Published in final edited form as:

Acta Neurochir Suppl. 2020 ; 127: 185–190. doi:10.1007/978-3-030-04615-6_29.

Cerebral Arterial Compliance in Polytraumazed Patients with Cerebral Vasospasm

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Abstract

The purpose was to determine the status of the cerebral arterial compliance (cAC) in a concomitant head injury and cerebral vasospasm (CVS) with and without the development of intracranial hematomas (ICH). In Materials and Methods, we examined 80 polytrauma patients with severe TBI and CVS. During or immediately after dynamic helical computed tomography angiography (DHCTA), the monitoring of the transcranial Doppler of the MCA was recorded bilaterally with 2-MHz probes. The cerebral blood volumes were calculated from the DHCTA data with complex mathematical procedures using the “direct flow model” algorithm. In Results, CAC was significantly decreased ($p < 0.001$) in both the first and second group TBI and CVS (with or without ICH) in comparison with normal data ($p < 0.001$) and TBI without CVS. The cAC was significantly decreased on the side of the former hematoma with CVS than on the contralateral side with CVS ($p = 0.003$). In Conclusion, the cAC in TBI and CVS gets significantly lower as compared to the normal condition ($p < 0.001$). After removal of the ICH and development of CVS, the compliance in the perifocal zone remains much lower ($p = 0.003$) as compared to compliance of the other brain hemisphere.

Keywords

Cerebral arterial compliance; Cerebral vasospasm; Intracranial hematomas; Traumatic brain injury

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Conflict of Interest: We declare that we have no conflicts of interest.

Introduction

Cerebral vasospasm (CVS) after traumatic brain injury (TBI) may dramatically affect the neurological and functional recovery. Secondary insults in patients with TBI and CVS are greatly affected by changes in the compliance and stiffness of cerebral vessels. The walls of downstream vessels have no external elastic membrane; therefore, the cerebral capillary network becomes vulnerable to intracranial and intravascular pressure surges [1, 2].

One of the features characterizing flexibility of the vascular network and its resistance to the changes is cerebral arterial compliance (cAC) [3, 4].

The state of the cAC at TBI and CVS is of great importance for the brain microcirculation. Since the brain is located within an inextensible cranial cavity and is surrounded by an incompressible fluid [5], the compensation of intracranial pressure surges caused by the pulse wave passage through the brain blood vessels occurs also through the reciprocal changes in arterial lumens [2, 6, 7].

Thus, the higher is the cAC, the greater is the compliance of a vascular wall, and, respectively, the better is the capacity of a vessel to change its lumen (i.e., vasomotion phenomenon) and thereby to maintain the adequate capillary bed perfusion.

Information on the compliance and stiffness of the cerebral vascular bed in the damaged brain after CVS is currently rather inconsistent [4, 8], and the aspects of the cAC reaction to the intracranial hematoma (ICH) development and the disturbed cerebral blood flow (CBF) in the case of TBI and CVS are still under investigation [9].

The purpose of our work was to determine the status of the cerebral arterial compliance in a concomitant head injury and posttraumatic cerebral vasospasm with and without the development of intracranial hematomas.

Materials and Methods

The study was approved by the Ethics Committee of the Nizhny Novgorod State Medical Academy and conformed to the standards of the Declaration of Helsinki. We examined 80 polytrauma patients with severe head injury and CVS who were treated at the Nizhny Novgorod Regional Trauma Center Level I in 2013–2015. The mean age of the patients with head injury was 35.5 ± 14.8 years (from 15 to 73 years, 38 women and 42 men). The criterion for the inclusion in the study was the CVS of the M1 and M2 segments of the middle cerebral artery revealed during the contrast-enhanced CT scanning of the brain.

All patients were divided into two groups. The first group included 41 polytraumatized patients with the CVS in the acute period head injury without the development of intracranial hematomas (ICH). The second included 39 polytraumatized patients with the developed CVS and the brain compression caused by epidural, subdural, or multiple hematomas (6, 29, and 4, respectively).

The average wakefulness level according to GCS (Glasgow Coma Score) was 9.7 ± 2.5 in the first group and 10.1 ± 2.5 in the second group. The severity of their state according to

ISS (Injury Severity Score) was 34.3 ± 8.2 in the first group and 35.2 ± 9.3 in the second group. Clinical outcomes are summarized in Table 1.

Dynamic Helical Computed Tomography Angiography

All patients were subjected to dynamic helical computed tomography angiography (DHCTA) by 64-slice tomograph Philips Ingenuity CT (Philips Medical systems, Cleveland, USA). Tomography was performed 1–12 days after TBI (mean 4 ± 3 days) in the first group and 2–8 days (mean 4 ± 2 days) after surgical evacuation of the hematoma in the second group.

The perfusion examination report included an initial contrast-free CT of the brain. Extended scanning was further performed of 16 “areas of interest,” 160 mm in thickness, within 60 s with a contrast agent (“Perfusion JOG” mode). The scanning parameters were 160 kVp, 160 mA, 70 mAs, 512×512 . The contrast agent Ultravist 370 (Schering AG, Germany) was administered with an automatic syringe injector (Stellant, Medrad, Indianola, PA, USA) into a peripheral vein through a standard catheter (20 G) at a rate of 4–5 mL/s in a dose of 30–50 mL per one examination.

After scanning, data were transferred to a PACS (JSC “KIR,” Kazan, Russia) and a Philips Extended Brilliance Workspace workstation (Philips HealthCare Netherland B.V., Best, the Netherlands) and MATLAB 2013b (The MathWorks Inc., Natick, MA, USA). Artery and vein marks were automatically recorded, followed by the manual control of indices in the time-concentration diagram. The so-called region of interest (ROI) was established based on subcortical areas of middle cerebral artery.

The computed tomography angiography source image (CTASI) analysis enabled to visualize the main vessels of the brain and to assess the state of their lumen. In all patients the minimal intensive projection analysis identified the local luminal narrowing middle cerebral artery (MCA) more than 30% of the diameter as compared to adjacent sections of the same vascular segment; based thereon an “angiographic” CVS was diagnosed. Vasospasm according to its severity is usually classified into three grades: mild, the vessel still has 70% of luminal flow; moderate, more than 50% of reduction of the lumen; and severe, less than 30% of luminal flow on angiography.

Perfusion maps were derived from the tissue time-attenuation curve on the basis of the change in X-ray attenuation, which is linearly related to iodinated contrast concentration on a per-voxel basis with time. Errors introduced by delay and dispersion of the contrast bolus before arrival in the cerebral circulation were corrected by block-circulant deconvolution algorithm. Quantitative perfusion indices, including CBF, were calculated on a voxelwise basis and were used to generate color-coded maps. The voxels with $\text{CBF} > 100 \text{ mL}/100 \text{ g}/\text{min}$ or $\text{CBV} > 8 \text{ mL}/100 \text{ g}$ were assumed to contain vessels and removed from the ROI.

During or immediately after DHCTA, the transcranial Doppler (TCD) of the MCA was recorded bilaterally with 2-MHz probes within 10 min (Sonomed 300 M, Spectromed, Russia). Amplitude of arterial blood pressure (ABPamp) and ECG-gated duration of the systole (Tsys) and the diastole (Tdia) were measured noninvasively (IntelliView MP5,

Philips Medizin Systeme, Germany). A complex of the neuromonitoring “Centaurus” was used during the study (Ver. 2.0, Nizhny Novgorod State Medical Academy, Russia).

The system view is shown in Fig. 1.

Statistical Analysis

CBF and cerebral blood volume (CBV) were calculated from DHCTA data by the “direct flow model” algorithm [10]. The systolic–diastolic values of the MCA diameters (D_{sys} and D_{dia}) were determined in DHCTA series in the proximal part of the M1 of both MCA. Amplitude of regional CBV oscillation (ΔCBV) was calculated as the difference between CBVs, which flowed through the MCA in systole (CBV_{sys}) and diastole (CBV_{dia}). We used Eqs. (1, 2, and 3) proposed by de Jong, Alexandrov, and Avezaat [11–13].

$$\Delta CBV = CBV_{sys} - CBV_{dia} \quad (1)$$

$$\Delta CBV = \frac{\pi}{4} \times D_{sys}^2 \times CBFV_{sys} \times T_{sys} - \frac{\pi}{4} \times D_{dia}^2 \times CBFV_{dia} \times T_{dia} \quad (2)$$

$$cAC = \Delta CBV \div ABP_{amp} \quad (3)$$

Reference range cAC was chosen according Ikdip K. as $0.105 \pm 0.043 \text{ cm}^3/\text{mmHg}$ [14].

The *t*-test for dependent samples was utilized to analyze differences in means of parameters between the ipsilateral and contralateral sides of the temporal lobes. Statistica 7.0 software (StatSoft Inc., USA) was used for the analysis. Data are presented as Mean $\pm$ SEM. Significance level was preset to $p < 0.05$.

Results

In the first group, the “angiographic” CVS was unilateral in 30 cases and bilateral in 11 cases, and in three cases it extended to segments of the anterior cerebral artery. In 15 cases the CVS was mild, in 21 cases it was moderate, and in 5 cases it was severe. The “angiographic” CVS coincided with the “dopplerographic” CVS in all patients with the severe CVS and in five patients with the moderate CVS. In the second group in 28 cases, the “angiographic” CVS was revealed on the side of the removed hematoma. In ten cases it was developed on the side opposite to the removed ICH, and in one case it was bilateral and included in addition to M1–2 also segments A1–2. In 13 cases the CVS was mild, in 16 cases it was moderate, and in 10 cases it was severe. The “angiographic” CVS coincided with the “dopplerographic” CVS in nine patients with the severe and moderate CVS.

Mean values of cAC were significantly decreased ($p < 0.001$) in both the first and second group of with TBI and CVS (with or without ICH) comparing to reference data ($p < 0.001$) and TBI without CVS.

The cAC was significantly decreased on the side of the former hematoma with CVS than on the contralateral side with CVS ($p = 0.003$). The acquired and analyzed data are summarized in Table 2.

Discussion

It has been proven that the constriction of cerebral arteries developing after traumatic subarachnoid hemorrhage (SAH) may result in reduction in CBF more distal than the spastic segment and depending on the state of the autoregulation may lead to brain ischemia and cerebral infarction [15, 16]. However, there are contradictory data on CVS as the cause of cerebral ischemia development after SAH. According to some reports, only in 20–30% of patients with the “angiographic” CVS some cerebral ischemia symptoms would develop [17]. Furthermore, the localization of the secondary ischemia in almost 25% of cases does not coincide with the territory of the spastic artery [10, 17, 18].

However, other researchers have reported high correlation between the “angiographic” CVS and secondary ischemia development. According to R. Crowley, only 3% of cerebral ischemia cases with SAH are either not followed by vasospasm, or it may be referred to a mild one [19].

Despite the fact that foregoing data mainly describe the dynamics of aneurysmal subarachnoid hemorrhages, they quite fully reflect total variety of cerebral microvascular reactions aimed at maintaining the adequate perfusion with available CVS, including a posttraumatic CVS [20–22].

It has been previously shown that cerebral microcirculation undergoes significant changes (especially in the compression of the brain by ICH), which remain even after hematoma’s removal [23].

At the same time, it is known that enveloped hematoma and the concomitant injury are factors that provoke posttraumatic CVS development [3, 4, 24].

Thus, the study of the cerebral arterial compliance state during the CVS formation in the acute period of head injury is obviously important for its prevention and timely diagnosis [25].

This study has shown that with developing CVS in the acute period (on the 2nd–third day after the accident) of a concomitant craniocerebral injury, the cAC significantly decreases as compared to the “normal” (reference) data.

One of the common causes of decreasing cAC is the development of a cytotoxic and vasogenic cerebral edema causing the compression of pial vessels [5, 26].

The indirect proof for this hypothesis is the identification of CT signs of cerebral edema in all 80 patients. However, as we have not carried out the blood-brain barrier breakdown study, we could not distinguish zones of ischemic injury and vasogenic edema.

Because of this limitation in our study, we have not been able to investigate the correlation between the change in cAC, the CVS development, and the secondary ischemia.

Another reason for the decreasing cAC may be a regional microvascular CVS due to the formation of a large amount of blood degradation products fallen into the subarachnoid cisterns. This effect is realized through the auto-oxidation of hemoglobin to methemoglobin with release of iron ions, which in turn cause the formation of superoxide radicals. Superoxide is supposed to cause change in the nitric oxide concentration and peroxide damage to the endothelium of pial vessels, thus causing the microvascular CVS development [27–29].

We have not used laser Doppler flowmetry (another limitation of the work); therefore, we could not directly examine the state of microvessels. However, taking into account that the “dopplerographic” CVS have coincided with the “angiographic” CVS in the most severe patients of the both groups, we assume that in this forth part of the patients (24% in first group, 23% in second group), the symptomatic character of a spasm took place, which was typical for its microvascular CVS [30].

Another possible reason for microvascular bed compression may be astrocytic endfeet swelling. Such swelling evolving in the first hours after injury may persist for a week thereafter [31, 32].

Finally, the compression of pial vessels both in a brain injury and in a vasospasm is associated with the dysfunction of pericytes—cells located in the basal pericapillary membrane. It was shown that the narrowing of arterioles and capillaries occurs because of the disturbance in the expression of endothelin-1 and pericytial receptors, types A and B, as well as the migration of over 40% of pericytes from the basal membrane [33–36].

All these factors as it has been shown above may result in the reduction of the total capillary bed lumen and accordingly to the decrease of cAC [37, 38]. It should be noted that the CVS formation after elimination of the brain compression by enveloped ICH changes even more the cAC value [39, 40].

It results in the sudden reduction of the number of functioning capillaries and in the decreasing cAC on the side of the vascular spasm and compression [4, 8]. The findings of our investigation may have the practical significance for optimizing the selection of individual regimens for brain edema therapy and vascular treatment with CVS available, which would prevent the development of cerebral perfusion disorders in patients with concomitant craniocerebral injury. Further studies are required to delineate the role of cAC in CVS.

Conclusion

The peripheral resistance of brain vessels in the cerebral vasospasm development in the acute period of a concomitant craniocerebral injury significantly increases as compared to the norm. The CVS formation in the perifocal zone after the removal of an enveloped

hematoma is followed by a significant decrease of the cAC as compared to the symmetric area of the opposite hemisphere.

Acknowledgments

D.B. was supported by NIH P20GM109089, DOD DM160142 and RSF No. 17-15-01263.

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Fig. 1.

The investigation system view. White arrow indicates a computer tomograph, blue arrow shows a TCD, red arrow shows ECG-ABP monitor, black arrow marks a syringe injector, and green arrow indicates cerebral oximeter

Table 1

Clinical outcome (Glasgow Outcome Score) of polytraumazed patients with CVS

	GOS 1 Good recovery	GOS 2 Moderate disability	GOS 3 Severe disability	GOS 4 Vegetative state	GOS 5 Death
Group 1	15	12	8	3	3
Group 2	16	9	8	3	3

Table 2

Comparison of the analyzed parameters

		CBF (mL/100 g/min)	ABPamp (mmHg)	CBV (cm³)	CAC (cm³/mmHg)
1	Group 1	33.1 ± 12.4	56.5 ± 15.6	2.3 ± 0.7	0.034 ± 0.029
2	Group 2 (ipsilateral sides with CVS)	28.3 ± 15.9	60.4 ± 21.6	2.1 ± 1.1	0.024 ± 0.027
3	Group 2 (ipsilateral sides without CVS)	37.4 ± 20.4	55.7 ± 29.2	2.4 ± 1.2	0.055 ± 0.049
4	Group 2 (contralateral sides with CVS)	29.6 ± 9.8	57.9 ± 17.9	2.2 ± 0.9	0.037 ± 0.029
	P (1–2)	0.183	0.563	0.510	0.097
	P (1–3)	0.312	0.892	0.764	0.172
	P (1–4)	0.215	0.892	0.351	0.34
	P (2–4)	0.695	0.616	0.282	0.003 ^a

CVS cerebral vasospasm, *CBF* cerebral blood flow, *ABPamp* amplitude of arterial blood pressure, *CBV* amplitude of regional cerebral blood volume oscillation, *cAC* cerebral arterial compliance

^aSignificant difference ($p < 0.01$)