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Original Research Article

Prevalence of metabolic syndrome in preeclampsia

Mehbooba Beigh[1], Muzafar Naik[2]*, Irshad Tali[1], Rifat Ara[1], Anjum Malik[1]

[1]Department of Obstetrics and Gynecology, [2]Department of General Medicine, SKIMS Medical College and Hospital, Srinagar, Jammu and Kashmir, India

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***Correspondence:**
Dr. Muzafar Naik,
E-mail: muzafarnaik@gmail.com

ABSTRACT

Background: Preeclampsia is associated with increased morbidity and mortality and it increases with severity of preeclampsia. Preeclampsia shares many characteristics of metabolic syndrome (MeS) which has led many investigators to elucidate this relationship. The aim and objective of this study was to assess the prevalence of MS in preeclampsia and its relation with its severity.
Methods: The study included 130 cases (41 gestational hypertension (GHTN), 27 mild pre-eclampsia (Mild PET), 47 severe preeclampsia (Severe PET), 13 pre-eclampsia superimposed on chronic hypertension (PSHTN) and 2 eclampsia) based on pre-specified maternal characteristics according to ACOG criteria after 20 th week of gestation. Two hundred normotensive pregnant females served as controls. The frequency of MS was assessed using pregnancy adaptation of MeS definition of the NCEP-ATP III criteria in cases and controls.
Results: Metabolic syndrome was found in 37.7% of preeclampsia group and 12% of control group ($p<0.00$). Among the components of MeS, preeclampsia group was having significantly higher sugars (30% Vs 20%) and body mass index (BMI) (23.8% Vs 7.5%) than controls. GHTN was seen in 31.5%, mild PET in 20.8%, severe PET in 36.2%, PSHTN in 10% and eclampsia in 1.5% of cases. MeS was seen in 57.4% of severe PET, 50% of eclampsia, 26.8% of GHTN, 25.9% of mild PET and 23.1% of PSHTN. The clinical course in preeclampsia with MeS was complicated by IUD (intrauterine death), IUGR (intrauterine growth retardation), preterm delivery, APH and pulmonary edema. Oligohydromnios was less common in preeclampsia with MeS.
Conclusions: The frequency of MeS was higher in preeclampsia group as compared to normotensive group. MeS was more significantly higher in patients with severe preeclampsia. In our study there were no demographic, clinical and laboratory predictors of MeS in preeclampsia. On the other hand, preeclampsia patients with MeS had significant maternofoetal complications. There is a need to screen for MeS in pregnant females from the first antenatal visit in order to predict severe preeclampsia.

Keywords: Metabolic syndrome, Preeclampsia, Triglycerides

INTRODUCTION

Pre-eclampsia is defined as new onset of hypertension and proteinuria after 20th weeks of gestation in a previously normotensive woman and it complicates 2–8% of pregnancies and contributes to considerable maternal, neonatal morbidity and mortality.[1-3] The majority of maternal deaths during pregnancy are caused by medical disorders and hypertension being the commonest cause. [4] The aetiology of preeclampsia remains unknown despite continued medical research. The pathophysiology of preeclampsia likely involves both maternal and fetal/placental factors although more with the former.

The risk of abnormal placentation and preeclampsia is the result of medical conditions associated with vascular

insufficiency (eg, hypertension, diabetes, systemic lupus erythematosus, renal disease, acquired and inherited thrombophilias).[5,6]

The majority of these medical disorders share a common pathogenesis and that is insulin resistance or in a broader term the metabolic syndrome (MeS).

The metabolic syndrome confers an increased risk of developing Type 2 diabetes.[7-10] Also, the metabolic syndrome is linked to cardiovascular disease (CVD) risk irrespective of age, sex and geographic distribution.

The National Cholesterol Education Program (NCEP)-adult treatment panel (ATP III) guidelines, which have gained widespread clinical use, define metabolic syndrome as three or more of five clinically ascertained risk factors: Abdominal obesity, low high density lipoprotein-C (HDL), elevated triglycerides (TG), blood pressure and fasting glucose.[11]

Recently there has been a study which demonstrates that inter-pregnancy metabolic syndrome predisposes to pre-eclampsia which further substantiates the fact that these two disorders share a common pathophysiology.[12] By elucidating the association of metabolic syndrome with severity of preeclampsia it may be possible to understand the pathophysiology and potentially predict the development of severe pre-eclampsia.

METHODS

This case-control study was performed in the department of Obstetrics and Gynecology in collaboration with General Medicine, at SKIMS medical college and hospital Srinagar from June 2015 to May 2017 for a period of two years after obtaining approval from institutional Ethical Clearance Committee. Written informed consent was taken from all women recruited. Antenatal women were enrolled in the study after 20th week of gestation.

CASES

Cases included were all patients with gestational hypertension, pre-eclampsia, pre-eclampsia superimposed on hypertension and eclampsia admitted to labor and delivery.

Cases (gestational hypertension, pre-eclampsia, pre-eclampsia superimposed on chronic hypertension and eclampsia) were identified based on pre-specified maternal characteristics according to ACOG criteria.[5,8]

Gestational hypertension

Gestational hypertension was defined as elevated blood pressure (≥140/90 mmHg on two measurements ≥6 h apart) without proteinuria after 20th week of gestation.

Pre-eclampsia

Pre-eclampsia was defined as elevated blood pressure (≥140/90 mmHg on two measurements ≥6 h apart) with proteinuria >300 mg/24 hours or ≥1 +proteinuria on spot urine examination after 20th week of pregnancy.

Pre-eclampsia superimposed on chronic hypertension

Pre-eclampsia superimposed on chronic hypertension was defined as new onset proteinuria > 300 mg/24 hours or ≥1 + proteinuria on spot urine examination in a patient of chronic hypertension after 20 th week of pregnancy.

Eclampsia

Eclampsia was defined as occurrence of seizure in a patient of pre-eclampsia without any personal history of seizures prior to pregnancy or family history of seizures.

Severity of pre-eclampsia

The severity of pre-eclampsia was determined by the involvement of following six sites prior to delivery CNS, renal, liver, hematogic, vascular and fetoplacental unit.

Assessment for metabolic syndrome

Metabolic syndrome was diagnosed by utilizing the pregnancy adaptation of MeS criteria of NCEP ATP III [13] laboratory and clinical criteria and it included (1) Blood pressure, (2) fasting glucose (as a measure of insulin resistance and/or glucose intolerance), (3) obesity (measured as hip to waist ratio or pre-pregnancy body mass index (BMI ≥ 30Kg/ m^2), (4) HDL and (5) TG.

The changes made were due to the metabolic abnormalities that occur during pregnancy, and it included.

Blood Pressure

As the study group involves pregnant patients with the diagnosis of hypertension (≥140/90 mmHg), and WHO utilizes 140/90 mmHg instead of 130/85 mm Hg therefore we took BP of 140/90 as criteria for diagnosis of HTN.

Blood sugar

The presence of gestational diabetes was defined by fasting plasma glucose ≥92 mg/dL or a single step OGTT using 75 grams in the fasting state with any single abnormal value of plasma sugar fasting >92 mg/dl, 1 hour >180 mg/dl and 2 hour >153 mg/dl utilizing International association of Diabetes and pregnancy study groups (IADPSG) criteria was used as a measure for insulin resistance.[21] Any woman who tested positive for gestational diabetes was considered positive for this factor.

Anthropometric assessment

Anthropometric measurement was done measuring height to the nearest centimeter without shoes with a wall mounted ruler and weight to the nearest to 0.1 Kg in light clothing. For the variable of obesity, we utilized pre-pregnancy BMI (calculated using reported height and weight before pregnancy-kg/m^2) given the impracticality of waist circumference in gravid women. BMI ≥30Kg/m^2 has been utilized in the World Health Organization (1999) diagnosis of metabolic syndrome.[15,15]

Lipid profile

Clinical endpoints for HDL was based on non-pregnant definition of <50 mg/dL but then TG levels were taken >250 mg/dl as modified by reports of lipid levels in pregnancy.[16]

Thus, our definition of metabolic syndrome included three out of five of the following components:

- Hypertension (BP ≥140/90 mm Hg),
- Diabetes (gestational or pre-gestational) Fasting blood sugar ≥92 mg/ dl or a single step OGTT using 75 grams in the fasting state with any single abnormal value of plasma sugar fasting >92 mg/dl, 1 hour >180 mg/dl and 2 hour >153 mg/dl.
- Pre pregnancy BMI ≥30 Kg/m^2,
- HDL ≤50 and
- TG ≥250.

Controls

Controls were enrolled from all normotensive women presenting for delivery at term (≥37 weeks) for spontaneous rupture of membranes, term labor, and induction of labor or caesarean section.

Exclusion criteria

- Multiple gestation pregnancies.
- Untreated hypothyroidism at the time of diagnosis of pre-eclampsia.
- Chronic hypertension without proteinuria.

Statistical analysis

Statistical data analysis was done utilizing SPSS 23. Normality of test was done by Shapiro-Wilk test. Median with Interquartile range (IQR) was calculated for age, body mass index, gestational age at delivery, systolic and diastolic BP, hemoglobin, platelet count, sugar fasting, triglycerides, high density lipoproteins, uric acid, lactate dehydrogenase (LDH), SGOT and SGPT as they were not normally distributed. Nonparametric data was analysed utilizing Mann-Whitney U test.

Nominal categorical data between the groups were compared using Chi-square test or Fisher's exact test as appropriate. Continuous categorical data between the groups were compared using sample t-test. $p<0.05$ was considered statistically significant.

RESULTS

A total of one hundred and thirty hypertensive pregnant females were included in this study. The cases included gestational hypertension, mild pre eclamptic toxaemia (PET), severe PET, eclampsia and pre-eclampsia superimposed on chronic hypertension.

Table 1: Baseline characteristic of hypertensive (cases) pregnant females and normotensive (controls) pregnant females.

	Hypertensive pregnant females	Normotensive pregnant females	p value
Age [years] (median (IQR)	31(6)	30 (5)	0.219
BMI [Kg/m^2] (Median (IQR))	31.26 (4)	30.65 (3)	0.065
SBP [mm Hg] (median (IQR))	160 (20)	110 (10)	0.00
DBP [mm Hg] (median (IQR))	106 (21)	70 (20)	0.00
Platelet [10 ^{9} cells/L] (median(IQR))	150 (108)	190 (86)	0.00
SGOT [Units/L] (median (IQR))	37 (73)	24 (13)	0.00
SGPT [Units/L] (median (IQR))	42.5 (75)	19 (13)	0.00
BSF [mg/dL] (median (IQR))	86 (19)	82 (17)	0.003
HDL [mg/dL] (median (IQR))	60.5 (24)	61 (18)	0.78
TG [mg/dL] (median (IQR))	270 (120)	261.5 (62)	0.00
Uric acid [mg/dL] (mediar (IQR))	9 (5)	6.15 (2)	0.00
LDH [mg/dL] (median (IQR))	405 (515)	264 (74)	0.00

Table 2: Contingency table showing distribution (percent) of metabolic syndrome among hypertensive pregnant females and normotensive pregnant females.

	Met syndrome absent		Met syndrome present		p value
	Count	%	Count	%	
Normotensive group (200)	176	88	24	12	p <0.00
Hypertensive group (130)	81	62.3	49	37.7	

Two hundred age and race matched normotensive pregnant females served as controls. The baseline characteristic of hypertensive pregnant females and normotensive pregnant females is shown in Table 1.

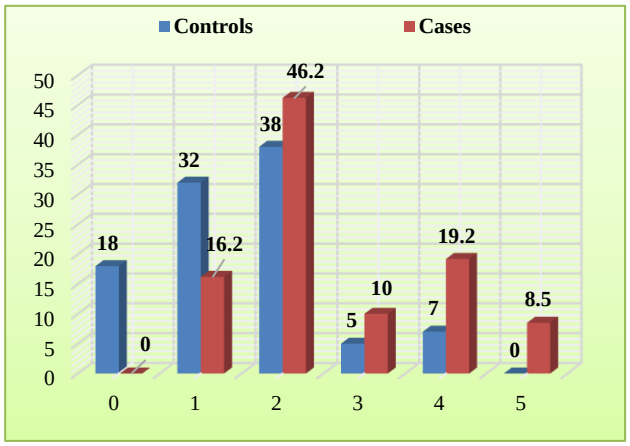


Figure 1: Clustered bar chart showing percent distribution of metabolic scores (0-5) in cases and controls.

The prevalence of metabolic syndrome in cases and controls is shown in Table 2. The metabolic scores of cases and controls are shown in Figure 1.

Table 3: Components of metabolic syndrome in hypertensive and normotensive pregnant females.

	Hypertensive group (130)		Control group (200)		p value
	(count)	(%)	(count)	(%)	
Elevated BMI (Kg/m^2)	31	23.8	15	7.5	0.000
Elevated BP (mm Hg)	130	100	0	0	0.000
Low HDL (mg/dL)	33	25.4	46	23	0.620
Elevated TG (mg/dL)	71	54.6	110	55	0.945
Abnormal sugar (mg/dL)	39	30	40	20	0.038

The distribution of components of metabolic syndrome in cases and controls is seen in Table 3.

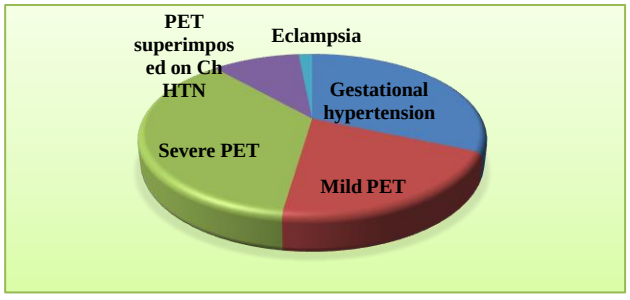


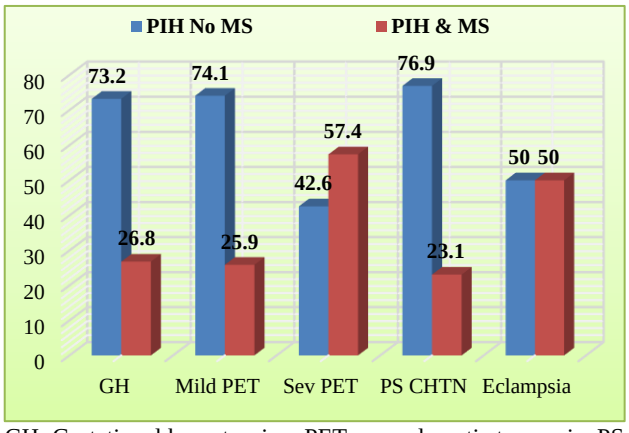
Figure 4: Three D Pie chart showing distribution of various types of hypertension in pregnant patients.

Table 4: Correlation of demographic and biochemical parameters of hypertensive pregnant females with and without metabolic syndrome.

	PIH without MS	PIH with MS	p Value
Age [years] (median (IQR)	31 (7)	30 (5)	0.723
BMI [Kg/m^2] (Median (IQR))	29.9 (3)	33.57 (4)	0.000
SBP [mm Hg] (median (IQR))	160 (20)	164 (78)	0.007
DBP [mm Hg] (median (IQR))	100 (22)	110 (19)	0.016
Platelet [10 ^{9} cells/L] (median(IQR))	161 (107)	137 (114)	0.169
OT [Units/L] (median (IQR))	37 (60)	36 (270)	0.380
PT [Units/L] (median (IQR))	41 (58)	47 (211)	0.073
BSF [mg/dL] (median (IQR))	83 (15)	97 (27)	0.000
Uric acid [mg/dL] (mean±SD)	9.446±3.21	9.28±3.24	0.769
HDL [mg/dL] (median (IQR))	63 (25)	54 (26)	0.001
TG [mg/dL] (median (IQR))	243 (100)	320 (101)	0.000

Characterization of preeclampsia is shown in 3 D pie chart in Figure 2.

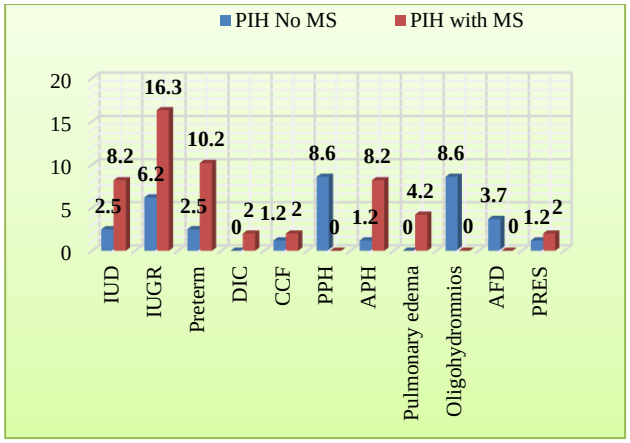
The correlation of demographic, clinical and biochemical parameters of PET with respect to metabolic syndrome is shown in Table 4.



GH: Gestational hypertension, PET: pre eclamptic toxaemia, PS CHTN: Preeclampsia superimposed on chronic hypertension.

Figure 3: Clustered 3 D bar chart showing distribution of metabolic syndrome in various types of hypertension in pregnant females.

Figure 3 shows percent distribution of MeS in subtypes of preeclampsia. Figure 4 shows complications in preeclampsia with metabolic syndrome.



IUD: Intrauterine death, IUGR: Intrauterine growth retardation, DIC: Disseminated intravascular coagulopathy, CCF: Congestive cardiac failure, PPH: Postpartum haemorrhage, APH: Anti-partum haemorrhage, AFD: Acute fetal distress, PRES: Posterior reversible encephalopathy syndrome.

Figure 6: Clustered bar chart showing various individual complications in PIH and metabolic syndrome.

DISCUSSION

Pregnancy is considered as an acid test for development for metabolic abnormalities, and the spectrum of these metabolic abnormalities include metabolic syndrome, dyslipidaemia and gestational diabetes mellitus. Metabolic syndrome (MeS) is being the cluster of all atherosclerotic risk factors.[17,19] The basic pathogenic mechanism of metabolic syndrome (MeS) is insulin resistance [20] in addition to an unhealthy diet, poor lifestyle, obesity and genetic factors.[17,21-23]

The presence of two components of MeS during pregnancy i.e. hypertension and glucose intolerance can lead to poor placental perfusion, endothelial dysfunction and abnormal placental development.[24] Placental hypoperfusion is a critical component in the pathogenesis of preeclampsia because it elaborates a variety of factors into the maternal bloodstream that alter maternal endothelial cell function and lead to the characteristic systemic signs and symptoms of preeclampsia.[25-31] Preeclampsia is a pregnancy specific disorder that affects 3-5% of pregnant worldwide.[32,33] It is a leading cause of maternal mortality in developing countries where access to healthcare is limited causing an estimated 60,000 maternal deaths worldwide per year.[32] It accounts for 20 % of maternal deaths and is the third biggest cause of maternal mortality in the United States.[34]

The prevalence of MeS in PET in our study was 37.7% as compared to 12 % in controls and is in accordance with studies conducted earlier.[35-37] The prevalence of MeS in hypertensive disorders of pregnancy has varied geographically from 7.6% in a Chinese study to 66% in an Iranian study.[38,39] The prevalence of MeS in a study in USA was 10.9% as compared to the normotensive counterparts (4.9%).[40] In our country also the prevalence of MeS in PET has been higher (10.9%) as compared to (4.9%) normotensive pregnant females.[41]

In present study, there was no significant elevation of Body Mass Index (BMI) in the hypertensive pregnant group as compared to the normotensive group. Studies conducted earlier also have found that hypertensive pregnant females were not significantly obese when compared with the normotensive pregnant females.[40,41] However hypertensive pregnant females were significantly obese in a study.[42] The difference can be explained by geographical distribution, ethnicity and genetic factors of the patients. The platelet count was significantly lower in cases as compared to the controls. This can be explained by the fact that in our study there was a high percentage of patients with severe PET.

The serum triglycerides levels were significantly higher in the hypertensive group as compared to normotensive controls. Our result is in accordance with observations made by others earlier.[43] This was also witnessed in a review of twenty two studies in which women with elevated triglycerides had twice the risk of preeclampsia, and the risk of PET further increased to four times when confounding factors like age, BMI and parity were adjusted for as compared with women with normal triglycerides.[16] However, there are studies which have demonstrated that triglycerides didn't show significant increase in PET patients.[40,41] Interestingly majority of investigators agree that hypertriglyceridemia could be involved in the pathogenesis of hypertensive disorders during pregnancy which is in accordance with our observation.[44-48]

We didn't report significant difference in the HDL levels between the PET patients and normotensive pregnant females. Again, present result is in accordance with observations made by earlier investigators.[16,43] Few investigators reported lower HDL in the PET cases as compared to the controls a difference that was statistically significant.[40,41]

In present study we found higher frequency of severe PET (36.2%) as compared to gestational hypertension (31.5%) and mild PET (20.8%). The frequency of preeclampsia superimposed on chronic hypertension was 10% and frequency of eclampsia was 1.5%. MeS was more common in severe PET and eclampsia as compared to gestational hypertension and mild PET a difference that was statistically significant. We didn't find any study that had evaluated the incidence of metabolic syndrome according to the severity of hypertension during pregnancy. This high incidence of MeS in severe preeclampsia can be explained by the fact that high triglyceride levels seem to increase the risk of placental vascular disorders, which trigger endothelial dysfunction, atherosclerosis and thrombosis.[44,45,49] The development of atherosclerosis in the placental spiral arteries of women

with preeclampsia indicates that elevated levels of triglycerides are involved in this disorder.[50]

There was no significant difference in age in the PET patients with MeS as compared to PET patients without MeS. These results are in accordance with observations made by previous investigators.[39] Present study indicated that higher BMI, blood pressure, triglycerides, sugar and low HDL were significantly associated with MeS in PET. This is in accordance with the results observed by earlier studies.[39] There was no statistically significant difference in platelets, liver function test, serum uric acid levels among PET with MeS group as compared to the PET without MeS group.

The overall complication rates in current study were higher in the PET with MeS although the difference was statistically insignificant. IUGR, IUD, preterm delivery and APH were significantly higher in PET with MeS, whereas oligohydromnios and PPH were significantly higher in PET without MeS. The reason being the endothelial dysfunction caused by metabolic syndrome leads to complications like IUGR, IUD, APH and preterm delivery whereas oligohydromnios was more common in PET without MeS group as sugars were significantly higher in the PET with MeS group and lastly there were two deaths in our study in the hypertensive pregnant group, one each in PET with MeS and PET without MeS and the difference was statistically insignificant.

Present study had a limitation and that needs to be mentioned. We diagnosed metabolic syndrome in the latter half of second trimester of pregnancy at a time when the likelihood of fetal malformations and perinatal adverse outcomes due to metabolic syndrome is already present. Also, is it the presence of metabolic syndrome prior to pregnancy or during the first trimester of pregnancy that is the prime factor leading to PET and it`s complications, is to be addressed by further studies. We conclude that in present study there were no predictors of MeS in PET with regards to age, parity history of PET in previous pregnancy, preterm delivery in previous pregnancy, thrombocytopenia and LFT abnormalities, thus formulating a trimester specific definition of metabolic syndrome especially for the first trimester and screening it in early pregnancy would be worthwhile in identifying at risk patients for severe PET.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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