Case report

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EARLY VASOSPASM FOLLOWING SPONTANEOUS SUBARACHNOID HEMORRHAGE: A CASE REPORT

Slavko Živković[1], Jovan Ilić[1], Vesna Stokanović[2], Radisav Mitić[1], Bojan Stanojević[1], Marija Djordjević[3]

Ultra early cerebral vasospasm (UEAV) occurs within 48 hours after aneurysm rupture, can be verified by angiographic imaging and usually has a poor outcome. The presented patient had clinical symptomatic UEAV, detected within a few hours of bleeding from anterior communicating artery aneurysm. The neurosurgical team decided on late operation, more precisely 18 days after the rupture of the aneurysm, because the patient was then giving clinical signs of cerebral vasospasm relief. Right pterygoid craniotomy was performed. The aneurysm was excluded from circulation by clipping without any incident. When treating patients with clinical signs of UEAV, the ruptured aneurysm site should be verified by angiographic imaging as soon as possible, while the appropriate treatment modality should be carefully and duly considered.

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Key words: *intracranial vasospasm, anterior communicating artery aneurysm, surgical clip*

[1]University Clinical Center Niš, Department of Neurosurgery, Niš, Serbia
[2]University Clinical Center Niš, Department of Radiology, Niš, Serbia
[3]University of Niš, Faculty of Medicine, Niš, Serbia

Contact: Jovan Ilić
112/12 Vizantijski Blvd., 18000 Niš, Serbia
E-mail: jovanilic94@gmail.com

Introduction

Cerebral vasospasm (CVS) after spontaneous subarachnoid hemorrhage (SAH) occurs in 20 to 30% of patients and represents a life-threatening condition (1). It involves a temporary contraction of the arteries, which results in a focal or diffuse reduction in caliber of blood vessels, and usually occurs 3 to 14 days after SAH (2). Diagnosis is made by Transcranial Doppler ultrasound (TCD), Magnetic Resonance angiography (MRA) or Computed tomography angiography (CTA) (3). The clinical presentation of a patient with symptomatic CVS depends on the affected intradural or surface brain artery and causes cerebral ischemia, which can be marked as delayed ischemic deficit (4).

On the other hand, there is much less data in the literature on early CVS, which occurs within 48 hours after aneurysm rupture, can be verified by angiographic imaging and usually has a poor outcome (5, 6).

In this case report, we present a patient with clinical symptomatic ultra-early vasospasm (UEAV), detected within a few hours of bleeding from the anterior communicating artery (ACoA) aneurysm, with an emphasis on therapeutic dilemmas.

Case report

A 30-year-old female patient was admitted to the emergency department due to spontaneous SAH. The symptoms occurred in the evening, on the day of admission, followed by a neck stiffness and severe headache, which she described as the worst in her life, followed by the loss of consciousness and emesis. The measured arterial pressure was 180/120 mmHg, and we obtained heteroanamnestic data that she did not suffer from hypertension and did not take any oral medications by then. Initial examination at the emergency department was performed by a neurosurgeon, who indicated CTA scan of the brain. Clinical examination showed that the patient was somnolent but conscious (Glasgow Coma Scale Score of 14), oriented to time, place and person, with neck stiffness (Hunt & Hess Score of 1) and without any recorded gross neurological deficit. CTA recorded SAH sagittal frontally, which filled basal cisterns with blood and expanded to the prepontine cistern, as well as an anteriorly directed

aneurysm of the ACoA, measuring 4 mm in diameter.

Therefore, she was hospitalized at the Neurosurgical department, where she was treated with hyperdynamic therapy for CVS, a calcium channel blocker nimodipine (Nimotop® in the dose of 60 mg administered orally every 4 hours), antiedematous and prophylactic anticonvulsant therapy. Moreover, Digital subtraction angiography (DSA) was performed the next morning (Figure 1), which showed a saccular aneurysm with a maximal diameter of 3.8x5 mm on ACoA, with the neck 3.1 mm wide. The aneurysmal dome, directed anteriorly, had a smaller aneurysm with a diameter of 3x2.6 mm, and another aneurysm was originating from the neck of the aneurysm, measuring 2 mm in diameter. Furthermore, at the origin of the right pericallosal artery, there was another sacullar aneurysm with a wide neck, measuring 3.2x2 mm in diameter, directed posteriorly. Vasospasm of both anterior cerebral arteries (ACA) was verified. Initially, the patient responded well to the therapy. Control CT angiography was performed 11 days after DSA, given that the patient's mental state had deteriorated.

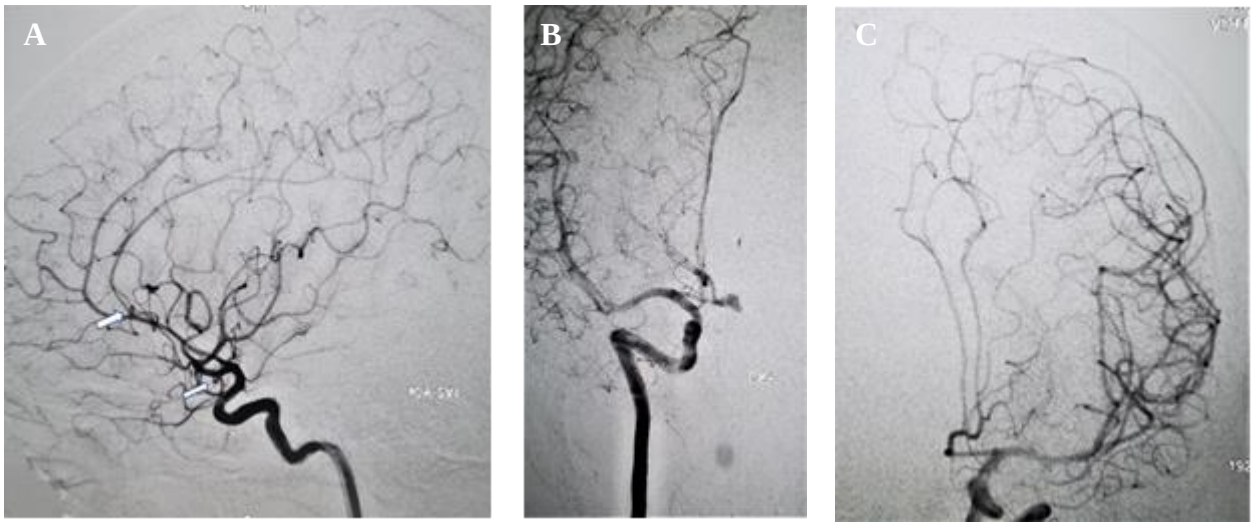

Figure 1. DSA finding (A, B, C) confirms previously seen aneurysms at ACoA and at the origin of pericallosal artery on the right. The CVS of both ACA is evident.

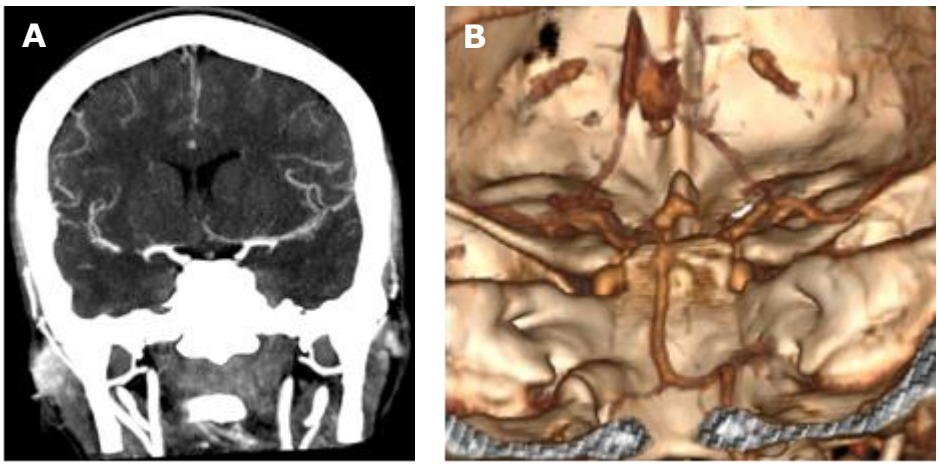

Figure 2. CTA (coronal plane- A; VR image- B) shows an ACoA aneurysm.
Bilaterally, the A1 segments of ACA are graceful, while the A2 segments are filiform in the proximal part, but distally without visible contrast, which indicates spasm of the aneurysm.

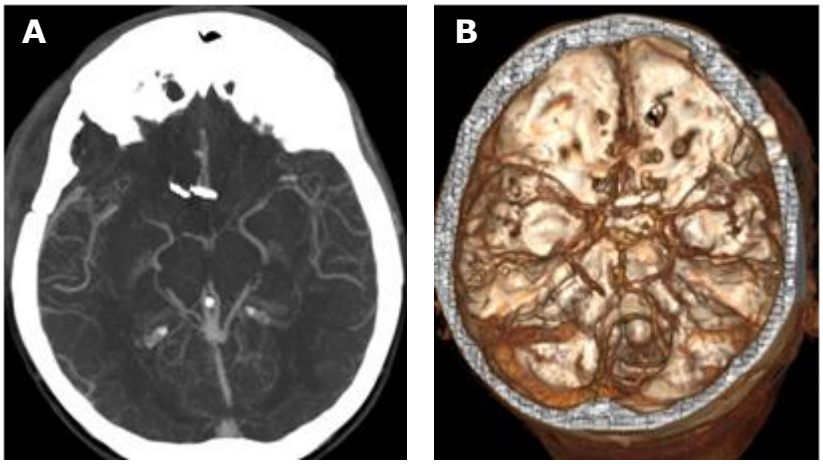

Figure 3. CT angiography (axial plane-A; VR imaging- B) shows surgical clips, which are placed correctly. Both ACA are transient and of a larger caliber in comparison with the previous CTA, but still in vasospasm, which indicates a relief of a CVS.

Compared with the initial CTA, regression of the amount of SAH was recorded and CVS of both ACA was still present. Since there was no re-hemorrhage and the CVS still persisted (Figure 2), the surgery was postponed. The patient was stabilized again after applied therapy.

After 7 days, due to the good clinical state of the patient, the neurosurgical council decided that the aneurysm should be treated surgically. Right pterygoid craniotomy was performed. The vasospasm of both ACA was verified. The aneurysm was excluded from the circulation by clipping without any incident. The bony lid was fixed back with surgical sutures and returned to the appropriate place and the soft tissues was sewn in layers. The postoperative recovery went smoothly. The patient was somnolent and without any gross neurological deficits. The further clinical course was complicated by the patient's mental state alteration and insipid diabetes. Due to the continued worsening of the patient's state to subcoma, as well as the respiratory insufficiency, the patients was transferred to the intensive care unit, when she was intubated and placed on mechanical ventilation in a BiPAP mode. The control CTA was performed (Figure 3), on which CVS of both ACA was still present. Six days after intubation, the patient became hypotensive, bradycardic, which resulted in cardiac arrest and death.

Discussion

Fawaz et al. reported UEAV in 4.6% of SAH patients, which were associated with younger age, poor neurological state during the first examination, as well as the presence of sentinel bleeding. Moreover, UEAV was linked with an increased chance of experiencing delayed cerebral ischemia (DCI), but was not related to poor neurological outcome after treatment (7). In contrast, Baldwin et al. found UEAV in 10% of SAH patients, which was not associated with symptomatic DCI (8). Furthermore, Qureshi et al. reported a significant relation between UEAV, symptomatic CVS and poor neurological outcome (9). Our patient, on the other hand, did not have a poor clinical grade on admission and during the first day of hospitalization, during which the vasospasm was verified by CTA and DSA, but she experienced cerebral ischemia and eventually a lethal outcome.

The etiology of UEAV has not been fully elucidated due to insufficient investigation. Predictive factors for the development of UEAV may be patient's poor clinical grade, hypertension, blood sodium level greater than 138 mM, high score (3 and 4) on the Fisher scale, larger dimensions of the ruptured aneurysm, and the previous history of SAH (7, 10). Some of the potential pathophysiological factors may be elevated intracranial pressure, inflammatory response to injury as well as the formation of subarachnoid blood clots. Moreover, key elements after subarachnoid clot formation are an increased concentration of nitric oxide (NO) scavengers, such as reactive oxygen species (ROS) and oxyhemoglobin, as well as increase in concentration of transmitters (serotonin, thromboxane A2, Endothelin-1 and thrombin), which are responsible for CVS (11). The patient we treated did not suffer from hypertension, did not have previous history of SAH, and had a lower score (2) on the Fisher CT scale.

Danura et al. consider that the incidence of CVS is more frequent after surgical clipping of a ruptured cerebral aneurysm. Furthermore, in a series of patients examined, they found that the CVS rate was 30% after clipping and 14% after endovascular treatment in patients with SAH, due to manipulation with arteries during surgery (12).

The open microsurgery has certain disadvantages, which could theoretically lead to CVS, such as the presence of new blood and its breakdown products during surgery in the subarachnoid space and the consequent formation of free radicals and lipid peroxides, as well as vasospasm after manipulation with arteries. The results of previously

conducted studies have shown a better outcome for patients and less often CVS after coiling compared to open microsurgery. These results could be explained by the fact that patients who were treated with coiling had a better neurological grade preoperatively (13-15). In our case, we opted for an open microsurgical approach in the patient, because of the possibility of irrigating the basal cisterns and removing blood from the subarachnoid space, guided by the experiences of some other authors (16, 17).

Ebeling et al. considered the right timing of surgery for the ruptured aneurysm, and pleaded that the patient should undergo a surgical treatment within 48-72 hours after rupture or within the 10-14 days following the rupture of an aneurysm (18). However, Ebeling et al. believe that the TCD examination in CVS could enable us to decide on the best and individual timing for surgery while daily examining the patient (18). Mahaney et al. Considered possible causes for unfavourable postsurgical outcomes in patients, which were operated on 3-6 days after rupture of an aneurysm. They explained it with secondary injuries due to impaired cerebral autoregulation. Furthermore, this pathophysiological mechanism has been described by some other authors as well (19). Patients with a high score of Fisher's scale (3 and 4) are considered to have better outcomes, if they undergo surgery during the first and second day after the aneurysm rupture, but the outcome is considered to be worse if they undergo surgery later. In contrast, patients with lower Fisher scale scores (1 and 2) experience more favorable outcomes if they undergo surgery early and later, while outcomes are less favorable with intermediate surgery. Consequently, Mahaney et al. Reported that during the follow-up examinations 90 days after surgery, patients who had undergone surgery during the first 2 days or after 6 days of aneurysm rupture had better outcomes (19). In accordance with these results, our neurosurgical team decided on late surgery, more precisely 18 days after the rupture of the aneurysm, because the patient was then giving clinical signs of CVS relief.

Conclusion

When treating a patient with clinical signs of UEAV, the ruptured aneurysm site should be verified by TCD, CTA or DSA as soon as possible, while the appropriate treatment modality should be carefully and duly considered.

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RANI VAZOSPAZAM NAKON SPONTANOG SUBARAHNOIDALNOG KRVARENJA: PRIKAZ SLUČAJA

Slavko Živković[1], Jovan Ilić[1], Vesna Stokanović[2], Radisav Mitić[1], Bojan Stanojević[1], Marija Đorđević[3]

[1]Univerzitetski klinički centar Niš, Klinika za neurohirurgiju, Niš, Srbija
[2]Univerzitetski klinički centar Niš, Klinika za radiologiju, Niš, Srbija
[3]Univerzitet u Nišu, Medicinski fakultet, Niš, Srbija

Kontakt: Jovan Ilić
Vizantijski bulevar 112/12, 18000 Niš, Srbija
E-mail: jovanilic94@gmail.com

Rani cerebralni vazospazam javlja se u roku od 48 sati nakon rupture aneurizme, a može se potvrditi angiografskim snimanjem i obično ima loš ishod. Prikazani bolesnik imao je klinički simptomatski rani cerebralni vazospazam, otkriven u roku od nekoliko sati nakon krvarenja iz aneurizme prednje komunikantne arterije. Neurohirurški tim odlučio se za kasniju operaciju, tačnije 18 dana nakon rupture aneurizme, jer je bolesnik tada davao kliničke znake popuštanja cerebralnog vazospazma. Urađena je desna pterigoidna kraniotomija. Aneurizma je isključena iz cirkulacije pomoću klipa, bez ikakvih incidenata. Prilikom lečenja bolesnika koji imaju kliničke znake ranog cerebralnog vazospazma, mesto rupture aneurizme treba, što je pre moguće, verifikovati angiografskim snimanjem, dok treba pažljivo i propisno razmotriti odgovarajući modalitet lečenja.

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Ključne reči: *cerebralni vazospazam, aneurizma prednje komunikantne arterije, hirurški klip*