# Serum visfatin level in obese Egyptian children and adolescents and its relation with metabolic syndrome

Ghada M. Anwar[a], Ayat A. Motawei[b], Amany Ibrahim[a], Ashraf Galal[b], Hassan M. Salama[b], Azza A. Aly[c] and Eman A. Moustafa[b]

***Objective*** **We aimed to study visfatin level in obesity and its relation with metabolic syndrome (MS).**

***Patients and methods*** **This case–control study included 42 obese children and adolescents and 39 age-matched and sex-matched nonobese children who served as controls. Full medical examination and anthropometric measurements were carried out. Fasting serum total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, blood sugar, insulin, and visfatin were measured. Homeostasis model assessment for insulin resistance (HOMA-IR) was calculated.**

***Results*** **Fasting serum visfatin level was significantly higher in obese children and adolescents compared with controls (8.7 ± 2.8 vs. 3.3 ± 2.3 ng/ml, *P* = 0.001). Visfatin showed significant positive correlations with waist circumference (*P* < 0.001), systolic blood pressure (*P* = 0.001), diastolic blood pressure (*P* = 0.01), and fasting blood glucose (*P* = 0.001). Visfatin was significantly higher in obese patients with MS than in patients without MS (10.03 ± 3.2 vs. 7.8 ± 2.2 ng/ml, *P* = 0.02).**

***Conclusion*** **Visfatin elevated in obese children. Visfatin might be used as a predictor test for the existence of MS in obese children. *Med Res J* 14:53–58 © 2015 Medical Research Journal.**

**Medical Research Journal** 2015, **14**:53–58

**Keywords: adolescents, children, insulin resistance, metabolic syndrome, obesity, visfatin**

[a]Pediatric Department, Cairo University, [b]Pediatric Department and [c]Chemical and Clinical Pathology Department, National Research Centre, Cairo, Egypt

Correspondence to Hassan M. Salama, MD, Pediatric Department, National Research Centre, El-Bohouth Street, Dokki, PO Box 12311, Cairo, Egypt
Tel: +20 100 651 4147; e-mail: hsalama27@gmail.com

**Received** 17 June 2015 **accepted** 11 August 2015

## Introduction

Childhood obesity is a global epidemic and is associated with a higher risk of chronic diseases such as hypertension, diabetes mellitus, and other metabolic disorders [1]. Adipose tissue is an endocrine organ producing and secreting biologically active adipokines [2]. Visfatin is a recently described adipokine and it is predominantly expressed in visceral adipose tissue and is upregulated in obese humans [3]. Visfatin is an adipokine that has been implicated in obesity, insulin resistance, and diabetes mellitus [4]. In normal-weight and insulin-sensitive individuals, visfatin is predominantly produced by subcutaneous adipose tissue [5]. However, in obese individuals, there is an increased synthesis of visfatin by macrophages in the visceral adipose tissue [6]. Visfatin exerts insulin-mimetic actions through insulin receptors [7]. This adipokine binds to the insulin receptor at a site distinct from insulin. By this mechanism, visfatin reduces glucose release from hepatocytes and stimulates glucose utilization in adipocytes. Visfatin has effects on lipid homeostasis similar to that of insulin, and it was also involved in adipocyte proliferation and differentiation and triglyceride metabolism [2]. Previous studies reported an increased circulating visfatin level in obese individuals [8] and type 2 diabetic patients [9]. Elevated visfatin production in obesity seems to be a compensatory response in obesity-induced insulin resistance [10]. Therefore, an increased visfatin level in the obese may also indirectly participate in the development of insulin resistance. However, data concerning the association between circulating levels of visfatin and insulin resistance are inconsistent [11]. There is lack of consensus on visfatin concentrations and their relationship with anthropometric and metabolic parameters in children.

The aim of this study was to determine the serum visfatin levels in obese Egyptian children and adolescents and its relation with their anthropometric measurements and metabolic syndrome (MS) risk factors.

## Patients and methods

### Patients

A total of 42 obese children and adolescents (23 male and 19 female) attending the Diabetes Endocrine and Metabolism Pediatric Unit, Cairo University, were studied. Their ages ranged between 5 and 14 years (mean = 9.2 ± 2.8 years). All of them had BMI greater than 95th percentile. No patients with pathological causes of obesity were included.

In addition, 39 age-matched and sex-matched healthy children and adolescents (22 male and 16 female) were included as the control group. They had a normal BMI, with no clinical evidence or family history of obesity or MS and they attended the hospital for minor illness.

The study was approved by the Ethics Committee of Cairo University School of medicine. Informed consent was taken from all participants included in the study.

### Methods

For all participants, complete medical history and clinical examination were performed. Anthropometric measurements, including weight, height, waist and hip circumferences, waist/

hip ratio, pubertal staging, and BMI, were performed. Obesity was defined as a BMI value higher than or equal to the 95th percentile value for age and sex. However, waist circumference measurement was plotted on centiles as followed by Fernández *et al.* [12]; all obese had a waist circumference more than the 90th percentile.

For laboratory investigations, the included cases and controls were made to fast for 12 h and then 5 ml of venous blood was drawn and the separated serum was stored at −80°C until analytic measurement of serum insulin, except for glucose, which was determined immediately after blood was drawn. Serum triglycerides, total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-c), and fasting blood sugar measurement was carried out using an autoanalyzer (Olympus-Au-400). Low-density lipoprotein cholesterol (LDL-c) was calculated using Friedewald's formula [13]. Adenocorticotropic hormone and cortisol were assessed to exclude Cushing syndrome.

Fasting serum insulin was estimated using the enzyme-linked immunoassay (ELISA) technique. Insulin resistance was estimated using HOMA-IR (homeostasis model assessment method): HOMA = [fasting insulin (μU/ml) × fasting glucose (mg/dl)/405] [14]. Fasting serum visfatin was assessed using ELISA, grossly hemolyzed or lipemic samples were excluded to avoid interference with the results. Detection range was 0.62–40 ng/ml. This assay recognizes recombinant and natural human visfatin. No significant cross-reactivity or interference was observed.

MS was diagnosed according to most recent guidelines; MS was considered if three or more of the following criteria were present:

(1) Abdominal obesity (waist circumference ≥ 90th percentile for age and sex).
(2) Fasting triglycerides 110 mg/dl or greater.
(3) HDL 40 mg/dl or less.
(4) Systolic/diastolic blood pressure ≥ 90th percentile for age, sex, and height.
(5) Fasting glucose of100 mg/dl or greater [15].

### Statistical methods

Statistical Package for Social Science (SPSS) program, version 17.0, was used for data analysis. Mean and SD were estimates of quantitative data, whereas frequency and percentage were estimates of qualitative data. Differences in clinical and biochemical characteristics were tested using Student's *t*-test for quantitative data and using the $\chi^2$-test for qualitative data. A two-sided *P*-value less than 0.05 was considered statistically significant. Receiver operator characteristic (ROC) curve was drawn to detect the best cutoff for the parameters studied. Standard deviation score was calculated for weight, height, and BMI using the WHO Anthro software, version 3.22, 2011.

## Results

Descriptive data of obese patients and controls are shown in Table 1. Acanthosis nigricans was found in 52.4% of obese patients. All cases had waist circumference more than the 90th percentile. An overall 40.5% of patients had only systolic hypertension, and 23.8% of patients had only diastolic hypertension, and 14.3% of patients had both systolic and diastolic hypertension. Laboratory investigations of the obese patients and controls are shown in Table 2. An overall 9.5% of obese children had high triglyceride, 47.6% of patients had high TC, 54.8% of patients had high LDL-c, 14.3% of patients had low HDL-c, and 2.4% of patients had high fasting blood sugar. None of the controls had abnormal laboratory results.

Acanthosis nigricans was found in 75% of patients with MS compared with 38.5% of patients without MS, with a *P*-value less than 0.05.

The comparison between the obese and control groups in their lipid profile, fasting blood sugar, fasting insulin, HOMA-IR, and visfatin showed statistically significant difference between the two groups in TC, HDL-c and LDL-c, fasting insulin, HOMA, and visfatin, whereas there was no statistically significant difference in the triglycerides and fasting blood sugar (Table 2).

The cutoff point of visfatin using ROC curve analysis is presented in Fig. 1.

Using ROC curve, cutoff point of visfatin for obesity in the studied group was 5.5. Visfatin was a good predictor for MS in obese patients as it had a sensitivity of 86.1% and a specificity of 86.5% and an area under the curve of 0.93.

When correlation analysis was performed, serum visfatin was positively correlated with age ($r = 0.5$, $P < 0.001$),

**Table 1** **Descriptive characteristics of the obese and control groups**

| | Obese group ($n=42$) | Control group ($n=39$) | *P*-value |
|---|---|---|---|
| Male/female ratio | 23/19 | 22/16 | 0.8 |
| Age (years) | 9.2 ± 2.8 | 9.1 ± 3.1 | 0.9 |
| Prepubertal/Pubertal patients | 18/24 | 21/18 | 0.2 |
| Height (SDS) | 0.9 ± 1.2 | −0.2 ± 1.3 | <0.001** |
| Weight (SDS) | 5.6 ± 2.2 | −0.5 ± 1.5 | <0.001** |
| BMI (kg/m$^2$) | 30.4 ± 5.9 | 16.98 ± 3.6 | <0.001** |
| BMI (SDS) | 3.5 ± 0.7 | −0.1 ± 1.1 | <0.001** |
| W/H ratio | 0.96 ± 0.0 | 0.9 ± 0.0 | <0.001** |
| SBP (mmHg) | 116.9 ± 11.6 | 106.7 ± 8.6 | <0.001** |
| DBP (mmHg) | 76.1 ± 9.5 | 67.2 ± 7.04 | <0.001** |

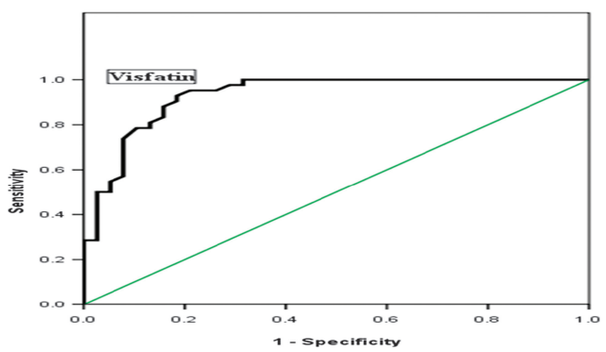
BMI was calculated using the WHO Anthro software, version 3.22, 2011.
Data are expressed as mean ± SD or ratio as indicated.
DBP, diastolic blood pressure; SBP, systolic blood pressure; SDS, standard deviation score was calculated for weight, height; W/H, waist/hip ratio.
**$P<0.001$, highly significant.

**Table 2 Lipid profile, fasting blood sugar, insulin, HOMA-IR, and visfatin in the obese and control groups**

| | Obese group (mean ± SD) ($n$ = 42) | Control group (mean ± SD) ($n$ = 39) | $P$-value |
|---|---|---|---|
| Total cholesterol (mg/dl) | 194.6 ± 35.9 | 163.2 ± 21.6 | <0.001** |
| Triglyceride (mg/dl) | 79.7 ± 42.4 | 69.6 ± 24.9 | 0.2 |
| HDL-c (mg/dl) | 60.8 ± 16.7 | 70.8 ± 24.2 | <0.01* |
| LDL-c (mg/dl) | 117.3 ± 34 | 79.9 ± 25.6 | <0.001** |
| Fasting blood sugar (mg/dl) | 93.5 ± 10.8 | 91.5 ± 6.7 | 0.3 |
| Fasting insulin (μIU/ml) | 16.97 ± 5.8 | 8.5 ± 4.5 | <0.001** |
| HOMA-IR | 3.9 ± 1.5 | 1.9 ± 0.9 | <0.001** |
| Visfatin (ng/ml) | 8.7 ± 2.8 | 3.3 ± 2.3 | <0.001** |

HDL-c, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment for insulin resistance; LDL-c, low-density lipoprotein cholesterol.
*$P$<0.01, significant.
**$P$<0.001, highly significant.

**Fig. 1**




Cutoff point of visfatin by means of receiver operator characteristic (ROC) curve analysis.

**Table 3 Correlation between fasting serum visfatin and the anthropometric measures and blood pressure measurement in the obese group**

| | $r$ | $P$-value |
|---|---|---|
| Age | 0.5 | <0.001** |
| Height (cm) | 0.50 | <0.001** |
| Height (SDS) | −0.20 | 0.3 |
| Weight (kg) | 0.60 | <0.001** |
| Weight (SDS) | 0.07 | 0.7 |
| BMI | 0.60 | <0.001** |
| BMI (SDS) | 0.06 | 0.7 |
| Waist circumference (cm) | 0.70 | <0.001** |
| Hip circumference (cm) | 0.70 | <0.001** |
| Waist/hip ratio | 0.04 | 0.8 |
| Systolic blood pressure (mmHg) | 0.50 | <0.001** |
| Diastolic blood pressure (mmHg) | 0.40 | <0.001** |

$r$, correlation coefficient; SDS, standard deviation score.
**$P$<0.001, highly significant.

height, weight, BMI, waist and hip circumference, and systolic and diastolic blood pressure (Table 3).

Moreover, serum visfatin was positively correlated with fasting blood glucose ($P$ = 0.001) ( Table 4).

Serum visfatin level was significantly higher in pubertal than in prepubertal obese patients (9.6 ± 3.1 vs. 7.5 ± 1.9 ng/ml, $P$ = 0.02). Acanthosis nigricans was found in 75% of patients with MS compared with 38.5% of patients without MS, with a $P$-value less than 0.05.

**Table 4 Correlation between fasting serum visfatin and the laboratory data in the obese group**

| | $r$ | $P$-value |
|---|---|---|
| Fasting blood glucose (mg/dl) | 0.50 | <0.001** |
| Total cholesterol (mg/dl) | 0.60 | −0.09 |
| Triglycerides (mg/dl) | 0.60 | 0.09 |
| HDL-c (mg/dl) | 0.40 | 0.1 |
| LDL-c (mg/dl) | 0.30 | −0.2 |
| Serum insulin (μIU/ml) | 0.20 | 0.2 |
| HOMA-IR | 0.08 | 0.3 |

HDL-c, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment for insulin resistance; LDL-c, low-density lipoprotein cholesterol; $r$, correlation coefficient.
**$P$<0.001, highly significant.

Comparison between anthropometric and blood pressure data in MS and non-MS obese patients is shown in Table 5, as BMI, waist circumference, head circumfrence, systolic blood pressure, and diastolic blood pressure were significantly higher in obese children with MS but W/H ratio was lower ($P$ = 0.008, 0.03, 0.002, <0.001, 0.001, and 0.05, respectively).

Moreover, comparison between laboratory data in MS and non-MS obese patients is described in Table 6.

## Discussion

Visfatin is known for its increasing levels in obese children and adults [1,16]. Our study shows the same previously established results wherein visfatin was elevated in obese children than in control children. Furthermore, Kim and colleagues concluded in 2013 that elevated plasma visfatin is probably augmented by central obesity in children and adolescents. They also concluded that visfatin levels are more related to visceral fat, especially in children and adolescents with lower adiposity [17]. In this study, our results showed a positive correlation between visfatin levels and both waist and hip circumference.

Central obesity is known to be related to the risk factors of cardiovascular disease. Visfatin and its genetic variants play an important role in morbidity and cardiovascular risk for obesity [18]. Lipid profile showed marked differences between obese and control children and adolescents in our study. TC and LDL-c levels were elevated, where HDL-c levels were decreased in obese children. However, no significant correlation was found between visfatin

**Table 5 Comparison between anthropometric and blood pressure data in metabolic syndrome and nonmetabolic syndrome obese patients**

| | Metabolic syndrome obese patients ($n=16$) | Nonmetabolic syndrome obese patients ($n=26$) | *P*-value |
|---|---|---|---|
| Height (SDS) | 0.7 ± 1.3 | 1.03 ± 1.2 | 0.5 |
| Weight (SDS) | 5.8 ± 2.1 | 5.6 ± 2.3 | 0.8 |
| BMI | 33.4 ± 7.5 | 28.5 ± 3.7 | <0.001** |
| BMI (SDS) | 3.5 ± 0.4 | 3.5 ± 0.8 | 0.8 |
| W/H ratio | 0.9 ± 0.04 | 0.97 ± 0.08 | <0.01* |
| Systolic blood pressure (mmHg) | 124.1 ± 7.7 | 112.3 ± 11.2 | <0.001** |
| Diastolic blood pressure (mmHg) | 81.9 ± 7.04 | 72.5 ± 9.2 | <0.001** |

SDS, standard deviation score; W/H, waist to hip ratio.
*$P<0.01$, statistically significant.
**$P<0.001$, highly significant.

**Table 6 Comparison between laboratory data in metabolic syndrome and nonmetabolic syndrome obese patients**

| | Metabolic syndrome patients ($n=16$) | Nonmetabolic syndrome patients ($n=26$) | *P*-value |
|---|---|---|---|
| Fasting blood glucose (mg/dl) | 96.8 ± 12.7 | 88.3 ± 8.04 | <0.01* |
| Total cholesterol (mg/dl) | 190.3 ± 43.8 | 197.3 ± 30.7 | 0.6 |
| Triglycerides (mg/dl) | 99.4 ± 56.6 | 67.2 ± 24.7 | <0.01* |
| HDL-c (mg/dl) | 54.6 ± 15.9 | 64.5 ± 16.4 | <0.01* |
| LDL-c (mg/dl) | 114.4 ± 34.2 | 119.1 ± 34.6 | 0.7 |
| Fasting insulin (μIU/ml) | 17.8 ± 5.5 | 16.5 ± 5.95 | 0.5 |
| HOMA-IR | 4.3 ± 1.7 | 3.6 ± 1.3 | 0.2 |
| Visfatin (ng/ml) | 10.03 ± 3.2 | 7.8 ± 2.2 | <0.01* |

HDL-c, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment for insulin resistance; LDL-c, low-density lipoprotein cholesterol.
*$P<0.01$, statistically significant.

and TC, LDL, and HDL-c levels. Similar results were elicited by Haider *et al*. [19], in 2006, who reported no correlation between lipid profile and visfatin in obese children. Jin *et al.* [20] observed that HDL-c levels were positively correlated with visfatin levels in adolescents. These different results could be contributed to the difference in the age group (11–18 years) of their study compared with ours.

In this study, visfatin levels showed a positive correlation with fasting blood glucose levels. There was no correlation with both HOMA-IR and fasting insulin levels. In 2014 Samsam-Shariat *et al.* [21] found a significant positive correlation between visfatin and fasting blood glucose levels in nondiabetic individuals. In an animal study conducted in 2011, recombinant mice visfatin lowered blood glucose levels directly [22]. Other authors showed that elevated visfatin is highlighted in obese children, especially those with insulin resistance. They even assume that visfatin might be used as an indicator of insulin resistance [23]. Such conflicting data may be attributed to the difference in the specificity of the immunoassays used in the different studies [24]. Moreover, Goktas *et al.* [25] (2013) concluded that correlations of visfatin with insulin resistance markers are tissue dependent.

When we compare obese children who are suspected of having MS with those who are not, we find more elevated levels of visfatin in cases with MS than in obese children without MS. The same was elicited by Kural *et al.* [26], who reported that the levels of visfatin were significantly higher in MS than in non-MS individuals. Chang *et al.* [27] also detected the same elevation of visfatin in MS cases and even suggested the use of visfatin as a diagnostic marker in MS. Furthermore, Belo *et al.* [28] found that obese children with metabolic risk factors have a higher visfatin level compared with obese children who do not have metabolic risk factors and controls. Nourbakhsh *et al.* [4] concluded that visfatin has a more prominent association with MS and thus can be used as a biomarker for MS. However, Pyrzak *et al.* [29] had found that adiponectin can be used as a biomarker for MS.

## Conclusion

Visfatin is increased in obese children and adolescents. Visfatin level is increased in children and adolescent with criteria of MS. Visfatin might be used as a marker for the diagnosis of MS.

## Acknowledgements

### Conflicts of interest

There are no conflicts of interest.

## References

1 Jaleel A, Aheed B, Jaleel S, Majeed R, Zuberi A, Khan S, *et al*. Association of adipokines with obesity in children and adolescents. *Biomark Med* 2013; **7**:731–735.
2 Fukuhara A, Matsuda M, Nishizawa M, Segawa K, Tanaka M, Kishimoto K, *et al*. Visfatin: a protein secreted by visceral fat that mimics the effect of insulin. *Science* 2005; **307**:426–430.
3 Berndt J, Klöting N, Kralisch S, Kovacs P, Fasshauer M, Schön MR, *et al.* Plasma visfatin concentrations and fat depot specific mRNA expression in humans. *Diabetes* 2005; **54**:2911–2916.
4 Nourbakhsh M, Gholinejad Z, Razzaghy-Azar M. Visfatin in obese children and adolescents and its association with insulin resistance and metabolic syndrome. *Scand J Clin Lab Invest* 2015; **75**:183–188.
5 Varma V, Yao-Borengasser A, Rasouli N, Bodles AM, Phanavanh B, Lee MJ, *et al*. Human visfatin expression: relationship to insulin sensitivity, intramyocellular lipid and inflammation. *J Clin Endocrinol Metab* 2007; **92**:666–672.

6 Panidis D, Farmakiotis D, Rousso D, Katsikis I, Delkos D, Piouka A, *et al*. Plasma visfatin levels in normal weight women with polycystic ovary syndrome. *Eur J Intern Med* 2008; **19**:406–412.
7 Taskesen D, Kirel B, Us T. Serum visfatin levels, adiposity and glucose metabolism in obese adolescents. *J Clin Res Pediatr Endocrinol* 2012; **4**:76–81.
8 Li L, Yang G, Li Q, Tang Y, Yang M, Yang H, Li K. Changes and relations of circulating visfatin, apelin and resistin levels in normal, impaired glucose tolerance, and type 2 diabetic subjects. *Exp Clin Endocrinol Diabetes* 2006; **114**:544–548.
9 Sethi JK, Vidal-Puig A. Visfatin: the missing link between intraabdominal obesity and diabetes? *Trends Mol Med* 2005; **11**:344–347.
10 Alghasham AA, Barakat YA. Serum visfatin and its relation to insulin resistance and inflammation in type 2 diabetic patients with and without macroangiopathy. *Saudi Med J* 2008; **29**:185–192.
11 Davutoglu M, Ozkaya M, Guler E, Garipardic M, Gursoy H, Karabiber H, Kilinc M. Plasma visfatin concentrations in childhood obesity: relationships to insulin resistance and anthropometric indices. *Swiss med wkly* 2009; **139** (1–2):22–27.
12 Fernández JR, Redden DT, Pietrobelli A, Allison DB. Estimated value for percentile regression for all children and adolescents combined, according to sex. *J Pediatr* 2004; **145**:439–444.
13 Friedwald WT, Levy RI, Frederickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. *Clin Chem* 1972; **18**:499–502.
14 Mebmet D, Mesut O, Ekrem G, Mesut G, Hilal G, Hamza K, Metin K. Plasma visfatin concentrations in childhood obesity: relationships to insulin resistance and anthropometric indices. *Swiss Med Wkly* 2009; **139** (1–2):22–27.
15 Pedrosa C, Bruno O, Isabel A, Carlos SP, Maria DV, Flora C. Obesity and metabolic syndrome in 7-9 years-old Portuguese schoolchildren. *Diabetol Metab Syndr* 2010; **2**:40.
16 Indulekha K, Surendar J, Anjana RM, Geetha L, Gokulakrishnan K, Pradeepa R, *et al*. Metabolic obesity, adipocytokines, and inflammatory markers in Asian Indians – CURES-124. *Diabetes Technol Ther* 2014; **17**:134–141.
17 Kim HJ, Park SY, Choi YJ, Han SJ, Lee KW, Kim DJ. Differential significance of plasma visfatin concentrations according to adiposity in children and adolescents. *Horm Res Paediatr* 2013; **79**:208–213.
18 Ooi SQ, Chan RM, Poh LK, Loke KY, Heng CK, Chan YH, *et al*. Visfatin and its genetic variants are associated with obesity-related morbidities and cardiometabolic risk in severely obese children. *Pediatr Obes* 2014; **9**:81–91.
19 Haider DG, Schaller G, Kapiotis S, Maier C, Luger A, Wolzt M. The release of the adipocytokine visfatin is regulated by glucose and insulin. *Diabetologia* 2006; **49**:1909–1914.
20 Jin H, Jiang B, Tang J, Lu W, Wang W, Zhou L, *et al*. Serum visfatin concentrations in obese adolescents and its correlation with age and high-density lipoprotein cholesterol. *Diabetes Res Clin Pract* 2008; **79**:412–418.
21 Samsam-Shariat SZ, Bolhasani M, Sarrafzadegan N, Najafi S, Asgary S. Relationship between blood peroxidases activity and visfatin levels in metabolic syndrome patients. *ARYA Atheroscler* 2014; **10**:218–226.
22 Naz R, Hameed W, Hussain M, Aslam M. Glucose lowering effect of visfatin in obese and insulin dependant diabetes mellitus. *Pak J Phusiology* 2011; **7**:4–7.
23 Tascilar ME, Cekmez F, Meral C, Pirgon O, Tanju IA, Canpolat FE, *et al*. Evaluation of adipocytokines in obese children with insulin resistance. *Turk J Pediatr* 2011; **53**:269–273.
24 Nusken KD, Nusken E, Petrasch M, Rauh M, Dotsch J. Preanalytical influences on the measurement of visfatin by enzyme immuno assay. *Clin Chim Acta* 2007; **382** (1–2):154–156.
25 Goktas Z, Owens S, Boylan M, Syn D, Shen CL, Reed DB, *et al*. Associations between tissue visfatin/nicotinamide, phosphoribosyltransferase (Nampt), retinol binding protein-4, and vaspin concentrations and insulin resistance in morbidly obese subjects. *Mediators Inflamm* 2013; **2013**:861496.
26 Kural B, Deger O, Erem C, Balaban Yucesan F, Aliyazicioglu R, Barlak Y. Is the combined use of insulin resistance indices, including adipokines, more reliable in metabolic syndrome? *Turk J Med Sci* 2014; **44**:1021–1028.
27 Chang X, Jiao Y, Lu J, Wang Y, Abudureyimu K, Zhang C, *et al*. Adiponectin and visfatin may serve as diagnosis markers for metabolic syndrome in Uygur population. *Int J Clin Exp Med* 2014; **7**:4322–4326.
28 Belo VA, Luizon MR, Lacchini R, Miranda JA, Lanna CM, Souza-Costa DC, *et al*. The effects of NAMPT haplotypes and metabolic risk factors on circulating visfatin/NAMPT levels in childhood obesity. *Int J Obes (Lond)* 2015; **39**:130–135.
29 Pyrzak B, Ruminska M, Popko K, Demkow U. Adiponectin as a biomarker of the metabolic syndrome in children and adolescents. *Eur J Med Res* 2010; **15** (Suppl 2):147–151.

# الملخص العربي

## مستوى الفيسفاتين فى مصل الدم وعلاقته بالأطفال والمراهقين المصريين البدناء وكذلك بمتلازمة التمثيل الغذائي لديهم

**غادة أنور*، أيات مطاوع†، أماني إبراهيم*، أشرف جلال†، حسن سلامة†، عزة على‡، إيمان مصطفى†**

**من أقسام *طب الأطفال، جامعة القاهرة،† طب الأطفال، ‡والتحاليل الطبية والكيميائية، المركز القومي للبحوث، جمهورية مصر العربية**

**تهدف الدراسة التالية الى ايجاد العلاقة بين مستوى الفسفاتين في الدم والسمنة في الاطفال وكذلك علاقته مع متلازمة التمثيل الغذائي في الاطفال المصريين. شملت الدراسة 42 من الأطفال والمراهقين الذين يعانون من السمنة المفرطة. وعدد 39 من الاطفال لا يعانون من السمنة والمتناغمين مع المجموعة السابقة سنا وجنسا واستخدموا كمجموعة ضابطة للدراسة. تم عمل فحص طبي شامل لكل الأطفال وكذلك أحذ المقاييس المختلفة لهم. بعد فترة الصيام المناسبة تم أخذ عينات الدم لقياس كل من الكولسترول الكلي والدهون الثلاثية، والكليسترول عالى الكثافة والكليسترول منخفض الكثافة والجلوكوز في الدم وهرمون الانسولين، والفيسفاتين. تم ايضا احتساب معامل مقاومة الانسولين بالجسم. وجد ان مستوى الفيسفاتين أعلى في الأطفال والمراهقين الذين يعانون من السمنة بالمقارنة بالمجموعة الضابطة (8.7 + 2.8 مقابل 3.3 + 2.3 نانوغرام / ملل $p=0.001$). أظهر الفيسفاتين ارتباطا إيجابيا مع محيط الخصر ($p<0.001$) وضغط الدم الانقباضي ($p=0.001$) وضغط الدم الانبساطي ($p=0.01$) والجلوكوز الصائم ($p=0.001$). مستوى الفيسفاتين كان أعلى كثيرا في الأطفال والمراهقين الذين يعانون من السمنة المفرطة والمقترنة مع متلازمة التمثيل الغذائي عن أقرانهم البدناء بدون الاقتران مع متلازمة التمثيل الغذائي (10.03 + 3.2 مقابل 7.8 + 2.2 نانوغرام / ملل $p=0.02$). نستنتج من الدراسة ان مستوى الفيسفاتين أعلى في الأطفال الذين يعانون من السمنة. قد يمكن استخدام الفيسفاتين كمؤشر ومقياس على وجود متلازمة التمثيل الغذائي لدى الأطفال والمراهقين البدناء.**