

The Relationship Between Intraabdominal Hypertension and Preeclampsia

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ABSTRACT

OBJECTIVE: We aimed to measure abdominal pressure and placental levels of malondialdehyde in patients with preeclampsia and investigate the relationship between intraabdominal pressure and clinical features of preeclampsia.

STUDY DESIGN: Study was conducted at a tertiary referral clinic. Study group consisted of patients with preeclampsia and control group consisted of normotensive pregnant women. Both placental malondialdehyde and intraabdominal pressure levels were studied in all patients. Intraabdominal pressure was assessed indirectly via a Foley bladder catheter both antepartum and postpartum period. Statistical comparisons among groups were made using the Mann-Whitney U test, independent T test and Chi-square test. Statistical significance was set as $p < 0.05$.

RESULTS: Study and control group consisted of 35 pregnant patients. The mean patients' age, gravidity, parity, weight and BMI were not different between study and control groups. In the study group, Caesarean, preterm delivery and abdominal hypertension rate were significantly higher whereas the mean neonatal birth weight was lower. The mean antepartum and postpartum intraabdominal pressure levels were significantly higher in study group. The mean intraabdominal pressure was highest in patients with oliguria (19.8 ± 1.8 cmH₂O). Abdominal hypertension was detected in 30 (86%) patients in study group and in 3 (9%) patients in control group. The mean placental malondialdehyde level was significantly higher in patients with intraabdominal hypertension.

CONCLUSIONS: Abdominal hypertension rate is very high in patients with preeclampsia. Abdominal hypertension may have an additional role in preeclampsia.

Keywords: Preeclampsia, Intraabdominal hypertension, Eclampsia, Oliguria

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Introduction

Preeclampsia is a hypertensive disorder of pregnant women and occurs in approximately 5 to 8 percent of all pregnancies (1). The etiology of preeclampsia is still unknown but placenta is thought to be the inciting organ in this syndrome.


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The occurrence of signs and symptoms of preeclampsia before 20 weeks of gestation is unusual. The gradual development of hypertension and proteinuria are the major clinical manifestations. All of the clinical features of preeclampsia may be explained as maternal responses to generalized endothelial dysfunction. The effects of endothelial dysfunction can predominantly be seen on renal, hepatic and cerebral systems. Intraabdominal hypertension (IAH), which is defined as a sustained intraabdominal pressure (IAP) greater than 15cmH₂O, has also multiple adverse effects on these systems (2,3). Clinical symptoms and organ failure that can be seen in patients with preeclampsia are similar to those of patients with abdominal hypertension.

Pregnancy and morbid obesity are causes of chronic intraabdominal hypertension. We expect that preeclamptic patients have greater intraabdominal pressure levels than normal pregnant women because of endothelial dysfunction, increased capillary leakage and increased third space volume. Increased abdominal pressure may have a role in clinic manifestations or pathogenesis of preeclampsia. Elevated abdominal pressure may cause placental hypoperfusion and increase oxidative stress in pregnant women. In literature, there is no

study investigating or defining intraabdominal pressure in preeclamptic patients. In this study we aimed to measure abdominal pressure and placental levels of malondialdehyde (MDA) in patients with preeclampsia and investigate the relationship between intraabdominal pressure and clinical features of preeclampsia.

Material and Method

This study was conducted in a University Hospital. Local institutional ethic committee approved our study protocol and written informed consent was obtained from all participants before enrolment. The study group consisted of patients who were diagnosed with preeclampsia and the control group consisted of normotensive pregnant women. Complete blood count, liver enzyme tests, blood urea nitrogen, serum creatinine, 24-hour urine collection for protein excretion, placental MDA and intraabdominal pressure levels were studied in all patients.

Gestational age was established based on last menstrual period and confirmed with first trimester ultrasonography. All patients were followed in hospital until discharge. The blood pressure measurements were taken on the right upper arm at the same level as heart while the patient was sitting for at least 15 minutes. The disappearance of the fifth Korotkoff sound indicated the diastolic pressure. The criteria of the National Institutes of Health (NIH) Working Group on High Blood Pressure in Pregnancy were used to classify patients. Hypertensive disorders of pregnancy were accepted when systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg in a woman who was normotensive prior to 20 weeks of gestation. Proteinuria was accepted as 300 mg or more of proteinuria in a 24-hour urine specimen. Oliguria was accepted when urine output is less than 400 mL/day. In a previously normotensive woman with new onset of hypertension and either proteinuria or end-organ dysfunction after 20 weeks of gestation was accepted as preeclampsia. Eclampsia was accepted as the occurrence of one or more generalized convulsions and/or coma in the setting of preeclampsia and in the absence of other neurologic conditions. Preeclamptic patients who had elevated liver enzymes and low platelet count were diagnosed as HELLP syndrome. Fetal growth restriction was accepted when the neonatal birth weight was below the 10th percentile for gestational age and gender (4).

Intraabdominal pressure (IAP) was measured both antepartum and postpartum period. Antepartum measurements were made at time of admission and postpartum measurements were made 24 hours after the birth. The IAP was assessed indirectly via a 16-Fr-Foley bladder catheter in dorsal supine position. The method described in the World Society of the Abdominal Compartment Syndrome guidelines was used to measure intraabdominal pressure (5). For each individual, a needle was used to Y-connect a manometer according to original Kron technique described by Kron and co-workers (6). The pressure was measured at the end-expiration after en-

suring that abdominal muscle contractions were absent. Intraabdominal hypertension was defined when intraabdominal pressure was greater than 15 cmH₂O.

Placental tissues were taken from one of the peripheral cotyledons for malondialdehyde (MDA) measurement. The sample was washed thoroughly in saline to remove maternal blood and was then dissected in saline to identify chorionic villi without associated decidua. Villous samples were transported to the laboratory on ice and stored at -80°C until assay. MDA levels were measured according to spectrophotometric method (MDA Bioxytech-MDA 586, Spectrophotometric Assay for MDA, catalogue number 21044).

Exclusion criteria included multifetal pregnancies, polyhydramnios, fetal abnormalities, maternal diabetes mellitus, maternal thrombophilia, maternal renal disease and severe maternal obesity BMI > 40 kg/m².

The Statistical Package for the Social Sciences (SPSS 11.0; SPSS Inc., Chicago, IL) version 11.0 was used for statistical analysis. Data normality was assessed by the Kolmogorov-Smirnov test. Statistical comparisons among groups were performed using the Mann-Whitney U test, independent T test and Chi-square test. All p values were two-tailed and statistical significance was set as $p < 0.05$.

Results

Study group consisted of 35 pregnant women with hypertensive disorders of pregnancy and control group consisted of 35 normotensive pregnant women. In study group, there were 10 patients with preeclampsia, 13 patients with eclampsia and 12 patients with HELLP syndrome. Antihypertensive medication was needed in 24 patients. Oliguria was detected in 11 patients. The mean gestational age in the study group was 33 weeks. The mean ALT and AST level were 200.2 ± 305.9 and 102.1 ± 128.6 respectively.

No significant difference was found in the mean age, gravidity, parity, weight and BMI between two groups. In study group, Caesarean, preterm delivery and abdominal hypertension rate were significantly higher whereas the mean neonatal birth weight was lower. The mean neonatal birth weight was 1910.9 ± 913.1 in study group and 3086.3 ± 620.6 grams in control group ($p < 0.05$).

In study group, although the mean gestational age and neonatal birth weight were significantly lower, the mean antepartum intraabdominal pressure was significantly higher. The mean antepartum intraabdominal pressure level was 18.3 ± 2.6 cmH₂O in study group and 13.3 ± 1.9 cmH₂O in control group. The mean intraabdominal pressure was highest in patients with oliguria (19.8 ± 1.8 cmH₂O). In study and control group, abdominal hypertension was detected in 30 (%86) and 3 (%9) patients respectively. The clinical characteristics of the patients and comparisons of the study and control group were summarized in table 1.

Table 1: Comparisons of patients' characteristics and intraabdominal pressure levels

	Patients with pregnancy related hypertension (n:35)	Normal pregnant women (n:35)	Significance (p)
Age	28.6±6.2	28.6±5.3	ns
Gravidity	2(1-8)	2(1-5)	ns
Parity	1(0-6)	1(0-3)	ns
Gestational age	33(24-40)	38(28-40)	<0.05
Weight (kg)	68.6±18.9	63.1±15.7	ns
BMI (kg/m ²)	27.0±7.9	24.2±5.7	ns
AST (SGOT)	200.2±305.9	14.6±5.2	<0.05
ALT (SGPT)	102.1±128.6	12.3±4.2	<0.05
Oliguria	11(n)	-	
Neonatal weeight (g)	1910.9±913.1	3086.3±620.6	<0.05
Cesarean delivery, (n)	29	19	<0.05
Placental MDA (µmol/L)	2.52±1.05	1.42±0.54	<0.05
Ante-partum IAP (cmH ₂ O)	18.3± 2.6	13.3±1.9	<0.05
Ante-partum systolic/diastolic pressure(mmHg)	166.0±21.3/108.9±18.6	110.3±7.1/71.1±6.8	<0.05
Post-partum IAP (cmH ₂ O)	13.6± 3.3	9.9±2.6	<0.05
Post-partum systolic/diastolic pressure (mmHg)	144.6±14.8/96.3±8.8	104.9±18.1/68.9±5.8	<0.05

BMI: Body mass index, IAP: Intraabdominal pressure, MDA: Malondialdehyde, Note: Values are expressed as mean ± SD and range in parenthesis

The mean placental MDA level was significantly higher in patients with intraabdominal hypertension (Figure 1). The intraabdominal pressure was decreased significantly in each group but it was still greater in study group 24h after the birth (13.6± 3.3 versus 9.9±2.6 cmH₂O).

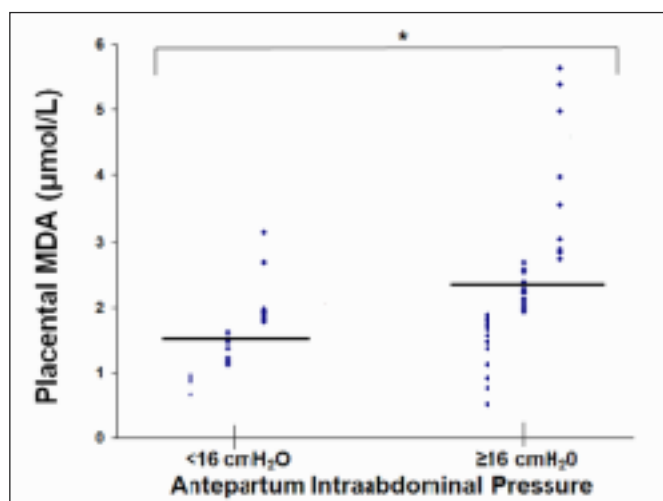


Figure 1: Relationship between intraabdominal hypertension and placental malondialdehyde

Discussion

This is the first clinical study investigating abdominal hypertension and its effects in patients with preeclampsia. In this clinical study we showed that antepartum and postpartum IAP level were significantly higher in patients with preeclampsia.

Abdominal hypertension was identified more than 80 per cent of preeclamptic patients. We also found that patients with severe preeclampsia complicated with oliguria had the highest IAP level. Placental MDA level was also higher in patients with abdominal hypertension.

Preeclampsia complicates 5-8% of pregnancies and is the second leading cause of maternal mortality in the United States (1,7). Vasoconstriction particularly in arterioles and endothelial damage play an important role in the pathogenesis of preeclampsia (8). We also know that preeclampsia has effects on nearly every maternal organ system, but predominately on the vascular, renal, hepatic and cerebral systems. Intraabdominal hypertension, especially grade III or grade IV has also multiple adverse effects on these systems. Abdominal hypertension shares some described diagnostic criteria with severe preeclampsia and HELLP syndrome. Additionally, increased capillary leakage due to endothelial damage is the same pathogenic factor with preeclampsia.

The abdominal cavity can be considered as a semi-closed compartment, so any changes in abdominal content may affect its pressure. When a critical volume is reached, the compliance of the abdomen wall abruptly drops, causing a progressive increase in intraabdominal pressure. Consistent increased intraabdominal pressure value greater than 15cmH₂O or 11mmHg is called intraabdominal hypertension (5). A normal value of intra-abdominal pressure is generally less than 10cmH₂O. Physiologic or pathologic IAP in pregnancy is not known. But pregnancy is accepted as one reason for chroni-

cally compensated state of abdominal hypertension. It has been reported that intraabdominal hypertension left untreated, its mortality rates up to 60% (9). At the systemic level as a result of elevated abdominal pressure venous return is diminished due to compression of vena cava inferior. The result is the decrease in preload and raised systemic vascular resistant because of compensatory vasoconstriction. In patients with IAP greater than 30mmHg, anuria is uniformly encountered (10). It is believed to be a result from the shunting of blood from the cortex to medulla, leading to a decreased glomerular filtration rate (11). Abdominal decompression reestablishes urine output and renal function (12). Elevated abdominal pressure impedes diaphragmatic movement (13). A rise in intrathoracic pressure causes increased intracranial pressure secondary to venous congestion. Finally cerebral perfusion pressure decreases (14).

It is plausible that abdominal hypertension has a potential role in the development or progression of preeclampsia. It is speculated that IAH may cause decreased abdominal perfusion and end organ perfusion including placenta, thus the activation of renin angiotensin aldosterone system (15).

Preeclamptic patients have less intravascular volume than normal pregnant women because of increased capillary leakage. During Caesarean delivery it is not uncommon to find moderately accumulations of fluid in the peritoneal cavity, so elevated intraabdominal pressure is expected in preeclamptic women. We found that the mean value of prepartum IAP was 18 cmH₂O in patients with preeclampsia and 66 percent of them had grade II abdominal hypertension. We assessed placental hypoperfusion damage by measuring MDA levels and found placental MDA levels higher in patients with intraabdominal hypertension. However, we found some correlation between intraabdominal hypertension and increased placental MDA levels, an unknown etiologic cause which may lead to placental ischemia can also be responsible for increased MDA levels. Because the most of the patients with intraabdominal hypertension also suffered from preeclampsia, we cannot clearly reveal direct correlation between intraabdominal hypertension and increased placental MDA in this study. This is one of the limitations of our study.

Intraabdominal pressure can be measured indirectly using intragastric, intracolonic, intravesical or inferior vena cava catheters (16). We measured intravesical pressure in stable position to determine IAP because intravesical measurement is less invasive, reliable and easier to perform than direct intraperitoneal measurement. It has been shown that there was strong correlation between the bladder pressure and directly measured intraabdominal pressure (17,18). The accurate measurement of intravesical pressure requires free movement of the bladder wall. The presence of intraperitoneal adhesions, pelvic hematomas, abdominal packs or a neurogenic bladder may affect the results (16). Intravesical pressure is the standard method to screen for intraabdominal hypertension (5).

In conclusion, we have detected that both antepartum and postpartum IAP were increased in patients with preeclampsia. Intraabdominal pressure showed correlation with both placental MDA levels and oliguria. Clinical signs and laboratory findings of preeclamptic patients may be associated with abdominal hypertension. Acute increases in intraabdominal pressure may be less well tolerated in pregnancy. We need further clinical studies to clearly express and reveal the effect of abdominal pressure on pathogenesis of preeclampsia.

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