Smoking and aneurysm rupture risk factor

Tobacco smoking is one of the most important risk factors for the formation of intracranial aneurysms and for aneurysmal subarachnoid hemorrhages [1) 2) 3)].

A dose-response relationship has been noted between intensity and duration of smoking consumption and increased risk of intracranial aneurysm rupture. As smoking is modifiable, this finding is important to managing patients with IAs to quit or reduce smoking prior to life-threatening subarachnoid hemorrhage [4)].

Current cigarette smoking, smoking intensity, and smoking duration are significantly associated with ruptured IAs at presentation. However, the significantly increased risk persists after smoking cessation, and smoking cessation does not confer a reduced risk of aneurysmal subarachnoid hemorrhage beyond that of reducing the cumulative dose [5)]

Data suggest that smoking, independent of hypertension, plays a critical role in aneurysm development, especially in younger patients, but that physiological mechanism exists for the repair of the damage induced by this toxic insult if cessation is possible [6)]

Results suggest that high cotinine levels in smokers with brain aneurysms are significantly associated with high rupture risk, independently of smoker status, age, and sex [7)]

The prevalence of smoking in patients who have suffered from SAH is higher than that in general adult population [8) 9)].

Smoking has also been suggested to contribute to the recurrence of aneurysms after endovascular coiling.

To improve the understanding of the impact of smoking on long-term outcomes after coil embolization of intracranial aneurysms, Brinjikji et al. studied a consecutive contemporary series of patients treated at their institution. The aims of this study were to determine whether smoking is an independent risk factor for aneurysm recurrence and retreatment after endovascular coiling.

All patients who had received an intrasaccular coil embolization of an intracranial aneurysm, who had undergone a follow-up imaging exam at least 6 months later, and whose smoking history had been recorded from January 2005 through December 2012 were included in this study. Patients were stratified according to smoking status into 3 groups: 1) never a smoker, 2) current smoker (smoked at the time of treatment), and 3) former smoker (quit smoking before treatment). The 2 primary outcomes studied were aneurysm recurrence and aneurysm retreatment after treatment for

endovascular aneurysms. Kruskal-Wallis and chi-square tests were used to test statistical significance of differences in the rates of aneurysm recurrence, retreatment, or of both among the 3 groups. A multivariate logistic regression analysis controlling for smoking status and for several characteristics of the aneurysm was also performed.

In total, 384 patients with a combined total of 411 aneurysms were included in this study. The aneurysm recurrence rate was not significantly associated with smoking: both former smokers (OR 1.00, 95% CI 0.61-1.65; p = 0.99) and current smokers (OR 0.58, 95% CI 0.31-1.09; p = 0.09) had odds of recurrence that were similar to those who were never smokers. Former smokers (OR 0.78, 95% CI 0.46-1.35; p = 0.38) had odds of retreatment similar to those of never smokers, and current smokers had a lower odds of undergoing retreatment (OR 0.44, 95% CI 0.21-0.91; p = 0.03) than never smokers. Moreover, an analysis adjusting for aneurysm rupture, diameter, and initial occlusion showed that former smokers (OR 0.65, 95% CI 0.33-1.28; p = 0.21) and current smokers (OR 1.04, 95% CI 0.60-1.81; p = 0.88) had odds of aneurysm recurrence similar to those who were never smokers. Adjusting the analysis for aneurysm rupture, diameter, and occlusion showed that both former smokers (OR 0.49, 95% CI 0.23-1.05; p = 0.07) and current smokers (OR 0.82, 95% CI 0.46-1.46; p = 0.50) had odds of retreatment similar to those of patients who were never smokers.

The results show that smoking was not an independent risk factor for aneurysm recurrence and aneurysm retreatment among patients receiving endovascular treatment for intracranial aneurysms at the authors' institution. Nonetheless, patients with intracranial aneurysms should continue to be counseled about the risks of tobacco smoking [10].

Tobacco use were not significantly associated with poor outcome after aneurysmal subarachnoid hemorrhage [11].

Previously established risk factors such as hypertension and smoking were identified as the most prevalent comorbidities, with disparity between subgroups, particularly women and African Americans [12].

The duration and timing of tobacco use, rather than the dose of tobacco per se, seem to be risk factors for delayed neurological deterioration after aneurysmal subarachnoid hemorrhage (aSAH). Although Krishnamurthy et al. did not find an association between tobacco use and overall clinical outcome after aneurysmal SAH, these results suggest that the distribution of various patterns of tobacco use within a given data set may influence the overall result [13].

Only 8 studies have investigated the incidence and epidemiology of aneurysmal subarachnoid hemorrhage (aSAH) in the United States. This is the first investigation in Indiana, which has some of the highest rates of tobacco smoking and obesity in the nation. The authors prospectively identified 441 consecutive patients with aSAH from 2005 to 2010 at 2 hospitals where the majority of cases are treated. Incidence calculations were based on US Census populations. Epidemiologic variables included demography; risk factors; Hunt and Hess scale; Fisher grade; number, location, and size of aneurysms; treatment type; and complications. Overall incidence was 21.8 per 100,000 population. Incidence was higher in women, increased with age, and did not vary by race. One third to half of patients were hypertensive and/or smoked cigarettes at the time of ictus. Variations by count were partially explained by Health Factor and Morbidity Rankings. Complications varied by treatment. These findings deviate from estimates that 6-16 per 100,000 people in the United States will develop aSAH and are double the incidence in a Minnesota population between 1945 and 1974. The results also deviate from the worldwide estimate of 9.0 aSAHs per 100,000 person-years. The predictive value of variations in Health Factor and Morbidity Rankings implicates the importance of future

research on multivariate biopsychosocial causation of aSAH [14].

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