SMOKERS WITH INTRACRANIAL ANEURYSMS WHO UNDERWENT NEUROSURGICAL OR ENDOVASCULAR TREATMENT HAVE NOT STOPPED SMOKING ONE YEAR AFTER SURGERY

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SUMMARY

Smoking increases the risk of forming, growing, and rupture of intracranial aneurysms. We retrospectively reviewed patients with intracranial aneurysms treated by neurosurgical or endovascular treatment − 154 patients (45 men, 109 women, 15 to 62 years, average 46.3 years, CI±1.72). We found 74% (114/154) of smokers − 80% (36/45) men and 71.6% (78/109) women, with the mean value of the Fagerström Test of Nicotine Dependence 4.4 (CI±0.40). The average age of smoking initiation was 18.2 years (CI±0.66), the average period of smoking 26.8 years (CI±2.13). The average number of cigarettes consumed daily was 18.2 (CI±1.58). With statistical significance $p<0.05$, the athero-index was lower in non-smokers than smokers: 3.4 (CI±0.56) vs. 4.5 (CI±0.51). HDL cholesterol was higher in non-smokers than smokers: 1.6 mmol/l (CI±0.25) vs. 1.4 (CI±0.10), and triglycerides were higher in smokers than non-smokers: 1.3 mmol/l (CI±0.16) vs. 1.9 (CI±0.35). Forty-two per cent of smokers (48/114) were controlled one year after the treatment; 18.8% of them stopped smoking, 41.7% reduced smoking, and 39.6% continued to smoke as extensively as before. The prevalence of smoking in our sample was higher than in the Czech population (28.2%). Only 18.8% of controlled smokers were able to quit one year after the intervention.

Key words: smoking, smoking cessation, intracranial aneurysm, subarachnoid hemorrhage, Fagerström Test for Nicotine Dependence

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INTRODUCTION

An intracranial aneurysm (IA) is a thin walled outpouching or dilatation of the brain artery. It originates in local infliction of the vascular wall by atherosclerosis, trauma, infection (mycotic aneurysms resulting from infected embolic material from a bacterial infection on one of the heart valves), neoplastic disease, and most often by congenital abnormality of the intima with abnormal thinning of the artery (1–3).

Many studies imply that smoking is a very important risk factor in the formation of intracranial aneurysms, with OR from 1.7 to 3.48 (4–6) (Table 1). Smoking and possibly also age and female sex seem to be risk factors for multiple intracranial aneurysms (OR of smoking 2.10; 95% CI, 1.06–4.13) (7). Cigarette smoking appears to increase the risk for growth of larger aneurysms (see e.g. the studies of The Department of Neurosurgery, University at Buffalo and University of Colorado in Denver). Smoking (OR 2.2; 95% CI, 1.1–4.5) and middle cerebral artery origin (OR 2.5; 95% CI, 1.3–4.9) seem to increase the risk of developing large aneurysms (6).

Smoking has also been proved to increase the risk of IA rupture (OR from 2.2 to 4.1 for former smokers, 5.4 for current smokers, respectively) that leads to subarachnoid hemorrhage (SAH) (6, 8, 9, 10) (Table 1). Hypotheses explaining the effects of smoking on IA rupture include enhanced systematic coagulability, inflammation within arterial walls, increased blood pressure, endothelial dysfunction, and the promotion of degradation of elastin within vessel walls by interfering with alfa1-antitrypsin (6, 11). Arterial hypertension is also a significant independent risk factor for aneurysmal SAH, OR from 2.4 to 6.8 (8, 9, 10, 12) (Table 1).

With regard to the minimal symptomatology of intracranial aneurysms, it is very important to support a healthy life style involving non-smoking. Cigarette smoking is the most important preventable cause of SAH, with a strong dose-response relationship having been shown in many studies. After smoking cessation the risk of aneurysmal rupture declines (11).

In the Czech Republic, with 10,429,692 inhabitants (13), the prevalence of smoking is 28.2% in the population older than 15 years (30.3% in men and 25.9% in women) (14).

Table 1. *Smoking and hypertension as risk factors for intracranial aneurysms and aneurysmal SAH*

	OR		
	of smoking for intracranial aneurysm	**of smoking for aneurysmal SAH**	**of hypertension for aneurysmal SAH**
Matsumoto et al., 1999	**1.7**		
Qureshi et al., 2000	**2.2** 95% CI 1.1–4.5		
Juvela, 2002	**3.48** 95% CI 1.14–10.64		
Qureshi et al., 2001		**5.4** 95% CI 3.7–7.8	**2.4** 95% CI 1.8–3.1
Kubota et al., 2001		**2.54**	**2.65**
Isaksen et al., 2002		**4.55** 95% CI 1.08–19.30	**2.46** 95% CI 1.52– 3.97
Kleinpeter and Lehr, 2002		**2.2** 95% CI 1.19–4.06	**6.8** 95% CI 3.53–13.14

This study does not seek to prove that smoking causes intracranial aneurysms. It addresses the resistance of our study sample to smoking cessation. We wanted to assess a sample of patients with diagnoses of intracranial aneurysms, find out the prevalence of smoking among these patients, and check their smoking status after one year.

METHODS

We retrospectively reviewed patients who had undergone the neurosurgical or endovascular treatment of intracranial aneurysms. The patients were informed about the impact of smoking on their disease and motivated to stop smoking (short intervention cca 10 minutes). We contacted all our patients one year after the neurosurgical or endovascular intervention to determine smoking status of those agreeable with follow-up examination.

The sample studied consists of patients with ruptured and unruptured intracranial aneurysms (IA) treated by neurosurgical or endovascular treatment at the Department of Neurosurgery of the Central Military Hospital, Prague, between January 2000 and December 2003.

A studied group consisted of 154 patients (45 men, 109 women, between the ages of 15 and 62) with the average age of the whole sample being 46.3 years (SD 10.8). Smoking status was determined by use of a questionnaire. "Smoker" was in our case everyone who, at the time of the study, smoked daily. Non-smokers included never-smokers and former smokers (ex-smokers for more than a year). There were not any occasional smokers in our sample. Patients were informed about the health hazards posed by smoking in respect of their disease; no other intervention was provided.

We also examined blood pressure and blood lipids such as total cholesterol (TC), triglycerides, HDL cholesterol, LDL cholesterol and athero-index (TC to HDL cholesterol ratio). Blood lipids were examined one year after the intervention. Statistical significance for the presented values was calculated by Independent Samples t-test.

Smoking status was checked up one year after the neurosurgical intervention in those patients who came for a follow-up examination. They answered the questions: "Do you smoke as extensively as a year ago?; Have you reduced your smoking (50% and fewer cigarettes daily compared to a year before)?; Did you stop smoking?"

RESULTS

Among our patients, 74% (114/154) were smokers − 80% (36/45) in men and 71.6% in women (78/109) (Fig. 1) were heavily nicotine dependent with a Fagerström Test for Nicotine Dependence (FTND) mean score of 4.4 (SD 1.94). All of them smoked daily and there was no occasional smoker among them. Their average age of initiation of regular smoking was 18.2 years (SD 3.40) and the average period of smoking was 26.8 years (SD 11.06). The average number of cigarettes smoked daily was 18.2 (SD 8.42). Smokers tend to have lower education compared to non-smokers – 15% of non-smokers had university education (11.11% men, 16.13% women) as opposed to 8.77% of smokers (13.89% men, 6.41% women).

There was a 2.85 times higher number of smokers than non-smokers in our sample – 4 times higher a number of smokers than non-smokers among men and 2.52 times higher a number of women smokers than non-smokers.

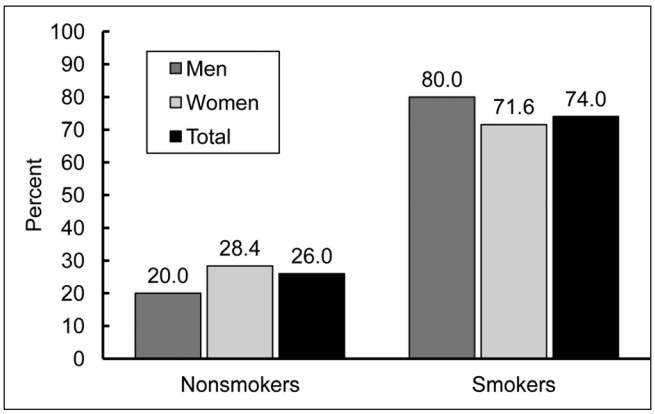

Fig. 1. *Smoking status among patients.*

Other risk factors for atherosclerosis in smokers and non-smokers were evaluated. For the whole sample total cholesterol, LDL cholesterol, and elevated blood pressure were positively associated with smoking, but without any statistical significance.

With statistical significance of $p<0.05$, the athero-index (TC/HDL) was lower in non-smokers than in smokers: 3.4 (CI±0.56) vs. 4.5 (CI±0.51), HDL cholesterol was higher in non-smokers than smokers: 1.6 mmol/l (CI±0.25) vs. 1.4 (CI±0.10), and triglycerides were higher in smokers than in non-smokers: 1.9 (CI±0.35) vs. 1.3 mmol/l (CI±0.16) (Fig. 3).

Forty-two per cent of smokers (48/114) were seen again one year after neurosurgical or endovascular treatment; 18.8% of them had stopped smoking (9/48), 41.7% (20/48) had reduced smoking (smoked daily at least 50% fewer cigarettes compared to a year before), and 39.6% (19/48) smoked the same amount of cigarettes as a year before (Fig. 2).

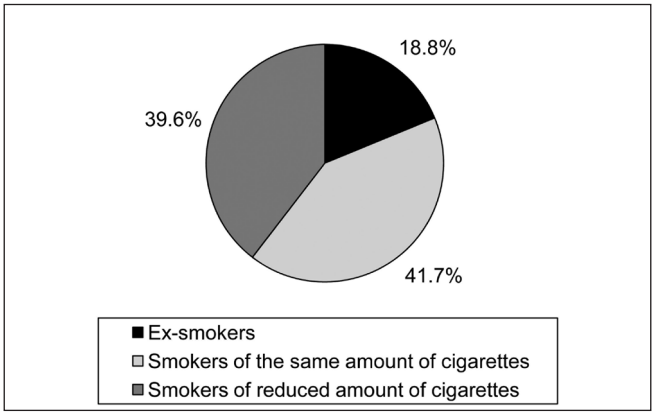

Fig. 2. *Smoking status after one year.*

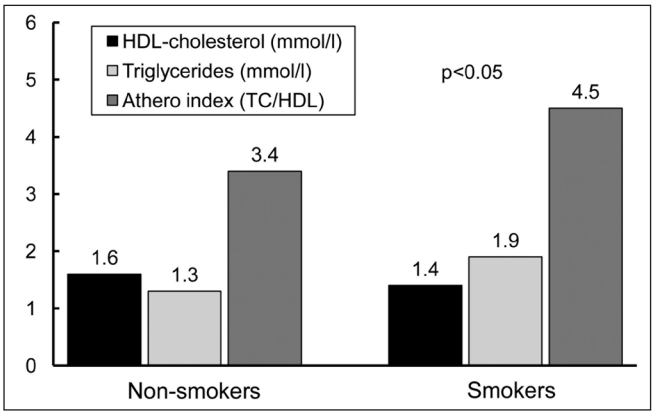

Fig. 3. *Blood lipids and smoking status.*

DISCUSSION

From the literature it is evident that cigarette smoking is a risk factor responsible for forming, growing, and rupture of intracranial aneurysms (4–6, 8–10, 12).

The prevalence of smoking in our sample was much higher compared to 28.2% prevalence found out in the Czech general population. It is alarming because IA severe enough to be treated by neurosurgical intervention is a very serious diagnosis. It could reflect the fact that many people don't realize the relationship between their diagnosis and cigarette smoking. The prevalence of smoking was determined from questionnaires. We could assume higher prevalence of smoking in the study sample in case of measuring expired air carbon monoxide.

Blood lipids were examined one year after the intervention. Their level may be influenced also by age, diet, and BMI.

Less than one third of the sample were men (29.2%). It would be interesting to compare a group of men and a group of women in the future phase of this study which is still continuing in order to test the role of sex in the formation of multiple intracranial aneurysms that has been suggested in some publications (7) as well as to explore the relationship between smoking and type, location, etc., of IA.

The average age of smokers was 47.3 years (SD 10.4) vs. years 43.4 in non-smokers (SD 11.6).

The study's smokers were heavily nicotine dependent regular, daily smokers, with a mean FTND of 4.4 (SD 1.94). They smoked average 18.2 (SD 8.42) cigarettes daily. The likelihood of a successful quit attempt without professional help is very low for nicotine dependent smokers. The quitting success rate in the general population with good tobacco control is only about 2% per year (15). It was 7.9% (9/114) in our sample, but if determined by measuring expired air carbon monoxide, the success rate could be lower. Fifty-eight per cent of smokers had lost to follow-up (66/114), so they were considered to be smokers.

CONCLUSIONS

Despite the short intervention and knowledge of highly risky behaviour, only 18.8% of smokers (7.9% of the sample) were able to quit smoking one year after treatment of the intracranial aneurysm. This demonstrates the resistance of the study sample to smoking cessation. Patients with known intracranial aneurysms should definitely stop smoking. Intensive treatment of tobacco dependence should be actively offered to this high risk group. The study still continues.

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Competing Interests:
None declared.

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