

Corpus callosum atrophy and cerebral blood flow in chronic alcoholics

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Abstract

The corpus callosum atrophy and cerebral blood flows were investigated in chronic alcoholics without Marchiafava–Bignami disease. Fifteen cases of chronic alcoholics and 15 age-matched healthy controls were studied. The sagittal plane of magnetic resonance imaging of the head was scanned into a computer and the corpus callosum was measured and the callosal index was calculated. Cerebral blood flows were measured using stable xenon computed tomography (CT) method. Regional cerebral blood flows in the frontal, temporal, parietal and occipital cortex, frontal, temporal and occipital white matter, caudate nucleus, putamen and thalamus were measured. The corpus callosum area, the thickness of the genu, the thickness of the trunk, the thickness of the splenium, and the callosal index were significantly smaller in the chronic alcoholic group than in the healthy control group. Blood flows in the cerebral cortex, thalamus and putamen were significantly lower in the chronic alcoholic group than in the healthy control group. Significant positive correlations were present between the corpus callosum atrophy and the cerebral cortex blood flows. Corpus callosum atrophy and decreased cerebral blood flows may be seen in chronic alcoholics without Marchiafava–Bignami disease. © 1999 Elsevier Science B.V. All rights reserved.

Keywords: Corpus callosum; Cerebral blood flow; Chronic alcoholics; Xenon computed tomography; Callosal index

1. Introduction

Marchiafava–Bignami disease, a rare neurological complication of chronic alcoholism, is well known to be characterized by demyelination and necrosis of the corpus callosum [1–4]. There are reports that the corpus callosum atrophy and decreased cerebral blood flows may be present in chronic alcoholics without Marchiafava–Bignami disease [5–22]. The damage of cerebral cortex in chronic alcoholism may result in corpus callosum atrophy and decreased cerebral blood flows. Because there are no reports on the relationship between corpus callosum atrophy and cerebral blood flows in chronic alcoholics, it was investigated in the present study.

2. Patients and methods

We studied 15 chronic alcoholics (mean age 55.4 ± 6.8 , all males) and 15 age-matched healthy males who do not drink alcohol (mean age 56.2 ± 6.9) with their informed consent. We defined chronic alcoholics as persons who drink more than 83 ml of pure alcohol equivalent every day for more than 10 years. The patients with Marchiafava–Bignami disease, Wernicke–Korsakoff syndrome, or the other central nervous system diseases were excluded. All patients drank alcohol at the night before the cerebral blood flow examination and on the day of examination all patients did not drink alcohol till the examination which was performed in the afternoon. Medications which influence cerebral blood flow were discontinued 2 weeks prior to the cerebral blood flow examination.

The sagittal plane of magnetic resonance imaging (MRI) of the head was scanned into a computer and the corpus callosum was measured using NIH image (Fig. 1) and the

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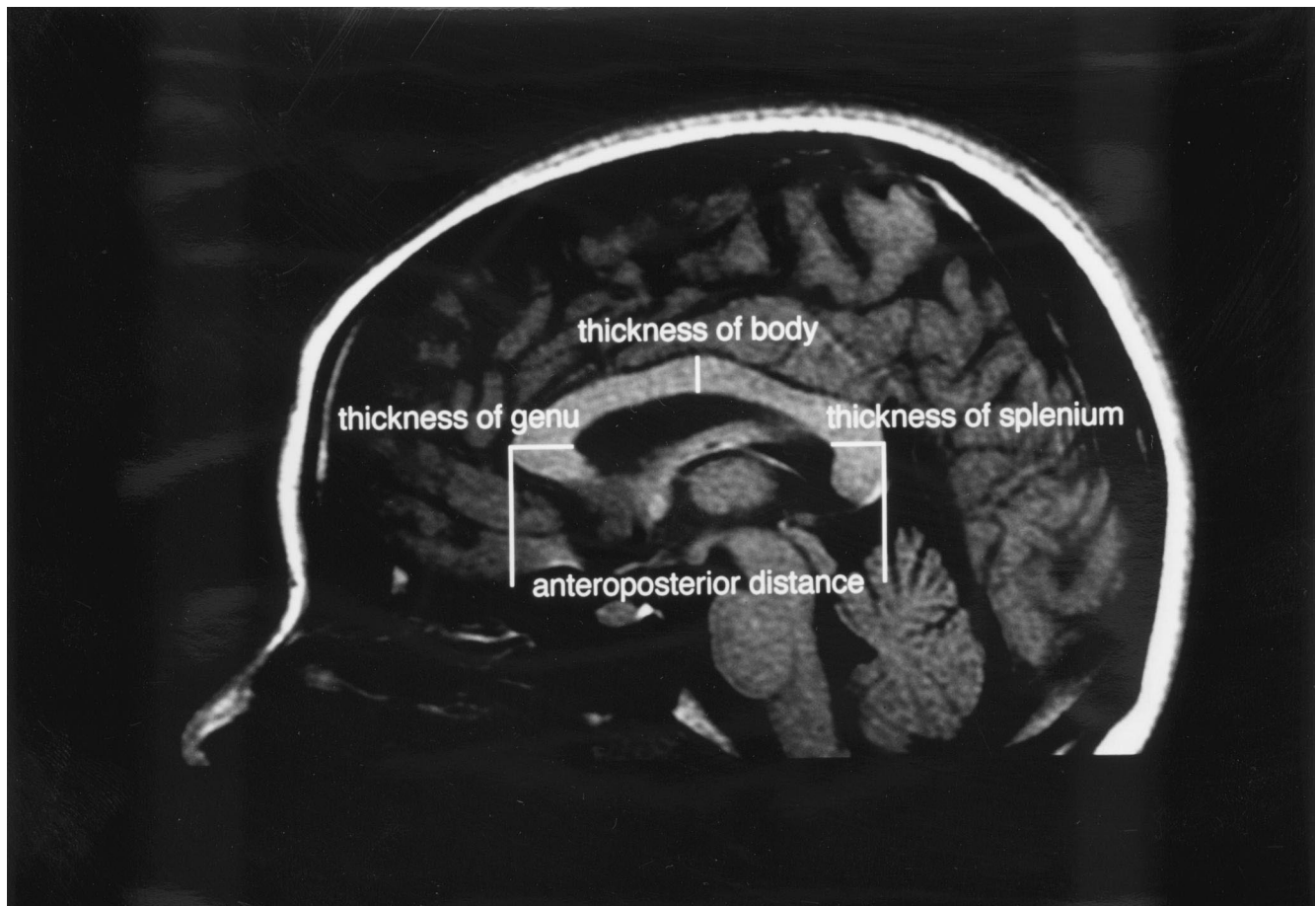


Fig. 1. The sagittal plane of T1-weighted image of head magnetic resonance imaging (MRI). The corpus callosum area, the anteroposterior distance, the thickness of the genu, the thickness of the body, and the thickness of the splenium were measured.

callosal index [corpus callosum area/(axial cerebral area + sagittal area)/2] was calculated [15].

The regional cerebral blood flow was measured using the stable xenon computed tomography (CT) method [11,12,17,18]. The basal ganglia section and the lateral ventricle section parallel to the orbito-meatal line were studied. The subjects inhaled room air followed by a mixture of 30% xenon and 50% oxygen for three minutes. Serial scanning was performed once before xenon inhalation, three times in the washin process and five times in the washout process of five minutes. The serial scanning program consisted of a total of 18 scans consisting of nine serial scans on each section. The xenon concentration in the end-tidal expired gas was continuously recorded by the thermoconductivity method. We used the xenon delivery and analysis system (AZ-7000 model, Anzai Sogyo, Tokyo, Japan) and the CT equipment (PreSage, Yokogawa Medical Systems, Tokyo, Japan). Round region of interest (ROI) with a diameter of 7 mm was used. Regional cerebral blood flows in the frontal, temporal, parietal and occipital cortex, frontal, temporal and occipital white matter, caudate nucleus, putamen and thalamus were measured.

Statistical analysis was performed using Mann–Whitney’s U tests for comparison between the two groups and using Spearman’s correlation coefficients for correlation between the measurements of the corpus callosum and the regional cerebral blood flows.

3. Results

Fig. 2 shows T1-weighted images of MRI and cerebral blood flow images of xenon CT in a chronic alcoholic and a healthy control.

Table 1 shows the mean and standard deviation of the measurements of the corpus callosum and the regional cerebral blood flows. The corpus callosum area, the thickness of the genu, the thickness of the body, the thickness of the splenium, and the callosal index were significantly smaller in the chronic alcoholic group than in the healthy control group. The blood flows in the frontal cortex, temporal cortex, parietal cortex, occipital cortex, thalamus, and putamen were significantly lower in the chronic alcoholic group than in the healthy control group.

Table 2 shows the correlation coefficients between the

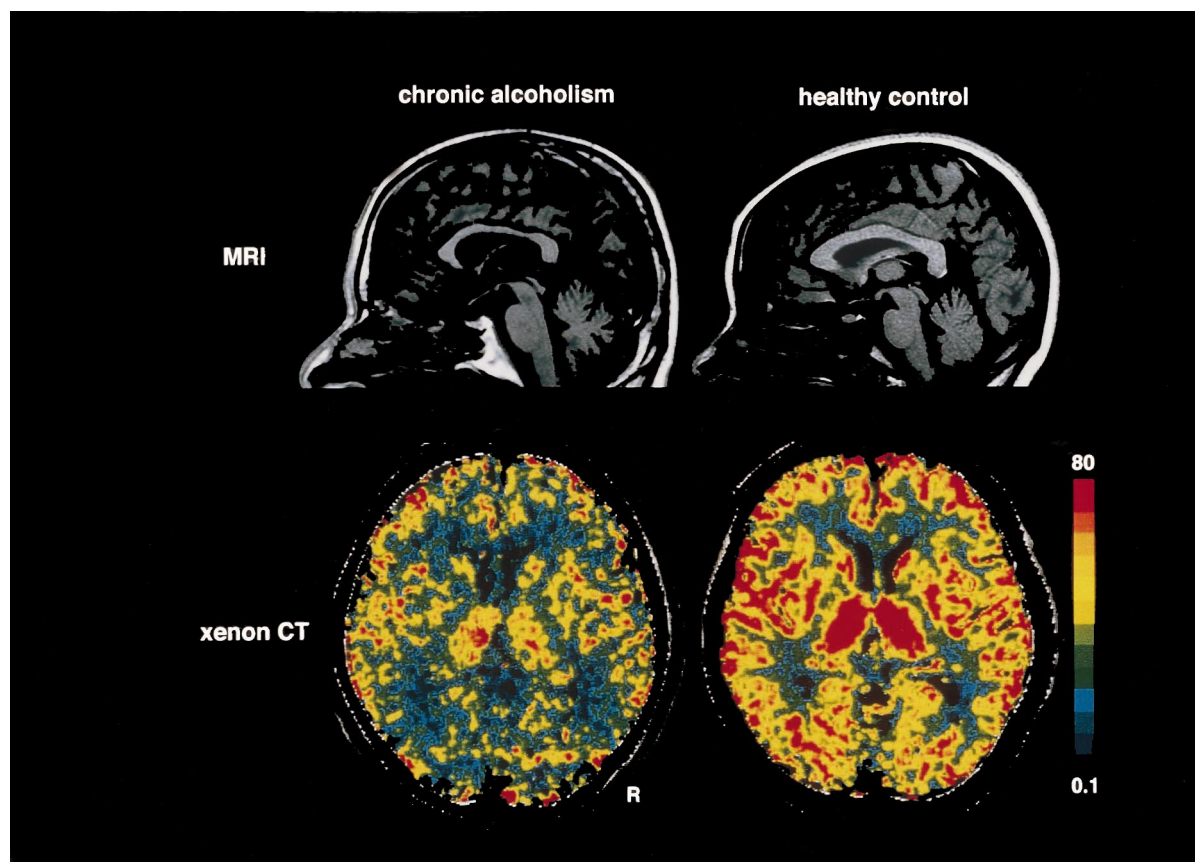


Fig. 2. The T1-weighted images of MRI and cerebral blood flow images of xenon CT in a chronic alcoholic and a healthy control. The corpus callosum atrophy and decreased cerebral blood flow were noted in a chronic alcoholic.

measurements of the corpus callosum and the regional cerebral blood flows in chronic alcoholics. A significant negative correlation was present between the corpus callosum atrophy and the lifetime alcohol intake. Signifi-

cant positive correlations were present between the corpus callosum atrophy and the cerebral cortex blood flows and between the corpus callosum atrophy and the thalamus blood flow. No significant correlations were present be-

Table 1

Mean and standard deviation of the measurements of the corpus callosum and the regional cerebral blood flows

	Chronic alcoholic group	Healthy control group
Corpus callosum area (mm ²)	445±97 ^a	568±104
Anteroposterior distance (mm)	70.3±8.2	72.7±8.5
Thickness of the genu (mm)	8.9±1.2 ^b	10.6±1.4
Thickness of the body (mm)	4.1±0.9 ^a	5.3±1.1
Thickness of the splenium (mm)	8.8±1.1 ^a	10.4±1.4
Callosal index	0.036±0.005 ^a	0.045±0.007
Frontal cortex blood flow (ml/100 g/min)	52.6±9.1 ^a	70.7±9.7
Temporal cortex blood flow (ml/100 g/min)	51.6±9.1 ^a	68.2±9.5
Parietal cortex blood flow (ml/100 g/min)	51.0±8.9 ^a	67.3±9.6
Occipital cortex blood flow (ml/100 g/min)	49.7±8.7 ^a	66.5±9.6
Frontal white matter blood flow (ml/100 g/min)	24.4±7.5	24.5±7.6
Occipital white matter blood flow (ml/100 g/min)	23.2±7.1	25.8±7.3
Thalamus blood flow (ml/100 g/min)	57.1±9.4 ^a	71.4±9.3
Caudate nucleus blood flow (ml/100 g/min)	78.2±9.6	82.4±9.4
Putamen blood flow (ml/100 g/min)	72.2±8.7 ^b	80.3±8.5

^a $P < 0.01$ vs healthy controls.

^b $P < 0.05$ vs healthy controls.

Table 2

Correlation coefficients between the measurements of the corpus callosum and the regional cerebral blood flows in chronic alcoholics

	Corpus callosum area	Thickness of genu	Thickness of body	Thickness of splenium	Callosal index
Age at examination	-0.38	-0.34	-0.40	-0.37	-0.36
Lifetime alcohol intake	-0.56 ^a	-0.54 ^a	-0.57 ^a	-0.53 ^b	-0.51 ^b
Frontal cortex blood flow	0.58 ^a	0.59 ^a	0.58 ^a	0.47	0.55 ^a
Temporal cortex blood flow	0.54 ^a	0.53 ^a	0.57 ^a	0.46	0.53 ^a
Parietal cortex blood flow	0.52 ^b	0.48	0.56 ^a	0.51 ^b	0.50 ^b
Occipital cortex blood flow	0.51 ^b	0.45	0.47	0.53 ^a	0.49 ^b
Thalamus blood flow	0.57 ^a	0.55 ^a	0.58 ^a	0.55 ^a	0.56 ^a
Putamen blood flow	0.46	0.46	0.47	0.42	0.43

^a $P < 0.01$.^b $P < 0.05$.

tween the corpus callosum atrophy and the cerebral white matter blood flows.

4. Discussion

The thickness of the body of the corpus callosum has been reported in chronic alcoholics [7,22]. But the other reports [19,20] suggested that the age and sex of the chronic alcoholics may influence the corpus callosum atrophy. In the present study, we compared the corpus callosum size between chronic alcoholic men and age-matched healthy men who do not drink alcohol. Because the corpus callosum size may be influenced by the cerebral size [23], we calculated the callosal index [15]. The significant decrease in callosal index in chronic alcoholics in the present study suggests that the corpus callosum atrophy is not due to cerebral atrophy.

Although the significant ($P < 0.05$) decrease in putamen blood flow may be due to multiple comparisons, the significant ($P < 0.01$) decreases in frontal cortex blood flow, temporal cortex blood flow, and thalamus blood flow are considered to be related to the corpus callosum atrophy.

Cerebral blood flows have been reported to be decreased in chronic alcoholics [5,6,8,13,14,21] but the relationship between corpus callosum atrophy and decreased cerebral blood flows has not yet been reported. The significant positive correlations between the corpus callosum atrophy and the cerebral cortex blood flows in the present study suggest that corpus callosum atrophy may be related to cortical damage in chronic alcoholism [22].

The corpus callosum atrophy in chronic alcoholism has been reported to be prominent in the genu and body [20] and the decrease in regional cerebral blood flows in chronic alcoholism has been reported to be prominent in frontal lobes [14]. The strongest correlation found in the present study was between the thickness of genu and the frontal cortex blood flow. This may be related to the fact that the nerve fibers connecting bilateral frontal lobes pass mainly the genu of the corpus callosum.

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