

Recurrent cerebral vasospasm after aneurysmal subarachnoid hemorrhage- A temporal “Marching Pattern” of progression

Keywords: cerebral vasospasm, Marching cerebral vasospasm, angiographic pattern

Abbreviations: CVS, cerebral vasospasm; aSAH, aneurysmal subarachnoid hemorrhage; CT, computed tomograph; ICA, internal carotid artery; MCA, middle cerebral artery; M1, proximal segment of middle cerebral artery; A1, proximal segment of anterior cerebral artery; M2, M3, distal segments of middle cerebral artery; A2, A3, distal segments of anterior cerebral artery; IA, intraarterial

Introduction

Cerebral vasospasm (CVS) following aneurysmal SAH is an important cause of morbidity and mortality and its pathogenetic mechanisms remain controversial. The available literature on CVS describes the angiographic patterns of evolution/resolution over time and the diffuse/focal nature of involvement of the cerebral vessels. However, the temporal pattern of evolution and mode of spread along the arterial tree in a given patient has been infrequently described.

We report a patient of aneurysmal SAH in the post-clipped status who developed recurrent episodes of vasospasm over a period of 2 weeks requiring repeated intraarterial spasmolysis. Temporally the CVS involved a new distal portion of the arterial tree each time spreading in a spasmodic squeezing pattern along the vessel length, a phenomenon we named as “Marching cerebral vasospasm.”

We discuss its possible pathogenetic mechanisms in light of available literature.

Case report

A 43-year old female without any relevant medical history was hospitalized after severe headache, neck pain, and self-limiting cramp of both upper extremities. At admission, she was awake and oriented without any focal neurological deficits. Brain computed tomography (CT) showed diffuse subarachnoid hemorrhage in all cisterns without intracerebral or intraventricular hemorrhage (Fisher grade 3, BNI grade 4) nor accompanying hydrocephalus. The CT angiography showed an anterior communicating artery aneurysm which was clipped six hours after the initial hemorrhage without any complications. A cisternal drain was left behind to drain cerebrospinal fluid. This drain was removed four days after surgery. Postoperative she was observed in our neurointensive care unit according to standard protocol. Due to the anatomy of the cranial vault, no transcranial Doppler could be performed.

At post hemorrhage day seven, the patient suddenly developed neurological deficits, including a decreased Glasgow coma score (GCS) E4M5V2, global aphasia and, a diffuse paresis of the right arm with normal leg function. She was diagnosed to have suffered symptomatic vasospasm. After a CT brain to rule out major infarcts

Volume 10 Issue 6 - 2020

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Received: August 12, 2020 | **Published:** December 18, 2020

a diagnostic cerebral angiogram was done along with intraarterial vasospasmolysis with nimodipine. After initial improvement, she again developed the same focal deficits, with now also a leg paresis, within twelve hours after spasmolysis which prompted us to perform a second emergency spasmolysis. She was extubated the next day and neurological examination showed global aphasia with improved right-sided hemiparesis. Some hours later she developed a status epilepticus for which she needed reintubation and sedation. Invasive multimodality monitoring (ICP, PtiO₂, and microdialysis) was placed right frontal. The PtiO₂ and microdialysis showed evidence for progressive functional deterioration and after interdisciplinary discussion, we decided to perform a third and even fourth spasmolysis over the next 4 days. These successive treatments lead to improved multimodality parameters.

The serial angiographic images are depicted in figures 1 to 4. We can appreciate that over a period of 6 days in the anterior circulation the CVS has progressed from terminal ICA, and M1M2 segments and spreading distally over the arterial tree to involve the M3 M4, A3 A4 segments, and distal PCAs. Each time after lysis the spasm got relieved and when it recurred it did not appear in a segment in which it existed but was more pronounced distally in a new segment of the same vessel as if the vessel is getting squeezed distally. We call this phenomenon a “Marching Pattern”.

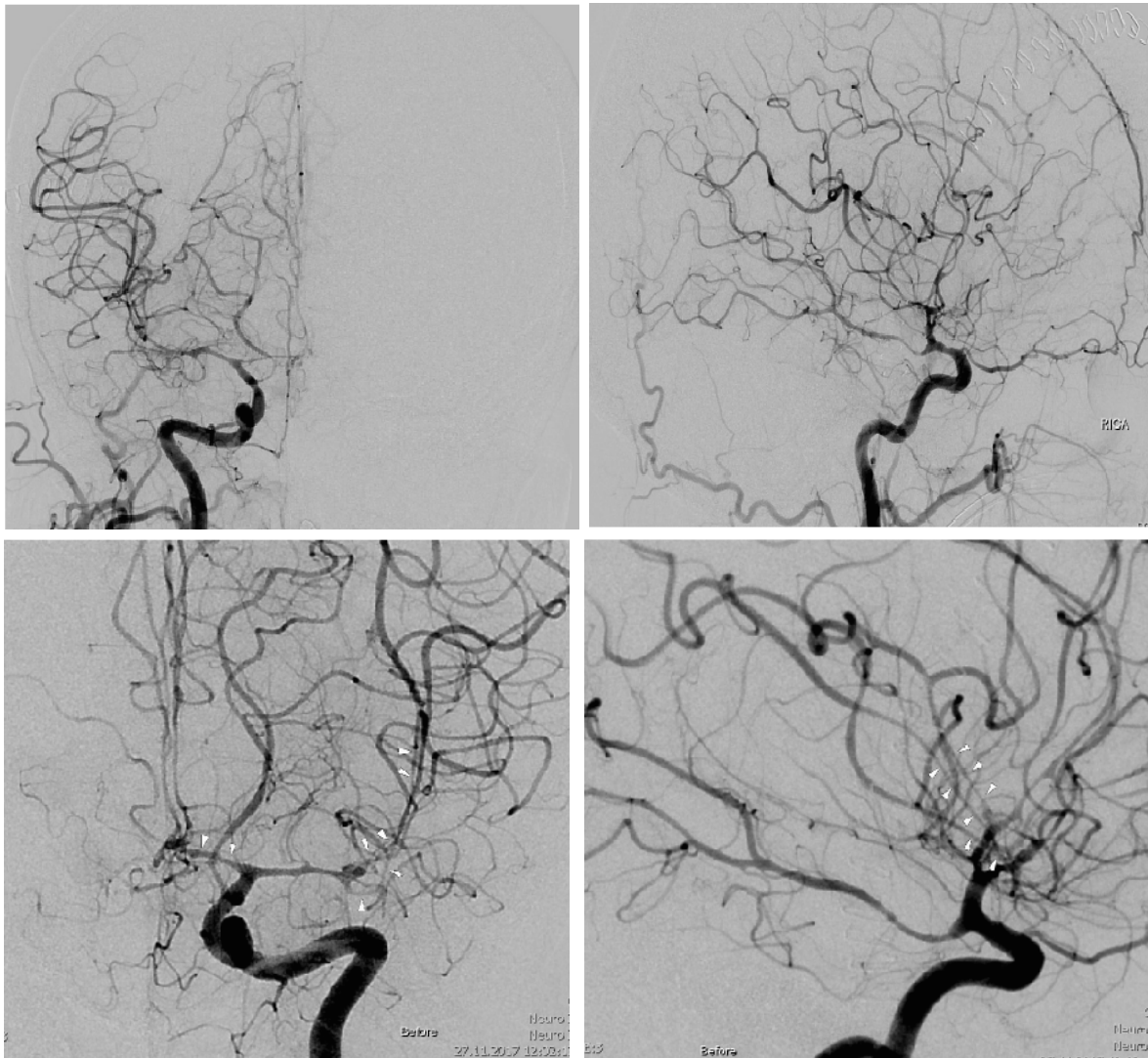


Figure 1 1st episode of CVS . Distal ICA, AI, M1, M2 and proximal PCOM in spasm, both sides.

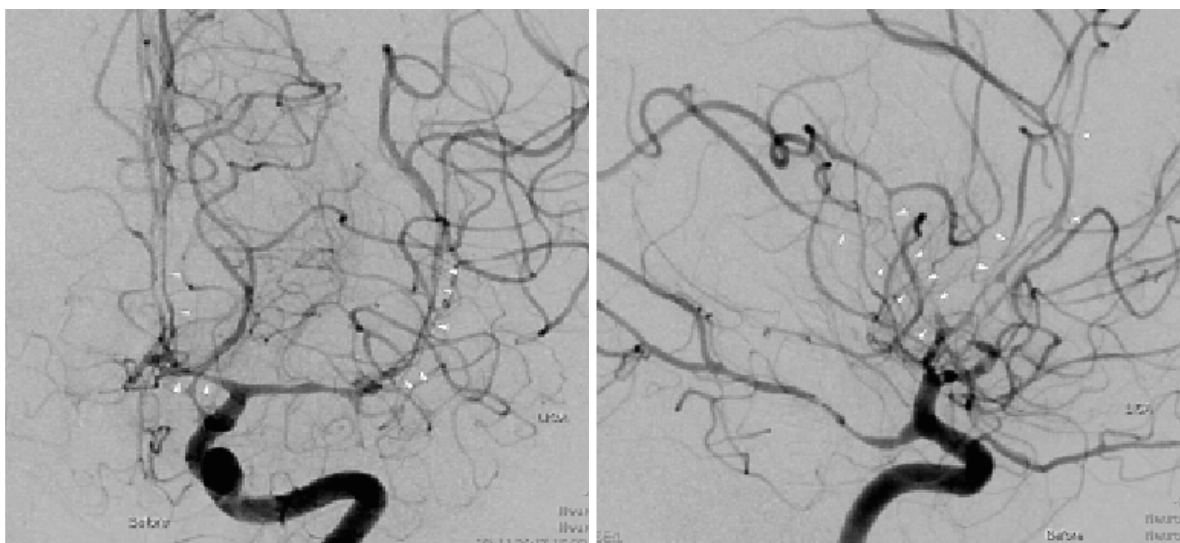


Figure 2 2nd episode of CVS- distal ICA and proximal PCOM are normal, CVS involves M1A1.

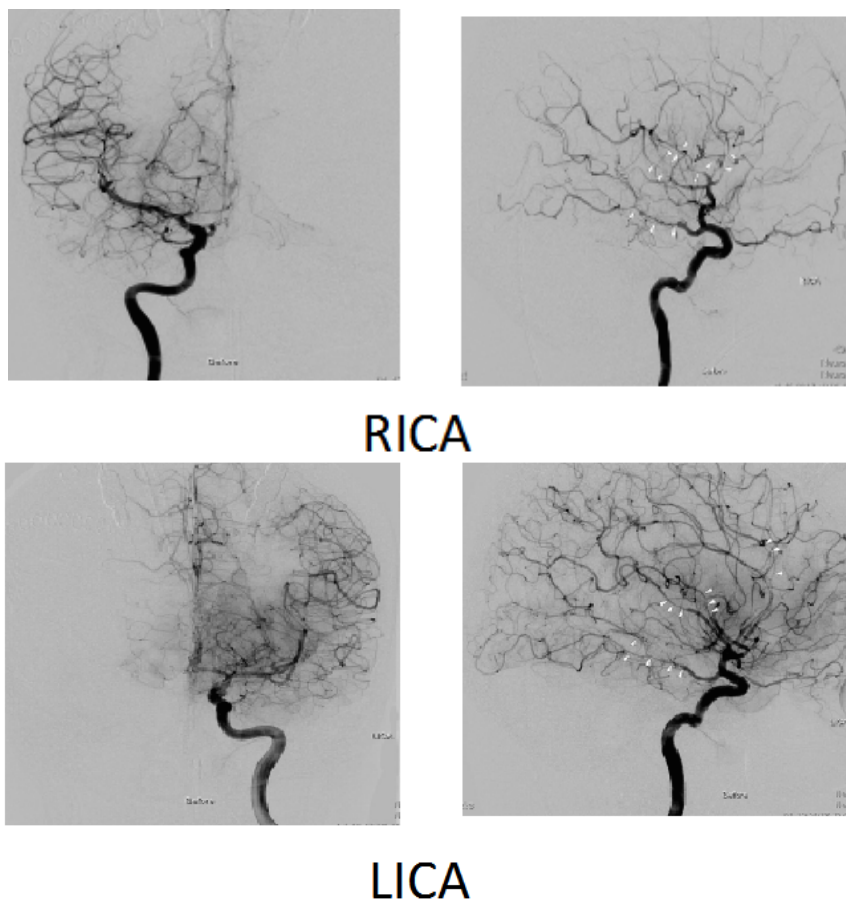


Figure 3 3rd episode of CVS. Proximal ICA, M1 looks normal. CVS seen in distal M2/M3.

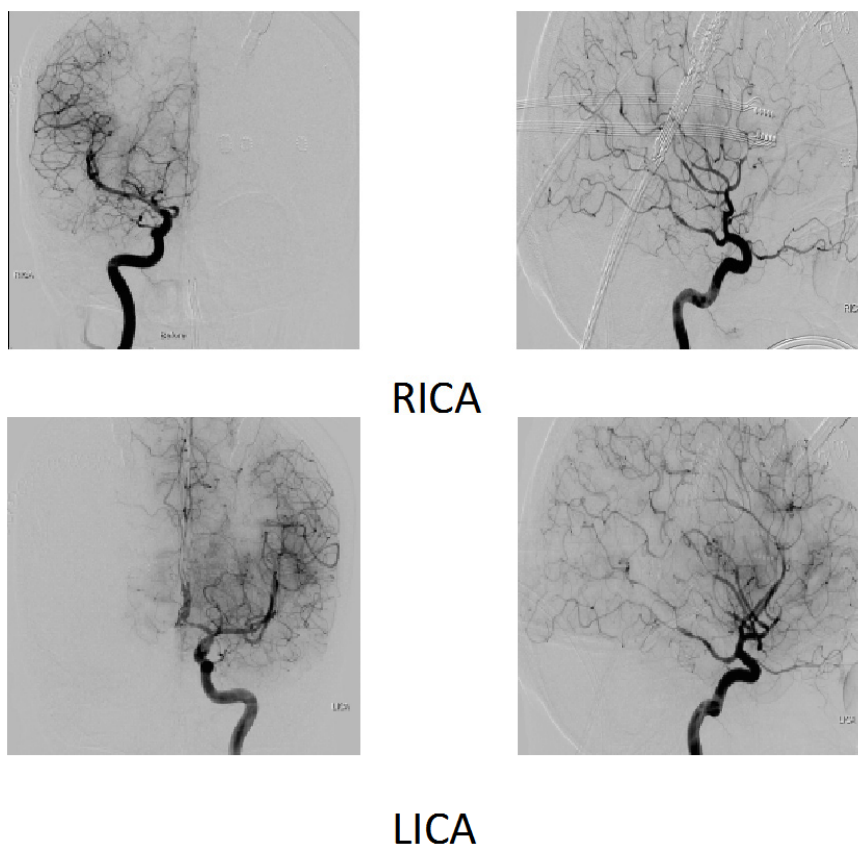


Figure 4 4th episode of CVS. Involves the M3, M4 and A3, A4 segments.

Discussion

CVS following aneurysmal subarachnoid hemorrhage (aSAH), is an important cause of significant morbidity and mortality.¹ It is consistently the leading cause of poor outcome and death, adversely affecting more than one in five of all patients who have suffered SAH.²

Worthwhile improvements in the available treatments have been made during the last 3 decades, resulting in a definite reduction of morbidity and mortality attributed to it from 25–30% in the 1970s to 15–20% in the 1980s, to 5–10% currently.³ Nonetheless, there remains an absence of consistently efficacious and ubiquitously applied preventive and therapeutic interventions for this cause of significant mortality and morbidity, even now. This probably reflects the fact that despite ongoing research efforts the underlying pathogenic mechanisms remain inconclusive.⁴

The angiographic descriptions of vasospasm after SAH and its progression/regression over time have been described as early as 1951.⁵ Various studies document the onset and resolution with serial angiograms. Vasospasm has its onset in man about Day 3 after subarachnoid hemorrhage, is maximal at Days 6 to 8, and is gone by Day 12.⁶

Sano et al described types of CVS and divided them into Type 1- extensive diffuse, Type 2 Multi segmental, and Type 3 Focal and found that surgical intervention in Type 1 had the worst results.⁷ In most of the above studies, onset and severity of spasm over a period of time were discussed but none documented that in a given patient there could be a defined pattern of how the vasospasm would spread over the intracranial arterial tree.

Jones et al described patterns of evolution of CVS temporally in a cohort of coiled and clipped patients.⁸ In their study they found perianeurysmal spasm affected all subjects. However, whereas spasm remained largely confined in the clipped group, patients who underwent coiling developed stepwise progression distally over time. In one of their illustrative cases, spasms involving bilateral A1, A2, M1, and M2 segments on post-bleed day 5 moves distally by day 7 to the posterior circulation; the right M1 and M2 segments had improved by then. By post-bleed day 15, much of the anterior circulation spasm had improved whereas the basilar and posterior cerebral arteries appear more affected. Their report does not talk of spasm spreading distally on the same-named vessels.

Contrary to this in our patient, the CVS was recurrent despite intra-arterial spasmolysis, and it demonstrated downstream spread into the same vascular territory but with the release of the spasm in the proximal portion of the same vessel in a squeezing fashion and that is why we call this a Marching Pattern. In one of the widely accepted theories of vasospasm, the volume of subarachnoid blood on presentation is an important predictor of angiographic vasospasm. The clot size and density and its clearance rate are important in determining the risk of vasospasm and delayed ischemic neuro deficits after the SAH.⁹

Subarachnoid hematoma evacuation during surgery was initially reported to reduce the incidence of vasospasm.¹⁰ However, further studies have shown that the method of aneurysm treatment does not affect clot clearance after aneurysmal subarachnoid hemorrhage.¹¹

In a review article on the pathogenesis of vasospasm Kolias et al have shown that after aneurysmal SAH, a multifaceted cascade of events is initiated, ultimately leading to vasospasm.¹² Breakdown products of blood in the subarachnoid space probably are the triggering factor, while calcium-dependent and independent vasoconstriction is

taking place during CVS. Lipid peroxides, an imbalance between endothelium-derived vasoconstrictor and vasodilator substances, nitric oxide toxicity, arachidonic acid metabolites, inflammatory cascades, disruption of neuronal mechanisms that regulate vascular tone, endothelial proliferation, and apoptosis, are among those factors that, acting through interconnected pathways, result in the development of vasospasm.

In yet another study it was shown that endothelial glycocalyx injury occurs in SAH, and might contribute to delayed cerebral ischemia by regulating cerebral micro thrombosis and delayed neuroinflammation.¹³ In an article titled "anatomical observations of the subarachnoid cisterns of the brain during surgery" Yasargil et al have observed that although often considered as anatomically distinct compartments, the basal cisterns are, in fact, only separated from each other by a trabeculated porous wall with various sized openings.¹⁴ These apertures can become plugged and partially or obliterated after subarachnoid hemorrhage and in cases of basal tumor. The arachnoid fibers and membranes are noted to be regularly thicker and tougher where the arteries pass through the trabeculated wall from one cisternal compartment to another.

Neurogenic factors, although not thought to be important in the normal physiological regulation of cerebral arteries, may become important under pathological conditions such as acute SAH. The presence of a rich plexus of adrenergic fibers within the adventitial layer of the pial vasculature has been amply documented. Further, there is evidence of differential innervation of nerve endings along the arterial tree and also between the anterior and posterior circulation. Also, the level of circulating catecholamines usually increases after SAH. It is possible, then, that this increase in circulating catecholamines, associated with abnormal sensitivity of the cerebral vessels to catecholamines and a differential innervation, is a factor in the complex genesis and progression of vasospasm.^{15,16} There is also evidence of sphenopalatine ganglion stimulation decreasing vasospasm and increasing cerebral blood flow after SAH in monkeys. This was associated with the opening of the blood-brain barrier.¹⁷ This also signifies the role of neurogenic mechanisms.

Conclusion

In light of the above literature review, we hypothesize that due to thickened arachnoidal bands near major vessels the SAH remained confined near proximal arteries initially and later the blood spread through opened cisterns (due to surgery) and also through naturally existing pores between cisterns. The vasospasm also followed the spread of the blood with its toxic metabolites as mentioned above and was influenced in some way by the neural innervations. Hence the "Marching pattern."

Acknowledgments

None.

Conflicts of interest

The authors declare no conflict of interest.

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