Smoking Cessation 1 Year Poststroke and Damage to the Insular Cortex

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Background and Purpose—Hospitalization as a result of stroke provides an opportunity to stop smoking that is often not taken up. The present study analyzes sociodemographic, psychological, and lesion-related variables to identify associated factors for smoking cessation during the first year after stroke.

Methods—We conducted a prospective longitudinal study with a 1-year follow-up of a cohort of 110 patients with acute stroke who were smokers at the time of diagnosis and were admitted consecutively between January 2005 and July 2007.

Results—On hospital release, 69.1% had given up smoking but at 1 year, only 40% had stopped smoking. Of the 110 patients, 27 (24.5%) had an acute stroke lesion in the insular cortex, of which 19 (70.3%) were nonsmokers at 1 year. Strongly associated factors in giving up smoking were the location of the lesion in the insular cortex (OR, 5.42; 95% CI, 1.95–15.01; P=0.001) and having the intention of giving up before the stroke, comparing precontemplating patients (without intention of giving up in the near future) with contemplating and prepared patients (intention of stopping in the near future; OR, 7.29; 95% CI, 1.89–28.07; P=0.004).

Conclusions—Of patients with stroke who were smokers, only 4 of 10 patients had stopped smoking 1 year after admission. Our results show that the variables best predicting smoking cessation in patients with a stroke diagnosis 1 year after hospital discharge are insular damage and the prestroke intention to stop.

Key Words: cessation ■ cerebral ■ cortex ■ motivation ■ stroke ■ smoking

Smoking has highly negative hemodynamic, hemostatic, and lipid effects; plays an important role in the development of atherosclerosis; and is a premature cause of death of millions of people in the world.1–5 Smokers are between 2 and 3 times more likely to have a stroke than nonsmokers and the risk is 2 times more likely if smoking addiction is associated with arterial hypertension.6–8 Tobacco addiction is also a risk factor for sustaining a cerebral or subarachnoid hemorrhage, especially in women.7,9,10 The risk of having a stroke falls on stopping smoking, especially between 2 and 5 years after cessation.9 Given these circumstances, smoking cessation has to be firmly encouraged during hospitalization.

Our understanding of the decisive factors for smoking cessation has deepened in an attempt to plan a more effective and efficient intervention for health promotion. It has been found that consumers go through different stages, processes, and levels of change before, during, and after giving up the habit.11 Whereas biological, psychological, and social factors have all been found to contribute to the decision to give up smoking, health tends to be the main motivation.12–14 Despite this, nicotine dependence rather than motivational factors has been seen as the main predictor of smoking cessation in the long term.15 Some authors have found an association between smoking cessation and living in institutions after the stroke,16 receiving specific information and education regarding changing habits,17 and greater discapacity.18 Noncessation has been associated with male sex, living alone, being a blue collar worker, not having discapacity,19 and younger age.20 A recent line of investigation has associated smoking cessation in stroke with insular cortex lesions.21 The study of a large number of cerebral systems involved in addictive behavior, together with improved understanding of activation through drug exposure of cortical areas such as the anterior cingulate cortex, the orbitofrontal cortex, and the insular cortex, has revealed that the insular cortex is necessary for the maintenance of tobacco addiction.22–24 Naqvi et al,21 in a retrospective study, observed that patients with insular damage gave up smoking more easily after stroke than patients who had lesions to other areas and, especially, that they had presented a situation of “disruption of smoking addiction.” However, a more recent prospective study with patients with ischemic stroke failed to find the same association.25

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The present study analyzes sociodemographic, psychological, and lesion-related variables to identify associated factors for smoking cessation during the first year after stroke.

Subjects and Methods

From a total of 1003 patients with acute stroke consecutively admitted to the Stroke Unit of the Department of Neurology of the Josep Trueta University Hospital of Girona (northeast Spain) between January 2005 and July 2007, 110 patients who were smokers at the time of diagnosis were prospectively evaluated in a longitudinal study with a 1-year follow-up. The reasons for excluded patients are set out in the Figure. Of the 208 active smokers, 38 patients who died in-hospital or were discharged with significant neurological deficits (Canadian Stroke Scale ≤4) or comprehension disorders that impeded follow-up, 16 nonresidents in Spain and 44 that declined to be followed during the first year postdiagnosis were excluded. Excluded patients were significantly older and had greater neurological deficit but had a similar prevalence of vascular risk factors and similar cigarette consumption per day.

The study protocol was approved by the hospital’s ethics committee. All patients were informed about the study protocol and signed an informed consent form.

Baseline Assessment

Variables Studied During Hospitalization

All cases were registered in the prospective stroke data bank of the Spanish Neurological Society (BAdISEn). The following data were recorded: sociodemographic variables, neurological deficit at admission and hospital discharge in accordance with the Canadian Stroke Scale,27 functional state at discharge by the Barthel Index,28 and the modified Rankin Scale,29 length of hospital stay, data related to the disease including classical vascular risk factors, neuroimaging characteristics, and the final etiopathogenic diagnosis of stroke, and the Trial of ORG 10172 in Acute Stroke Treatment classification for determination of the subtype of ischemic stroke.30 A diagnosis of hemorrhagic stroke was considered if the clinical signs were compatible with stroke and an intracerebral hemorrhage was found at CT scan and/or MRI.

Furthermore, data related to smoking history such as the number of cigarettes per day until the day of the event, level of nicotine dependence before the diagnosis as per the Fagerström Test,31 and the stage of change at which the patients were before the stroke according to the Transtheoretical Model of Prochaska, Norcross, and Diclemente were recorded.11 This model includes: (1) precontemplation stage: no immediate intention of stopping smoking; (2) contemplation stage: intending to stop smoking in the next 6 months; (3) preparation stage: considering stopping smoking in the next month with an attempt at stopping smoking in the past year; (4) action stage: stopped smoking for <6 months; and (5) maintenance: smoke-free for at least 6 months.

CT scan or MRI was performed at admission and on follow-up at 5±2 days after stroke onset. The second CT scan or MRI was performed in all patients to confirm the final topography and lesion volume. Three investigators classified the lesion location independently with disagreements being resolved by consensus. Lesions affecting the insular cortex were later classified by hemisphere (right or left side) and subtype (ischemic or hemorrhagic). The volume of the cerebral infarct or hemorrhage was measured by an experienced neurologist applying the largest diameters method with the formula $0.5 \times A \times B \times C$ (where $A =$ largest diameter of the infarction, $B =$ largest perpendicular diameter of the infarction, and $C =$ largest vertical diameter).32 These evaluations were done during the acute phase of stroke and before the potential smoking cessation on follow-up was known.

During hospitalization, patients were managed in accordance with European Stroke Organisation Stroke Guidelines. Patients and relatives followed a full health education training program as part of the standard integral acute phase stroke treatment.

Follow-Up Assessment

Follow-up evaluations were performed between 3 and 6 months and at 1 year after the stroke. When the patient missed the first follow-up visit, follow-up was performed by telephone to avoid information loss. All patients were visited as outpatients at 1 year to verify abstinence.

Smoking cessation, the stage of change at each of the controls performed, the days of abstinence from tobacco, the date of cessation or relapse, and the number of cigarettes (if the patient continued smoking) were studied throughout the follow-up period.

The difficulty in giving up smoking and the urge to start smoking again were evaluated through 2 questions: (1) How difficult was it for you to give up smoking? (from 1 = no difficulty to 7 = maximum degree of difficulty); and (2) since giving up smoking, have you felt the urge to start smoking again? (yes, no, sometimes).

The “disruption of smoking addiction” variable was defined as the presence of smoking cessation from the moment of the stroke, the absence of relapses, having stopped smoking without difficulties (<3 on the scale of difficulty), and not having had the urge to smoke since the moment of having stopped smoking. This variable, which was described by Naqvi et al.,21 has been used in the present investigation as a means of comparing the results.

Carbon monoxide levels in expired air were recorded during outpatient visits as an objective biomarker to verify cessation in the group of patients who self-informed that they had stopped smoking. Measurements were performed with a Micro CO Meter (Micro Medical Ltd). Carbon monoxide values of >8 ppm were considered as positive of active smoking.33

Statistical Analysis

The Student $t$ test and the analysis of variance test were used to compare groups in the case of continuous variables and the $\chi^2$ test was used for categorical variables. A stepwise logistic regression analysis was performed to assess variables predicting smoking cessation 1 year after stroke diagnosis. Demographic variables were included in the first block with the “enter” method. In the second block, variables related to smoking history and variables related to the stroke (neurological deficit and functional deficit on discharge) were entered stepwise. In the third block, the location of the lesion was also entered stepwise (affecting the insular cortex/not affecting the insular cortex). Results were expressed as adjusted ORs and corresponding 95% CIs.

Results

Baseline characteristics of patients are shown in Table 1. Men were significantly older and had a higher prevalence of vascular risk factors. Ninety-eight patients (89.1%) presented with ischemic stroke and 12 (10.9%) had intracranial hemorrhagic lesions. In 27 patients (24.5%), the location of the lesion affected the insular cortex. The distribution of ischemic
stroke subtypes was as follows: 24.1% large-artery atherosclerosis (including large-artery thrombosis and artery-to-artery embolism); 13.8% cardioembolism; 33.3% small-artery occlusion, 5.7% stroke of other determined cause; and 23% stroke of undetermined origin.

Patients smoked an average of 27.6 cigarettes per day (SD, 15.4), had a mean score of 6.1 points (SD, 2.1) in the Fagerström Test, and had started smoking at a mean age of 17.1 years (SD, 5.1). On having the stroke, 85.5% of the sample declared that they were not considering stopping smoking in the near future (they were in the precontemplation phase at admission) and 15.6% of patients had previously attempted to stop smoking. One third of patients perceived the association of stroke with the consumption of tobacco, whereas one third did not perceive any association and a further third responded that they did not know.

There was a progressive increase along the study period in the percentage of people who did not intend to give up smoking (precontemplators), especially in the group of patients without insular cortex lesions (Table 2). At discharge, 69.1% of patients referred to having given up smoking during hospitalization, specifically 88.9% of patients with an insular cortex lesion group had stopped smoking in comparison with 62.7% in those without (P=0.05). At the first follow-up visit, only 51.8% of the patients of the study had stopped smoking: 81.5% of patients with an insular cortex lesion as compared with 42.2% of those without (P<0.001). One year after stroke, 40% had stopped smoking completely (n=44), of which 37 had not smoked for ≥6 months (maintenance stage).

No statistical differences were observed between smoking cessation and the type of lesion (ischemic or hemorrhagic) or between cessation and the side of the lesion (right or left) either in the sample as a whole or when studied separately by whether the lesion was in the insular cortex. Comparing smoking cessation with the location of the lesion, 70.4% of the participants (19 of the 27) with an insular cortex lesion had stopped smoking in comparison with 30.1% (25 of the 83) of those without (P<0.001).

Biological confirmation of having stopped smoking by the carbon monoxide measurement in expired air showed only 3 patients to have levels ≥8 ppm in the first follow-up visit and 2 patients at the 1-year visit (for statistical purposes, these were recorded as smokers). None of these patients had insular cortex lesions.

In studying the difficulty in giving up smoking according to the type of lesion, patients with insular cortex lesions gave significantly lower scores (mean score, 3.8; SD, 2.1) than those without this type of lesion (mean score, 5.1; SD, 2.4; P<0.05).

Table 1. Baseline Characteristics of the Subjects*

<table>
<thead>
<tr>
<th>Variables Assessed at Hospitalization</th>
<th>All Strokes Current Smoking (n=110)</th>
<th>Men (n=96)</th>
<th>Women (n=14)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>55.6 (10.7)</td>
<td>57.1 (10.2)</td>
<td>44.9 (7.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Living with their family</td>
<td>84 (77.8)</td>
<td>71 (75.5)</td>
<td>13 (92.9)</td>
<td>NS</td>
</tr>
<tr>
<td>Cigarettes per d</td>
<td>27.6 (15.4)</td>
<td>28.3 (15.4)</td>
<td>22.8 (15)</td>
<td>NS</td>
</tr>
<tr>
<td>Tobacco dependence (Fagerström Test)</td>
<td>6.1 (2.1)</td>
<td>6.1 (2.1)</td>
<td>5.8 (2.2)</td>
<td>NS</td>
</tr>
<tr>
<td>Prochaska stage</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>prior stroke (precontemplation)</td>
<td>94 (85.5)</td>
<td>82 (85.4)</td>
<td>12 (85.7)</td>
<td>NS</td>
</tr>
<tr>
<td>Previous attempts at stopping</td>
<td>14 (15.6)</td>
<td>12 (15.6)</td>
<td>2 (15.4)</td>
<td>NS</td>
</tr>
<tr>
<td>Age at onset of regular smoking, y</td>
<td>17.1 (5.1)</td>
<td>17.1 (5.3)</td>
<td>16.6 (3.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Vascular risk factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension (n=108)</td>
<td>51 (47.2)</td>
<td>49 (52.1)</td>
<td>2 (14.3)</td>
<td>0.007</td>
</tr>
<tr>
<td>Diabetes (n=108)</td>
<td>17 (15.7)</td>
<td>16 (17)</td>
<td>1 (7.1)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypercholesterolemia (n=109)</td>
<td>36 (33)</td>
<td>30 (31.6)</td>
<td>6 (42.9)</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>9 (8.2)</td>
<td>9 (9.4)</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Alcohol consumption (≥40 g/d)</td>
<td>20 (18.2)</td>
<td>19 (19.8)</td>
<td>1 (7.1)</td>
<td>0.006</td>
</tr>
<tr>
<td>Prior stroke</td>
<td>21 (19.1)</td>
<td>20 (20.8)</td>
<td>1 (7.1)</td>
<td>NS</td>
</tr>
<tr>
<td>Stroke subtype</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic stroke</td>
<td>98 (89.1)</td>
<td>86 (89.6)</td>
<td>12 (85.7)</td>
<td>NS</td>
</tr>
<tr>
<td>Hemorrhagic stroke</td>
<td>12 (10.9)</td>
<td>10 (10.4)</td>
<td>2 (14.3)</td>
<td></td>
</tr>
<tr>
<td>Canadian Stroke Scale on admission</td>
<td>8.2 (2.2)</td>
<td>8.3 (2.2)</td>
<td>7.7 (2.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Canadian Stroke Scale at discharge</td>
<td>8.7 (1.8)</td>
<td>8.7 (1.8)</td>
<td>8.7 (2)</td>
<td>NS</td>
</tr>
<tr>
<td>Barthel Index at discharge</td>
<td>76.2 (32.8)</td>
<td>76.2 (32.6)</td>
<td>75.7 (36.1)</td>
<td>NS</td>
</tr>
<tr>
<td>Modified Rankin Scale</td>
<td>2.02 (1.6)</td>
<td>2.07 (1.6)</td>
<td>1.6 (1.9)</td>
<td>NS</td>
</tr>
<tr>
<td>Hospitalization, d</td>
<td>6.3 (4.6)</td>
<td>6.5 (4.8)</td>
<td>4.9 (2.9)</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS indicates nonsignificant; SD, standard deviation.
*Values are means (SD) or no. (percentages).
Insular Damage (n=27) | Noninsular Damage (n=83) | P
---|---|---
On discharge | | NS
Precontemplation | 3 (11.1) | 18 (21.7) |
Contemplation | 0 | 12 (14.5) |
Preparation | 0 | 1 (1.2) |
Action | 24 (88.9) | 52 (62.7) |
At first follow-up | 0.004
(3–6 mo poststroke) | | |
Precontemplation | 4 (11.8) | 28 (33.7) |
Contemplation | 0 | 10 (12) |
Preparation | 1 (3.7) | 10 (12) |
Action | 22 (81.5) | 35 (42.2) |
One year poststroke | 0.001
Precontemplation | 5 (18.5) | 34 (41) |
Contemplation | 0 | 10 (12) |
Preparation | 3 (11.1) | 14 (16.9) |
Action | 1 (3.7) | 6 (7.2) |
Maintenance | 18 (66.7) | 19 (22.9) |

Numbers in parentheses are percentages. NS indicates nonsignificant.

Twenty patients (18.2%) were classified as falling within the “disruption of smoking addiction” variable. The percentage of patients with these characteristics was higher in the group of patients with an insular cortex lesion but this did not reach significance (29.6% versus 14.5%, P=0.09).

Patients who had stopped smoking had a lesser perception of difficulty in stopping smoking, higher frequency of intention of stopping smoking before the stroke, and a greater proportion of insular cortex lesions than patients who continued smoking 1 year after the stroke (Table 3). The logistical regression analysis revealed the strongly associated factors for smoking cessation to be having the lesion at the insular cortex (OR, 5.42; 95% CI, 1.95–15.01; P=0.001) and having had the intention of stopping smoking before the stroke (evaluated by comparing precontemplation stage patients with contemplation and preparation stage patients; OR, 7.29; 95% CI, 1.89–28.07; P=0.004; Table 4).

Finally, the neuroimage data of the 27 patients with insular cortex lesions were evaluated. The mean lesion volume of the 8 patients who did not give up smoking was 22.5 cm³ (interquartile range, 14.9–95.6 cm³) and the mean lesion volume of the 19 patients who stopped smoking was 24.7 cm³ (interquartile range, 9–75 cm³). Differences between the 2 groups were not significant.

Discussion

The present study shows a statistically significant relationship between smoking cessation and the lesion in the insular cortex as a consequence of stroke. Although this was not conclusive at discharge, at the first follow-up visit, 8 of 10 patients with an insular cortex lesion had stopped smoking in comparison with 4 of 10 who did not have this lesion, and at 1 year of the stroke, 7 of 10 patients with an insular cortex lesion had stopped smoking as compared with 3 of 10 who did not have a lesion in this part of the brain. In our research, the probability of stopping smoking was 5.4-fold greater in patients with a lesion at the insular cortex than in those who did not have a lesion in this area. Furthermore, and in agreement with Naqvi et al., patients with insular cortex lesions presented significantly lower scores on the scale used to evaluate the difficulty in giving up smoke. We have also

Table 4. Final Logistic Regression Model*

<table>
<thead>
<tr>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>1.03</td>
<td>0.24–4.40</td>
</tr>
<tr>
<td>Age</td>
<td>1.04</td>
<td>0.99–1.08</td>
</tr>
<tr>
<td>Intention to change†</td>
<td>7.29</td>
<td>1.89–28.07</td>
</tr>
<tr>
<td>Insular damage</td>
<td>5.42</td>
<td>1.95–15.01</td>
</tr>
</tbody>
</table>

OR indicates odds ratio; CI, confidence interval.

*Logistic regression model adjusted for sex and age.
†Intention to change: patients in contemplation (intending to stop smoking in the next 6 months) + patients in preparation (considering stopping smoking in the next month with an attempt at stopping smoking in the past year).
observed a trend among patients with an insular cortex lesion to present less urge to smoke with a greater proportion of patients with “disruption of smoking addiction,” although, unlike in Naqvi et al,21 this did not reach statistical significance.

At discharge, 7 of 10 patients stated that they had stopped smoking but this had fallen to just 5 of 10 at the first follow-up visit and only 4 of 10 at 1 year. Eight earlier studies on smoking cessation in patients with stroke have given data relating to the profile of smokers, the prevalence of smoking, and related factors,16–19,21,25,34,35 but only the study of Sienkiewicz-Jarosz et al35 that investigated factors relating to smoking cessation in 98 patients with acute stroke focused on a group with a similar sociodemographic profile to the present study in terms of age and the type of convalescence. The demographic characteristics of the population of the present study mirror those of the general population in our country with a higher percentage of smokers in adults <65 years and a higher proportion in men.36,37

We found that most patients were not considering smoking cessation at the time of having the stroke, although no comparison can be made here with other studies because, to the best of our knowledge, this factor has not previously been considered. Having said that, in comparing the situation in Europe and the United States, the highest incidence of people in the precontemplation stage is found in Europe, whereas the greatest incidence of people in the preparation stage is in the United States.38 This difference is believed to be due to the greater pressure to smoke cessation exercised by the health authorities of the United States over many years.

Only one third of the patients in our sample associated stroke with their smoking habit. This fact is indicative of the lack of awareness regarding stroke risk factors in the general population and highlights the need to improve the information given so as to make progress in the primary prevention of this disease. The fact that the importance of the association with this disease is learned when smokers are hospitalized makes it vital for healthcare professionals to work together with patients from early on in their period of hospitalization to successfully achieve a change in conduct.39,40 In other words, as Prochaska and DiClemente proposed (1986),41 a process of consciousness-raising needs to be gone through in which the patient becomes clear that he or she has a problem to initiate the process of change. When the patient is fully aware that smoking is a problem, he or she will no longer see this behavior as something that is worthwhile.

With regard to cessation of smoking, the previously cited studies report cessation from 21.7% to 43% between 3 and 6 months, which are slightly below those of the present study.16,19,25,35 At 1 year, lower percentages have been reported to those in the present study (30%)34 as well as similar percentages (41.1%).16

In the present study, smoking cessation has not been associated to female sex as was the case in other cited studies16,19 nor to worse functional dependence.18,19,34 With regard to the variables related to smoking history, our results agree with Sienkiewicz et al,35 in which people who had stopped smoking presented less urge to smoke. However, in contrast to this study, patients who had stopped smoking in the present sample were not found to have had a different level of tobacco consumption and less tobacco dependence as those who did not stop smoking during the follow-up period.

To the best of our knowledge, this is the first study to investigate smoking cessation in a sample of patients with stroke that prospectively associates cessation with biological and psychological variables during the first year postdiagnosis. The present research has found an association between cessation and the cerebral lesion produced by the stroke. Naqvi et al21 in an earlier retrospective study of 69 patients found a greater incidence of cessation in patients with insular damage, but without statistical significance, and significantly greater “disruption of smoking addiction” in patients with insular lesions. Bienkowski et al25 have investigated the same phenomenon more recently in 87 patients with ischemic stroke and did not find an association between smoking cessation and the insular cortex lesion.

The main limitation of the study was the difficulty in following the patients, because although patients typically attend neurological visits, there tends to be a higher level of nonattendance when following up risk factors, especially in those who continue smoking. For this reason, not all patients were visited twice at outpatients, and those who failed to attend either of the 2 programmed visits were followed up by telephone. Further possible limitations are the relatively small sample size, the necessary exclusion criteria (severely ill patients and those who could not be followed up), and not having studied the impact that the home environment might have on the success in giving up smoking. On the other hand, among the strengths of the present study are the fact that information provided by the patients with regard to smoking cessation was contrasted with a biomarker (the measurement of carbon monoxide) and that detailed information was recorded regarding the smoking history of patients.

The results of the present study demonstrate that the variables that best predict smoking cessation in patients with a diagnosis of stroke 1 year after hospital discharge are the location of the lesion at the insular cortex and the intention to stop smoking before the stroke. The results should make it possible to conduct antismoking and health education sessions with patients with stroke with a more precise understanding of the process of giving up smoking with regard to the factors that predispose and determine cessation. Furthermore, it is hoped that the results will encourage further investigation in this line.

Disclosures

None.

References


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