The association between intracranial pressure and optic nerve sheath diameter on patients with head trauma

Associação entre a pressão intracraniana e o diâmetro da bainha do nervo óptico em pacientes com traumatismo cranioencefálico

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ABSTRACT

Background: Although intracranial pressure (ICP) monitoring is the gold standard method for measuring intracranial pressure after traumatic brain injury, optic nerve sheath diameter (ONSD) measurement with ultrasound (US) is also used in the evaluation of ICP. Objective: To investigate the association between a series of ONSD measurements by US and changes in clinical presentation of the patient. Methods: Prospective study including 162 patients with traumatic brain injury. Age, sex, cerebral CT findings, ONSD levels by US at minutes 0, 60, and 120, Glasgow Coma Scale (GCS) within same period, change of consciousness, treatment, and mortality data were reviewed. The association of ONSD levels with GCS, change of consciousness, treatment, and mortality was evaluated. Results: There was no difference in ONSD changes in the patients’ sample within the period (p=0.326). ONSD significantly increased in patients who died (p<0.001), but not in those who survived (p=0.938). There was no significant change in ONSD of the patients who received anti-edema therapy (p=0.801), but significantly increased ONSD values were found in those who received anti-edema therapy (p=0.03). Patients without change of consciousness did not have any significant change in ONSD (p=0.672), but ONSD values increased in patients who consciousness became worse, and decreased in those who presented a recovery (respectively, p<0.001, p=0.002). A negative correlation was detected between ONSD values and GSC values measured at primary, secondary, and tertiary time periods (for all p<0.001). Conclusions: ONSD follow-up may be useful to monitor ICP increase in patients with acute traumatic brain injury.

Keywords: Brain Injuries, Traumatic; Optic Nerve Neoplasms; Glasgow Coma Scale; Mortality.

RESUMO

Antecedentes: Embora o monitoramento da pressão intracraniana (PIC) seja o método padrão-ouro para medir a pressão intracraniana após lesão encefálica traumática, a medição do diâmetro da bainha do nervo óptico (DBNO) com ultrassom (US) também é usada na avaliação da PIC. Objetivo: Investigar a associação entre uma série de medidas de DBNO por US e mudanças na apresentação clínica do paciente. Métodos: Estudo prospectivo incluindo 162 pacientes com traumatismo cranioencefálico. Idade, sexo, achados de TC cerebral, níveis de DBNO por US nos minutos 0, 60 e 120, Escala de Coma de Glasgow (GCS) no mesmo período, mudança de consciência, tratamento e dados de mortalidade foram revisados. A associação dos níveis de DBNO com GCS, mudança de consciência, tratamento e mortalidade foi avaliada. Resultados: Não houve diferença nas mudanças de DBNO na amostra de pacientes no período (p=0,326). O DBNO aumentou significativamente em pacientes que morreram (p<0,001), mas não naqueles que sobreviveram (p=0,938). Não houve mudança significativa no DBNO dos pacientes que receberam terapia antiedema (p=0,801), mas valores significativamente aumentados de DBNO foram encontrados naqueles que receberam terapia antiedema (p=0,03). Pacientes sem alteração da consciência não tiveram alteração significativa no DBNO (p=0,672), mas os valores do DBNO aumentaram nos pacientes que pioraram a consciência e diminuíram naqueles que apresentaram recuperação (respectivamente, p<0,001, p=0,002). Detectou-se correlação negativa entre os valores de DBNO e os valores de GSC medidos nos períodos primário, secundário e terciário (para todos, p<0,001). Conclusões: O acompanhamento do DBNO pode ser útil para monitorar o aumento da PIC em pacientes com lesão cranioencefálica aguda.

Palavras-chave: Lesões Encefálicas Traumáticas; Neoplasias do Nervo Óptico; Escala de Coma de Glasgow; Mortalidade.

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Conflict of interest: There is no conflict of interest to declare.
Authors' contributions: BED, KÇ: contributed substantially to the conception or design of the study; KÇ: contributed to the collection, analysis, or interpretation of data; BED, KÇ: contributed to the writing or critical review of the manuscript; BED, KÇ: contributed to the approval of the final version to be published.
Received on October 14, 2020; Received in its final form on November 04, 2020; Accepted on November 09, 2020.
INTRODUCTION

Traumatic brain injury (TBI) is a health condition that affects the whole society and especially young adults. TBI is responsible from one third of trauma-related deaths. TBI may develop due to primary effect of the trauma or as a result of secondary effects such as hypoxia, hyperkapnia, hypotension, increase in intracranial pressure, and hyperglycemia. Although it is not possible to avoid primary TBI due to acute effects of the impact, brain damage may be reduced by minimizing metabolic causes and cerebral edema in secondary injuries. The cause for brain edema developed after secondary injury is vasogenic, and this induces cytotoxic edema and causes increase in intracranial pressure (ICP). On the other hand, increased ICP may cause more edema due to decrease in cerebral perfusion. If it is not intervened, it may progress to herniation and death. Therefore, detection and monitoring of ICP has a vital importance.

Although ICP monitoring is the gold standard approach, it is an invasive procedure and often it cannot be performed due to complications and requirement of technical equipment. It has been specified that the optic nerve sheath diameter (ONSD) increases along with the increase of ICP due to the extension of the dura mater surrounding the optic nerve, and the most adequate measurement site is 3 mm distant from the distal side of the ocular globe. Several studies were conducted through brain tomography (CT), magnetic resonance imaging, and ultrasound (US) and consistent results were obtained. A meta-analysis stated that ONSD thickness wider than 5 mm measured by US is an indicator for the increase in ICP, and sensitivity and specificity of such measurement are 99 and 77%, respectively.

The aim of this study was to investigate the association between a series of ONSD measurements by US and changes in clinical presentation of patients.

METHODS

This study was conducted prospectively on 162 adult patients with TBI following approval of the local ethical committee of Medical Faculty within Abant Izzet Baysal University.

Patient selection

One hundred and sixty-two adult patients who were monitored due to TBI between January, 1st, 2020 and June, 1st, 2020 were prospectively reviewed.

Patients with metabolic, orbital, or intracranial pathology that may cause the increase of ONSD, patients <18 years old, patients with indication of urgent surgery who were monitored in the emergency clinic for less than 2 hours, patients with isolated cranial fracture, and those who have rejected to give consent were excluded from the study.

RESULTS

Age, sex, cerebral CT findings, ONSD levels by US at minutes 0, 60, and 120, Glasgow Coma Scale (GCS) within same period, change of consciousness, treatment, and mortality data of patients were reviewed. The association of ONSD levels with GCS scores, change of consciousness, treatment, and mortality was evaluated.

Optic nerve sheath diameter measurement method

The ONSD measurement was performed by a well-trained emergency medicine specialist certificated by the Health Minister of Turkey using a 7.5 MHz linear probe. A thin layer of gel was applied on both eyes of the patient lying in the supine position. ONSD was measured with a Sonosite Plus 180 model linear transducer US machine 3 mm posterior to the eye globe at sagittal and transverse positions. The averages ONSD of transverse and sagittal measurements on the right and left eyes were calculated.

Statistical analysis

Data analysis was performed with the Statistical Package for the Social Sciences (SPSS) version 22 program. Median and interquartile range (IQR) were used to demonstrate quantitative data; qualitative data were expressed in number of cases (n) and percentile (%). The distribution of quantitative data was evaluated by Kolmogorov-Smirnov test. Time-dependent changes in numeric variables were analyzed by Friedman Test. The Wilcoxon test was utilized to detect significant differences. Detection of significance of repetitive measurements and affecting factors was performed through the Greenhouse-Geisser analysis. Mann-Whitney U test was used to analyze the differences in measurement values between two groups. Pearson’s chi-square test was utilized for analysis of categorical variables. Correlation between time-dependent changes of two different numeric variables was analyzed through Spearman’s Correlation test. A p<0.05 was accepted as significant.
patients who survived (p=0.007). There was no significant difference in patients who died at referral even with higher ONSD values (p=0.941). The differences in ONSD between 60 and 120 minutes, 0 and 60 minutes, 60 and 120 minutes, and 0 and 120 minutes were significantly higher in patients who died (respectively p=0.006, p<0.001, p<0.001, p=0.013, p<0.001). There was a significantly higher difference in GCS scores between 0 and 60 minutes, 60 and 120 minutes, and 0 and 120 minutes in patients who died (for all; p<0.05). GCS scores difference between 0 minutes and 60 minutes was similar between patients who died and those who survived (p=0.05) (Table 1).

There was no difference in time-dependent ONSD changes in the patients of our study (p=0.326). It was detected that mortality was associated with change in ONSD (p<0.001). Anti-edema treatment did not significantly affect ONSD change (p=0.05). Change of consciousness was significantly associated with change in ONSD (p<0.001) (Table 2).

There was no difference in ONSD change in the patients of our study within the time period (p=0.102). ONSD significantly increased in patients who died (p<0.001), and there was no change in patients who survived (p=0.938). ONSD did not vary in patients who received anti-edema therapy (p=0.831), but it significantly increased in those who did not receive anti-edema therapy (p<0.03). Patients without change of consciousness did not have a significant change in ONSD (p=0.672); ONSD values increased in patients who

| Table 1. Clinical and demographic characteristics of patients. |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                 | Total (n=162)   | Alive (n=146)   | Dead (n=16)    | p-value         |
| Age, median (IQR) | 34 (45)         | 30 (42)         | 48 (68)        | 0.668           |
| Sex, n (%)       |                 |                 |                | 0.563           |
| Male             | 119 (73.5)      | 106 (72.6)      | 13 (81.3)      |                 |
| Female           | 43 (26.5)       | 40 (27.4)       | 3 (18.8)       |                 |
| Off-vehicle traffic accident | 26 (16)     | 23 (15.8)       | 3 (18.8)       |                 |
| IVTA             | 72 (44.4)       | 67 (46.9)       | 5 (31.3)       |                 |
| Mechanism of trauma, n (%) | 5 (3.1) | 0 (0)           | 5 (31.3)       | <0.001          |
| Firearm injury   | 28 (17.3)       | 27 (18.5)       | 1 (6.3)        |                 |
| Pounding         | 31 (19.1)       | 29 (19.9)       | 2 (12.5)       |                 |
| High fall        |                 |                 |                |                 |
| CT findings, n (%) |                 |                 |                |                 |
| Bone fracture    | 45 (27.8)       | 36 (24.7)       | 9 (56.3)       | 0.015           |
| Epidural hematoma | 34 (21)     | 24 (16.4)       | 10 (62.5)      | <0.001          |
| Subdural hematoma | 48 (29.6)    | 44 (30.1)       | 4 (25)         | 0.780           |
| Subarachnoid hemorrhage | 94 (58) | 84 (57.5)       | 10 (62.5)      | 0.794           |
| Contusion        | 38 (23.5)       | 33 (22.6)       | 5 (31.3)       | 0.534           |
| Cerebral edema   | 24 (14.8)       | 20 (13.7)       | 4 (25)         | 0.262           |
| Change of consciousness, n (%) | 23 (14.2) | 13 (8.9)        | 10 (62.5)      | <0.001          |
| None             | 128 (79)        | 122 (83.6)      | 6 (37.5)       |                 |
| Negative         | 11 (6.8)        | 11 (7.5)        | 0 (0)          |                 |
| Positive         | 114 (70.4)      | 108 (74)        | 6 (37.5)       | 0.007           |
| Anti-edema therapy, n (%) | 48 (29.6) | 38 (26)         | 10 (62.5)      |                 |
| Present          |                 |                 |                |                 |
| None             |                 |                 |                |                 |
| Minute 0         | 6.60 (1.2)      | 6.40 (0.6)      | 6.60 (1.2)     | 0.941           |
| Minute 60        | 6.50 (1.2)      | 6.50 (1.2)      | 6.70 (0.2)     | 0.006           |
| Minute 120       | 6.50 (1.2)      | 6.45 (1.2)      | 6.7 (0.2)      | <0.001          |
| Difference between minutes 0 and 60 | 0 (0)       | 0 (0)           | 0.2 (0)        | <0.001          |
| Difference between 60 and 120 | 0 (0.10) | 0 (0.1)         | 0 (0.3)        | 0.013           |
| Difference between minutes 0 and 120 | 0 (0.2)    | 0 (0.2)         | 0.4 (0.85)     | <0.001          |
| GCS, median (IQR) |                 |                 |                |                 |
| Minute 0         | 9 (1)           | 9 (1)           | 5.5 (3)        | <0.001          |
| Minute 60        | 9 (1)           | 9 (1)           | 5.3 (8)        | <0.001          |
| Minute 120       | 9 (1)           | 9 (1)           | 4.5 (2.75)     | <0.001          |
| Difference between minutes 0 and 60 | 0 (0)       | 0 (0)           | 0 (0)          | 0.050           |
| Difference between minutes 60 and 120 | 0 (0)     | 0 (0)           | -1 (2)         | <0.001          |
| Difference between minutes 0 and 120 | 0 (0)      | 0 (0)           | -1.5 (2.75)    | <0.001          |

IQR: interquartile range; ONSD: optic nerve sheath diameter; GCS: Glasgow Coma Scale.
consciousness became worse, and decreased in those who presented recovery (respectively; p<0.001, p=0.002) (Table 3). A negative correlation was detected between ONSD values and GSC values measured at primary, secondary, and tertiary time periods (for all p<0.001). Furthermore, a negative correlation was detected between ONSD values differences and GCS scores differences measured within the same time periods (for all p<0.001) (Table 4).

**DISCUSSION**

Non-invasive ICP measurement techniques have been sought to replace the invasive measurement, which is accepted as the gold standard, but causes complications (infection, hematoma, etc.) in 5% of cases, cannot be applied in many centers, and cannot be used in bleeding disorders and extremely high brain pressures⁵. Fortunately,

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**Table 2.** Optic nerve sheath diameter change within the study period.

| Type III sum of squares | df | Mean square | F     | p-value |
|-------------------------|----|-------------|-------|---------|
| Totally                 | 0.069 | 1.500 | 0.046 | 1.078 | 0.326 |
| Mortality               | 2.033 | 1.633 | 1.245 | 39.037 | <0.001 |
| Anti-edema therapy      | 0.212 | 1.498 | 0.142 | 3.348 | 0.050 |
| Change of consciousness | 2.316 | 3.288 | 0.704 | 22.877 | <0.001 |

**Table 3.** Optic nerve sheath diameter change and differences within the period 0, 60, and 120 minutes.

|                | 0 minute Median (IQR) | 60 minutes Median (IQR) | 120 minutes Median (IQR) | p-value |
|----------------|-----------------------|-------------------------|--------------------------|---------|
| Totally        | 6.6 (1.2)             | 6.5 (1.2)               | 6.5 (1.2)                | 0.102   |
| Mortality      | Alive                 | 6.6 (1.2)               | 6.5 (1.2)               | 6.45 (1.2) | 0.938   |
|                | Dead                  | 6.4 (0.6)               | 6.7 (0.2)               | 6.7 (0.2) | <0.001abc |
| Anti-edema therapy | Receiving        | 6.55 (1.2)             | 6.5 (1.2)               | 6.5 (1.2) | 0.801   |
|                | Not receiving         | 6.55 (1.2)             | 6.6 (1.3)               | 6.6 (1.2) | 0.003abc |
| Change of consciousness | None             | 6.6 (1.2)             | 6.5 (1.2)               | 6.5 (1.2) | 0.672   |
|                | Negative              | 6.2 (1.2)             | 6.7 (1.0)               | 6.7 (0.4) | <0.001abc |
|                | Positive              | 6.5 (1.5)             | 6.2 (1.5)               | 5.9 (1.5) | 0.002abc |

*p<0.05 for changes between 0 and 60 minutes; †p<0.05 for changes between 60 and 120 minutes; ‡p<0.05 for changes between 0 and 120 minutes.

**Table 4.** Association between Glasgow Coma Scale and optic nerve sheath diameter.

| GCS                      | Minute 0 | Minute 60 | Minute 120 | The difference between minutes 0 and 60 | The difference between minutes 60 and 120 | The difference between minutes 0 and 120 |
|--------------------------|----------|-----------|------------|----------------------------------------|------------------------------------------|----------------------------------------|
| **ONSDF**                | r        | p         | r          | p                                      | r                                        | p                                      |
| Minute 0                 | -0.231   | 0.003     | -0.378     | <0.001                                 | -0.485                                   | <0.001                                 |
| Minute 60                | r        | p         | -0.269     | 0.001                                   | -0.303                                   | <0.001                                 |
| Minute 120               | r        | p         | -0.498     | <0.001                                 |                                          |                                        |

GCS: Glasgow Coma Scale; ONSD: optic nerve sheath diameter.
it has been shown to be consistent with measurements made by US²⁸.

The elevation of ONSD due to the increase of ICP in patients with TBI was shown in several studies¹¹,¹²,¹³,¹⁴. Although the importance of urgent intervention for ICP increase, there is no method developed for ICP monitoring in the emergency clinic including the acute trauma period.

Many studies have shown that adult men are more exposed to trauma²⁵,²⁶,²⁷,²⁸. Mortality prevalence was reported to be independent from gender in many studies, but there are conflicting findings on the association between the age and mortality¹⁵,¹⁹,²⁰. Unlu et al. reported that mortality is not dependent on age¹⁵; Kara et al. stated that trauma in elderly has high mortality²⁵; and Adıyaman et al. found that age is a significant factor for mortality in the long-term¹⁹. In line with the literature, young males were more frequent in our study. There was no significant association of mortality with age and gender. Since adult males usually drive more and are more involved in social life as well as violence episodes such as fights and firearm injury, we believe that they are more exposed to trauma than women. Although age is associated with increased mortality due to natural increased catabolism and co-morbidities, we believe that age and sex contribute to the association because the main factor determining mortality is trauma severity, and men are more exposed to traumatic events than women.

Many studies show that TBI is commonly caused by traffic accidents²¹,²². Motorized vehicle accidents are responsible for the majority of TBI-associated deaths among young adults²². Dur et al. stated that mortality due to high falls and traffic accidents are the most common in trauma patients admitted to the intensive care unit; however, no statistical significance was reported²⁴. Kara et al. reported no association between mortality and type of trauma²⁰. Although the most common form of injury was traffic accident in line with the literature, TBI was detected more in penetrating injuries. This may be related to the higher damage on the brain tissue in firearm injury cases.

Previous studies stated that the most common TBI cases are subarachnoid hemorrhage (SAH) and subdural hematoma²⁵,²⁶. Siwicka-Gieroba et al. reported that patients with TBI developed SAH and epidural and subdural hematoma; however, deaths commonly occurred due to intracranial hemorrhage²⁷. A study conducted on patients with TBI specified that death cases have higher hemorrhage and shifts and pressure on basal cisterns²⁸. In this study, the most common TBI was SAH and subdural hematoma; TBI leading to death was more common in patients with epidural hemorrhage and bone fracture. Depending on the severity of the trauma, the risk of rupture of venous formations can be higher, thus increasing the death rate in SAH and subdural hemorrhage. However, we believe that the mortality rate increased due to trauma and bone fracture severity and the interruption of the bone integrity in firearm injuries followed by the damage to the brain tissue by bone fragments. Furthermore, epidural hemorrhage might progress to death because of arterial origin and concomitant bone fractures indicating the high energy trauma.

Previous studies stated that ONSD is over 5 mm when ICP is over 20 mmHg; ONSD of patients with TBI elevated up to 6.3 to 6.4 mm²⁹,³⁰,³¹. Moreover, ONSD is higher in patients with lower GCS, higher ICP, and in those who die from the injury²⁷,²⁹,³²,³³. A previous study conducted on patients with TBI reported that death cases have lower GCS and higher ONSD²⁵. Sekhon et al. indicated in their study that every increase of 1 mm in ONSD doubles the risk of death. In line with the literature, we also detected that death cases had lower GCS and higher ONSD.

Along with primary TBI, secondary injuries may cause cerebral edema, increase in ICP, and deterioration of the clinical presentation²⁵,³⁴,³⁵. Studies reported that ONSD is an independent factor for morbidity and mortality, and monitoring of ICP and performance of required interventions are associated with lower rates of mortality³⁶,³⁷. Thotakura et al. performed ONSD measurement on adults with head trauma in an interval of 24 to 48 hours and reported that patients with a decreasing trend in ONSD values presented a good clinical progress, and none of them were treated surgically³⁸. In the present study, patients who died had lower GCS, worse clinical progress, and elevated ONSD values. ONSD significantly increased in patients who died and/or presented worsening clinical progress. There was no significant change in ONSD values of survivors. We believe that ICP increased within a short period due to primary or secondary causes, and the ONSD increased accordingly. However, we consider that edema was not resolved in survivors within 2 hours, and the treatment would reduce ONSD within a wider period of time. This indicates that patients with severe ONSD elevation have higher risk of death, and this may guide the physician for necessary adjustments in the treatment plan.

There are medical and surgical decompression approaches for cases with ICP increase. Although hypertonic saline and mannitol are therapies to reduce ICP, they may constitute a risk in some cases. However, cases receiving anti-edema therapy needed a longer period for treatment response²⁹,³⁹. There was no change in ONSD in patients who received anti-edema therapy, and an increase of ONSD was detected in patients who were not treated, probably due to the severity of the TBI as well as the increase of ICP due to secondary causes.

Our study had some limitations; first, vital parameters of the patients were not recorded, and their effects on ICP was not investigated. Second, evaluation of a new lesion (i.e., shift, hemorrhage) and comparison with any former lesion were not performed in control brain CT scan of the patients. Patients who underwent surgical intervention for acute brain injury were not observed within 2 hours; therefore,
these patients were excluded from the study, preventing the evaluation of the effect of surgical decompression on ONSD. Subgroups (type of anti-edema medication, ventilation rate, elevation status of the head) of the anti-edema therapy and the effect of the treatment were not investigated.

Consequently, ONSD follow-up may be used to monitor ICP increase of the patients with acute TBI. We believe that repetitive ONSD measurements would be useful to determine possible effects of secondary damage and trauma severity during patient treatment.

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