Anosmia After Perimesencephalic Nonaneurysmal Hemorrhage

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Background and Purpose—Anosmia frequently occurs after aneurysmal subarachnoid hemorrhage not only after clipping, but also after endovascular coiling. Thus, at least in part, anosmia is caused by the hemorrhage itself and not only by surgical treatment. However, it is unknown whether anosmia is related to rupture of the aneurysm with sudden increase in intracranial pressure or to the presence of blood in the basal cisterns. Therefore, we studied the prevalence of anosmia in patients with nonaneurysmal perimesencephalic hemorrhage.

Methods—We included all patients admitted to our hospital with perimesencephalic hemorrhage between 1983 and 2005. Patients were interviewed with a structured questionnaire. We calculated the proportion of patients with anosmia with corresponding 95% CIs.

Results—Nine of 148 patients (6.1%; 95% CI, 2.8% to 11%) had noticed anosmia shortly after the perimesencephalic hemorrhage. In 2, the anosmia had disappeared after 8 to 12 weeks; in the other 7, it still persisted after a mean period of follow-up of 9 years.

Conclusions—Anosmia occurs in one of every 16 patients with perimesencephalic hemorrhage, which is lower than previously reported rates after coiling in patients with subarachnoid hemorrhage but higher than rates after coiling for unruptured aneurysms. These data suggest that blood in the vicinity of the olfactory nerves plays a role in the development of anosmia.

Key Words: anosmia • perimesencephalic • subarachnoid hemorrhage

Patients with an aneurysmal subarachnoid hemorrhage often report a loss of smell not only after clipping, but also after coiling and this has an important impact on quality of life.1 Anosmia can occur after intracranial surgery1,2 and after subarachnoid hemorrhage, it often has been attributed to the operative treatment of the aneurysm. Recent studies have shown that anosmia also occurs in patients with subarachnoid hemorrhage and endovascular occlusion of the aneurysm. Thus, at least in part, the anosmia is caused by the hemorrhage itself.

Although the cause of perimesencephalic hemorrhage has not yet been identified, the invariably good clinical condition at onset, the often more gradual onset of headache, and the localized nature of the blood on CT all argue against spurting of blood under arterial pressure and favor venous oozing of blood.3 A venous source is further supported by the normal arteriograms and often abnormal findings in venous drainage in patients with perimesencephalic hemorrhage.4 We therefore studied the prevalence of anosmia in patients with perimesencephalic hemorrhage.

Methods

Patients

From a prospectively collected database of patients admitted to the University Medical Center Utrecht with subarachnoid hemorrhage, we retrieved data on all patients admitted between 1983 and 2005 who met the following criteria: CT scan performed within 72 hours after the onset of the headache showing a perimesencephalic pattern of hemorrhage5 and absence of a saccular aneurysm on CT angiography or conventional angiography. Hydrocephalus was defined as a bicaudate index exceeding the upper limit for age.6 The study was approved by the Medical Ethics Committee of our hospital.

Follow-Up

We contacted the general practitioner of eligible patients to find out if the patient was still alive. Subsequently, we sent a letter to all patients who were still alive to announce a telephone call. If the patient’s phone number was unknown, we sent a letter asking the patient to contact us. Patients were interviewed by telephone with a standardized, previously used questionnaire.1 The questionnaire comprised questions on whether patients had noticed any degree of loss of smell or taste after the hemorrhage. Patients were interviewed by telephone with a standardized, previously used questionnaire.1 The study was approved by the Medical Ethics Committee of our hospital.

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16 patients; 13 patients had died during the follow-up period, one patient was a UK resident, another was imprisoned, and one had emigrated. Therefore, the cohort consisted of 148 patients, 61 (41.2%) of whom were women. The mean age at time of the hemorrhage was 54 years (range, 24 to 81 years). The Table describes baseline characteristics of the patients. None of the patients had symptoms or signs of Parkinson disease or had developed Parkinson disease during follow-up. Mean follow-up was 7.5 years (range, 1 to 23 years). None of the patients had severe head trauma or intracranial surgery during follow-up.

Nine (5 women and 4 men) of the 148 patients (6.1%; 95% CI, 2.8% to 11%) had noticed a loss of smell after the hemorrhage; all had noticed the loss of smell already during the clinical course, which typically lasted a few days. Two reported a complete recovery after 8 to 12 weeks. The mean period of follow-up was 9 years. None of the 19 patients with hydrocephalus had anosmia; the corresponding relative risk is 0.0 (95% CI, 0.0 to 3.5).

Discussion

In patients with a perimesencephalic nonaneurysmal hemorrhage, the prevalence of anosmia is one in 16. The prevalence in patients with endovascular treatment of the aneurysm after subarachnoid hemorrhage is one in 6,7 but in patients with endovascular treatment of an unruptured aneurysm, the prevalence of anosmia is negligible.8 Therefore, the anosmia should be attributed, at least in part, to the hemorrhage itself and is related not only to the suddenly increased intracranial pressure from a ruptured aneurysm, but also to the presence of blood in the basal cisterns. This finding that subarachnoid blood is related to anosmia is further supported by case reports of patients with hemosiderosis of the central nervous system and anosmia.9,10

Like in a previous study, we found no relation between anosmia and risk factors such as age, sex, and hydrocephalus, but the power of our study was limited.

The study population was retrieved from a large cohort of patients admitted since 1983 and none of the patients was lost. The data were collected retrospectively and the prevalence of anosmia might therefore have been underestimated, because patients might have forgotten about the temporary loss of smell. Moreover, the diagnosis of anosmia was based on an interview by telephone and was not verified with smelling tests. Nevertheless, all 9 patients were confident that the anosmia had not been present before the hemorrhage and had started shortly after the perimesencephalic hemorrhage.

Our cohort of patients with perimesencephalic hemorrhage is a good setting to study whether the occurrence of anosmia should be attributed to aneurysmal rupture or to the presence of blood in the basal cisterns. Our data suggest that blood in the vicinity of the olfactory nerves contributes to the development of anosmia.

Disclosures

None.

References

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