Current PSYCHIATRY

CASES THAT TEST YOUR SKILLS

Eight weeks after remission, Ms. F's depressive symptoms resurface. She's tired, sleep-deprived, and has trouble functioning at work. Did a recent automobile accident contribute to her mood reversal?

The painful truth about depression

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HISTORY Initial symptoms

Ms. F, age 39, presented with depression, severe anxiety, and disturbed sleep. She denied psychiatric or medical history, but reported that her depressive symptoms hampered her performance at work and led to work-related stress.

Ms. F's Beck Depression Inventory (BDI) score at baseline was 21, indicating moderate depression. The psychiatrist diagnosed her as having generalized anxiety disorder and adjustment disorder with depressed mood.

The patient was started on paroxetine, 10 mg/d. Five weeks later, her anxiety had decreased significantly and her BDI score had dropped to 10, indicating normal mood. Both the patient and clinician decided at this point that Ms. F had reached remission. The patient demanded that paroxetine be stopped, saying that she "does not like medication." The psychiatrist reluctantly agreed.

Eight weeks later, during a follow-up examination, Ms. F complained of severely depressed mood with frequent crying spells. She complained of fatigue, nausea, headaches, decreased appetite, and dizziness. Her work performance, which had improved during the paroxetine trial, was again compromised. Her BDI score was 32, indicating severe depression.

Was the psychiatrist justified in stopping paroxetine therapy after 5 weeks?

Dr. Fishbain's observations

Premature paroxetine discontinuation cannot be ruled out as a cause for Ms. F's relapse. Sood et al[1] found that duration of antidepressant therapy beyond treatment guidelines correlated with longer times to relapse.

By contrast, Ms. F's initial therapy duration

fell short of the 6 to 8 weeks recommended by the American Psychiatric Association.[2]

Patients commonly cite adverse events as a reason for wanting to stop antidepressant therapy.[3] Ms. F reported no adverse effects, however; she said only that she did "not like medication." Despite her insistence to the contrary, antidepressant therapy probably should not have been stopped.

FURTHER HISTORY A painful discovery

After questioning, Ms. F told the psychiatrist that she had been involved in a motor vehicle accident 2 weeks before the follow-up visit and had since been suffering lower back and neck pain.

After more questioning, Mr. F revealed that the pain was disrupting her sleep. She was getting about 4 hours of fragmented sleep per night, resulting in lack of energy during the day. The pain made it hard for her to sit, further impairing her work performance.

Ms. F was restarted on paroxetine, 10 mg/d titrated across 6 weeks to 60 mg/d for her depression, and zolpidem, 10 mg at bedtime, to help her sleep. After 6 weeks, her BDI score improved to 20, and she was less labile. Her depressive symptoms persisted, however, as did her pain, fatigue, headaches, nausea, dizziness, and sleep disturbances.

What role did Ms. F's pain play in her relapse? How can clinicians detect somatic symptoms and gauge their effect on mood?

Dr. Fishbain's observations

Pain most likely caused Ms. F's depression relapse. McBeth et al[4] have demonstrated that pain can contribute to depression's development. In another study,[5] 43.4% of subjects who met criteria for major depression reported painful symptoms. The presence of a chronic painful condition was also found to contribute to major depression.[5]

Nakao et al[6] screened 2,215 outpatients who were referred with mind/body complaints. Patients who were diagnosed with major depression had significantly higher rates of fatigue (86% vs. 65%), insomnia (79% vs. 58%), nausea/vomiting (51% vs. 40%), and low-back pain (36% vs. 24%) than those who were not. Within the major depression group, somatic symptoms were more abundant in patients with severe depressive episodes than in those with mild depressive episodes (5.8 vs. 3.7, $P < 0.05$).

Depression prevalence appears to increase when somatic symptoms are considered in the diagnosis. Posse and Hallstrom[7] used a two-stage design to screen for depression. In the first stage, depression prevalence was 1.8%. In the second stage, 62 patients with high somatic complaint scores were re-evaluated. Of this group, 41 were diagnosed with a major depressive disorder or dysthymia.

Patients with continued pain after depression treatment are at high risk for depression recurrence.[8] Diffuseness of pain and extent to which it interferes with daily activities strongly predict depression.[9]

Diagnostic challenges. Patients with depression often present to their primary care physicians with somatic rather than behavioral symptoms, making it hard for the family doctor to diagnose depression.[10] By contrast, when presenting to a psychiatrist, depressed patients tend to discuss their emotional symptoms but not their physical complaints.[11] This is because patients often:

- attribute physical symptoms to an unrelated medical illness
- consider aches and pains a normal part of aging
- or are not aware that psychiatrists can treat physical symptoms.[11]

Psychiatrists in the past have emphasized emotional symptoms while barely addressing physical symptoms. This trend is changing, however, as the link between physical pain and depression has become clearer.

Be sure to include chronic pain or other somatic symptoms in the systems review. Screening tools such as the Visual Analogue Scale can measure pain intensity, while the Cornell Medical Index can uncover somatic symptoms. No all-inclusive tool exists to help detect depression-related somatic symptoms. however.

Should the psychiatrist continue to address Ms. F's depressive symptoms, or should the focus shift to her somatic symptoms?

Dr. Fishbain's observations

Patients who do not respond to depression treatment (ie, achieve >50% symptom reduction), or who respond without achieving remission, usually have residual physical symptoms—often fatigue, sleep disturbance, decreased appetite, anxiety, sexual dysfunction, and/or pain.[12-14] Severe pain and other somatic symptoms are likely prolonging Ms. F's depression, despite increased paroxetine dosages.

Paykel et al[8] have associated residual depression symptoms with early relapse of depression. In their study, 94% of depressed patients with lingering depressive symptoms had mild to moderate physical symptoms. By contrast, degree of physical symptom improvement has been shown to correlate with likelihood of depression remission.[15]

Although emotional symptoms improve with antidepressants,[16] some evidence[17] indicates that physical symptoms associated with depression may be less responsive.

Also, because many psychiatrists have been taught to track emotional symptoms and only some physical symptoms, somatic symptoms of depression often are not targeted for treatment.[17] Lack of rating scales to track somatic symptoms compounds this problem.[17]

Psychiatrists need to target both the physical and emotional symptoms of depression. When pain prolongs depression, it should be the primary target of antidepressant drug therapy (*Algorithm*).

To date, several meta-analyses[18-20] have demonstrated that antidepressants have a separate analgesic effect on all forms of chronic pain. Evidence[21,22] also indicates that the dual-action antidepressants—such as amitriptyline, bupropion, venlafaxine, and (awaiting FDA approval) duloxetine—have a more-consistent analgesic effect than do the serotonin reuptake inhibitors.

The analgesic effects of bupropion, duloxetine, and venlafaxine have not been compared with those of tricyclics or other older antidepressants. If one of the newer dual-action antidepressants does not reduce somatic conditions or produce an adequate response, consider switching to a tricyclic.

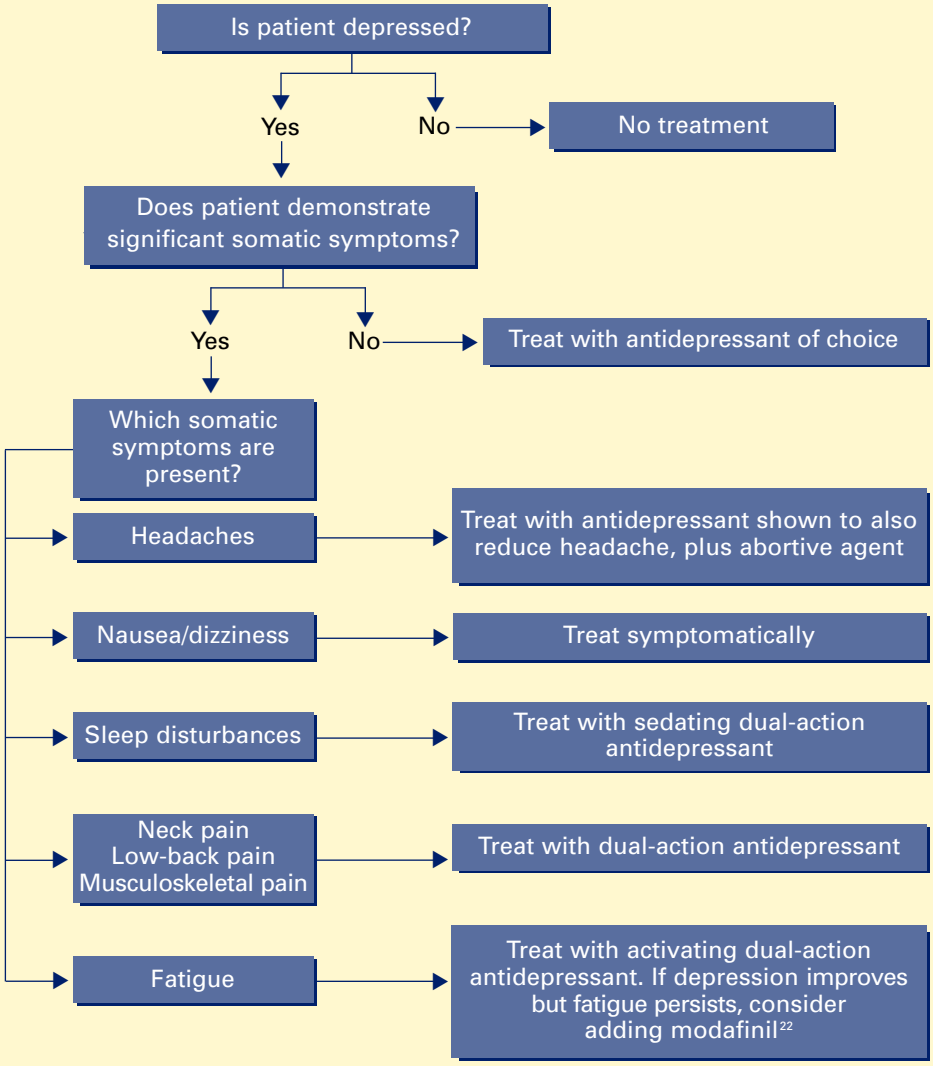
TREATMENT No pain, some gain

Another psychiatrist who specializes in pain medicine targeted some of Ms. F's somatic symptoms with antidepressants. Paroxetine and zolpidem were discontinued and the patient was started on:

- venlafaxine, 37.5 mg bid, titrated to 225 mg/d across 2 weeks. Because of its activating properties, venlafaxine was chosen to address Ms. F's pain and daytime fatigue.
- amitriptyline, 50 mg at bedtime nightly, to promote sleep
- prochlorperazine, 10 mg as needed, and meclizine, 25 mg as needed, to treat her nausea and dizziness, respectively.

Algorithm

Suggested drug treatment of depression with somatic symptoms



Ms. F also was advised to take an abortive migraine compound (Midrin, 2 tablets at headache onset and 1 additional tablet every half-hour as needed, maximum 5 tablets per day). Mr. F's primary care physician also referred her to a physical therapist to treat her neck and low-back pain; workup revealed no surgically treatable problem.

Four weeks later, Ms. F reported that her somatic symptoms significantly improved and that she was sleeping nearly 8 hours per night. Her BDI score was 12, indicating normal mood. She was functioning much more effectively at work and could once again routinely perform her daily activities.

Ms. F continued her medication regimen for 8

Related resources

- Fishbain DA, Cutler R, Rosomoff HL, Rosomoff RS. Chronic pain-associated depression: antecedent or consequence of chronic pain? A review. *Clin J Pain* 1997;13:116-37.

DRUG BRAND NAMES

Amitriptyline • Elavil
Duloxetine • Cymbalta
Meclizine • Antivert
Modafinil • Provigil
Paroxetine • Paxil
Prochlorperazine • Compazine
Venlafaxine • Effexor
Zolpidem • Ambien

DISCLOSURE

Dr. Fishbain is a consultant to Eli Lilly and Co. and a speaker for Purdue Pharmaceuticals.

months, after which she was lost to follow-up. At her most recent visit, her depression remained in remission. Her pain persisted, though at a lower intensity.

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Bottom Line

Pain and other somatic symptoms of depression can stall remission and hasten relapse. Target both physical and emotional symptoms when treating depression. Choose antidepressants that exhibit both analgesic and psychotropic effects.