ORIGINAL ARTICLE

# RELATIONSHIP OF SERUM VISFATIN LEVELS WITH SERUM ELECTROLYTES, LIVER PROFILE, HEPATIC ENZYMES AND ANTHROPOMETRIC PARAMETERS IN PREGNANT WOMEN WITH PREECLAMPSIA AND ECLAMPSIA DURING 3RD TRIMESTER

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**Background:** Eclampsia and preeclampsia are among the serious complications of gestation and threaten the lives of both mother and foetus. A protein called visfatin, one of these adipokines, is evaluated for its effects on serum electrolytes, lipid profile and hepatic enzymes in preeclamptic and eclamptic patients. **Methods:** A sum of 234 pregnant women were enrolled in this cross-sectional study and divided in to 2 main groups, i.e., Group A (eclamptic/preeclamptic) Group B (control) pregnant women respectively. Serum visfatin levels (ng/mL), serum electrolytes and liver enzymes were determined for every patient, using relative diagnostic kits. Anthropometric measurements were also noted. **Results:** A total of 234 women (cases; n=160, controls; n=74) with gestation age of ≥20 weeks participated in this study. Group A had 86 (36.75%) women with preeclampsia and 74 (31.62%) women with eclampsia whereas Group B had 74 (31.62%) normotensive pregnant women. A strong significantly positive association was recorded for systolic ($R^2$=78.78; *p*-value <0.000) and diastolic blood pressure (BP) ($R^2$=78.52; *p*-value <0.000). Similar result was obtained for serum sodium ions ($R^2$=3.09; *p*-value <0.002) and chloride ions ($R^2$=7.36; *p*-value <0.000). Alkaline phosphatases (ALP) ($R^2$=63.47; *p*-value <0.000) had also shown a strong positive and statistically significant association with visfatin levels. **Conclusion:** Serum visfatin significantly decreased the sodium and chloride levels whereas the levels of potassium remained unaffected. A very strong and positive association of visfatin levels with levels of bilirubin and alkaline phosphatases was also observed (ALP) but it found no effect on aspartate transferases (AST).

**Keywords:** Preeclampsia; Eclampsia; Visfatin; Lipids; Electrolytes

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## INTRODUCTION

Eclampsia and preeclampsia are among the most serious and misunderstood complications that occurs during gestation and threaten the lives of both mother and fetus.[1] In Pakistan eclampsia and preeclampsia is one of the major causes of mortality and 1 out of 89 pregnant women die because of it.[2]

In human body the adipose tissues, through its multisystem effects, play a key role in regulating the endocrine functions, such as by secretion of plasma adipocytokines. A protein called visfatin[3], is one of these adipokines whose dysregulation leads to causation of disorders associated with obesity such as preeclampsia (during gestation)[4], hypertension[3], hyperlipidaemia[5], type-2 diabetes, small-for-gestational age foetus (SGA) etc[6].

In both normal pregnancy and the pathogenesis of preeclampsia, the relationship of serum levels of visfatin is highly anticipated.[7,8] The levels of visfatin proteins have been found raised in both endothelial dysfunction and vascular damage.[9,10] Besides adipose tissues and visceral adipocytes[11], visfatin is also expressed in adipose tissue such as neutrophils, macrophages and lymphocytes and is considered as an important marker of inflammation as well[12]. Evidences from the different studies propose that there is a twofold rise in the mean serum concentrations of visfatin in pregnant ladies that present with preeclampsia.[13,14] Furthermore, the concentration of visfatin is also found to be dependent on the severity of the preeclampsia.[15] Moreover, some studies also found that in preeclamptic women visfatin concentration revealed a negative correlation with Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), without showing any relation with body-mass-indices (BMI) and age of the patient.[16,17] But on the contrary some studies also suggested a very strong and positive correlation of circulating visfatin with BMI (overweightness)[18,19], depots of visceral fats[19] and waist-to-hip ratios[17].

Visfatin seems to affect many body activities and functions by promoting eclampsia and preeclampsia.[7,8] It could also be assumed that it may influence the functions of other body organs as well. Therefore, it's important to identify the clinical and epidemiological factors that could help us predict this condition. Our study focuses on to evaluate the relationship and effects of serum visfatin proteins levels on serum electrolytes and liver enzymes in pregnant women that present with eclampsia and preeclampsia and compare them with normal normotensive pregnant women during their 3rd trimesters.

## MATERIAL AND METHODS

This cross-sectional study was conducted in 3 main government teaching hospitals, named Medical Teaching Institute-Khyber Teaching Hospital (MTI-KTH); Medical Teaching Institute-Hayatabad Medical Complex (MTI-HMC), and Medical Teaching Institute-Lady Ready Hospital (MTI-LRH), all situated in Peshawar, Khyber Pakhtunkhwa (KP), Pakistan. The study was conducted from December 2013 to January 2018, after the approval from both Advance Research and Research Borad (ASRB) and ethical board of Khyber Medical University, Peshawar, Pakistan. Total of 11189 pregnant women were scrutinized to segregate potential subjects; out of which 234 (cases; n=160, controls; n=74) pregnant women, with mean gestation age of ≥20 weeks, proteinuria (+ive on dip-stick technique) and persistent hypertension (≥140/90mm of Hg) with or without edema, were included in this study. All the participants were further divided in to 2 main groups, i.e., Group A and Group B. Group A had 86 (36.75%) women with preeclampsia and 74 (31.62%) women with eclampsia whereas Group B had 74 (31.62%) normotensive pregnant women. All the potential participants were motivated to enrol themselves in the study and then a well-informed written consent was duly signed by all the willing study subjects.

Body mass indexes (BMI), weight and height were also noted using standard protocols. The diastolic and systolic blood pressures were recorded from left arm with the help of mercury Sphygmomanometer (Yamasu® -600 Japan). Three readings were taken at a time and their means were taken into account for the record.

Each participant donated about 5ml of her blood through aseptic technique to an adept phlebotomist. The blood was collected and stored in Gel and EDTA tubes (BD® USA) for further processing. All samples went through centrifugation at 3000 revolutions per minute (rpm) for at least 10 minutes to obtain cell free serum with the help standard centrifuge machine (Sarlorius® AG Weender Landhasses 94–108 Germany). Another 5 ml, uncoagulated, blood sample was also collected from each participant and stored in vacutainer tubes (BD® USA).

Visfatin concentrations (ng/mL), in serum, were measured using enzyme linked immune sorbent assay (ELISA) (Biovision® Research Products-CA94043-USA); serum electrolytes, liver functions tests through COBAS (automated) chemistry analyzer and other haematological parameters were measured with Sysmex (Automated) haematology analyzer. Roche® diagnostic kits were used to determine proteinuria.

MINTAB® 17 was used to carry out the comprehensive analysis of data. The data was presented as medians±interquartile ranges. Anderson-Darling test gave us the parametric and non-parametric nature of the data. Kruskall Wallis test, Mann-Whitney Test, Spearman rho test and Binary logistic regression were also applied where applicable to find significance ($p$-value ≤0.05).

## RESULTS

The median age of group A and B were 30±13.75 years and 31.1±12 years respectively. A total of 11189 pregnant women were scrutinized to segregate potential subjects; out of which 234 women (cases; n=160, controls; n=74) with gestation age of ≥20 weeks participated in this study. Group A had 86 (36.75%) women with preeclampsia and 74 (31.62%) women with eclampsia whereas Group B (controls) had 74 (31.62%) normotensive pregnant women.

Anthropometric relations did not show any significant changes ($p$-value>0.05) except for BMI and lean-body-mass ($p$-value<0.05) which were highly significant. A significant difference was seen in visfatin serum's concentrations (Table-1). The serum electrolytes (Na+ and Cl-) concentrations were found significantly decreased in "group A" except for serum potassium levels ($K^+$) in which no significant change was observed (Table-2).

Moreover, a strong and positive correlation was also noted for height (r=0.160, $p$-value 0.043) and of both group A and group B. For the remaining indices the association was insignificant (Table-3).

Furthermore, a strong significantly positive association was recorded for systolic ($R^2$=78.78; $p$-value <0.000) and diastolic blood pressure (BP) ($R^2$=78.52; $p$-value <0.000) (Table-6). Alkaline phosphatases (ALP) ($R^2$=63.47; $p$-value <0.000) had also shown a strong positive and statistically significant association with visfatin levels but did not affect aspartate transferases (AST) (Table-7). Similar result was obtained for serum $Na^+$ ($R^2$=3.09; $p$-value <0.002) and serum $Cl^+$($R^2$=7.36; $p$-value <0.000). For the remaining indices, the association was insignificant (Table-8).

**Table-1: Comparison of patients' demographic and anthropometric data and vesfatin levels.**

| Variables | Controls (n=74) | E/PE Patients (n=160) | *p*-Value* |
|---|---|---|---|
| | Med±IQR | Med±IQR | |
| **Age Group (year)** | 31.1±12 | 30±13.75 | **0.033** |
| **Height (m)** | 1.55±0.166 | 1.58±0.17 | 0.475 |
| **Weight (kg)** | 71±14 | 69±15 | 0.114 |
| **BMI (kg/m$^2$)** | 30.139±8.767 | 28.8235±8.38 | 0.069 |
| **Lean Body Mass (kg)** | 42.25±6.51 | 52.75±8.80 | **0.001** |
| **Lean mass (%)** | 71.59±7.13 | 76.33±7.8 | **0.02** |
| **Fat Mass (kg)** | 16.69±8.50 | 16.37±7.37 | 0.99 |
| **Body Fat (%)** | 26.85±11.27 | 22.55±7.71 | 0.36 |
| **Body Water (%)** | 49.4±7.63 | 52.05±5.21 | 0.43 |
| **BP (Systolic) (mm of Hg)** | 111±9.8 | 160±42.2 | **<0.001** |
| **BP (Diastolic) (mm of Hg)** | 71.3±10.1 | 100±10.2 | **<0.001** |
| **Visfatin (ng/mL)** | 1.95±1.81 | 3.78±3.87 | **<0.001** |
| **Bilirubin** | 4.30±0.43 | 0.81±0.39 | **<0.001** |

Kruskall-Wallis test implied for **p*-value; Med±IQR= Median±Inter Quartile Range; BP= Blood Pressure; E/PE= Eclamptic/Preeclamptic (Group A); Controls= Group B

**Table-2: Comparison of serum electrolytes in eclamptic/preeclamptic patients and controls**

| Electrolytes (mEq/L) | Controls (n=74) | E/PE Patients (n=160) | *p*-Value* |
|---|---|---|---|
| | Med±IQR | Med±IQR | |
| Potassium | 4.10±0.90 | 4.11±0.52 | 0.618 |
| Sodium | 141.00±9.00 | 136.05±5.54 | **<0.001** |
| Chloride | 99.00±4.00 | 103.05±8.11 | **<0.001** |

Mann-Whitney Test implied for **p*-value; Med±IQR= Median±Inter Quartile Range; E/PE= Eclamptic/Preeclamptic (Group A); Controls= Group B

**Table-3: Correlation analysis of visfatin with anthropetric and clinical parameters in eclamptic/preeclamptic patients and controls**

| Variables | r | | *p-Value | |
|---|---|---|---|---|
| | Controls (n=74) | &E/PE Patients (n=160) | Controls (n=74) | &E/PE Patients (n=160) |
| **Age Group (year)** | 0.071 | 0.069 | 0.549 | 0.369 |
| **Weight (kg)** | -0.086 | 0.006 | 0.468 | 0.945 |
| **Height (m)** | 0.101 | 0.160 | 0.390 | **0.043** |
| **BMI (kg/m$^2$)** | -0.128 | 0.104 | 0.277 | 0.192 |
| ****BP (Systolic) (mm of Hg)** | 0.028 | 0.012 | 0.816 | 0.882 |
| ****BP (Diastolic) (mm of Hg)** | -0.049 | -0.019 | 0.676 | 0.816 |

**p*-value based on Spearman rho test; **BP= Blood Pressure; &E/PE= Eclamptic/Preeclamptic (Group A); Controls= Group B

**Table-4: Correlation analysis of visfatin with hepatic enzymes in eclamptic/preeclamptic patients and controls**

| Enzymes (units/L) | r | | *p-Value | |
|---|---|---|---|---|
| | Controls (n=74) | &E/PE Patients (n=160) | Controls (n=74) | &E/PE Patients (n=160) |
| #ALP | 0.127 | -0.154 | 0.281 | 0.052 |
| $ALT | -0.050 | 0.044 | 0.671 | 0.579 |

**p*-value based on Spearman rho test; #ALP= Alkaline Phosphatase; $AST= Aspartate Transferase; E/PE= Eclamptic/Preeclamptic (Group A); Controls= Group B

**Table-5: Correlation analysis of visfatin with electrolytes in eclamptic/preeclamptic patients and controls**

| Electrolytes (mEq/L) | r | | *p-Value | |
|---|---|---|---|---|
| | Controls (n=74) | E/PE Patients (n=160) | Controls (n=74) | E/PE Patients (n=160) |
| Potassium | 0.094 | -0.056 | 0.424 | 0.479 |
| Sodium | 0.138 | -0.041 | 0.242 | 0.604 |
| Chloride | -0.080 | -0.134 | 0.500 | 0.091 |

**p*-value based on Spearman rho test; &E/PE= Eclamptic/Preeclamptic (Group AB); Controls= Group B.

**Table-6: Analysis of visfatin association with anthropometric data and blood pressure in eclamptic/preeclamptic patients and controls.**

| Variables | Odds Ratio (CI-95%) | $R^2$%-adjusted | **p*-Value |
|---|---|---|---|
| Age (years) | 0.9978 (0.9647–1.0320) | 0.00 | 0.897 |
| Weight (Kg) | 0.9830 (0.9577–1.0090) | 0.23 | 0.196 |
| Height (m) | 2.4575 (0.2117–28.5281) | 0.00 | 0.473 |
| BMI (kg/m$^2$) | 0.9624 (0.9180–1.0089) | 0.52 | 0.110 |
| BP (Systolic) (mm of Hg) | 1.2017 (1.1385–1.2684) | 78.78 | **0.000** |
| BP (Diastolic) (mm of Hg) | 1.3702 (1.22691.5302) | 78.52 | **0.000** |

Binary logistic regression implied to find **p*-value; CI= Confidence Interval

**Table-7: Analysis of visfatin association with hepatic enzymes in eclamptic/preeclamptic patients and controls**

| Enzymes (Units/L) | Odds Ratio (CI-95%) | $R^2$%-adjusted | *p-Value |
|---|---|---|---|
| #ALP | 1.0835 (1.0581–1.1096) | 63.47 | **0.000** |
| $ALT | 0.9895 (0.9709–1.0084) | 0.07 | 0.275 |

Binary logistic regression implied to find *$p$-value; CI= Confidence Interval; #ALP= Alkaline Phosphatase; $AST= Aspartate Transferase

**Table-8: Analysis of visfatin association with serum electrolytes in eclamptic/preeclamptic patients and controls.**

| Electrolytes (mEq/L) | Odds Ratio (CI-95%) | $R^2$%-adjusted | *$p$-Value |
|---|---|---|---|
| Potassium | 1.0673 (0.6818–1.6575) | 0.00 | 0.786 |
| Sodium | 0.9276 (0.8819–0.9757) | 3.09 | **0.002** |
| Chloride | 1.1478 (1.0793–1.2205) | 7.36 | **0.000** |

Binary logistic regression implied to find *$p$-value; CI= Confidence Interval

# DISCUSSION

Eclampsia and pre-eclampsia are among the most serious and misunderstood complications that occurs during gestation and threaten the lives of both mother and foetus.[1] In Pakistan eclampsia and preeclampsia is one of the major causes of mortality and 1 out of 89 pregnant women die because of it.[2]

In human body the adipose tissues, through its multisystem effects, play a key role in regulating the endocrine functions, such as by secretion of plasma adipocytokines. A protein called visfatin[3], is one of these adipokines whose dysregulation leads to causation of disorders associated with obesity such as preeclampsia (during gestation)[4], hypertension[3], hyperlipidaemia[5], type-2 diabetes, small-for-gestational age foetus (SGA) etc[6].

The levels of visfatin proteins, an adipokine in nature, has been found raised in both endothelial dysfunction and vascular damage.[9,10] In our study serum visfatin concentrations were also found significantly raised in preeclamptic patients which is confirmed by other studies as well.[11,20,21] But this is also not always the case; some studies have even reported decrease in its levels.[22] The reasons behind this dissimilarities may be due to changes in abnormalities in metabolism, disease severity, and genetic variability. Our results also account significant positive correlation of both systolic and diastolic blood pressure (BP) with serum visfatin concentrations[23,] but some studies also revealed the opposite[24].

Moreover, our study did not conclude an association of BMI with visfatin levels which conforms with other prior study.[17] Moreover, some studies found that in preeclamptic women visfatin concentration revealed a negative correlation with Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), without showing any relation with BMI and age of the patient.[16,17] But on the contrary some studies also suggested a very strong and positive correlation of circulating visfatin and BMI (overweightness)[18,19], depot of visceral fats[19], waist-to-hip ratios[17]. Our study also showed a very strong and positive association of visfatin levels with levels of bilirubin and alkaline phosphatases (ALP), but it found no effect on aspartate transferases (AST).

Furthermore, the effects of visfatin on certain electrolytes like potassium ($K^+$), sodium ($Na^+$) and chloride ($Cl^-$) were also evaluated and a significant increase in serum chloride and sodium ions levels was recorded in both eclamptic and preeclamptic patients when compared with normal patients.[25] However, no effect on serum levels of potassium were observed. Majority (almost 90%) of potassium ions are found intracellular whereas sodium ions are found extracellular; for every two ions of potassium to enter cell three sodium ions are translocated out of the cell.[26] We also know that these ions are one of the major regulators of the blood pressure as well.[27] But the regulation of these ions itself depends the activity of sodium-potassium-ATPase pump (Na/P-ATPase Pump).[26] Thus, it can be assumed that eclampsia/preeclampsia may be indication of irregularity in the above mentioned ion pump, which may lead to the alter levels of potassium, sodium and chloride ions in the serum.[28] In addition to that the significant rise in chlorine levels in the eclamptic and preeclamptic patients could also result in increasing osmolality which in turn could inhibit the dilatation of vessels and thus causing vasoconstriction.[29]

# CONCLUSION

Serum visfatin had a significant effect on serum electrolytes and hepatic enzymes. It also has significantly decreased the sodium and chloride levels whereas the levels of potassium remained unaffected. A very strong and positive association of visfatin levels with levels of bilirubin and alkaline phosphatases was also observed (ALP) but it found no effect on aspartate transferases (AST).

# AUTHORS' CONTRIBUTION

AS: Conception and study design, acquisition of data, drafting the manuscript, critical review, approval of final version to be published. MWL: Acquisition of

data, drafting the manuscript, approval of final version to be published. SI and NK: Analysis and interpretation of data, drafting the manuscript, approval of final version to be published. SF: Acquisition, analysis and interpretation of data, critical review, approval of final version to be published. RN: Acquisition, analysis, and interpretation of data, critical review, drafting the manuscript, approval of final version to be published

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