlations was found between serum visfatin level and weight, BMI, and waist circumference in obese children of our study. This was in agreement with the

studies of *Berndt et al. (2005)*, *Dominik et al. (2006)* and *Haider et al. (2006)*. *Jian et al. (2006)* showed a negative correlation between visfatin level and BMI in a Chinese obese adult population. The reasons for these conflicting results may be ethnic heterogeneity, different population characteristics (children, men, women) and confounding factors such as gender.

Our work showed significant higher levels of TC, TG, LDL-C and triglycerides (except HDL-C) in the obese children compared with control group. This was in agreement with study of *Mehmet et al. (2009)*. A positive correlation for TG was found in obese children, which was similar to the result of *Haider et al. (2006)*, who did not observe a correlation between serum concentrations of visfatin and lipids in obese children. However, *Mehmet et al. (2009)* reported a positive correlation between plasma concentrations of visfatin and HDL-cholesterol levels in adolescent children and in female adults. Human visfatin gene is located at 7q22.3, which has been reported to be a linkage region for insulin resistance syndrome *Jian et al. (2006)* reported that a single nucleotide polymorphism at different loci of visfatin gene was associated with triglyceride and total cholesterol levels. These reports suggest that visfatin may play a role in lipid homeostasis. However, the underlying mechanism is currently unknown (*Chen et al., 2006*).

In our study, significantly higher fasting blood glucose were found in the obese children compared with control group. This was in agreement with *Mehmet et al. (2009)*. There was no

significant correlation found between visfatin and fasting blood glucose which was similar to the study of *Berndt et al. (2005)*. Moreover, *Araki et al. (2008)*, in a population-based study of adult women, reported that no relationship was found between visfatin and metabolic parameters including fasting serum glucose.

The results of our study showed that serum leptin level was significantly higher in obese than in children of normal weight. This was in agreement with *Eun et al. (2012)* who have reported that serum leptin level were higher in obese than in children of normal weight. Markedly elevated leptin levels have been shown in obese human compared with non-obese humans (*Orel et al., 2004*). Elevated serum leptin concentration is a feature of obesity and abdominal adiposity (*Huang et al., 2004*). However, leptin levels severely decline in underweight human subjects compared with normal weight humans. The lack of the inhibiting effect of leptin as shown by higher energy intake in obese children who had higher serum leptin levels in our study suggested the possibility of the occurrence of leptin resistance in obese children, as reported by previous human studies (*Kolaczynski et al., 1996*). However, no correlations was found between serum leptin level with weight, BMI, and waist circumference in obese children in our study. In the present study, there was no correlation between serum concentration of leptin with level of lipids and fasting blood glucose. This was in agreement with *Eun et al. (2012)* who reported that leptin level was not correlated with lipids profile and fasting blood glucose. Leptin is a well-known adipokine involved in the long-term regulation of body weight,

dietary intake, and energy expenditure (*Crowley, 2008*). It has been proposed that this dual leptin restraint is the major regulatory arm of the feedback communication between the periphery and the hypothalamus for weight homeostasis. Leptin has been thought to contribute to body weight regulation by controlling food intake and energy expenditure at the hypothalamic level. Leptin abnormalities have been proposed to increase the propensity to obesity. Besides its role in metabolic disorders and obesity, leptin also has an important regulatory role on body hormonal and gonadal functions (*Carro et al., 1997 and Garcia-Mayor et al., 1997*).

The present study also showed that obese children have significantly higher systolic and diastolic blood pressures in obese children compared with control group and these findings were similar to study of *Mehmet et al. (2009)*.

In our study, the obese children showed higher significant difference in fasting blood glucose, TG, LDL-C, T.C, diastolic and systolic blood pressures, and they had lower HDL-C compared with non obese children. These were in agreement with *Stovitz et al. (2008)* who found that, in obese child, back pain was the most common complaint followed by foot and knee pain. Pain in the knees and hips were associated with increased weight and/or body mass index (BMI). *Hainsworth et al. (2012)* reported that pain occurred primarily in the lower extremities and with physical activity. Patients reporting current pain had a significantly higher body mass index than those reporting no pain. These findings suggested that pain was common in

severely obese youth, and pain should be recognized as a comorbidity of pediatric obesity. Routinely screening in severely obese children and adolescents for pain presence and intensity is recommended .

### CONCLUSION

Obesity has a significant impact on the health and well-being of these children and may contribute to ongoing health problems such as musculoskeletal pain and bone/joint dysfunction in later life.

### REFERENCES

1. **Araki S, Dobashi K, Kubo K, Kawagoe R, Yamamoto Y and Kawada Y (2008):** Plasma visfatin concentration as a surrogate marker for visceral fat accumulation in obese children. *Obesity*,16(2):384–8.
2. **Berndt J, Kl?ting N, Kralisch S, Kovacs P, Fasshauer M and Sch?n MR (2005):** Plasma visfatin concentrations and fat depot-specific mRNA expression in humans. *Diabetes*, 54(10):2911–6.
3. **Bouhours N, Gatelais F, Boux de Casson F, Rouleau S, and Coutant R (2007):** The insulin-like growth factor-I response to growth hormone is increased in prepubertal children with obesity and tall stature. *J Clin Endocrinol Metab.*, 92(2):629–35.
4. **Carro E, Senaris E R, Considine RV, Casanueva F F and Dieguez C (1997):** Regulation of in vivo growth hormone secretion by leptin. *Endocrinology* ,138:2203-2206.
5. **Chen MP, Chung FM, Chang DM, Tsai JC, Huang HF, Shin SJ and Lee YJ (2006):** Elevated plasma level of visfatin/pre-B cell colony-enhancing factor in patients with type 2 diabetes mellitus. *J Clin Endocrinol Metab.*, 91(1):295-9.
6. **Considine R V, Sinha M, Heiman A, Kriauciunas T, Stephens M, Nyce M, Ohannesian J and Marco C (1996).** Serum immunoreactive-leptin concentrations in normal-weight and obese humans. *N. Engl. J. Med.*, 334:292-295.
7. **Crowley VE (2008):** Overview of human obesity and central mechanisms regulating energy homeostasis. *Ann Clin Biochem*, 45:245–55.
8. **Danese A, Dove R, Belsky DW, Henchy J, Williams B, Ambler A and Arseneault L (2014):** Leptin deficiency in maltreated children, *Transl Psychiatry*, 23:4:e446.
9. **Daniels SR (2009):** Complications of obesity in children and adolescents. *Int J Obes (Lond)* , 33(suppl 1):S60–S65.
10. **Dominik G, Gregor H, Georg S, Daniel W, Kurt W, Oswald W, Stylianos K, and Michael W (2006):** *Journal of Pediatric Gastroenterology and Nutrition* .43:548Y549 Lippincott Williams & Wilkins, Philadelphia Department of Clinical Pharmacology, Institute for Medical and Chemical Laboratory Diagnostics, and Department of Pediatrics, Division of Nutrition and Metabolism, Medical University of Vienna.
11. **Eun K, Min S C, Soo H J, and Kyung R M (2012):** *Pediatric Gastroenterology, Hepatology & Nutrition Department of Pediatrics, School of Medicine, Chosun University, Gwangju, Korea*,15(3)166-174.
12. **Francisca L, Carlos D, Juan G R and Oreste G (2007):** Adipokines as emerging mediators of immune response and inflammation. *Nature Clinical Practice Rheumatology* .3, 716-724
13. **Fukuhara A, Matsuda M, Nishizawa M, Segwa K, Tanaka M, Kishimoto K, Matsuki Y, Murakami M, Ichisaka T, Murakami H, Watanabe E, Takagi T, Akiyoshi M, Ohtsubo T, Kihara S, Yamashita S, Makishima M, Funahashi T, Yamanaka S, Hiramatsu R, Matsuzawa Y and Shimomura I (2005):**Visfatin: a protein secreted by visceral fat that mimics the effects of insulin. *Science*, 307: 426-430.
14. **Garcia-Mayor R, Andrade MA, Rios M, Lage M, Dieguez C and Casanueva FF (1997):** Serum leptin levels in normal children: relationship to age, gender, body mass index, pituitary-gonadal hormones and pubertal stage. *J Clin Endocrinol Metab.*, 82: 2849-55.

15. Haider DG, Holzer G and Schaller G (2006): The adipokine visfatin is markedly elevated in obese children. *J Pediatr Gastroenterol Nutr* , 43(4):548–9.
16. Hainsworth KR1, Miller LA1, Stolzman SC1, Fidlín BM1, Davies WH1, Weisman SJ1 and Skelton JA1 (2012): Pain as a Comorbidity of Pediatric Obesity. *Infant Child Adolesc Nutr.*, 1;4(5): 315-320.
17. Hitt HC, McMillen RC, Thornton-Neaves T, Koch K and Cosby AG (2007): Comorbidity of obesity and pain in a general population: results from the southern pain prevalence study. *J Pain*,8: 430–436.
18. Huang L, Wang Z and Li C (2004): Modulation of circulating leptin levels by its soluble receptor. *J Biol Chem.*,276: 6343–9 .
19. Janssen I, Katzmarzyk PT and Boyce WF (2005): Health Behaviour in School-Aged Children Obesity Working Group. Comparison of overweight and obesity prevalence in school-aged youth from 34 countries and their relationships with physical activity and dietary patterns. *Obes Rev.*,6: 123–132.
20. Jian WX, Luo TH and Gu YY (2006):The visfatin gene is associated with glucose and lipid metabolism in a Chinese population. *Diabet Med.*,23:967–73.
21. Kolaczynski JW, Ohannesian JP, Considine RV, Marco CC and Caro JF (1996): Responses of leptin to Short-term fasting and prolonged overfeeding in human. *J Clin Endocrinol Metab.*, 81: 4162-5.
22. Long AC, Palermo TM, Manees and AM (2008): Brief report: using actigraphy to compare physical activity levels in adolescents with chronic pain and healthy adolescents. *J Pediatr Psychol.*, 33:660–665.
23. Mehmet D, Mesut O, Ekrem G, Mesut G, Halil G, Hamza K and Metin K (2009): S W I S S M E D W K L Y.1 3 9 ( 1 – 2 ): 22-27 Department of Paediatrics, Faculty of Medicine, Kahramanmaras Sutcu Imam University, Kahramanmaras, Turkey.
24. Moschen AR, Geiger S, Gerner R and Tilg H (2010): Pre-B cell colony enhancing factor/NAMPT/visfatin and its role in inflammation-related bone disease. *Mutat Res* ., 690(1-2):95-101.
25. Nusken KD, Nusken E, Petrasch M, Rauh M and D?tsch J (2007): Preanalytical influences on the measurement of visfatin by enzyme immunoassay . *Clin Chim Acta*, 382 (1-2):154–6.
26. Orel M, Lichnovska R, Gwozdziejczova S, Zlamalova N, Klementa I, Merkunova A and Hrebicek J (2004): Gender differences in tumor necrosis factor alpha and leptin secretion from subcutaneous and visceral fat tissue. *Physiol Res* 53: 501-505. Relation to the metabolic syndrome. *Endocr Rev.*, 21(6):697–738.
27. Sandeep S1, Velmurugan K, Deepa R and Mohan V (2007): Serum visfatin in relation to visceral fat, obesity and type 2 diabetes mellitus in Asian Indians. *Metabolism*, 56(4): 565-70.
28. Smith S M, Sumar B and Dixon K A (2014): Musculoskeletal pain in overweight and obese children, *Int J Obes (Lond)*, 38(1): 11–15.
29. Stovitz SD1, Pardee PE, Vazquez G, Duval S and Schwimmer JB (2008): Musculo-skeletal pain in obese children and adolescents. *Acta Paediatr*, 97(4):489-93.

حجازي مجاهد التميمي\* - محمود فرج سالم\*\* - البكري محمد ثروت\*\*\*

أيمن السعيد صادق\*\*\*\*

أقسام الطب الطبيعي والروماتيزم والتأهيل\* والباثولوجيا الإكلينيكية\*\* والأطفال\*\*\* والقلب\*\*\*\*  
بكلية الطب - جامعة الأزهر (القاهرة ودمياط)

**خلفية البحث:** زيادة معدل السمنة أصبح مشكلة هامة من مشاكل الصحة العامة لدى الأطفال ويعكس معه مشاكل أخرى. والسمنة لدى الأطفال قد تسبب ارتفاع ضغط الدم والإصابة بالسكر وأمراض القلب والأوعية الدموية.

**الهدف من البحث:** مقارنة القياسات البشرية ( الطول والوزن و مؤشر كتلة الجسم ومحيط الخصر ) ضغط الدم الانبساطي و الانقباضي ، لمحة الدهون ( TC ، TG ، HDL-C ، LDL-C ) ، وصوم الجلوكوز في الدم، مستوى هرمون الليبتين ، ومستوى الفوسفاتين وألم الهيكل العظمى بين الأطفال البدناء(ذوى السمنة)مع الأطفال غير البدناء. وكذلك دراسة العلاقة بين ألم الهيكل العظمى و مستوى هرمون الليبتين ، ومستوى الفوسفاتين والوزن و مؤشر كتلة الجسم ومحيط الخصر.

**طرق وأشخاص البحث:** وتألفت عينة الدراسة من مجموعتين. المجموعة ( 1 ) ثلاثون طفلا غير بدناء مع مطابقة السن مع اعتبارهم المجموعة الضابطة و المجموعة ( 2 ) 60 طفلا يعانون من السمنة المفرطة الذين تتراوح أعمارهم بين 11-15 عاما بصحة جيدة. تم تقييم دراسة وصفية الحالات والشواهد في طب الأطفال وأمراض الروماتيزم وأمراض القلب والباثولوجيا الإكلينيكية ، مستشفيات جامعة الأزهر ودمياط والقاهرة ، مصر في الفترة من ديسمبر 2013 إلى ديسمبر 2014 مع الموافقة المستنيرة. وقد تم أخذ تاريخ دقيق ولفحص البدني بما في ذلك القياسات البشرية ، تم احتساب وزن الجسم والطول، و قياس محيط الخصر ، و مؤشر كتلة الجسم ( BMI ) ( بالكيلو جرام / M2 ) وذلك في مؤشر السمنة بشكل عام. وقد تم تحديد الأطفال البدناء هم ذوي مؤشر كتلة الجسم < 95 المئوية ، والذين يعانون من مؤشر كتلة الجسم > 85 المئوية غير البدناء . وكذلك دراسة العلاقة بين ألم الهيكل العظمى و مستوى هرمون الليبتين ، ومستوى الفوسفاتين والوزن و مؤشر كتلة الجسم ومحيط الخصر.

**النتائج:** أظهر الأطفال البدناء أعلى أهمية مختلفة في السكر الصائم في الدم، TG، LDL-C، TC وضغط الدم الانبساطي والانقباضي و كان لديهم انخفاض HDL-C مقارنة مع الأطفال غير البدناء وهذا التنبؤ من متلازمة التمثيل الغذائي لهؤلاء الأطفال اللذين يعانون من السمنة المفرطة في المستقبل. وقد أظهرت الدراسة أن ارتباط ملموس بين مؤشرات القياسات البشرية (الوزن، الطول، مؤشر كتلة الجسم، ومحيط الخصر ) والعمر وضغط الدم الانقباضي و ضغط الدم الانبساطي مع الفوسفاتين في مجموعة الاطفال الذين يعانون من السمنة المفرطة. وقد وجد ارتباطا ملموسا بين الليبتين، FBS، الكوليسترول الكلي، HDL-C و LDL-C مع الفوسفاتين في مجموعة الاطفال اللذين يعانون من السمنة المفرطة، وقد وجد أن هناك ارتباطا ولكن ايجابيا بين الفوسفاتين والشحوم الثلاثية ولا توجد علاقة ذات قيمة احصائية بين مؤشرات القياسات البشرية (الوزن، الطول، مؤشر كتلة الجسم، ومحيط الخصر) وضغط الدم الانقباضي وضغط الدم الانبساطي والعمر مع هرمون الليبتين في المجموعة الاطفال اللذين يعانون من السمنة المفرطة. لا توجد علاقة ذات قيمة احصائية بين FBS، الكوليسترول الكلي والدهون الثلاثية، HDL-C، و LDL-C مع هرمون الليبتين في الأطفال الذين يعانون من السمنة المفرطة. وقد ظهر ارتباطا كبيرا بين من ألم الورك و ألم الركبة وآلام القدمين وآلام الظهر مع الوزن والخصر ومؤشر كتلة الجسم.

**الخلاصة:** السمنة لها تأثير كبير على صحة الأطفال و يمكن أن تسهم في مشاكل صحية مستمرة مثل آلام العضلات والعظام والخلل في الحياة في وقت لاحق.