Evaluation of cerebral hemodynamics by CT perfusion in patients with intracranial aneurysms

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Aims and objectives

The rupture of intracranial aneurysms causes about 50-70% of all nontraumatic subarachnoid hemorrhage, leading to disability in 46% of patients [2]. Meantime the frequency of asymptomatic aneurysms detected accidentally is several times greater. The appearance and the growth of the aneurysms are not only closely related to the changes of local hemodynamics, but, in contrary, have a direct impact on the speed and direction of blood flow as well as on the wall shear stress in the parent artery. It is proved that the blood flow and volume within the aneurysm and in the parent artery depend on the size of the aneurysmal sac [1,6,7]. The purpose of the study is to determine the effect of intracranial aneurysms on collateral blood flow and to assess the relationship of these changes on the size and location of aneurysm.
Methods and materials

Between 01.01.13 to 30.07.14 the CT-data of 41 patients with 47 intracranial aneurysms without rupture were evaluated, including six patients with multiple aneurysms.

CT-angiography and CT-perfusion were performed with slice thickness 5mm following reconstruction to 0.6mm. CT perfusion was performed with the slab thickness of 8cm, captured of the cerebral cortex up to the apex of parietal and frontal lobes, the area of interest had been revealed previously by native CT. Total iodinated contrast agent used to each patient, was 100 ml.

The data achieved were processed on a workstation using MIP and Volume rendering reconstructions. The CT perfusion data were analyzed using VPCT Neuro Stroke by computation parameters of cerebral blood flow CBF (ml / 100g/min), blood volume CBV (ml/100g), mean transit time MTT (s). Average values for normal gray matter of the brain for CBF = 39-55ml/100g/min, CBV=3-4ml/100g, MTT=2-4c.
Results

During the study none of the patients had been diagnosed with a stroke or postischemic changes.

30 of intracranial aneurysms with a maximum size up to 15mm were diagnosed, 18 of them of internal carotid artery, 6 - of middle cerebral artery, 4 - of anterior communicating artery, 2 - of basilar artery. No significant perfusion deviations in the majority of patients in this group were found (Fig. 1 on page 6, Fig. 2 on page 6) except 3 patients. In 1 patient with miliary aneurysm of ophthalmic segment of the right internal carotid artery revealed a local reduction of CBF in the right temporal lobe to 35ml/100g/min, with an unchanged CBV and increased MTT to 5c. In 2 patients with aneurysms of ophthalmic segment of the internal carotid artery up to 10-12mm a similar deficit of perfusion was diagnosed in the frontal and temporal lobes with a decrease of CBF to 32-35ml/100g/min, the unchanged CBV (up to 3.5ml /100g) and increased MTT to 5-6s.

10 intracranial aneurysms with a maximum size from 16mm to 25mm were revealed, among them 5 of the internal carotid artery, 2 - of middle cerebral artery, 1 - of anterior communicating artery, 1 - of posterior cerebral artery, 1 - of basilar artery. The majority of these patients (70%) observed different changes of perfusion parameters, primarily as widespread hypoperfusion in the temporal and occipital lobes in the area of blood supply of the parent artery of aneurysm, with reduced CBF on average 44-49%, symmetrical CBV (maximum 8% reduction on the side of aneurysm), increased MTT to 38-45% in comparison with the opposite site (Fig. 3 on page 7, Fig. 4 on page 8). Minimum absolute CBF values for these patients were 29ml/100g/min, CBV - 2ml /100g, maximum value of MTT was 7c, which may correspond to significant neurological deficit [9].

Giant aneurysms with a maximum size over 25mm were diagnosed in 7 patients, 6 patients revealed saccular aneurysms of various locations, with partially lumen thrombosis in 4 cases preserving contrast of about 1/3-1/2 volume. In 1 patient a fusiform nontrombosed aneurysm of the basilar artery was found.

Patients with partially thrombosed giant aneurysms presented predominantly local perfusion deficits, with decreasing CBF to 7-10ml /100g/ min, CBV to 1-1.5ml /100g and increasing MTT to 7-9s. These data are typical for deep irreversible ischemia [9].

In 3 patients with giant non-thrombosed aneurysms of the internal carotid and basilar arteries a vast area of hypoperfusion observed extending to white and gray matter, sometimes covering almost the entire hemisphere on the side of aneurysm (Fig. 6 on page 10, Fig. 7 on page 11), with a decrease in CBF to 15-29ml /100g/ min, CBV to 2-3ml /100 g, an increase in MTT to 7-9s, corresponding to deep chronic ischemia [9].

Comparing the anatomical features of revealed aneurysms and the prevalence and depth of brain hypoperfusion, it was found that the most important role in the developing of
hemodynamic deficit played the size and degree of lumen thrombosis, but not the precise location of the aneurysm (e.g., ophthalmic or communicating segment of internal carotid artery). Table 1 presents a summary of localization and extending of hypoperfusion, depending on size and degree of thrombosis of diagnosed aneurysms.

Table 1. Influence of size and degree of thrombosis of aneurysm on regional perfusion of the brain

<table>
<thead>
<tr>
<th>Size (mm)</th>
<th>Normal perfusion (N)</th>
<th>Perfusion pathology (N)</th>
<th>Predominantly local perfusion disturbances around the aneurysmal sac (N)</th>
<th>Widespread perfusion disturbances of the parent vessel (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>CBF (ml/100g/min)</td>
<td>CBV (ml/100g)</td>
</tr>
<tr>
<td>#3</td>
<td>8 (N=8)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4-15</td>
<td>19 (N=22)</td>
<td>3</td>
<td>27-35</td>
<td>3.3</td>
</tr>
<tr>
<td>16-25</td>
<td>3 (N=10)</td>
<td>7</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>#25</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>without thrombotic mass (N=3)</td>
<td>4 (N=4)</td>
<td>7-17</td>
<td>1-1.2</td>
<td>6-8</td>
</tr>
</tbody>
</table>
Fig. 1: CT-angiography of the patient with aneurysm of communicative segment of right carotid artery

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Fig. 2: CT perfusion of the same patient as in the Fig.1. No significant changes of perfusion are detected, #BF, CBV and MTT are symmetrical

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**Fig. 3:** CT-angiography of patient with 2 aneurysms of communicative segments of internal carotid arteries (right up to 7mm, marked with the red arrow, left up to 20mm, marked with the white arrow).

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**Fig. 4:** the same patient as in Fig.3. CT perfusion data demonstrate the normal perfusion of the right hemisphere (accordingly to the aneurysm 7mm in diameter), but the moderate hypoperfusion of the left temporal and occipital lobes (accordingly to the aneurysm 20mm in diameter) with a decrease of CBF up to 49% and CBV up to 19% in comparison with the right side (short red arrow).

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**Fig. 5:** CT-angiography of the patient with the giant fusiform aneurysm of basilar artery (white arrow)

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Fig. 6: the same patient as in Fig.5. CT perfusion data reveal the large area of deep hypoperfusion extending to both occipital lobes, right temporal lobe, brainstem and both thalamus (short red arrows) with a decrease of CBF to 27ml/100g/min and CBV to 2ml/100g. According to the native CT no sings of acute ischemia or posts ischemic changes were detected.

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Fig. 7: the same as in the Fig.6

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Conclusion

Every time after detection of intracranial aneurysm without rupture the neurosurgeon faces a dilemma: whether the patient requires surgery or can be observed in the dynamics. In the most cases the decision is based on the assessment of so called risk factors for aneurysm rupture, including localization, size and shape of the aneurysmal sac, its pulsation and the relationship of the aneurysm with the parent vessel [2]. That's why the majority of studies in this field are devoted to the "hemodynamic behavior" of aneurysm itself, especially to arterial wall shear stress and blood flow changes, and practically ignore such an important aspect as the effect of aneurysm on the blood flow in the parent artery and the changes of the blood supply to the relevant area of cerebral cortex. Simultaneously the standard CT or MRI examination of the brain and intracranial arteries doesn't include any methods of perfusion evaluation [4,5], so that the state of the microcirculation of the brain remains unclear.

The relationship of vascular lesions and peripheral(capillary) blood flow are discussed from the early 1970s, but the accent was made on the possibility of arterial embolism [8], so the impact of intracranial aneurysms on cerebral hemodynamics is limited to the development of local ischemia at the level of "mass effect" due to compression of giant aneurysm or brain ischemia of the microembolic origin, most often in patients with fusiform and partially thrombosed giant aneurysms of the internal carotid and middle cerebral arteries [4,8,10]. The incidence of thromboembolic cerebral arteries in patients with aneurysms is still being debated and is approximately 5-59% [8].

In our study different types of perfusion disorders were diagnosed in patients with 17 from 47 aneurysms, including 70% of aneurysms of 16-25mm in diameter and 100% giant aneurysms (more than 25mm in diameter), which corresponds to a high risk of thromboembolism in the literature. However the chronic ischemic and post-ischemic changes were detected only in 7 patients with aneurysms (41%) and predominantly (in 6 cases) were localized around the aneurysmal sac due to "mass effect". No characteristic CT signs of embologenic ischemia in patients of this group were observed [9]. On the contrary, the decrease in CBF and increase in MTT in the cerebral cortex in all patients was diffuse, relatively uniform, without clear boundaries, which is also unusual for embolism. Hypoperfusion observed predominantly in the case of aneurysms larger than 16 mm, regardless of location and shape. Prevalence and severity of hypoperfusion depended only on the degree of aneurysm lumen thrombosis. Partially thrombosed aneurysms with less functioning lumen caused mainly local perfusion lesions, possibly related to "mass effect", while non-thrombosed aneurysms were accompanied by extensive hypoperfusion in the blood supply area of the parent artery. All cases of decrease in CBF were compensated by well developed collaterals, as indicated by
symmetrical CBV on the average 2-3ml/100g, so that no ischemic changes of that zone were diagnosed by native CT relatively.

The above described changes in patients with intracranial aneurysms more than 16mm in diameter force to look for another cause for the development of hypoperfusion in the area of parent artery blood supply except embolism. In studies of Tateshima S. (2007), D. Sforza (2009), JR Cebral (2011), it is emphasized that the rate of blood flow within the aneurysmal sac dramatically slows down and is only about 14-17% of the parent artery flow [7,10,11]. But the flow velocity in a parent vessel before aneurysm exceeds the speed in the same vessel after aneurysm. So perhaps every large aneurysm within each cardiac cycle adds an extra slower and turbulent blood portion from the aneurysmal sac to the parent vessel. This may lead to the change in the volume blood flow in the artery after aneurysm and the disturbance of normal adaptive response of the peripheral capillars, resulting eventually to the development of brain hypoperfusion. Probably long-existing impaired blood flow at the level of the aneurysm becomes more common, and includes several mechanisms of rebuilding microcirculation, though the hypothesis requires further investigation and confirmation. Nevertheless, the presence of neurologically significant microcirculatory disorders of the brain in patients with intracranial aneurysms is of great importance, as it increases the risk of ischemic changes not only during surgery and in the postoperative period, but prior to surgery (for example, in patients with arterial hypertension) [3]. Revealed violations of perfusion in patients with intracranial aneurysms may lead to the expansion of the indications for surgery and require greater use of neuroprotection during surgery and in the postoperative period.
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