Visfatin as an Early Marker for the Diagnosis of Metabolic Syndrome in Obese Adults: A Cross-sectional Study

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ABSTRACT

Introduction: It is well-established that obesity plays a significant role in the development of metabolic syndrome. Visfatin is a novel adipocytokine predominantly secreted in adipose tissue, associated with a wide range of biological effects including glucose and lipid metabolism. Visfatin levels are significantly linked to inflammation and the development of obesity-related metabolic disorders. Unfortunately, the roles of visfatin in obesity, particularly in the Indian population, are scarce.

Aim: To study the role of serum visfatin in diagnosing metabolic syndrome in overweight and obese adults.

Materials and Methods: A comparative cross-sectional study was conducted at the Department of Biochemistry, Tomo Riba Institute of Health and Medical Sciences, Naharlagun, Arunachal Pradesh, India, between September 2022 and October 2023. A total of 200 subjects (50 controls, 50 overweight individuals, 50 obese individuals without metabolic syndrome, and 50 obese individuals with metabolic syndrome), aged 20-70 years, were enrolled as study participants. Anthropometric parameters, lipid profiles, and fasting glucose were analysed using an auto analyser. Serum visfatin levels were measured by Enzyme-linked Immunosorbent Assay (ELISA). Statistical analysis was performed using the t-test, and categorical data were analysed using the Chi-square test. Correlation analysis was done by Pearson's correlation at a significance level of 5%.

Results: The control group consisted of 17 males and 33 females with a mean age of 41.5±13.4 years, the overweight group consisted of 13 males and 37 females with a mean age of 37.1±10.9 years, the obese without metabolic syndrome group consists of 16 males and 34 females with a mean age of 40.6±12.7 years, and obese with metabolic syndrome group had 23 males and 27 females with a mean age of 42.0±9.2 years. Serum visfatin levels (ng/mL) were significantly elevated in the overweight (1.7±0.3), obese without metabolic syndrome (4.3±3.2), and obese with metabolic syndrome (10.9±6.6) groups compared to the controls (1.0±0.2). Serum visfatin levels were positively correlated with Body Mass Index (BMI) (r=0.51, p<0.001), Waist to Hip Ratio (WHR) (r=0.41, p<0.001), Neck Circumference (NC) (r=0.50, p<0.001), Fasting glucose (r=0.44, p<0.001), Total Cholesterol (TC) (r=0.41, p<0.001), Triglycerides (TG) (r=0.39, p<0.001), Low-Density Lipoprotein-cholesterol (LDL-c) (r=0.39, p<0.001), Very Low-Density Lipoprotein (VLDL) (VLDL) (r=0.39, p<0.001), Systolic Blood Pressure (SBP) (r=0.52, p<0.001), and Diastolic Blood Pressure (DBP) (r=0.45, p<0.001), and negatively correlated with High-Density Lipoprotein-cholesterol (HDL-c) (r=-0.20, p<0.002).

Conclusion: The present study revealed a good relationship between serum visfatin and the anthropometric and biochemical parameters. The current data belief is that visfatin may be a promising biomarker for predicting metabolic syndrome and its associated disorders particularly in overweight and obese adults.

Keywords: Body fat, Enzyme-linked immunosorbent assay, Lipid profile, Obesity

INTRODUCTION

There is now evidence from many studies that overweight and obesity have reached epidemic proportions globally, and perhaps India is undergoing a rapid epidemiological transition from underweight to an overweight/obese population [1,2]. Epidemiological studies have described that the prevalence of men and women living with overweight/obesity is observed to be 38.4% and 36.2%, respectively [3,4]. The Indian National Family Health Survey-4 reported that in the 10-year period from 2005 to 2006 to 2015 and 2016, obesity among women between the ages of 15 and 49 years increased from 13% to 21%. During the same period, obesity among men between the ages of 15 and 49 years increased from 9.3% to 19% [5]. Recent reports suggest that obesity is one of the most neglected public health problems and is also associated with unemployment, social disadvantages, and reduced socio-economic productivity [6].

The medical literature abounds with evidence that obesity, particularly central obesity, is a risk factor for metabolic syndrome. Metabolic syndrome has also been found to be associated with a 2 fold increased risk of cardiovascular mortality, type 2 diabetes mellitus, and certain cancers such as endometrial, breast, ovarian, prostate, liver, gall bladder, kidney, and colon [7,8]. Recently, an elegant study revealed that the prevalence of metabolic syndrome has dramatically increased in India. It has been found that the overall pooled prevalence of metabolic syndrome among the adult population was 30% [9].

It is commonly accepted that adipose tissue is an endocrine organ that produces molecules with important functions in the human body called adipokines. Visfatin is a recently discovered adipocytokine with a 55-kDa protein consisting of 491 amino acids, previously called Pre-β cell Colony Enhancing Factor (PBEF). Visfatin is secreted by macrophages (CD 14+) in visceral adipose tissue induced by hypoxia [10,11]. Under hypoxic conditions, hydroxylase processes are rendered inactive; consequently, hypoxia-inducible factor-1a is stabilised and moves into the nucleus where it binds to hypoxia response elements within target genes and initiates the transcription of visfatin [12,13].

Visfatin is synthesised and released by inflammatory cells in adipose tissue as well as adipocytes [11]. It has the ability to activate human leukocytes, induce the synthesis of proinflammatory cytokines and adhesion molecules, regulate the maturation of leukocytes B, and inhibit apoptosis of neutrophils [14]. Visfatin was first discovered by Fukuhara A et al., as an adipokine, and it has been stated that visfatin is secreted from visceral fat only and its blood shows insulin-mimetic effects [10]. Recent studies have mentioned that visfatin production is not limited to visceral fat alone and it is expressed and secreted in many other tissues such as adipose tissue, chondrocytes, heart,

pancreas, liver, skeletal muscle, and plays a vital role in glucose and lipid metabolism [15,16]. Visfatin may be a promising biomarker for predicting obesity, insulin resistance, metabolic syndrome, diabetes mellitus, cardiovascular diseases, and cancer [17,18]. Contradictory results have been documented regarding analyses correlating visfatin and obesity. Chan, TF et al., have reported positive correlations between visfatin and obesity [19], whereas Pagano C et al., have demonstrated low plasma visfatin levels in obesity [20].

To the best of the authors knowledge, a similar study has not been reported so far with respect to serum visfatin levels in Indian overweight and obese adults with and without metabolic syndrome. It is better to identify adults with overweight and obesity who are at risk of metabolic syndrome at an early stage to prevent the future development of type 2 diabetes mellitus, cardiovascular disease, and cancer. The present study, therefore, aimed to determine the levels of serum visfatin in overweight and obese adults with and without metabolic syndrome in the Indian population and its correlation with anthropometric and biochemical parameters.

MATERIALS AND METHODS

The present comparative cross-sectional study was conducted at the Department of Biochemistry, Tomo Riba Institute of Health and Medical Sciences, Naharlagun, Arunachal Pradesh, India, between September 2022 and October 2023. This study was approved by the Institutional Ethics Committee (IEC No. TRIHMS/ETHICS/01/2019-20/14). Written informed consent forms from all participants were obtained before the commencement of the study.

Inclusion criteria: A total of 200 subjects in the age group of 20-70 years (50 subjects with overweight, 50 subjects with obesity without metabolic syndrome, 50 subjects with obesity with metabolic syndrome, and 50 healthy controls) were included in the present study. Subjects with a BMI of 18.5-24.9 Kg/m^2 were considered healthy controls, subjects with a BMI of 25-29.9 Kg/m^2 were considered overweight, and subjects with a BMI >30 were considered obese. Subjects with metabolic syndrome were identified using International Diabetes Federation (IDF) criteria [21].

Exclusion criteria: Adults with secondary causes of obesity such as Cushing's syndrome, polycystic ovary syndrome, hypogonadism, hypothyroidism, and drug-induced obesity, adults with a known history of diabetes, cardiovascular diseases, cancer, and relevant drug treatment, and also adults undergoing treatment for any other co-morbid conditions, were excluded.

Sample size calculation: The sample size was calculated by a qualified statistician assuming alpha=0.001, beta=0.1 (power=90%).

$$\frac{2s^2 (Z_{1-b}+Z_{1-a/2})^2}{(m_1-m_2)^2}$$

Based on the previous study [17], the sample size was calculated to be 200.

Study Procedure

Anthropometric measurements such as height, weight, BMI, WHR, and NC were recorded. Weight was measured using a beam balance to the nearest 0.1 kg, and height was measured to the nearest centimetre using a non stretchable tape. Abdominal girth was measured at the level of the umbilicus with the subject relaxed and in a standing posture. Hip girth was measured at the widest point of the hips at the level of the greater trochanter with the subject standing with both feet together. Waist-to-hip ratio was calculated from these measurements. NC was measured at the midway point of the neck between the mid-cervical spine and mid-anterior neck to within 1 mm using a non stretchable plastic tape with subjects standing upright; in men with a laryngeal prominence, it was measured just below the prominence.

According to IDF criteria, a person is defined as having metabolic syndrome if they have central obesity (waist circumference >90 cm in men, >80 cm in women) along with any 2 of the following factors: 1) TG 150 mg/dL or higher; 2) HDL levels <40 mg/dL in men or <50 mg/dL in women; 3) BP 130/85 mm Hg or higher; 4) Fasting blood glucose >100 mg/dL [21]. Blood pressure levels were also recorded for all subjects using a mercury sphygmomanometer. Venous blood samples were collected after 12 hours of fasting from all subjects. Serum was separated, and the samples were stored at -20°C until analysis. The lipid profile, which includes TC, TG, LDL-c, HDL-c, and Fasting glucose, was analysed by a fully auto analyser (Agappe Mispa Nano Plus). All samples for the lipid profile and fasting glucose were analysed immediately. Serum visfatin levels (Fine Biotech Co., Ltd.) were measured using the Alere ELISA Reader AM 2100 [Table/Fig-1].

S. No.	Parameters	Method	Reference range*	Cut-off range
1.	Serum Total Cholesterol (TC)	CHOD-PAP	≤200 mg/dL	3-600 mg/dL
2.	Serum Triglycerides (TG)	GPO-PAP	40-140 mg/dL	2-1000 mg/dL
3.	Serum Low Density Lipoprotein-cholesterol (LDL-c)	Selective solubilisation method	5-130 mg/dL	1-700 mg/dL
4.	Serum High Density Lipoprotein-cholesterol (HDL-c)	Selective inhibition method	Males: 35-80 mg/dL Females: 42-88 mg/dL	1-150 mg/dL
5.	Fasting glucose	GOD-POD	74-100 mg/dL	1-600 mg/dL
6.	Serum Visfatin	Sandwich ELISA	Range mentioned in the kit insert: 0.313-20 ng/mL (Sensitivity: 0.188 ng/mL)	

[Table/Fig-1]: Method and reference ranges for the biochemical parameters are shown in the table.
CHO-PAP: Cholesterol oxidase peroxidase; GPO-PAP: Glycerol-3-phosphate oxidase-phenol-aminophenazone; GOD-POD: Glucose oxidase-peroxidase coupled
*The Reference range mentioned for lipid profile and fasting glucose was taken from the respective kit inserts (Agappe Mispa nano). There is no defined standard reference range for serum visfatin, as it is a special parameter that has not been studied extensively. The Reference range for serum visfatin was taken from the kit which we have used (Fine Biotech Co., Ltd Catalogue No.: EH0651)

STATISTICAL ANALYSIS

Statistical analysis of the data was done by using Statistical Package for Social Sciences (SPSS) version 23.0 (SPSS Inc., Chicago, IL, USA). Descriptive analysis: The categorical variables were analysed using frequency and percentages, and the continuous variables were analysed by calculating Mean±Standard Deviation (SD). For inferential analysis, the numerical data were analysed using the t-test and correlation, while the categorical data were analysed using the Chi-square test. Correlation analysis was done by Pearson's correlation coefficient at a 5% level of significance.

RESULTS

The present study examined 200 subjects (50 healthy controls, 50 overweight, 50 obese without metabolic syndrome, and 50 obese with metabolic syndrome). Out of the 200 subjects, 69 were men and 131 were women with an age range of 20-70 years. A total of 48 subjects were excluded from the study due to refusal of the study protocol, incomplete data, or previous diagnosis of various disorders. The final sample size was 200 after excluding the 48 subjects.

The anthropometric parameters were found to be significantly higher in overweight individuals (except for WHR) and in those who were obese with or without metabolic syndrome. Serum visfatin levels were significantly higher in overweight individuals (1.7±0.3 ng/mL), obese individuals without metabolic syndrome (4.3±3.2 ng/mL), and obese individuals with metabolic syndrome (10.9±6.6 ng/mL) (p<0.001) compared to the control group (1.0±0.2 ng/mL) [Table/Fig-2].

Serum visfatin levels were positively correlated with BMI (r=0.51, p<0.001), WHR (r=0.41, p<0.001), NC (r=0.50, p<0.001), Fasting glucose (r=0.44, p<0.001), TC (r=0.41, p<0.001), TG (r=0.39,

Parameter	Healthy control (n=50)	Healthy control vs overweight (n=50)	Healthy control vs obese without Met S (n=50)	Healthy control vs obese with Met S (n=50)
Age (Years)	41.5±13.4	37.1±10.9	40.6±12.7	42.0±9.2
BMI (Kg/m²)	21.5±1.8	27.2±1.2**	31.4±1.9**	32.8±2.7**
WHR	0.79±0.059	0.81±0.063ⁱ	0.9±0.0**	0.9±0.1**
NC	14.1±0.8	14.6±0.7*	15.8±1.0**	16.2±1.1**
Glucose (mg/dL)	90.2±8.8	95.2±10.7*	94.9±8.5*	108.5±10.4**
TC (mg/dL)	167.5±32.3	177.1±37.7ⁱ	190.4±26.8*	219.6±42.3**
TG (mg/dL)	121.9±33.0	162.8±6.8**	150.2±41.2**	242.0±9.61**
HDL-c (mg/dL)	48.8±11.6	43.2±9.1*	49.1±6.2ⁱ	40.6±7.0*
LDL-c (mg/dL)	92.2±26.2	104.3±36.1ⁱ	107.5±22.4*	135.0±40.5**
VLDL (mg/dL)	24.1±6.8	32.5±13.6*	29.8±8.2*	48±18.8**
SBP (mmHg)	116.2±6.4	117.6±5.6ⁱ	117.8±6.8ⁱ	133.8±10.7**
DBP (mmHg)	79.6±6.7	79.4±5.5ⁱ	79.8±6.8 NS	87.8±8.2**
Visfatin (ng/mL)	1.0±0.2	1.7±0.3**	4.3±3.2**	10.9±6.6**

[Table/Fig-2]: Comparison of various parameters between healthy control vs other groups.
**p<0.001; *p<0.005; Ip >0.05; BMI: Body mass index; WHR: Waist to hip ratio; NC: Neck circumference; TC: Total cholesterol; TG: Triglycerides; HDL-c: High-density lipoprotein-cholesterol; LDL-c: Low-density lipoprotein-cholesterol; VLDL: Very low density lipoprotein; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; Met S: Metabolic syndrome

p<0.001), LDL-c (r=0.39, p<0.001), VLDL (r=0.39, p<0.001), SBP (r=0.52, p<0.001), and DBP (r=0.45, p<0.001), and negatively correlated with HDL-c (r=-0.20, p<0.002). The relationship between serum visfatin, biochemical, and anthropometric parameters for all subjects is shown in [Table/Fig-3].

Parameter	r-value	p-value
BMI	0.511	0.001
WHR	0.416	0.001
NC	0.503	0.001
Glucose	0.447	0.001
TC	0.414	0.001
TG	0.394	0.001
HDL-c	-0.206	0.002
LDL-c	0.391	0.001
VLDL	0.394	0.001
SBP	0.522	0.001
DBP	0.450	0.001

[Table/Fig-3]: Pearson's correlation analysis between serum visfatin and anthropometric, biochemical variables of the study subjects.
*(Yes including controls); BMI: Body mass index; WHR: Waist to hip ratio; NC: Neck circumference; TC: Total cholesterol; TG: Triglycerides; HDL-c: High density lipoprotein-cholesterol; LDL-c: Low density lipoprotein-cholesterol; VLDL: Very low density lipoprotein; SBP: Systolic blood pressure; DBP: Diastolic blood pressure

In the present study, when comparing serum visfatin levels between males and females in each group, it was found that although males had slightly higher serum visfatin levels in all groups than females, the difference was statistically non significant. The descriptive statistics between males and females in each group are shown in [Table/Fig-4].

The present study observed a positive correlation between serum visfatin and anthropometric parameters such as BMI, WHR, and NC, as well as the lipid profile which includes TC, TG, LDL-c, VLDL, and Fasting glucose in both males and females. Serum visfatin was negatively correlated with HDL-c in both males and females, but the correlation was found to be weak [Table/Fig-5].

DISCUSSION

It is well established that visfatin is an adipocytokine, and its increased levels could play a vital role in central obesity [7,8]. Central obesity is a risk factor for metabolic syndrome, and metabolic syndrome has also been found to be associated with an increased risk of cardiovascular mortality, type 2 diabetes mellitus, and cancers [9].

In the past decade, there has been a surge in the prevalence of metabolic syndrome in Indian adults, especially in urban areas [22,23]. Mohammad GS reported metabolic syndrome prevalence ranges from 25-45%, and Dakshinamurthy S et al., reported it to be 38.2% [22,23]. The present study shows elevated levels of serum visfatin in overweight individuals, as well as in obese individuals with and without

Parameter	Control		Overweight		Obese without Met S		Obese with Met S	
	Males (n=17)	Females (n=33)	Males (n=13)	Females (n=37)	Males (n=16)	Females (n=34)	Males (n=23)	Females (n=27)
Age (Years)	42.1±12.7	41.1±13.9	37.6±12.2	36.8±10.6	42.1±10.8	39.8±13.5	41.5±9.1	42.3±9.5
BMI (Kg/m²)	21.7±1.7	21.3±1.7	27.0±1.2**	27.2±1.1**	30.8±1.2**	31.7±2.1**	33.1±3.3**	32.5±1.9**
WHR	0.78±0.06	0.78±0.06	0.8±0.07ⁱ	0.81±0.06ⁱ	0.86±0.05**	0.86±0.04**	0.88±0.05**	0.87±0.04**
NC	14.6±0.69	13.8±0.74	14.9±0.65ⁱ	14.5±0.68*	16.4±1.0**	15.4±0.87**	16.4±1.0**	15.9±1.0**
Glucose (mg/dL)	88.8±8.0	90.8±9.2	95.7±15.1ⁱ	95.0±8.8ⁱ	96.6±11.6ⁱ	93.9±6.5ⁱ	108.2±9.8**	108.7±11.0**
TC (mg/dL)	164.6±37.5	168.9±29.7	166.9±40.4ⁱ	180.6±36.5ⁱ	183.1±25.8ⁱ	193.8±26.9**	223.0±41.0**	216.6±43.9**
TG (mg/dL)	120.6±34.3	122.5±32.7	171.7±61.9*	159.6±70.6*	137.8±18.2*	156.0±47.5**	261.1±132.1**	225.6±45.1**
HDLC (mg/dL)	48.5±11.6	48.9±11.7	41.3±6.5ⁱ	43.8±9.8ⁱ	47.0±3.4ⁱ	50.1±6.9ⁱ	39.3±7.1**	41.7±6.7**
LDL (mg/dL)	87.4±29.7	94.5±24.2	93.6±33.6ⁱ	107.9±36.6ⁱ	103.5±25.5ⁱ	109.3±20.9*	139.9±38.1**	130.7±42.5**
VLDL (mg/dL)	24.0±6.9	24.1±6.8	34.2±12.3*	31.8±14.1*	27.3±3.6ⁱ	30.9±9.4*	51.5±25.8**	45.0±9.0**
SBP (mmHg)	118.2±8.0	115.1±5.0	116.9±6.3ⁱ	117.8±5.3ⁱ	119.3±6.8ⁱ	117.0±6.7ⁱ	135.2±11.6**	132.5±9.8**
DBP (mmHg)	78.2±6.3	80.3±6.8	79.2±4.9ⁱ	79.4±5.7ⁱ	80.0±6.3ⁱ	79.7±7.1ⁱ	88.2±10.2**	87.4±5.9**
Visfatin (ng/mL)	0.95±0.20	1.0±0.19	1.76±0.33**	1.64±0.34**	4.6±3.9**	4.1±2.9**	11.5±6.8**	10.4±6.4**

[Table/Fig-4]: Comparison between males and females of different groups.
**p<0.001; *p<0.005; ⁱp>0.05; BMI: Body mass index; WHR: Waist to hip ratio; NC: Neck circumference; TC: Total cholesterol; TG: Triglycerides; HDL-c: High density lipoprotein-cholesterol; LDL-c: Low density lipoprotein-cholesterol; VLDL: Very low density lipoprotein; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; Met S: Metabolic syndrome

Parameter	Males (n=69)		Females (n=131)	
	r-value	p-value	r-value	p-value
BMI	0.54	0.001	0.48	0.001
WHR	0.48	0.001	0.35	0.001
NC	0.51	0.001	0.47	0.001
Glucose	0.41	0.001	0.46	0.001
TC	0.57	0.001	0.29	0.001
TG	0.42	0.001	0.34	0.001
HDL-c	-0.28	-0.01	-0.14	- 0.11
LDL-c	0.57	0.001	0.25	0.003
VLDL	0.42	0.001	0.34	0.001
SBP	0.50	0.001	0.51	0.001
DBP	0.45	0.001	0.43	0.001

[Table/Fig-5]: Pearson's correlation analysis between serum visfatin and anthropometric, biochemical variables for males and females.
BMI: Body mass index; WHR: Waist to hip ratio; NC: Neck circumference; TC: Total cholesterol; TG: Triglycerides; HDL-c: High density lipoprotein-cholesterol; LDL-c: Low density lipoprotein-cholesterol; VLDL: Very low density lipoprotein; SBP: Systolic blood pressure; DBP: Diastolic blood pressure

metabolic syndrome. In contrast, one study has shown less visfatin in obesity [20]. Taken together, the present findings suggest that visfatin is synthesised and released by inflammatory cells in adipose tissue as well as adipocytes [11], and subclinical inflammation in overweight and obesity may enhance the expression of visfatin [24]. Similarly, the current study findings also suggest that inflammation is a condition which sits at the very base of atherogenesis, which is a major consequence of metabolic syndrome, and adipose tissue impacts all organs by the synthesis of adipokines [25,26].

In the present study, anthropometric parameters such as BMI and NC were significantly elevated in overweight individuals and in obese individuals with and without metabolic syndrome, whereas WHR was non significant in overweight individuals and elevated in both obese individuals with and without metabolic syndrome. Visfatin levels positively correlated with anthropometric parameters. At this juncture, it is appropriate to mention that body fat possibly influence the concentration of circulating visfatin in overweight and obesity, and these findings are consistent with those reported by others [24,27,28]. Fasting glucose levels were significantly elevated in overweight individuals and in obese individuals with and without metabolic syndrome, and we found a positive correlation between visfatin and fasting glucose.

The present study findings strongly suggest that visfatin influences glucose metabolism, as plasma visfatin levels may be related to progressive β cell deterioration and increased insulin resistance, possibly mediating the link of the adipocytokine with diabetes. Visfatin can be stimulated under a hyperglycemic environment and may play a role in the pathogenesis of diabetes through interaction with the insulin receptor, phosphorylating tyrosine and insulin substrates 1 and 2. The results of this study are also consistent with other studies that have shown a positive relation between visfatin and glucose [24,29]. On the other hand, some studies have reported no association between visfatin and diabetes [30,31].

Previously published studies, as well as observations from the present study, show increased levels of TC, TG, LDL-c, and VLDL in overweight individuals and in obese individuals with and without metabolic syndrome compared to the control group. There was a substantial reduction in HDL-c levels in the other groups compared to the control group. Serum visfatin showed positive correlations with TC, TG, LDL-c, and VLDL, while the association between visfatin concentration and HDL-c was negative. Therefore, the increase in TC and LDL-c levels may be attributed to high saturated fat diets resulting from increased fat intake, which will impede the process of filtering LDL-c particles as it reduces the effectiveness of the LDL-c receptors [32]. The increase in TG levels may be caused

by obesity-induced insulin resistance, as the decrease in insulin levels inhibits the activity of the lipoprotein lipase enzyme, leading to a decrease in the removal of TG in the chylomicron and VLDL [33]. The decrease in HDL-c occurs because of high fat intake, causing an increase in TG and TC levels in tissues and blood vessels, which in turn creates an impediment to HDL-c and reduces its efficiency in transporting cholesterol from tissues to the liver. The current data belief is that visfatin influences lipid homeostasis similar to insulin and is responsible for adipocyte proliferation, differentiation, and TG metabolism [34]. However, one study found no association between visfatin and TC, TG, LDL-c, and HDL-c [17].

There are no marginal differences in SBP and DBP between the overweight and obese individuals without metabolic syndrome group when compared to the control group. However, we found a significant elevation in the obese individuals with metabolic syndrome group. Furthermore, in this study, serum visfatin levels positively correlated with SBP and DBP. The possible biological mechanism could be that the renin-angiotensin system and sympathetic nervous system interact with proinflammatory cytokines. These cytokines affect vascular function and endothelium-derived factors involved in blood pressure regulation. Endothelial dysfunction is associated with many forms of hypertension. Therefore, visfatin measurement might have potential benefits in the detection of hypertension. The present study is also in agreement with an earlier study reported [35].

Limitation(s)

The potential limitation of the present study was the lack of detailed measurement of physical activity and dietary habits of the study participants.

CONCLUSION(S)

The findings of the present study show higher serum visfatin levels in overweight individuals, as well as in obese individuals with and without metabolic syndrome, and a consistent positive correlation between serum visfatin and anthropometric, clinical, and biochemical parameters in all the groups. HDL-c levels were found to be negatively correlated with serum visfatin. These findings imply that visfatin is synthesised and released by inflammatory cells in adipose tissue as well as adipocytes, and subclinical inflammation in overweight and obesity may enhance the expression of visfatin. Inflammation is a major consequence of metabolic syndrome. Possibly body fat influences the concentration of circulating visfatin. The current data belief is that visfatin may be a promising biomarker for predicting metabolic syndrome and its associated disorders, particularly in overweight and obese adults. Furthermore, future research on a larger sample size should endeavor to elucidate the exact mechanism of visfatin in overweight and obesity.

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