A combined conventional ROI and Voxel based T2 relaxometry analysis in alcohol use disorders

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Target audience: Researchers working in the field of drug abuse and neuroimaging.

Purpose: Magnetic Resonance Imaging (MRI) techniques have contributed significantly to our understanding of the effects of heavy alcohol use on structural, biochemical and functional changes in the human brain, which might underlie the neuropsychological impairments that are observed in alcohol dependent individuals. However, very few studies specifically sought to examine the alterations in associated hemodynamic or paramagnetic properties (as reflected by T2 relaxation times (T2-RT)) in this population, that too using the conventional ROI based approaches[1]. Taking this into consideration, we performed a T2 relaxometry study, using Voxel based relaxometry (VBR) and conventional ROI analysis to identify regions of T2-RT alterations in alcohol dependent subjects as compared to controls and to further identify the correlation of T2-RT alterations with the neuropsychological test scores of brain dysfunction.

Material and methods: The T2 mapping sequence (standard Carr-Purcell-Meiboom- Gill (CPMG) multi-echo acquisition (12 echoes; echo times, TE = 13.3–159.6 ms (spaced at equal intervals); repetition time, TR = 6.7 s; slice thickness = 3 mm; no. of slices = 36 slices; image matrix: 256*128; field-of-view (FOV) = 180 cm; scan time, T scan = 13.31 min) was acquired for alcohol dependent subjects (n=25) and healthy controls (n=20) matched for age, sex and education. The T2 maps in our study were generated by fitting the relaxation time curves to a modified exponential model, i.e., a mono-exponential decay with inclusion of a baseline, i.e. $S(t)=S(0)\exp(-t/T2)+k$, where S(t) is the signal acquired at time=t and k is the baseline signal level. VBR analysis followed methods previously described[2]. ROI analysis was also performed to quantify T2-RT changes in areas which showed T2-RT alterations in VBR. PGIBBD (PGI-Battery of Brain Dysfunction) test battery subtests, namely, memory scale, Koh's block, Bender-Gestalt (BG) and Nahor-Benson (NB) test, were administered to all subjects for their neuropsychological evaluation. The whole study was carried out using 3T whole body MR system (Magnetom Skyra, Siemens, Germany) with a 32 channel head coil.

Results and discussion: VBR results showed clusters of increased T2-RT in cerebellum bilaterally, parahippocampal gyrus (LPHG) bilaterally, right anterior cingulate cortex(RACC), left superior temporal gyrus (LSTG), left calcarine gyrus (LCaG) and left middle frontal gyrus (LMFG) in alcohol dependents as compared to controls (Fig.1;table 1). ROI analysis also revealed similar trend of increased T2-RT in these areas as was observed in VBR. We also observed some very significant correlations between alterations in T2-RT and the dysfunction scores of neuropsychological tests. T2-RT alterations in AUD subjects with raw scores of neuropsychological tests, we observed some very significant correlations. The T2-RT in right cerebellum (RC; r= 0.64, $p\leq0.001$) and left middle frontal gyrus (LMFG; r=0.73, $p\leq0.001$) was positively correlated with dysfunction scores of Kohs block. Similarly, T2-RT in left para hippocampal gyrus (PHCG; r=-0.49, $p\leq0.01$) and left superior temporal gyrus (LSTG; r= -0.48, $p\leq0.01$) were negatively correlated with memory scores. T2-RT in RC (r=0.55, $p\leq0.05$) and LCaG (r= 0.57, $p\leq0.01$) was positively correlated with dysfunction scores of NB test. T2-RT in left middle frontal gurus (LMFG; r= 0.54, $p\leq0.01$) and left calcarine gyrus (LCaG; r=0.79, $p\leq0.01$) was positively correlated with dysfunction scores of BG test. These T2-RT alterations observed in our study are suggestive of unseen tissue disruption or demyelination or gliosis in chronic alcoholism, which in part, underlie the impaired cognitive abilities in these subjects as evident by our correlation analysis.

Conclusion: Our results are consistent with the previous studies suggesting tissue disruption or gliosis or demyelination as a possible reason for prolonged T2-RTs. This damage to brain tissue which is evident as prolonged T2-RT could possibly underlie the impaired cognitive abilities noticeable in alcohol dependent subjects.

References

1. Agartz I, Saaf J, Wahlund LO, Wetterberg L (1991). T1 and T2 relaxation time estimates and brain measures during withdrawal in alcoholic men. Drug and Alcohol Dependence, 1991; 29: 157-169.
2. Pell GS, Briellmann RS, Waites AB, Abbott DF, Jackson GD (2004). Voxel-based relaxometry: a new approach for analysis of T2 relaxometry changes in epilepsy. Neuroimage, 2004; 21:707–713.

Table 1: Results for the whole brain VBR analysis for Alcohol dependents> controls contrast

Area	MNI coordinates (x, y, z)			Activation size (voxels)	T value
RC	38	-46	-44	166	5.22
RPHG	-18	-10	-24	137	8.05
RACC	2	36	-4	117	5.03
LSTG	-50	2	-2	77	4.98
LCaG	-2	-64	10	75	5.01
LC	-18	-38	50	62	5.95
LMFG	-28	32	42	60	5.75
LPHG	20	-20	17	53	5.21

Table 2: Results for ROI analysis in both the groups

ROI placement	Alcohol dependent subjects (n=25)	Control subjects(n=25)
RC	84.05± 2.1	73.8 ±2.1*
LC	80.53 ±2.2	74.19 ±3.5*
LPHG	71.65 ±1.4	68.63 ±2.2*
RPHG	70.51±1.2	69.52±1.1
RACC	70.29± 6.1	69.3 ±4.4
LSTG	67.26 ±4.1	58.2 ±6.9*
LCaG	74.06 ±5.6	66.84± 4.3*
LMTG	70.69 ±4.4	65.62± 8.1*

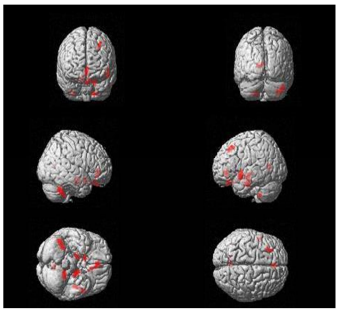

Fig.1 Results of VBR analysis showing regions with increased T2-RT in alcohol dependent subjects as compared to controls.