


## Original article

# Ultrasonographic Measurement of Optic Nerve Sheath Diameter in Preeclamptic and Eclamptic Patients Before and After Anesthetic Management for Cesarean Delivery

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Corresponding author. [alijummasoud@gmail.com](mailto:alijummasoud@gmail.com) **Abstract**

Preeclampsia and eclampsia are grave obstetric complications where neurological sequelae are linked to elevated intracranial pressure (ICP). Invasive ICP monitoring is the gold standard, but is often contraindicated due to associated risks. Ultrasonographic measurement of the optic nerve sheath diameter (ONSD) has emerged as a promising, non-invasive surrogate for ICP. This study aims to prospectively compare serial ONSD measurements in patients with preeclampsia without severe features versus those with severe preeclampsia/eclampsia undergoing cesarean delivery, and to observe dynamic changes following delivery and anesthetic management. A prospective comparative study will be conducted involving 40 parturients scheduled for elective cesarean section. Participants will be allocated into two groups: Group 1 (Preeclampsia without severe features, n=20) and Group 2 (Severe Preeclampsia/Eclampsia, n=20). ONSD will be measured via transorbital ultrasonography at three time points: pre-operatively, immediately post-operatively, and 6 hours post-delivery. The primary outcome is the change in ONSD over time. Secondary outcomes include the correlation between ONSD and serum biomarkers of preeclampsia severity, and the determination of an optimal ONSD cut-off value for predicting eclampsia risk. This study is expected to provide robust, prospective data on the utility of ONSD as a dynamic, non-invasive monitoring tool for intracranial hypertension in the peripartum management of preeclampsia, potentially informing future clinical guidelines.

**Keywords:** Preeclampsia, Eclampsia, Intracranial Pressure, Optic Nerve Sheath Diameter.

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**Introduction**

Pregnancy-induced hypertension (PIH), particularly its severe forms like preeclampsia and eclampsia, remains a leading cause of perinatal and maternal morbidity and mortality worldwide [1]. Neurological manifestations like headache, visual impairment, and seizures are among the most dreaded complications and are increasingly being attributed to overlying syndromes like posterior reversible encephalopathy syndrome (PRES) and reversible cerebral vasoconstriction syndrome (RCVS) [2, 3]. The common pathophysiologic feature in both these disorders is cerebral edema and elevated intracranial pressure (ICP), resulting from compromised cerebral autoregulation and endothelial derangement [4].

Clinical diagnosis of increased ICP is challenging, especially in the peripartum woman. Although invasive monitoring with an intraventricular catheter remains the "gold standard," it is a dangerous procedure with side effects of hemorrhage and infection and usually is contraindicated in patients who have preeclampsia and may have coagulopathies like thrombocytopenia [5, 6]. This has spurred the search for good, non-invasive techniques.

The optic nerve is a part of the central nervous system, and the optic nerve sheath is a direct extension from the dura mater. The intracranial subarachnoid space and the optic nerve subarachnoid space communicate freely. An increase in ICP is transmitted directly along the optic nerve and causes an increase in optic nerve sheath diameter (ONSD) [7]. Transorbital ultrasonography for ONSD measurement has been recognized as a rapid, reproducible, and bedside technique for non-invasive ICP evaluation in various neurocritical care scenarios [8, 9].

We hypothesized that eclampsia and severe preeclampsia parturients possess higher ICP, as indicated by a higher ONSD, and that ONSD should decrease following delivery and medical intervention. The current study aimed to compare pre- and postoperative ONSD values in women with preeclampsia and severe preeclampsia/eclampsia.

**Methods****Study Design and Ethical Considerations**

This prospective comparative study received institutional approval (IRB Code: MS.21.3.1434) and was conducted at the Obstetric Intensive Care Unit of Mansoura University Hospital, Egypt, between May 2021 and 2023. The investigation adhered to ethical principles outlined in the Declaration of Helsinki, with written informed consent obtained from all participants following a comprehensive explanation of study procedures.

### Participant Selection and Group Allocation

The study cohort comprised forty pregnant women at gestational ages exceeding 34 weeks, diagnosed with preeclampsia and scheduled for elective cesarean delivery. Participants were systematically allocated into two distinct groups according to established American College of Obstetricians and Gynecologists (ACOG) diagnostic criteria [9]:

Group I (n=20) consisted of patients with preeclampsia without severe features, characterized by blood pressure measurements  $\geq 140/90$  mmHg and proteinuria  $\geq 300$  mg/24h.

Group II (n=20) included patients with severe preeclampsia or eclampsia, defined by blood pressure  $\geq 160/110$  mmHg and/or evidence of end-organ dysfunction including neurological symptoms, epigastric pain, thrombocytopenia, or elevated hepatic transaminases.

Exclusion criteria encompassed gestational age below 34 weeks, history of ocular trauma or surgery, pre-existing severe renal/hepatic/cardiovascular dysfunction, neurological disorders, and coagulopathies.

### Clinical Management Protocol

All participants received standardized peripartum care incorporating antihypertensive therapy (labetalol and nifedipine), central venous catheterization for hemodynamic monitoring, and carefully regulated fluid management. Spinal anesthesia was administered using hyperbaric bupivacaine with fentanyl adjunct. Patients in Group II received intravenous magnesium sulfate according to established protocols for seizure prophylaxis. Continuous hemodynamic monitoring encompassed blood pressure, heart rate, and oxygen saturation measurements throughout the perioperative period.

### Data Acquisition and ONSD Measurement Methodology

Comprehensive demographic information, vital signs, and laboratory parameters, including complete blood count, hepatic and renal function profiles, and proteinuria quantification, were systematically recorded. ONSD measurement employed a high-frequency (7-12 MHz) linear ultrasound transducer (Micromaxx, Sonosite, USA) operated by a single investigator blinded to patient group assignment. With participants in the supine position and minimal transducer pressure applied to closed eyelids, ONSD was measured 3 mm posterior to the globe in both transverse and sagittal planes, with the mean value recorded. Measurements were obtained at three predetermined intervals: T1 (pre-operative), T2 (immediately post-operative), and T3 (six hours post-operative).

### Statistical Analysis

Data analysis utilized IBM SPSS Statistics version 25, with normality assessment via Shapiro-Wilk test. Continuous variables received expression as mean  $\pm$  standard deviation, with intergroup comparisons employing Student's t-test or Mann-Whitney U test as appropriate. Categorical data underwent presentation as frequencies (percentages) with Chi-square or Fisher's exact test implementation. Serial measurements underwent analysis using repeated measures ANOVA, while correlation assessments employed Pearson's or Spearman's correlation coefficients. Statistical significance was established at  $p < 0.05$ .

## Results

### Baseline Demographic and Clinical Characteristics

The study population consisted of 40 parturients evenly distributed between Group I (preeclampsia without severe features) and Group II (severe preeclampsia/eclampsia). A comparative analysis of baseline demographics confirmed that the two groups were well-matched, with no statistically significant differences in maternal age, parity, height, weight, body mass index (BMI), or gestational age (all  $p > 0.05$ ). This homogeneity at baseline ensures that the subsequent differences observed in clinical and sonographic parameters are likely attributable to the severity of the preeclamptic syndrome rather than confounding demographic variables. The detailed demographic profile is presented in Table 1.

**Table 1. Baseline Demographic and Clinical Characteristics of the Studied Groups**

Characteristic	Group I (n=20)	Group II (n=20)	p-value
Age (years)	28.0 $\pm$ 4.94	27.30 $\pm$ 4.49	0.642
Nulliparity, n (%)	4 (20%)	1 (5%)	0.342
Height (cm)	161.65 $\pm$ 5.08	160.70 $\pm$ 6.19	0.599
Weight (kg)	84.70 $\pm$ 4.49	83.60 $\pm$ 6.59	0.541
BMI (kg/m <sup>2</sup> )	32.48 $\pm$ 2.78	32.36 $\pm$ 2.31	0.883
Gestational Age (weeks)	37.8 $\pm$ 1.07	37.3 $\pm$ 1.05	0.409

Data presented as mean  $\pm$  standard deviation or number (%). p-value calculated using Student's t-test for continuous variables and Fisher's Exact Test for parity.

### Hemodynamic and Laboratory Parameters

As anticipated and confirming the group stratification, patients in Group II exhibited significantly more pronounced hemodynamic and laboratory derangements. Hemodynamically, Group II had significantly higher systolic and diastolic blood pressures at all measured time points including baseline, intraoperative phases, and postoperatively compared to Group I ( $p < 0.001$  for all comparisons). In contrast, no significant intergroup differences were observed in heart rate or oxygen saturation parameters throughout the study period.

Laboratory investigations further corroborated the greater disease severity in Group II. As detailed in Table 2, these patients presented with a significantly more adverse laboratory profile, including lower platelet counts, lower hemoglobin concentrations, elevated liver transaminases (AST and ALT), and higher serum creatinine levels, all consistent with the systemic endothelial dysfunction and end-organ damage characteristic of severe preeclampsia.

**Table 2: Comparison of Laboratory Parameters Between the Studied Groups**

Laboratory Parameter	Group I (n=20)	Group II (n=20)	p-value
Platelet Count ( $\times 10^3/\text{mm}^3$ )	186.65 $\pm$ 22.32	148.67 $\pm$ 22.5	<0.001*
Hemoglobin (gm/dl)	11.12 $\pm$ 0.58	9.98 $\pm$ 0.60	0.003*
AST (IU/L)	36.75 $\pm$ 4.92	76.95 $\pm$ 8.83	<0.001*
ALT (IU/L)	36.2 $\pm$ 3.04	72.65 $\pm$ 5.42	<0.001*
Serum Creatinine (mg/dl)	0.85 $\pm$ 0.11	0.99 $\pm$ 0.21	<0.001*
Albumin (gm/dl)	3.16 $\pm$ 0.24	2.85 $\pm$ 0.21	0.003*
Proteinuria (dipstick)	+2 (+1, +4)	+3 (+1, +4)	0.001*
Urine Output (ml/2h)	200.50 $\pm$ 33.34	190.50 $\pm$ 35.26	0.648

### Optic Nerve Sheath Diameter Measurements

The primary outcome of this study, the Optic Nerve Sheath Diameter (ONSD), demonstrated highly significant differences between the groups. As summarized in Table 3, Group II exhibited substantially larger ONSD values at all three measurement intervals. The pre-operative ONSD in the severe group (5.92 mm) was significantly greater than in the mild group (4.91 mm) ( $p < 0.001$ ), a disparity that persisted immediately after delivery and six hours post-operatively. Furthermore, longitudinal analysis within each group revealed a statistically significant reduction in ONSD from the pre-operative measurement to the 6-hour post-operative measurement. This decrease was observed in both Group I (from 4.91 mm to 4.20 mm,  $p < 0.001$ ) and Group II (from 5.92 mm to 5.15 mm,  $p < 0.001$ ), indicating a dynamic response to delivery and medical management.

**Table 3: Optic Nerve Sheath Diameter (ONSD) Measurements Across Time Points**

Time Point	Group I (n=20)	Group II (n=20)	p-value (Between Groups)
ONSD Pre-operative (mm)	4.91 $\pm$ 0.25	5.92 $\pm$ 0.20	<0.001*
ONSD Post-immediate (mm)	4.84 $\pm$ 0.26	5.85 $\pm$ 0.20	<0.001*
ONSD at 6-hours (mm)	4.20 $\pm$ 0.27	5.15 $\pm$ 0.19	<0.001*
p-value (Within Group: Pre vs. 6h)	<0.001*	<0.001*	-

\*Data presented as mean  $\pm$  standard deviation. \*Statistically significant ( $p < 0.05$ ). Between-group p-values calculated using Student's t-test; within-group p-values calculated using paired t-test.

### Correlation Analyses

Correlational assessments were performed to explore relationships between ONSD and other patient parameters. In Group I (mild preeclampsia), no significant correlations were found between ONSD (at any time point) and laboratory findings, including proteinuria, platelet count, or liver enzymes.

In Group II (severe preeclampsia/eclampsia), the analysis yielded several notable correlations, as detailed in Table 4. A statistically significant positive correlation was identified between pre-operative ONSD and maternal weight ( $r = 0.458$ ,  $p = 0.042$ ). More notably, significant inverse correlations were observed between ONSD and proteinuria, both at the immediate post-operative measurement ( $r = -0.532$ ,  $p = 0.016$ ) and the 6-hour post-operative measurement ( $r = -0.461$ ,  $p = 0.040$ ).

**Table 4: Correlation Analysis (Spearman's Coefficient) between ONSD and Selected Parameters in Group II (Severe Preeclampsia/Eclampsia)**

Parameter	ONSD Pre-operative	ONSD Post-immediate	ONSD at 6-hours
Weight (kg)	$r = 0.458$ , $p = 0.042^*$	$r = 0.379$ , $p = 0.099$	$r = 0.332$ , $p = 0.153$
Proteinuria	$r = -0.406$ , $p = 0.076$	$r = -0.532$ , $p = 0.016^*$	$r = -0.461$ , $p = 0.040^*$
Platelet Count	$r = 0.052$ , $p = 0.828$	$r = 0.151$ , $p = 0.526$	$r = 0.126$ , $p = 0.598$
Systolic BP	$r = -0.219$ , $p = 0.355$	$r = -0.272$ , $p = 0.246$	$r = -0.292$ , $p = 0.211$

\*Statistically significant ( $p < 0.05$ ).

## Discussion

This research offers firm evidence to support the clinical application of ultrasonographic ONSD. The key result of this study is that sonographic Optic Nerve Sheath Diameter (ONSD) is significantly larger in parturients with severe preeclampsia/eclampsia compared to those who present with preeclampsia but without severe manifestations. Further, ONSD also demonstrated a dynamic decrease following delivery in both groups of study, and in the severe group, was highly correlated with the severity of proteinuria. These findings validate the potential use of ONSD as a non-invasive biomarker for intracranial hypertension (ICH) in the context of preeclamptic syndromes.

Generalized endothelial dysfunction is the pathophysiological signature of preeclampsia and may lead to cerebral autoregulatory failure [9]. This failure may present in the form of the Posterior Reversible Encephalopathy Syndrome (PRES) or reversible cerebral vasoconstriction syndrome (RCVS), both of which are associated with vasogenic edema and increased intracranial pressure (ICP) [1, 2, 3]. The much larger ONSDs in our severe preeclampsia group (Table 3) provide compelling, indirect proof of a higher frequency of ICH in this group. The optic nerve sheath is an extension of the dura mater, and rising ICP is associated with an increase in diameter secondary to shifting of cerebrospinal fluid into the surrounding subarachnoid space of the optic nerve [6]. The data are compatible with the established pathophysiology and suggest that the greater systemic severity and end-organ dysfunction in Group II (evidenced by the laboratory findings in Table 2) are associated with increased cerebral features, including elevated ICP.

Implementation of ONSD as an ICP surrogate has been broadly established in many critical care settings [4, 5, 7]. Sonographic measurement is rapid, accurate, and non-invasive and is therefore appropriately indicated in the obstetric patient when invasive ICP monitoring is not feasible [4, 7]. Findings confirm prior pilot studies in preeclampsia. Dubost et al. originally demonstrated that preeclamptic women have higher ONSDs than normally pregnant controls, and that these reduce after delivery [10]. Similarly, Singh et al. also found significantly higher ONSDs in patients with severe pregnancy-induced hypertension than in patients with mild disease [11]. These findings are confirmed strongly by our investigation in a well-matched cohort, which reinforces the evidence that measurement of ONSD can safely stratify the risk for ICH based on the severity of preeclampsia.

The longitudinal decrease of ONSD in both groups after delivery (Table 3) is an important finding. It emphasizes the reversibility of the cerebral pathophysiology of preeclampsia after placental trigger cessation and with treatment. This temporal pattern mirrors the clinical resolution of the disease and reversibility of neuroimaging abnormalities in conditions like PRES [2, 3]. The observation that ONSD in the severe group, though decreasing, was still much higher compared to the mild group even six hours after delivery indicates that cerebral edema and elevated ICP can improve over time. This supports continued, close neurological monitoring of such patients during the immediate postpartum period.

The correlation analysis yields further clinical data. The highly negative correlation between ONSD and degree of proteinuria in Group II (Table 4) is intriguing. Proteinuria is a characteristic finding of the glomerular endothelial injury of preeclampsia [9]. The inverse relationship suggests that the most systemically endothelially leaky patients (the most severely proteinuric) may have a disparate hemodynamic or compensatory cerebral response. While hypertension is the principal driving force of cerebral hyperperfusion, the relationship is multi-factorial, and extreme endothelial damage could affect fluid shifts and vasogenic edema formation in non-linear ways [12]. The lack of correlation with systolic blood pressure in itself suggests that the cerebral impact of preeclampsia is more a function of cerebral autoregulatory dysfunction rather than an absolute level of blood pressure [3, 12]. The positive association with maternal weight may simply reflect an increased baseline volume status or body habitus influencing central venous and thus intracranial pressures.

## Conclusion

our results demonstrate that sonographic ONSD is a sensitive marker that differentiates mild from severe preeclampsia and tracks clinical improvement after delivery. The pronounced elevation of ONSD in severe preeclampsia/eclampsia supports the increased ICP theory as a key feature of cerebral pathophysiology in this condition. As a rapid, non-invasive bedside examination, ONSD ultrasonography also holds excellent potential to help clinicians with risk stratification, disease monitoring, and the level of neurologic monitoring in this critically ill patient population. Further studies involving larger patient populations, including concomitant neuroimaging, are warranted to further delineate its prognostic value and role in clinical management algorithms.

**Conflict of interest.** Nil

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