Depression and Parkinson's Disease

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Parkinson's disease (PD) is generally considered a neurological disorder. However, because of the frequency of mood and other psychiatric complications, PD could also be considered as a neuropsychiatric disease. In fact, James Parkinson himself observed in 1817 that depression is commonly associated with PD.

The estimated prevalence of depression in PD varies according to the type of mood assessment applied (interview versus self-report questionnaire), the diagnostic criteria or definition of depression (the number and type of symptoms required), and the research setting (community-based versus neurology clinic). The data available suggest that at any given time, 20 to 40 percent of individuals with Parkinson's disease are experiencing a depression of some type and this is a higher rate than that found in the general population.

Depression can be difficult to diagnose in PD because of the overlap between symptoms of depression and PD. For example, the biological symptoms typical of depression, such as low energy, insomnia or excessive
sleep, weight loss, diminished sexual function and an emotionless face can be directly related to the neuroanatomical disruption characteristic of PD. These symptoms are not necessarily evidence of depression and conversely, the psychomotor slowing of depression might be accidentally overlooked in a patient with PD.

Depression in PD could be considered an understandable reaction to a disabling chronic illness, however some researchers believe depression may be a part of the disease and caused by neurological changes. This notion is supported by the fact that sometimes depression precedes the diagnosis of PD. Of course, the cause of depression in patients with PD could be a combination of a subjective reaction to the illness and the brain changes brought about by the disease. The consensus is that depressive symptoms should be addressed and treated by physicians regardless of whether they are part of PD itself or due to a separate cause.

The psychiatric complications of PD require attention because they can exacerbate the already considerable physical challenges brought on by the disease. In fact, an international survey of patients with PD discovered that depressive symptoms were the most important factor in patient quality-of-life ratings. Another reason to treat depression in PD is that reports of caregiver burden correlate significantly with patients' depression and quality-of-life.

It has been recommended that optimal anti-Parkinson symptoms treatment should be the first step in the treatment of PD. It is notable that some PD medications may have an antidepressant effect of their own. The antidepressant effect of dopamine agonists, such as pramipexole, is probably due to stimulation of D3 dopamine receptors, whereas the drug's effect on PD symptoms is related to the D2 dopamine receptor. Dopamine agonists may be helpful in particular for patients who experience on-off motor fluctuations, with their depressive symptoms being related to the "off" periods.

When the use of an antidepressant is considered in a patient with PD, its potential side effects and interactions with PD drugs must be weighed against the effects of the depression itself. Although it should be noted that the majority of the studies did not include a placebo control, there is evidence that antidepressant medications can have a definitely positive effect on depression in PD.

There are different classes of antidepressant medications. The selective-serotonin reuptake inhibitors (SSRIs: e.g., Zoloft®, Paxil®, and Celexa®) are prescribed most often in PD patients with depression. In general, the SSRIs are safer and better tolerated by patients than the tricyclic antidepressants (TCAs: e.g., Elavil®, Tofranil®, and Pamelor®). The
SSRIs have fewer cardiac and cognitive side effects and in addition, they can effectively treat anxiety and pain, which also are common in patients with PD.

Research studies have shown that PD symptoms can worsen as a result of SSRI use, but this adverse effect occurred only in a small minority of patients, and the effect was reversed after discontinuation of the medication. The combination of Selegiline and SSRIs could potentially result in a deadly serotonin syndrome. One author states that when Selegiline is being taken at a dose greater than or equal to 20 mg per day, it should not be used together with SSRIs. Finally, more research is necessary to determine the best therapeutic ranges for antidepressant medications in individuals with PD.

Some patients with depression either experience medication interaction effects, and do not respond to pharmacotherapy for depression or simply are reluctant to take another medication. For these patients in particular, effective psychotherapeutic options would of course be valuable. To date, there have been very few studies of the effectiveness of psychotherapeutic techniques, such as cognitive behavioral therapy (CBT), for the treatment of depression in PD. The research that has been done suggests non-pharmaceutical approaches can help to improve mood, quality of life, and health outcomes. In one study, those individuals with the most severe depression seemed to benefit the most from CBT. The use of coping strategies and other techniques for managing grief and other emotional distress still can be effective in this context.

Attending a Parkinson support group can be very useful.

Now with the growing trend toward use of online support groups they may also serve patients with PD well. Communicating with a group via computer allows one to access support without leaving the home. For individuals who have difficulty typing, a friend or spouse could possibly do the computer keyboard typing. A recent study in California found that participation in an online support group that included professional facilitators positively affected mood and quality of life in a group of patients with PD.

In non-demented PD patients, electroconvulsive therapy (ECT) can be an effective choice for depression when other treatments have failed. This treatment requires close management by a psychiatrist.

Regular exercise sometimes can help improve both physical and mental health in individuals with PD.

The usual cause of the onset of delusions, hallucinations, and paranoia in PD is either the addition of a new PD drug, such as amantadine, a dopamine agonist, or Selegiline, or an increase in levodopa. A head injury or a metabolic imbalance can also be responsible for these types of behavioral changes. If the dramatic change in behavior is actually due to a primary psychotic depression, rather than an
adverse medication or an injury, both an antidepressant and an atypical antipsychotic medication are called for. At the same time, a reduction in or elimination of one or more of the patient’s anti-parkinsonian drugs might be necessary.

DBS surgery, including bilateral subthalamic nucleus (STN) surgery, can result in the appearance or exacerbation of personality, anxiety, or mood disorders in some PD patients. In particular, a history of major depression is a risk factor for a significant post-operative mood disorder, even when surgery results in marked improvement in motor functioning.

In general, it should be emphasized that current antidepressant medications and psychotherapy have shown effectiveness for patients with PD, and so both the depressive symptoms and motor symptoms of the illness should be addressed.

This article was adapted from the December 2006 issue of the APDA Madison, WI I&R Center. It is based on a number of previously published articles, and references are available upon request from the APDA national office.

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