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ORIGINAL ARTICLE

Maternal and Fetal Plasma Urocortin Levels in pregnancies Complicated by Preeclampsia

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Abstract

Background: ***Preeclampsia is a multisystem illness that manifests in the latter half of pregnancy as end-organ failure, proteinuria, or new-onset hypertension.***

Aims: ***seeks to assess urocortin levels in the plasma of the mother and fetus in 70 singleton pregnancies complicated by preeclampsia and in an additional 70 healthy, normotensive singleton pregnancies.***

Patients and Methods: ***After receiving approval from the research and ethical council, this case-control study was carried out at El Hussein University Hospital between November 2022 and December 2023.140 pregnant women having singleton pregnancies were included in this investigation and divided into two categories. The control group consisted of 70 healthy, normotensive singleton pregnancies that went on to deliver a healthy term baby. Pregnant women with singletons exacerbated by preeclampsia (n = 70; one case against one healthy control) made up the study group).***

Results: ***We recommend that an additional study be done on this biochemical marker since it has a strong correlation with preeclampsia and elevated levels of urocortin in both the mother and the fetus in preeclamptic instances.***

Conclusion: ***Preeclampsia cases have higher plasma urocortin levels in the mother and fetus than in the control group. Additionally, preeclampsia patients have a considerable impact on the middle cerebral artery and uterine artery Doppler indices.***

Keywords: Maternal Plasma; Fetal Plasma; Urocortin Levels; pregnancies; Preeclampsia

1. Introduction

Preeclampsia was defined in 2013 by the American College of Obstetricians and Gynecologists (ACOG) as the new onset of hypertension (the blood pressure of at least 140 mmHg systolic and 90 mmHg diastolic) on two separate times at least four hours apart during 20 weeks of gestation, aggravated through a single or multiple of the following: Proteinuria., malfunctioning mother organs, such as pulmonary edema, thrombocytopenia (platelet count<100,00/dL), headaches or vision problems, reduced liver function (ALAT or ASAT≥ 70 U/l) or renal insufficiency (serum creatinine concentrations≥100 µmol/L; 1.1 mg/dL).[1]

In 2018, ACOG updated the previous definition by adding that (the headache is unresponsive to acetaminophen and not accounted for by alternative diagnosis); as regards liver involvement, it added(+/-right upper quadrant or epigastric abdominal pain) and as regards serum creatinine, it added (or a doubling of the serum creatinine concentration in the absence of another renal disease).[2]

Also, preeclampsia was defined in 2018 by the International Society for the Study of Hypertension in Pregnancy as a new onset of hypertension (blood pressure of ≥140 mmHg systolic and≥90 mmHg diastolic) after 20 weeks of gestation accompanied by one or more of the following new-onset conditions at or after 20 weeks gestation: Proteinuria., Maternal organ malfunction, including Hematological complications (thrombocytopenia with platelet count below 150,000/dL, DIC, hemolysis),

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liver involvement (elevated transaminases with or without right upper quadrant or epigastric abdominal pain), renal insufficiency (creatinine>90 µmol/L; 1 mg/dL), neurological complications (examples include eclampsia altered mental status, blindness, stroke, hyperreflexia with clonus, severe headaches with hyperreflexia, persistent visual scotomata), or uteroplacental dysfunction (such as fetal growth restriction, abnormal umbilical artery Doppler wave). [3]

Eclampsia is a life-threatening manifestation of preeclampsia, defined by the new onset of tonic-clonic, focal, or multifocal seizures in the pregnant or postpartum patient without an alternative explanation.[4] Because trophoblasts incorrectly invade the mother's decidua, uteroplacental circulation is restricted, which in turn reduces the amount of blood that moves through the mother to the fetoplacental unit. This is why the human placenta is essential in pathogenesis. Reduced oxygen and nourishment transfer from the mother to the growing fetus is the result of decreased uteroplacental blood flow. This could lead to a decrease in intrauterine growth (IUGR), a characteristic of pregnancy-related hypertension diseases.[5]

A neuropeptide belonging to the corticotropin-releasing factor (CRF) family, urocortin has 40 amino acids. It exhibits a high affinity for the CRF receptor types 1 and 2.[6]

Maternal and fetal circulation can be used to measure urocortin, which is expressed by gestational tissues like the amnion, chorion, decidua, trophoblast, and myometrium. The concentrations of maternal plasma urocortin remain stable during pregnancy in both the initial and third trimesters.[7]

The current study aims to evaluate urocortin levels in preeclamptic pregnant women, both in the mother and the fetus.

2. Patients and methods

This case-control study was carried out at El-Hussein University Hospital between November 2022 and December 2023 after receiving approval from the research and ethics committee.

Sample Size: 140 pregnant women were included in the study and divided into two groups. The control group consisted of 70 healthy normotensive pregnant women with singleton pregnancies progressing to deliver a healthy-term baby, and the case group consisted of 70 pregnant women having singleton pregnancies complicated by preeclampsia (one case versus one healthy control).

Inclusion Criteria for the study group: The study comprised pregnant women between the ages of 18 and 35. All of the pregnant women had term singleton pregnancies and preeclampsia.

Inclusion Criteria For the control group: The study comprised women between the ages of 18 and 35 who were carrying a singleton pregnancy at term and who were in good health with normal blood pressure.

Exclusion Criteria for both groups: Thrombophilias, collagen vascular disease, pregestational or gestational diabetes mellitus, repeated pregnancies, fetal abnormalities, and any medical condition or systemic disease history should not be considered a risk factor for pregnancy.

Steps:

The study procedures were followed, and women who met the requirements for inclusion or exclusion were included in the analysis. After counseling and an explanation of the technique, a signed and informed agreement to take part in the study was obtained from the patient. Every patient was questioned, and a thorough medical history was obtained, encompassing the individual's history, pregnancy, and gynecology.

After that, every patient was checked out, both locally and generally, for any medical condition. Each subject had ten milliliters of blood drawn; two milliliters were placed on EDTA for a complete blood count (CBC), and three milliliters were allowed to clot. The serum was then extracted and used for the remaining laboratory tests, which included tests for kidney and liver function (serum creatinine level, serum urea level, and serum glutamic-pyruvic transaminase, or SGOT and SGPT). A thorough urine analysis was performed on them.

Then trans abdominal ultrasound and uterine, umbilical, and middle cerebral arteries Doppler were performed for all pregnant women included in the study after hospitalization and before delivery (As regards Doppler indices criteria, RI and PI values above the 95th percentile standardized for the gestational age were considered abnormal for the umbilical and uterine arteries, and below the 10th percentile for the middle cerebral artery).[8] Finally, we collected umbilical cord blood samples from all case and control groups during delivery and before cord clamping to asses fetal urocortin level.

Urocortin measurement:

The ELISA kit (cat. #ELK2877) was used to measure the plasma urocortin level in accordance with the manufacturer's instructions.

Test Principle:

This kit uses sandwich enzyme immunoassay as its test principle. An antibody specific to Urocortin 1 (UCN1) has been pre-coated on the microtiter plate included in this kit. Following the addition of standards or samples to the appropriate microtiter plate wells, a biotin-conjugated antibody specific for Urocortin 1

(UCN1) is added. Each microplate well is then filled with horseradish peroxidase (HRP)-conjugated Avidin, and the mixture is incubated. Only in the wells containing Urocortin 1 (UCN1) will the colors of the enzyme-conjugated Avidin and biotin-conjugated antibody change once the TMB substrate solutions are introduced. A sulfuric acid solution is added to terminate the enzyme-substrate reaction, and the color change is determined spectrophotometrically at a wavelength of 450 nm plus or minus 10 nm. Next, each sample's optical density (OD) is compared to the standard curve to determine the amount of Urocortin 1 (UCN1) present in it.

Reagent Preparation:

Before use, all kit parts, including the sample, should be warmed to room temperature (18-25°C). If the chemicals and strips needed for the present experiment won't be used all at once, please remove them from the kit. Keep the remaining strips and reagents as directed. Use double-distilled water to dilute the 25×Wash Buffer into 1×Working Concentration. Centrifuge the Standard for about one minute at 1000 ×g using the regular working solution.

Before reconstituting the Standard with 1.0 mL of Standard The diluted solution Buffer, give it a gentle shake (not till it foams) and allow it to sit at room temperature for ten minutes. The stock solution has a 1000 pg/mL concentration of the Standard. As indicated in the photo below, Please arrange seven tubes in a double dilution process using the Diluted Standard and 0.5 mL of Standardized Diluent Buffer. To guarantee that every tube is thoroughly mixed before the subsequent transfer, pipette the solution upward and downward several times. Organize the diluted Standard at seven points: 125 pg/mL, two hundred pg/mL, a thousand pg/mL, 62.5 pg/mL, 31.25 pg/mL, 15.63 pg/mL, plus the blank, which is the last EP tube containing the standard diluent, at 0 pg/mL. Please utilize the new Standard Solution for every experiment to ensure the authenticity of the results. After every dilution, swap out the pipette tip while reducing the Standard's concentration from a high to a low value. The last tube is blank, so please remember that and avoid pipetting the solution from the previous tube into it.

The stock quantities of streptavidin-HRP and biotinylated antibodies should be quickly centrifuged or spun before use. Bring their focus down to a level appropriate for work. Using Biotinylated Antibody Diluent and HRP Diluent, 100 times each. TMB Substrate: Do not repour the remaining solution back into the vial; instead, aspirate the necessary dosage using sterile tips.

Primary outcome measures: Plasma urocortin levels in the mother and fetus were estimated for the case and control groups.

Secondary outcome measures: The relationship between the Doppler results (UA RI, UTA RI, and MCA RI) in both groups and the maternal and fetal plasma urocortin levels.

Statistical analysis:

IBM© Corp. Armonk, NY's SPSS© Statistics version 27 was used for statistical analysis. The independent-sample t-test is used to compare inter-group differences when comparing numerical variables, which are displayed as mean and standard deviation. When categorical variables are provided as ratios, numbers, or percentages, the Pearson chi-squared test or Fisher's exact test is utilized to assess differences between groups. When contrasting ordinal data, the linear-by-linear association is used. The Pearson correlation is used to test correlations. With a correlation coefficient (Pearson's r) of less than 0.2, you get a very weak correlation, moderate correlation (0.4 to 0.59), high correlation (0.6 to 0.79), and very strong correlation (≥0.8). When categorical variables are provided as ratios, numbers, or percentages for assessing group differences, the Pearson chi-squared test or Fisher's exact test is utilized. When contrasting ordinal data.

3. Results

Table 1. Maternal and fetal urocortin in cases and controls.

VARIABLE	PRE-ECLAMPSIA (N=70)		CONTROL (N=70)		MEAN DIFFERENCE	95% CI		P-VALUE†
	Mean	SD	Mean	SD		Lower	Upper	
MATERNAL UROCORTIN (PG/ML)	262.6	37.0	209.9	53.6	52.7	37.3	68.1	<0.001
FETAL UROCORTIN (PG/ML)	253.9	33.9	211.9	53.0	42.0	27.1	56.9	<0.001

†. Independent-samples t-test. 95% CI = 95% confidence interval, SD = standard deviation.

The mean of maternal and fetal serum urocortin levels in cases and controls which were significantly higher in cases group.

Table 2. Receiver-operating characteristic (ROC) curve analysis for discrimination between PE cases and controls using maternal or fetal urocortin.

	AREA UNDER THE ROC CURVE					CLASSIFIER EVALUATION METRICS					DIFFERENCE BETWEEN AUCS				
VARIABLE	AUC	SE	Lower 95% CI	Upper 95% CI	p-Value	Gini Index	Max K-S	Cutoff (pg/ml)	Sensitivity (%)	Specificity (%)	Difference	SE	Lower 95% CI	Upper 95% CI	p-Value
MATERNAL UROCORTIN	0.793	0.038	0.719	0.867	**<0.001**	0.586	0.471	265.5	56	91	0.039	0.276	-0.010	0.088	0.117
FETAL UROCORTIN	0.754	0.041	0.674	0.833	**<0.001**	0.507	0.414	251.3	57	84					

95% CI = 95% confidence interval, AUC = area under the ROC curve,

Gini index = (2 * AUC)-1, Max K-S = maximum Kolmogorov-Smirnov metric, SE = standard error.

Both maternal and fetal urocortin have fair to good discriminative value (AUC=0.793 and 0.754, respectively) with no statistically significant difference between both variables (difference between AUCs=0.039, 95% CI= -0.010 to 0.088, p-value=0.117). Best cutoff for maternal urocortin is >265.5 pg/ml (sensitivity=56%, specificity=91%) and for fetal urocortin is >251.3 pg/ml ml (sensitivity=57%, specificity=84%).

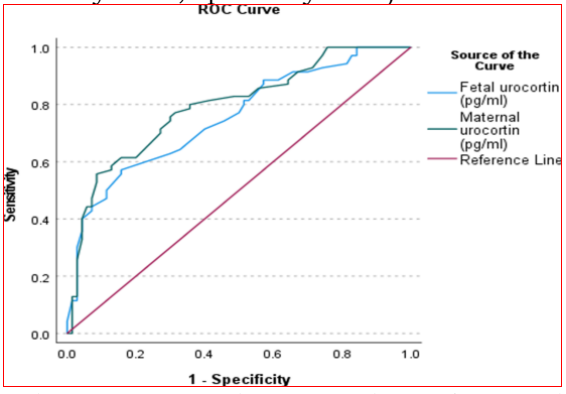

Figure 1. Receiver-operating characteristic (ROC) curves for discrimination between PE cases and controls using maternal or fetal urocortin.

Table 3. Arterial Doppler measures in cases and controls.

VARIABLE	PRE-ECLAMPSIA (N=70)		CONTROL (N=70)		MEAN DIFFERENCE	95% CI		P-VALUE †
	Mean	SD	Mean	SD		Lower	Upper	
UA RI	0.59	0.09	0.58	0.09	0.0	0.0	0.0	0.401
MCA RI	0.75	0.11	0.82	0.12	-0.1	-0.1	0.0	0.001
UTA RI	0.58	0.13	0.50	0.11	0.1	0.0	0.1	<0.001

†. Independent-samples t-test. 95% CI = 95% confidence interval, SD = standard deviation.

The middle cerebral artery resistance index (MCA RI) was significantly lower in preeclampsia group which indicate affection of fetal cerebral circulation due to preeclampsia. Uterine artery RI was significantly higher in preeclampsia group.

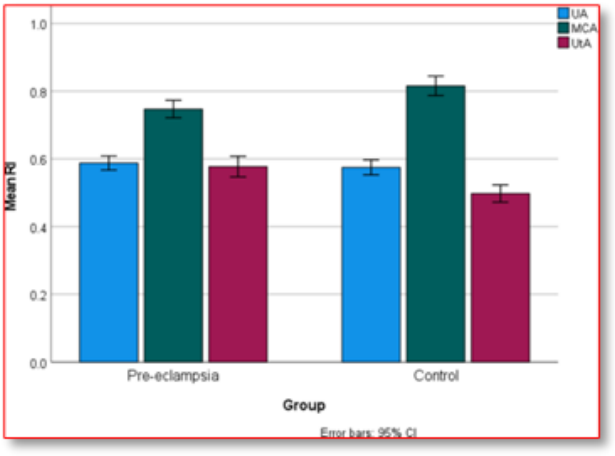

Figure 2. Mean UA RI, MCA RI and UtA RI in cases and controls. Error bars represent 95% confidence interval.

Table 4. Correlations of maternal and fetal urocortin with arterial Doppler measures in PE cases, controls and all study population.

VARIABLE		ALL PATIENTS (N=140)		PE (N=70)		CONTROL (N=70)	
		Maternal urocortin	Fetal urocortin	Maternal urocortin	Fetal urocortin	Maternal urocortin	Fetal urocortin
MATERNAL UROCORTIN	Pearson r	-	0.872**	-	0.897**	-	0.816**
	p-Value	-	<0.001	-	<0.001	-	<0.001
FETAL UROCORTIN	Pearson r	0.872**	-	0.897**	-	0.816**	-
	p-Value	<0.001	-	<0.001	-	<0.001	-
UA RI	Pearson r	-0.003	-0.025	-0.209	-0.146	0.060	-0.013
	p-Value	0.969	0.768	0.083	0.229	0.619	0.913
MCA RI	Pearson r	-0.213*	-0.164	-0.231	-0.223	0.008	0.054
	p-Value	0.012	0.053	0.054	0.064	0.945	0.656
UTA RI	Pearson r	0.135	0.087	-0.002	0.023	-0.059	-0.129
	p-Value	0.112	0.306	0.987	0.850	0.628	0.286

*. Correlation is significant at the 0.05 level (2-tailed). **. Correlation is significant at the 0.01 level (2-tailed).

There is very strong positive correlation between both variables in PE cases (r=0.897, p <0.001), controls (r=0.816, p<0.001) and all study population (r=0.872, p <0.001).

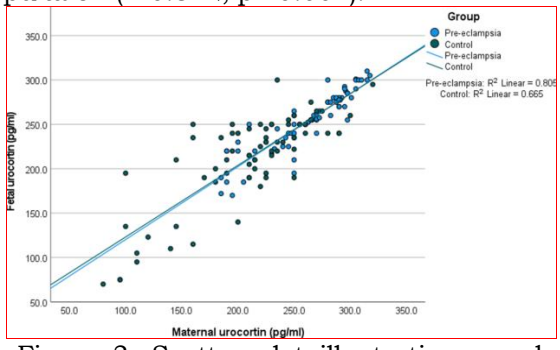

Figure 3. Scatter plot illustrating correlation between maternal and fetal urocortin in PE cases, controls and all study population.

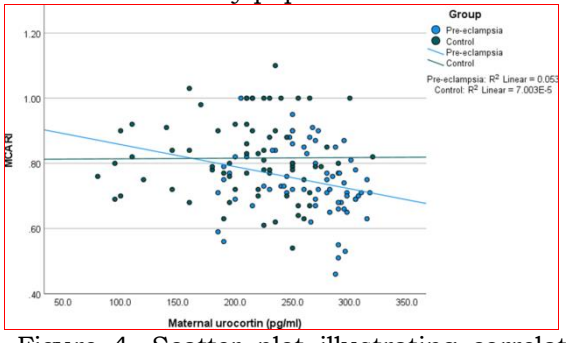

Figure 4. Scatter plot illustrating correlation between maternal urocortin and MCA RI in PE cases, controls and all study population.

There is weak negative correlation between both variables in all study population (r -0.213, p=0.012).

Sub-Group Analysis for Main Outcome Measures

Table 5. Comparison of Doppler measures and urocortin levels in cases with severe or non-severe PE.

VARIABLE	NON-SEVERE PET(N=24)		SEVERE PET (N=46)		MEAN DIFFERENCE	95% CI		
	Mean	SD	Mean	SD		Lower	Upper	p-Value†
UA RI	0.57	0.09	0.60	0.09	-0.03	-0.074	0.013	0.167
MCA RI	0.80	0.12	0.72	0.10	0.07	0.022	0.126	0.006
UTA RI	0.51	0.15	0.62	0.10	-0.11	-0.169	-0.051	<0.001
MATERNAL UROCORTIN (PG/ML)	251.1	34.1	268.6	37.5	-17.42	-35.687	0.848	0.061
FETAL UROCORTIN (PG/ML)	245.3	28.9	258.3	35.8	-13.04	-29.918	3.848	0.128

†. Independent-samples t-test. 95% CI = 95% confidence interval, SD = standard deviation.

The mean of uterine artery RI was significantly higher in severe PET group than in non-severe PET group and the mean of MCA RI was significantly affected in severe PET group than the other group.

Although there was no statistically significance as regard serum maternal and fetal urocortin levels in both groups but there was trend to be increased in severe PET group than non-severe PET group.

Table 6. Correlations of maternal and fetal urocortin with arterial Doppler measures in all PE cases and in cases with severe or non-severe PE.

VARIABLE		ALL PE (N=70)		NON-SEVERE PE (N=24)		SEVERE PE (N=46)	
		Maternal urocortin	Fetal urocortin	Maternal urocortin	Fetal urocortin	Maternal urocortin	Fetal urocortin
MATERNAL UROCORTIN	Pearson r	-	0.816**	-	0.776**	-	0.940**
	p-Value	-	<0.001	-	<0.001	-	<0.001
FETAL UROCORTIN	Pearson r	0.816**	-	0.776**	-	0.940**	-
	p-Value	<0.001	-	<0.001	-	<0.001	-
UA RI	Pearson r	0.060	-0.013	-0.148	0.067	-0.308*	-0.288
	p-Value	0.619	0.913	0.489	0.755	0.037	0.053
MCA RI	Pearson r	0.008	0.054	-.408*	-0.432*	-0.038	-0.051
	p-Value	0.945	0.656	0.048	0.035	0.800	0.738
UTA RI	Pearson r	-0.059	-0.129	-0.174	-0.030	-0.064	-0.079
	p-Value	0.628	0.286	0.416	0.890	0.670	0.601

*. Correlation is significant at the 0.05 level (2-tailed). **. Correlation is significant at the 0.01 level (2-tailed).

There is very strong positive correlation between both variables in all PE cases (r=0.816, p<0.001) and in cases with severe PE (r=0.940, p <0.001), and strong positive correlation in cases with non-severe PE (r=0.776, p <0.001). There is moderate negative correlation between both variables in cases with non-severe PE (r= -0.432, p=0.035).

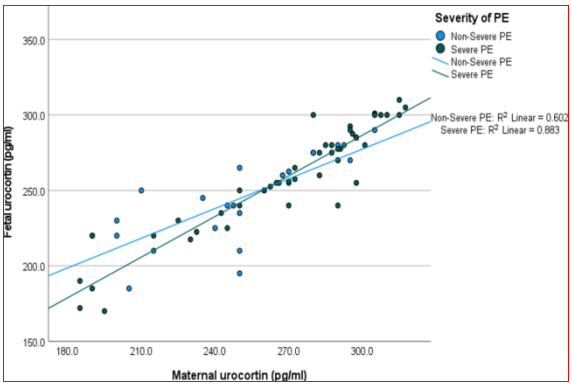

Figure 5. Scatter plot illustrating correlation between maternal and fetal urocortin in all PE cases and in cases with severe or non-severe PE.

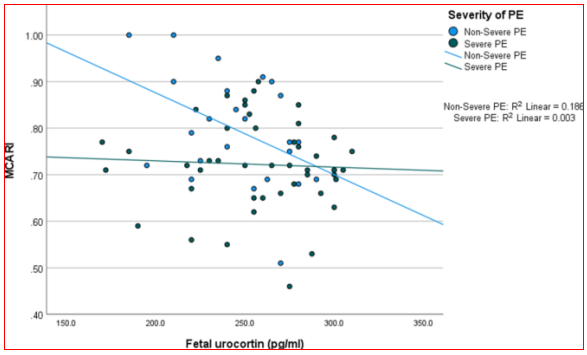

Figure 6. Scatter plot illustrating correlation between fetal urocortin and MCA RI in all PE cases and in cases with severe or non-severe PE.

4. Discussion

A pregnancy-related condition with a prevalence of 4.6% (95% CI, 2.7-0.2) in all pregnancies is preeclampsia. In the world, it is still a major factor in maternal and infant morbidity and death.[9]

Preeclampsia puts women at risk for potentially fatal conditions such as disseminated intravascular coagulation (DIC), cerebral hemorrhage, pulmonary edema, acute damage to the kidneys, liver failure, or rupture, as well as placental abruption.[2]

The human placenta releases many vasoactive substances to decrease vascular resistance and ensure a consistent blood supply for the fetal-placental unit. One of the components is corticotrophin-releasing factor (CRF), which is a neuropeptide produced in the placenta. It is found in high levels in women with hypertension problems during pregnancy, and especially in those with preeclampsia.[10,11]

Urocortin is a peptide consisting of 40 amino acids. It is a member of the CRF family and shares 45% sequence similarity in both rat and human CRF.[12] The expression of this phenomenon is observed in gestational tissues, including the amnion, chorion, decidua , trophoblast , and myometrium. It can be measured in both maternal and fetal circulation, with steady levels discovered in mother plasma from the first to the third trimester of pregnancy.[13]

Florio et al., One hundred and forty pregnant women participated in the study; seventy of them were healthy, normotensive singletons in the control group. A healthy-term infant was delivered as a result of these pregnancies. Seventy singleton pregnancies with severe hypertension disorders during pregnancy made up the study group. A healthy control group was contrasted with each instance. Three additional categories were applied to the pregnancies: preeclampsia (19 cases), preeclampsia with intrauterine growth restriction (PE/IUGR; 15 occurrences), and gestational hypertension (36 cases). All samples analyzed showed measurable levels of urocortin; however, maternal levels were significantly (P<0.0001) higher in the study group than in the control group. In particular, in comparison to the control patients, the subgroups with gestational hypertension (P<0.001), preeclampsia (P<0.001), and PE/IUGR (P<0.001) had significantly higher urocortin levels.[13]

Compared to control (P<0.001) and gestational high blood pressure (P<0.001) patients, patients with preeclampsia (P<0.001) and PE/IUGR (P<0.001) had an average UtA resistance index that was considerably higher. The UtA resistance index was the same in the preeclampsia, PE/IUGR, and control groups, as well as in the groups experiencing hypertension throughout pregnancy. In comparison to control (P<0.001) and gestational hypertension (P<0.001) patients, patients with preeclampsia (P<0.001) and PE/IUGR (P<0.001) showed a statistically significant rise in the UCA pulsatility index. The UCA pulsatility index did not, however, significantly differ between the preeclampsia and PE/IUGR subgroups or between the groups with gestational hypertension and those without. However, for either group, the MCA pulsatility index remained unchanged.

In the study of Florio et al., The levels of urocortin I in maternal subjects were considerably elevated in comparison to the control group in GH (the P<0.05), PE (the P<0.001), and PE+FGR (P<0.001). The urocortin levels in the PE+FGR group were the highest, considerably (P<0.001) surpassing those in the PE and GH groups. The levels of umbilical cord were substantially greater (P<0.0001) in cases of gestational hypertension (GH), preeclampsia (PE), and preeclampsia with fetal growth restriction (PE+FGR) in contrast to the control cohort. Furthermore, compared to GH and PE, the levels were considerably greater (P<0.001) in PE+FGR instances. The concentrations were significantly higher (P<0.0001) and exhibited a relationship with the mother's levels.[14]

Our study revealed a statistically significant increase in the average levels of urocortin in both maternal and fetal serum among individuals with

preeclampsia.

As regards the ROC curve analysis, both maternal and fetal urocortin have fair to good discriminative values (AUC=0.793 and 0.754, respectively) with no statistically significant difference between both variables (difference between AUCs=0.039, 95% CI= -0.010 to 0.088, p-value=0.117). Best cutoff for maternal urocortin is >265.5 pg/ml (sensitivity=56%, specificity =91%) and for fetal urocortin is >251.3 pg/ml ml (sensitivity=57%, specificity=84%).

There is a very strong positive correlation between maternal and fetal urocortin in PE cases (r=0.897, p<0.001), controls (r=0.816, p<0.001), and all study populations (r=0.872, p<0.001) also there is very strong positive correlation between maternal and fetal urocortin in all PE cases (r=0.816, p<0.001) and cases with severe PE (r=0.940, p<0.001), and strong positive correlation in cases with non-severe PE (r=0.776, p<0.001).

As regards Doppler findings from our study, the middle cerebral artery resistance index (MCA RI) was significantly lower in the preeclampsia group, which indicates an affection of fetal cerebral circulation due to preeclampsia. The uterine artery RI was significantly higher in the preeclampsia group.

There is a weak negative correlation between maternal urocortin and MCA RI in all study populations (r -0.213, p=0.012).

4. Conclusion

Preeclampsia cases have higher plasma urocortin levels in the mother and fetus than in the control group. Preeclamptic patients have a considerable impact on the middle cerebral artery and uterine artery Doppler indices. Additionally, in our study, there is a correlation between the maternal urocortin level and MCA RI, which is a weak negative in all study populations and a moderate negative in the non-severe preeclampsia subgroup. Also, as regards fetal urocortin level and MCA RI, there is a moderate negative correlation between them in the non-severe preeclampsia subgroup..

Disclosure

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Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

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