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Alcohol Dependence Syndrome Patients: Cortical Atrophy Score on MRI

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ABSTRACT
Chronic ethanol intoxication may lead to loss of subcortical white matter, cerebral atrophy, Wernicke's encephalopathy, Marchiafava-Bignami disease, osmotic demyelination syndrome, etc. Moderate/heavy alcohol consumption in older people has been associated with reduced total brain volume, increased ventricle size. Several studies have shown that abstinence can reverse much of the cognitive damage caused by heavy drinking. This is a hospital based, cross sectional case study conducted on all alcohol dependence syndrome patients attending outpatient and inpatient department. A total of 55 patients in the age group of 18-50 yrs, full filling the inclusion criteria were included in the study which was conducted over a period of 18 months. MR imaging examination was performed on a Siemens Magnetom Avento 1.5 Tesla MR system. Routine brain protocol sequences like Axial-T1, T2, FLAIR, SWI, DWI, Sagittal-T2, Coronal-T2 and MPRAGE were performed. In cortical atrophy, the predominantly involved lobe was frontal lobe which showed more atrophic changes compared to other lobes, followed by parietal and temporal lobe atrophy, least atrophy changes were seen in occipital lobe.

INTRODUCTION

Alcohol has been repeatedly shown in both animal and human studies to cause cerebral volume loss and functional cognitive deficits. Even in adolescents who consume large amounts of alcohol in a binge-type pattern, there is evidence for volume loss in areas such as the rostrum of the corpus callosum and cerebellum. Abstinence from alcohol has been seen to cause neuronal regeneration and often partial reversal of clinical effects and is the mainstay of management[1]. Neuronal damage especially in a binge-type pattern of drinking has been suggested to be due to the induction of pro-inflammatory cytokines and oxidative enzymes that result in neuronal death. As a consequence, there is wallerian degeneration and resultant reduction in white matter volume[2]. Computed tomography and MRI studies of chronic alcohol abuse subjects demonstrate diffuse loss of both grey and white matter affecting the frontal lobes, limbic system and cerebellum compared to control subjects with relative sparing of the corticospinal tracts. Hepatic encephalopathy (HE) is a functional and potentially reversible syndrome occurring during acute and chronic liver failure or after porto systemic shunt surgery. It is characterized by psychiatric, cognitive, and motor abnormalities (Flapping tremors). Pathogenic mechanisms responsible for HE are due to the accumulation in blood of several compounds like manganese and ammonia, which can then enter the brain and induce disturbances in astrocyte and neuron function. The substance has a neurotoxic effect, inducing reactive gliosis and selective neuronal loss in basal ganglia and midbrain structures[3]. He can occur during acute liver malfunction which can be from any cause or can complicate chronic liver disease. He clinically manifests as a neuropsychiatric syndrome manifesting as a wide spectrum of psychiatric and behavioral disturbances, as well as motor disorders. In acute HE, T2 prolongation may affect the cerebral cortex. In chronic hepatic encephalopathy (CHE), foci of T2 hyperintensities resembles those seen in small vessel disease. The chronic phase is characterized by symmetric T1 hyperintensities noted in the basal ganglia (more often the globuspallidus), the subthalamic nucleus, tectal plate, hypothalamus, and adenohypophysis. The T1 hyperintensity is caused by deposition of manganese. DWI can show diffusion restriction with corresponding decrease in ADC[4]. MRS depicts an increase in the glutamine and glutamate peak and a decrease in the myoinositol and choline peaks.

MATERIALS AND METHODS

This is a hospital based, cross sectional case study conducted on all alcohol dependance syndrome patients attending outpatient department and inpatient admitted in hospital. A total of 55 patients in the age group of 18-50 yrs, full filling the inclusion criteria were included in the study.

Inclusion Criteria:

- Patient willing to give informed consent.
- Patients aged between18 to 50 yrs who have been diagnosed with alcohol dependence syndrome according to the International Classification of Disease criteria (ICD-10).

Exclusion Criteria:

- Patient not willing to give informed consent.
- Patient with a history of major medical and neurological illness.
- Patient with a history of major psychiatric illness.
- Patient with a history of seizure disorder unrelated to alcohol consumption.
- Patient with contraindication for MRI scans such as Pacemakers, metallic implants or metallic foreign body.
- Patient with a history of other substance abuse (except nicotine).

The Study group consisted of 55 patients, who fulfilled the alcohol dependence syndrome criteria according to the International Classification of Disease criteria (ICD-10). Alcohol use disorder identification test scale was applied and individual with alcohol dependence score of >15 (for men) were considered for study. Hemodynamically stable patients, referred to the department of Radio diagnosis BMCRI, for imaging studies were included in the study after duly taking an informed consent. Details of the study were collected and documented as per the proforma attached. All patients were screened before entry into the MRI scanning room for ferromagnetic objects, cardiac pacemakers, aneurysm clips etc. Patients were examined in the supine position on the MRI machine after proper positioning and immobilization of the head was obtained. The head coil was used for the scan. Initial topogram of the head was obtained and sequences were planned according to the MRI brain (plain without contrast) protocol.

RESULTS AND DISCUSSIONS

19/22 (86%) patients of cortical atrophy showed atrophy of frontal lobe. 18/22(81%) patients of cortical atrophy showed parietal lobe atrophy. Similarly temporal lobe involvement were seen in 16/22(72%) and occipetal lobe involvement were seen in 9/22 (40%) cortical atrophy patients.

In our study out of 22 cortical atrophy patients, 11/22 (50%) patients showed cortical atrophy score of 1. 8/22(36.3%) patients show score of 2. Remaining 3/22 (13.6%) showed no changes of atrophy.

In our study out of 22cortical atrophy patients, 12/22 (54.7%) patients show cortical atrophy score of 1.

Table 1: Cortical atrophy distribution among four lobes

Lobes	Cortical atrophy
Frontal	86%
Parietal	81%
Temporal	72%
Occipital	40%

Table 2: Cortical atrophy score distribution in frontal lobe

Cortical atrophy score	Frontal lobe Involvement (%)
0	13.6%
1	50.1%
2	36.3%

Table 3: Cortical atrophy score distribution in parietal lobe

Cortical atrophy score	Parietal lobe Involvement (%)
0	18.1%
1	54.7%
2	27.2%

Table 4: Cortical atrophy score distribution in temporal lobe

Cortical atrophy score	Temporal lobe Involvement (%)
0	27.3%
1	72.7%
2	0%

Table 5: Cortical atrophy score distribution in occipital lobe

Cortical atrophy score	Occipital lobe Involvement (%)
0	59.1%
1	40.9%
2	0%

Table 6: Cortical atrophy score distribution involving frontal lobe among category 2 and category 3 patients

Score	Category 2	Category 3
0	50%	0%
1	33.33%	56.2%
2	16.66%	43.8%

X2 =9.339, p value = 0.009

Table 7: Cortical atrophy score distribution involving parietal lobe among category 2 and category 3 patients

Score	Category 2	Category 3
0	33%	12.5%
1	50%	56.3%
2	17%	31.2%

X2 =1.413, p value = 0.49.

Table 8: Cortical atrophy score distribution involving temporal lobe among category 2 and category 3 patients

Score	Category 2	Category 3
0	50%	18.7%
1	50%	81.3%

X2 =2.148, p value = 0.149.

Table 9: Cortical atrophy score distribution involving occipital lobe among category 2 and category 3 patients

Score	Category 2	Category 3
0	83%	50%
1	17%	50%

X2 =2.006, p value = 0.157.

Table 10: Cortical atrophy distribution among category 1, category 2 and category 3 patients

Cortical atrophy	Category 1	Category 2	Category 3
Present	0%	20%	88.9 %
Absent	100%	80%	11.1%

X2 =27.593, p value = 0.002

Table 11: Mean duration of problem drinking in patients with cortical atrophy compared to those without cortical atrophy

	Numbers	Mean duration of problem drinking
No cortical atrophy	33	12.91
Cortical atrophy	22	23.05

6/22(27.2%) patients show score of 2. Remaining 4/22 (18.1%) showed no changes of atrophy.

In our study out of 22 cortical atrophy patient, 16/22 (72.7%) patients show cortical atrophy score of 1. 6/22(27.3%) patient showed no changes of atrophy.

In our study out of 22 cortical atrophy patients, 9/22 (40.9%) patients show cortical atrophy score of 1. 13/22(59.09%) patient showed no changes of atrophy.

In our study, 2/6 (33.3%) of Category 2 patients showed score of 1, 3/6(50%) and 1/6 (16.66%) showed score of 0 and 2 respectively. 9/16 (56.2%) of category 3 patients showed score of 1,7/16(43.8%) showed score of 2. Frontal lobe was most common lobe to get atrophied.

Most of the atrophic changes were seen in patients who had duration of alcohol intake of greater then 20 yrs, there was significant association between duration of drinking and frontal lobe atrophy and results were statistically significant ($p<0.05$).

In our study 3/6 (50%) of Category 2 patients showed score of 1, 2/6(33.3%) and 1/6 (17%) showed score of 0 and 2 respectively. 9/16 (56.3%) of Category 3 patients showed score of 1, 2/16(12.5%) and 5/16 (31.2%) showed score of 0 and 2 respectively. Most of the atrophic changes were seen in patients who had duration of alcohol intake of greater than 20 yrs but the results were statistically not significant ($p> 0.05$).

In our study, 3/6 (50%) of Category 2 patients showed score of 1, remaining 3/6(50%) showed no changes of atrophy. 13/16 (81.3%) of Category 3 patients showed score of 1, remaining 3/16(18.7%) showed no changes of atrophy. But results of association between atrophy and duration of drinking wa not statistically significant (P value > 0.05).

In our study,1/6 (17%) of Category 2 patients showed score of 1, remaining 5/6(83.3%) showed no changes of atrophy. 8/16 (50%) of Category 3 patients showed score of 1, remaining 8/16(50%) showed no changes of atrophy. But results of association between atrophy and duration of drinking was not statistically significant (P value > 0.05)

16/18 (88.9%) patients of category 3 showed changes of cerebral atrophy. 6/30 (20%) patients of Category 2 showed atrophy changes.

Most of the atrophic changes were seen in category 3 patients, there was significant association between duration of drinking and cortical atrophy and results were statistically significant ($p<0.05$).

There is a definite difference in the duration of problem drinking between those who were normal and those with cortical atrophy, namely 12.9 years versus 23.05 years for those with cortical atrophy. The statistical significance of this difference by t test is t = 8.7, significant at < 0.05 level of probability.

Most common finding seen in our study subjects was cerebral atrophy which was seen in 22/55 (40%) patients. Most of the atrophic changes were seen in group 3. 16/18 (88.9%) patients of group 3 showed changes of cerebral atrophy. 6/30 patients of group 2

showed atrophy changes. No atrophy changes were seen in group 1 patients. In a similar study done by john[5] which was a cross-sectional study of 50 patients, 29/50(58%) patients showed cerebral atrophy. In another cross-sectional study done by Somsubhra Chattopadhay[6], cortical atrophy changes were seen in 60% of alcohol dependence patients. In patients with cerebral atrophy most common lobe to show changes of atrophy was frontal lobe.19/22 (86%) patients of cortical atrophy showed atrophy of frontal lobe. Next common lobe to be involved was parietal lobe. 18/22(81%) patients of cortical atrophy showed parietal lobe atrophy. Similarly temporal lobe involvement were seen in 16/22(72%) and occipital lobe involvement were seen in 9/22 (40%) cortical atrophy patients. Our study findings were consistent with similar study done by Sullivan[7], which showed that cortical volume loss are more in frontal lobes, followed by parietal, temporal and occipital lobes respectively. Another study done by Somsubhra Chattopadhay[6] also showed frontal and parietal lobes to be the predominantly involved lobes, with temporal and occipital lobes being less involved.

Extent of Involvement of Cerebral Cortex: Frontal Lobe:

- 11/22 (50%) patients showed cortical atrophy score of 1. 8/22(36.3%) patients show score of 2. Remaining 3/22 (13.6%) showed no changes of atrophy.
- 2/6 (33.3%) of Category 2 patients showed score of 1, 3/6(50%) and 1/6 (17%) showed score of 0 and 2 respectively.
- 9/16 (56.2%) of category 3 patients showed score of 1, 7/16(43.7%) showed score of 2.

Parietal Lobe:

- 12/22 (54%) patients show cortical atrophy score of 1. 6/22(27.2%) patients show score of 2. Remaining 4/22 (18.1%) showed no changes of atrophy.
- 3/6 (50%) of Category 2 patients showed score of 1, 2/6(33.3%) and 1/6 (17%) showed score of 0 and 2 respectively.
- 9/16 (56.2%) of Category 3 patients showed score of 1, 2/16(12.5%) and 5/16 (31.2%) showed score of 0 and 2 respectively.

Temporal Lobe:

- 16/22 (72.7%) patients show cortical atrophy score of 1. 6/22(27.2%) patient showed no changes of atrophy.
- 3/6 (50%) of Category 2 patients showed score of 1, remaining 3/6(50%) showed no changes of atrophy.
- 13/16 (81.2%) of Category 3 patients showed score of 1, remaining 3/16(18.7%) showed no changes of atrophy.

Occipital Lobe:

- 9/22 (40.9%) patients show cortical atrophy score of 1. 13/22(59.09%) patient showed no changes of atrophy.
- 1/6 (17%) of Category 2 patients showed score of 1, remaining 5/6(83.3%) showed no changes of atrophy.
- 8/16 (50%) of Category 3 patients showed score of 1, remaining 8/16 (50%) showed no changes of atrophy.

72.7% of the atrophy changes were seen in category 3 patients compared to 27.3% atrophy changes seen in category 2 patients. There was a positive association noted between duration of drinking and atrophic changes and the results were statistically significant (P value < 0.005). This association was similar to the john et al. study[5]. Global cortical atrophy scale score of 2 which indicates moderate amount of atrophy were seen only in frontal and parietal lobes. In our study Frontal lobes showed more severe atrophy changes when compared to parietal lobe. There was a positive association noted between duration of drinking and frontal lobe atrophic changes and the results were statistically significant (P value<0.005). Most of the patients who showed score 2 cerebral atrophy changes belonged to patients who had history drinking for greater than 20 years. One study conducted by kubota [8] showed increased incidence of frontal lobe shrinkage in chronically heavy drinkers. There is a definite difference in the duration of problem drinking between those who were normal and those with cortical atrophy, namely 12.9 years versus 23.05 years for those with cortical atrophy. The statistical significance of this difference by t test is t = 8.7, significant at < 0.05 level of probability. This result was similar to study done by john[5]. Both temporal and occipital lobes showed atrophy score of 1 indicative of mild atrophy. (40.9%) and (72.7%) patients show cortical atrophy score of 1 in occipital lobe and temporal lobe respectively. Atrophy score of 2 was not seen in both temporal and occipital lobes. Thus there is less severe involvement of both temporal and occipital lobes compared to frontal and parietal lobes. But the results of association between duration of drinking with parietal, temporal and occipital lobes were not statistically significant with P value >0.005.

CONCLUSION

In patients with cerebral atrophy most common lobe to show changes of atrophy was frontal lobe.19/22 (86%) patients of cortical atrophy showed atrophy of frontal lobe. Next common lobe which showed

changes of atrophy was parietal lobe. 18/22(81%) showed parietal lobe atrophy. Similarly temporal lobe involvement were seen in 16/22(72%) and occipital lobe involvement were seen in 9/22 (40%) cortical atrophy patients.

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