

CASE SERIES ON NON -INVASIVE PREDICTORS OF RAISED INTRACRANIAL PRESSURE IN SEVERE TRAUMATIC BRAIN INJURY: AN OBSERVATIONAL STUDY OF OPTIC NERVE SHEATH DIAMETER AND NCCT BRAIN FINDINGS

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ABSTRACT

Background: Raised intracranial pressure (ICP) is a crucial indicator of morbidity and mortality among patients with severe traumatic brain injury (TBI). Early detection of intracranial pressure (ICP) is essential for timely intervention. Unfortunately, monitoring ICP is often limited by due the invasiveness of the procedure, cost, and associated risks. Hence, non-invasive predictors such as optic nerve sheath diameter (ONSD) and computed tomography (CT)-based scoring systems like the Marshall and Rotterdam scores have gained importance. **Objective:** To evaluate the role of ONSD, Marshall CT classification, and Rotterdam score as non-invasive predictors of raised ICP in patients with severe TBI. **Materials and Methods:** This observational case series was conducted among patients presenting with severe TBI. ONSD was measured using bedside ultrasonography, and all patients underwent non-contrast CT (NCCT) brain imaging. CT findings were categorized using the Marshall classification and Rotterdam scoring system. The correlation between increased ONSD values, CT-based scores, and clinical indicators of raised ICP was analyzed. **Results:** The study demonstrated that patients with clinical features suggestive of raised ICP showed significantly increased ONSD values. Higher Marshall grades and Rotterdam scores were consistently associated with radiological signs of intracranial hypertension, including midline shift, compressed basal cisterns, and intracranial hemorrhages. A positive correlation was observed between ONSD measurements and both CT-based scoring systems. The Rotterdam score showed slightly better prognostic value due to its inclusion of additional parameters such as traumatic subarachnoid hemorrhage and intraventricular hemorrhage. **Conclusion:** ONSD measurement, along with Marshall and Rotterdam CT scores, serves as reliable and complementary non-invasive predictors of raised ICP in severe TBI. Their combined use enhances diagnostic accuracy and aids in early risk stratification and management. These modalities offer a practical alternative in settings where invasive ICP monitoring is not feasible, thereby contributing to improved patient outcomes.

INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of disability, especially in low-resource setting countries.^[1] Managing patients with head injury is very critical in the emergency department, and elevated intracranial pressure (ICP) is one of the determinants in predicting poor neurological outcomes.^[2] Timely detection, along with early management of increased ICP, enhances survival and functional recovery. The major challenge in measuring ICP is the invasiveness of the procedure, which requires skilled physicians, as well as the risks

of infection and haemorrhage.^[3] Identifying a non-invasive method with high predictive power is the need of the hour.

Numerous non-invasive techniques for measuring ICP are available, with varying sensitivities and specificities. Measuring the diameter of the optic nerve sheath by ultrasonography is a good alternative because it is closely adjacent to the subarachnoid space.^[4,5] Similarly, fundus examination also detects increased ICP by identifying papilledema, though its accuracy is not high in acute conditions.^[6] Neuroimaging with non-contrast computed tomography (NCCT) showing effacement of basal

cisterns, cerebral oedema, and midline shift serves as a radiological marker of increased ICP.^[7]

Studies comparing these non-invasive procedures and their correlation with increased ICP are limited.^[4,8] Additionally, the use of this procedure as a screening tool among brain-injured patients at the emergency department is not known. We present a case series of 7 patients with severe brain trauma to evaluate the use of non-invasive predictors, such as optic nerve sheath diameter, Marshall Index, and Rotterdam Score, on NCCT brain imaging to predict raised intracranial pressure. Our findings will provide valuable information on the feasibility of using this bedside assessment to detect raised ICP at the emergency department.

MATERIALS AND METHODS

Study design and setting: Case series of ten patients with head injury presenting at the MGM Medical College, Kamothe, Navi Mumbai, a tertiary care centre.

Study population: Patients with severe head injury presenting in the at Emergency department during the study period were included based on the following criteria

Inclusion Criteria

Patients aged ≥ 18 years presenting with severe traumatic brain injury (Glasgow Coma Scale ≤ 8) within 24 hours of head trauma were included.

Exclusion Criteria

Pre-existing ocular pathology, orbital trauma, penetrating head injury, known intracranial pathology (tumour, hydrocephalus)

Study tool

The non-invasive predictor tool used in the study was the Glasgow Coma Scale (GCS), Optic Nerve Sheath Diameter (ONSD), Marshall Scale and Rotterdam CT Score. Increased ONSD was considered when the value was more than 5.0 mm, an abnormal Marshall Scale was considered for grade IV and above, while an abnormal Rotterdam score was considered for scores of 4 and above.

RESULTS

Case 1: A 69-year-old male with a history of a fall from a bike presented with severe headache, profuse sweating, vomiting and decreased sensorium. No seizure was present. Patient was afebrile, with pulse rate 86/minute, blood pressure (BP) 190/101 mmHg, and SpO₂ 100% at 8 L/min. GCS score was E2V1M5 with the left pupil non-reactive to light and mid dilated, while the right pupil was reactive to light. ONSD was 6.0 mm, Marshall score class IV and Rotterdam 5. NCCT brain findings reported a large left subdural hematoma (SDH) causing compression over the left lateral ventricle with midline shift.

Case 2: A 65-year-old male with an alleged history of a railway track accident by an unknown mechanism. There was a history of loss of

consciousness but no vomiting, ENT bleed or seizure. On examination, the patient was afebrile with pulse 108, BP 185/122 mmHg and SpO₂ 100% on ventilator support. GCS score was E1V1M1 with both pupils reactive to light. ONSD was 5.8 mm, Marshall score class IV and Rotterdam 4. The NCCT brain showed a right frontoparietal extradural hematoma (EDH) with a right parietal depressed skull fracture with pneumocephalus, multiple diffuse intraparenchymal bleeds in the bilateral cerebral hemispheres.

Case 3: A 35-year-old male with a known history of seizure disorder had an episode of seizure followed by a fall. The patient had multiple episodes of vomiting. On examination, the patient had a pulse of 102, BP 100/70 mmHg and SpO₂ 100% on pressure-regulated volume control (PRVC) ventilation. GCS was E1VtM3 with the left pupil non-reactive to light and the right pupil reactive to light. ONSD was 5.6 mm, Marshall Score class IV and Rotterdam 5. NCCT brain showed left frontotemporoparietal SDH causing mass effect.

Case 4: A 40-year-old male sustained a head injury and left upper limb amputation following a hit by a train. There was no headache, vomiting, seizure, ENT bleed or chest or abdomen injury. On examination, vitals were stable with GCS on arrival E2V2M5, which deteriorated to E1V2M4. The bilateral pupil was reactive to light. ONSD was 5.7 mm, Marshall score class IV and Rotterdam 5. The NCCT brain showed a right parietooccipital depressed fracture with small EDH and pneumocephalus. There was also a right supraorbital ridge and a right zygomatic fracture.

Case 5: A 45-year-old male with an alleged history of road traffic accident (RTA) and fall from a bike presented with a history of loss of consciousness (unknown duration). There was bilateral nasal bleed with multiple episodes of vomiting but no seizure. On examination, the patient had a pulse of 116, BP 98/60 mmHg, and SpO₂ 100% on pressure-regulated volume control (PRVC) ventilation. GCS was E2VtM5 with bilateral pupils reactive to light. ONSD was 4.6 mm, Marshall score class III and Rotterdam 4. The NCCT brain showed right temporal contusion with left sylvian SAH, left high parietal contusion and left temporal bone fracture.

Case 6: A 22-year-old female with a history of a fall from height presented with loss of consciousness. There was one episode of vomiting with no ENT bleed or seizure or chest or abdominal trauma. Pulse was 80/min, BP 150/100 mmHg with SpO₂ 98% on PRVC mode. GCS was E1VtM1 with the left pupil reactive to light, dilated and fixed, while the right pupil was reactive to light. ONSD was 4.8 mm, Marshall score class VI and Rotterdam 6. NCCT brain showed multiple contusions in the bilateral parietal and occipital regions with edema with hypodensity in the pons (hypoxic injury)

Case 7: A 52 years old male with history of slip and fall from bike and loss consciousness with no history of seizures, ENT bleed, chest or abdominal trauma.

On examination pulse was 105/min, BP 150/90 mm Hg, SpO2 100 on room air. GCS was E1V2M4 with bilateral pupil reactive to light. ONSD was 5.5 mm, Marshall score class II and Rotterdam 3. NCCT brain showed contusion in left frontal lobe.

Case 8: A 40-year-old male with an unknown mechanism of trauma sustained a head injury and was taken to a private hospital, where intubation was done due to low GCS, and later referred to our hospital. Patient was unconscious with pulse 60 /min, BP-118/70 mmHg, SpO2 97% on PRVC. GCS was E1VtM1 with bilateral pupils constricted. ONSD was 5.6 mm, Marshall score class III and Rotterdam 5. The NCCT brain showed signs of right parieto-occipital SDH with contusion.

Case 9: A 22 year old male with history of RTA slip and fall, sustained head injury with loss of consciousness with no vomiting, seizures, ENT bleed or trauma at other sites. On arrival, GCS was E2VtM4 with pulse 108/min, BP 130/90 and SpO2 100 % on PRVC mode. Patient was intubated on sedation. ONSD was 4.8 mm, Marshall score class IV and Rotterdam 4. CT brain showed a right temporoparietal thin rim of SDH, left parietal small contusion with right orbital fracture.

Case 10: A 26 years old male sustained head injury due to fall from bike. Patient was admitted elsewhere, and intubation was done with conservative management initially, following which extubation was done after 2 days. He developed multiple episodes of seizures, hence re-intubated and referred to our centre. On examination, pulse 112/min, BP 156/60, SpO2 was 100% on PVRC. GCS was E1VtM1 with bilateral pupil fixed, absent gag and cough reflex. ONSD was 5.5 mm, Marshall score class III and Rotterdam 5. CT brain showed bilateral multiple hemorrhagic contusions with diffuse cerebral edema with inter hemispheric SDH with pneumocephalus.

The age of patients with severe TBI ranged between 22 and 69 years of age with 9 out of 10 being males. Our study found four out five patients with SDH to have an elevated ONSD (>5.0 mm).



Figure 1: Case 1 showing a large left subdural hematoma (SDH) causing compression over the left lateral ventricle with midline shift

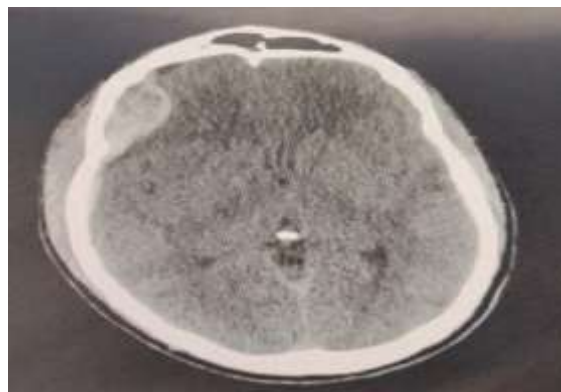


Figure 2: Case 2 showing right frontoparietal extradural hematoma (EDH) with pneumocephalus

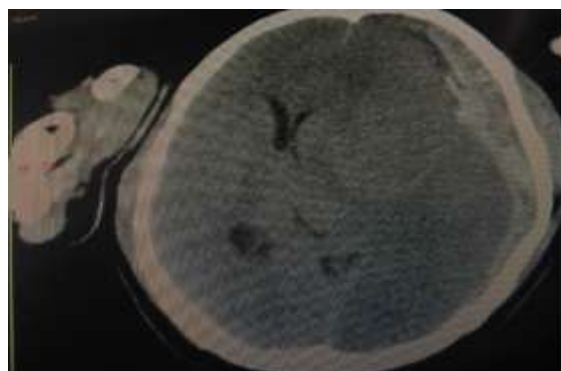


Figure 3: Brain CT of Case 3 showing left frontotemporoparietal SDH with mass effect.

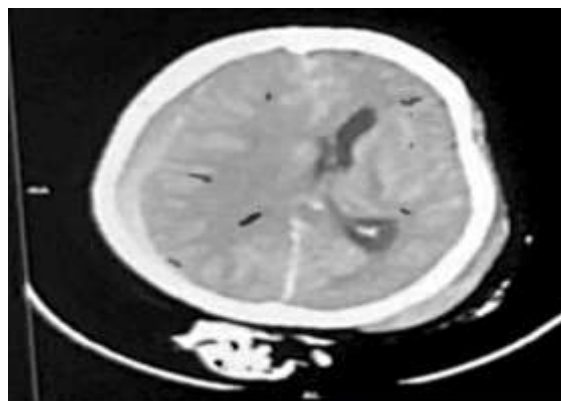


Figure 4: Case 8 showing right parieto-occipital SDH with contusion

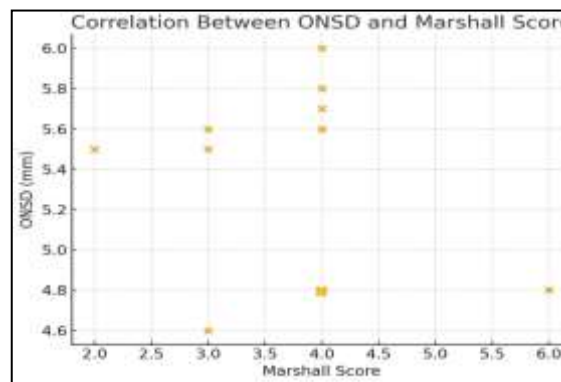


Figure 5: Correlation between the ONSD and Marshall score (N=10)

Table 1: Summary of ONSD, Marshall score and Rotterdam score (N=10)

Parameter	Abnormal (N)	Percentage
Raised ONSD (>5.0mm)	8/10	80%
Marshall CT Score	6/10	60%
Rotterdam CT Score	9/10	90%

DISCUSSION

In this case series of 10 patients with severe TBI, we evaluated the utility of ONSD measurement and non-contrast CT (NCCT) brain findings as predictors of raised ICP. Our observations suggest that both modalities offer clinically relevant insights, with certain distinctions based on the type of intracranial pathology.

We observed that patients with subdural hemorrhage (SDH) demonstrated higher ONSD values compared to those with subarachnoid hemorrhage (SAH). This finding may be explained by the pathophysiological differences between these entities. SDH typically produces a mass effect with progressive compression of adjacent brain parenchyma, leading to a sustained increase in ICP and consequent distension of the optic nerve sheath. In contrast, SAH is often associated with diffuse meningeal irritation and transient elevations in ICP, which may not result in the same degree of optic nerve sheath expansion. Mehsam et.al,^[9] also found focal mass lesions to correlate more strongly with elevated ONSD than diffuse injuries. In their study, greater ONSD values were found to be significantly associated with increased mortality and also delayed recovery among TBI patients.

ONSD measurement, performed via bedside ultrasonography, offers several advantages including rapidity, repeatability, and non-invasiveness. It reflects changes in cerebrospinal fluid dynamics due to the continuity between the intracranial subarachnoid space and the optic nerve sheath. A study by Kasinathan et al,^[10] found the ONSD value of more than 5.23 mm to have a diagnostic accuracy of 80.7% to predict elevated ICP, with a sensitivity and specificity of 82% and 78% respectively. Previous literature suggests a threshold ONSD of approximately 5–6 mm for predicting raised ICP, although variability exists depending on patient population and measurement technique.^[11] Our findings reinforce the utility of ONSD as a practical adjunct in emergency and critical care settings, particularly where invasive monitoring is not feasible.

We found that the Rotterdam CT score demonstrated better correlation with clinical indicators of raised ICP compared to the Marshall classification. This may be attributed to the more comprehensive nature of the Rotterdam score, which incorporates additional variables such as basal cistern status, intraventricular hemorrhage, and traumatic subarachnoid hemorrhage. In contrast, the Marshall classification, though widely used, is relatively limited in scope and may not fully capture the spectrum of secondary brain injury. The superiority of the Rotterdam score

observed in our study aligns with previous research indicating its stronger prognostic value for mortality and unfavorable outcomes in TBI.^[12] Its ability to stratify injury severity more precisely may also explain its better performance in reflecting raised ICP states.

The Rotterdam score is developed to address the limitations of Marshall system, mainly to classify patients presenting with multiple types of injuries.^[13,14] Although both systems are valuable in predicting an outcome, since Rotterdam score is a more recently developed tool, it incorporates additional variables such as subarachnoid hemorrhage and should be a preferable measure.^[15]

The combined use of ONSD and CT scoring systems may further enhance diagnostic accuracy. While ONSD provides a dynamic, bedside assessment of ICP, CT imaging offers structural and etiological insights. Together, they can guide early decision-making, including the need for escalation of care, neurosurgical intervention, or transfer to higher centers.

However, our study has several limitations. The small sample size (n=10) limits the generalizability of the findings. Additionally, the absence of direct invasive ICP measurements precludes definitive validation of ONSD and CT parameters against gold-standard values. Inter-observer variability in ONSD measurement and CT interpretation may also influence results. Future studies with larger cohorts and correlation with invasive ICP monitoring are needed to establish standardized thresholds and validate these findings.

CONCLUSION

The findings of this study suggest that ONSD measurement using ultrasonography serves as a feasible and non-invasive tool that correlates well with radiological scoring systems such as the Marshall classification and Rotterdam score. This reinforces its role as a valuable screening tool, especially for use in emergency and critical care settings where rapid decision-making is essential since invasive ICP monitoring is not feasible. Among the two CT-based scores, the Rotterdam may provide more detailed prognostic information due to its incorporation of additional parameters, such as traumatic subarachnoid haemorrhage and intraventricular haemorrhage. Additionally, the combined use of ONSD measurement with Marshall and Rotterdam scores can enhance overall diagnostic accuracy, allowing better risk stratification, improved prognosis prediction, and potentially better outcomes in severe TBI patients.

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