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Case Report

Uncommon Cerebral Vasospasms after Neurosurgical Operations: A Diagnostic Challenge. A Case Report

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Abstract

Background: Even neurosurgical standard operations, not giving reason for cerebral Vasospasms (*VS*) per se, may involve symptomatic spasms with subsequent disseminated subcortical infarcts away from the operation site and should give reason to consider vasospasms.

Methods & Clinical Setting: Occurrence of subcortical, small spot-shaped infarcts offside the operation area drew suspicion on symptomatic cerebral *VS* Transcranial Doppler-sonography (TCD), cranial Computed Tomography Angiography (CTA), CT perfusion (CTP) and Magnetic Resonance Tomography (MRI) angiography (MRA) and perfusion (MRP) proved vasospasms and standard vasospasmolytic therapy was induced.

Results: Probably, ipsilateral sequential *VS* of the proximal and distal Medial Cerebral Artery (MCA) led to unexpected infarcts. Pattern of infarcts were initially disseminated sub-cortical evolving to segmental lesions. Even though infarcts established could not be changed under vasospasmolytic therapy, there did not occur any further infarcts in vulnerable brain-at-risk areas identified by perfusion CT. For the first patient, the associated neurological deficits receded partly, the second one recovered completely.

Conclusion: Subcortical, progressive infarcts after neurosurgical interventions could be induced by vasospasms of the large brain-supplying vessels without an obvious trigger. Disseminated "embolic-like" infarcts should give reason to consider cerebral *VS* for saving at least brain-at-risk areas.

Introduction

Even following neurosurgical interventions usually not implicating a risk for vasospasm, perfusion-disturbing *vs* of brain-supplying arteries with corresponding brain ischemia may occur.

This complication is of major importance for the patient concerned and a challenge for the treating physician, since the conventional framework conditions for *vs* are missing [1-6], but diagnosis and potential therapy in case of imminent brain infarcts should take place immediately to save brain-at-risk-areas. We would like to raise awareness of this rare and serious but treatable complication.

Subjects and Methods

Patient 1: A male patient, 46 years, no pre-existing diseases, active smoker. Rupture and hemorrhage by cavernoma in right Sylvian fissure bleeding 4 weeks ago, no remaining neurological deficit (Figure 1a). For microsurgical resection, the fissure was widely opened for avoiding the use of a brain retractor and minimization of manipulation on the superficial sylvian branches of MCA. The M3 branches were exposed, covered by gauze sponges, the proximal segments were not been visualized. The surrounding brain tissue of cavernoma was slightly brownish. A minor bleeding of the cavernoma whilst mobilization and en-bloc resection occurred, but blood clots in the fissure removed by irrigation. A local administrable vasodilatator was not used.

On the first Post-Operative Day (POD), CT revealed edema around the operation situs with consecutive midline shift by approx. 3 mm and minimal residual blood film in the fissure. The patient was awake, however revealed hemiplegia left sided, Level of Strength 0 (LOS 0) and dysphasia [7], [Adnex: BMRC]. Due to the distinctive perifocal edema the patient obtained Mannitol 15%, 4x125 ml/24hrs.

On the second POD, an additional reduction of vigilance (GCS9) caused non-invasive respiratory support. Unfortunately, only on the third POD a new CT was performed, that revealed a segmental brain infarction (4 x 2,5 cm) and disseminated small sub-cortical infarcts (up to 1x2 cm in diameter) off the operation area (Figure 1b and 1c).

The infarct pattern gave reason to consider vasospasm-induced infarcts and TCD revealed relevant spasms of the ipsilateral proximal MCA with a pathological mean flow of 160 cm/sec. A CTA and CTP confirmed vasospasm-suspicious stenoses, revealing a punctum maximum in the right posterior M2 branch, serious caliber irregularities in the remaining MCA, a diminished Cerebral Blood Volume (CBV) corresponding to the situs and an overlapping delayed Time To Drain (TTD) in the posterior MCA territory (Figure 2a-b), but no abnormalities in other vascular territories. Suspected *VS* led to anti-vasospastic therapy using nimodipine intravenously (10ml/h/2mg/h) and an induced hypertension following spasm therapy after aneurysmal Subarachnoid Hemorrhaghe (aSAH) [5], due to somnolence no oral application. Mannitol stopped. Treatment covered by emergency indication.

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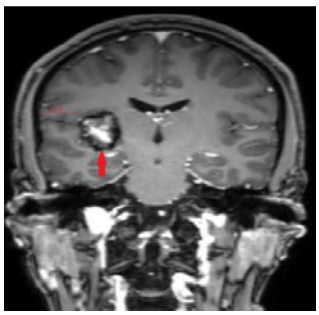

Figure 1a: Preoperative MRI image (T1w after applying contrast agent, coronary layering), 1. Patient, Cavernoma (arrow).

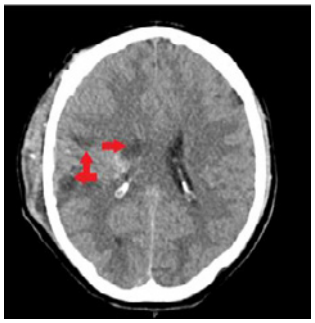

Figure 1b: POD: Subcortical, embolic type hypodensitiesand diffuse edema on the right side following removal of cavernoma in the native CCT (3rd POD, axial layering, brain window).

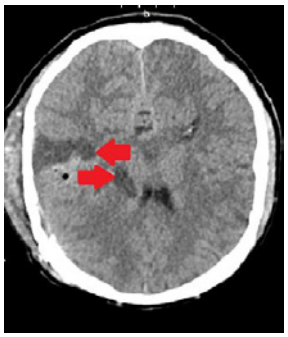

Figure 1c: 3rd POD, Segmential infarktion, (axial layering, brain window).

24hrs later, an improvement of vigilance and speech disorder appeared, in the course, plegia of the left leg decreased during several days to paresis (LOS 3), dysarthria remitted completely, unfortunately, the left arm remained a high-grade paresis with a spastic bending position (LOS 1). After normalization of flow parameters, Nimodipine was tapered over eight days. Importantly, there did not occur any additional in brain-at-risk-areas.

Patient 2: A male patient, 35 years, spontaneous membranous, chronic subdural hematoma (cSDH) on the right with midline shift of 7 mm to the left (Figure 3a). Due to structure of the cSDH, a bone flap was performed for removing reachable parts of the membranous hematoma. The arachnoid layer was untouched, no contact of blood and cortex vessels occurred. Prior diseases: Common-ALL 2010, condition after two therapy phases with Tacrolimus in 2011.

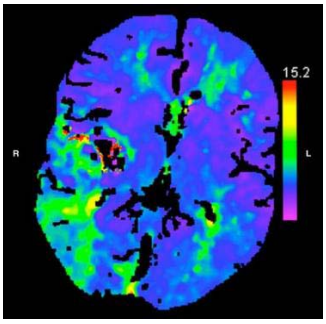

Figure 2a: CT-P with delay of TTD in the MCA on the right side exceeding the operation area (axial layering, Time to Drain).

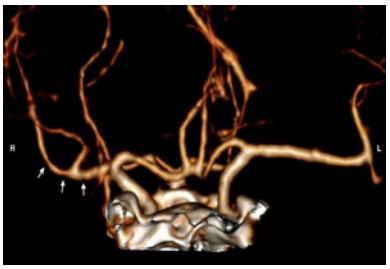

Figure 2b: Caliber irregularities and stenosis *VS* of MCA on the right side in the CTA (Volume Rendering Technology reconstruction, VRT, arrows). No Disturbances on left side and in territory of anterior cerebral artery.

The patient was extubated soon after operation and did not reveal any neurological findings. CT control on the first POD showed a residual hematoma beyond the area of trepanation, thin fresh layers of blood in the area of the trepanation defect with an equally less severe perifocal edema and regressive midline shift. Moreover, however, there were several small, spotty infarction-like lesions in the supply area of the MCA on the right side (Figure 3b), 1st POD). During the following night, the vigilant patient first developed a fluctuating paresis on the left progressing in to a high-grade hemiparesis (LOS2) on the second POD with hemihypesthesia. Immediately induced CT showed a typical segment infarct in the territory of the posterior M2 branch of the right MCA (Figure 3c).

The infarction pattern suggested symptomatic *VS*. TCD on the same day confirmed *VS* with pathological mean flows of 165 cm/s at the first segment (M1) and 150 cm/s in the peripheral branches of the right MCA. The remaining brain arteries revealed normal flow rates. Imaging diagnostics (CTA and CTP) (Figure 4a-b) substantiated *VS*-suspicious tenoses in the M1 and M2 section of the right MCA with overlapping perfusion disorders by a delay of Mean Transit Time (MTT) and TTD in the entire posterior MCA territory (Figure 4a-b), but no impact on other vascular trunks. A vasospasmolytic therapy with nimodipine 6x60mg orally and induced hypertension begun. The vigilant patient agreed to treatment. During treatment, the paresis solved completely in the following days. Cranial MRI, MRA and MRP on the ninth POD revealed the known infarct, but no additional ischemia (not shown). After normalization of TCD measurements, nimodipine was tapered over a week. The patient was discharged free of symptoms.

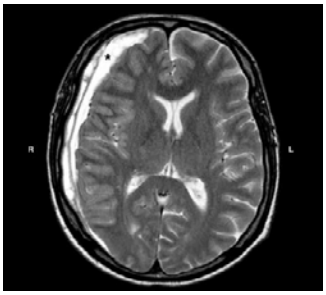

Figure 3a: Preoperative MRI image (T2w, axial layering) 2. Pat, subdural hematoma on the right side (star).

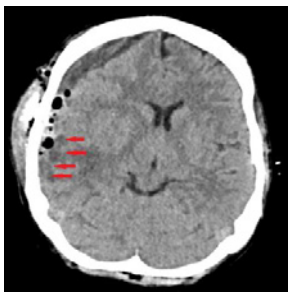

Figure 3b: 2nd POD. Narrow residual hematoma and subcortical, disseminated (4 arrows, axial layering, brain window).

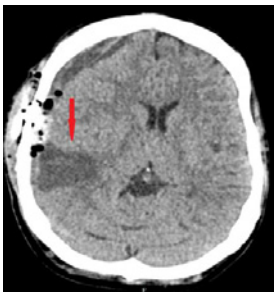

Figure 3c: Segment infarct (single arrow) on the right side native CCT (axial layering, brain window).

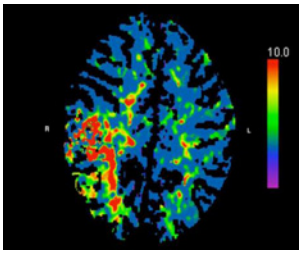

Figure 4a: CTP with delay of MTT in the MCA on the right side exceeding the infarct region.

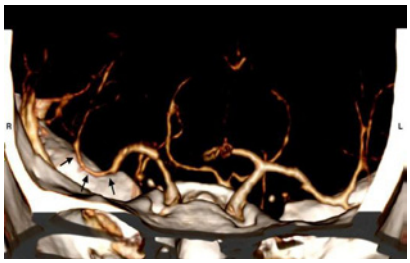

Figure 4b: CTA (VRT reconstruction) with caliber irregularities and stenosis of the MCA on the right side (arrows) *VS*.

Discussion

Vasospasm of the large brain-supplying arteries frequently occur following aSAH where blood around the vascular trunks accumulates. Delayed occurrence of *VS* is characteristic [2,5,6] *VS* of the large vessels following resection of Meningioma, skull base and pituitary tumors [1,8] is well-known, possibly explained by manipulation on vessels. Beyond mechanical mechanisms [3,9,10], triggers for *VS* may be genetically disposition or for example thermic, pharmaceutical and inflammatory and oxidative stress induced irritations of the vessels [11-17]. Vasospasm also exist in case of the call Fleming syndrome, familial hemiplegic migraine, Prinz-Metals's Angina and cellular hyper-contractility syndrome [11,12,15]. Moreover, cigarette smoking and the surgical access evoking manipulations may provoke a spasm [18,19].

Regarding the first patient, during exposure, a minimal manipulation of M3 branches of MCA occurred, the previous bleeding of cavernoma may induce a hyper-contractility of vessels and the thin blood layer remained postoperatively, as well smoking, may have a vague impact. Each individual factor on its own could probably not have triggered a spasm, but possibly the combination of these factors could have. Unfortunately, we considered this possibility too late, and heavy impact neurological deficits were fixed and immutable. At least we were able to prevent additional areas of the brain that were disturbed by perfusion from developing into manifest infarcts. In the second patients, the etiology of the non-traumatic membranous subdural hematoma in a young man is obscure and there is no bibliographic reference for extended spasms of proximal vessels following surgery of a chronic subdural hematoma, except one case report dealing with an infectious one, possibly leading to of an inflammatory cascade and a consecutive vasoconstriction [20]. In this case, only a condition after Tacrolimus therapy was available as a potential trigger. But, we were alert as soon as multiple embolic-type small infarctions showed up and a fluctuating clinical course appeared. Probably, we started diagnostics and spasmolytic therapy early enough, saving affected but still vital brain parenchyma, and the patient is free of a neurological deficit.

Conclusion

Disseminated, etiologically unclear, small subcortical infarcts should give reason to consider the existence of cerebral vasospasms and to induce corresponding diagnostics.

Fast diagnosis is of decisive clinical importance for the patients for saving still vital but endangered parenchyma ("brain-at-risk") from the development of manifest infarcts. Diagnosis and corresponding treatment in the first case were delayed, neurological deficits remained. As far as the second patient is concerned, treatment was clinically effective or initiated fast enough, because we were sensitized to this issue and he recovered completely.

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Adnex:

BMRC scale for muscle strength.

Memorandum no. 45. London: Her Majesty's Stationery Office; 1976

Grade 0: No contraction.

Grade 1: Flicker or trace contraction.

Grade 2: Active movement, with gravity eliminated.

Grade 3: Active movement against gravity.

Grade 4: Active movement against gravity and resistance.

Grade 5: Normal power.