CT BRAIN CHANGES IN CHRONIC ALCOHOLIC PATIENTS IN KARAIKAL

Dr. Chandra kala, Dr.Kanimozhi, and Dr.AvikalpKumar

Department of Radiology, Vinayaka Missions, Medical College and Hospital, Vinayaka Mission Research Foundation (Deemed to be University), Karaikal, Puducherry, India

Background Brain damage is caused by chronic alcoholism. Rural India has few published data. The aim of this study is to examine changes in the brain of chronic alcoholics living in rural areas using computed tomography (CT).

Methods An analysis of chronic alcoholic patients referred from addiction centers at our tertiary care hospital over the course of two years was conducted in this prospective cross-sectional study. Students' t-test and chi-square test comparisons were performed on nonalcoholic controls who were aged and sex-matched. In order to calculate the correlation coefficient, Pearson's correlation coefficient was used. The study included patients and controls from 30 to 45 years of age and 46 to 60 years of age. In each case, a CT scan was performed without contrast and indices of brain damage were calculated from the CT scan.

Results A total of 50 alcoholics were included in the study, 35 of whom were aged 30 to 45 and 15 of whom were aged 46 to 60. There were 50 non-alcoholic, age-matched controls. In the older age group, ethanol values were higher than in the younger age group. Both groups had

significantly higher CT scan indices of ventricular changes, cortical changes, and subcortical changes than controls. Hemoglobin values decreased significantly in both age groups and indices of brain damage increased significantly with increasing alcoholism grade in both age groups.

Conclusion A prospective epidemiological study shows that brain atrophy CT indices are significantly altered by ethanol consumption, and that ethanol consumption duration and amount are related. There is a strong link between chronic alcoholism and brain atrophy, as observed in this study.

Keywords: A study of alcoholism, ventricular index, dilation, computed tomography scan, brain atrophy, epidemiological

Introduction

Drinking excessive amounts of alcohol in a habitual manner can lead to chronic alcoholism. Different types of alcohol abuse can affect and damage the brain in different ways, depending on a variety of factors, including age, gender, history of abuse, diet, and vulnerability of specific brain areas. In different subgroups of alcohol-induced brain injuries, multiple methods have been used for studying etiopathogenesis. Cognitive and emotional functions can be studied using behavioral neuroscience techniques. The extent of pathology and abnormal neurotransmitter levels in the brain have also been studied with postmortem brain biopsies. Damaged brain areas can be identified using neuroimaging techniques [1].

In addition to cerebral atrophy, dilated ventricles, and cerebellar atrophy, chronic alcoholism has numerous alcohol-related effects on the brain [2].

In rural India, there are few published studies on the effects of alcohol on the brain of alcoholics. It was therefore the goal of this study to compare brain changes in chronic alcoholics in rural Indian patients with changes in non alcoholics of the same age and grade of alcoholism and to correlate these changes with the grades of alcoholism [3].

Materials and Methods

A cross-sectional prospective study was conducted at our tertiary care teaching institute's department of radiodiagnosis, serving rural communities as a referral center. Deaddiction clinic referred the majority of the patients. In accordance with institutional ethics guidelines, the study was approved [4].

Participants in the study were chronic alcoholics referred to the radiology department between the ages of 30 and 60. In order to define alcohol dependence syndrome, we used the 10th revision of the International Classification of Diseases. Systemic medical illnesses including hypertension, diabetes mellitus, abuse of any other drugs, head injury, intellectually disabled patients, and patients with cerebrovascular diseases, infectious diseases, degenerative diseases, and brain tumors were not included in the study. The presence of stigmata of liver cirrhosis, an abdominal examination, and an abdominal ultrasonography were used to exclude cirrhosis of the liver [5].

A control group of 50 non-alcoholics age- and sex-matched to the group under study was included as a comparison. Controls had a normal CT scan and were non-alcoholic patients with headache or dizziness, no systemic illnesses, drugs, or trauma. As a control, normal age-related

changes in the brain were compared with the changes in the brain of alcoholics. There were two age groups of patients and controls: 30 to 45 and 46 to 60 [6].

Each case was documented on a proform after an informed written consent was obtained. Philips MDCT (16 slice) was used to perform a CT scan on the patient [7].

Nonenhanced head CT scan was done in each case, from base of head to vertex in supine position, arms by side of the body, scanogram length of 256 mm, plane of imaging: 15 degrees cephalic tilt to inferior orbital meatal line and parallel to skull base to reduce the radiation dose to the orbits. There were 140 kVs, 120 mAs, 250 mm wide fields of view, and a scanning time of 3 minutes using 140 kVs with 120 mAs [8].

Different indices such as the bicaudate index, ventricular index, Evan's index, and maximum transverse diameter of third ventricle were used to assess the ventricular size and cerebral and cerebellar changes [9].

The bifrontal index measures the maximum width of the anterior horns of the lateral ventricles as compared with the inner skull width at the same level.

A bicuated index is defined as the *width of the lateral* ventricles at the same level as the inner skull.

An index derived from the relationship between the widths of the anterior horns of the lateral ventricles and the diameter of the inner skull [10].

In the lateral ventricle, the venetricular index indicates how wide the lateral ventricle is in relation to the anterior horns, which have the same width.

Atrophy of the cortex: The sum of the widths of the four largest sulci at the two highest scanning levels divided by the inner skull's largest diameter [11].

A correlation was found between the patients' brain CT scan results and their hemoglobin levels.

In order to convert alcohol consumption into standard ethanol consumption in grams, various amounts and types of alcohol have been converted into conversion factor 5:

1 mL of ethanol = 0.79 g of pure ethanol. For example, 40 mL of whisky $(40\%) \times 0.79$ (conversion factor) = 12.6 g of ethanol [12].

- 40 mL of whisky $(42.8\%) \times 0.79$ (conversion factor) = 13.5 g of ethanol.
- 40 mL of country liquor $(40\%) \times 0.79$ (conversion factor) = 12.6 g of ethanol.
- 40 mL of illicit liquor $(56\%) \times 0.79$ (conversion factor) = 17.6 g of ethanol.

Statistical Analysis

Categorical data were expressed as percentages and continuous variables as mean + standard deviation. Student's t-test and chi-square test were used to compare indices for alcoholics between 30 and 45 years of age and 46 to 60 years of age with controls of similar age. Based on Pearson's correlation coefficient, a correlation coefficient was calculated [13].

To determine if ethanol intake can be correlated with cerebral and cerebellar atrophies, several stepwise analyses were performed with adjustments for age. Statistical significance was determined by a p-value of 0.05 [14].

Results

Among the 50 participants,35was between the ages of 30 and 45 (younger age group) and 15 were between the ages of 46 and 60 (older age group). Fifty non-alcoholic controls were included in the study, 25 for each age group. In comparison with controls, alcoholics had a mean age of 43 years and 43 years, respectively. Younger people tended to consume country liquor while older people tended to consume whiskey. In comparison to younger age groups, older age groups consumed more ethanol. Most younger patients had only consumed alcohol for a short period of time (2-5 years). In the older group, the highest proportion of alcoholics (39.2%).

Discussion

CT scan is a widely available, reliable, and economical imaging technique that can detect gross neuropathological changes in the brain such as cortical atrophy, ventricular enlargement, and other intracranial changes. Noncontrast CT head scan in alcoholics in our study showed marked changes in all the CT indices of cortical, ventricular, and subcortical changes [16].

These indices were significantly altered when compared with age-matched controls and were related to the years of alcohol consumption and the daily amount of alcohol consumed.

As also reported by other authors, we observed diffuse and fairly symmetrical cerebral cortical atrophy, dilated lateral and third ventricles, and shrinkage of the cerebellar hemisphere and vermis (80% of alcoholics in 30–45 and 46–60 years age groups, respectively) [17].

Frontal lobes showed the most marked shrinkage (78 and 86% in younger and older age groups, respectively) with widening of the interhemispheric fissures and frontal horns of the lateral ventricles. Similar observations have been made by others [18].

The pathophysiology of alcohol effects on the brain has been studied and numerous hypotheses proposed. Alcohol and its metabolite directly cause neurotoxic effect resulting in demyelination and degeneration of the nervous tissue and indirectly by decreasing the blood flow to certain areas of cerebrum and cerebellum due to reduced flow rates in vessels, stasis, and aggregation of blood cells. The above effects are proportional to the amount of ethanol in blood [19].

Pathological and neuroimaging studies show that chronic alcoholism leads to diffuse white matter volume loss with relative sparing of the gray matter. Few neuroimaging studies have suggested a causative role of smoking tobacco in brain atrophy in alcoholics. This hypothesis was however refuted by a recent pathological study that measured regional brain volumes and found no significant effects of smoking on either global or selected regional gray matter volumes in smokers or smoking alcoholics. We did not study the effects of smoking [20].

As reported by Whitehead et al, we also observed a significant reduction in hemoglobin levels with increase in grades of alcoholics in both age groups.

We did not include patients older than 60 years because age-related atrophic changes of the brain start manifesting at this stage. Female's brains are more vulnerable to alcohol-related changes than males. We included only males in patient and control groups, since alcohol intake by females is considered a social stigma in India, especially in the rural population [21].

Conclusion

We conclude from our epidemiological study that chronic alcoholism is a risk factor for brain atrophy. There is more or less symmetrical shrinkage of the cortex of the cerebral hemispheres with dilation of the lateral and third ventricles. Alcoholics had a greater degree of central and frontal brain atrophy as compared with controls [22].

Reference:

- Crews FT, Bechara R, Brown LA, Guidot DM, Mandrekar P, Oak S, Qin L, Szabo G, Wheeler M, Zou J. Cytokines and alcohol. Alcoholism: Clinical and Experimental Research. 2006 Apr;30(4):720-30.
- 2. González-Reimers E, Santolaria-Fernández F. Brain atrophy in alcoholics. In Handbook of Behavior, Food and Nutrition 2011 (pp. 2993-3010). Springer, New York, NY.
- 3. Donadon MF, de Lima Osório F. Recognition of facial expressions by alcoholic patients: a systematic literature review. Neuropsychiatric disease and treatment. 2014;10:1655.
- Mahapatra R, Kaliyappan A, Chinnakali P, Hanumanthappa N, Govindarajalou R, Bammigatti C. Prevalence and Risk Factors for Resistant Hypertension: Cross-Sectional Study From a Tertiary Care Referral Hospital in South India. Cureus. 2021 Oct 14;13(10).

- Harris GJ, Jaffin SK, Hodge SM, Kennedy D, Caviness VS, Marinkovic K, Papadimitriou GM, Makris N, Oscar-Berman M. Frontal white matter and cingulum diffusion tensor imaging deficits in alcoholism. Alcoholism: Clinical and Experimental Research. 2008 Jun;32(6):1001-13.
- Allen AM, Therneau TM, Mara KC, Larson JJ, Watt KD, Hayes SN, Kamath PS. Women with Nonalcoholic Fatty Liver Disease Lose Protection against Cardiovascular Disease–a Longitudinal Cohort Study. The American journal of gastroenterology. 2019 Nov;114(11):1764.
- Poulomi M. Can Dual Energy CT using Iodine Mapping Predict the Nuclear Grade of Clear Cell Type of Renal Cell Carcinoma (Doctoral dissertation, Christian Medical College, Vellore).
- Flach PM, Thali MJ, Germerott T. Times have changed! Forensic radiology—a new challenge for radiology and forensic pathology. American Journal of Roentgenology. 2014 Apr;202(4):W325-34.
- LeMay M. Radiologic changes of the aging brain and skull. American journal of neuroradiology. 1984 May 1;5(3):269-75.
- Synek V, Reuben JR, Du Boulay GH. Comparing Evans' index and computerized axial tomography in assessing relationship of ventricular size to brain size. Neurology. 1976 Mar 1;26(3):231-.
- Pfefferbaum A, Zatz LM, Jernigan TL. Computer-interactive method for quantifying cerebrospinal fluid and tissue in brain CT scans: effects of aging. Journal of Computer Assisted Tomography. 1986 Jul 1;10(4):571-8.

- 12. Babor TF, Higgins-Biddle JC, Saunders JB, Monteiro MG. The alcohol use disorders identification test. Geneva: World Health Organization; 2001.
- Hansen JP. CAN'T MISS—Conquer Any Number Task by Making Important Statistics Simple. Part 1. Types of Variables, Mean, Median, Variance, and Standard Deviation. The Journal for Healthcare Quality (JHQ). 2003 Jul 1;25(4):19-24.
- 14. Kuo MC, Lu YC, Tai CH, Soong BW, Hu FC, Chen ML, Lin CH, Wu RM. COQ2 and SNCA polymorphisms interact with environmental factors to modulate the risk of multiple system atrophy and subtype disposition. European Journal of Neurology. 2022 Oct;29(10):2956-66.
- 15. Orchard TJ, Temprosa M, Goldberg R, Haffner S, Ratner R, Marcovina S, Fowler S, Diabetes Prevention Program Research Group. The effect of metformin and intensive lifestyle intervention on the metabolic syndrome: the Diabetes Prevention Program randomized trial. Annals of internal medicine. 2005 Apr 19;142(8):611-9.
- 16. Filley CM, Halliday W, Kleinschmidt-DeMasters BK. The effects of toluene on the central nervous system. Journal of Neuropathology & Experimental Neurology. 2004 Jan 1;63(1):1-2.
- 17. Sandhu GS, Nagrale HR. Computed tomography evaluation of brain in chronic alcoholics. Journal of Neurosciences in Rural Practice. 2020 Jan;11(01):063-71.
- Geschwind N, Galaburda AM, editors. Cerebral dominance: The biological foundations. Harvard University Press; 1984.
- 19. Pereira RB, Andrade PB, Valentão P. A comprehensive view of the neurotoxicity mechanisms of cocaine and ethanol. Neurotoxicity research. 2015 Oct;28(3):253-67.

- 20. Kril JJ, Halliday GM. Brain shrinkage in alcoholics: a decade on and what have we learned?. Progress in neurobiology. 1999 Jul 1;58(4):381-7.
- 21. Aylward EH, Nopoulos PC, Ross CA, Langbehn DR, Pierson RK, Mills JA, Johnson HJ, Magnotta VA, Juhl AR, Paulsen JS, PREDICT-HD Investigators. Longitudinal change in regional brain volumes in prodromal Huntington disease. Journal of Neurology, Neurosurgery & Psychiatry. 2011 Apr 1;82(4):405-10.
- 22. Rehm J, Baliunas D, Borges GL, Graham K, Irving H, Kehoe T, Parry CD, Patra J, Popova S, Poznyak V, Roerecke M. The relation between different dimensions of alcohol consumption and burden of disease: an overview. Addiction. 2010 May;105(5):817-43.