

Migraine and Intracerebral hemorrhage

A [metaanalysis](#) found an increased risk of [hemorrhagic stroke](#) (HS) in subjects with migraine ¹⁾, although potential sources of heterogeneity can be found among the studies included in that analysis. Most studies did not distinguish between [intracerebral hemorrhage](#) (ICH) and [subarachnoid hemorrhage](#) (SAH) when assessing the outcomes and some of them did not report the outcomes separately for MA and migraine without aura (MO). In addition, in the available studies little consideration was given to migraine activity and duration ²⁾.

The case-control study published in The Journal of Headache and Pain by Gaist and colleagues adds important data gathered from general practice to the link between migraine and vascular diseases. In that study, data from 1,797 subjects with ICH and 1,340 subjects with SAH from a large epidemiological dataset, The Health Improvement Network (THIN), were reviewed and frequency-matched with control subjects for sex, age (± 1 year), and calendar year of diagnosis. After adjustment for sex, age, calendar year, smoking, alcohol, body mass index, hypertension, previous cerebrovascular disease, oral contraceptive use, and health services utilization, the authors did not find an increased risk of overall HS or of ICH or SAH in subjects with migraine compared with non-migraineurs. Analysis according to migraine type showed that neither MA nor MO were associated with an increased risk of HS, ICH or SAH.

Only subjects with a long history (≥ 20 years) of migraine had an increased risk of ICH compared to control subjects, and even they did not show an increased risk of SAH ³⁾.

It should be noted that migraine and HS have some vascular comorbidities, particularly hypertension, which is one of the most important risk factors for HS; however, the great majority of the studies investigating the association between migraine and HS adjusted their statistical models for hypertension. Future studies should be planned to verify the presence of any vascular vulnerability in migraine in order to explain the increased vascular risk of migraineurs and to develop new therapies acting on both migraine and the associated vascular risk ⁴⁾.

Case series

1990

Three women with well-documented migraine associated with intracerebral hemorrhage are described. In each case, migraine headaches began during adulthood. Unusually severe and protracted headache heralded the onset of fixed neurological deficits associated with lobar intracerebral hemorrhage. Striking carotid artery tenderness was characteristic. Except for a history of migraine, no cause for intracerebral hemorrhage could be established. In each case arteriography showed extensive spasm of the appropriate extracranial or intracranial artery. Surgical pathology following evacuation of two hematomata demonstrated signs of vessel wall necrosis associated with subacute inflammatory changes. Vasospasm associated with severe migraine attacks may result in ischemia of intracranial vessel walls, leading to necrosis and subsequent vessel rupture when perfusion pressure is restored ⁵⁾.

Case reports

1989

A patient with classic migraine who developed an intracranial hemorrhage 36 h after the onset of a migraine attack and had a good recovery over a period of six days. Including this patient there were five cases till 1989, all females, with this complication of migraine. It is postulated that the condition is fairly common and under reported ⁶⁾.

1993

A case of intracerebral hemorrhage which occurred during a migraine in a 62 year-old woman who had migraine since the age of 20. The patient was normotensive. The angiograms were normal. Cerebral hemorrhage in migraine appears to be very rare: we found 6 reported cases that were compared with our own. The hypothesis of recirculation in an infarcted tissue and the possible role of dissecting aneurysms are examined ⁷⁾.

1998

A normotensive migrainous woman experienced multiple intracerebral hemorrhages. No drug abuse was recorded, but bleeding occurred after the use of several antimigrainous drugs. Angiography revealed severe vasospasm of both anterior cerebral arteries. Subsequent brain MRI and MRI angiography failed to show any vascular abnormalities. Echocardiography and an exhaustive biological evaluation ruled out an alternative condition favoring intracerebral hemorrhage. Multiple intracerebral hemorrhages in migraine may exceptionally be related to vasospasm leading to arterial wall injury. Intracranial arterial vasospasm could be precipitated by excessive vasoactive therapy ⁸⁾

1999

Raabe and Krug report a case of [bilateral basal ganglia hemorrhage](#) which occurred during an attack of classical [migraine](#). The patient had a history of migraine associated with [aura](#) of neurological deficit for 10 years and a history of arterial [hypertension](#) for 20 years, which was treated with [propranolol](#). [Intracerebral hemorrhage](#) during an attack of migraine is very rare and up to now the existence of true migraine-induced intracerebral haemorrhage has been controversial. The case of bilateral occurrence of the haemorrhages supports the theory of the existence of migraine-induced damage of the wall of intraparenchymal vessels during vasoconstriction and focal ischaemia at the beginning of a migraine attack. Subsequent vessel rupture may occur during the following period of increased cerebral blood flow especially with coexisting arterial hypertension ⁹⁾.

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