Ocular Ultrasound to Detect Intracranial Hypertension in Trauma Patients

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Background: Increases in intracranial pressure (ICP) after head trauma require a rapid recognition to allow for adequate treatments. The aim of this study was to determine whether dilation of the optic nerve sheath, as detected by ocular ultrasound at the bedside, could reliably identify increases in ICP assessed with an intraparenchymal probe in adult head trauma patients.

Methods: Eleven head trauma injured adult patients admitted to the intensive care unit with a Glasgow Coma Scale score ≤8, with cerebral contusion confirmed by computed tomography scan, and that required invasive ICP monitoring, were enrolled in the study. ICP values ≤20 mm Hg were considered as normal. Patients with acute or chronic ocular lesion were excluded. Ten nontrauma intensive care unit patients, with no ICP monitoring, were enrolled as control group. Invasive arterial pressure was monitored, and optic nerve sheath diameter (ONSD) was assessed by ultrasound in all the patients.

Results: Head trauma patients without intracranial hypertension had ONSD values, assessed by ultrasound, equivalent to those measured in control patients (5.52 mm ± 0.36 mm vs. 5.51 mm ± 0.32 mm). ONSD, instead, significantly increased to 7.0 mm ± 0.58 mm, when ICP rose in value to ≥20 mm Hg (p < 0.0001 vs. normal ICP and control). ONSD values were significantly correlated to ICP values (r = 0.74, p < 0.001).

Conclusions: When ICP was higher than 20 mm Hg, the ONSD diameter increased, whereas when the ICP was below 20 mm Hg, the ONSD returned to values equivalent to those assessed in control nontrauma patients. Accordingly, ocular ultrasound may be considered as a good alternative for a rapid indirect evaluation of head trauma patients’ ICP.

Key Words: Ultrasound, Head trauma, Optic nerve.

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Increases in intracranial pressure (ICP) after head trauma require a rapid recognition to allow for the adequate treatment. Accordingly, cranial computed tomography (CT) followed by invasive monitoring of ICP with an intraparenchymal probe, if required, are the recommended interventions.1

When a CT scan is not readily available, i.e., in out-of-hospital emergencies, an alternative approach to detect a possible intracranial hypertension should be considered. Thus, methods for ICP monitoring that can be easily performed in different settings and conditions have been of major interest. Ultrasound evaluation of the optic nerve sheath diameter (ONSD) has appeared as the most reliable solution.2–4

The optic nerve has an intraorbital portion that can be visualized and accurately measured by ultrasound. A direct communication between the perineural cerebral spinal fluid and the craniospinal subarachnoid space exists, and therefore when ICP increases, cerebral spinal fluid is displaced into the intraorbital sheath, and its diameter is consequently enlarged.5

The aim of this study was therefore to determine whether dilation of the optic nerve sheath, as detected at the bedside by ultrasound, could reliably identify raises in ICP assessed with an intraparenchymal probe in adult head trauma patients.

PATIENTS AND METHODS

This study was performed in the intensive care unit (ICU) of the Cannizzaro Hospital of Catania, Italy, from September 2009 to September 2010. Institutional ethical approval was obtained. Because patients’ care was not changed because of the specific intervention studied, namely the optic nerve sheath ultrasound, a non-invasive procedure, the ethical Committee waived the requirement for informed consent.

Head trauma injured adult patients admitted to the ICU with a Glasgow Coma Scale score ≤8, with cerebral contusion or hemorrhage confirmed by CT scan, and that required sedation, mechanical ventilation, and invasive ICP monitoring, were enrolled in the study. Patients with acute or chronic ocular lesion were excluded.

Patients were sedated with intravenous administration of midazolam and remifentanil. Arterial pressure was monitored with an intravascular catheter (20 G, Vygon arterial Leader Cath) inserted into the radial artery. All the hemodynamic parameters were continuously monitored. Gender, age, temperature, plasma electrolytes, blood oxygen, and carbon dioxide partial pressure were also evaluated.

All the patients had an intraparenchymal probe inserted by a neurosurgeon for the measurement of ICP (Codman Microsensor Kit). An ICP ≤20 mm Hg was considered as normal, whereas values >20 mm Hg were classified as abnormal and treated.6 If needed, noradrenalin, dobutamine,
or dopamine were used to increase mean arterial pressure (MAP) and, as a result, the cerebral perfusion pressure (CPP). CPP was calculated as the difference between MAP and ICP.

Ultrasound scan of the optic nerve sheath was performed bilaterally and in accordance to established protocols. In brief, after a thick layer of ultrasound gel was applied over the closed upper eyelid a 10-MHz linear probe (Model Vivid Expert, General Electric Medical System, Milwaukee, WI) was placed over the temporal area of the eyelid. Attention was paid to avoid pressure on the eye during the ultrasound examination. The field was then reduced to a depth of 4 cm, and a two-dimensional mode was used. The ONSD was measured 3 mm behind the globe using an electronic caliper ad an axis perpendicular to the optic nerve. In addition, the ONSD was measured in both the sagittal and the transverse plane. Each measurement was repeated twice, and values were averaged. These ONSD ultrasounds were performed within 1 hour of the insertion of ICP probe, and then repeated daily until the intracranial catheter removal. Additional ultrasound scans were repeated if episodes of rapid increase of ICP occurred. This protocol design produced from a minimum of 12 to a maximum of 28 measurement per each patient.

Ultrasound examination of the eye was performed by investigators trained in ocular nerve ultrasound (G.C. or G.M.). Interobserver variability was calculated on measurements obtained from the initial three patients where ocular ultrasound assessments were performed by the two investigators at the same time. Because of the little information available in literature regarding the normal ONSD, an additional group of ICU patients admitted for nontraumatic reasons served as control. These control patients required sedation and ventilation, equivalent to the trauma patients, but they did not have invasive ICP monitoring.

For measurements between groups, analysis of variance and Scheffe’s multicomparison techniques were employed. Linear correlation was calculated using the Pearson correlation coefficient. Measurements are reported as mean ± standard deviation. A value of \( p < 0.05 \) was considered significant.

**RESULTS**

A total of 11 patients fulfilled the inclusion criteria. Six had normal ICP in all the observations, two had continuously high ICP resistant to the treatment, and three had either normal or high ICP in relationship to the concurrent clinical conditions and consequent treatment. Ten nontrauma ICU patients were included in the control group. Data in Figures 1 and 2 are reported as the values obtained concurrently to a condition of normal ICP, increased ICP, or in control patients.

The ONSD in the control patients was 5.51 mm ± 0.32 mm (average between right and left eye). This value was not significantly different to that measured in head trauma patients without intracranial hypertension (Fig. 1). The measurements obtained in head trauma patients during the condition of normal ICP were, in fact, 5.52 mm ± 0.36 mm. ONSD, instead, significantly increased to 7.0 mm ± 0.58 mm, when ICP rose in value to >20 mm Hg (\( p < 0.0001 \) vs. normal ICP and control, Fig. 1). ONSD values were significantly correlated to ICP values (\( r = 0.74, p < 0.001 \)). The interobserver coefficient of variation for the ocular ultrasound measurements was 4.2%.

In head trauma patients, when ICP was >20 mm Hg, CPP significantly decreased from 78.4 mm Hg to 70 mm Hg (\( p < 0.02 \), Fig. 2), despite the higher MAP (\( p = 0.05 \)), in comparison with head trauma patients with ICP <20 mm Hg.

**Figure 1.** ONSD in the right (RE) and left (LE) eye in head trauma patients with ICP ≤20 mm Hg and >20 mm Hg and in control nontrauma patients. Data are shown as mean ± standard deviation. *\( p < 0.0001 \) vs. trauma patients with ICP ≤20 mm Hg and control nontrauma patients.

**Figure 2.** MAP and CPP in head trauma patients with ICP ≤20 mm Hg and >20 mm Hg and in control nontrauma patients. Data are shown as mean ± standard deviation. *\( p = 0.05 \) vs. trauma patients with ICP ≤20 mm Hg; †\( p < 0.02 \) vs. control nontrauma patients; **\( p < 0.02 \) vs. trauma patients with ICP ≤20 mm Hg.
There were no significant differences in age, gender, body temperature, blood-gas analysis, or plasma electrolytes concentrations among the three groups of patients.

**DISCUSSION**

This investigation in a small number of patients supports the potential utility and advantage of optic nerve sheath ultrasound for a rapid detection of ICP increases in the head trauma patient. When the ICP was higher than 20 mm Hg, the ONSD was ~7 mm, whereas when the ICP was below 20 mm Hg, the ONSD decreased to ~5.5 mm, a value equal to the control nontrauma patients.

Although CT scan is readily available in most emergency settings, in particular situations, i.e., long-distance transportation or disaster scenes, it cannot be accessible. The process of identification of a possible underlying intracranial hypertension is thereby delayed. In such settings, a rapid and noninvasive method would be useful to determine whether patient’s ICP is elevated or to monitor its changes. Invasive ICP monitoring requires a cranial trephination and, when intraparenchymal probes are used, brain tissue penetration. It is therefore an invasive, cumbersome, and potentially dangerous method, which has to be reserved to severely brain damaged patients in whom an ICP increases is highly probable. Furthermore, monitoring ICP can become a more challenging task in small peripheral hospitals without neurosurgical facilities.

The ocular nerve is part of the central nervous system and in case of raised pressure in the cerebrospinal fluid its sheath inflates. Association between increases in optical nerve sheath diameter and radiologic signs of intracranial hypertension detected by CT scan has been previously confirmed. When needed, ocular ultrasound therefore can be rapidly performed at the patient’s bedside and help to identify conditions of normal and high ICPs or rapid changes in ICP. In our study, the instances of stably high ICP and rapid raises of ICP were accompanied by increases in the optical nerve sheath diameter, assessed by ultrasound, from ~5.5 mm to 7 mm. A cutoff value of sheath diameter, however, remains to be identified to allow for a more accurate diagnosis.

Based on the literature and on our own experience, the learning curve of optic nerve sheath ultrasound skill acquisition is rapid and does not represent a limit for the introduction of this method in the clinical practice. Beside the rapidity and simplicity of the management of this ultrasound examination, the technique has the great advantages of being easily repeatable and noninvasive. These aspects might be of additional importance for the care of unstable patients who cannot be mobilized to be subjected to CT scan or cannot have an intracranial probe implanted because of coagulation alterations. However, conditions such as ocular trauma, optic nerve injury, and neuritis have to be mentioned as a limit in the applicability of this approach.

In conclusion, cranial CT scan followed by insertion of an intraparenchymal probe remains the gold standard for ICP monitoring in head trauma injured patients. Nevertheless, in many circumstances, ocular ultrasound may be considered as a good alternative for a rapid indirect evaluation of head trauma patients’ ICP. Accordingly, the capability of this approach to be used for triage and for assessment of effectiveness of treatment of intracranial hypertension is anticipated.

**REFERENCES**


