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Depression and ventricular enlargement

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ABSTRACT - Compared with controls, 46 depressed patients showed increased ventricular-brain ratio (VBR). Patients with poor outcome, single episode, onset at 50 years or over, or without melancholia were found to have greater VBR than controls.

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Recent research in schizophrenia using CT scans has revealed morphological abnormalities, such as ventricular enlargement and cortical atrophy (1-3). Studies in depression also confirmed abnormalities similar to those in schizophrenia, though not as prominent (4-9).

In this study CT scans were applied to depressed patients in order to elucidate the relationship between ventricular size and clinical features.

Material and methods

We studied 37 women and 9 men, aged 25 to 77 years (mean 53 years), who met the criteria of DSM III for major affective disorder (2 bipolar disorder and 44 major depression).

The control group consisted of 11 volunteers and 35 patients with headache or dizziness as their chief complaints, and no neurological abnormalities (30 women and 16 men, aged 27 to 78 years (mean 52 years)). All patients and volunteers had given their informed consent to participate.

CT scans were obtained using Hitachi CTHF and EMI 1010. The width of slices was 10 mm. Ventricular-brain ratio (VBR) was calculated at the level where the ventricles were at their largest.

Results

In depressed patients VBR was larger than in controls (Table 1). A significant positive correlation was obtained between VBR and age in both groups of subjects (patients r = 0.48, P < 0.01; controls r = 0.54, P < 0.01).

We then divided depressed patients into subgroups by melancholia, age at onset, number of episodes, and outcome. Patients with melancholia had larger VBR than controls (n = 23), though this was

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Table 1
Comparison of VBR between depressed patients and normal controls

	Patients	Controls	t	P
All patients $(n = 46)$	11.2 ± 3.5	9.1 ± 2.4	3.27	0.001
With melancholia $(n = 23)$	10.6 ± 3.3	8.9 ± 2.8	1.86	N.S.
Without melancholia $(n = 23)$	11.7 ± 3.7	9.3 ± 2.1	2.76	0.008
Onset $\geq 50 \ (n = 24)$	12.8 ± 3.8	9.8 ± 2.7	2.98	0.004
Onset $\leq 50 \ (n=22)$	9.5 ± 2.3	8.3 ± 1.8	1.93	N.S.
Single episode $(n = 23)$	11.9 ± 3.3	8.9 ± 2.3	3.67	< 0.001
Recurrent episodes $(n = 23)$	10.4 ± 3.7	9.3 ± 2.5	1.12	N.S.
Good outcome group $(n = 13)$	9.9 ± 2.2	8.8 ± 2.6	0.21	N.S.
Poor outcome group $(n = 31)$	11.7 ± 3.8	9.2 ± 2.4	3.08	0.003

not statistically significant. Those without melancholia, however, showed much larger VBR than controls (n = 23). Patients whose age at onset was 50 years or over were found to have much larger ventricles than controls (n = 24), while the difference between VBR at earlier onset of depression and that of controls (n = 22) was not significant. Similarly, patients with single episode had larger VBR compared with controls (n = 23), whereas those with multiple episodes did not.

In addition, we defined "poor outcome group" as depressed patients who continued to manifest affective symptoms at least 9 months after their first visit to the hospital; those who remitted completely from the index episode within 9 months were defined as "good outcome group". Patients in the "good outcome group" (13 cases) did not have larger ventricles compared with controls, whereas those in the "poor outcome group" had.

Discussion

It has been demonstrated in the present study that depressed patients have larger ventricles than controls and that patients without melancholia, a late onset, single episode or poor outcome displayed the same tendency. These findings could suggest that non-melancholic, non-recurrent, late onset and refractory depression has "organicity"; while endogenous, recurrent, early onset and good response to treatment is "functional".

Jacoby et al. (4) first reported on computed tomography in the elderly with affective disorder. They stated that a subgroup of patients with enlarged ventricles emerged, whose first depression had begun later in life, and who at the time of the study were older and showed more "endogenous" features than the remaining subjects. The discrepancies between our findings of non-melancholic depressives tending to have enlarged ventricles and Jacoby's results might be due to the difference in criteria; we used DSM III, in contrast to the Newcastle Depression Scale. In a subsequent study Jacoby et al. (5) reported on the 2-year follow-up, in which mortality was significantly higher in patients who had previously shown ventricular enlargement.

Pearlson et al. (6) found that of 16 manic-depressive patients two had a significant ventricular enlargement. Tanaka et al. (7) found in depressive patients of 50 years and over a significant enlarge-

ment of the maximum width of the interhemispheric fissure and the maximum width between the sylvian fissure and inner skull, compared with controls. Standish-Barry et al. (8) studied 55 patients with severe affective illness who underwent psychosurgery after a pneumo-encephalographic and computerized axial tomographic study, and suggested that the ventricular enlargement in some patients could be due to brain structure abnormality. Targum et al. (9) have recently reported that five of 20 delusional depressives in contrast to none of 18 non-delusional depressives had ventricular brain ratios > 2 SD of the mean of 26 neurological controls. These earlier examinations made no mention of age of onset, melancholia of DSM III, number of episodes or outcome.

Our findings suggest that in depression there may be one subtype with cerebral morphological abnormalities, probably degenerative change.

Further follow-up studies using other approaches, such as neuroendocrinological, neurophysiological and neuropharmacological, are needed to clarify the clinical significance of CT abnormalities in depression.

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