https://doi.org/10.17116/engneiro201781670-76

Correlation of Intracranial Pressure and Diameter of the Sheath of the Optic Nerve by Computed Tomography in Severe Traumatic Brain Injury

A.M. TURKIN[1*], A.V. OSHOROV[1], E.L. POGOSBEKYAN[1], A.S. SMIRNOV[1], A.S. DMITRIEVA[2]

[1]Burdenko Neurosurgical Institute, Moscow, Russia, 125047; [2]Evdokimov Moscow State University of Medicine and Dentistry, Moscow, Russia, 127473

Background. Noninvasive techniques to evaluate intracranial pressure (ICP) are important for everyday practice in intensive care and neurosurgery departments. CT data can be used to evaluate the optic nerve sheath diameter (ONSD) and, indirectly, the ICP value. The ONSD value is an additional criterion in deciding on invasive monitoring of ICP.
Objective. To analyze a correlation between CT-based ONSD and the results of invasive measurements of ICP in patients with severe traumatic brain injury.
Material and methods. The study evaluated 41 patients with severe traumatic brain injury within the first 48 h after injury. Invasive monitoring of ICP (Codman & Shurtlett, MA, USA) was performed during 7±1.7 days. ONSD was measured using axial CT scans (CereTom, Neurologica Danvers, MA, USA) with a slice thickness of 2.5 mm. The ONSD value was measured at a distance of 3 mm from the posterior eyeball contour. The patients were allocated in a group with normal ICP (10 patients) and a group with high ICP (31 patients). ONSD served as an ICP classifier. The data were processed using ROC analysis.
Results. According to the CT data, the optimal threshold ONSD value was 6.35 mm in patients in the acute TBI period. The sensitivity was 0.93 (95% CI 0.84—1.00), the specificity was 0.80 (95% CI 0.50—1.00), and AUC was 0.87 (95% CI 0.69—1.00).
Conclusion. We found a correlation between the CT-based ONSD and the median ICP (R=0.32, $p<0.05$). An ONSD value of 6.35 mm and more is one of the signs of previous or existing ICP.

Keywords: optic nerve sheath diameter, computed tomography, traumatic brain injury, intracranial pressure.

Development of intracranial hypertension (ICH), i.e. increase in intracranial pressure (ICP) over 20 mmHg for more than 5 minutes, is the main cause of secondary brain damage and adverse outcomes in patients with severe traumatic brain injury (TBI) [1—3]. Edema of the brain matter that develops as a result of primary traumatic brain damage is one of the leading causes of ICH in patients with TBI, along with such factors as disruption of venous outflow, liquorodynamics, autoregulation of cerebral blood flow, failure of spatial compensation mechanisms, etc. [4—7]. All these mechanisms contribute to the development of secondary ischemic brain damage [4, 7—12].

According to the international recommendations of the Brain Trauma Foundation [3], as well as the recommendations adopted by the Association of Neurosurgeons of the Russian Federation [2], invasive ICP monitoring is indicated only for TBI patients whose condition is assessed as less than 9 points on the Glasgow Coma Scale (GCS) in the presence of pathological changes in the brain based on the data of computed tomography (CT). In the absence of pathological changes at CT, the ICP sensor is implanted when any two criteria are met: age over 40 years, arterial systolic pressure less than 90 mmHg, presence of poznotonic reactions of decortication or decerebration.

Such brain CT data as displacement of the median structures of more than 5 mm, narrowing of the basal cisterns and convexital subarachnoidal fissures, are only indirect criteria of increasing ICP and cannot be used to predict its dynamics. In patients with traumatic brain injury the assessment of changes in ICP is possible only with continuous measurement using an invasive ICP sensor [6].

Modern technological advances led to the development of mobile transport CTs, which greatly facilitates the performance of this diagnostic procedure, making it possible to use it even for non-transportable patients and expanding the options for non-invasive ICP assessment. Along with generally accepted criteria for evaluating the state of the brain matter and intracranial contents, the use of CT in the department of neuroreanimation makes it possible to measure the diameter of the sheath of the intraorbital part of the optic nerve.

Measurement of the optic nerve sheath diameter (ONSD) is a non-invasive method for diagnosis of ICP and can be used as an additional criterion when deciding on the use of the invasive ICP measurement [13, 14]. The term "optic nerve sheath diameter" (ONSD) is commonly accepted and widely used in foreign literature. In this case, the optic nerve sheath is visualized as a cylinder, which can be stretched when the ICP rises, leading to an increase in its diameter. In our work we decided to use the literal Russian translation of the term and the corresponding abbreviation.

Since intraorbital sections of the optic nerves are surrounded by a solid and arachnoid membranes and are communicating with the subarachnoid space of the brain, the increase in ICP is transmitted to the intrathecal space of the optic nerve through increase in liquor pressure,

*e-mail: turkin@nsi.ru

causing it to stretch and increasing ONSD. Many experimental and clinical studies [15—17] have demonstrated that ONSD increases in diameter for a few minutes after an increase in ICP and it reaches its maximum value at ICP 35-45 mmHg.

Measurement of ONSD has become a routine practice in intensive therapy of patients with CNS lesions and polytrauma and is performed with the help of ultrasound [18—20]. However, successful implementation of this examination require adherence to a standardized procedure by a highly qualified specialist.

MRI can also be used for these purpose, but it is usually limited by the severity of the patient's condition, the duration of the examination, the need for anesthesia [7]. It has been shown that the measurements of ONSD using CT and MRI correlate well with each other [21].

The purpose of the study was to calculate the correlation between ONSD and ICP values from invasive monitoring data, and to carry out a statistical analysis of the data obtained.

Materials and Methods

The work is a single center retrospective cohort study. The data of the Burdenko Neurosurgical Institute (FGAU NNPTCN named after NN Burdenko of the Russian Ministry of Health) from a prospectively collected database of patients who sustained severe traumatic brain injury in the period from 2004 to 2013 were used for the analysis.

The inclusion criteria were: the diagnosis of severe TBI at the time of hospitalization (GCS score 8 points or less), age over 16 years, admission to the Institute within the first 2 days after the injury, the availability of CT scan data at the time of admission to the intensive care unit, blood pressure monitoring data, ICP for the duration of stay in the ICU in the specified period of time.

The exclusion criteria were: presence of cranio-orbital trauma, hospitalization after more than 2 days from the moment of the injury, absence of neuromonitoring data, presence of artifacts on CT scan in the projection of the optic nerve, and history of craniotomy and decompressive trepanation, signs of basal liquorrhea.

Based on the presented criteria, 41 patients (11 women and 30 men) with a severe TBI (GCS 8 points or less) were enrolled in the study. The mean age was 30±11 years. All patients have indications for invasive measurement of ICP based on the severity of their condition, clinical-neurological picture and CT data.

Distribution of patients by GCS scores is presented in **Fig. 1**. There were 19 (40%) patients with isolated TBI, and 16 (33%) with closed TBI. All patients underwent CT scan upon admission to ICU (CereTom, Neurologica Danvers MA, USA). Scanning was carried out with a slice thickness of 2.5 mm. All the patients were in the intensive care unit on ALV, intensive therapy was performed in accordance with international recommendations [3]. Parenchymal ICP ("Codman & Shurtlett, MA", USA) was measured in all patients with the average duration of monitoring of 7±1.7 days.

The ICP sensor was installed in the intensive care unit. The sensor was implanted into the white matter of the brain into the premotor zone according to a conventional technique to a depth of 2 cm from the inner bone plate through the trephination hole in the projection of the Kocher point. The implantation side was selected based on the nature of the injury. In case of a diffuse lesion, the sensor was implanted in the subdominant hemisphere; in case of a focal lesion, it was installed to the side of greater damage to the medulla. The sensor was calibrated according to the manufacturer's instructions on the surface of the sterile saline solution at the water-air interface. The values of the main parameters (BP, ICP) were recorded using Software ICM + ("Cambridge", United Kingdom) with a frequency of 100 Hz. The ICP parameters used for further analysis are presented in **the Table**.

The evaluation of ONSD was performed "blindly" by two radiologists (DAS and TAM). ONSD measurements were carried out on a range of 25-300 units. The ONSD value was measured at a distance of 3 mm from the posterior eyeball contour **(Fig. 2)**. The larger of the two ONSD measurements was chosen for further statistical analysis. Only the data of the first CT scan after the hospitalization were analyzed.

Figure 3 shows the dynamics of ICP in a patient with severe TBI, who underwent CT (**see Figure 2**), and met the requirements for invasive ICP monitoring based on clinical-neurological and neuroimaging criteria. Despite the fact that at the time of the installation of the sensor the ICP was at the level of 5—6 mmHg, the patient subsequently had repeated and multiple episodes of ICP increase up to the level exceeding 20 mmHg, which required targeted intensive therapy.

The ICP parameters were recorded for each patient using the Software ICM + ("Cambridge", United Kingdom) and were used for subsequent statistical analysis **(see Table)** as a mean, median and maximum values, as well as standard deviations, the total duration of ICH (ICP>20 mmHg) over the entire monitoring period. Based on the data obtained, correlation and ROC analysis was performed using the R-project software package (www.r-project.org).

All patients were divided in two groups: the first group included 10 patients with normal ICP, and the second group included 31 patients with the development of ICH. ROC-analysis was performed to assess the possibility of using ONSD as the classifier of ICH. Calculated quantitative characteristics of ROC analysis: Area Under Curve (AUC), optimal threshold value and corresponding values of sensitivity and specificity. 95% confidence intervals for AUC, sensitivity, and specificity were calculated using the bootstrap replication method by generating pseudo-sample sets.

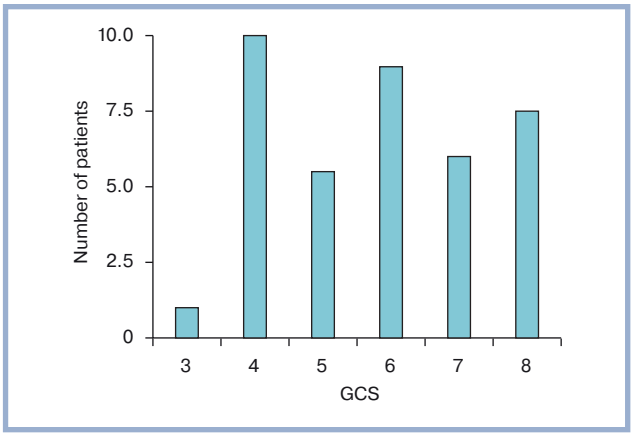

Fig. 1. **Distribution of patients with severe TBI by Glasgow Coma Scale (in points).**

Results

In 31 (76%) out of 41 patients, the total duration of the recorded ICH (ICP>20 mmHg) was more than 1 hour.

Based on the results of the ROC analysis, a threshold value of CT-based ONSD equal to 6.35 mm was established for both groups of patients. This ONSD value corresponds to a point with a maximum total sensitivity of 0.93 (95% CI 0.84—1.00) and specificity of 0.80 (95% CI 0.50-1.00) and AUC of 0.87 (95% CI 0.69—1.00) (**Figure 4**).

The distribution of the optic nerve sheath diameter was considered normal, as values of $p>0.05$ were obtained in the Shapiro-Wilk tests. The Pearson correlation coefficient between the diameters of the sheath of the left and right optic nerves is 0.78 ($p<0.05$, 95% CI 0.62—0.88) **(Fig. 5)**. Such high correlation indicates narrow spread of the ONSD of the left and right eye in each patient under study.

Nonparametric Spearman correlation between ONSD and mean ICP is 0.32 (95% CI 0.05—0.63, $p<0.05$).

Spearman correlation between ONSD and median ICP is 0.32 (95% CI 0.02—0.60, $p<0.05$). Since for most patients in ICH group (23 of 31), the distribution of ICP during the monitoring period had abnormal at equal correlation coefficients $r=0.32$ for mean ICP, the median ICP was used for further analysis.

Discussion

The first studies of ICP dynamics were conducted by Lundberg in 1960. He was the first to perform continuous measurement of cerebrospinal fluid pressure in the ventricles of the brain. Later, various methods of invasive ICP measurement were developed and introduced into practice: in the subarachnoid and subdural spaces, in the parenchyma of the brain. Each of these methods has its own advantages and disadvantages [22, 23].

The installation of an intracranial pressure sensor of any type is a surgical intervention and is associated with the development of hemorrhagic and infectious complications, as well as the problem of the ICP "zero value" drift [24]. There are a number of non-invasive techniques that allow one to indirectly assess the presence of ICH: quantitative papillometry, ophthalmodinamometry, assessment of the swelling of the tympanic membrane, measurement of ONSD (by different methods), transcranial dopplerography, MRI, CT [25].

According to the literature [26], the accurate measurement of ONSD can be performed using ultrasound or MRI, and the values obtained by these methods are in good agreement with each other. The data obtained by MRI and CT are also well correlated [21]. In the case of severe TBI, the primary diagnostic method is CT, which, in contrast to ultrasound, is an operator-independent method of examination.

Examined ICP parameters

Parameter	All patients	Patients without ICH	Patients with ICH
Mean age, years (standard deviation)	41	10	31
Number of men, %	32 (12)	32 (16)	32 (11)
Number of women, %	30 (73)	8 (80)	22 (71)
GCS, median (25—75 quantiles)	11 (27)	2 (20)	9 (29)
GOS, median (25—75 quantiles)	6 (4—7)	7 (5—7)	6 (4—7)
Marshall scale, median (25—75 quantiles)	3 (3—4)	3 (3—4)	3 (3—4)
Average ONSD on the left, mm (deviation)	2 (2—3)	2 (2—2)	2 (2—4)
Average ONSD on the right, mm (deviation)	6.06 (0.6)	6.06 (0.71)	6.77 (0.46)
Average ONSD of the largest optic nerve, mm	6.52 (0.75)	5.75 (0.62)	6.77 (0.62)
(standard deviation)	6.73 (0.66)	6.08 (0.71)	6.94 (0.49)
Number of lethal outcomes, %	6 (15)	0 (0)	6 (19)
Number of adverse outcomes, %	22 (54)	5 (50)	17 (55)
Median ICP (standard deviation)	16.45 (6.11)	10.95 (4.18)	18.22 (5.59)
Mean ICP (standard deviation)	12.26 (4.86)	8.1 (5.00)	13.61 (4.03)
Duration of ICP >20 mmHg (standard deviation)	16.84 (32.93)	3.02 (6.37)	21.30 (36.73)

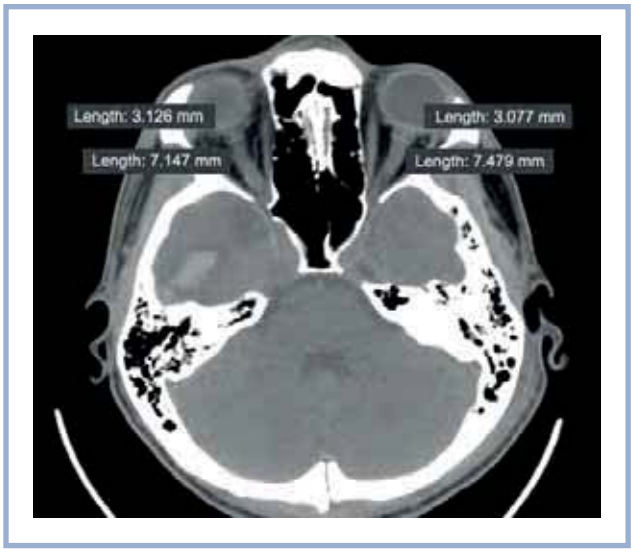

Fig. 2. **CT of the patient with severe TBI and intracranial hypertension. ONSD on the left, 7.48 mm, on the right, 7.15 mm. Intracerebral hematoma in the basal parts of the right temporal lobe.**

The increase in ONSD with an increase in ICP is associated with the peculiarities of the optic nerve structure. An increase in ICP alongside with the depletion of the mechanisms of spatial compensation, causes redistribution of CSF from the intracranial to extracranial spaces, which is accompanied by an expansion of the optic nerve sheaths and an increase in ONSD. These changes are most pronounced in the distal third of the optic nerve, closer to the eyeball [15]. It has been shown experimentally that the most pliant part of the optic nerve sheath is located in the region of its ampullar part, therefore it is generally accepted practice to evaluate ONSD at a distance of 3 mm from the posterior wall of the eyeball [16]. Normal CT-based values of ONSD in this area were calculated in a study of 300 patients without clinical and radiological signs of ICH. According to the results of the study, ONSD values were in the range from 4.94±1.51 to 5.17±1.34 mm [27].

Measurements of ONSD using of MRI showed that at a diameter of less than 5.3 mm the development of ICH is unlikely, whereas with a ONSD of more than 5.82 mm, the probability of ICH development is 90% [20].

M. Sekhon et al. [14] identified a strong correlation between the ONSD and ICP values in severe acude TBI. Using the threshold value of 6 mm, the authors obtained an area under the curve AUC=0.83 (95% CI 0.73-0.94) with a true positive value in 67% of cases, false-positive in 92%. The authors concluded that the CT-based measurement of ONSD is more accurate criterion for development of ICH (R^2=0.56) compared to such CT signs (R^2=0.21) as compression of the lateral ventricles, smoothing of the border of white and gray matter, lateral displacement of more than 5 mm, and compression of basal cisterns. They also showed that nosocomial mortality doubles with an increase in ONSD of 1 mm (ratio of probability of occurrence and non-occurrence of the event, odds ratio (OR) 2.0, 95% CI 1.2—3.2, p=0.007). Our results on the correlation between ONSD and ICP are fairly close to the data of M. Sekhon et al. [14], since the studies were conducted on similar groups of patients in an early period after the injury and were compared with the analysis of invasive ICP measurement.

Despite the results obtained, which are consistent with the data of other authors, we want to emphasize a

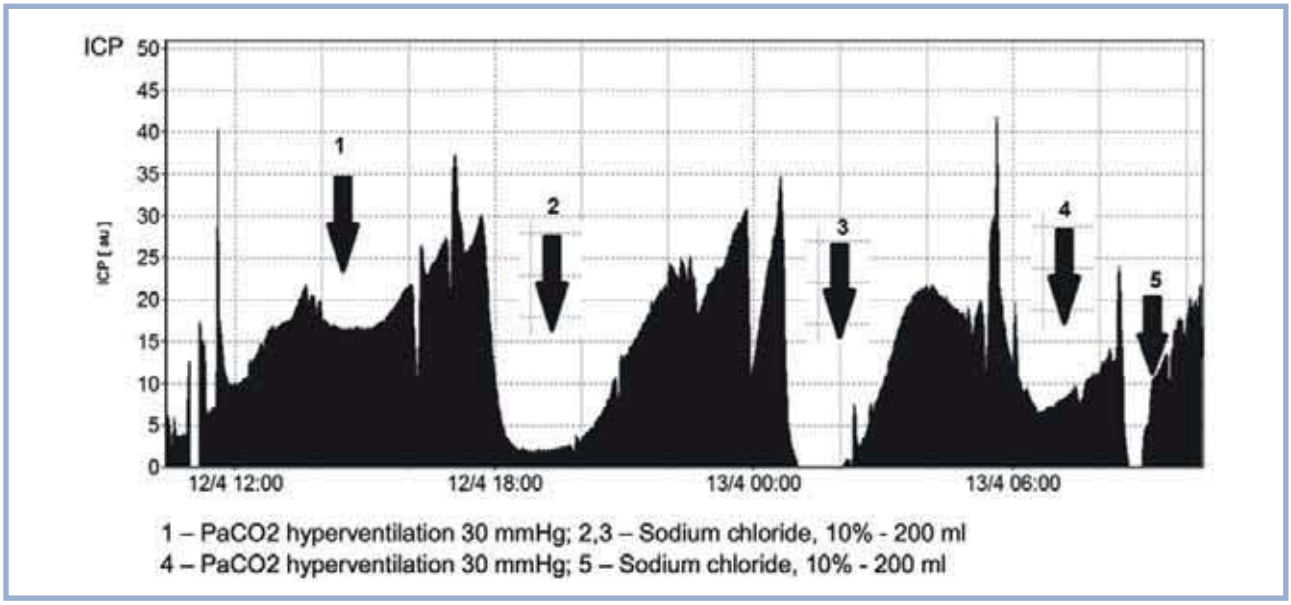

Fig. 3. **Dynamics of ICP in the patient after a CT scan (see Figure 2) and deciding on invasive monitoring of ICP (data for the first 24 hours are presented).**

ICP at the moment of the sensor installation was 5—6 mmHg.

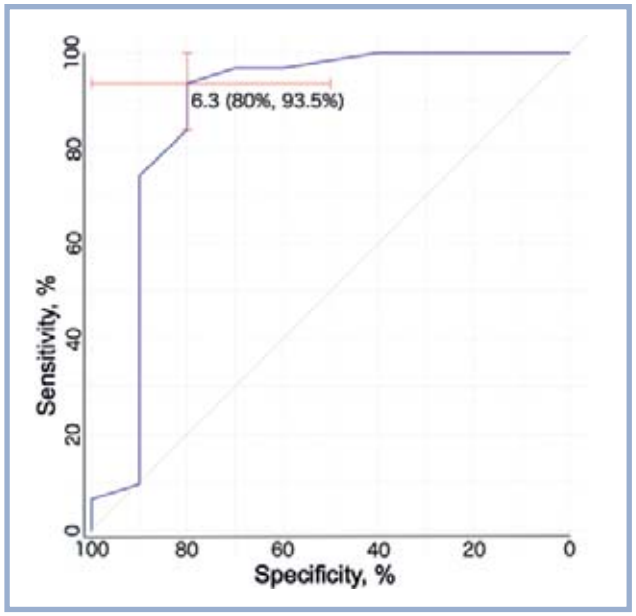

Fig. 4. **ROC-analysis curve for ONSD as a criterion for the development of intracranial hypertension (ICP above 20 mmHg).**

The area under the curve reflects the diagnostic accuracy of the ONSD measurement in identifying ICP above 20 mmHg.

number of important points that should be taken into account in clinical practice.

First of all, CT is a screening diagnostic method that provides information on the state of the injured brain and adjacent tissues at a particular time point.

Secondly, we analyzed a limited number of patients (41 patients) who were admitted to the Institute within the first 48 hours from the moment of the injury. According to the literature [28], this period is associated with the maximum risk of ICH development due to traumatic brain damage and formation of cerebral edema. Therefore, we did not rule out a possibility that some of the patients could have undergone ICH prior to hospitalization and the beginning of invasive ICP monitoring. We would like to remind that all patients had severe TBI and were in coma, which indicates the severity of the primary brain damage and, accordingly, the high probability of ICH. Therefore, some patients could have had overextended sheath of optic nerves with corresponding high ONSD values already at the time of hospitalization. This fact can explain rather low, but reliable correlation of ICP and ONSD, which was lower than in the studies by Sekhon et al. (r=0.7).

According to literature data [15, 16], ONSD is a fairly dynamic parameter which can rapidly increase after the increase in ICP. Long exposure to ICP over 35—55 mmHg can promote overextention of the sheaths, which can persist in the future even at normalization of ICP. In our work, we compared only ONSD at the time of hospitalization with parameters of invasive measurement of ICP (maximal, mean, median). Preliminary analysis showed that ONSD values were significantly correlated with median and mean ICP values, but since most patients had abnormal ICP distribution, we chose median values for the analysis. In our opinion, the low correlation coefficient between ONSD and the median ICP can also be attributed to the "noisiness" of the ICP (**Figure 3**).

Thirdly, the objective of our retrospective study was to evaluate the correlation between ONSD and ICP and we have achieved this objective. In addition, we estimated the threshold value for ONSD, which can, potentially,

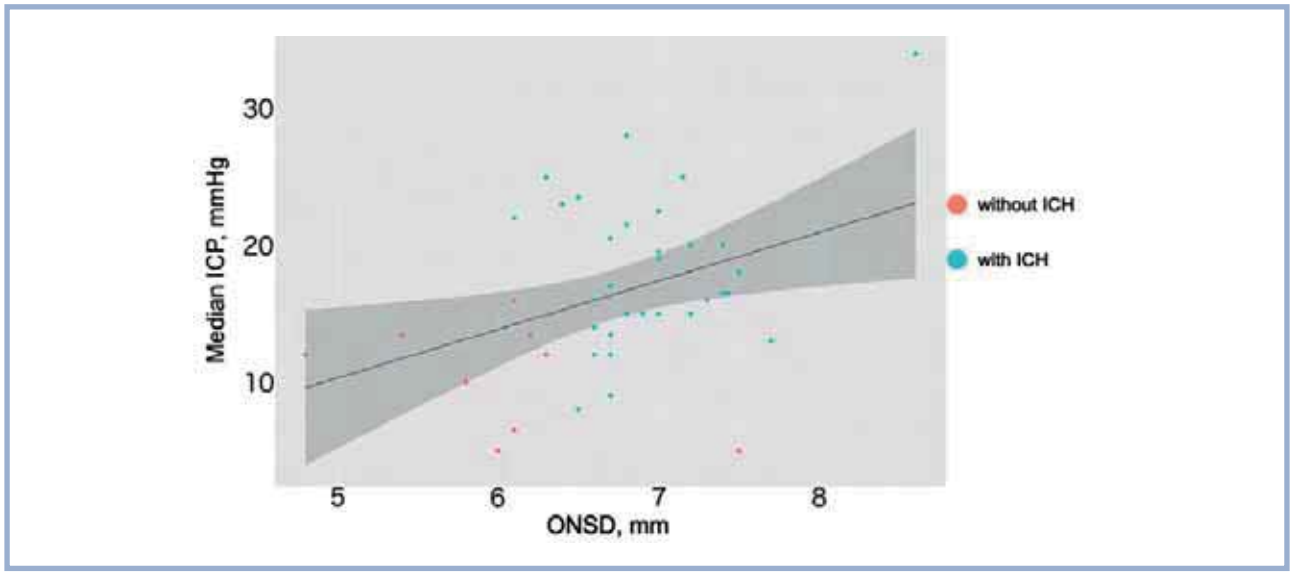

Fig. 5. **Correlation between ONSD (mm) and ICH (mmHg).**

The straight line is constructed using the least squares method, dark gray area around it represents a 95% confidence interval. The scatter of the points demonstrates that the values of ONSD in patients with normal ICP are usually lower than in patients with high ICP (above 20 mmHg).

allow a clinician to identify a group of patients which has already experienced ICH, with ICH that developed at the time of CT examination, or, which is of the highest particular importance, with a high probability of ICH development in the short term after the CT. We would like to note that ONSD values in the group of patients with ICH (**Table 1**) significantly exceeded those in the group with normal ICP. According to our data, 76% of the subjects developed ICH with a total duration of more than 1 hour, despite the full range of preventive and curative measures in accordance with accepted guidelines. In our opinion, higher than the threshold ONSD value at the time of the primary examination of patients with severe TBI may be an additional indication for invasive ICP monitoring, as it can reveal both the likelihood of the previous ICH and the likelihood of ICH development in the future.

However, it should be noted that the interpretation of ONSD values may be difficult in case of liquorrhea, cranio-orbit-facial trauma, after craniotomy, and drainage of the ventricular system.

Conclusions

A correlation between the CT-parameter ONSD and the median ICP in patients with severe TBI was identified with a correlation coefficient of 0.32 ($p<0.05$). The threshold ONSD value was 6.35 mm, with sensitivity of 0.93 (95% CI 0.84—1.00), specificity of 0.80 (95% CI 0.50—1.00), and AUC of 0.87 (95 % CI 0.69—1.00).

Authors declare no conflict of interest.

REFERENCES

1. Konovalov AN, Potapov AA, Likhterman LB. *Brain cranial trauma. Clinical guidelines in 3 books*. M. 1998-2002. (In Russ.).
2. Potapov AA, Krylov VV, Gavrilov AG, Kravchuk AD, Likhterman LB, Petrikov SS, Talypov AE, Zakharova NE, Oshorov AV, Sychev AA, Alexandrova EV, Solodov AA. Guidelines for diagnostics and treatment of severe traumatic brain injury. Part 2. Intensive care (treatment) and neuromonitoring. *Zhurnal Voprosy Neirokhir N.N. Burdenko*.2016;80(1):98-106. (In Russ.)

   https://doi.org/10.17116/neiro201680198-106
3. Carney N, Totten AM, O'Reilly C, Ullman JS, Hawryluk GW, Bell MJ, Bratton SL, Chesnut R, Harris OA, Kissoon N, Rubiano AM, Shutter L, Tasker RC, Vavilala MS, Wilberger J, Wright DW, Ghajar J. Guidelines for the management of severe traumatic brain injury, 4th ed. *Neurosurgery*. 2016;80(1):6-15. https://doi.org/10.1227/NEU.0000000000001432
4. Potapov AA. *Pathogenesis and differential treatment of local and diffuse brain injury*: Doctoral dissertation. M. 1989. (In Russ.).
5. Likhterman LB, Potapov AA, Kravchuk AD. Modern approaches to the diagnosis and treatment of craniocerebral trauma and its sequelae. *Jurnal Voprosy Neirokhir N.N. Burdenko*. 1996;1:35-37. (In Russ.). PMID: 8711970
6. Potapov AA, Zakharova NE, Pronin IN, Kornienko VN, Gavrilov AG, Kravchuk AD, Oshorov AV, Sychev AA, Zaitsev OS, Fadeeva LM, Takush SV. Prognostic value of ICP, CPP and regional blood flow monitoring in diffuse and focal traumatic cerebral lesions. *Voprosy neurokhirurgii im. N.N. Burdenko*. 2011;75(3):3-18. (In Russ.). PMID: 22066252
7. Zakharova N, Kornienko V, Potapov A, Pronin I. Neuroimaging traumatic brain injury. *Neuroimaging of Traumatic Brain Injury*. 2014;1-159.
8. Likhterman LB, Potapov AA, Kravchuk AD, Okhlopkov VA. Clinical classification and conceptual approaches to the treatment of of subsequently traumatic brain injury. *Voprosy Neirokhir N.N. Burdenko*. 1999;3:3. (In Russ.).
9. Potapov AA, Zakharova NE, Kornienko VN, Pronin IN, Aleksandrova EV, Zaitsev OS, Likhterman LB, Gavrilov AG, Danilov GV, Oshorov AV, Sychev AA, Polupan AA. Neuroanatomical basis for traumatic coma: clinical and magnetic resonance correlates. *Voprosy neyrokhirurgii im. N.N. Burdenko*. 2014;78(1):4-13. (In Russ.). PMID: 24761591
10. Eisenberg HM, Frankowski RF, Contant CF, Marshall LF, Walker MD. High dose barbiturate control of elevated intracranial pressure in patients with severe head injury. *J Neurosurg*. 1988;69:15-23.

    https://doi.org/10.3171/jns.1988.69.1.0015
11. Gjerris F, Brennum J. The cerebrospinal fluid, intracranial pressure and herniation of the brain. In: Paulson OB, Gjerris F, Sørensen PS, eds. *Clinical neurology and neurosurgery*. Copenhagen: FADL's Forlag Aktieselskab. 2004;179-196.
12. Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF, Eisenberg HM, Foulkes MA, Marshall LF, Jane JA. Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg*. 1991;66:883-890.
13. Kimberly HH, Noble VE. Using MRI of the optic nerve sheath to detect elevated intracranial pressure. *Crit Care*. 2008;12:181.

    https://do.org/ 10.1186/cc7008
14. Sekhon MS, Griesdale DE, Robba S, McGlashan N, Needham E, Walland K, Shook AC, Smielewski P, Czosnyka M, Gupta AK, Menon DK. Optic nerve sheath diameter on computed tomography is correlated with simultaneously measured intracranial pressure in patients with severe traumatic brain injury. *Intensive Care Med*. 2014;40:1267-1274.

    https://doi.org/10.1007/s00134-014-3392-7
15. Helmke K, Hansen HC. Fundamentals of transorbital sonographic evaluation of optic nerve sheath expansion under intracranial hypertension II. Patient study. *Pediatr Radiol*. 1996;26:706-710.
16. Hansen HC, Helmke K. Validation of the optic nerve sheath response to changing cerebrospinal fluid pressure: ultrasound findings during intrathecal infusion tests. *J Neurosurg*. 1997;87:34-40.

    https://doi.org/10.3171/jns.1997.87.1.0034
17. Launey Y, Nesseler N, Le Maguet P, Mallédant Y, Seguin P. Effect of osmotherapy on optic nerve sheath diameter in patients with increased intracranial pressure. *J Neurotrauma*. 2014;31:984-988.

    https://doi.org/10.1089/neu.2012.2829
18. Tayal VS, Neulander M, Norton HJ, Foster T, Saunders T, Blaivas M. Emergency department sonographic measurement of optic nerve sheath diameter to detect findings of increased intracranial pressure in adult head injury patients. *Ann Emerg Med*. 2007;49:508-514.

    https://doi.org/10.1016/j.annemergmed.2006.06.040
19. Rajajee V, Vanaman M, Fletcher JJ, Jacobs TL. Optic nerve ultrasound for the detection of raised intracranial pressure. *Neurocrit Care*. 2011;15:506-515. https://doi.org/10.1007/s12028-011-9606-8
20. Geeraerts T, Launey Y, Martin L, Pottecher J, Vigué B, Duranteau J, Benhamou D. Ultrasonography of the optic nerve sheath may be useful for detecting raised intracranial pressure after severe brain injury. *Intensive Care Med*. 2007;33:1704-1711. https://doi.org/10.1007/s00134-007-0797-6
21. Kalantari H, Jaiswal R, Bruck I, Matari H, Ghobadi F, Weedon J, Hassen GW. Correlation of optic nerve sheath diameter measurements by computed tomography and magnetic resonance imaging. *Am J Emerg Med*. 2013;31:1595-1597. https://doi.org/10.1016/j.ajem.2013.07.028
22. Zhong J, Dujovny M, Park HK, Perez E, Perlin AR, Diaz FG. Advances in ICP monitoring techniques. *Neurol Res*. 2003;25:339–350.

    https://doi.org/10.1179/016164103101201661
23. Oshorov AV. *Monitoring of cerebral perfusion pressure and autoregulation of cerebral blood flow in intensive care of severe brain injury*: Dis. .d-ra med. nauk. M. 2016. (In Russ.).
24. Saladino A, White JB, Wijdicks EF, Lanzino G. Malplacement of ventricular catheters by neurosurgeons: A single institution experience. *Neurocrit Care*. 2009;10:248-252. https://doi.org/10.1007/s12028-008-9154-z

25. Sahu S, Swain A. Optic nerve sheath diameter: A novel way to monitor the brain. *J Neuroanaesthesiol Crit Care*. 2017;4:13-18. https:/doi.org/10.4103/jnacc.jnacc_73_16

26. Bäuerle J, Schuchardt F, Schroeder L, Egger K, Weigel M, Harloff A. Reproducibility and accuracy of optic nerve sheath diameter assessment using ultrasound compared to magnetic resonance imaging. *BMC Neurol*. 2013;13:187. https:/doi.org/10.1186/1471-2377-13-187

27. Vaiman M, Abuita R, Bekerman I. Optic nerve sheath diameters in healthy adults measured by computer tomography. *Int J Ophthalmol*. 2015;8:1240-1244. https://doi.org/10.3980/j.issn.2222-3959.2015.06.30

28. Miller JD, Becker DP, Ward JD, Sullivan HG, Adams WE, Rosner MJ. Significance of intracranial hypertension in severe head injury. *J Neurosurg*. 1977;47(4):503-516. https://doi.org/10.3171/jns.1977.47.4.0503

Received: 26.04.17

Commentary

Intracranial hypertension is one of the most important problems in neurosurgery and neuroreanimatology, which arises in treatment of patients with severe traumatic brain injury (TBI) associated with both primary traumatic brain injuries and secondary changes and subsequent edema of the brain, disruption of perfusion parameters and liquorodynamic disturbances, and it requires a complex of individualized medical interventions.

This work reflects the constant drive in medicine to reduce the invasiveness in both medical and diagnostic practice at any stage of the patient's management. Computed tomography has long gone beyond the stationary diagnostic units, and mobile and compact CT machines are widely used in operating and resuscitation rooms.

The article analyses retrospective data of the measurement of the CT-cased optic nerve sheaths diameter (ONSD) in 41 patients in the acute period of TBI (within 2 days after the injury) and evaluates a correlation between them and the results of the invasive measurement of intracranial pressure (ICP) by the parenchymal sensor.

This method is highly relevant since CT-based ONSD measurement make it possible to indirectly assess the presence of intracranial hypertension without resorting to the invasive ICP monitoring, which naturally reduces the number of complications. There are quite a few publications devoted to this topic in foreign literature, but the number of Russian articles is quite low. The low prevalence of this technique is due, among other things, to the fact that intracranial pressure is a very labile indicator.

The authors rightly note that in the acute period of TBI ICP can vary a lot, whereas the diameter of the optic nerve sheath appears to be a more inert indicator of intracranial hypertension. There are may be cases when the patient already had a history of low intracranial pressure, and the ONSD remains elevated.

In the article, the authors consider cases in which the patients with certain to anatomical features or racial origins who had no history of TBI, have enlarged optic nerves with wide perineural spaces. Although this does not concern the topic of this paper, it would be interesting to investigate the dynamics of the restoration of the optic nerve sheaths after the normalization of intracranial pressure.

Overall, the work is interesting and relevant, well-presented and contains the results of the correlation analysis. The article can be useful to neuroradiologists, neurosurgeons and, of course, resuscitators. Resuscitators who have only just started their career can learn important information about various forms of non-invasive control of changes in intracranial pressure. It will also be of interest to neurophysiologists studying the features of liquorodynamics in patients with intracranial lesions.

M.B. Dolgushin (Moscow, Russia)