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ECT AND T_2 RELAXOMETRY : A STATIC WATER PROTON MAGNETIC RESONANCE IMAGING STUDY

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ABSTRACT

The aim of the study was to detect brain oedema with electroconvulsive therapy (ECT). Magnetic resonance imaging (MRI) T_2 relaxation time which is an indicator of brain water was measured one day prior to the first ECT and at two hours after second ECT in five depressive patients prescribed right unilateral (RUL) ECT. MRI T_2 relaxation time was measured in hippocampus (Hc) and thalamus (Th) bilaterally. No significant change in T_2 relaxation time following ECT occurred in any of the four regions. ECT did not produce any detectable brain oedema and hence the treatment may be considered safe. The study needs to be replicated in a larger sample and also in bilateral (BL) ECT patient group.

Key words : Magnetic resonance imaging, electroconvulsive therapy, depression

MRI is a non-invasive procedure to measure structural changes in the brain. That ECT results in brain damage is not supported in literature. Animal studies using electroconvulsive shock have shown only subtle changes in brain (Petito et al.,1977). Recent review on long term effects of ECT have shown no structural changes in the brain on MRI (Devanand et al.,1991, Coffey et al.,1991). However, these studies were based on gross anatomical changes rather than fine alterations of the water levels in the brain. If ECT were to have any effects, it could be at the level of alterations in the water content of the brain. Computerized tomography scans done immediately following ECT failed to demonstrate any changes in the brain (Jayakumar et al.,1992). Recent studies have hypothesized that ECT produces damage to the blood brain barrier (BBB) resulting in the brain oedema (Scott et al.,1990). Brain oedema is detectable by MRI T_1 and T_2 relaxation times. This damage in BBB was implicated to be associated with short term memory disturbance (Diehl et al.,1994). No attempt has hitherto been made to replicate these findings. This study was conducted with the aim of detecting changes in MRI T_2 relaxation time two hours after second RUL ECT in our population.

MATERIAL AND METHOD

Five right handed (3 males and 2 females) consenting patients with severe depressive disorder (ICD-10; WHO,1992) constituted the sample. The mean age of the sample was 34.6 years (range 26-46 years) and had a mean score of 28.6 (range 24-33) on the 17-item Hamilton Rating Scale for depression (Hamilton,1960). Inclusion criteria included : first episode of depression, drug naive and none had received ECT for the current episode. Patients with mental retardation, substance abuse/dependence, seizures or suffering from any other organic disorders were excluded from the study. All patients received modified RUL ECT under general anesthesia using thiopentone (3 mg/kg bw) and succinylcholine (0.75 mg/kg bw) under EEG monitoring. Stimulus dose was assessed

using titration method at first ECT session. All patients received moderately (2-2½ times threshold) supra threshold dose at second ECT sessions. All patients had seizures at the very first stimulus in second ECT session.

All patients underwent MRI scanning one day prior to first ECT and two hours after second ECT. MRI was measured using magneton vision 1.5 tesla whole body superconducting system. MRI T_2 relaxation time was measured by a neuroradiologist who was unaware whether the particular scan was pre- or post-ECT scan. Data for T_2 quantification was collected with Dual Echo Multi Planar (DEMP) sequences. Regional T_2 values were determined using proprietary software. The signal intensity was recorded for a 20 mm^3 uniform defined circular region of interest (ROI) placed within each selected brain region. The ROI included Hc and Th (implicated in memory; Lezak,1995) in both the cerebral hemispheres. Paired-sample t-test statistic was used to measure changes in MRI T_2 relaxation time from pre- to post-ECT sessions. Significance (α) was set at $p<0.05$.

RESULTS

There was no significant change in the MRI T_2 relaxation times before first ECT and two hours after second ECT in any of the ROI studied (Table)

TABLE 1
MRI T_2 RELAXATION TIME (MSEC) {MEAN±SD (RANGE)}

ROI	Pre-ECT	Post-ECT	t=; p=; sig
Hippocampus			
Right	152.4±16.8 (133-175)	156.2±11.5 (140-165)	0.89; 0.42; ns
Left	156±14.1 (134-170)	149.8±12.9 (132-164)	0.72; 0.51; ns
Thalamus			
Right	106.2±5.2 (99-112)	104.6±5.4 (97-110)	0.81; 0.46; ns
Left	108±4.7 (104-116)	106.6±2.1 (104-109)	0.86; 0.44; ns

DISCUSSION

This study was carried out to detect changes in MRI T_2 relaxation time with ECT. T_2 relaxation time was measured as it is more sensitive than T_1 to index brain water content (Bederson et al.,1986). MRI before ECT and two hours after second ECT did not differ significantly in T_2 relaxation times. No change was detected in any of the four brain regions. These regions were specifically chosen as they have been implicated in memory functions (Lezak,1995) and that these regions are at risk for damage in chronic epileptics (Adams,1979). The findings are in agreement with Scott et al. (1990) where increase was found only in MRI T_1 relaxation time following ECT but not in T_2 relaxation time. Mander et al. (1987) used only MRI T_1 relaxometry.

However these findings differ from Diehl et al. (1994) in which the methodology including MRI standards were similar to our study. Hence, the negative results of the present study merit explanation. (a) RUL ECT may not produce any structural change in brain detectable on routine MRI examination. (b) The increase in MRI T_2 relaxation time after the second ECT may not be of sufficient magnitude. Therefore a cumulative effect across multiple ECT treatments should also be considered. (c) Small ROI were used in this study; increasing the ROI and thereby the signal may provide a larger magnitude of change. (d) Small sample size could result in a type-II error. (e) It may be noted that Diehl et al. (1994) used one-tailed t-test statistics and reported significant difference in T_2 relaxation times whereas the present study used two-tailed t-test. It is arguable that brain-water changes following ECT, if any, are transient and reversal takes place within two hours after seizure. However, scott et al. (1990) and Mander et al. (1987) found increase in MRI T_1 relaxation time two hours and 4-6 hours respectively following ECT. Our study showed no such change indicating no brain-oedema following ECT.

There is also a need to measure the acute effects of BLECT using MRI. Studies using magnetic resonance spectroscopy and diffusion weighted scan may help in further understanding the changes in brain after ECT. In conclusion, RUL ECT does not produce any acute changes

in brain parenchyma detectable by MRI. It can hence be considered a safe procedure.

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