



THE SMOKING PARADOX IN ISCHEMIC STROKE: A SINGLE CENTER RECORD.

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Background:

A better outcome in smokers compared to nonsmokers was first observed in myocardial infarction and, more recently, in ischemic stroke [1]. Smoking has been associated with more thrombogenic vascular occlusions, greater susceptibility to spontaneous or therapeutic thrombolysis [2] and higher reperfusion rate [3]. Smokers are younger and with less comorbidities, but paradox is still confirmed after adjustment for cofactors.

Aim:

The aim of our study was to to evaluate the relationship between smoking status and reperfusion in ischemic stroke.

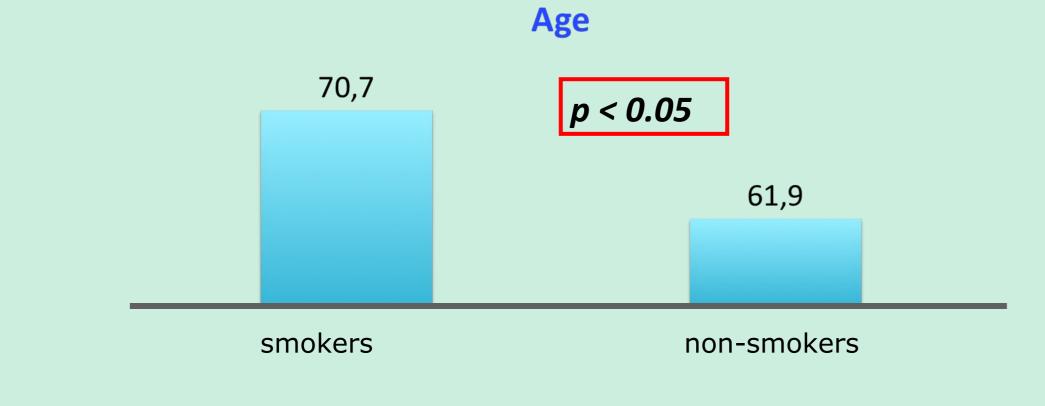
Methods:

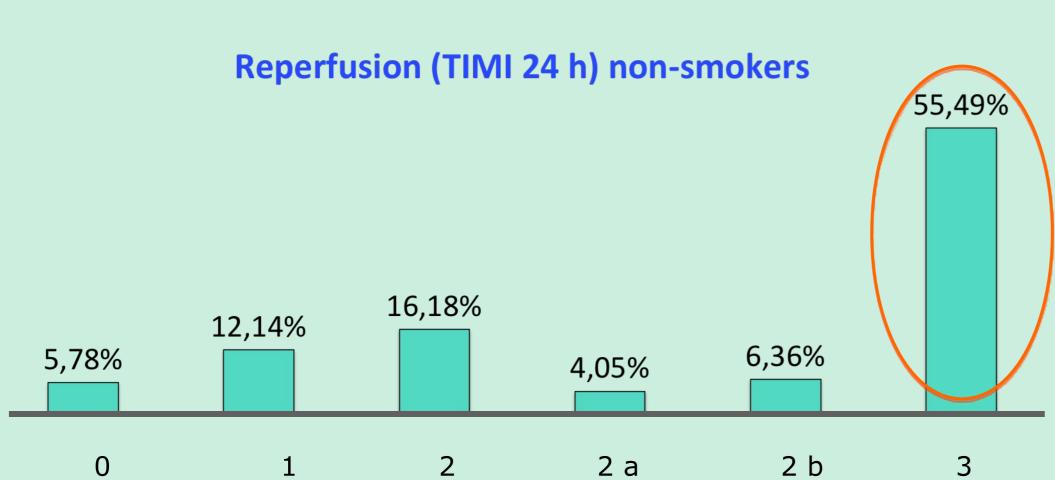
We analysed data from patients consecutively admitted to our centre for ischemic stroke, from September 2009 to May 2014, for which we had data about reperfusion. Patients included were 247, 132 male and 115 female, mean age 69.1 yrs (11.2 SD), 19% (n=47) were smokers, 69.6% (n=172) non smokers, 4.9% (n=12) ex-smokers; for 16 patients (6.5%) it wasn't possible to obtain smoking status. This last group and ex-smokers were excluded from further analysis. Patients were divided into three groups, depending on the

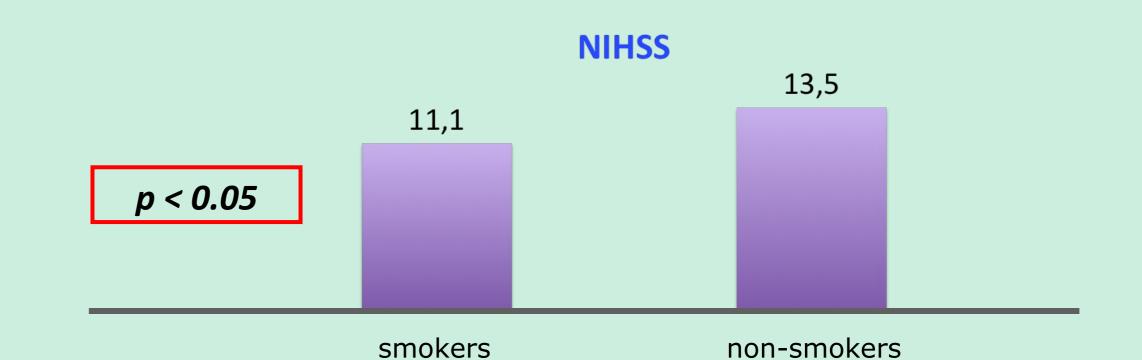
treatment they received: IV rt-PA, endovascular or no therapy. Reperfusion was defined as a complete restoration of blood flow (TIMI 3) at 24 hours on CTA. Secondary endpoints included favorable and very favorable outcome (90-day modified Rankin score \leq 2 or \leq 1), 24-hours and 7-days NIHSS and any intracerebral hemorrhage.

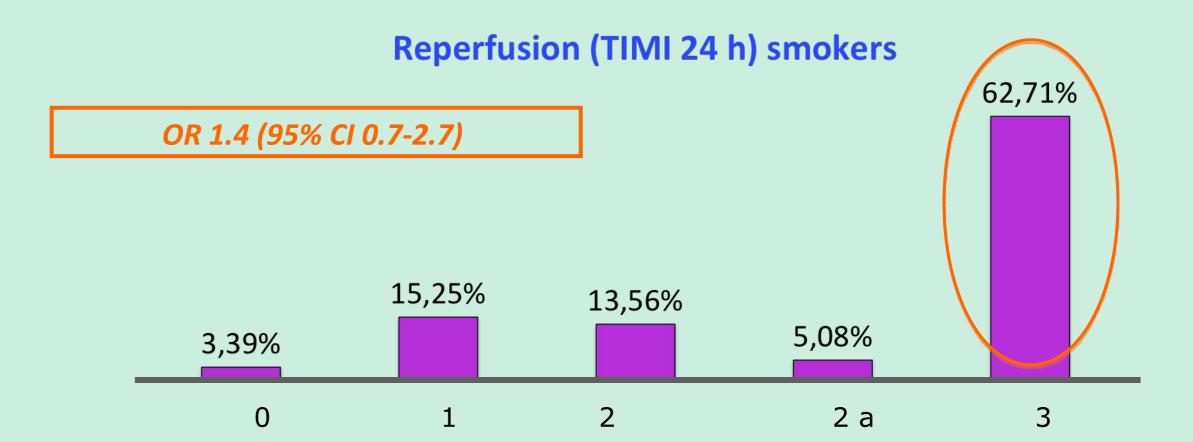
Results:

Smokers were younger than non-smokers (median 61.9 [SD 11.7] years versus 70.7 [SD 10.4] years; p < 0.001) and had a milder stroke at baseline (median NIHSS 11.1 [SD 5.8] versus 13.5 [SD 7.1]; p < 0.05). Median NIHSS at 24 hours was 9.5 in non-smokers and 8.2 (SD 6.4) in smokers, without any significant difference. Median 7-days NIHSS was similar in smokers and non-smokers (6.3 versus 6.9). Smoking was not significantly associated with reperfusion (OR 1.4; 95% CI 0.7-2.7) even if there was a higher percentage of reperfusion in smokers (62.7% versus 55.5%). A very favourable outcome was not significantly different (OR 0.9; 95% CI 0.2-3.5) and not even a favourable one (OR 0.6; 95% CI 0.1-2.6). There was no difference regarding hemorrhagic transformation. These findings were similar in all subgroups.









Conclusions:

In our study smokers are younger and have a milder stroke; they also have a higher reperfusion rate even if in a non significantly way. Main limitation of this study is the small sample. Smoking paradox is a concept that needs further studies to identify underlying pathophysiologic mechanism and a patient-target population with a favorable response to treatments and prognosis

References:

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