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ACUTE TRANSIENT FORM OF HYDROCEPHALY: DIAGNOSTIC ASPECTS AND THERAPEUTIC TACTICS

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SUMMARY
This article presents diagnostic data and treatment tactics for acute transient hydrocephalus in 228 patients, which complicated the course of traumatic brain injury and stroke for the period 2019-2022. The purpose of the study: carry out analysis the diagnostic aspects characterizing intracranial hypertension syndrome of predominantly hydrocephalic origin with the development of acute transient form hydrocephaly as traumatic brain injury, acute cerebrovascular accident (stroke) and to develop optimal methods of its treatment. The analysis of 228 treated patients with acute transient form hydrocephalus predetermined the timely identification of its characteristic signs of development, which complicated the course of primary brain damage on days 8-14 with traumatic brain injury and on days 4-7 from the moment of stroke. Which allowed us to establish direct indications for non-invasive and invasive monitoring of intracranial hypertension syndrome of a hydrocephalic nature in the acute stage of traumatic brain injury and stroke.

KEYWORDS: Traumatic brain injury, stroke, acute hydrocephalus, neuroimaging, monitoring of intracranial pressure.

RELEVANCE

According to the World Health Organization (WHO), the increase in traumatic brain injury, cerebrovascular diseases (CVD) and their complications in the form of stroke (TBI - 2% annual increase, acute cerebrovascular accident (ACVA, stroke) - 1.5% annual increase in the morbidity structure), high mortality rate population (about 1.5 million people die annually), the duration of temporary and permanent disability (disability reaches up to 3 million people), also cause an increase in the frequency of intracranial complications, causing them to be a medical and social problem of modern medicine.[1,2]

Despite significant improvements in the technical equipment of medical institutions, modern advances in neurology, neurosurgery and neuroreanimatology, the results of treatment of patients with TBI, strokes and their intracranial complications remain unsatisfactory for clinicians.[3]

One of the most dangerous intracranial complications of cerebral pathology, in particular TBI and stroke, is the acute form of hydrocephalus, which in some cases is transient in nature.[4;5;6;7;8]

The relevance of the problem of ATFH is due to difficulties in solving a set of problems, including identifying characteristic clinical and neurological lesions, determining the choice of diagnostic and therapeutic tactics, the need for surgical intervention, timing and methods of their implementation.[9;10;11;12;13;14]

When choosing an adequate treatment and diagnostic tactic, the clinician, of course, first of all, is guided by the results of both clinical-neurological and paraclinical examination methods.[15;16]

Compliance with certain principles of diagnostic methods in combination with the analysis of clinical and neurological manifestations of the disease makes it possible to obtain objective criteria for treatment and prognosis of the outcome of the disease and, thereby, justify the feasibility of using one or another tactic for the management and treatment of patients.[17;18;19]

The purpose of the study was carry out analysis the characteristic diagnostic aspects in the diagnosis of intracranial hypertension in patients with ATFH due to TBI, stroke and determine the tactics of its treatment.

Material and methods of research. An analysis of the results of treatment of 228 patients with TBI (n=136) and stroke (n=92) admitted to the Bukhara branch of the Republican Research Center for Emergency Medicine for the period 2019–2022 was carried out, in whom the development of ATFH was noted, which amounted to 30.2 % (n=755 patients).

Men – 174 (76.3%), women – 54 (23.7%) observations. The age median of the patients was 47.15±16.95 years (from 18 to 84 years). All patients underwent a comprehensive examination. Methods for non-invasive ICP monitoring carried out and compared with data from neuroimaging signs of ATFH.

In our work, we used the calculation of liquorocranial (ventricular cranial) coefficients (morphometric) based on CT monitoring: VCK-bodies of the ventricles; VCK-1 (Evans index); VCK-3rd ventricle; Width of the 3rd ventricle; Dimensions of the optic nerve (width dON) (formula 1; Fig. 1-4); Determination of the ratio of the size dON to the width of the 3rd ventricle - HgS. (formula 2).

When calculating the gradient of the ratio of the width dON and the size of the 3rd ventricle, the ICP coefficient was higher than 1.0, we diagnosed ICP with hypertensive syndrome (HtS) of parenchymal origin (cerebral edema). If the ICP coefficient was less than 1.0, we interpreted ICH due to hydrocephalic syndrome (HgS - hyperproduction, dyshyporesorption of cerebrospinal fluid, occlusion of cerebrospinal fluid tracts). The normal ICP coefficient in healthy individuals was approximately 1.0 (Table 1).

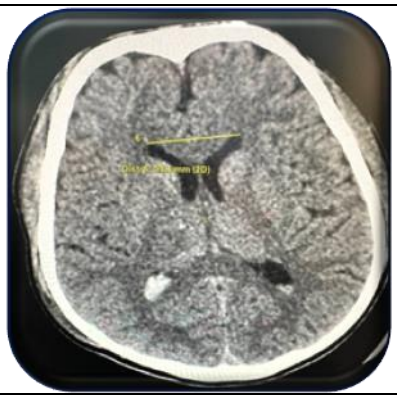


Fig. 1

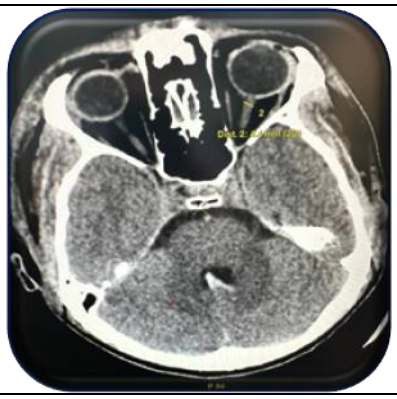


Fig. 2

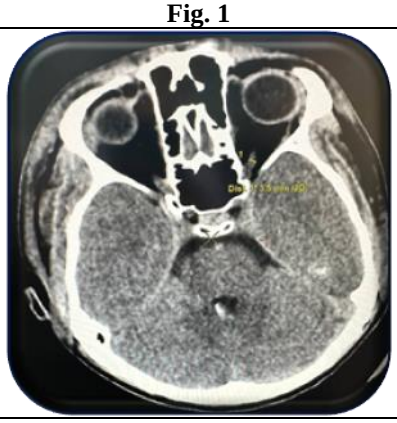


Fig. 3

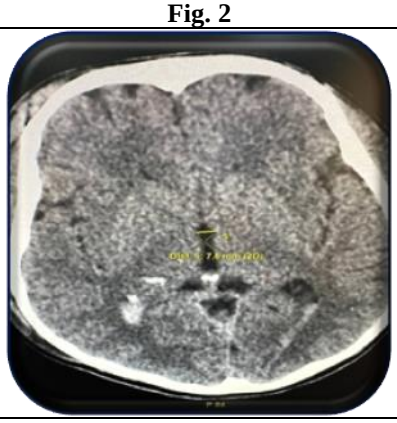


Fig. 4

CT morphometric: Fig.1-calculation of VKK-1 (Evans index); Fig.2-measurement of the width of the optic nerve behind the eyeball by 2 mm (d1); Fig.3-measurement of the width of the optic nerve at the exit from the cranial cavity by 2 mm (d2); Fig.4-measurement of the width of the 3rd ventricle.

Method for calculating the intracranial pressure coefficient (formulas).

Formula 1	**Formula 2**
Determination of the average value (width) of the optic nerve (dON)	Coefficient for calculating intracranial pressure (ICPk)
	ICP coefficient in the control group (healthy) ICPk=1.0
$d_s = \frac{d_1+d_2}{2}$	$ICP_k = \frac{d_s}{V_3}$

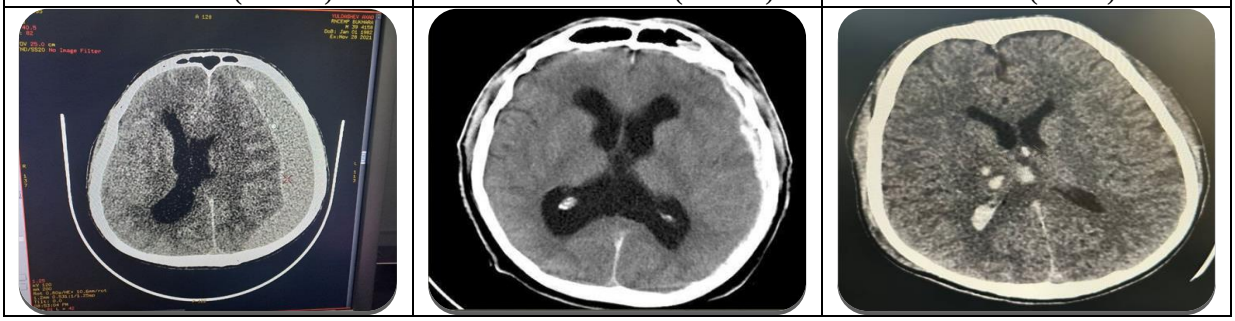
Table 1: Characteristics of the intracranial pressure coefficient.

Intracranial pressure calculation factor (ICPk)	
hypertensive parenchymal syndrome (HtS) - swelling of brain tissue; (ICPk>1.0)	hydrocephalic syndrome (HgS) - acute transient form of hydrocephalus; (ICPk<1.0)
$HtS = \frac{d_s(5,2mm)}{V_3(4,2mm)} = 1,23$	$HgS = \frac{d_s(5,4mm)}{V_3(6,8mm)} = 0,79$

Results and their discussion: When assessing clinical and neurological manifestations, the presence and severity of neurological symptoms of ATF, as well as the choice of the scope of therapeutic and diagnostic measures, the leading importance given to focal prolapses and brainstem disorders (table 2 and 3).

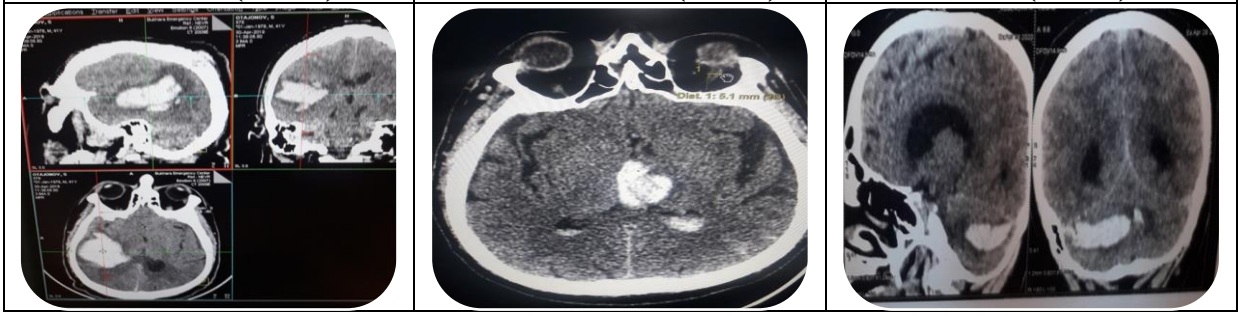
The distribution of patients depending on the type and localization of damage to brain structures characterized as follows: convexital - 117 (51.3%), convexital-basal - 58 (25.4%) and basal (including intraventricular and posterior fossa localization) - 53 (23.3%) (Fig. 5).

Localization of injury of traumatic etiology (n=136)		
Convexital 75(55.15%)	Convexital-basal 44(32.35%)	Basal 17(12.5%)



a)

Localization of damage of non-traumatic etiology (n=92)		
Convexital 42(45.65%)	Convexital-basal 14(15.22%)	Basal 36(39.13%)



b)

Figure 5: CT characteristics of primary brain injury/damage: a) with TBI; b) with stroke.

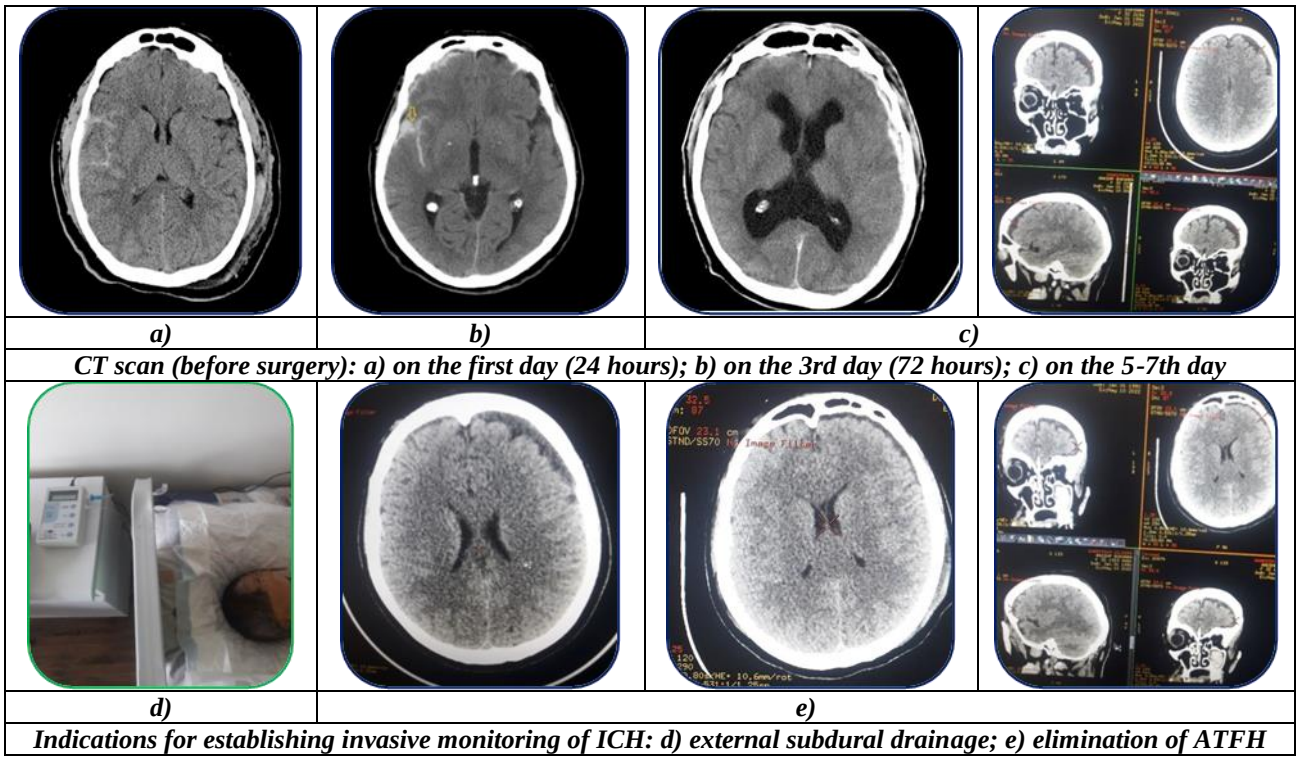
General clinical signs of the development of ICH were characterized by a decrease in pulse (PS<60 beats/min), an increase in blood pressure (BP>140/90mmHg) and with the development of ATPH they changed on days 8-14 against the background of TBI and on days 4 -7 days from the moment of stroke. Stagnant ONH reflected hydrocephalic-hypertensive syndrome and, according to the number of observations, increased by 8-14 days in case of TBI and 4-7 days in case of stroke.

An examination by an otolaryngologist carried out on 154 (67.54%) patients with the characteristic examination of the eardrum. In all observations, its bulging was noted - a sign of increased ICP. CT monitoring made it possible to determine predictors of the risk of developing ATFH with its traumatic etiology on days 8-14 and non-traumatic origin on days 4-7 (tables 4 and 5).

Monitoring of ATFH with ICH syndrome using a neuroimaging method predetermined the installation of active invasive monitoring (on days 3-7, 10-14) in 40 (42.39%) patients.

Clinical example 1. *Observation of ATFH of traumatic etiology with convexital-basal localization of damage. Diagnosis: "TBI. Brain contusion of moderate severity. Traumatic subarachnoid hemorrhage of the right basal frontotemporal region. Acute traumatic subdural hematoma of the left hemisphere.*

Complications: Brain edema. Acute transient hyporesorptive hypersecretory form of hydrocephalus (mixed type).



a) ***b)*** ***c)***

CT scan (before surgery): a) on the first day (24 hours); b) on the 3rd day (72 hours); c) on the 5-7th day

d) ***e)***

Indications for establishing invasive monitoring of ICH: d) external subdural drainage; e) elimination of ATFH

Table 2: Data from an objective assessment of the neurological status in patients with ATFH due to TBI.

Neurological criterion		**upon admission**		**p**	**with the development of ATFH**		**p**
		abs	**M(%±m)**		**abs**	**M(%±m)**	
Level of consciousness (GCS)	Clear (15)	8	5,88±2,02	χ^2=56,353 p=0,000	2	1,47±1,03	χ^2=44,882 p=0,000
	Moderate stunning (13-14)	37	27,21±3,82		19	13,97±2,97	
	Deep stun (11-12)	41	30,15±3,94		29	21,32±3,51	
	Stupor (9-10)	33	24,26±3,68		42	30,88±3,96	
	Superficial coma (6-8)	11	8,09±2,34		31	22,79±3,60	
	Deep coma (3-5)	6	4,41±1,76		13	9,56±2,52	
p		Wilcoxon signed rank test W=-10,000; p=0,000					
Total		**136**	**100,00**		**136**	**100,00**	
Neurological criterion		**upon admission**		**with the development of ATFH**		**McNemar criterion**	
		abs	M(%±m)	abs	M(%±m)	χ^2	p
Cranial nerves disorders	Anisocori	51	37,50±4,15	119	87,50±2,84	66,015	0,000
	Nystagmus	83	61,03±4,18	124	91,18±2,43	39,024	0,000
Movement disorders	Monoparesis	9	6,62±2,13	23	16,91±3,21	-	0,000
	Monoplegia	3	2,21±1,26	11	8,09±2,34	-	0,008
	Hemiparesis	43	31,62±3,99	89	65,44±4,08	44,022	0,000
	Hemiplegia	6	4,41±1,76	13	9,56±2,52	-	0,016
Disturbances of the sensitive sphere	Hypoesthesia	9	6,62±2,13	24	17,65±3,27	-	0,000
	Hyperesthesia	52	38,24±4,17	112	82,35±3,27	58,017	0,000

Meningeal symptoms	78	57,35±4,24	127	93,38±2,13	47,020	0,000
Pathological symptoms	61	44,85±4,26	125	91,91±2,34	62,016	0,000

Table 3: Data from an objective assessment of the neurological status in patients with ATFH due to ACVA.

Neurological criterion		**upon admission**		**p**	**with the development of ATFH**		**p**
		abs	**M(%±m)**		**abs**	**M(%±m)**	
Level of consciousness (GCS)	Clear (15)	0	0,00	χ^2=20,717 p=0,000	0	0,00	χ^2=41,261 p=0,000
	Moderate stunning (13-14)	17	18,48±4,05		4	4,35±2,13	
	Deep stun (11-12)	23	25,00±4,51		11	11,96±3,38	
	Stupor (9-10)	32	34,78±4,97		39	42,39±5,15	
	Superficial coma (6-8)	14	15,22±3,74		25	27,17±4,64	
	Deep coma (3-5)	6	6,52±2,57		13	14,13±3,63	
p		Wilcoxon signed rank test W=-7,693; p=0,000					
Total		92	100,00		92	100,00	

Neurological criterion		**upon admission**		**with the development of ATFH**		**McNemar criterion**	
		abs	**M(%±m)**	**abs**	**M(%±m)**	χ^2	**p**
Cranial nerves disorders	Anisocori	40	43,48±5,17	78	84,78±3,74	36,026	0,000
	Nystagmus	52	56,52±5,17	89	96,74±1,85	35,027	0,000
Movement disorders	Monoparesis	0	0,00	0	0,00		
	Monoplegia	0	0,00	0	0,00		
	Hemiparesis	40	43,48±5,17	78	84,78±3,74	36,026	0,000
	Hemiplegia	12	13,04±3,51	14	15,22±3,74		0,500
Disturbances of the sensitive sphere	Hypoesthesia	12	13,04±3,51	14	15,22±3,74		0,500
	Hyperesthesia	40	43,48±5,17	89	96,74±1,85	47,020	0,000
Meningeal symptoms		62	67,39±4,89	76	82,61±3,95	47,020	0,000
Pathological symptoms		52	56,52±5,17	92	100,00	38,025	0,000

Table 4: Characteristics of neuroimaging monitoring of ICH in ATFH of traumatic etiology.

Indicator	**Dynamics of ICH monitoring (n=136)**				**Friedman criterion**	
	0–3 days	**4–7 days**	**8–14 days**	**15–21 days**		
	M(±m)	**M(±m)**	**M(±m)**	**M(±m)**	χ^2	**p**
VCK-bodies (18–26%)	17,43±0,16	20,56±0,16	21,72±0,14	20,70±0,23	49,882	0,000
VCK-1 (24,0-29,1%)	27,28±0,21	32,03±0,24	33,07±0,28	29,07±0,48	56,989	0,000
VCK-3v (2,7-4,3%)	2,72±0,04	4,27±0,07	4,37±0,06	3,66±0,11	61,069	0,000
dON (4,0-4,5 mm)	3,77±0,05	5,52±0,04	5,30±0,06	5,02±0,20	48,080	0,000
v3 (4-8 mm)	3,53±0,06	6,42±0,09	6,17±0,10	4,69±0,28	57,228	0,000
HgS (≥1≤) (ICPk)	1,08±0,01	0,86±0,01	0,82±0,01	1,07±0,01	71,760	0,000
Reliability of differences in relation to the previous study period						
Wilcoxon signed rank test	0–3 days <---> 4–7 days		4–7 days <---> 8–14 days		8–14 days <---> 15-21 days	
	«W»	«P»	«W»	«P»	«W»	«P»
VCK-bodies (18–26%)	-9,821	0,000	-4,072	0,000	-2,379[c]	0,017
VCK-1 (24,0-29,1%)	-9,947	0,000	-0,770	0,441	-3,814	0,000
VCK-3v (2,7-4,3%)	-9,955	0,000	-0,663	0,507	-3,654	0,000
dON (4,0-4,5 mm)	-9,856	0,000	-0,495	0,621	-2,645	0,008
v3 (4-8 mm)	-9,974	0,000	-0,524	0,600	-3,632	0,000
HgS (≥1≤) (ICPk)	-9,979	0,000	-6,184	0,000	-3,823	0,000

Table 5: Characteristics of neuroimaging monitoring of ICH during ATFH due to stroke.

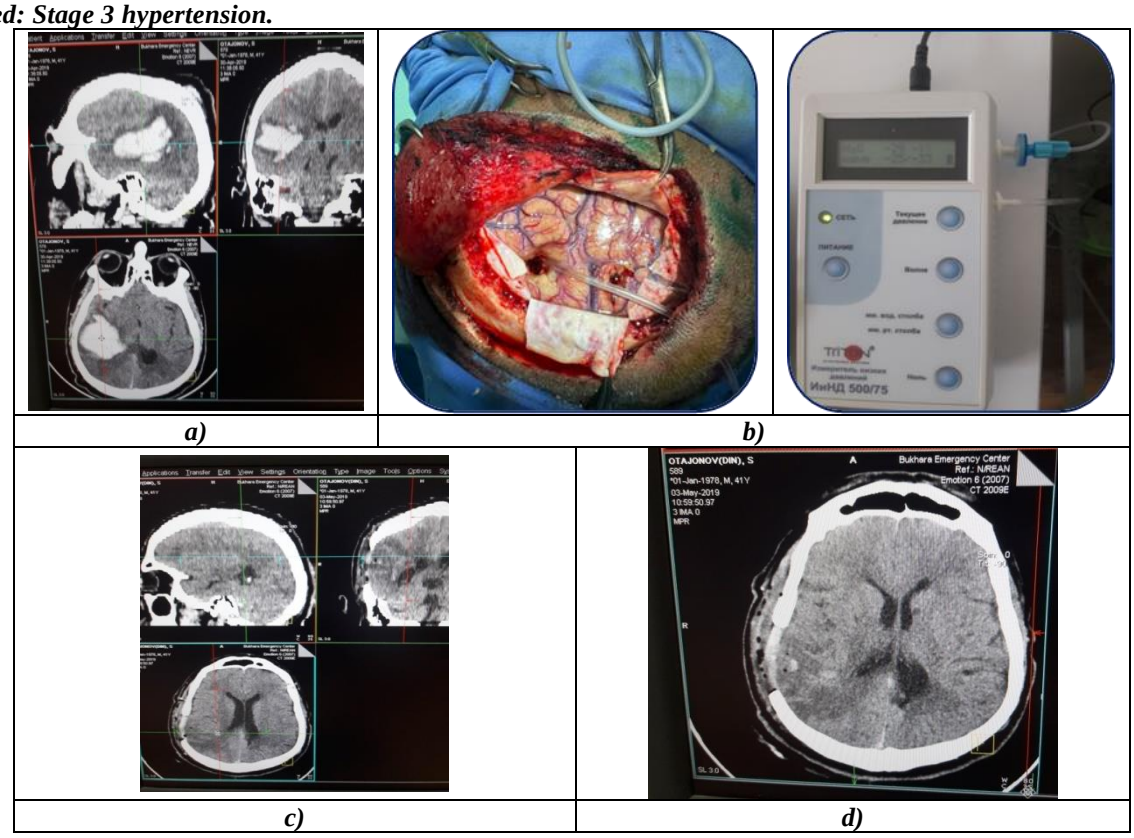
Indicator	Dynamics of ICH monitoring (n=92)				Friedman criterion	
	0–3 days	4–7 days	8 – 14 days	0–3 days		
	M(±m)	M(±m)	M(±m)	M(±m)	χ^2	p
VCK-bodies (18–26%)	17,48±0,31	21,83±0,20	20,22±0,18	20,68±0,39	27,495	0,000
VCK-1 (24,0-29,1%)	27,78±0,32	33,66±0,45	31,52±0,27	29,95±0,73	31,734	0,000
VCK-3v (2,7-4,3%)	2,70±0,06	4,49±0,09	4,22±0,09	3,90±0,18	23,807	0,000
dON (4,0-4,5 mm)	3,90±0,08	5,49±0,07	5,26±0,07	5,01±0,18	21,495	0,000
v3 (4-8 mm)	3,67±0,10	6,54±0,14	6,04±0,14	5,50±0,32	23,532	0,000
HgS (≥1≤) (ICPk)	1,06±0,01	0,84±0,01	0,87±0,01	0,91±0,01	25,944	0,000
Reliability of differences in relation to the previous study period						
Wilcoxon signed rank test	0 – 3 days <---> 4 – 7 days		4 – 7 days <---> 8 – 14 days		8 – 14 days <---> 15-21 days	
	«W»	«P»	«W»	«Z»	«W»	«P»
VCK-bodies (18–26%)	-7,930	0,000	-3,699	0,000	-2,138	0,033
VCK-1 (24,0-29,1%)	-8,081	0,000	-2,383	0,017	-2,317	0,020
VCK-3v (2,7-4,3%)	-8,088	0,000	-0,331	0,741	-2,050	0,040
dON (4,0-4,5 mm)	-7,840	0,000	-1,233	0,217	-2,051	0,040
v3 (4-8 mm)	-8,059	0,000	-0,663	0,508	-2,093	0,036
HgS (≥1≤) (ICPk)	-8,063	0,000	-3,901	0,000	-1,163	0,245

Clinical example 2. *Observation of ATFH against the background of stroke: convexital localization of the lesion.*

Diagnosis: ACVA (stroke) of the parenchymal-hemorrhagic type in the MCA basin on the right. Intracerebral stroke-hematoma of the right hemisphere.

Complications: Brain edema. Sopor. Hunt-Hess-III, according to WFNS-IV. Acute transient hyporesorptive mixed form of hydrocephalus.

Related: Stage 3 hypertension.



a) CT signs of putamenal stroke hematoma on the right (before surgery); b) visualization during craniotomy with removal of stroke hematoma and establishment of invasive monitoring of ICP in the right lateral ventricle; c) control CT with calculation of ICP and ICP on the 5th day after surgery: signs of the development of moderate ATFH and d) CT monitoring of ICP: signs of regression of ATFH on the 9th day after surgery

According to the study data, when assessing the outcome and results of the ATFH treatment using the modified Rankin scale, we noted 0 points (no symptoms) in 13 patients (5.7%). In 31 (13.6%) patients with ATFH, a score of 1 point (no significant impairment of life activity). In 53 observations (23.7%) the ATFH score was 2 (mild impairment of vital functions, the patient is able to cope without assistance). In 37 patients (22.4), the outcome rated 3 points (moderate impairment of vital functions, the patient is able to walk independently). 4 points (severe impairment of vital functions, needs outside help) assessed in 20 (11.0%) patients. A 5-point score (severe impairment of vital functions, the patient is "bedridden") established in 41 patients (18.0%). In addition, 6 points noted in 33 (14.5%) cases of ATFH - death. According to the data obtained, in 67.5% - 154 observations, an improvement in condition noted in the immediate period.

CONCLUSIONS

Thus, we noted

1. Typical diagnostic predictors of the development of ATFH observed against the background of TBI on days 8-14 and against the background of stroke on days 4-7 from the moment of the cerebrovascular accident.
2. When identifying signs of the development of ATFH, patients must undergo dynamic neuroimaging monitoring in comparison with data from non-invasive paraclinical research methods to determine the development or absence of ICH with a hydrocephalic component.
3. Based on the data obtained, the study confirming the development of signs of ATFH made it possible to predetermine the indications for the use of an invasive "active" method of monitoring ICP either by subdural drainage of cerebrospinal fluid or by installing an external ventricular drainage.

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