ELSEVIER

Anxiety Disorders
18 (2004) 423–431

JOURNAL OF Anxiety Disorders

Anxiety sensitivity and modulation of the serotonergic system in patients with PD

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Received 21 March 2002; received in revised form 16 April 2002; accepted 25 November 2002

Abstract

Anxiety sensitivity, i.e., the fear of anxiety-related bodily sensations, is one of the most studied cognitive variables in panic disorder (PD). However, the effects of selective serotonergic antipanic agents on this variable have not yet been investigated. The present study examines the effects of 6 weeks of treatment with citalopram on anxiety sensitivity in patients with PD. Twenty patients entered the study. On day 0, before starting drug treatment, after 1 week and after 6 weeks of treatment, each patient was evaluated with the Anxiety Sensitivity Index (ASI); the severity of clinical symptomatology was assessed with standardized psychometric scales. Results showed a significant reduction of anxiety sensitivity after 6 weeks of treatment. There was a significant correlation between decrease of anxiety sensitivity and anticipatory anxiety, while no correlations were found between panic attacks and agoraphobic avoidance. These results suggest that antipanic drug treatment decreases anxiety sensitivity.

Keywords: PD; Carbon dioxide; Anxiety sensitivity; Cognitive

1. Introduction

Anxiety sensitivity is a dispositional cognitive variable that reflects the fear of anxiety-related bodily sensations that arise from beliefs that these sensations have

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0887-6185/$ – see front matter
doi:10.1016/S0887-6185(02)00295-5

harmful consequences (Taylor, 1999). For example, subjects with high anxiety sensitivity may believe that a rapid heart rate signifies an impending heart attack, whereas subjects with low anxiety sensitivity will merely regard this as unpleasant (McNally, 1994). Anxiety sensitivity is a construct conceptually distinct from trait anxiety (McNally, 1996; Rapee & Medoro, 1994) and may be a cognitive risk factor for developing panic disorder (PD) (McNally, 1994; McNally & Lorenz, 1987; Schmidt, Lerew, & Jackson, 1999). Anxiety Sensitivity can be measured by the Anxiety Sensitivity Index (ASI) (Peterson & Reiss, 1992; Reiss, Peterson, Gursky, & McNally, 1986).

Anxiety sensitivity may be a predictor of the maintenance of PD in patients who remain untreated (Ehlers, 1995). Consequently, treating PD may have an impact on the level of anxiety sensitivity. Reductions in ASI scores of patients with PD following cognitive/behavioral treatment (CBT) have already been reported (McNally & Lorenz, 1987; Shear, Pilkonis, Cloitre, & Leon, 1994; Telch et al., 1993). These studies indicate that exposure and cognitive restructuring interventions reduces anxiety sensitivity. However, it is not clear if an explicit focus on fear of anxiety is necessary for the modification of anxiety sensitivity. Other treatments, such as pharmacotherapy, may also lead to significant changes in anxiety sensitivity. It is possible that fear of anxiety sensations is maintained by presence of panic attacks. If so, any treatment that controls these attacks might correspondingly diminish the fear of anxiety sensations. Alternatively, cortical serotonin changes might change cognitions directly, i.e., without psychological mediation, as some studies on depression have highlighted (Imber et al., 1990; Simons, Garfield, & Murphy, 1984).

Few studies have examined the effects of pharmacotherapy on ASI scores; all of these found a decrease of anxiety sensitivity (Mavissakalian, Perel, Talbott-Green, & Sloan, 1998; Otto, Pollack, Sachs, Rosenbaum, & Fava, 1991). Otto et al. (1991) reported a reduction of ASI scores from a mean of 31.3 at baseline to 22.2 after 6 months of drug treatment (67% of the sample with benzodiazepines alone, 8% with antidepressants alone and 25% with a combination of the two drugs). Mavissakalian et al. (1998) found that mean ASI scores decreased from 33.6 to 12.5 after 24 weeks of treatment with imipramine, with the mean score lower than 20 by 8 weeks. These studies suggest that fear of the consequences of anxiety-related bodily sensation can be normalized by biological treatments. Among the main brain systems, several experimental evidences suggest that the serotonergic one plays a key role in the pathogenetic mechanism of Anxiety Disorders and, in particular, of PD (Coplan, Gorman, & Klein, 1992; Gorman, Kent, Sullivan, & Coplan, 2000; Roy-Byrne & Cowley, 1998) and the efficacy of selective serotonergic agents in the treatment of PD supports the idea of an important role of the modulation system in the treatment of PD (Kent, Coplan, & Gorman, 1998; Perna, Bertani, Caldirola, Smeraldi, & Bellodi, 2001). We hypothesize that modulation of the serotonergic system is able to improve not only the core symptoms of PD, but also the catastrophic misinterpretation of anxiety symptoms. To test this idea, we have evaluated effects of 6 weeks