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Case Report

Neuromonitoring Severe Traumatic Brain Injury by Transcranial Doppler and Diameter of the Optic Nerve Sheath. Case Report

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Abstract

In patients with severe traumatic brain injury (TBI) initial neurological evaluation and follow-up are difficult, especially in the context of multiple trauma.

Multimodal neuromonitoring is essential in the management of traumatic brain injury as it allows early individualized therapeutic behavior that prevents secondary brain injury.

N. Martin; D. Kelly and colleagues describe the importance of changes in cerebral blood flow (CBF) following traumatic brain injury and define three hemodynamic phases during the first two weeks after trauma. Phases are characterized by transcranial Doppler (TCD) measurements, jugular venous oxygen saturation (SJVO2) through catheterization of the jugular bulb, and cerebral perfusion pressure.

Phase I (hypoperfusion phase) occurs on the day of injury; Phase II (hyperemia phase, days 1-3) and Phase III (vasospasm phase, days 4-14).

We present a case of multiple trauma associated with severe TBI. The objective is to discuss the use of neurological monitoring after the limitation of invasive intracranial pressure (ICP) monitoring.

Keywords: Severe Traumatic Brain Injury; Cerebral Blood Flow; Intracranial Hypertension; Transcranial Doppler; Optic Nerve Sheath

Abbreviations

FAST: Focused Assessment with Sonography in Trauma; RASS: Richmond Agitation-Sedation Scale; APACHE: Acute Physiology and Chronic Health disease Classification System; SOFA: Sequential Organ Failure Assessment

Introduction

In patients with severe traumatic brain injury (TBI) initial neurological evaluation and its follow-up are particularly difficult, especially in the setting of multiple trauma. In early management, neuromonitoring is essential to know the underlining pathophysi-

ological evolution, which allows for early therapeutic measures. Martin., *et al*. [1] and Kelly., *et al*. [2], describe the importance of the changes in cerebral blood flow (CBF) after severe traumatic brain injury and classify them into three phases described by transcranial doppler (TCD), cerebral perfusion pressure (CPP) and oximetry in the jugular bulb: initial phase (day 0) of hypoperfusion, phase II (days 1-3) of hyperemia and phase III (days 4-14) of vasospasm. Based on the presentation of a patient who suffered multiple trauma associated with severe TBI, the continuous evaluation after interrupting intracranial pressure (ICP) monitoring will be discussed.

Objective

Present the case of a 21-year-old patient with polytrauma and severe TBI, whose evolution could not be followed nor monitored with ICP monitoring.

Methods

Indirect determination of CBF by Flow Velocities with DTC Rimed® 2Mhz equipment pulsed with M mode. Both middle cerebral arteries were explored through the temporal window and the basilar artery through the transforaminal window. Measurement of the optic nerve sheath diameter (ONSD) was done by Sonoacer 3® ultrasound with a 5-12 MHz linear soft tissue transducer. The optic nerve sheath (ONS) is measured 3 mm behind the retina, at the retro bulbar level, here a transverse line is drawn from edge to edge of the ONS, this area has been described as the point where maximum ONSD occurs due to the increase in ICP. Three measurements were made on both eyes, and then the values were averaged. The cut-off point estimated in all studies is 5.4 mm or more, which correlates with ICP values greater than 20 mmHg (Figure 1).

Figure 1: Optic nerve sheath measurement technique.

The same qualified operator performed the procedures, both for TCD and for ONSD determination.

Case Report

A 21-year-old female patient with no medical history of relevance was admitted to the first institution for multiple trauma with severe TBI as a consequence of a car crash. She had a fracture of the orbit and left wrist, fracture of the femur and exposed fracture of the tibia and fibula Gustillo IIIb of the right lower limb, fracture of the medial malleolus of the left lower limb, and contusion of the right pulmonary field. The evaluation was performed in the emergency department, the Glasgow Coma Scale (GSC) score was 4/15, and airway protection was done by orotracheal intubation together with computed tomography (CT) of the brain without contrast, a depressed fracture of the left frontal bone and the orbit was found. FAST ultrasound was negative for abdominal bleeding. After evaluation, the traumatology department decided to place an external tutor in the lower limb.

She was referred to our institution where a new brain CT was performed, which revealed right frontal and temporal contusion as well as subarachnoid hemorrhage in the left temporoparietal cortex. An Intracranial Pressure Catheter (PIC) was placed and she entered therapy under sedoanalgesia, GCS 3/15, RASS -5, non-reactive pupils with miosis, APACHE Score: 20 points, and SOFA: 7 points.

The patient progressed with GCS 8/15 (E: 3 V: 1 M: 4), under sedoanalgesia with propofol and morphine. The ICP catheter was removed 6 hours after its placement by the Neurosurgery department, which for technical reasons did not place it again.

Given the inability to continue ICP monitoring in the context of multiple trauma with TBI, TCD and ONSD monitoring was used.

On the 6th day post-TBI, the first evaluation with TCD was performed, which showed increased mean velocities (MV) with normal pulsatility index (PI), a pattern compatible with cerebral reperfusion (Phase 2) (Table 1 and Figure 2).

Measurements on days (7-8-9) showed a similar symmetrical increase in MV with TCD.

	Right		Left	
Arteries	**Mean velocity**	**PI**	**Mean velocity**	**PI**
Middle cerebral	127	0,81	124	0,72
Anterior cerebral	71	0,86	99	0,4
Vertebral	57	0,81	76	0,6
Basilar	Mean velocity: 63		PI: 0,84	

Table 1: First TCD on the 6th day post TBI.

Figure 2: One TCD sample from the 6th-day post-TBI.

This sustained increase in MV was accompanied by changes to the ONSD (Table 2).

	Right eye	Left eye
ONSD (DIA6)	5,2	6
ONSD (DIA8)	6	5,8

Table 2: ONSD days 6-8-10.

On day 10, the TCD evaluation showed a decrease in MV, again accompanied by changes in ONSD (Table 3 and Figure 3).

	Right		Left	
Arteries	**Mean velocity**	**PI**	**Mean velocity**	**PI**
Middle cerebral	83	0,9	85	0,85
Anterior cerebral	52	0,77	68	0,79
Vertebral	47	0,87	47	0,77
Basilar	Mean velocity: 51		PI: 0,84	
ONSD (DAY10)	4		4	

Table 3: Mean velocity and pulsatility index on TCD and ONSD at the 10th day.

Figure 3: One TCD sample from the 10th-day post-TBI.

These findings were compatible with the cessation of reperfusion and a decrease in ICP. The interruption of sedation with propofol was done and changes were monitored with TCD and MV (Table 4).

	RIGHT		LEFT	
Arteries	**Mean velocity**	**PI**	**Mean velocity**	**PI**
Middle cerebral	105	0,94	97	0,68
Anterior cerebral	64	0,66	66	0,69
Vertebral	50	0,74	54	0,78
Basilar	Mean velocity: 59		PI: 0,86	

Table 4: Mean Velocity and, PI and TCD with the suspension of sedation.

These results were compatible with the recovery of brain autoregulation by the patient.

In the second week of hospitalization, improvement was noticed with a GCS score of 11/15 (E: 4 V: 1 M: 6) and right hemiparesis. TCDs with speeds within normal parameters expected for the evolutionary time of TBI were found.

Discussion and Conclusion

The use of TCD allows for diagnostic evaluation of the different phases of neurological damage in an estimated non-invasive way and without complications for the patient, which makes it a practical tool for the emergency department and intensive care unit. Performing early TCD along with other diagnostic studies allows

for more accurate predictions of the clinical course and can help avoid unnecessary studies or maneuvers.

Due to the referral from another institution, it was not possible to perform TCD measurements on the first day after TBI and no evidence of the initial hypoperfusion phase was found, which would have allowed us to estimate not only the severity of the patient but also the risk of developing intracranial hypertension (IHT) and the result on discharge from ICU [3]. Interindividual heterogenicity could influence the evolution of the phases by extending or shortening them; in this case prolongation of the reperfusion phase was seen as an increase in mean velocities towards day 9.

ICP measurement was not performed due to the removal of the ICP catheter at 6 hours of hospitalization, and the ONSD was then chosen as a way to monitor IHT. Considering the association between TBI and IHT and its probable consequence. Secondary brain injury, mainly arterial hypotension and ischemic cerebral hypoxia may require adjustments in the management of sedoanalgesia [4]. In this case when elevated MV and ONSD values were observed sedoanalgesia was put in place until decreasing values were obtained.

ONSD has a sensitivity and specificity for the detection of IHT of 88% and 93% respectively. A cut-off point of 5 mm or more correlates with ICP values greater than 20 mmHg [5,6]. Hansen and Helmke [7] carried out the first studies measuring ONSD, which showed that the increase in intracranial pressure in cadavers was reflected in an increase in ONSD by more than 50%. Other studies have shown that the measurement of ONSD for the detection of IHT with a standardized cut-off point of 5 mm can achieve a sensitivity and specificity of 80 to 95% and 80 to 100%, respectively [8]. The meta-analysis by Dobourg., *et al.* [9] included six studies with statistically similar characteristics; two of them included patients with non-traumatic hemorrhage and hemorrhage secondary to trauma; analyzing a total of 231 patients. This study shows the correlation between ONSD and IHT, using ICP measurement with an intraventricular catheter as control and the gold standard; inferring that a cut-off point of the ONSD above 5.4 mm presents a good correlation with an ICP above 20 mmHg (S 90%, E 85%).

The monitoring and follow-up of IHT in a patient with TBI with CT are at least controversial for some authors [10]. Since the clinical course of TBI is very dynamic, serial CT could only demonstrate or confirm gross changes that are usually clinically evident and that may lead to active clinical and surgical behavior. Therefore, its routine use in critically ill patients should be evaluated on individual bases with the results of physical examination and complementary studies. Furthermore, the cost-effectiveness of the procedure and the risk that it may bring should always be considered.

Indirect measurement of blood flow through TCD can estimate the clinical course of a patient with severe TBI. It is a non-invasive and reliable practice that allows close monitoring of neuronal tissue damage and recovery stages. The importance of having this study in the initial evaluation upon arrival of the patient is reinforced, as it allows for close monitoring of the damage induced by trauma.

Ethical Aspects

By the principles in the Declaration of Helsinki of the WMA (World Medical Association), this case report is developed by the principles of non-maleficence and confidentiality, since the identification data of the patient to whom the report corresponds are not disclosed.

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