Prolactin response to TRH in patients with panic disorder

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Received 16 September 1998; received in revised form 1 March 2000; accepted 8 March 2000

Abstract

The effects of TRH administration (400 μg, i.v.) on the release of prolactin were examined in 15 patients who met DSM-III-R criteria for panic disorder and 15 normal control subjects. Four hundred micrograms TRH was given via IV route. Blood samples were taken before TRH administration (baseline values) and at 15, 30 and 60 min. The results demonstrate that prolactin responses to TRH did not differ between panic disorder patients and normal control subjects. When only women were evaluated, the findings indicate that women with PD tend to show excessive prolactin responses to TRH. The findings are discussed in view of findings from earlier reports.

Keywords: Panic disorder; TRH; Prolactin

1. Introduction

A relationship between panic disorder (PD) and depressive disorder is supported by the frequent comorbidity of these disorders and the similarity of their pharmacological therapies. In numerous studies, decreased thyrotropin (TSH) responses to protirelin (TRH) were obtained in 25–30% of patients with major depressive disorder (MDD) (Loosen and Prange, 1982). Another finding from those studies suggests that normal or abnormal prolactin responses to TRH can be observed in depressive patients and that
these abnormal responses can be in blunted or excessive forms (Baumgartner et al., 1988).

Roy-Byrne et al. (1986) found significantly lower prolactin responses to TRH in patients with PD compared to normal controls, which was a similar finding to those observed in patients with MDD. This finding could not be supported by two later studies in which there was no significant difference in prolactin responses to TRH between patients with PD and control groups (Castellani et al., 1988; Stein and Uhde, 1991).

The present study examined prolactin responses to TRH in 15 PD patients and compared their test results with those of 15 normal control subjects. To date, except in one study (Stein and Uhde, 1991), gender differences have not been evaluated in respect of prolactin responses to TRH in patients with PD. Therefore, a secondary issue was to evaluate whether gender differences might effect prolactin responses to TRH in PD patients and normal control subjects.

2. Methods

2.1. Subjects

Fifteen patients who had been referred to the Psychiatry Outpatient Clinic of the Department of Psychiatry, Istanbul Faculty of Medicine, were included in the study. All of the patients met DSM-III-R (APA, 1987) criteria for PD and none of them met DSM-III-R criteria for past or current major depressive episode. Of the 15 PD patients, three had comorbid generalized anxiety disorder, one had comorbid simple phobia and one had comorbid somatization disorder. The control group consisted of 15 healthy volunteers matched by age and sex who did not have any current or past psychiatric disorder, as established by unstructured clinical interview. The matching for age included ±1 year difference. In the panic group, the mean age was 33.67 (S.D. 7.95), and in the control group, it was 33.80 (S.D. 7.93). There were seven women and eight men in both groups. In the PD group, the mean age of onset was 28.3 (S.D. 6.10), the mean duration of the disorder was 66.21 months (S.D. 90.40), the longest period free of panic attacks since the onset was 16.81 months (S.D. 30.07), and the mean number of attacks in the previous month was 17.00 (S.D. 24.83). The mean Beck Depression Inventory (BDI) score in the PD group was 23.36 (S.D. 12.60) and the mean Hamilton Rating Scale for Anxiety (HRSA) score was 30.79 (S.D. 7.04).

Detailed medical histories were taken from all of the subjects in both groups and all of them underwent a thorough physical examination by an internist, to exclude subjects with any kind of organic disorder in the past or present. Nine patients in the PD group had recently received trials of antidepressants and/or benzodiazepines. Another six PD patients were medication free for at least 1 month prior to testing. Control subjects had never received psychotropic drugs. Both groups had at least 2 weeks in which all of their medications were discontinued prior to the administration of the neuroendocrine tests. Informed consent was given by all of the patients and the healthy controls before they were included in the study.

2.2. Procedures

A semi-structured interview form prepared by the authors was used to evaluate the clinical features of the patients including the age of onset, duration of the disorder and number of attacks in the last month.

The diagnoses of PD and other Axis I disorders were made by Structured Clinical Interview for DSM-III-R (SCID; Spitzer et al., 1987). The translation and adaptation of the SCID into Turkish was done by Soria et al. (1990). PD patients were given a semi-structured interview form prepared by the authors, the BDI (Beck et al., 1961) and the HRSA (Hamilton, 1959) during the day before TRH testing.

After an overnight fast, the subjects were placed in a reclined chair and instructed to relax for 20 min before the test. At 09.00 h, 400 μg TRH was given with slow i.v. injection over 1 min. Blood samples were taken before TRH administration (baseline values) and at 15, 30 and 60 min. After TRH administration, subjects in both groups reported urge to void, nausea, palpitations, acceler-
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