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Original Research Article

Evaluation of Serum Visfatin, a Marker of Endothelial Dysfunction and Total Oxidant Status Using Tbars in Post-Menopausal Women

P. Saraswathi[1], S. Santhini[2]

[1]Assistant Professor, Department of Biochemistry, Central lab, Tamilnadu Government Multi Super Specialty Hospital, Omandurar Estate, Chennai

[2]Assistant Professor, Department of Biochemistry, Government Vellore Medical College, Vellore

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Corresponding Author: Dr. P. Saraswathi

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Abstract:

Background: Menopause is associated with hormonal decline, particularly estrogen deficiency, which predisposes women to cardiovascular disease, metabolic dysfunction, and oxidative stress. Visfatin, an adipocytokine implicated in endothelial dysfunction and inflammation, alongside markers of oxidative stress such as Thiobarbituric Acid Reactive Substances (TBARS), may provide insights into cardiovascular risk in postmenopausal women. Aim of this study is to evaluate serum visfatin levels and oxidative stress status in obese and non-obese postmenopausal women.

Methods: This cross-sectional study was conducted in a tertiary care hospital with 55 postmenopausal women, stratified into obese (n=30) and non-obese (n=25) groups based on BMI. Serum visfatin was measured using ELISA, and oxidative stress was assessed by TBARS assay. Clinical, anthropometric, hematological, and biochemical parameters were recorded. Statistical analysis included Student's t-test and Pearson's correlation, with $p<0.05$ considered significant.

Results: Obese postmenopausal women exhibited significantly elevated serum visfatin (19.58 ± 1.18 vs. 11.37 ± 5.12 nmol/mg, $p<0.01$) and TBARS levels (5.97 ± 1.00 vs. 4.48 ± 0.36 ng/L, $p<0.05$) compared to non-obese women. Positive correlations were observed between BMI and visfatin ($r=0.951$, $p=0.001$) and between BMI and TBARS ($r=0.553$, $p=0.05$). Additionally, obese women showed increased protein carbonyls and reduced antioxidant capacity.

Conclusion: Obesity in postmenopausal women is associated with elevated visfatin and oxidative stress markers, indicating early endothelial dysfunction and increased cardiovascular risk. These findings highlight visfatin and TBARS as potential biomarkers for cardiovascular risk assessment in postmenopausal women. Lifestyle modification, weight reduction, and antioxidant supplementation may help mitigate these risks. Further longitudinal studies are needed to establish causality and therapeutic implications.

Keywords: Visfatin, TBARS, Oxidative Stress, Obesity, Postmenopausal Women.

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Introduction

Menopause represents a critical health transition characterized by hormonal decline, particularly estrogen deficiency, leading to increased cardiovascular disease risk, metabolic dysfunction, and oxidative stress in women [1]. This postmenopausal phase creates a complex interplay of inflammatory, metabolic, and vascular changes that significantly impact women's health outcomes.

Visfatin, also known as nicotinamide phosphoribosyltransferase (NAMPT), is an emerging 52-kDa adipocytokine with dual functions as both an intracellular NAD+ biosynthesis enzyme and an extracellular insulin-mimetic cytokine [2]. Elevated serum visfatin levels are associated with obesity, type 2 diabetes, cardiovascular disease, and inflammatory disorders, making it a potential biomarker for metabolic and vascular dysfunction [3]. Endothelial dysfunction, an early manifestation of cardiovascular disease, is particularly relevant in postmenopausal women due to estrogen deficiency compromising vascular homeostasis [4]. Visfatin has emerged as a marker of endothelial dysfunction, promoting endothelial cell activation, increasing vascular permeability, and stimulating inflammatory cytokine production, thereby directly impacting cardiovascular health [5].

Post-menopausal women experience heightened oxidative stress due to estrogen deficiency, aging, and metabolic changes [6]. Estrogen's antioxidant properties normally protect against reactive oxygen species, but its decline results in compromised antioxidant defense mechanisms and increased sus-

ceptibility to oxidative damage, contributing to cardiovascular disease and other age-related disorders. Total Oxidant Status (TOS) and Thiobarbituric Acid Reactive Substances (TBARS) serve as reliable biomarkers for evaluating oxidative stress levels, with TBARS primarily measuring lipid peroxidation products like malondialdehyde [7]. These markers consistently show elevated levels in post-menopausal women, reflecting increased oxidative damage and providing insights into cellular integrity.

Obesity significantly affects post-menopausal women, with prevalence rates increasing substantially following menopause [8]. The combination of hormonal changes and metabolic alterations creates a pro-inflammatory environment that exacerbates cardiovascular risk. Obese individuals typically exhibit elevated visfatin concentrations, which may contribute to systemic inflammation and endothelial dysfunction [9].

Despite growing interest in visfatin and oxidative stress biomarkers, limited research has specifically examined their relationships in post-menopausal women, particularly when stratified by obesity status. The evaluation of serum visfatin levels and oxidative stress parameters in this population represents a critical research priority for identifying reliable biomarkers for cardiovascular risk assessment and therapeutic monitoring [10].

Based on this aim of our study is to evaluate serum visfatin level and oxidative stress status in obese and non-obese post-menopausal women. Thereby to compare serum visfatin level between obese and non-obese post-menopausal women and also to analyze Total Oxidative Status (TBARS) in post-menopausal women.

Materials and Methods

This cross-sectional study was conducted in the Department of Biochemistry in collaboration with the Department of Obstetrics and Gynaecology at a tertiary care teaching hospital after obtaining approval from the Institutional Human Ethics Committee. Written informed consent was obtained from all participants. A total of sixty post-menopausal women were enrolled under gynecologist supervision, of which fifty-five completed the study. Participants were divided into two groups based on BMI: Group I included 30 obese women and Group II included 25 non-obese women. The inclusion criterion was post-menopausal women at least five years past menopause. Women with thyroid, liver, renal diseases, hysterectomy, ischemic heart disease, or cancer were excluded. Fasting venous blood samples (5 mL) were collected, and serum was separated for baseline investigations. For estimation of visfatin, total oxidant status, and antioxidant status, serum samples were stored at –80°C until analysis.

Clinical and anthropometric measurements included BMI, waist-hip ratio, and blood pressure. Hematological parameters assessed were hemoglobin, total WBC, platelet, and RBC counts. Biochemical investigations included fasting glucose, urea, creatinine, uric acid, lipid profile, and liver function tests. Special parameters studied were serum visfatin by ELISA and oxidative stress markers.

Lipid peroxidation was assessed by the thiobarbituric acid reactive substances (TBARS) method, based on the reaction of malondialdehyde (MDA) with TBA to form a red pigment, measured at 535 nm and expressed as nmol/mL. Serum visfatin was measured using a Sandwich-ELISA kit, with results interpreted colorimetrically at 450 nm. The assay had a detection range of 0.31–20 ng/mL, sensitivity of 0.19 ng/mL, and required 100 µL of serum per well.

Statistical analysis was done using SPSS version 25.0, Software. Unpaired student's t test was applied for comparison of the groups and Pearson's correlation coefficient was applied for analysing the relationship between BMI and serum visfatin, TBARS, FRAP, protein carbonyl. The level of significance was fixed at $p<0.05$.

Results

We started with analysing the baseline parameters between group I (obese) and group II (non-obese) post menopausal women. There was significant difference in WHR and serum Total cholesterol among the two groups but no significant difference with respect to blood pressure, serum AST and blood urea levels. Also there was significant difference in Total cholesterol in obese women compared to non obese women. There is no significant difference in other lipid profile parameters in obese and non obese post menopausal women.

Table 1: Comparison of Serum Visfatin, Tbars, In Obese & Non Obese Post-Menopausal Women.

Parameter	Group I (OBESE) n= 30	Group II (Non Obese) n= 25	p value
TBARS (ng/l)	5.97±1.00	4.48±0.36	<0.05*
VISFATIN (nmol/mg)	19.58±1.18	11.37±5.12	<0.01**

Table 1 shows serum visfatin, TBARS in obese and non obese post menopausal women. Serum visfatin and TBARS were significantly elevated in obese women.

Table 2: Pearson Correlation Analysis of Bmi with Parameters.

Parameter	R Value	P Value	Correlation
Visfatin	0.951	0.001	Positive
Tbars	0.553	0.05	Positive

Table 2 shows Pearson correlation analysis between visfatin,TBARS and BMI. There was significant positive correlation between BMI and visfatin,TBARS in obese women and also there was significant negative correlation between visfatin and FRAP. In obese, there was about 1.5 fold increase in serum visfatin levels.

There was significant increase in protein carbonyls in obese women than non obese women.

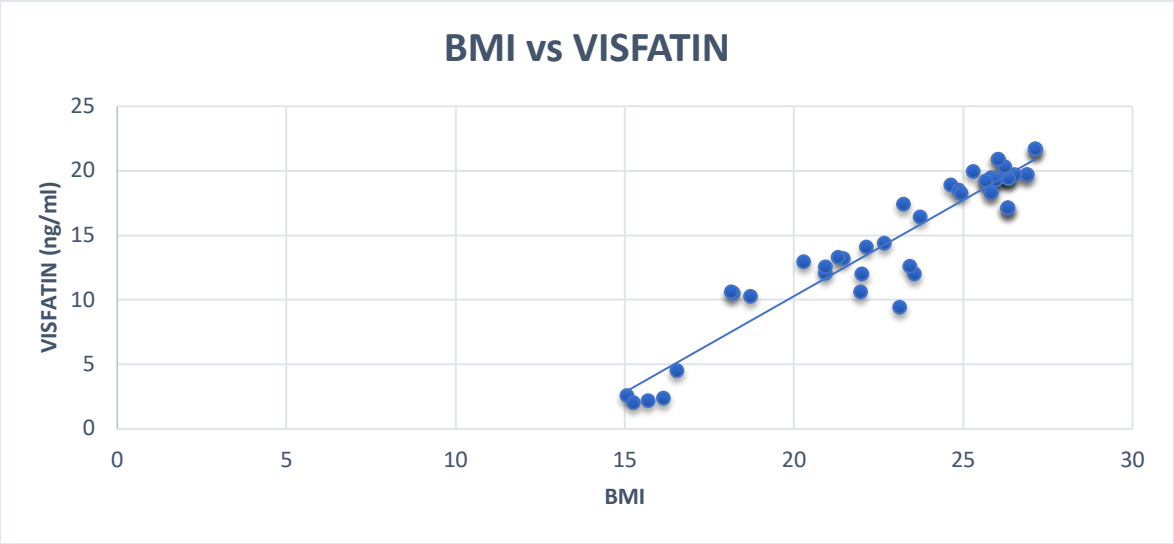

Figure 1: Pearson Correlation of BMI Vs Visfatin in Obese and Non-Obese Post-Menopausal Women.

Discussion

According to Leuzzi C et al. [11], cardiovascular disease (CVD) is the primary cause of morbidity and mortality among women globally. Gender-based disparities in CVD are lessened after menopause and the resulting loss of hormonal effects, however the exact cause is still unknown. The apparent cardioprotective effects of endogenous estrogen appear to prevent CVD in premenopausal women. Despite the fact that earlier research has highlighted the benefits of estrogen, there is currently no solid proof that it lowers the risk of CVD.

According to the BMI Guidelines provided by the Prevention and Management of Obesity and Metabolic Syndrome Group, postmenopausal women with a BMI of less than 22.5 kg/M2 were classified as non-obese and those with a BMI of more than 22.5 kg/M2 as obese in the current study. Regarding age, there was no discernible difference. According to certain research by Casiglia E et al, menopause is typically blamed for cardiovascular problems [12].

Due to an increase in total body fat, postmenopausal women often gain weight starting in the first year of menopause and undergo a redistribution of body fat from a gynoid to an android pattern. Body shape and fat distribution were not differentiated by BMI. Regardless of BMI, central obesity, which is defined by a comparatively high distribution of belly fat, has been linked to an increased risk of death. WHR, a measure of central obesity, was considerably higher in obese women than in non-obese women. According to Yangbo Sun et al., women who were normal weight but had central obesity, as indicated by their waist circumference [WC], were more likely to die than those who were normal weight but did not have central obesity. [13]

A pro-atherogenic lipid profile linked to menopause includes elevated triglyceride (TG) and low-density lipoprotein cholesterol (LDL-C) and decreased HDL-C levels, central adiposity, and elevated diastolic blood pressure, all of which increase the risk of cardiovascular disease. Since there was no discernible difference in blood pressure or lipid profiles between the groups in this trial, the greater risk of CVD in obese women could not be explained by these factors. According to NCEP (2002), elevated levels of triglycerides and low-density lipoprotein cholesterol (LDL-C) and decreasing levels of high-density lipoprotein cholesterol (HDL-C) were recognized risk factors for CVD. [14] Postmenopausal women who were obese had significantly higher levels of TBARS ($p<0.05$) and lower levels of total antioxidant capacity ($p<0.05$) than those who were not.

Cardiovascular illness is associated with higher levels of malondialdehyde (MDA), an end product of lipid peroxidation that is frequently evaluated as thiobarbituric acid (TBA) reactive substances (TBARS). An obvious sign of increased free radical production was elevated TBARS. In hypercholesterolemic individuals, lipid peroxidation has also been linked to the development of early carotid atherosclerosis. One of the best indicators of a 3-year increase in carotid wall thickness was found to be TBARS (Salonen J T et al). Therefore, a postmenopausal obese person's higher plasma TBARS would raise their risk of CVD. [15]

In recent years, endothelial dysfunction—the first and most important stage in the development of the atherosclerotic process—has been shown to be indicated by visfatin/Nampt (P. M. Vanhoutte et al). Endothelial dysfunction may be a contributing factor to oxidative stress in obesity. [16] Obese postmenopausal women had considerably higher levels of visfatin, a hallmark of endothelial dysfunction. Visfatin/Nampt levels have been found to be strongly positively correlated with poor vascular endothelial function in patients with type 2 diabetes. Visfatin/Nampt circulating levels have been suggested by Kadoglou et al. (2010) as a sign of progressive carotid atherosclerosis in patients with type 2 diabetes. [17] Additionally, a substantial positive connection between visfatin and BMI was found in the current investigation. Additionally, there was a notable difference between the two groups in the case of WHR.

In human unstable atherosclerotic lesions, visfatin expression was found to be elevated. Several researchers have suggested that visfatin/Nampt is a marker of atherosclerosis since elevated levels of the protein in the blood have been linked to the formation of atherosclerotic plaques [17]. Circulating inflammatory markers including IL-6 and MCP-1 have been shown to favourably correlate with visfatin/Nampt levels in coronary artery disorders (CAD), and more especially in acute coronary syndrome. Accordingly as per a study by TB Dahl et al, it has been suggested that the localization of visfatin/Nampt within the lesions may be connected to the instability of atherosclerotic plaque. [18]

In addition to the smooth muscle of atherosclerotic plaques, foam cells of unstable plaques from individuals who experienced an acute myocardial infarction also showed increased expression of visfatin/Nampt. The production of various cytokines and chemokines by endothelial cells, such as IL-6, IL-8, or MCP-1, is further upregulated by visfatin/Nampt. This promotes the adherence of human THP-1 monocytes to endothelial cells. Reactive oxygen species produced intracellularly have been implicated as the causative agents of visfatin/Nampt-induced endothelial cell inflammation. Additionally, a strong positive connection between oxidative stress indicators and BMI was identified. The ability of antioxidants in plasma to quench radicals has been measured comprehensively by total antioxidant status. The interaction and collaboration of different antioxidants determines the antioxidant state of plasma. According to earlier research done by Demirnag R et al, patients with coronary artery disease (CAD) had a decline in their overall antioxidant level [19]. According to a previous study done by Vincent K H et al, obese people had lower plasma levels of FRAP than non-obese controls. [20]

However, TBARS significantly increased in the current study, suggesting that obese postmenopausal women were experiencing increased oxidative stress. The overall antioxidant capacity significantly decreased, which may be the result of decreased availability of antioxidants or increased ROS production and antioxidant enzyme inactivation. It is found that, in comparison to non-obese postmenopausal women, obese postmenopausal women had considerably higher levels of serum visfatin, an early indicator of endothelial dysfunction, which was linked to oxidative stress and lower total antioxidant capacity.

Conclusion:

Obese postmenopausal women had considerably higher levels of serum visfatin, an early indicator of endothelial dysfunction, which was linked to oxidative stress and a reduction in total antioxidant capacity. When compared to postmenopausal women who are not fat, this may raise their risk of CVD. The effects of weight loss and dietary antioxidant supplements on lowering the risk of CVD in postmenopausal women require more research.

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