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RESEARCH ARTICLE

ULTRASOUND GUIDED MEASUREMENT OF OPTIC NERVE SHEATH DIAMETER IN CRITICALLY ILL PATIENTS AND ITS CORRELATION WITH RADIOLOGICAL EVIDENCE OF RAISED ICP; FOLLOWED BYITSVARIATION POST LUMBAR PUNCTURE.

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Abstract

Objectives: The diagnosis of raised intracranial pressure is very crucial in critically ill patients. The optic nerve sheath (ONS) is in continuum with the subarachnoid space; thus, an increase in ICP will result in a corresponding increase in the optic nerve sheath diameter. Ideally the best method to measure intracranial pressure is invasive intracranial devices but, in many situations, direct ICP monitoring or even imaging of the brain is not possible. In such conditions immediate diagnosis and intervention proves to be lifesaving. Hence optic nerve sheath diameter assessment by ultrasonography (US-ONSD) is emerging to be a quick and reliable tool in detecting raised intracranial tension. This study aims to use bedside optic nerve sheath diameter (ONSD) measurements by ultrasonography and its correlation with clinical and radiological evidence of raised intracranial tension and the subsequent changes following medical intervention and lumbar puncture (LP) procedure.

Materials and methods: A prospective observational study including 150 total participants from the emergency department of Bapuji Hospital, Davangerewas taken up, for a duration of six months; from January 2022 to June 2022. Group A consisted of patients above the age of 18 years presenting with clinical features of raised intracranial tension - fever, headache, vomiting, seizures, altered sensorium evaluated using Glasgow coma scale and cerebrovascular accident. Group B, those patients who did not have any clinical signs of raised ICP, otherwise healthy subjects were taken as controls. Ultrasonographic measurements of the optic nerve sheath, 3mm behind the globe in supine position was done consecutively for 2 days using a 10 MHz linear array probe, this diameter was then compared to the ONSD in MRI/CT brain scans and correlated. Midline shift, edema, effacement of sulci and gyri with effaced ventricles on radiographic analysis suggested raised ICP. In 19 patients' diagnostic lumbar puncture was performed as indicated. Prior to the procedurepharmacological measures to reduce edema was administered to the patients and subsequently US-ONSD was measured and compared to the same 30 minutes post the procedure. Appropriate statistical analysis was done and data interpreted.

Results: There was a highly significant difference noted in the ONSD between the healthy subjects and patients presenting with raised ICP.

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Mean difference was 0.8307 mmwhichsuggested that US-ONSD was higher among patients when compared to healthy subjects. Also,thedifferencebetween clinical and radiological ONSD was-0.15 suggesting radiological ONSD to be higher than clinical ONSD but the variationwas small; calculated using Hedge's g. Pre and post lumbar puncturedifference in US-ONSD was found to be 0.22 mm. Amongst patients who underwent lumbar puncture 68% patients found reduction in symptoms which was resembled by the decrease in US-ONSD.

Conclusion: Bedside measurement of US-ONSD is a useful tool to identify raised ICP. It has an advantage of being non-invasive and hence can be repeated multiple times and applied in many situations. Also changes in US-ONSD following pharmacological intervention and lumbar puncturebrings to light its reliability in identifying changes in ICP once the pressure is reduced.

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Introduction:-

The diagnosis of raised intracranial pressure is very crucial in critically ill patients. Various causes can lead to increased ICP such as traumatic brain injury, intracranial infections, stroke, intracranial hemorrhage, brain tumors, hydrocephalus, arachnoid cysts, central nervous system venous outflow impairment, idiopathic intracranial hypertension, or hepatic encephalopathy. The main goal following identification of raised intracranial pressure after the primary insult is to prevent secondary brain injury. This occurs as a consequence of a series of pathophysiologicevents reducing perfusion, oxygen and metabolite delivery to the brain, and clearance of metabolic waste and toxins from the brain. [1,2,3,4].

Ideally the best method to measure intracranial pressure is invasive intracranial devices, the gold standard being an External ventricular drain (EVD) connected with an external fluid-filled transducer which allows for ICP monitoring and therapeutic CSF drainage at the same time but in many situations even imaging of the brain becomes an impossible task. In such conditions immediate diagnosis and intervention proves to be lifesaving. [5,6,7] The use of point-of-care ultrasound (POCUS) for diagnostic assessment has recently become widespread in emergency and critical care services, to aid physical assessment of such patients.

The intraorbital portion of the optic nerve, a part of the central nervous system, extends from the ocular bulb to the optic canal and is surrounded by cerebrospinal fluid and optic nerve sheath (ONS), a membrane continuous with the dura mater of the brain. The perioptic subarachnoid space is a prolongation of the intracranial subarachnoid space, specifically, the chiasmal cistern; as the ONS is distensible, acute variations of cerebrospinal fluid pressure determine changes occurring within minutes in optic nerve sheath diameter (ONSD) [8,9,10,11,12]

Optic nerve sheath diameter ultrasound has been shown to correlate with increased ICP, thus appearing as a promising non-invasive and radiation-free bedside tool to assess elevated ICP. Hence optic nerve sonography has been applied to a variety of patients presenting with clinical symptoms of raised ICP and those who are at a risk of intracranial hypertension, including traumatic and non-traumatic brain injury, intracranial hemorrhage, metabolic disorders (hepatic failure), seizures of any etiology, suspected intracranial lesions, hypoxic injury, intracranial infections.

The aim of this study is todiscuss the basic principles of ultrasound measurement of the ONSD and its application to critically ill patients presenting with clinical features suggesting raised ICP and thus correlating its usefulness.

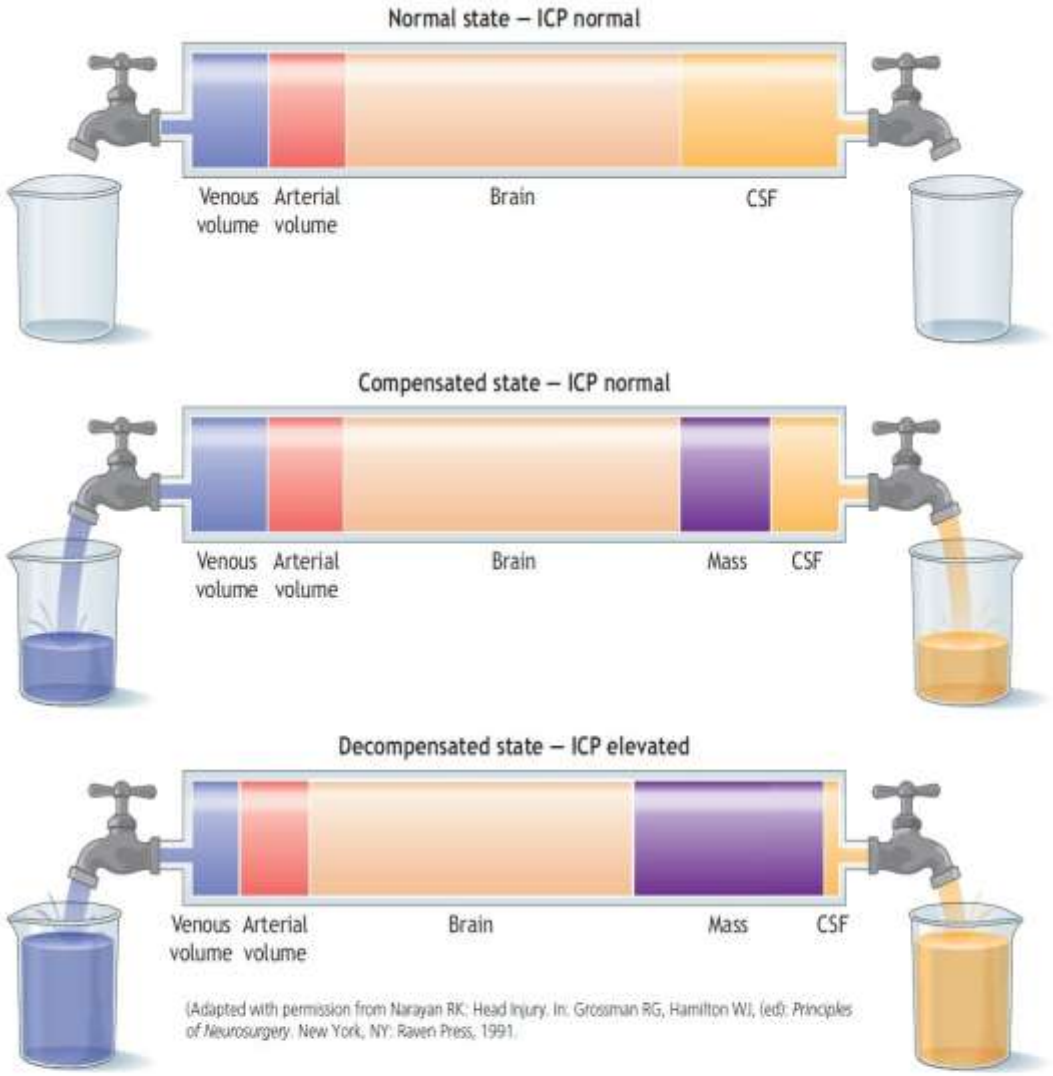
Pathophysiology of Raised Intracranial Pressure (ICP)

Thecranial part of the skull encases the brain, and bony structure (frontal, ethmoid, sphenoid, occipital, paired parietals, and paired temporal bones). As the cranium is made of solid bone, its structure is fixed and therefore the volume contained within cannot be changed. Components include brain tissue and the blood supply (mostly venous blood from within dural sinuses and arterial blood) and the cerebrospinal fluid (CSF). The volume of each of these components is restricted by the fixed space within the cranium.

In physiology, these three components exist in equilibrium with each other to satisfy this fixed volume; therefore, if the volume of one component increases, the volume of another must decrease. CSF secretion must be equal to the absorption rate, and at the same time, the arterial cerebral blood flow has to equal the effluent venous drainage to maintain this equilibrium.

The intracranial pressure (ICP) is the pressure within the cranium of the skull. Due to the fixed nature ofthe cranium, any increase in volume in the intracranial components willcause an increase in pressure. Therefore, in the absence of pathology, an equilibrium between these three components must be maintained to preserve a normal intracranial pressure.

The Monro-Kellie doctrine states thatif the volume of one of the components within the cranium increases, the volume of a different component must decrease to maintain this equilibrium and sustain a normal ICP.The normal value for intracranial pressure is 5–15mmHg. A value above 20mmHg usually signifies the point at which intervention may be required to avoid significant or life-threatening consequences.



The Monro-Kellie Doctrine Regarding Intracranial Compensation for Expanding Mass.
The volume of the intracranial contents remains constant. If the addition of a mass such as a hematoma results in the squeezing out of an equal volume of CSF and venous blood, the ICP remains normal. However, when this compensatory mechanism is exhausted, there is an exponential increase in ICP for even a small additional increase in the volume of the hematoma.

Under normal conditions, the intracranial volume is constant, and maintaining a steady ICP depends on the volume of the intracranial compartments (brain + CSF + blood); an increase in one component will cause a compensatory decrease in one or both.[13,14]Raised ICP can result from any pathological condition increasing the volume of any of the three components or from the addition of a fourth component (e.g., intracranial hemorrhage, cerebral edema, or mass), overwhelming the compensatory mechanisms. Once the reserve is exhausted, the intracranial compliance will decrease, and slight elevations in the intracranial volume will lead to dramatic changes in ICP [13,14] CO2, O_2 and blood vessels size influence ICP in the critically ill patient [13,14]

Cerebral blood flow (CBF) is driven by cerebral perfusion pressure (CPP), which is defined as mean arterial pressure (MAP) minus intracranial pressure (CPP = MAP−ICP). Cerebrovascular autoregulation (CA) is tightly linked to CPP. It refers to the capacity of the cerebral circulation to alter the vascular arteriolar resistance to maintain a constant CBF as mean arterial pressure (MAP, and thus CPP) varies. In healthy adults, CA is normally operational across a wide range of MAPs, from 50 to 150 mm Hg. Beyond the limits of autoregulation, CBF becomes pressure passive. ICP elevations can compromise the CPP leading to secondary ischemic brain injury. In the face of high ICP, brain ischemia can be partially counteracted by increasing the MAP through manipulation of the cardiac output and arterial pressure. Increased ICP can further compromise the brain parenchyma through herniation syndromes [15,16,17,18,19,20,21] ICP fluctuates under physiologic conditions, including body posture (orthostatism vs. clinostatism), cardiorespiratory variations, electroencephalography (EEG) activity, and changes of the intrathoracic (ITP) and intra-abdominal pressure (IAP; if central venous pressure exceeds ICP) [22,24,24,25,26]ICP is referenced at the level of the Foramen Of Monro.

Optic Nerve and Its Measurement

Anatomy and Physiology of Optic Nerve

The intraorbital portion of the optic nerve, developmentallyis a part of the central nervous system. It extends from the ocular bulb to the optic canal and is surrounded by cerebrospinal fluid and optic nerve sheath (ONS), a membrane made up of leptomeninges in continuity with the dura mater of the brain.

The optic nerve is approximately 40 mm long and 3 mm wide,whereas the optic nerve sheath has a thickness of approximately 1 mmwith an average diameter of 0.4 mm. From in to out, the sheath consists of the pia mater, the subarachnoid space, the arachnoid mater, and the dura mater.The subarachnoid space features a structure of arachnoid trabeculae, septa, and stout pillars. Under normal conditions, it holds approximately 0.1–0.2 mL of cerebrospinal fluid

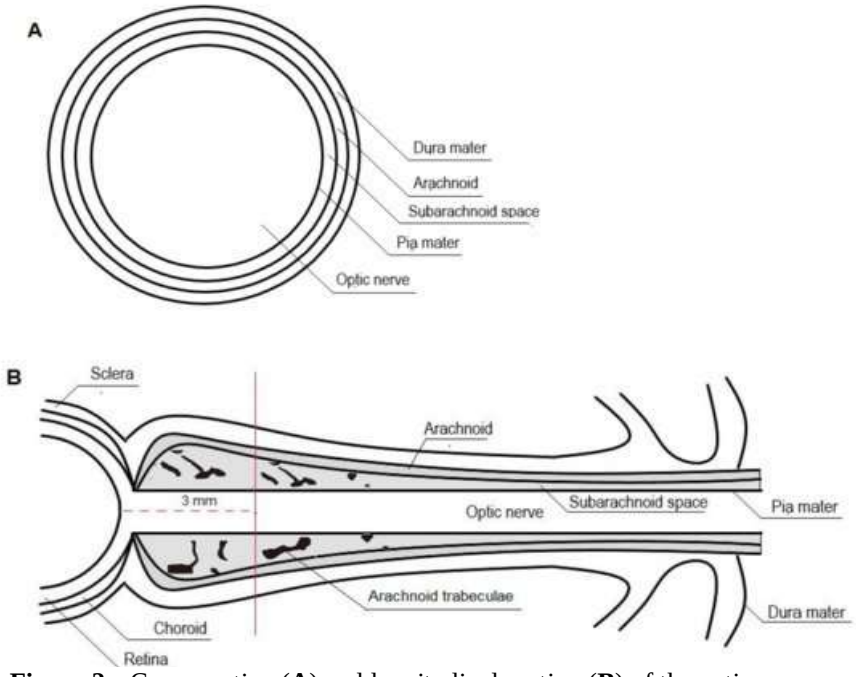


Figure 2:- Cross-section (**A**) and longitudinal section (**B**) of the optic nerve.

The perioptic subarachnoid space is a prolongation of the intracranial subarachnoid space, specifically, the chiasmal cistern.As the ONS is distensible, optic nerve sheath diameter (ONSD) changes rapidly with changing cerebrospinal fluid pressure. The ONSD is constant as long as the ICP remains within normal ranges. When ICP rises, maximum ONSD fluctuations occur in the anterior subarachnoid compartment, 3 mm behind the globe, rather than in the posterior perineural one. It has been suggested that this non-uniform enlargement may be the result of the asymmetrical distribution of the arachnoid trabeculae, with lower density in the retrobulbar ONS. Moreover, the anterior compartment of the ONS is the thinnest of the entire segment and, therefore, the most distensible.

Ultrasonographic Technique for Optic Nerve Sheath Diameter (ONSD) Measurement

Ocular ultrasound is performed using a high-frequency linear transducer (10 MHz) with the patient lying supine, the head in a neutral position and both eyes closed.

The probe is gently placed in an axial plane on the closed upper eyelid with the pointer towards the temporal side of the head; using a thick layer of sterile coupling ultrasound gel. B mode is used. A transverse sonographic section allows for visualization of the globe and the structures of the retrobulbar area, including the optic nerve in its longitudinal course [27,29,29].

Image of the optic nerve complex is seen as a homogenous hypoechoic band extending posteriorly from the bulb′s base with a hyperechoic nerve sheath and echogenic retrobulbar fat surrounding it, colordoppler may be used to facilitate optic nerve identification through visualization of the central retinal artery and vein running inside.

By convention, the ONSD measurement is performed 3 mm posterior to the papilla base by manual cursor placement on the outer contours of the optic nerve sheath. [8,9,10]

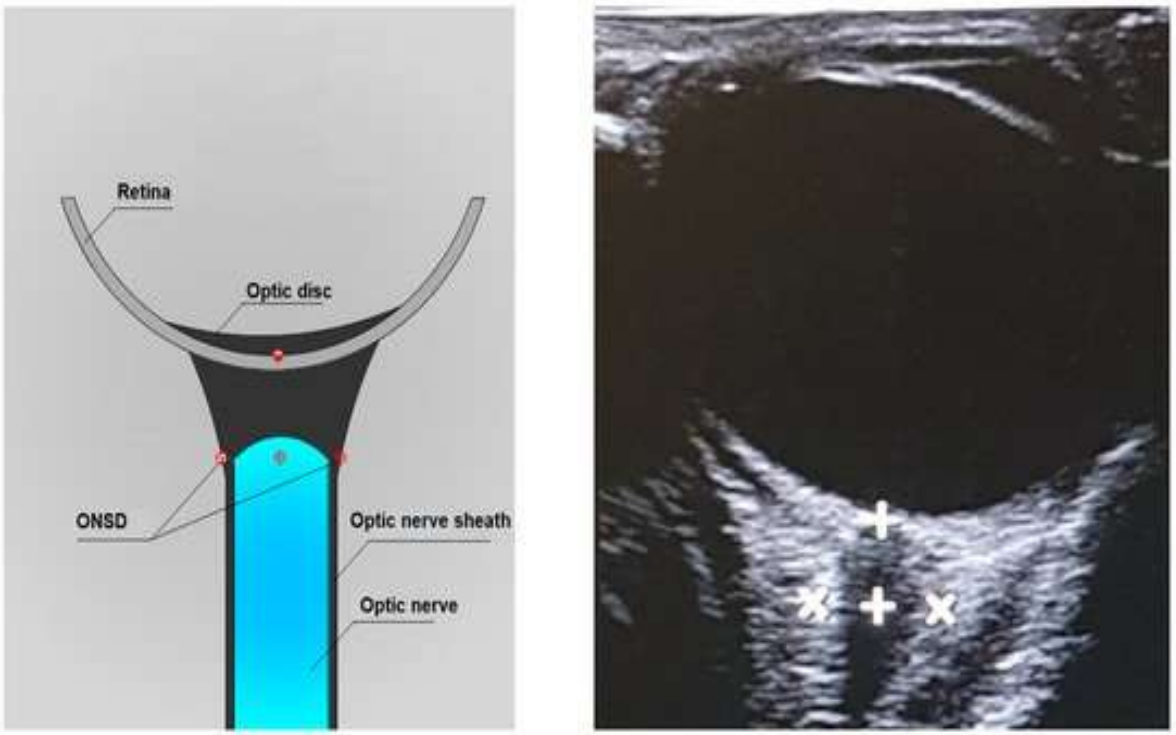
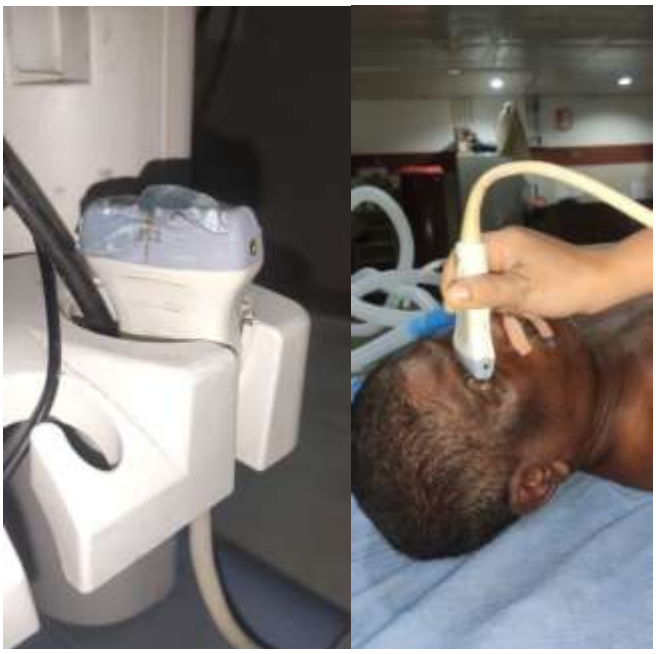
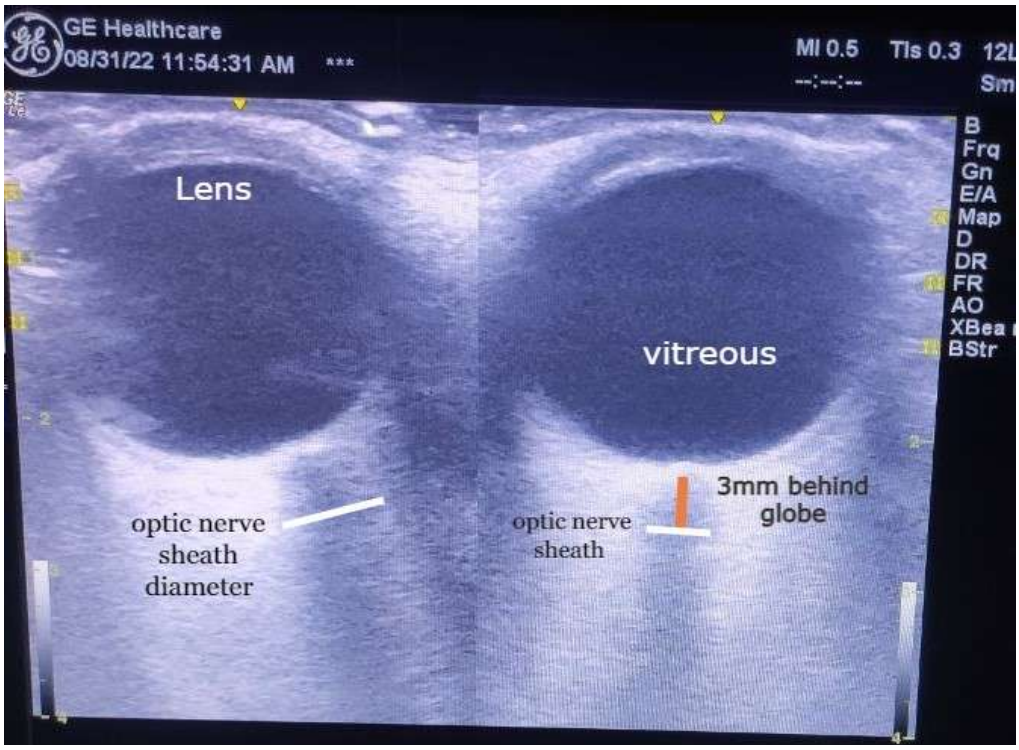


Figure 3:- Axial transbulbar approach ONSD measurement.





Materials And Methods:-

A prospective observational study on 150 adult individuals was conducted after Institutional Review Board's permission over a period of 6 months between January 2022 to June 2022. The patients were divided into two groups; A and B. Group A were, 75 healthy controls who did not present with clinical features of raised ICP. Group B included a total of 75 patients who were admitted during this period with symptoms of fever, headache, vomiting and altered sensorium with possibility of elevated ICP. Patients with a history of optic neuritis, high myopic, optic nerve trauma, are excluded from the study.

All these patients were examined in the supine position using a 10 MHz phased linear array probe on the closed eyelids. The structures of the eye were visualized to align the optic nerve directly opposite the probe, with the ONSD width perpendicular to the vertical axis of the scanning plane. The optic nerve sheath diameter was measured 3.00 mm behind the globe in both the eyes. The ONSD measurements were obtained averaging two readings from each eye to create a binocular ONSD measurement. [51,52]

All the individuals admitted were above the age of 18 years. In the controls a mean binocular ONSD was 3.96 mm. All the patients presenting with symptoms of raised ICP had a mean ONSD of 4.79 mm. [53,54,55] Imaging of the head CT/MRI was done if indicated in group B patients only. The finding of CT/MRI was reported by the radiologist, and they were correlated with bedside US-ONSD measurement [56] The patient's imaging result was considered to be positive for raised ICP if the radiologist's impression described findingssuggestive of elevated ICP such as cerebral edema, midline shift, mass effect, effacement of sulci, collapse of ventricles, compression of cisterns and ONSD >4.95 mm on T2 MRI. Amongst the subjects, 19 underwentlumbar puncture for diagnosis as indicated. Prior to the procedure pharmacological interventions to reduce edema was administered to the patients and US-ONSD was measured and compared to the same30 minutes post the procedure which was also compared to the clinical reduction in symptomsof raised ICP.The waiver of the individual consent was requested as the intervention was completely harmless, non-chargeable to the patient and no patient identification was used.

The statistical analysis was performed using the statistical software package SPSS (Chicago, IL, USA) version 26.0 for MS Windows. Continuous variables were summarized through the calculation of mean and standard error. Data was quantitative so it was subjected to paired t test and unpaired t test whenever applicable. P <0.05 (two tailed) was considered statistically significant. Hedge's g was used to check the size of the effect between the correlations.

Results:-

This was a prospective observational study which included150 adult individuals divided into two groups. The mean US-ONSD of the control and study group was 3.967 ± 0.345 mm and 4.797 ± 0.592 mm respectively (Table 1). There was highly significant (p = 0.001) difference between the groups for clinical ONSD with a mean difference of 0.830 (95% CI 0.674 – 0.987) mm which suggested that clinically ONSD was in patients as compared to healthy subjects. The Hedge's g was found to be 1.7, signifying a very large size of difference between the groups.

Table 1:- Mean clinical ONSD among the study subjects.

Study Groups	Frequency	Mean clinical ONSD (± SD)
Group A	75	3.967 ± 0.345
Group B	75	4.797 ± 0.592

ONSD: Optic Nerve Sheath Diameter

Radiological and clinical ONSD was compared, imaging showing cerebral oedema, midline shift, mass effect, effacement of sulci, collapse of ventricles, compression of cisterns and ONSD > 4.95 mm on T2 MRI was considered to be raised ICP.Out of the 75 patients that were admitted with clinical features of raised ICP, 72 patients had a mean radiological ONSD of 4.95 ± 0.69 mm. In this study 3 patients did not show a raised ICP on imaging, but ONSD was found to be 4.80 ± 0.20 mm. The difference in mean ONSD showed a statistically significant difference between the cases and controls with a **P** value < 0.001. There was a very high positive significant correlation between the clinical and radiological ONSD. Hence with increase in clinical ONSD, radiological ONSD also increases significantly (Table 2). Paired sample test showed a statistically significant mean difference of -0.15mm (95% CI: - 0.1953 to - 0.1047) with radiological ONSD higher than that of clinical ONSD.

Table 2:- Correlation between clinical and radiological ONSD.

Variable	Frequency	Mean (± SD)	Correlation coefficient	P value
Clinical ONSD	72	4.799 ± 0.604	0.965	0.001
Radiological ONSD	72	4. 950 ± 0. 694		

* P value < 0.05, statistically significant
ONSD: Optic Nerve Sheath Diameter

From group B, 19 patients underwent lumbar puncture for diagnosis.Prior to the procedure pharmacological anti-edema measures were administered to the patients and subsequently US-ONSD measured and compared to the same 30 minutes post procedure and the results analyzed. Data was tabulated as mentioned in Table no3. It was observed that the mean Pre intervention US-ONSD was 5.179 ± 0.327mm while mean post intervention US-ONSD was 4.958 ± 0.285mm, with a highly significant positive correlation.

Table 3:- Correlation between mean pre and post intervention (pharmacological measures and diagnostic lumbar puncture) US-ONSD.

Variable	Frequency	Mean (± SD)	Correlation coefficient	P value
Pre LP US-ONSD	19	5.179 ± 0.327	0.970	0.001*
Post LP US-ONSD	19	4. 958 ± 0. 694		

* P value < 0.05, statistically significant
LP US-ONSD: Lumbar puncture Ultrasonographic Technique for Optic Nerve Sheath Diameter

In this study it was observed that there was a statistically significant difference between Pre and post LP US-ONSD with a mean difference of 0.22 mm (95% CI: 0.179 to 0.262). Pre LP US-ONSD was found to be higher than Post LP US-ONSD with P value 0.001 (Hedge's g = 0.71). Hence,some reduction in ICP was noted which was determined by the decrease in US-ONSD.

Discussion:-

Emergency physicians on daily basis encounter patients presenting with clinical features of raised intracranial pressure, some examples being stroke, meningitis, meningoencephalitis, subarachnoid haemorrhage, encephalopathy due to sepsis or a metabolic component, liver cell failure presenting with hepatic encephalopathy and post resuscitation syndrome. According to the Monro-Kelly doctrine explained above, the cranium and the vertebral canal form a rigid container surrounded by a relatively inelastic dura. Any increase in its contents, that is, brain, blood, or CSF, will tend to resist an increase in the ICP until the compensatory mechanisms fail. Hence once the ICP reaches around 20-25 mmHg, even small changes in the brain volume will lead to marked elevation in the ICP but small increases do not lead to an immediate increase in ICP as the CSF gets displaced into the spinal canal.

Initially when the intracranial mass increases there is very minimal raise in the ICP due to the compensatory mechanisms but as the mass grows in size; that is more than 100-200 ml the regulatory mechanisms fail leading to drastic increase in ICP which is sustained even when only small changes in the mass occur. As the ICP goes on increasing and equals mean arterial pressure, blood flow to the intracranial space reduces thus leading to reduction in blood supply to the brain (as according to the relationship between cerebral perfusion pressure and mean arterial pressure determined by the formula CPP=ICP-MAP). The reduction in blood supply eventually leads to widespread ischemia and brain infarction.

The optic nerve sheath (ONS) is anatomically continuous with the dura mater and has a trabeculated arachnoid space through which CSF slowly percolates. On ultrasound examination, optic nerve appears homogeneous with low internal reflectivity compared with the high reflectivity of the nerve sheath; this was utilized by Ossoinig**et al** when he performed the first ultrasound measurement of the optic nerve using an A-scan technique, and subsequently described standardized A-scanning.[31]

Use of ocular sonography has proven to be invaluable addition to physical exam, several authors have investigated the relation between the ONSD and ICP; Munawar **et al**,[32] Chen L, Wang L, Hu Y**et al** [33] and Wang, J**et al**. [34] each demonstrated a positive linear relation between these two variables in neurosurgical patients and in particular, an immediate change in ONSD with change in ICP. Optic nerve sheath diameter was measured 3 mm behind the globe as the ultrasound contrast is greatest, the results are more reproducible, and anatomically the anterior nerve is most distensible. Hansen **et al**. presented data using a transorbital B-scan approach for the measurement of ONSD, [35] this approach allowed them to select a distance behind the globe to consistently measure the nerve, which was difficult to attain using A-scan techniques. Helmke and Hansen [36,37,38] demonstrated in cadaver studies that the ONSD increased by up to 60% at a distance of 3 mm behind the globe compared with only 35% at 10 mm thus confirming Liu and Kahn's [39] observations. Furthermore, they went on to show that the optimal experimental

scanning position was longitudinal (axial) where the least interobserver variability was found although there was no significant difference in measurement by lateral, axial, or transverse projection.

In our study, the average ONSD in the control group was 3.967 ± 0.345 mm and in study subjects was 4.797 ± 0.592 mm. Similar study conducted by Rajajee **et al**. who concluded that bedside measurement of ONSD is an accurate non-invasive method to identify ICP > 20 mmHg in a heterogeneous group of patients with acute brain injury with ONSD > 4.8 mm has greatest accuracy [69]. Also, in our study about 3 individuals did not have raised ICP on imaging, but their US-ONSD was found to be increased. We found for mean US-ONSD of 4.797 mm, sensitivity for detecting raised ICP was 77.8% (95% CI: 83-100%) and specificity 97%. Beare **et al**. evaluated ONS ultrasound as a non-invasive method of detecting raised ICP in African children. They concluded that sensitivity and specificity of detecting raised ICP on CT for ONSD 4.2 mm was 100% and 86% respectively. The patients with raised ICP are 51 times more likely to have a positive ONSD.It was also noted that papilledema was not found in the acute situation as it takes hours to days to develop.In this study it was also observed that there was a statistically significant difference between pre and post intervention US-ONSD, intervention included pharmacologic measures to reduce ICP along with diagnostic lumbar puncture; the mean difference being 0.22 mm (95% CI: 0.179 to 0.262). Prior to any intervention US-ONSD was found to be higher with P value 0.001 (Hedge's g = 0.71). Hence it was noted that there were significant changes in the US-ONSD which could be attributed to the medical interventions done and not to the lumbar puncture procedure as the volume of CSF collected was only for diagnostic purpose and not therapeutic.

The results were encouraging as it gave us a window for the possibility of dynamic monitoring of ICP following any methods to reduce ICP with serial measurements of US-ONSD and hence allowing for evaluation of the treatment given. Acute rise in ICP can be difficult to diagnose because the symptoms are nonspecific, and direct measurement of ICP has the attendant risks of intracranial hemorrhage and infection. In our study 72 cases had raised ICP on imaging and 73 patients had high US-ONSD on presentation with clinical signs and symptoms of raised ICP.

Conclusion:-

Early detection of raised ICP can be challenging when invasive devices are unavailable. Clinical signs of raised ICP such as headache, vomiting and altered sensorium are not always specific and are difficult to interpret. Also, in sedated patients such clinical signs are most often always missed especially when ischemic brain injury is already established and appear late. Furthermore, a normal CT scan does not always exclude raised ICP.

In conclusion, bedside sonographicmeasurement of ONSD is a useful test to identify raised ICP, being non-invasive, can be repeated multiple times as it is devoid of ionizing radiation and hence can be applied in a broad range of settings. Further studies are required to validate its usefulness for dynamic monitoring of ICP in patients presenting withraised intracranial pressure.

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Footnotes
Source of Support:
Nil.

Conflict of Interest:
None declared.

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