Lateral ventricular enlargement in schizotypal personality disorder


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Abstract

Although an increase in the ratio of ventricular space to brain (ventricle-brain ratio, VBR) on computed tomography (CT) has been among the most robust findings in chronic schizophrenia, VBR has not been investigated in a large, well-characterized clinical population of patients with schizotypal personality disorder (SPD), a clinical entity with a phenomenologic, genetic, biological, and treatment response relationship to chronic schizophrenia. Accordingly, CT scans were obtained in 36 male SPD patients, 23 males with other personality disorders, 133 male schizophrenic patients, and 42 male normal volunteers. The mean body of the lateral VBR was significantly greater in the SPD patients than in the patients with other personality disorders. The VBR of the SPD patients did not differ significantly from either that of the normal volunteers or the schizophrenic patients but was intermediate between the two groups. There were no correlations with either psychotic-like or deficit-related symptoms of SPD in either the SPD or total personality disorder cohorts. SPD patients, like schizophrenic patients, may have increased VBRs compared with patients with other personality disorders; their VBRs fall between the means of schizophrenic patients and normal control subjects.

Keywords: Computed tomography; Ventricle-brain ratio; Schizophrenia; Personality disorder

1. Introduction

Enlargement in the ventricle/brain ratio (VBR) as determined by computed tomography (CT) has been one of the most robust and consistent findings in schizophrenia, although there is considerable heterogeneity in VBR values in schizophrenic patients (Shelton and Weinberger, 1986; Marks and Luchins, 1990; Raz and Raz,
Increased VBR values have been correlated, although not necessarily consistently, with phenomenological and biological characteristics of schizophrenia, including poor premorbid adjustment, cognitive and social impairment, fewer positive symptoms, decreased treatment response, low cerebrospinal fluid (CSF) levels of homovanillic acid, and impaired smooth pursuit eye movement (Shelton and Weinberger, 1986; Marks and Luchins, 1990; Raz and Raz, 1990; Bogerts et al., 1991; Van Horn and McManus, 1992). A greater VBR compared with control values has been reported in other psychotic schizophrenia-related disorders, including schizotypal disorder (Schulz et al., 1983; DeLisi et al., 1992; Kenny and Schulz, 1993), as well as in other psychotic disorders such as bipolar affective disorder (Rieder et al., 1983) and psychotic depression (Targum et al., 1983). However, VBR has not been extensively investigated in the schizophrenia-related personality disorders, which share common genetic and biological substrates with schizophrenia.

Schizotypal personality disorder (SPD), the prototype of the schizophrenia-related personality disorders, is phenomenologically, genetically, and biologically related to chronic schizophrenia (Siever et al., 1990, 1993a, 1993b), but it differs from chronic schizophrenia in the absence of overt, sustained psychosis and in the concomitant lesser likelihood of long-term institutionalization and neuroleptic treatment. Thus, the evaluation of VBR values in SPD affords an opportunity to help determine whether VBR abnormalities can be observed across the schizophrenia spectrum or are restricted to the psychoses.

In one pilot study, a small group of patients with schizophrenia-related personality disorders demonstrated increased lateral VBRs compared with normal control subjects (Cazzullo et al., 1991) and, in another, adolescents with psychotic schizophrenia spectrum disorders demonstrated increased VBR values, but this sample included only three patients with schizotypal disorder and none with SPD (Schulz et al., 1983). In contrast, in 31 patients with borderline personality disorder, no increase in VBR was found compared with normal control values (Lucas et al., 1989). Among offspring of schizophrenic mothers, increased cortical and cerebellar abnormalities were associated with increased genetic loading for schizophrenia and with diagnoses of both schizophrenia and schizotypal personality disorder, while increased periventricular area was associated with an interaction between genetic loading and obstetrical complications and with diagnoses of schizophrenia but not necessarily schizotypal personality disorder (Schulsinger et al., 1984; Cannon et al., 1989, 1993). To date, there have been no studies of VBR in adequate clinical samples of patients with schizotypal personality disorder compared with patients with other personality disorders (OPD). The OPD cohort provides a comparison group with comparable psychopathology but specific failure to meet criteria for a diagnosis of SPD.

To determine whether increased VBR is associated with the full spectrum of schizophrenia-related disorders rather than just with chronic severe psychosis, the VBRs of male SPD patients were compared with VBRs of male OPD patients and normal control subjects. As a secondary comparison group, the VBR means of chronic schizophrenic patients evaluated in an identical protocol in our center were compared with the VBRs of the patients with personality disorders. As the patients with personality disorders were under 60 years of age, the schizophrenic sample was also restricted to males younger than age 60.

Correlational analyses were carried out of the VBR in relation to the total number of schizotypal symptoms, as well as the psychotic-like and deficit-related criteria of SPD in the total and SPD samples (Siever, 1991; Siever et al., 1993b). In addition to the bodies of lateral ventricles, frontal horn, posterior horn, and third ventricle VBRs were also compared between groups in secondary analyses to explore the possibility of regional ventricular differences between groups. It was hypothesized that SPD patients would demonstrate a greater lateral VBR than the OPD and normal control comparison groups but not the schizophrenic comparison group. It was also hypothesized that the ventricular enlargement would be correlated with the presence of deficit-like symptoms of SPD.
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