

Pharmacotherapy of somatoform disorders

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Abstract

Objective: This paper reviews the published literature on the pharmacologic management of somatoform disorders. **Methods:** Using Medline, the author identified all articles published between 1970 and 2003 on this topic, selecting the best-designed studies for inclusion. **Results:** The review reveals that patients with the obsessional cluster of somatoform disorders (hypochondriasis and body dysmorphic disorder [BDD]) respond well to serotonin reuptake inhibitors (SRIs). Less is known about the pharmacologic

responsiveness of patients with the primarily somatic cluster of somatoform disorders (somatization, pain), a patient group that is common in the health provider's office. **Conclusions:** Improvements in the design of future clinical trials are needed. A particular focus needs to be applied to study the neglected area of the pharmacologic treatment of syndromal and subsyndromal somatization and pain disorders.

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Introduction

Over the last decade, there has been a resurgence of hope that pharmacologic strategies might be helpful for patients with somatoform disorders. Recent reports support that hope and point to a need for additional research to investigate the efficacy of novel pharmacologic strategies for patients with illness fears and unexplained bodily sensations.

In DSM-IV [1], the disorders included under the somatoform heading are somatization disorder, undifferentiated somatoform disorder, pain disorder, hypochondriasis, body dysmorphic disorder (BDD), conversion disorder, and the residual category somatoform disorder not otherwise specified. This review paper will focus on the pharmacotherapy of hypochondriasis, BDD, somatization disorder, and pain disorder.

Two terms commonly used in any discussion of somatoform disorders include unexplained or "functional" somatic symptoms and hypochondriasis. These terms differ in crucial ways [2]. The former is a term used to describe somatic symptoms not caused by physical disease or tissue

damage. The latter is a term that indicates an unrealistic fear or belief that one has a disease, most often based on the perception of an unexplained somatic symptom. To the extent that these two terms indicate different phenomena and perhaps different pathophysiology, the treatment response to one type of somatoform disorder (e.g., hypochondriasis) may have only limited bearing on the treatment responsiveness of another type of somatoform disorder (e.g., somatization disorder).

The overarching category of somatoform disorders includes conditions that share the common feature of physical symptoms that induce undue discomfort, distress, or dysfunction. In the case of hypochondriasis and BDD, the disorders carry the additional component of intrusive unpleasant thoughts about disease or bodily appearance, compulsions to check for reassurance, and an accompanying negative appraisal of bodily symptoms that results in fear or avoidance. In these disorders, the meaning and implications of the symptoms are more distressing than the symptoms themselves. In the case of somatization disorder and pain disorder, the symptoms themselves are the primary focus of discomfort and distress. Because the terms hypochondriasis and somatization disorder are often used interchangeably by primary care clinicians, it is worth emphasizing that in hypochondriasis the fear of a serious illness preoccupies the patient and the compulsive checking

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serves to temporarily reduce the anxiety, creating a mental state and behavioral response that is quite similar to obsessive–compulsive disorder. In somatization disorder, on the other hand, the primary concern is not catastrophic, life-threatening illness but concern about multiple unexplained somatic symptoms.

Hypochondriasis and BDD then might be considered to fall primarily within an “obsessional/cognitive cluster,” whereas somatization and pain disorders would fall primarily within a “somatic/sensory cluster.” A somatoform disorder that may not fit well into either of these clusters is conversion disorder. Conversion disorder, unlike the other somatoform disorders, requires a stressor to precede the onset of the loss of function. Given the oft-cited symbolic significance to the part of the nervous system that is affected and given the lack of conscious awareness by the patient of the relationship between the stressor and the area of somatic dysfunction, it is clear that patients with conversion symptoms have more of a dissociative process at work rather than a primarily obsessional or somatizing one. Within any one individual, there may be a mix of these various processes but most often one predominates over the others. Further investigation of this cluster concept is required.

The majority of research on the pharmacotherapy of somatoform disorders over the last decade has been conducted on the obsessional cluster of somatoform disorders. To the extent that hypochondriasis or BDD falls within the domain of “obsessive–compulsive spectrum” disorders [3,4], it should not be surprising that patients with these disorders would have a preferential pharmacologic response to agents also found to be helpful for the obsessive–compulsive disorders. At present, it remains an open, relatively unexamined question whether the agents demonstrated to be helpful for patients with hypochondriasis and BDD would also be helpful for patients with the more somatic-focused somatoform disorders (e.g., somatization disorder).

Pharmacotherapy of hypochondriasis

It is well known that hypochondriasis may emerge as a secondary feature of other primary psychiatric disorders, such as panic disorder or “masked” major depression, and that treatment of the underlying disorder will lead to a resolution of the hypochondriacal preoccupation. Kellner et al. [5] demonstrated that about one-third of patients with melancholic depression had scores on a hypochondriasis scale that reached a threshold identified as being characteristic of patients with hypochondriasis. After these patients were treated with amitriptyline, the hypochondriacal features resolved along with the depression. Similarly, Noyes et al. [6] reported that hypochondriasis scores among patients with panic disorder declined in parallel with the resolution of the panic attacks as a result of pharmacotherapy.

Although clinicians have long been aware of the benefit of pharmacotherapy for secondary hypochondriasis accompanied by either an overt affective disorder or panic disorder, the clinical impression until the late 1980s was that pharmacotherapy would not benefit those with primary hypochondriasis [7]. The pessimism in regard to the treatment of primary hypochondriasis paralleled the pessimism that existed toward the treatment of all obsessional disorders. The pharmacologic revolution in the early 1990s shattered this pessimism as clinicians and researchers became aware that clomipramine and a new class of pharmacologic agents (serotonin reuptake inhibitors [SRIs]) were helpful for patients with obsessional disorders.

As SRIs became available and more widely used over the last 10 years, case reports and clinical case series suggested that these agents might be helpful for hypochondriasis: clomipramine [8,9], fluvoxamine [10], fluoxetine [11,12], and citalopram [13]. In one case report [10], a patient who showed no benefit to 80 mg/day of fluoxetine for 12 weeks subsequently responded very well to 300 mg/day of fluvoxamine. Therefore, patients with hypochondriasis who fail to respond to one SRI may experience benefit from an alternative SRI.

Uncontrolled open-label series have suggested efficacy associated with fluoxetine [14], fluvoxamine [15], paroxetine [16], nefazadone [17], and imipramine [18]. The fluoxetine trial lasted 12 weeks and used a flexible dosing regimen such that patients started on 20 mg/day and had dose increases as needed to 80 mg/day. In this trial, 10 of 14 (70%) study completers were responders with 4 of the 14 rated as being nearly symptom free. The mean end dose was 52 mg/day (S.D. 28 mg). Of interest, that trial demonstrated that patients without other Axis I comorbidity (6 of 7 patients) were as likely or more likely to benefit than patients with Axis I comorbidity (4 of 7 patients). Also, as measured by the Whiteley Index, although disease conviction and disease fear improved significantly, bodily preoccupation did not improve. The fluvoxamine trial consisted of 2 weeks of placebo followed by 10 weeks of fluvoxamine, starting at 50 mg/day and increasing weekly by 50 mg to the target dose of 300 mg/day. The responder rate to fluvoxamine of 72.7% among the 11 patients who completed at least 6 weeks was comparable to the rate reported in the fluoxetine study. Unlike the fluoxetine study, there was significant improvement in bodily preoccupation, as well as disease phobia and conviction. The 12-week paroxetine trial entered 11 patients and used a flexible dosing schedule to a target maximum of 60 mg/day. Of the 9 patients who completed the trial, 8 were rated as improved in hypochondriasis, 5 of whom were considered virtually symptom free. The mean completer dose was 31 mg/day (S.D. 17.9 mg). In the nefazadone open-label trial, 11 patients entered and 9 completed the 8 weeks of treatment (mean dose: 432 mg/day), 5 of whom were rated at least much improved with significant improvement noted in a variety of areas on the Kellner Illness Attitudes Scale,

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