BRAIN GLUCOSE METABOLISM IN BORDERLINE PERSONALITY DISORDER

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Summary — We searched for regional cerebral metabolic disturbances in patients with borderline personality disorder (BPD). Ten inpatients with BPD, no current DSM-III-R Axis I diagnosis and free of any psychotropic substances, were compared with 15 age-matched control subjects using positron emission tomography with 2-deoxy-2-[18F]fluoro-D-glucose and semiquantitative analysis of regional glucose metabolic activity. We found relative hypometabolism in patients with borderline personality disorder at the level of the premotor and prefrontal cortical areas, the anterior part of the cingulate cortex and the thalamic, caudate and lenticular nuclei. This study shows significant cerebral metabolic disturbances in patients with borderline personality disorder. These metabolic disturbances, which are similar to some of those described in other psychiatric entities, may help to understand the characteristic clinical aspects of this disorder.

Introduction

Borderline personality disorder (BPD) as defined in 1980 by the DSM-III criteria (APA, 1980) has compiled a series of patients formerly diagnosed as having emotionally unstable character, hysteroide dysphoria, pseudoneurotic schizophrenia, etc. (Gunderson et al., 1981). Patients with BPD present affective or psychotic symptoms, impulsiveness, episodic behavioral dyscontrol, pathological responses to psychological stress and chronic social malfunctioning. BPD can be identified with good reliability (Gunderson et al., 1981; Spitzer et al., 1979).

From a phenomenological point of view, this syndrome has been associated with several psychiatric and organic pathological conditions including affective disorders (Akiskal et al., 1985; Carroll et al., 1981; Pope et al., 1983; Schultz et al., 1989), schizophrenia (Gunderson et al., 1975, 1979; Schultz et al., 1989), schizoaffective psychoses (Andrulonis et al., 1982), atypical psychoses (Andrulonis et al., 1982; Mitsuda & Fukuda, 1974) and epilepsy (And-
The pathophysiological processes involved in the BPD syndrome are not well understood. Noradrenergic and endogenous opiate dysfunction (Van der Kolk, 1987), serotonin (Brown et al., 1982; Coccaro et al., 1989; Hollander et al., 1994) and dopamine (Bonnet & Redford, 1982; Lucas et al., 1987) system dysfunction have been proposed as possibly involved in BPD.

Although the biological bases of this clinical entity have largely been discussed in the literature (for a review see Korzekwa et al., 1993), only a few introductory results on the use of positron emission tomography (PET) with 2-deoxy-2-[18F]fluoro-d-glucose (FDG), have been reported in BPD (De La Fuente et al., 1992, 1993, 1994; Goyer et al., 1994). This is in contrast to the great bulk of PET data described for other psychiatric disorders. As some of these pathological conditions already studied with PET share common symptoms and are thought to be associated to BPD, we believe it interesting to study regional cerebral metabolism in BPD using PET with FDG because it may offer clues to a better understanding of this disorder.

Methods

Patient data

Ten patients (eight women and two men, mean age = 34.2; range 24 to 45) fulfilling the DSM-IIIIR criteria for BPD and with a score of at least 7 in the Gunderson and Kolb's diagnostic interview for borderlines (Gunderson et al., 1981) were recruited. Exclusion criteria were: current DSM-IIIIR axis I disturbances, abnormal physical or neurological examinations, abnormal standard biological blood tests, abnormal electrocardiogram, history of brain lesion, convulsion or generalized seizure or standard EEG traits of epilepsy. Depressed patients were excluded only if they met the DSM-IIIIR criteria for major
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