

MIGRAINE HISTORY IN PATIENTS WITH ANEURYSMAL SUBARACHNOID HEMORRHAGE DEVELOPING CEREBRAL VASOSPASM AND DELAYED CEREBRAL ISCHEMIA: A CASE SERIES

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Objectives. to describe the characteristics of patients with a history of migraine developing cerebral vasospasm and/or delayed cerebral ischemia (DCI) following aneurismal subarachnoid haemorrhage (aSAH).

Background. Cerebral vasospasm and DCI are major secondary vascular complications of aSAH. Migraine, a recognized cerebrovascular risk factor, may share pathophysiological mechanisms with vasospasm and DCI.

Methods. we retrospectively reviewed consecutive patients admitted to the Stroke Unit of Tor Vergata University Hospital with a diagnosis of aSAH between January 2022 and December 2025. Among these, we identified migraineurs who developed cerebral vasospasm and/or DCI. Collected data included demographics, vascular risk factors, aneurismal and aSAH features, clinical severity at admission with WFNS and Hunt-Hess scales, radiological severity assessed using the modified Fisher scale (mFS). Cerebral vasospasm was assessed by transcranial doppler ultrasound; patients with symptoms consistent with vasospasm underwent digital subtraction angiography.

Results. among 96 patients, 5 migraineurs (4 episodic, 1 chronic; none with aura; all female, mean age 35.2 years) developed vasospasm and/or DCI. Smoking was the only vascular risk factor identified. At admission, all patients had WFNS and Hunt-Hess grade 1-2; 3 patients presented with mFS grade 1-2 and 2 with grade 4. Cerebral vasospasm was documented in 4 patients, 3 of whom symptomatic. Three patients fulfilled criteria for DCI, including 1 without evidence of macrovascular vasospasm. All patients achieved favorable 3-month outcomes.

Results. Neurovascular complications occurred despite mild clinical presentation and low radiological burden in most of selected patients.Although current evidence doesn't support a clear association between migraine and cerebral vasospasm after aSAH [1], previous studies have suggested a higher occurrence of DCI among migraineurs [2]. Metabolic and vascular dysfunction of the migrainous brain may represent a potential vulnerability factor for secondary neurovascular complications following aSAH, possibly through shared mechanisms [3] involving altered cerebrovascular reactivity, endothelial dysfunction, neuroinflammation and cortical hyperexcitability. Notably, the occurrence of DCI in 1 patient without angiographic vasospasm further supports the multifactorial pathophysiology of DCI.

Conclusions. It could be interesting to investigate with prospective studies whether migraine history could independently increase the susceptibility to secondary vascular complications post-aSAH, especially in patients with a high migraine burden.