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Ultrasonographic Optic Nerve Sheath Diameter as A Surrogate Measurement of Intracranial Pressure in Preeclampsia

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ABSTRACT

Background: Preeclampsia affects five to ten percent of pregnant women and accounts for about twelve percent of maternal mortality. It is the third most common cause of maternal mortality worldwide. Papilledema is an indirect and late indicator of raised intracranial pressure (ICP), whereas a pressure rise in optic nerve sheath (and the resulting enlargement of the optic nerve diameter) is a more dynamic process. The retrobulbar optic nerve sheath diameter (ONSD) can be measured at a position 3 mm posterior to the globe, where ultrasound contrast is greatest with more reproducible results.

Objective: To evaluate the correlation between ultrasonographic measurement of ONSD with the degree of severity of preeclampsia.

Patients and methods: This prospective observational study was conducted at Mansoura University Hospital Intensive Care Unit from July 2019 to June 2020. Just after delivery, 175 pregnant females aged between 18 and 45 years old were enrolled for participating in this study. Out of them, 25 females were excluded.

Results: As regarding body weight, there was a significant difference between the control group (79.9±6.36 kg) and preeclamptic without severe feature group (87.7±10.58 kg). The mean body weight in severe preeclampsia group was (91.5 ±14.73kg) which showed no significant difference from that of the non-severe group. ONSD values showed significant difference between the studied groups. The control group had ONSD mean value of 4.85±0.32 mm, mild preeclamptic group had mean value of 6.05±0.096 mm, while severe preeclampsia group had a mean value of 6.76±0.25 mm.

Conclusion: Ultrasonographic measurement of ONSD provides a non-invasive, quick and readily accessible tool for evaluation of raised intracranial pressure (ICP).

Keywords: Intracranial pressure, Optic nerve sheath diameter, Preeclampsia, Ultrasonographic.

INTRODUCTION

Preeclampsia is an exclusive complication of pregnancy that is defined as the presence of high blood pressure with proteinuria after twenty weeks of gestation [(1)]. However, in the absence of proteinuria, preeclampsia can be diagnosed by the presence of hypertension in ass0ciation with thrombocytopenia, impaired liver function, renal insufficiency, pulm0nary edema, or new-onset cerebral or visual disturbances[(2)].

Major complications that threaten the preeclamptic patient include severe hypertension and hypertensive emergencies (Intracranial hemorrhage, hypertensive encephalopathy), acute renal failure, congestive heart failure, placental abruption, disseminated intravascular coagulation (DIC), increased intracranial pressure (ICP), retinal detachment and pulmonary edema[(3)]. Preeclampsia can also cause fetal growth restriction and early delivery, and in some cases can lead to fetal death by increasing the incidence of placental complications[(4)].

Raised ICP is one of the consequences of preeclampsia[(5)]. The most precise method of ICP measurement is the direct invasive measurement of the intraventricular or subdural pressure. This invasive method is not practical in emergency departments and carries the risk of intracerebral hemorrhage and infection[(6)].

ICP can be non-invasively measured by computed tomography (CT) scan, but only the secondary characteristics of raised ICP can be visualized[(7)]. Nowadays, CT scans are readily available in many hospitals, but in many cases, they are still not easily accessible and the transfer of the patient could be problematic[(8)]. Therefore, the measurement and monitoring of ICP should be performed using a non-invasive, simple, reproducible and bedside method especially f0r emergency department patients[(9)].

Changes in the optic nerve sheath diameter (ONSD) is an important clinical and radiographic demonstration of raised ICP. Several studies have reported a significant relationship between elevated ICP (independent of the underlying cause) and the increase in ONSD[(10)]. From a physiologic standpoint, it can be stated that the increase in ICP exerts a pressure on the subarachnoid space around the optic nerve expanding the nerve sheath[(11)]. Several studies have proved the correlation between the high ICP in other pathologies and ultrasonographic measurement of ONSD[(12)].

Thus it is expected that a positive relationship between preeclampsia (as one of the causes of increased ICP) and ONSD exists and if confirmed, the routine use of bedside ultrasonography to measure ONSD can be used to monitor the increase in ICP. This method can be also considered as a potential tool for monitoring the effects of preeclampsia and anticipating its potential risks and complications[(13)].

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This study was designed to evaluate the correlation between ultrasonographic measurement of ONSD with the degree of severity of preeclampsia.

PATIENTS AND METHODS

This prospective observational study was conducted at Mansoura University Hospital Intensive Care Unit from July 2019 to June 2020.

Ethical approval:

This study was approved by Institutional Review Board (IRB), code number: MS.19.05.645, Mansoura Faculty of Medicine. All participants provided written informed consent. For the unconscious patients, this consent was obtained by a first degree relative. It was carried out according to ethical principles set by Declaration of Helsinki and good clinical practice.

Just after delivery, 175 pregnant females aged between 18 and 45 years old were enrolled for participating in this study. Out of them, 25 females were excluded.

Exclusion criteria:

History of ocular surgeries, presence of ocular ulcers, severe myopia, and any clinical or morphological condition that prevents ultrasound examination of the orbital area.

Sample size calculation:

Power analysis for one way ANOVA (analysis of variance) with three groups was conducted in G power to determine a sufficient sample size using an alpha (type 1 error) of 0.05, a power of 0.8 and a large effect size (f= 0.4). Based on the aforementioned assumptions, the desired sample size is 130 cases. To avoid the expected drop out, the total sample size was increased to 150 patients.

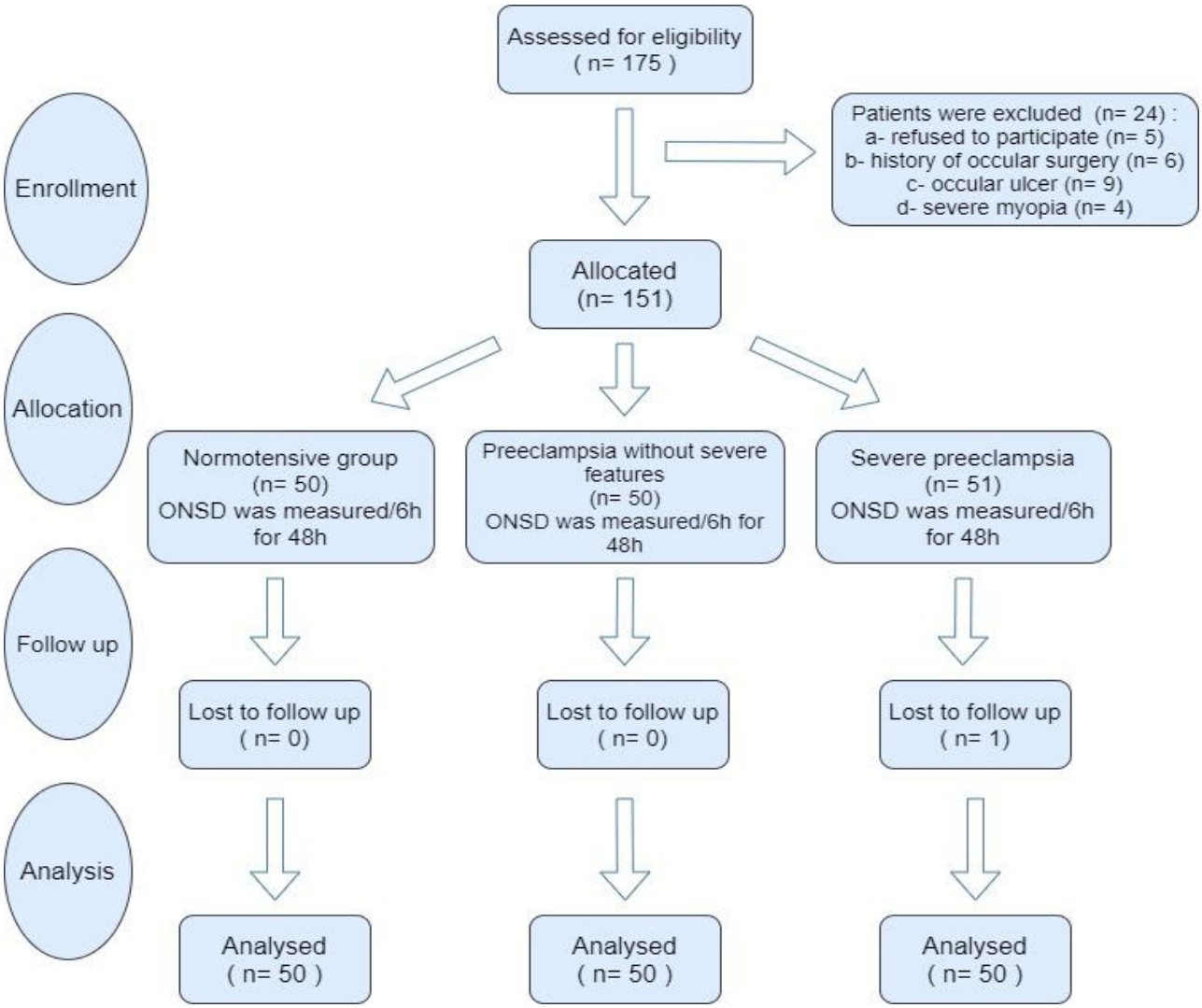

Figure (1): Flowchart demonstrating cases enrolled in the study.

Patients were allocated according to severity of preeclampsia as defined by the **American College of Obstetricians and Gynecologists** [14] into three study groups:

Group (1): normotensive pregnant women as a control group (n=50).

Group (2): preeclamptic patients without severe features (n=50).

Group (3): patients with severe preeclampsia (n=50).

Patients' management:

Patients were admitted just after delivery and were managed according to our institutional treatment protocols that were not modified during the study. All preeclamptic patients including preeclamptic without severe features were regarded as possible of developing any complication of preeclampsia and attempts were done to reduce external stimuli and exposure to intense light to minimize the risk of seizures.

A detailed history was taken and included information regarding obstetric history, symptoms and signs of preeclampsia, impending eclampsia, history of any organ system involvement, history of investigations performed and treatment taken, history of medication for hypertension and any significant past medical or surgical history.

General examination was conducted for evaluation of the level of consciousness, presence of pallor, cyanosis or icterus, checking for pulse rate and volume, checking the respiratory and cardiovascular systems for aspiration and signs of congestive heart failure, assessing the motor tone, patellar and plantar reflexes and searching for tongue bite in cases of eclampsia.

Blood samples were collected for complete blood count, liver function tests, renal function tests, serum uric acid, lactate dehydrogenase, serum calcium and magnesium and urine catheter was inserted for collecting 24 hour urine protein and guiding fluid therapy.

Arterial blood pressure was non-invasively measured every 15 minutes and mean values were recorded every six hours.

Ultrasound guided central venous catheter (CVC) was inserted to estimate central venous pressure (CVP) and guide fluid therapy accordingly, especially in cases of pulmonary edema, refractory oliguria, intractable hypertension and hypovolemia requiring massive resuscitation.

Technique of ONSD measurement:

ONSD measurement was performed in two axes of transverse and sagittal planes. The reported ONSD corresponded to the mean of four values obtained for each patient (transverse and sagittal planes for both eyes).

Patients were placed in supine position with the upper part of the body and the head at 30 degree above the horizontal position. After application of a thick layer of gel, a 7.5 MHz linear probe was placed on the temporal area of the eyelid, the hand holding the probe was placed on the forehead of the patient to prevent excessive pressure being exerted on the eye.

The placement of the probe was adjusted to give a suitable angle for displaying the entry of the optic nerve into the globe. The field was reduced to a depth of 4 cm. The two-dimensional mode was used and ONSD was measured 3 mm behind the globe using an electronic caliper and an axis perpendicular to the optic nerve (Figures 2-4).

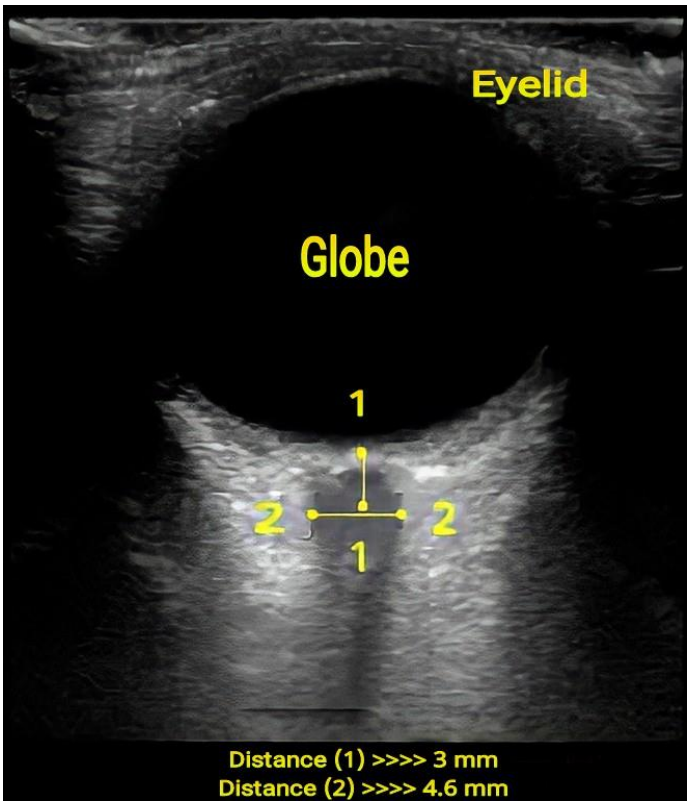

Figure (2): Sonographic measurement of ONSD of a case of the normotensive group.

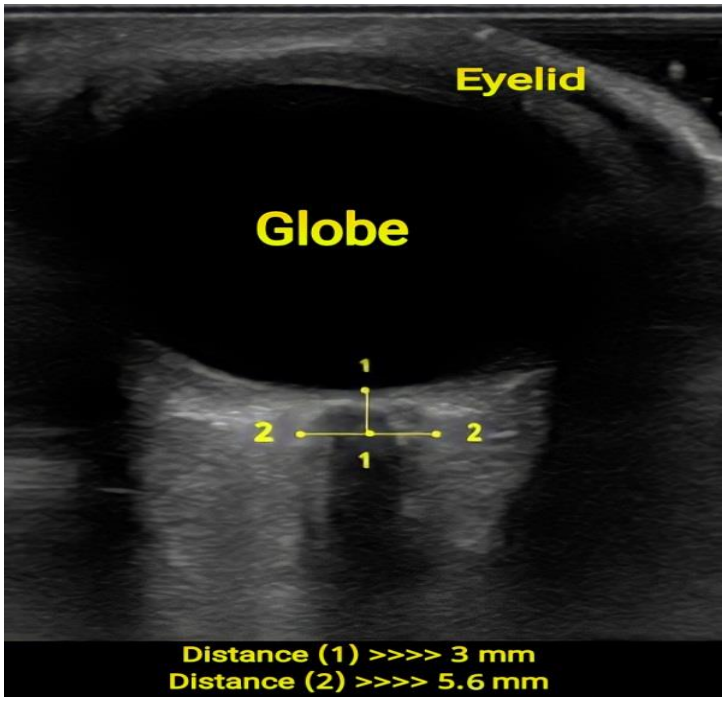

Figure (3): Sonographic measurement of ONSD of a case of the non-severe preeclampsia group.

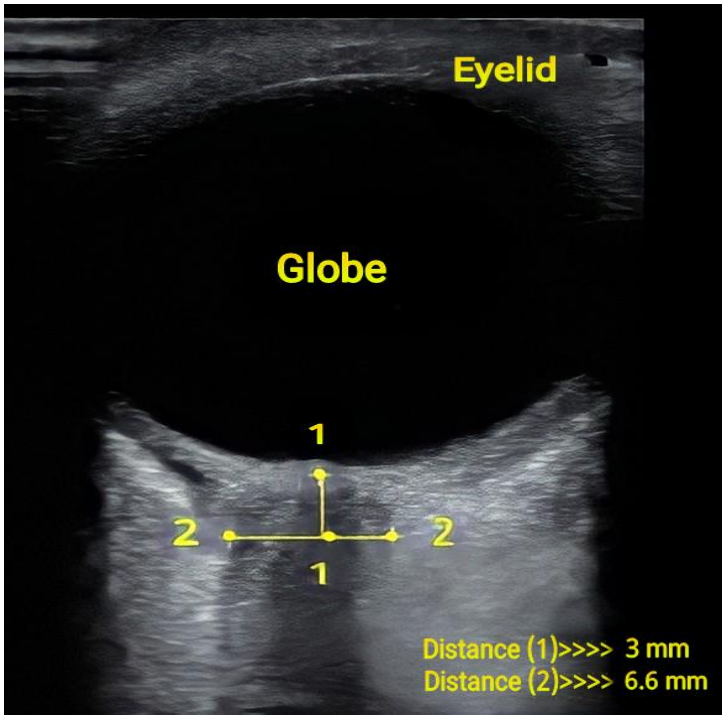

Figure (4): Sonographic measurement of ONSD of a case of the severe preeclampsia group.

Data collection:

A checklist was used to record the age, body mass index (BMI), gravidity, gestational age by weeks and history of intrauterine device (IUD) use. Ultrasonographic measurement of ONSD after delivery and then every six hours for 48 hours. Non-invasive monitoring of arterial blood pressure and mean values were recorded every six hours. Liver enzymes, platelets, serum uric acid, serum calcium and magnesium were daily recorded.

Statistical analysis and data interpretation:

Data were fed to the computer and analyzed using IBM SPSS Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp. Qualitative data were described using number and percent. Quantitative data were described using median (minimum and maximum) for non-parametric data and mean, standard deviation for parametric data after testing normality using Kolmogrov-Smirnov test. Significance of the obtained results was judged at the (0.05) level.

Chi-Square test was used for comparison of qualitative data. Monte Carlo test was used as correction for Chi-Square test when more than 25% of cells have count less than 5 in tables (>2*2). Fisher Exact test was used as correction for

Chi-Square test when more than 25% of cells have count less than 5 in 2*2tables. One-way ANOVA test was used to compare parametric quantitative data with post hoc Tukey test to detect pair-wise comparison.

RESULTS

In this prospective observational study, a total of 175 postpartum females were enrolled for and 25 cases were excluded or discontinued the intervention. The final analysis was done on 150 patients.

The three groups were comparable in terms of age, gestational age, mode of delivery and parity.

As regard body weight, there was a significant difference between the control group and preeclamptic without severe feature group. Regarding history of IUD use, there was a significant difference between the groups (Table 1).

Table (1): Demographic (age, weight, BMI) and obstetric (gestational age, mode of delivery, pregnancy outcome, IUD use, parity number) characteristics of the studied groups

	Group(1)	**Group(2)**	**Group(3)**	**test of significance**	**Within group significance**
	N=50	N=50	N=50		
Age (years)	27.72±6.26	28.32±6.40	27.26±7.37	F=0.3315 P=0.730	-
Weight (kg)	79.9±6.36	87.76±10.58	91.50±14.73	F=14.24 P<0.001*	P1=0.001* P2=0.001* P3=0.094
BMI (Kg/m^2)	28.01±3.39	31.67±4.13	32.34±5.08	F=15.05 P<0.001*	P1<0.001* P2<0.001* P3=0.425
Gestational age (weeks)	38.58±0.64	38.46±0.95	36.06±3.73	F=19.87 P<0.001*	P1=0.791 P2<0.001* P3<0.001*
Mode of delivery n (%) **Vaginal** **Caesarean section**	 12 (24.0) 38 (76.0)	 9 (18.0) 41 (82.0)	 10 (20.0) 40 (80.0)	 χ^2=0.569 P=0.752	-
Pregnancy outcome n (%) **Single(low birth weight)** **Single (average weight)** **Twin** **Triplets**	 **3 (6.0)** **46 (92.0)** **1 (2.0)** **0 (0.0)**	 **7 (14.0)** **41 (82.0)** **2 (4.0)** **0 (0.0)**	 18 (36.0) 28 (56.0) 2 (4.0) 2 (4.0)	 P=0.004*	 P1=0.329 P2=0.001* P3=0.048*
History of IUD use n (%)	28 (56.0)	6 (12.0)	3 (6.0)	P<0.001*	P1<0.001* P2<0.001* P3=0.295
Number of primigravida n (%)	22 (44)	23 (46)	26 (52)	P=0.463	-

Parameters described as mean± SD (standard deviation) and number (%).
F: One Way ANOVA test MC: Monte Carlo test
p1: difference between group 1 and 2
p2: difference between group 1 and 3
p3: difference between group 2 and 3
*statistically significant
BMI (Body Mass Index),
IUD (Intra-uterine device)

Various relevant laboratory markers for severity of preeclampsia were measured and compared among the groups. There was a significant difference in mean levels of platelets, aspartate transaminase, alanine transaminase, uric acid and LDH (Table 2).

Nine cases were presented by complete HELLP (hemolysis, elevated liver enzyme levels, and low platelet levels) and four cases were complicated by partial HELLP syndrome.

Table (2): Laboratory findings (Hemoglobin, platelet count, albumin, creatinine, uric acid, bilirubin, AST, ALT, calcium, magnesium, LDH and 24 h urine protein) distribution among studied groups
AST: Aspartate Aminotransferase.

Parameters	Time of assessment	Group(1)	Group(2)	Group(3)	Within group significance
		N=50	N=50	N=50	
Hemoglobin (gm/dl)	First day	11.06±1.11	11.01±0.77	10.13±1.25	P1=0.003*
	Second day	11.21±1.01	11.38±0.72	10.39±1.02	P2=0.001*
					P3=0.001*
Platelet count (*10^3 / mm3)	First day	267.44±58.04	208.48±50.2	149.56±9.48	P1<0.001*
	Second day	272.42±58.93	225.04±48.70	146.68±8.9	P2<0.001*
					P3<0.001*
Albumin (gm/ dl)	First day	3.67±0.44	2.97±0.14	2.87±0.41	P1<0.001*
	Second day	3.73±0.38	3.11±0.16	2.97±0.45	P2<0.001*
					P3=0.096
Creatinine (mg/ dl)	First day	0.79±0.12	0.97±0.11	0.99±0.28	P1<0.001*
	Second day	0.759±0.08	0.917±0.13	0.942±0.24	P2<0.001*
					P3=0.894
AST (IU/L)	First day	29.58±4.96	40.34±7.28	79.60±6.45	P1=0.909
	Second day	28.42±4.57	39.12±5.31	69.34±7.5	P2=0.001*
					P3=0.894
ALT (IU/L)	First day	23.84±4.38	35.80±5.73	74.50±4.93	P1=0.067
	Second day	22.46±3.47	34.22±4.95	61.76±6.24	P2<0.001*
					P3<0.001*
Bilirubin (mg/ dl)	First day	0.99±0.09	1.07±0.086	1.02±0.19	P1=0.001*
	Second day	0.969±0.11	1.032±0.10	1.00±0.18	P2=0.027*
					P3=0.193
24 hour Urine protein (mg)	First day	196.38±21.55	430.46±75.02	1324.82±337.1	P1<0.001*
	Second day	174.54±17.96	357.50±49.03	1117.42±152.57	P2<0.001*
					P3<0.001*
Calcium (mg/dl)	First day	9.38±0.19	8.02±0.12	7.81±0.29	P1<0.001*
	Second day	9.45±0.17	8.19±0.12	8.02±0.28	P2<0.001*
					P3<0.001*
Magnesium (mg/dl)	First day	1.79±0.07	1.44±0.08	1.39±0.15	P1<0.001*
	Second day	1.79±0.08	1.61±0.09	1.59±0.15	P2<0.001*
					P3<0.001*
Uric acid (mg/dl)	First day	4.78±0.13	5.53±0.91	7.65±1.59	P1=0.003*
	Second day	4.69±0.14	5.17±0.86	7.19±1.51	P2<0.001*
					P3<0.001*
LDH (IU/L)	First day	177.92±13.34	187.0±8.41	465.12±71.24	P1<0.001*
	Second day	164.0±10.13	171.88±7.16	409.22±54.12	P2<0.001*
					P3<0.001*

ALT: Alanine Aminotransferase.
LDH: Lactate Dehydrogenase.
p1: difference between group 1 and 2
p2: difference between group 1 and 3
p3: difference between group 2 and 3
*statistically significant

There was a significant difference in blood pressure measurement between the studied groups. However, nine cases of severe preeclamptic group were complicated by eclampsia despite border line increase in blood pressure (Tables 3 and 4).

Table (3): Systolic blood pressure distribution among studied groups

SBP (mmHg)	Group(1)	Group(2)	Group(3)	test of significance	Within group significance
Time	N=50	N=50	N=50		
Zero	119.20±10.07	155.0±5.05	187.40±15.36	F=481.08 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
6 h	117.60±9.16	148.20±3.88	169.60±10.44	F=492.58 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
12h	114.80±8.39	140.60±5.12	160.70±9.37	F=430.89 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
18h	115.40±6.76	135.0±5.44	151.94±20.50	F=101.17 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
24h	116.60±5.57	128.8±5.58	150.5±6.41	F=428.04 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
30h	117.0±6.78	123.60±5.25	144.40±7.33	F=241.10 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
36h	117.0±6.14	120.40±3.48	139.90±6.89	F=235.47 P<0.001*	P1=0.003* P2<0.001* P3<0.001*
42h	116.40±7.76	116.40±4.85	135.80±9.28	F=110.80 P<0.001*	P1=1.0 P2<0.001* P3<0.001*
48h	116.40±8.51	115.40±5.03	131.50±8.70	F=70.31 P<0.001*	P1=0.512 P2<0.001* P3<0.001*

F: One Way ANOVA test
p1: difference between group 1 and 2
p2: difference between group 1 and 3
p3: difference between group 2 and 3
*statistically significant
Parameters described as mean± SD
Zero: Time of admission just after delivery.

Table (4): Diastolic blood pressure distribution among studied groups

DBP (mmHg)	Group(1)	Group(2)	Group(3)	Test of significance	Within group significance
Time	N=50	N=50	N=50		
Zero	77.60±6.56	98.20±4.82	116.80±5.89	F=572.30 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
6 h	75.40±5.42	91.60±4.68	108.40±4.68	F=558.08 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
12h	76.6±5.93	89.60±2.82	104.20±6.73	F=323.48 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
18h	76.0±4.95	87.70±4.65	100.0±7.14	F=222.38 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
24h	76.40±4.84	85.80±4.98	97.50±7.02	F=171.73 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
30h	76.0±4.95	82.0±4.95	95.70±5.98	F=180.56 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
36h	76.0±4.95	78.40±3.70	91.50±7.16	F=116.68 P<0.001*	P1=0.03* P2<0.001* P3<0.001*
42h	74.60±5.03	76.60±4.78	86.90±7.69	F=60.89 P<0.001*	P1=0.097 P2<0.001* P3<0.001*
48h	74.0±4.95	72.50±4.32	82.50±7.02	F=47.24 P<0.001*	P1=0.179 P2<0.001* P3<0.001*

F: One Way ANOVA test
p1: difference between group 1 and 2
p2: difference between group 1 and 3
p3: difference between group 2 and 3
*statistically significant
Parameters described as mean± SD
Zero: Time of admission just after delivery.

ONSD values showed significant difference between the studied groups (Table 5).

Table (5): ONSD values among studied groups

ONSD (mm)	Group (1)	Group (2)	Group (3)	Test of significance	Within group significance
Time	N=50	N=50	N=50		
Zero	4.85±0.32	6.05±0.096	6.76±0.255	F=789.08 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
6 h	4.798±0.27	5.93±0.105	6.56±0.24	F=812.77 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
12h	4.74±0.25	5.78±0.13	6.37±0.22	F=818.99 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
18h	4.72±0.20	5.59±0.17	6.21±0.19	F=793.13 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
24h	4.74±0.169	5.41±0.194	6.09±0.19	F=643.39 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
30h	4.74±0.15	5.22±0.19	5.92±0.24	F=449.78 P<0.001*	P1<0.001* P2<0.001* P3<0.001*
36h	4.74±0.17	5.03±0.19	5.79±0.23	F=362.66 P<0.001*	P1=0.003* P2<0.001* P3<0.001*
42h	4.72±0.17	4.85±0.19	5.62±0.27	F=254.03 P<0.001*	P1=1.0 P2<0.001* P3<0.001*
48h	4.69±0.20	4.72±0.18	5.42±0.35	F=132.46 P<0.001*	P1=0.512 P2<0.001* P3<0.001*

F:One Way ANOVA test
p1: difference between group 1 and 2
p2: difference between group 1 and 3
p3: difference between group 2 and 3
*statistically significant
Parameters described as mean± SD
Zero: Time of admission just after delivery.

DISCUSSION

Increase in cerebral edema is considered to be the main factor which results in further deterioration of severe preeclamptic patients to eclampsia. **Loureiro *et al.*** [15] found criteria for vasogenic edema in 100% of their 17 patients by diffusion weighted imaging. **Schwartz *et al.*** [16] showed evidence of brain edema in 20 out of 28 patients (72%) using the same methods.

In this study there was a significant difference in the mean body weight between the normotensive and preeclampsia without severe features groups. The mean body weight of the severe preeclampsia group showed no significant difference from that of the non-severe group. This is consistent with findings from other studies by **Agyemang *et al.*** [17] **and Tesfaye *et al.*** [18]**.**

We also observed that mean body mass index (BMI) of the normotensive group was significantly different from that of preeclampsia without severe features group, but there was no significant difference between severe preeclampsia and non-severe preeclampsia groups. This is consistent with a study by **Alkholy *et al.*** [19] where their values were 25.4±1.2, 26.5±0.9 and 27±1.1 kg/m^2 in normal pregnant, mild preeclampsia and severe preeclampsia groups respectively.

This observed association is thought to be as a result of the role mediated by adipose tissue which is hormonally active tissue and produces several inflammatory mediators that can act to alter endothelial function rendering the woman more vulnerable to develop preeclampsia. Consequently, not only obesity, but also excessive weight gain during pregnancy has been associated with increased concentrations of inflammatory factors which might predispose to preeclampsia [20]**.**

In our study, gestational age of severe preeclampsia group (36.06±3.73 weeks) showed a significant difference from that of non-severe group (38.46±0.95 weeks) and the normotensive (38.58±0.64 weeks).

Similarly, **Vyakaranam *et al.*** [21] reported same results where gestational age was 36.4±3.5, 36.9±2.9 and 35.8±2.6 weeks for control, mild and severe preeclampsia groups respectively.

As regard pregnancy outcome, we observed a high incidence of low birth weight (36%) in severe preeclampsia group. This incidence was 3% and 7% in the normotensive and non-severe preeclampsia groups respectively. These findings were similar to those of **Anselmini *et al.*** [22] who reported an incidence of 13.5%, 14.5% and 59% in the control, mild and severe preeclampsia groups respectively. This was attributed to abnormal placentation and insufficient placental functioning resulting in intrauterine growth restriction.

We also observed a higher incidence of multifetal pregnancies in the severe preeclampsia group (4% with twins and 4% with triplets), while it was 4% with twins in the non-severe group and only 2% with twins in the normotensive group. Similarly, **Sharami *et al.*** [23] reported an incidence of 4.5%, 9.1% and 9.9% in the control, mild and severe preeclampsia groups respectively. This finding was supported by previous studies that reported a two to threefold increase in incidence of preeclampsia in multifetal pregnancies and attributed this to multiple implantation with increased placental mass or relative placental ischemia [24].

In the present study, the prevalence of IUD use in the normotensive group was 56% which showed a significant difference from the other two groups (12% in preeclampsia without severe features and 6% in severe preeclampsia). This finding is consistent with a study performed in a cohort of South American women that reported a 40% decreased risk of preeclampsia among women with IUD in situ at the time of conception [25]. The mechanism through which IUD use reduces the risk of preeclampsia may be through a process involving endometrial injury. IUD use causes some level of mechanical injury to the endometrium, as evidenced by a chronic inflammatory response to copper IUD and an altered cytokine profile to both copper and hormonal IUDs. Endometrial injury has been demonstrated to improve implantation and subsequent placentation. There is evidence that some level of decidual injury increases the invasion potential of trophoblastic cells to the maternal spiral arteries, a process that when is inadequate may underlie preeclampsia [26].

Regarding the number of primigravida, the three groups showed no significant difference and this was supported by previous studies by **Alkholy *et al.*** [19] **and Deshmukh *et al.*** [27]**.**

In our study, the mean basal hemoglobin levels were 11.06 ±1.11, 11.01±0.77 and 10.13±1.25 gm/dl in the normotensive, preeclampsia without severe features and severe preeclamptic group respectively, which were significantly different and this was attributed to the large number of cases complicated by HELLP (hemolysis, elevated liver enzymes and low platelet count) syndrome (9 cases were complicated by complete HELLP and 4 cases were complicated by partial HELLP syndrome). In agreement with our study, **Deshmukh *et al.*** [27] reported a significant decrease of the mean hemoglobin values with the severity of preeclampsia and their values were 10.67±1.16, 9.93±1.2 and 9.46±1.98 gm/dl in the control, non-severe preeclampsia and severe preeclampsia group respectively. **Akhtar *et al.*** [28] reported contradictory results and their values were 10.58±0.9, 11.23±0.86 and 11.56±1.28 gm/dl in the normotensive, mild preeclampsia and severe preeclampsia groups respectively. There was no significant difference between the preeclamptic groups. Similar results were observed by **Heilmann *et al.*** [29] where there was a significant difference between the normotensive and preeclamptic group. But, no such significance was observed between preeclamptic subgroups. They attributed their observation to failure of normal plasma expansion as the loss of serum protein and the increase in capillary endothelial permeability led to a decrease in intravascular volume and increase tissue edema.

CONCLUSION

Ultrasonographic measurement of optic nerve sheath diameter (ONSD) provides a non-invasive, quick and readily accessible tool for evaluation of raised intracranial pressure (ICP). The results of our study showed that there was a correlation between higher ONSD measurements and severity of preeclampsia. Using a cut off value of ONSD, we can predict the risk of eclampsia.

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