MORPHOLOGY OF THE SEQUELAE OF INCREASED INTRACRANIAL PRESSURE

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Summary. *With the introduction of the concept of primary and secondary brain injuries, it became clear that the outcome of one particular cranial-cerebral injury greatly depends on the secondarily initiated mechanisms, which are actually resulting of raised intracranial pressure (ICP). We can conclude about the existence of the raised ICP during person was alive, at the postmortem examination only by its effects on the brain tissue i.e. the signs of internal herniation as sequelae of it. This paper discusses our findings on the sequelae of raised ICP based on neuropathological examination of 80 forensic cases of closed head injury with a survival until 1,5 months. Our findings indicate that the herniation of the brain is going to occur in the first 10,5 days in 90% of the cases and in nearly half of them this deadly consequence can occur in the first 48 hours, which is of great clinical importance.*

Keywords: *ischemia, herniation, secondary brain injuries*

Introduction: With the introduction of the concept of primary and secondary brain injuries, it became clear that primary brain injuries (focal and diffuse brain injuries inflicted directly by forces of impact) are not decisive for the outcome of one particular cranial-cerebral injury, but greatly depend on the secondarily initiated mechanisms. The latter are actually result of raised intracranial pressure (ICP) [1, 4, 9, 10, 11].

The intracranial cavity is a space of limited volume, where three main contents are present: the brain 80%; the blood 2-11%; and the cerebrospinal liquor 10%. When the equilibrium of these contents is impaired, an increase in ICP occurs. The ICP is a clinical parameter which can be measured only ante mortem. Normal values are below 2 kPa (1 kPa =7,5 mm Hg), elevation to 3 kPa is considered mild, to 4 kPa moderate, and values exceeding 5 kPa are considered as severe intracranial hypertension [14]. The lethal upper limit of ICP is of 8-10 kPa.

During the post-mortem examination, what we can conclude about the increased ICP is only by its effects on the brain tissue and the occurrence of the signs of internal herniation [9, 11, 14, 15].

The herniation of the brain represents the movements of particular parts of the brain, from one compartment to another. The increased pressure in the supratentorial region leads to herniation against the edge of the tentorium cerebelli i.e. transtentorial herniation where the most exposed part is the temporal lobe uncus, including the hippocampus and the parahippocampal region. The increased pressure in the infratentorial compartment leads to herniation through the foramen magnum i.e. infratentorial herniation. This is associated with brainstem compression and death. The midline shift of the medial parts of the brain hemispheres (gyrus cinguli) to the left or right under the falx cerebri is known as subfalcine herniation [10, 14].

In this study, the sequelae of increased intracranial pressure i.e. signs of internal herniation have been analyzed in order to emphasize the characteristic morphological appearance of those injuries in the post-mortem examination of the brain and their correlation with the time of survival. **The overall purpose** of this study has been to contribute to the neuropathological criteria for determining the sequelae of increased intracranial pressure in the daily forensic medicine practice.

Material and methods: 80 cases with fatal closed head injury (57 male, 23 females, age ranged from 5 to 94 years), already presented in another study [5], have been now analyzed for the appearance and distribution of hypoxic-ischemic brain injury and the signs of internal herniation.

The inclusion criteria included post-mortem interval up to 24 h and the availability of data concerning: clear evidence of the type of the traumatic event [5] the known time of survival and full autopsy information. Clinical information was obtained for cases that survived long enough to be clinically investigated.

The survival period ranged from instantaneous death to 1.5 months (12 of the examined cases died quickly after the traumatic event, 25 of them survived 24 hours, 22 cases survived 1 week and the rest 21 cases survived more than 1 week, the longest survived 1,5 month).

All cases have been subjected to a forensic neuropathological examination of fixed brains in 10% buffered formalin [6, 8, 13].

Finding uncal notching or hemorrhages and necroses in the hippocampus and the parahippocampal area and infarctions of the inferior surfaces of both occipital lobes resulting from posterior cerebral artery

compression have been considered to be a sign of transtentorial herniation. The characteristic finding of the cerebellar tonsillar notching and the secondary brainstem hemorrhages which typically occur in the midline of the midbrain and pons (the so-called Duret hemorrhages) have been considered to be a sign of infratentorial herniation [5,10].

Results and discussion: Using the criteria given above, signs of the internal herniation have been perceived in 46 (57,5%) of the cases, Table 1.

Table 1. Finding signs of herniation in the examined cases.

Type of internal herniation	Number of cases (abs.46)	%
Transtentorial herniation	16	20
Infratentorial herniation	9	11,25
Subfalcine herniation	1	1,25
Transtentorial and infratentorial herniation	12	15
Transtentorial and subfalcine herniation	2	2,5
Infratentorial and subfalcine herniation	3	3,75
Transtentorial, infratentorial and subfalcine herniation	3	3,75

Table 2 presents the time of survival for all cases diagnosed with internal herniation. Using the data on Table 2, the distribution of the time of survival in cases with herniation was explored i.e. the dependence between the time of survival and the occurrence of herniation, Fig. 1.

Table 2. Time of survival for all cases diagnosed with internal herniation

Case No.	Type of the herniation	Time of survival
1	Transtentorial herniation	9 days
2	Infratentorial herniation	10 days
3	Subfalcine and transtentorial herniation	2 days
4	Infratentorial herniation	2-4 hours
5	Infratentorial herniation	6 days
6	Transtentorial herniation	until 1 hour
7	Subfalcine and infratentorial herniation	2 days
8	Infratentorial herniation	until 1 hour
9	Transtentorial and infratentorial herniation	3 days
10	Transtentorial and infratentorial herniation	12 days
11	Transtentorial and infratentorial herniation	10 days
12	Transtentorial and infratentorial herniation	10 days
13	Transtentorial herniation	immediately
14	Transtentorial and infratentorial herniation	2 days
15	Transtentorial and infratentorial herniation	1,5 month
16	Subfalcine herniation	3 weeks
17	Subfalcine and infratentorial herniation	3 days
18	Subfalcine herniation, transtentorial and infratentorial herniation	8 days
19	Transtentorial herniation	until 1 hour
20	Transtentorial herniation	immediately
21	Transtentorial herniation	immediately
22	Transtentorial herniation	15 days
23	Subfalcine herniation, transtentorial and infratentorial herniation	4 days
24	Transtentorial and infratentorial herniation	5 days
25	Subfalcine and infratentorial herniation	7 days
26	Transtentorial herniation	4 hours
27	Subfalcine herniation, transtentorial and infratentorial herniation	6 hours

28	Transtentorial herniation	immediately
29	Transtentorial herniation	6 hours
30	Transtentorial herniation	immediately
31	Transtentorial herniation	until 1 hour
32	Infratentorial herniation	10 days
33	Transtentorial and infratentorial herniation	2-4 hours
34	Infratentorial herniation	4 days
35	Transtentorial and infratentorial herniation	7 hours
36	Infratentorial herniation	10 days
37	Infratentorial herniation	2 days
38	Transtentorial herniation	6 days
39	Transtentorial herniation	6 days
40	Transtentorial and infratentorial herniation	24 hours
41	Subfalcine and transtentorial herniation	7 days
42	Transtentorial herniation	2 days
43	Infratentorial herniation	3 days
44	Transtentorial and infratentorial herniation	minutes
45	Transtentorial and infratentorial herniation	8 days
46	Transtentorial herniation	2 days

Upon the data from the Table 2, it has been explored the interdependence between the occurrence of any type of herniation in the 46 of the examined cases and the survival time, presented on Table 3.

Table 3. The interdependence of survival time with the occurrence of the herniation in 46 of the examined cases.

No of cases, in total	Cases with herniation	Percentage
Until 10,5 days	42	91%
Until 2days, 6 hours	23	50%
Until 24 hors	14	30%

The results shown on the graph analysis (Fig. 1.) demonstrate that:
– in 91% of the cases with herniation, the herniation occurred within the first 10,5 days after the injury;
– in 50% of them, the herniation occurred in less than two days and 6 hours;
– in 30% of the cases, the herniation occurred within in the first 24 hours.

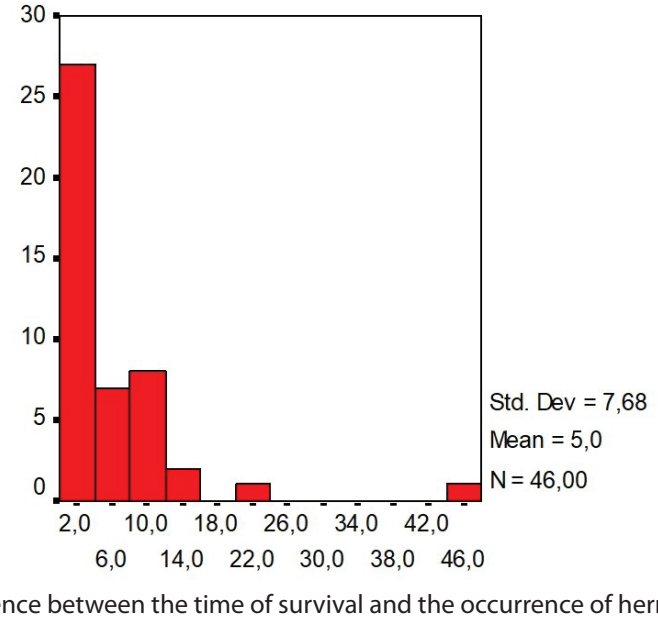

Fig. 1. Dependence between the time of survival and the occurrence of herniation.

Hence, 80 cases of fatal closed head injury in this study were analyzed with detailed forensic neuropathological examination [6, 13] for the occurrence of secondary brain changes resulting from the increased ICP.

Signs of internal herniation as the sequelae of the raised ICP were present in 57,5% of the examined cases, which is in accordance with other studies (56% by Adams et al. 1982 [2] and 55% of 85 examined cases by Adams et al. 2011[3]). Signs of transtentorial herniation (Fig. 2) have been found in 33 (41, 25%) of the cases. Signs of infratentorial herniation (Fig. 3) have been found in 27 (33,75%) of the cases, whereas signs of subfalcine herniation have been found in 9 (11,25%) of the examined cases. In a study with 434 analyzed cases [11], signs of transtentorial herniation have been found in two thirds of the cases, and signs of infratentorial herniation in 68% of cases.

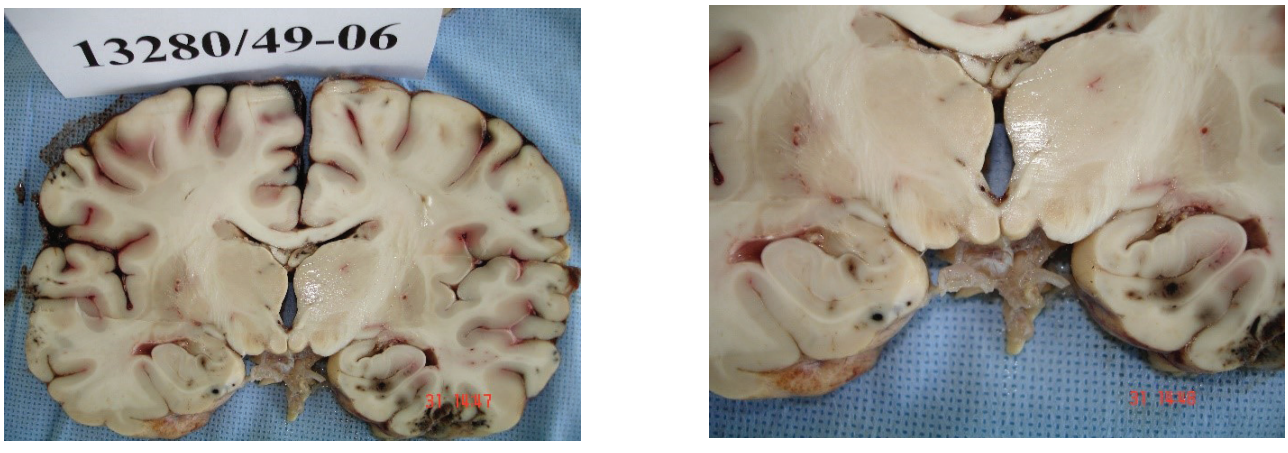

Fig. 2. **a. b.** Signs of transtentorial herniation. Transtentorial herniation. Case with survival time of 3-4 hours and the brain weight of 1.503 grams. On the section of the level of mammilary bodies have been seen hemorrhages in the hippocampus and the parahippocampal region on both sides.

Fig. 3. Signs of infratentorial herniation. Infratentorial herniation. The secondary Duret hemorrhages which are typically midline located in the midbrain and pons can be seen. a. Case with a survival of 7 hours and the weight of the brain of 1.487 grams; b. Case with a survival of 8 days and the brain weight of 1.512 grams.

In 91% of the cases with internal herniation, as shown by the results in this study, herniation occurred within the time frame of 10,5 days postinjury, implying that the threat of internal herniation is highest in the first 10 days after injury. Accordingly, in 50% of the cases herniation occurred in less than 2 days and 6 hours and in 30% of the cases it occurred in the first 24 hours. This analysis is mostly of clinical importance, obtaining information about the occurrence threat of internal herniation in cases with closed head injuries and possible time window for therapeutic intervention. From a forensic neuropathological point of view, besides the correlation with the survival time and proof for the existence of raised ICP ante mortem, this study emphasizes the morphological feature of herniation as it has been classically outlined [7,8,12].

Conclusion: Hence, the results of the present study show that in order to perceive the existence of the raised ICP during person has been alive, it is essential to perform postmortem the detail forensic neuropathological examination of the brain and to observe the signs of herniation as the sequelae of the raised

ICP. Herniation of the brain is deadly complication of closed head injury and in 91% of the closed injury cases it is going to occur in the first 10,5 days and in nearly half of them this deadly consequence can occur in the first 48 hours, which is of great clinical importance to take measures of avoiding them.

References:

1. Adams, H.J., J.A.N. Corsellis, L.W. Duchen.- *Greenfield's neuropathology*. 4th edition. *John Wiley&Sons. New York*, 1984.
2. Adams J.H., D.I. Graham, L.S. Muray, G. Scott. Diffuse axonal injury due to nonmissile head injury in humans: an analysis of 45 cases. *Ann.Neurol.,* 12, 1982, 557-563. doi: 10.1002/ana.410120610
3. Adams J.H., B. Jannet, L.S. Murray, M.G. Teasdale, T.A. Gennarelli, D.I. Graham. Neuropathological findings in disabled survivors of a head injury. - *J Neurotrauma.,* 28, 2011, 701-709. doi. 10.1089/neu.2010.1733
4. Adams, H., D.E. Mitchell, D.I. Graham., D. Doyle. Diffuse brain damage of immediate impact type. Its relationship to "primary brain stem damage" in head injury.- *Brain*, 100, 1977, (3):489-502. doi: 10.1136/jnnp.54.6.481
5. Davceva N., V. Janevska, B. Ilievski, G. Petrushevska, Z. Popeska. The occurrence of acute subdural haematoma and diffuse axonal injury as two typical acceleration injuries.- *J Forensic Leg Med.,* 19, 2012, 480-484.
6. doi: 10.1016/j.jflm.2012.04.022. Epub 2012 May 23.
7. Davceva N., Janevska V, Ilievski B, R. Jovanovic. The importance of the detail forensic-neuropathological examination in the determination of the diffuse brain injuries. - *Soud Lek.,* 57, 2012, 2-6. https://pubmed.ncbi.nlm.nih.gov/22724588/
8. Dolinak D., E. Matshes. *Medicolegal neuropathology. CRC Press New York 2002*.
9. Finnie J.W. Forensic pathology of traumatic brain injury – Review. *Veterinary Pathology.* 2016, Vol 53 (5) 962-978. https://doi.org/10.1177/0300985815612155
10. Graham D.I., J.H. Adams, D. Doyle. Ischemic brain damage in fatal non-missile head injuries.- J *Neurol Sci.,* 39,1978, 213-34. doi: 10.1016/0022-510x(78)90124-7.
11. Graham D.I., T.A. Gennarelli, T.K. McIntosh. Diffuse (multifocal) brain damage. In: Graham DI, Lantos PL (eds) - *Greenfield's neuropathology 7th edn, vol.1. Arnold, London* 2002, 854-875
12. Graham D.I., A.E. Lawrence, J.H. Adams, D. Doyle, D.R. Mclellan. Brain damage in non-missile head injury secondary to high intracranial pressure.- *Neuropathol Appl Neurobiol.,* 13, 1987, 209-217. doi: 10.1111/j.1365-2990.1987.tb00184.x.
13. Itabashi H.H., J.M. Andrews, U. Tomiyasu, S. Erlich, L. Sathyavagiswaran. *Forensic neuropathology. A practical review of the fundamentals. Elsevier* 2007.
14. Kalimo H., P. Saukko, D. Graham. Neuropathological examination in forensic context. *Forensic Sci Int.,* 146, 2004, 73-81. doi: 10.1016/j.forsciint.2004.06.022.
15. Miller J.D., J.W. Ironside. Raised intracranial pressure, oedema and hydrocephalus. In*: Graham DI, Lantos PL (eds) - Greenfield neuropathology, 6th edn. Arnold, London*, 1997, 157-195.
16. Oehmichen M., R.N. Auer, H.G. Konig. Forensic neuropathology and associated neurology. - *Springer-Verlag Berlin Heidelberg* 2006.

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FATAL INJURIES DUE TO BULLET FIREARMS

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***Summary*:** *Injuries due to firearms are a major public health problem, especially because they occur more frequently in young people, lead to human loss or disability of the population, and the mortality due to these injuries is greatly high. The work aims to highlight the frequency, dynamics, structure as well as some general peculiarities of lethal traumas produced by bullet firearms. The analysis includes 36 cases of fatal trauma caused by bullet, investigated at the Center for Forensic Medicine, during 2019-2023. The study determines that lethal trauma from bullet firearms accounts for 1.3% of all violent deaths and 59 % of all firearm deaths. More frequently, in 86% cases, men are traumatized, from urban localities, mainly in winter and summer months, and being drunk, which can be explained by their more frequent use of firearms for various purposes or this being a conception of interpersonal supremacy between men, as well as supremacy over women, respectively. The seasonal frequency can also be incident to annual holidays, in association with the consumption of alcohol. Mostly, the fatal injuries produced by bullets are localized at the level of the head, as transfixing wounds, and the death more frequently occurs at the people's home (aggressor/sufferer).*

***Keywords*:** *Firearm injuries, gunshot wounds, lethal trauma, violent death, traumatic objects*