

Resistant somatoform symptoms: Try CBT and antidepressants

Preferred strategy for 'mismatched' category of illnesses

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Treatment-resistant somatoform disorders are chronic (duration >1 year), can cause significant functional impairment, and respond poorly to routine care.

In the somatoform category, DSM-IV-TR includes diverse diagnoses such as conversion disorder, hypochondriasis, pain disorder, and body dysmorphic disorder. But like mismatched shoes, these disorders do not fit together well—one reason they are often misdiagnosed and ineffectively treated. This article describes:

- debate about how to categorize somatoform disorders—as psychological or physiologic
- evidence supporting psychotherapy and antidepressants to help patients with treatment-resistant somatoform disorders.



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continued



Box 1

Somatoform disorders: Interacting psychiatric and biologic processes

Psychobiologic causes of somatoform disorders are poorly understood. In a recent review, Rief and Barsky¹ emphasized that somatoform symptoms such as abdominal pain, headaches, or dizziness “are not strictly mental events, but are associated with a diversity of biological processes.” They propose that the following factors might contribute to somatoform disorders.

Autonomic physiologic arousal may lead patients to misperceive the meaning of normal bodily symptoms, but most studies have been equivocal or correlate closely with changes in the cardiovascular system. For example, patients with somatoform spectrum disorders who performed mentally distressing tasks did not have the same decrease in heart rate after completing the task as normal controls did, suggesting a deficit in autonomic reactivity.

Hypothalamic-pituitary-adrenal (HPA) axis studies also have been equivocal. Some have found low cortisol in patients with somatoform disorders—suggesting commonalities with

conditions such as posttraumatic stress disorder—but other studies have found normal or even elevated cortisol. Although a relationship between the HPA axis and somatoform disorders is likely, its nature remains unclear or may be indirect.

Serotonin is known to alter pain perception in major depressive disorder, so this neurotransmitter also probably plays a role in somatoform disorders. Low serotonin—mediated in part by alterations in branched-chain amino acid concentration—may be linked to increased pain perception.

Perception and filtering of body signals. A signal-filtering model of somatoform symptoms proposes that physical sensations enter consciousness influenced by numerous factors. These signals are then sent to a filter system, which itself is subject to factors that may decrease its activity. Cortical perception of distress may occur and symptoms begin to manifest if enough factors come into play.

WHICH CATEGORY?

Somatoform disorders are common in primary care. A medical utilization survey of 1,500 primary care patients found somatization symptoms in >20%.³ Controlling for comorbid psychiatric or medical illness did not change the study’s findings, which suggests that somatization is a distinct entity and not a symptom of another underlying disorder.

Little is known about somatoform disorders’ pathophysiology (*Box 1*),¹ but their unifying theme is that psychological factors contribute to, amplify, or alter the presentation of physical illness. Not only do these disorders not form a coherent DSM category, but—as described by Mayou et al²—the lack of clearly defined thresholds between normal and pathologic behaviors is one of

numerous problems that complicate diagnosis and treatment (*Box 2, page 107*).

Psychosomatic diad. Despite DSM-IV’s claims to etiologic neutrality, the origin of somatoform disorders’ physical symptoms clearly is meant to be psychological. As Lipowski⁴ said, somatization is “a tendency to experience and express somatic distress and symptoms unaccounted for by pathological findings, to attribute them to physical illness, and to seek medical help for them. It is often assumed that somatization becomes manifest in response to psychosocial stress brought about by life events that are personally stressful to the individual.”

Kroenke and others,^{5,6} however, have pointed out 2 shortcomings of this definition:

- the difficulty in knowing when a physical

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symptom truly is unexplained, especially in patients with comorbid medical illness³

- the instability of somatoform diagnoses (in a cohort examined with the same questionnaire 12 months apart, 43% of “lifetime somatic symptoms” patients reported at the first screening were not reported at the second).⁶

Kroenke³ suggests using “physical symptom disorder” as an etiologic-neutral descriptor of unexplained physical symptoms. He would place this category on Axis III and shift the causal emphasis from psychological to unexplained. This category would replace somatization disorder, undifferentiated somatoform disorder, and pain disorder in DSM.

Similarly, Mayou et al² contend that because most patients with somatoform disorders are treated by primary care physicians, having their disorders understood as psychiatric does not serve them well.

Psychiatric component. Conversely, patients with somatization disorder often have psychological symptoms, and many have personality disorders. The number of somatic symptoms with unexplained cause may be a normally distributed trait, with somatization disorders at the extreme end of the spectrum. Thus:

- Hypochondriasis could be reconsidered as health anxiety disorder because it features anxiety about potential illness.²
- Conversion disorders might be regrouped with other disorders focused on dissociation.²
- Body dysmorphic disorder might be regrouped with obsessive-compulsive disorder.⁷

These changes would shift focus away from the disorders’ physiologic presentations, emphasize the psychiatric disorders to which they likely are related, and provide insight into treatments and clinical investigations.

Pain disorder could be removed from DSM because of persistent concerns about the validity of

Box 2

Problems with DSM categorization of somatoform disorders

- **Somatoform disorders** lack clearly defined thresholds that establish a difference between normal and pathologic behaviors
- **Somatoform disorders** do not form a coherent category, and exclusion criteria are ambiguous
- **By existing**, the category suggests that some disorders are physical and others are mental, leaving little room for intermediate or mixed conditions
- **Patients reject** the term “somatoform” because it conveys doubt about the reality of their conditions
- **Somatoform disorders** are incompatible with some cultures’ views of mental illness (for example, the DSM translation used in China does not include the somatoform category)
- **Nonspecific** somatoform illness subcategories cannot achieve established reliability standards in studies examining diagnoses
- **Medical-legal cases** and insurance entitlements are complicated by unclear descriptions of somatoform disorders

Source: Reference 2

this diagnostic category. Tyrer⁸ reviewed his clinical experience and reported shifting from a view that people with excessive pain had a psychiatric disorder to the view that living with chronic pain produces a profile similar to that of a person with a psychiatric disorder.

Physiologic component. Others recommend caution before radically altering DSM’s categorizations. Rather than shift symptoms to Axis III—as Kroenke suggests—Starcevic⁹ would use unexplained physical symptoms as an organizing principle and group disorders with common features, such as somatization disorder, conversion disorder, pain disorder, and undifferentiated somatoform



Box 3

Treatment approach to patients with somatoform disorders

- **Carefully evaluate** for mood, anxiety, and substance use disorders
- **Assess over-the-counter**, prescription, and illicit drugs the patient may be using to control pain and other physical symptoms
- **Review** medical and psychiatric records, laboratory and radiographic findings
- **Discuss** the patient's case with his or her primary care physician, and provide formal feedback or a consultation letter
- **Treat** comorbid psychiatric disorders with medications such as selective serotonin reuptake inhibitors or venlafaxine, which have improved somatoform symptoms in randomized, controlled trials
- **Offer** the patient cognitive-behavioral therapy (10 to 20 sessions) that focuses on reducing somatoform symptoms, and advise the primary care provider to schedule monthly follow-up intervals

disorder. Body dysmorphic disorder and hypochondriasis—focusing on dysfunctional appraisal of physical symptoms—would likely move elsewhere.

Hiller and Rief¹⁰—who advocate strongly for keeping somatoform disorders in DSM—suggest 4 categories: monosymptomatic, polysymptomatic, hypochondriasis, and body dysmorphic disorder. They believe grouping diagnoses in this way would improve and refine existing nosology.

NEW TREATMENT APPROACHES

As the categorization debate continues, a treatment approach is developing that includes cognitive-behavioral therapy (CBT) and antidepressants to address the psychological and physiologic effects of resistant somatoform disorders (*Box 3*).

Consultation letters. Sending a consultation letter to the patient's primary care physician is considered the standard of care (*Box 4, page 114*).¹¹ In the study that introduced the consultation letter,¹² patients with somatization disorder were randomly assigned to treatment (a consultation letter) or control (treatment as usual). Health care utilization costs declined approximately 50%—largely because of decreased hospitalization—when patients' physicians received consultation letters, compared with no change for usual treatment.

Consultation letters may reduce health care spending but are less effective in improving symptoms. Evidence is changing treatment as psychotherapies have been found to help patients with somatoform disorders.

Group psychotherapy. In a controlled trial, primary care patients with somatization disorder received short-term group CBT or treatment as usual, with follow-up 6 months later. Those in the CBT group—who had received patient education and relaxation training—showed moderate but significant improvement in physical illness and somatic preoccupation, hypochondriasis, and medication use. Usual-care patients did not improve.¹³

CBT vs relaxation. A group of 191 inpatients described as “highly impaired” by somatization syndrome— ≥ 8 DSM-IV somatoform symptoms—was evaluated for psychopathology, subjective health status, and life satisfaction. They then were randomly assigned to somatization-focused CBT (“soma”) or relaxation training and compared with 34 control patients. At 1-year follow-up, doctor visits had declined significantly in patients who received CBT (“soma”), and their somatoform symptoms were reduced compared with controls.¹¹

Psychotherapy vs listening. In a randomized, controlled trial, 102 patients with chronic refractory irritable bowel syndrome were assigned to receive exploratory psychotherapy or supportive listening. After 12 weeks, psychotherapy was more effective in improving physical and psychological symptoms,

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Dose Dependency of Adverse Events in Short-Term, Placebo-Controlled Trials—Extrapyramidal Symptoms—In an acute-phase controlled clinical trial in schizophrenia, there was no significant difference in ratings scales incidence between any dose of oral olanzapine (5+2.5, 10+2.5, or 15+2.5 mg/d) and placebo for parkinsonism (Simpson-Angus Scale total score >3) or akathisia (Barnes Akathisia global score ≥2). In the same trial, only akathisia events (spontaneously reported COSTART terms akathisia and hyperkinesia) showed a statistically significantly greater adverse events incidence with the 2 higher doses of olanzapine than with placebo. The incidence of patients reporting any extrapyramidal event was significantly greater than placebo only with the highest dose of oral olanzapine (15+2.5 mg/d). In controlled clinical trials of intramuscular olanzapine for injection, there were no statistically significant differences from placebo in occurrence of any treatment-emergent extrapyramidal symptoms, assessed by either rating scales incidence or spontaneously reported adverse events.

Other Adverse Events—Dose-relatedness of adverse events was assessed using data from a clinical trial involving 3 fixed oral dosage ranges compared with placebo. The following treatment-emergent events showed a statistically significant trend: asthenia, dry mouth, nausea, somnolence, tremor.

Vital Sign Changes—Oral olanzapine was associated with orthostatic hypotension and tachycardia in clinical trials. Intramuscular olanzapine for injection was associated with bradycardia, hypotension, and tachycardia in clinical trials (see PRECAUTIONS).

Weight Gain—In placebo-controlled 6-week schizophrenia studies, weight gain was reported in 5.6% of oral olanzapine patients (average 2.8-kg gain) compared to 0.8% of placebo patients (average 0.4-kg loss); 29% of olanzapine patients gained >7% of their baseline weight, compared to 3% of placebo patients. During continuation therapy (238 median days of exposure), 56% of patients met the criterion for having gained >7% of their baseline weight. Average gain during long-term therapy was 5.4 kg.

Laboratory Changes—Olanzapine is associated with asymptomatic increases in SGPT, SGOT, and GGT and with increases in serum prolactin and CPK (see PRECAUTIONS). Asymptomatic elevation of eosinophils was reported in 0.3% of olanzapine patients in premarketing trials. There was no indication of a risk of clinically significant neutropenia associated with olanzapine in the premarketing database.

In clinical trials among olanzapine-treated patients with baseline random triglyceride levels of <150 mg/dL (N=659), 0.5% experienced triglyceride levels of ≥500 mg/dL anytime during the trials. In these same trials, olanzapine-treated patients (N=1185) had a mean triglyceride increase of 20 mg/dL from a mean baseline of 175 mg/dL. In placebo-controlled trials, olanzapine-treated patients with baseline random cholesterol levels of <200 mg/dL (N=1034) experienced cholesterol levels of ≥240 mg/dL anytime during the trials more often than placebo-treated patients (N=602; 3.6% vs 2.2% respectively). In these same trials, olanzapine-treated patients (N=2528) had a mean increase of 0.4 mg/dL in cholesterol from a mean baseline of 203 mg/dL, which was significantly different compared to placebo-treated patients (N=1415) with a mean decrease of 4.6 mg/dL from a mean baseline of 203 mg/dL.

ECG Changes—Analyses of pooled placebo-controlled trials revealed no statistically significant olanzapine/placebo differences in incidence of potentially important changes in ECG parameters, including QT, QTc, and PR intervals. Olanzapine was associated with a mean increase in heart rate of 2.4 BPM compared to no change among placebo patients.

Other Adverse Events Observed During Clinical Trials—The following treatment-emergent events were reported with oral olanzapine at multiple doses ≥1 mg/d in clinical trials (8661 patients, 4165 patient-years of exposure). This list may not include events previously listed elsewhere in labeling, those events for which a drug cause was remote, those terms which were so general as to be uninformative, and those events reported only once or twice which did not have a substantial probability of being acutely life-threatening. **Frequent events** occurred in ≥1/100 patients; **infrequent events** occurred in 1/100 to 1/1000 patients; **rare events** occurred in <1/1000 patients. **Body as a Whole**—**Frequent:** dental pain, flu syndrome; **Infrequent:** abdomen enlarged, chills, face edema, intentional injury, malaise, moniliasis, neck pain, neck rigidity, pelvic pain, photosensitivity reaction, suicide attempt; **Rare:** chills and fever, hangover effect, sudden death. **Cardiovascular**—**Frequent:** hypotension; **Infrequent:** atrial fibrillation, bradycardia, cerebrovascular accident, congestive heart failure, heart arrest, hemorrhage, migraine, pallor, palpitation, vasodilatation, ventricular extrasystoles; **Rare:** arteritis, heart failure, pulmonary embolus. **Digestive**—**Frequent:** flatulence, increased salivation, thirst; **Infrequent:** dysphagia, esophagitis, fecal impaction, fecal incontinence, gastritis, gastroenteritis, gingivitis, hepatitis, melena, mouth ulceration, nausea and vomiting, oral moniliasis, periodontal abscess, rectal hemorrhage, stomatitis, tongue edema, tooth caries; **Rare:** aphthous stomatitis, enteritis, eruption, esophageal ulcer, glossitis, ileus, intestinal obstruction, liver fatty deposit, tongue discoloration. **Endocrine**—**Infrequent:** diabetes mellitus; **Rare:** diabetic acidosis, goiter. **Hemic and Lymphatic**—**Infrequent:** anemia, cyanosis, leukocytosis, leukopenia, lymphadenopathy, thrombocytopenia; **Rare:** normocytic anemia, thrombocytopenia. **Metabolic and Nutritional**—**Infrequent:** acidosis, alkaline phosphatase increased, bilirubinemia, dehydration, hypercholesterolemia, hyperglycemia, hyperlipemia, hyperuricemia, hypoglycemia, hypokalemia, hyponatremia, lower extremity edema, upper extremity edema; **Rare:** gout, hyperkalemia, hypernatremia, hypoproteinemia, ketosis, water intoxication. **Musculoskeletal**—**Frequent:** joint stiffness, twitching; **Infrequent:** arthritis, arthrosis, leg cramps, myasthenia; **Rare:** bone pain, bursitis, myopathy, osteoporosis, rheumatoid arthritis. **Nervous System**—**Frequent:** abnormal dreams, amnesia, delusions, emotional lability, euphoria, manic reaction, paresthesia, schizophrenic reaction; **Infrequent:** akinesia, alcohol misuse, antisocial reaction, ataxia, CNS stimulation, cogwheel rigidity, delirium, dementia, depersonalization, dysarthria, facial paralysis, hypesthesia, hypokinesia, hypotonia, incoordination, libido decreased, libido increased, obsessive compulsive symptoms, phobias, somatization, stimulant misuse, stupor, stuttering, tardive dyskinesia, vertigo, withdrawal syndrome; **Rare:** circumoral paresthesia, coma, encephalopathy, neuralgia, neuropathy, nystagmus, paralysis, subarachnoid hemorrhage, tobacco misuse. **Respiratory**—**Frequent:** dyspnea; **Infrequent:** apnea, asthma, epistaxis, hemoptysis, hyperventilation, hypoxia, laryngitis, voice alteration; **Rare:** atelectasis, hiccup, hypoventilation, lung edema, stridor. **Skin and Appendages**—**Frequent:** sweating; **Infrequent:** alopecia, contact dermatitis, dry skin, eczema, maculopapular rash, pruritus, seborrhea, skin discoloration, skin ulcer, urticaria, vesiculobullous rash; **Rare:** hirsutism, pustular rash. **Special Senses**—**Frequent:** conjunctivitis; **Infrequent:** abnormality of accommodation, blepharitis, cataract, deafness, diplopia, dry eyes, ear pain, eye hemorrhage, eye inflammation, eye pain, ocular muscle abnormality, taste perversion, tinnitus; **Rare:** corneal lesion, glaucoma, keratoconjunctivitis, macular hypopigmentation, miosis, mydriasis, pigment deposits lens. **Urogenital**—**Frequent:** vaginitis; **Infrequent:** abnormal ejaculation, amenorrhea, breast pain, cystitis, decreased menstruation, dysuria, female lactation, glycosuria, gynecomastia, hematuria, impotence, increased menstruation, menorrhagia, metrorrhagia, polyuria, premenstrual syndrome, pyuria, urinary frequency, urinary retention, urinary urgency, urination impaired, uterine fibroids enlarged, vaginal hemorrhage; **Rare:** albuminuria, breast enlargement, mastitis, oliguria. (*Adjusted for gender).

The following treatment-emergent events were reported with intramuscular olanzapine for injection at one or more doses ≥2.5 mg/injection in clinical trials (722 patients). This list may not include events previously listed elsewhere in labeling, those events for which a drug cause was remote, those terms which were so general as to be uninformative, and those events reported only once or twice which did not have a substantial probability of being acutely life-threatening. **Body as a Whole**—**Frequent:** injection site pain; **Infrequent:** abdominal pain, fever. **Cardiovascular**—**Infrequent:** AV block, heart block, syncope. **Digestive**—**Infrequent:** diarrhea, nausea. **Hemic and Lymphatic**—**Infrequent:** anemia. **Metabolic and Nutritional**—**Infrequent:** creatine phosphokinase increased, dehydration, hyperkalemia. **Musculoskeletal**—**Infrequent:** twitching. **Nervous System**—**Infrequent:** abnormal gait, akathisia, articulation impairment, confusion, emotional lability. **Skin and Appendages**—**Infrequent:** sweating. **Postintroduction Reports**—Reported since market introduction and temporally (not necessarily causally) related to olanzapine therapy: allergic reaction (eg, anaphylactoid reaction, angioedema, pruritus or urticaria), diabetic coma, jaundice, pancreatitis, priapism, rhabdomyolysis, and venous thromboembolic events (including pulmonary embolism and deep venous thrombosis). Random cholesterol levels of ≥240 mg/dL and random triglyceride levels of ≥1000 mg/dL have been rarely reported.

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Literature revised March 20, 2006

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although the difference was statistically significant only in women. After 1 year, patients who received psychotherapy remained well and control patients who declined psychotherapy had relapsed.¹⁴

CBT vs usual treatment. In a randomized controlled trial, 84 patients with somatization disorder received 10 CBT sessions or treatment as usual. CBT's goals were to:

- reduce physiologic arousal though relaxation techniques
- enhance activity regulation through increasing exercise and meaningful pleasurable activities and pacing activities
- increase awareness of emotions
- modify dysfunctional beliefs
- enhance communication of thoughts and emotions
- reduce spousal reinforcement of illness behavior.

The Clinical Global Impression Scale for Somatization Disorder showed significantly greater improvement in the group receiving CBT. Somatic symptoms as measured by patients' self-ratings also improved more in the CBT group.¹⁵

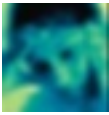
Psychotherapy's success in these and other studies supports the idea that somatoform spectrum disorders resemble other conditions—such as mood and anxiety disorders—that respond to psychological treatment.

ANTIDEPRESSANT THERAPY

Controlled trials also have shown that some antidepressants are more effective than placebo in improving somatoform symptoms.

St. John's wort. In a randomized, placebo-controlled, double-blind trial, 184 patients with somatoform disorders but not major depression received St. John's wort extract, 300 mg bid, or placebo. After 6 weeks, 45% of patients responded to St. John's wort, compared with 21% for placebo ($P=0.0006$). Six measures determined response; St. John's wort and placebo were equally well tolerated.¹⁶

continued



Box 4

Consultation letter for somatization: Discourage saying 'it's in your head'

Describe somatoform disorder, its relapsing course, and low morbidity and mortality rates

Encourage the primary care physician to:

- serve as the patient's primary doctor and avoid fragmented care from numerous sources
• schedule regular appointments with the patient
• perform physical exams at each visit
• eliminate unnecessary tests or hospitalizations
• avoid statements such as "it's all in your head" when medical tests are negative

Source: Reference 13

Extended-release venlafaxine. A pilot study enrolled 112 adult primary care patients with multi-somatoform disorder (≥3 medically unexplained, bothersome physical symptoms plus ≥2-year history of somatization) and comorbid major depressive disorder, generalized anxiety disorder, or social anxiety disorder.

Primary outcome was change in the 15-item Patient Health Questionnaire (PHQ-15) somatic symptom severity score. After 12 weeks, PHQ-15 scores declined significantly (P <0.0001) in both groups but did not improve significantly more with venlafaxine ER than with placebo (-8.3 vs -6.6, respectively, P=0.097).

Citalopram. In an 8-week trial, investigators compared the efficacy of a selective serotonin reuptake inhibitor (SSRI) and a selective noradrenaline reuptake inhibitor (SNRI) on pain symptoms in 35 patients with somatoform pain disorder. Patients were randomly assigned to double-blind treatment with the SSRI citalopram, 40 mg/d (n=17), or the SNRI reboxetine, 8 mg/d (n=18).

In patients receiving citalopram, scores decreased significantly from baseline on the Present Pain Intensity scale (3.5 vs 2.8, P=0.045) and Total Pain Rating Index of the McGill Pain Questionnaire (41.9 vs 30, P=0.004), but these scores did not change significantly in patients receiving reboxetine.

The authors concluded that citalopram was moderately effective for somatoform pain disorder in this small trial. Although antidepressants' efficacy for somatoform symptoms may be mediated through changes in comorbid mood and anxiety disorders, these authors observed that citalopram's analgesic effect appeared to be independent of how patients rated their depressive symptoms.

TREATMENT RECOMMENDATIONS

Based on the evidence and our experience, we recommend offering CBT to patients with recent symptom onset and insight into their comorbid

Somatoform disorders are often undiagnosed and difficult to treat. Improved diagnostic criteria and better collaboration with primary care may lead to better outcomes and more evidence-based treatment. Cognitive-behavioral therapy and pharmacotherapy are emerging treatment options for patients with refractory illness.

BottomLine

mood and anxiety disorders. If the patient does not improve after 8 to 12 sessions, consider adding an antidepressant such as:

- citalopram, 20 to 60 mg/d
- venlafaxine XR, 150 to 375 mg/d.

For patients with chronic somatization, start with combined pharmacotherapy and CBT.

Side effects are a frequent concern in this patient population, so titrate dosages slowly. Aim for the target antidepressant dosages used to treat major depression, and avoid declaring a treatment failure without first completing adequate trials. Once the patient is stable on medication, continue for at least 1 somatization-free year.

Allow patients to discuss their physical concerns, and attempt to support them in their suffering. At the same time, help them focus on attaining realistic goals for occupational and social functioning.

Work closely with the primary care provider in treatment planning to avoid sending the patient mixed messages. Communicating in the spirit of respect and collaboration with primary care colleagues can help prevent “splitting,” in which the patient may come to idealize one practitioner and devalue the other.

Remember that patients with somatization can become medically ill. Remind their primary care providers to perform expected evaluations as dictated by objective findings.

References

1. Rief W, Barsky AJ. Psychobiological perspectives on somatoform disorders. *Psychoneuroendocrinology* 2005;30(10):996-1002.
2. Mayou R, Kirmayer LJ, Simon G, et al. Somatoform disorders: time for a new approach in DSM-V. *Am J Psychiatry* 2005;162(5):847-55.
3. Barsky AJ, Orav EJ, Bates DW. Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. *Arch Gen Psychiatry* 2005;62(8):903-10.
4. Lipowski ZJ. Somatization: the experience and communication of psychological distress as somatic symptoms. *Psychother Psychosom* 1987;47(3-4):160-7.
5. Kroenke K. Physical symptom disorder: a simpler diagnostic category for somatization-spectrum conditions. *J Psychosom Res* 2006;60(4):335-9.
6. Simon GE, Gureje O. Stability of somatization disorder and somatization symptoms among primary care patients. *Arch Gen Psychiatry* 1999;56(1):90-5.

Related resources

- ▶ VHA/DoD clinical practice guideline for the management of medically unexplained symptoms: chronic pain and fatigue (brief summary). www.guideline.gov/summary/summary.aspx?doc_id=3415.
- ▶ Abbey SE. Somatization and somatoform disorders. In: Levenson JL, ed. *The American Psychiatric Publishing textbook of psychosomatic medicine*. Washington, DC: American Psychiatric Publishing; 2005:271-96.

DRUG BRAND NAMES

Citalopram • Celexa
Venlafaxine extended-release • Effexor XR

DISCLOSURE

Dr. Marcangelo reports no financial relationship with any company whose products are mentioned in this article or with manufacturers of competing products.

Dr. Wise is a consultant to or speaker for Eli Lilly and Co., GlaxoSmithKline, and Pfizer.

7. Phillips KA, McElroy SL, Hudson JI, Pope HG Jr. Body dysmorphic disorder: an obsessive-compulsive spectrum disorder, a form of affective spectrum disorder, or both? *J Clin Psychiatry* 1995; 56(suppl 4):41-51.
8. Tyrer S. Psychosomatic pain. *Br J Psychiatry* 2006;188:91-3.
9. Starcevic V. Somatoform disorders and DSM-V: conceptual and political issues in the debate. *Psychosomatics* 2006;47(4):277-81.
10. Hiller W, Rief W. Why DSM-III was right to introduce the concept of somatoform disorders. *Psychosomatics* 2005;46(2):105-8.
11. Bleichhardt G, Timmer B, Rief W. Cognitive-behavioural therapy for patients with multiple somatoform symptoms—a randomised controlled trial in tertiary care. *J Psychosom Res* 2004;56(4):449-54.
12. Smith GR Jr, Monson RA, Ray DC. Psychiatric consultation in somatization disorder. A randomized controlled study. *N Engl J Med* 1986;314(22):1407-13.
13. Lidbeck J. Group therapy for somatization disorders in general practice: effectiveness of a short cognitive-behavioural treatment model. *Acta Psychiatr Scand* 1997;96(1):14-24.
14. Guthrie E, Creed F, Dawson D, Tomenson B. A randomised controlled trial of psychotherapy in patients with refractory irritable bowel syndrome. *Br J Psychiatry* 1993;163:315-21.
15. Allen LA, Woolfolk RL, Escobar JI, et al. Cognitive-behavioral therapy for somatization disorder: a randomized controlled trial. *Arch Intern Med* 2006;166(14):1512-8.
16. Muller T, Mannel M, Murck H, Rahlfs VW. Treatment of somatoform disorders with St. John's wort: a randomized, double-blind and placebo-controlled trial. *Psychosom Med* 2004;66(4):538-47.
17. Kroenke K, Messina N 3rd, Benattia I, et al. Venlafaxine extended release in the short-term treatment of depressed and anxious primary care patients with multisomatoform disorder. *J Clin Psychiatry* 2006;67(1):72-80.
18. Aragona M, Bancheri L, Perinelli D, et al. Randomized double-blind comparison of serotonergic (citalopram) versus noradrenergic (reboxetine) reuptake inhibitors in outpatients with somatoform, DSM-IV-TR pain disorder. *Eur J Pain* 2005;9(1):33-8.