Effects of Chronic Alcohol Dependence and Chronic Cigarette Smoking on Cerebral Perfusion

S. Gazdzinski[1], T. C. Durazzo[2], F. Ezekiel[2], G. H. Jahng[1], D. J. Meyerhoff[1]

[1]Radiology, University of California San Francisco, San Francisco, California, United States, [2]VA Medical Center, San Francisco, California, United States

Introduction: Previous research has associated chronic alcohol dependence with decreased cerebral blood flow (CBF), especially in frontal and temporal lobes. However, the potential effects of chronic cigarette smoking in alcohol-dependent individuals were not considered. Approximately 80% of alcoholics smoke regularly [1], and chronic cigarette smoking, independent of alcohol consumption, is associated with globally decreased CBF [e.g., [2]]. We tested the hypothesis that chronic cigarette smoking increases chronic alcohol induced CBF deficits, especially in the frontal and parietal gray matter.

Methods: We studied 23 alcohol dependent individuals in treatment, who were abstinent for 6±3 days and 12 healthy light drinking, non-smoking control participants (nsCON, 42.4 ± 10 years of age). In the alcohol dependent group, 15 were smokers (sALC, 47.3 ± 9) and 8 non-smokers (nsALC, 53.1 ± 8). sALC smoked the last cigarette 2.6±1.1 hours prior to the perfusion measurement (the acute effects of smoking on CBF are currently not clear [2]). Cerebral perfusion was measured with DIPLOMA [3] pulsed arterial spin labeling using single shot EPI acquisitions in five 8mm thick slices 2mm apart, above the circle of Willis and oriented 5° off the orbital meatal line. Imaging parameters were TR=2.5s, TE=15ms, TI_2 (time between labeling pulse and the excitation pulse)=1500ms with in-plane resolution of 2.3 x 2.3 mm^2. T1-weighted 3D MPRAGE images were also acquired with TR/TI/TE=10/300/4ms for tissue segmentation. MPRAGE images were automatically segmented into probabilistic maps of gray matter (GM), white matter (WM) and CSF in frontal and parietal lobes. These maps were co-registered to the perfusion image, thus allowing for calculation of mean perfusion in frontal and parietal GM and WM. Regional GM and WM perfusion was calculated from voxels containing at least 90% GM tissue and 100% WM tissue, respectively.

Results: Univariate analysis of variance indicated significant group differences in frontal GM perfusion ($p = 0.05$). Follow-up comparisons, covaried for age, revealed significantly lower frontal GM perfusion in sALC than nsCON (-7%, $p=0.03$), and a trend for lower frontal GM perfusion in sALC compared to nsALC (-6%, $p=0.07$). No differences in frontal WM or parietal lobar perfusion were observed between nsALC and nsCON.

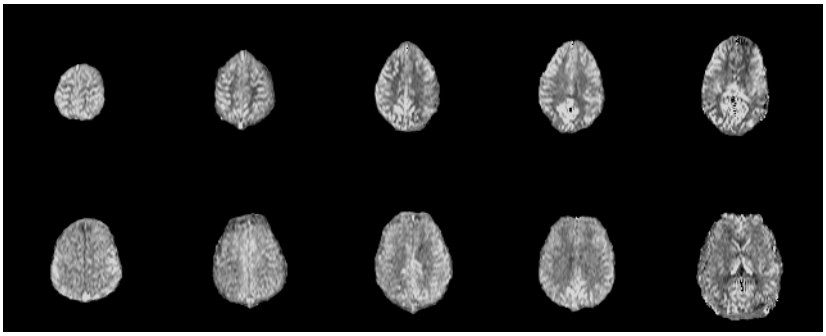

Fig. 1. Consecutive subtraction images for nsCON (upper) and sALC (lower). Notice the weaker GM – to – WM contrast in the sALC, suggesting lower GM perfusion.

Discussion: In other studies, we have shown via ^{1}H MR spectroscopic imaging that chronic cigarette smoking exacerbates chronic alcohol induced neuronal injury and cell membrane damage in frontal lobes [4]. The preliminary perfusion results of this study also suggest that chronic cigarette smoking magnifies alcohol-associated abnormalities in frontal gray matter perfusion. Mechanisms for reduced perfusion in sALC may be related to compromised vascular function secondary to impaired vasomotor reactivity [5] and/or atherosclerosis [6]. Our smoking related findings indicate that the effects of chronic cigarette smoking should be considered in future studies on alcohol dependent individuals and also in other common pathologies where chronic smoking is a co-morbid factor.

References:

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