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Non-Invasive Assessment of Cerebral Edema in Traumatic Brain Injury: A Comparative Review of Radiological Imaging, Optic Nerve Sheath Diameter, and Ophthalmoscopy

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A B S T R A C T

Traumatic brain injury (TBI) initiates a destructive cascade of secondary pathophysiological processes, primarily post-traumatic cerebral edema, which drives intracranial hypertension and severely worsens clinical outcomes. Although invasive intracranial pressure (ICP) monitoring remains the historical gold standard, its practical utility is limited by procedural risks such as infection and hemorrhage, alongside a shortage of neurosurgical resources in resource-limited settings. Consequently, establishing reliable non-invasive diagnostic strategies has become crucial in modern neurocritical care. This review provides a comparative analysis of three key non-invasive modalities operating along the craniospinal axis: radiological imaging (CT/MRI), ultrasound-guided optic nerve sheath diameter (ONSD) measurement, and ophthalmoscopy. While neuroimaging serves as the macro-structural baseline for identifying mass effect and edema volume, point-of-care ultrasound of the ONSD acts as a dynamic, real-time proxy for acute ICP fluctuations. Concurrently, ophthalmoscopy evaluates the visual endpoint of sustained intracranial hypertension through papilledema assessment. By synthesizing the diagnostic sensitivity, latency, and operational limitations of each method, this paper establishes an integrated clinical framework to optimize bedside triage and therapeutic monitoring for patients with cerebral edema.

Introduction

Traumatic brain injury (TBI) represents an escalating global epidemiological crisis, standing as a primary contributor to mortality, permanent neurological deficits, and severe socioeconomic burdens across modern healthcare systems (5, 21). While the primary insult is determined instantly at the moment of mechanical impact, the ultimate neurological outcome of the patient is largely dictated by a secondary sequence of biochemical, metabolic, and cellular cascades that evolve over hours and days post-injury (11). Among these

secondary pathological processes, the development of post-traumatic cerebral edema stands out as the most critical and deadly driver of patient deterioration (22, 24). The abnormal and rapid accumulation of fluid within the rigid, unyielding confines of the bony cranium triggers a predictable escalation in intracranial pressure (ICP), causing a profound reduction in cerebral perfusion pressure (CPP), global tissue ischemia, and ultimately leading to catastrophic brain herniation syndromes (4, 8, 25).

Pathophysiologically, cerebral edema in the injured brain manifests through a highly dynamic and overlapping interplay of cytotoxic and vasogenic mechanisms (26, 27). Cytotoxic edema develops early at the cellular level, where energy failure compromises ATP-dependent sodium-potassium pumps, inducing intracellular water shifts and astrocyte swelling. Conversely, vasogenic edema arises from the subsequent structural disruption of the blood-brain barrier (BBB) and microvascular endothelial tight junctions, permitting the uncontrolled extravasation of plasma proteins and fluid into the extracellular parenchyma (28, 30). In clinical neuroresuscitation, managing this expanding volume of edema demands precise, early identification and continuous risk stratification. Historically, tracking these shifts relied exclusively on invasive intracranial monitoring tools, such as intraventricular catheters and intraparenchymal microtransducers (31, 34). Although these methods offer precise numerical measurements of ICP, they expose vulnerable patients to serious procedural complications, including bacterial ventriculitis, localized parenchymal hemorrhage, and significant technical failure rates, while strictly requiring immediate neurosurgical expertise and high-cost equipment that are rarely available in peripheral or secondary-tier emergency centers (36).

To circumvent these clinical hazards, modern neurocritical care has focused intensely on exploring and validating reliable, non-invasive diagnostic surrogates that can mirror changing intracranial hemodynamics at the bedside (38). Within the spectrum of non-invasive options, three distinct modalities have gained profound clinical relevance by targeting separate anatomical and physiological segments along the craniospinal axis (40). Radiological imaging via Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) serves as the primary macro-structural baseline, visualizing overt tissue shifts, midline deviations, and ventricular compression. In contrast, point-of-care ultrasound measurement of the optic nerve sheath diameter (ONSD) acts as a highly dynamic, real-time physiological proxy for acute ICP spikes by instantly detecting micro-fluidic shifts within the retrobulbar subarachnoid space, which communicates directly with the intracranial subarachnoid pathways. Simultaneously, ophthalmoscopy stands as the fundamental clinical endpoint assessment, identifying visible papilledema and microvascular engorgement on the optic disc surface resulting from prolonged intracranial hypertension (1, 3). Although these three diagnostic approaches are frequently evaluated as isolated techniques in scientific literature, they are fundamentally connected through the contiguous anatomy of the cerebrospinal fluid (CSF) compartments. Intracranial fluid accumulations and elevated pressure waves propagate directly from the damaged parenchymal tissue into the subarachnoid space of the optic nerve, eventually presenting as structural disc changes on the fundus surface. Therefore, a comprehensive comparative review that juxtaposes the diagnostic accuracy, response latencies, and specific clinical boundaries of radiological imaging, ONSD, and ophthalmoscopy is highly warranted. This paper aims to synthesize current medical evidence and establish an optimized, multimodal non-invasive framework to enhance emergency triage, refine therapeutic interventions against cerebral edema, and improve prognostic reliability in acute TBI management (7).

Radiological Imaging in Cerebral Edema Assessment

In the immediate acute phase of traumatic brain injury, cranial computed tomography (CT) serves as the primary diagnostic foundation and neuroimaging modality globally. The clinical utility of CT is driven by its exceptional speed, high sensitivity to acute intracranial hemorrhages, and the ability to visualize macro-structural displacements resulting from expanding cerebral edema volume (2, 16). When evaluating post-traumatic brain edema, specific CT semantic signs reflect the underlying pathophysiology, including the progressive loss of grey-white matter differentiation due to cytotoxic cell swelling, continuous compression or complete effacement of the basal cisterns, and a clear midline shift away from the injured hemisphere. To standardize these morphological findings and establish a baseline correlation with intracranial hypertension, clinicians widely employ the Marshall and Rotterdam CT classification scales (37). A high Rotterdam score, characterized by compressed basal cisterns and a midline

shift greater than 5 mm, serves as a strong, clinically validated predictor of elevated intracranial pressure (ICP) exceeding 20 mmHg. However, conventional CT possesses fundamental physical constraints; it operates purely as a static, non-continuous anatomical snapshot that requires hazardous transport of an unstable neurocritical patient away from the resuscitation bay (17, 18). Furthermore, CT is fundamentally limited in its resolution during the hyper-acute phase, frequently failing to differentiate early, micro-level cytotoxic tissue changes from expanding vasogenic extracellular fluid accumulations (19).

To overcome the micro-structural resolution boundaries of computed tomography, magnetic resonance imaging (MRI) provides an advanced, multi-parametric approach to non-invasively dissecting the specific components of post-traumatic cerebral edema. Through specific sequences such as Diffusion-Weighted Imaging (DWI) and the calculation of the Apparent Diffusion Coefficient (ADC) maps, MRI can reliably separate cellular cytotoxic edema from interstitial vasogenic edema (32). Cytotoxic edema presents on DWI as restricted water diffusion with a corresponding drop in ADC values, reflecting intracellular water entrapment driven by cellular membrane pump failure. Conversely, vasogenic edema displays increased ADC values due to the enhanced free movement of water molecules within the expanded extracellular matrices resulting from blood-brain barrier degradation.

Additionally, T2-weighted Fluid-Attenuated Inversion Recovery (FLAIR) sequences provide exceptional sensitivity for identifying subtle, non-hemorrhagic parenchymal contusions and diffuse axonal injury zones that are completely invisible on standard CT scans (37). Despite these diagnostic advantages, the routine utilization of MRI within the acute resuscitation window of severe TBI remains heavily constrained. The lengthy acquisition protocols, the absolute requirement for specialized MR-compatible monitoring equipment, and the isolation of the patient inside a high-magnetic-field gantry make MRI highly unfeasible for the continuous or rapid monitoring of hyper-acute cerebral edema progression (33).

Optic Nerve Sheath Diameter (ONSD) Ultrasonography

To overcome the dynamic monitoring limitations and intra-hospital transport risks associated with serial radiological imaging, point-of-care ultrasound (POCUS) measurement of the optic nerve sheath diameter (ONSD) has emerged as a rapid, repeatable, and non-invasive bedside surrogate for detecting acute increases in intracranial pressure. Anatomically, the optic nerve is enclosed within a continuation of the three intracranial meninges—the dura, arachnoid, and pia mater—creating a continuous retrobulbar subarachnoid space that communicates directly with the chiasmatic cistern (9). Because of this direct anatomical continuity, any expansion of cerebral edema or subsequent acute spikes in intracranial pressure (ICP) induce an immediate, fluid-kinetic displacement of cerebrospinal fluid (CSF) into this retrobulbar sheath, causing a measurable circumferential expansion of its diameter (20).

The standardized technique for performing ONSD ultrasonography involves placing a high-frequency linear transducer gently over the closed superior eyelid, utilizing an ample amount of acoustic coupling gel to avoid exerting mechanical pressure on the orbit. Measurements are strictly obtained at a depth of 3 mm posterior to the junction where the optic nerve meets the globe (23). This specific region serves as the clinical benchmark because the dural sheath is highly elastic and exhibits maximum mechanical distensibility in response to transmitted pressure waves. In severe traumatic brain injury cohorts, an ONSD threshold ranging between 5.0 mm and 5.5 mm is widely recognized as a highly sensitive indicator of elevated intracranial pressure exceeding the critical threshold of 20 mmHg (29).

Despite its dynamic operational advantages, ONSD tracking possesses critical limitations that must be addressed within a comparative clinical framework. First, the technique is highly operator-dependent, requiring standardized training and a verified learning curve to minimize inter-observer variability and prevent false-positive interpretations (39). Second, localized craniomaxillofacial or ocular pathologies—such as direct orbital trauma, retrobulbar hematomas, severe facial fractures, or pre-existing optic nerve conditions like advanced glaucoma—completely invalidate the diagnostic accuracy of the ultrasound measurements. Lastly, while ONSD changes rapidly during acute pressure variations, it does not provide a precise, continuous numerical wave tracer like an invasive intraparenchymal probe, acting instead as a semi-quantitative screening instrument that signals the need for definitive therapeutic interventions or emergency decompressive surgery (35).

Ophthalmoscopy and Fundus Photography

Ophthalmoscopy represents the classic clinical modality for assessing the visual and structural endpoints of sustained intracranial hypertension resulting from expanding cerebral edema. The pathophysiological hallmark detected via this method is papilledema, which is defined as mechanical swelling of the optic nerve head induced by elevated intracranial pressure (ICP). Because the subarachnoid space surrounding the optic nerve is completely continuous with the intracranial subarachnoid space, elevated cerebrospinal fluid (CSF) pressure waves propagate anteriorly along the nerve (12). This localized pressure gradient dynamic halts normal axoplasmic transport within the optic nerve fibers at the lamina cribrosa, leading to axonal stasis, severe cellular swelling, and secondary vascular engorgement on the visible surface of the optic disc.

In modern neurocritical care settings, traditional direct ophthalmoscopy is increasingly supplemented or replaced by non-mydriatic fundus photography and digital imaging systems. These advanced modalities allow clinicians to capture high-resolution images of the ocular fundus without the absolute requirement of pharmacological pupillary dilation (mydriasis) (35). This is a critical advantage in neuroresuscitation, as preserving the patient's natural pupillary reflexes is paramount for ongoing, serial neurological examinations and the early detection of impending brain herniation signs. To standardize the severity of the edema, clinicians utilize grading systems such as the Frisén scale, which categorizes papilledema from Stage 1 (early C-shaped halo of axonal blurring) to Stage 5 (severe, total obscuration of major retinal vessels and disc boundaries) (14).

Despite its high diagnostic specificity for confirming elevated ICP, ophthalmoscopy possesses significant clinical limitations regarding latency and acute diagnostic sensitivity. The primary constraint is a pronounced temporal latency (time lag); papilledema is not an instantaneous physical reaction (13, 15). It typically requires several hours to days (often 24 to 48 hours) of sustained intracranial hypertension to develop visible structural changes on the optic disc surface. Consequently, in the hyper-acute phases of severe traumatic brain injury, a completely normal ophthalmoscopic exam does not rule out the rapid development of early cytotoxic cerebral edema or dangerous pressure spikes (6). Furthermore, the evaluation remains highly subjective and operator-dependent when digital systems are unavailable, and pre-existing ocular comorbidities can frequently mimic or confound the diagnostic interpretation of post-traumatic disc swelling (10).

Comparative Discussion and Clinical Integration

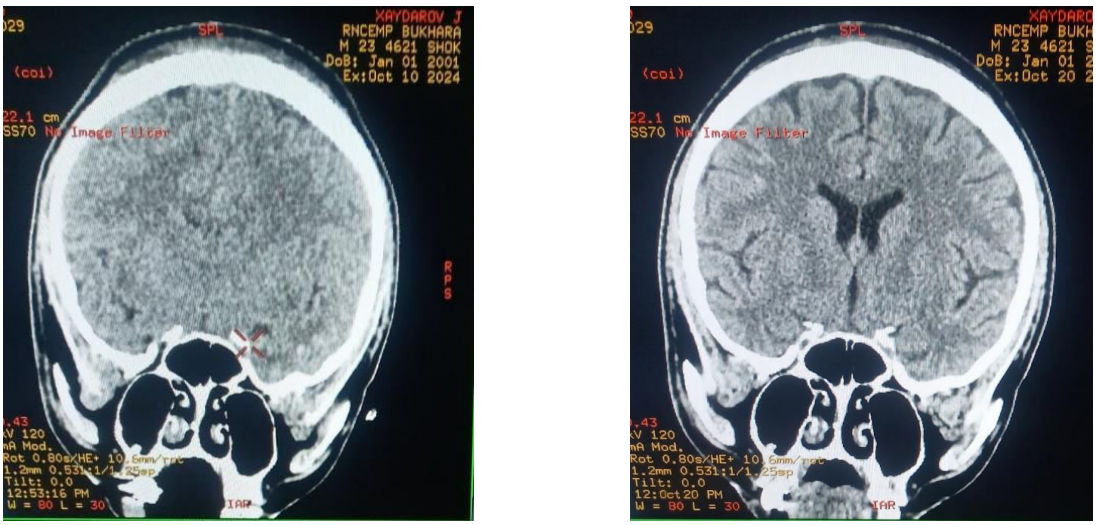
Evaluating post-traumatic cerebral edema through a purely isolated diagnostic lens often fails to capture the rapidly evolving pathophysiology of secondary brain injury. The three non-invasive modalities reviewed in this paper—radiological imaging, optic nerve sheath diameter (ONSD) ultrasonography, and ophthalmoscopy—are fundamentally interconnected through the contiguous anatomy of the craniospinal axis, yet each operates within entirely distinct temporal and structural dimensions. Understanding these distinct characteristics allows neurocritical care clinicians to transition from a single-modality approach to an integrated, multimodal non-invasive monitoring framework. Radiological neuroimaging via cranial CT and MRI serves as the structural baseline of this diagnostic triad. It offers unmatched macro-structural resolution, allowing for the direct visualization of mass effect, ventricular effacement, and physical tissue displacements such as midline shifts.

However, neuroimaging is inherently constrained by its static nature; it represents a single moment in time and carries significant clinical risks associated with intra-hospital transport, making it entirely unfeasible for continuous or rapid serial monitoring at the bedside.

To bridge this temporal gap, point-of-care ONSD ultrasonography functions as a highly dynamic, real-time physiological proxy. Because fluid shifts from expanding parenchymal edema propagate instantly into the retrobulbar subarachnoid space, ONSD measurements can detect acute, micro-fluidic changes within minutes of an intracranial pressure wave. This makes ocular ultrasound an exceptional tool for the rapid detection of acute neurological deterioration directly at the bedside, signaling the immediate need for hyperosmolar therapies or emergency surgical decompression long before a repeat CT scan can be arranged.

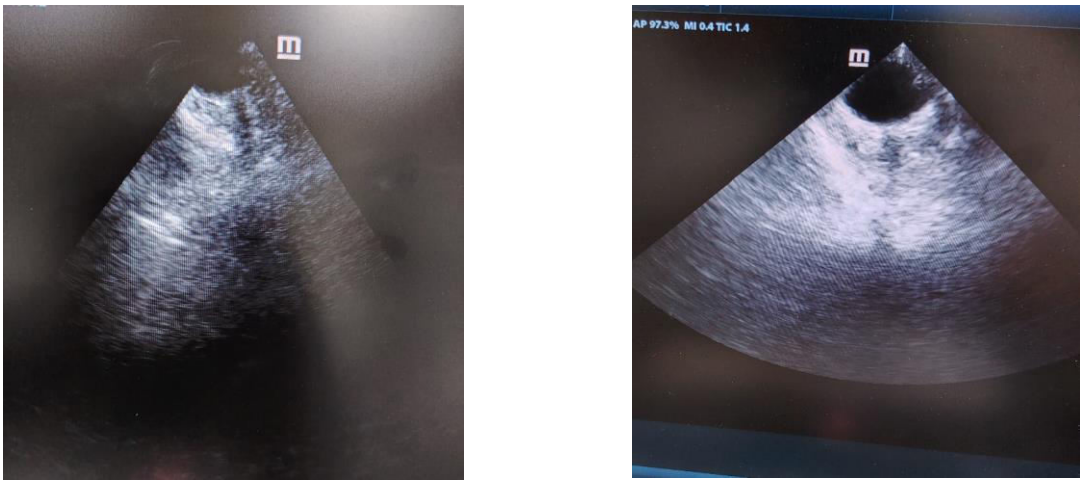
Ophthalmoscopy and fundus photography occupy the final segment of this clinical continuum, representing the structural and visual endpoint of sustained intracranial hypertension.

Figure.1 An example of dynamic change in multispiral computed tomography



Day of hospitalization **Day 10**

Figure.2 An example of dynamic change in Optic Nerve Sheath Diameter (ONSD) Ultrasonography



Day of hospitalization **Day 10**

Figure.3 An example of dynamic change in Ophthalmoscopy and Fundus Photography

Day of hospitalization **Day 10**

While ONSD ultrasonography captures the immediate, elastic expansion of the retrobulbar sheath during acute pressure spikes, ophthalmoscopy visualizes the downstream axoplasmic stasis and subsequent mechanical swelling of the optic nerve head (papilledema). The primary clinical limitation of ophthalmoscopy is its pronounced temporal latency, as visible papilledema requires hours to days of sustained pressure to manifest. Consequently, a normal fundoscopic examination in the hyper-acute phase of severe TBI is highly expected and must never be misinterpreted as an absence of expanding cerebral edema. Conversely, the presence of fully developed papilledema serves as a definitive indicator of chronic or subacute intracranial hypertension, making it invaluable for intermediate and long-term prognostic tracking.

By combining these three modalities, clinicians can establish a comprehensive clinical timeline: cranial CT establishes the initial anatomical landscape and surgical eligibility, bedside ONSD ultrasound provides continuous, real-time screening for acute pressure fluctuations, and non-mydriatic fundus photography tracks the cumulative structural impact on the optic nerve over time. This integrated approach maximizes the unique diagnostic advantages of each method while mitigating their individual limitations, ultimately refining therapeutic monitoring and improving safety in resource-limited or pre-hospital neurocritical care environments.

In conclusion, the effective management of post-traumatic cerebral edema in traumatic brain injury necessitates early detection, precise risk stratification, and continuous monitoring to prevent irreversible secondary ischemic damage. While invasive intracranial pressure monitoring remains a historical benchmark, its procedural hazards and resource-intensive nature underscore the critical clinical necessity for reliable non-invasive alternatives.

This comparative review highlights that radiological imaging, ONSD ultrasonography, and ophthalmoscopy each provide unique, highly complementary insights into the intracranial compartment. Integrating these distinct modalities into a unified, multimodal framework allows clinicians to balance structural detail with real-time physiological responsiveness. Ultimately, leveraging the combined advantages of these non-invasive tools enhances bedside triage, optimizes targeted therapies against cerebral edema, and improves prognostic accuracy without exposing vulnerable neurocritical patients to the inherent risks of invasive interventions.

Author Contributions

Visolat Khamzaevna Sharipova: Investigation, formal analysis, writing—original draft. Temur Khayrullaevich Ashurov: Validation, methodology, writing—reviewing.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

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