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Case Report

Prolonged Cerebral Hyperemia after Aneurysmal Subarachnoid Hemorrhage: Report of Two Cases

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Abstract

Prolonged cerebral hyperemia is an underrecognized complication of aneurysmal subarachnoid hemorrhage (SAH), characterized by a sustained increase in cerebral blood flow (CBF) in the absence of increased metabolic demand. This entity may be associated with cerebral edema, seizures, and neurological deterioration, and requires a diagnostic and therapeutic approach different from vasospasm or delayed cerebral ischemia. We present two cases of patients with SAH who developed sustained hyperemia and headache, without evidence of vasospasm, and with good response to outpatient management with indomethacin.

Keywords: *Subarachnoid Hemorrhage, Cerebral Hyperemia, Autoregulation, Intracranial Pressure, Metabolic Dysfunction.*

Introduction

Aneurysmal subarachnoid hemorrhage (SAH) is a severe neurological emergency, responsible for high morbidity and mortality. The most extensively studied complications, such as vasospasm and delayed cerebral ischemia (DCI), have historically guided therapeutic strategies. However, a subgroup of patients present with a less recognized phenomenon: prolonged cerebral hyperemia, characterized by a sustained increase in cerebral blood flow (CBF) without a corresponding rise in energy demand. This state may lead to cerebral edema, seizures, and signs of increased intracranial pressure (ICP), and requires a radically different therapeutic approach from vasospasm. [1,2]

Case Reports

Case 1

An 18-year-old female patient, with no relevant past medical history, presented with sudden onset of “thunderclap” headache associated with vomiting at home, prompting admission to our institution. Brain CT revealed subarachnoid hemorrhage with ventricular involvement, FISHER grade 4, secondary to a dissecting aneurysm of the distal branch of the right posterior choroidal artery. Digital subtraction angiography was performed by the endovascular neurosurgery team, and the aneurysm was successfully embolized with Magic Glue. Preventive vasospasm treatment with nimodipine 50 mg every 6 hours was initiated.

Five days after symptom onset, transcranial Doppler showed signs of vasospasm. Hemodynamic support with vasopressors (norepinephrine) was administered to maintain adequate cerebral perfusion pressures (CPP).

The patient improved in velocity readings, with no vasospasm, but with diffuse hyperemia in all arteries of the circle of Willis explored, along with intermittent headache that improved with indomethacin 75 mg as rescue therapy. Control brain CT showed complete resorption of the hemorrhage and aneurysm exclusion.

The patient was discharged in good general condition, without neurological deficits, but with intermittent holocranial headache of moderate intensity, worsened in the supine position, improved on standing, and relieved by indomethacin 75 mg. During outpatient follow-up, hyperemia signs persisted on Doppler, progressively improving until velocities normalized for the patient's age and complete headache resolution was observed at 6 months post-discharge.

Case 2

A 43-year-old female with no significant medical history suffered a moderate traumatic brain injury in a motor vehicle accident, presenting to the emergency department with global amnesia. Brain CT revealed subarachnoid hemorrhage, FISHER grade 1, with angiography showing no evidence of aneurysms. The patient improved clinically from the amnesia but developed intermittent headaches, with no new hemorrhage detected on follow-up CT scans.

Outpatient transcranial ultrasound revealed a hyperemia pattern, for which indomethacin 75 mg was prescribed. The patient experienced symptomatic improvement, with Doppler velocities normalizing at 3 months after discharge.

Discussion

Sustained cerebral hyperemia after SAH is an underdiagnosed phenomenon, occurring in up to 10–20% of patients according to studies using Xenon-CT and brain PET. Its recognition is crucial, since conventional treatment for vasospasm may be counterproductive. Pathophysiologically, it is associated with neuroinflammatory activation, mitochondrial dysfunction, and production of vasodilators such as nitric oxide and prostacyclins, leading to inappropriate increases in CBF. This alteration is favored by loss of autoregulation, common in severe SAH, where cerebral flow becomes passively dependent on cerebral perfusion pressure (CPP).

Prolonged cerebral hyperemia has been defined as a sustained state (≥72 h) by transcranial Doppler, with elevated mean flow velocities in MCA and a Lindegaard Index (MCA/ICA) < 3 and low/normal pulsatility index (suggesting hyperemia and not vasospasm). It can also be defined using other diagnostic methods such as CT/MR perfusion or PET/SPECT when available. Some authors recommend using two distinct methods for confirmation [1–6].

The literature supports that SAH alters autoregulation and promotes flow–metabolism uncoupling through nitric oxide/endothelin-1, neuroinflammation, and mitochondrial dysfunction; these mechanisms relate to DCI and sustained hyperemia phenotypes. The decoupling between flow and metabolism generates a paradoxical condition: hyperperfusion in a hypoactive brain, predisposing to vasogenic edema and seizures. The clinical case presented reflects this situation, with no evidence of ischemia but a hemodynamic pattern of hyperemia.

From a diagnostic point of view, TCD is a useful tool to detect diffuse increases in velocity with low pulsatility index, suggesting hyperemia without focal vasospasm. Advanced monitoring by tissue oxygen pressure ($PbtO_2$), microdialysis, or evaluation of optimal CPP (CPP_{opt}) can confirm dysregulation and guide therapy [3,5,6,7,9].

Clinically, hyperemia does not respond favorably to induced hypertension, which may even worsen the condition. The inadequate use of vasopressors may increase perfusion pressure in a brain unable to autoregulate, precipitating cerebral edema [8].

Treatment is based on three pillars: control of triggering factors (fever, hypercapnia, hyperglycemia, seizures), personalized CPP-directed monitoring, and symptomatic management. In the presented case, outpatient management with indomethacin was effective.

Indomethacin, a non-steroidal anti-inflammatory drug with cerebral vasoconstrictive effects, has studies in trauma patients and one case in SAH showing that intravenous boluses of indomethacin (30–50 mg) significantly reduced ICP and improved CPP. This drug inhibits prostaglandins, produces dose-dependent cerebral vasoconstriction, reduces basal cerebral blood flow (≈20–30%), and attenuates CO_2 reactivity, thus decreasing ICP in hyperemic contexts. This has been confirmed in animal models, although at high doses it may induce hypoperfusion and ischemia.

Adverse effects such as reversible cerebral vasoconstriction syndrome (RCVS) have also been reported after its use in spontaneous cortical hemorrhage. In our case, the association with clinical improvement was observational; a larger number of cases would be necessary to determine a causal relationship. However, we have not found reports of its use orally and on an outpatient basis for sustained post-SAH hyperemia, probably due to the low detection of sustained hyperemia in peripheral follow-up of these patients [9,12].

Conclusion

Prolonged cerebral hyperemia after SAH represents a pathophysiological entity distinct from vasospasm, which must be considered during outpatient follow-up. Its recognition allows treatment to be directed toward individualized brain protection. Despite its clinical relevance, prospective studies are required to define specific therapeutic strategies and validate the use of agents such as indomethacin in this context.

Conflicts of Interest

During the preparation of this work the authors used Chat GPT 5 in order to translate the paper from Spanish to English. After using this tool, the authors reviewed and edited the content as needed and takes full responsibility for the content of the published article.

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