Parkinson’s Disease: You Make the Call

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PROLOGUE
Parkinson’s disease is reviewed and discussed during a two-hour lecture, presented to third-year doctor of pharmacy candidates. During the prior year, the students had lectures addressing the basic pharmacology of anti-Parkinson’s agents. This discussion introduces the students to a chronic and complex disease state that requires the pharmacist to monitor drug therapy for the entire life of the patient. It is also one of the initial lectures that challenges the student to think in clinical terms. Mr. Timothy Nigra, a man with Parkinson’s, is presented as a case study that unfolds in the context of the discussion and spans the last 12 years of his life. The case study is used to demonstrate to the students that pharmacists deal with real problems in real people. Today’s pharmacist is responsible for drug therapy outcomes and this case illustrates the need for continued contact and collaboration with the patient as well as the patient’s other health care providers.

The various classes of anti-Parkinson’s drugs available to treat patients are presented. Mr. Nigra is introduced early in the discussion so that the patient slowly comes alive, thereby allowing the students to become intimately involved with the drug therapy issues faced by the patient and his other health professionals. In this lecture and exercise the teacher serves as a facilitator, allowing students to begin to experience the “art” of providing pharmaceutical care while encouraging them to become proactive members of Mr. Nigra’s health care team. Likewise, the case and discussion present some of the difficulties in separating out physical signs and symptoms due to a disease and the role of drug therapy in maintaining symptom control while limiting drug complications.

INTRODUCTION
Parkinson’s disease is a chronic, debilitating neurologic disorder afflicting approximately one percent of people over the age of 50 in the United States(1). The impact of this disease is similar to Alzheimer’s in that it not only affects the patient but also greatly impacts immediate family members, friends, and other social relationships. This disorder was first characterized in an essay written in 1817 by Dr. James Parkinson. His description included statements such as: “involuntary tremulous motion,” “propensity to bend the trunk forward,” and the patients “pass from a walking to a running pace”(2). Parkinson’s disease is characterized by the following four classic features: bradykinesia, tremor (the classic “pill rolling” motion of the thumb and forefinger), muscle rigidity, and loss of postural reflexes. Parkinson’s is insidious and slowly progressive and signs are often not noted until 80 percent of the dopaminergic neurons are lost or become nonfunctional(3). Initially, Parkinson’s responds well to drug therapy, but as neuronal damage ensues, drug therapies become less efficacious.

MR. NIGRA’S INTRODUCTION
May 12, 1998: Your pharmacy career has just started and your interest in neurology has provided you the opportunity to work with “Neuron Associates” a group of neurologists. Your first patient is Timothy Nigra, a 73 year old, African American male, who is 5’ 11” and weighs 78.6 kg. He recently is experiencing hand tremors, the primary reason for seeing his neurologist Dr. Mary Dura-Mater. Mr. Nigra is also worried that he is “slowing down” and wonders if he isn’t anemic. Past medical history includes Type 2 diabetes diagnosed three years ago, hypertension of seven years duration, and benign prostatic hypertrophy (BPH) diagnosed in the past year. He is a retired farmer and has been married for 54 years and has five daughters and two sons. Currently he works at the local school district and smokes one pack of cigarettes a day. Drug Regimen: Metformin 1,000 mg bid, Enalapril 20 mg qd, ASA 325 mg qd, Multivitamin with minerals qd. Blood pressure is stable at 128/84 and HbA1c is 7.4 percent. Recent laboratory tests do not indicate any evidence of anemia. Dr. Dura-Mater does not prescribe any therapy at this time since the disease is not appreciably affecting Mr. Nigra’s daily activities.

PATHOPHYSIOLOGY OF PARKINSON’S DISEASE
The extrapyramidal system (EPS) is the functional unit in the CNS that modulates motor activities and is responsible for posture, muscle tone, and voluntary muscle function. Subcortical masses of gray matter known as the basal ganglia comprise the EPS( ). Dopaminergic pathways in the brain include the nigrostriatal (extrapyramidal), mesolimbic, mesocortical, and tuberohypophyseal. The nigrostriatal tract is the primary dopaminergic system in the brain affected by Parkinson’s and neuronal loss and degeneration of dopamine producing cells in the substantia nigra of the midbrain are the hallmark pathoneurologic findings in Parkinson’s disease(5).

The cholinergic neurons in the striatum are normally under the inhibitory influence of dopamine. When dopamine levels decrease in the striatum, cholinergic excess occurs thus upsetting the normal balance of dopamine and acetylcholine in this area of the brain. Decreased dopamine and increased acetylcholine levels are targeted pharmacologically to regain neurotransmitter balance and to treat the symptomatology of Parkinson’s(6).
Table I: Vocabulary of Parkinson’s disease

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tr>
<td>Akinetia</td>
<td>Inability to adequately initiate or implement involuntary movement, (attempt to pick up a pencil but make very tentative motions that do not accomplish the task)</td>
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<tr>
<td>Bradykinia</td>
<td>Slowness of movement, (walk across the floor in a slow, shuffling motion)</td>
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<tr>
<td>Dysskinesia</td>
<td>Involuntary movements of the musculature (jerky movements of the neck)</td>
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<tr>
<td>Dystonia</td>
<td>Abnormal tonicity of the muscles resulting in sustained postures or muscle contractions, (take shoes off and flex the toes and feet)</td>
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<tr>
<td>Festination</td>
<td>Acceleration of gait associated with Parkinson’s disease, (start walking slow and then speed up, with feet being unable to keep up with the upper body and almost fall or run into the wall)</td>
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<tr>
<td>Hypomimia</td>
<td>Masklike facies, (expressionless, staring gaze with infrequent blinking)</td>
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<tr>
<td>Myoclonus</td>
<td>Clonic spasm or twitching of the muscles, (twitch shoulder and arm in violent swinging motion over the head)</td>
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**SIGNS AND SYMPTOMS**

How does a person with Parkinson’s appear or present? As previously mentioned, there are four classic features of Parkinson’s which we can readily recite. But how do these people appear if you were to meet one patient today. Upon physical assessment the patient may exhibit cogwheel rigidity (ratchethlike movements of a limb when passively stretched), hypotension, and demonstrate a shuffling gait with a simian posture. Additionally, many patients experience festination, or the tendency to speed up as they walk an observation made by Dr. Parkinson in 1817. Diminished or nonexistent arm swing when walking is also common. If you were to sit across the table from a patient, hypomimia (masklike facies) with a decreased blink rate may be observed as well as, hypophonia (soft voice), and sialorrhea (drooling). The fact that Muhammed Ali, Pope John Paul II, and Reverend Billy Graham all have Parkinson’s disease is noted to have the students “fix” images of these famous people in their minds as representations of Parkinson’s.

Common symptoms include constipation, sweating, keratitis sicca, and intermittent immobility referred to as “freezing.” Depression and abnormal sleeping habits are associated conditions frequently present in patients with Parkinson’s disease. Many of the symptoms of Parkinson’s disease may also occur due to drug treatment. Sweating and constipation in a patient may be due to the disease, a drug, or a combination of both. At this point, the vocabulary describing abnormal muscle movement is introduced and demonstrated (see Table I).

What signs/symptoms of Parkinson’s is Mr. Nigra experiencing? This question requires the student to match the characteristics of the disease in a person in the early stages of Parkinson’s. In addition to the classic tremor, the students should recognize the “slowing down” as evidence of bradykinesia, one of the hallmark signs. This stresses the point that patients don’t come with a description of their disease but we often need to interpret what the patient says. The students are not being trained as diagnosticians, but they need to be able to identify the presenting signs and symptoms of Parkinson’s and understand how these may be monitored by pharmacists as drug therapy is instituted.

**PHARMACOLOGICAL THERAPY**

Currently, five broad classes of drugs are available to treat Parkinson’s. Another class of drugs known as the catechol-O-methyltransferase inhibitors (COMT inhibitors) are currently under intense investigation and may represent an additional drug class in the future. In Parkinson’s disease the main pharmacologic attempt is to increase dopamine and its activity in the EPS. Unfortunately, Parkinson’s is accompanied by progressive neuronal loss leading to drug failures overtime. One of the main challenges in treating patients is to separate a drug’s side effects from the disease itself. Also, with concurrent depression, insomnia, and other confounding conditions and comorbidities, treatment is often complex and highly individualized. The statement by Cipolle, “Drugs don’t have doses-people have doses,” is particularly applicable to the management of patients with Parkinson’s disease, as therapeutic response and incidence and type of side effects is highly variable and patient specific(7).

**MAO-B INHIBITORS**

Selegiline selectively inhibits the monoamine oxidase B (MAO-B) enzyme in the brain at therapeutic doses(8). MAO-B is involved in the metabolism of dopamine in the striatum and by effectively inhibiting this enzyme’s function, more dopamine is available to the neurons. Amphetamine and methamphetamine are two byproducts of selegiline metabolism, both of which may contribute to increase brain levels of dopamine(9). Additionally, selegiline may offer some neuroprotective effects by inhibiting free radical formation, although this is an area of current debate(10,11). Recommended dosing of selegiline is 5 mg bid and early morning and early afternoon administration will help avoid evening insomnia. In addition to insomnia, other side effects may include confusion, hallucinations, nausea, and orthostatic hypotension. A severe drug interaction between selegiline and meperidine resulting in pronounced agitation and delirium accompanied by muscle rigidity, sweating, and elevated body temperature has been documented and these two drugs should not be prescribed concurrently(12).

**Nine Months Later:** On February 27, 1999 Mr. Nigra’s tremor is worsening as is his bradykinesia. He is prescribed selegiline 5 mg pc breakfast and lunch. The students are then asked to counsel Mr. Nigra on his new therapy. Students offer their suggestions and it is stressed that the following points need to be made: take after breakfast and lunch, discuss orthostatic hypotension and offer suggestions how to minimize it, report any unusual symptoms, particularly signs of hypertensive crisis, i.e., chest pain, headache (HA). Also, instruct the patient that if xerostomia is experienced it can be relieved with gum or candy, ice chips or a saliva substitute.

The students are then asked to monitor the effectiveness of this patient’s therapy. This should consist of regular phone contact with the patient as well as periodic face-to-face meetings with the patient. Response to therapy should be correlated with a decrease in symptoms and any side effects should be addressed. Blood pressure should be monitored and questions concerning dry mouth, abnormal movements or changes in mental status should be discussed as well the patients perceptions on how the treatment is work-
ing. Also, since selegiline can potentially exacerbate BPH, changes in urinary habits should be addressed. It is agreed upon that Mr. Nigra will receive a phone call every month, and will be visited at home or seen at the clinic by the pharmacist every three to four months.

**Carbidopa/Levodopa**

Treatment with levodopa is the cornerstone of therapy for Parkinson’s Disease as it has been proven to clinically alleviate signs and symptoms and decreases disability(13). Levodopa is combined with the peripherally acting dopa-decarboxylase inhibitor carbidopa to prevent the peripheral conversion of levodopa to dopamine, as this conversion of levodopa contributes to increased side effects and patient discomfort. Carbidopa in doses of 50 to 100 mg per day prevents peripheral decarboxylation of dopamine(14). Initial doses of carbidopa/levodopa are in the range of 25/100 mg given two to three times daily. Dosing adjustments are made based on patient response, and levodopa doses as high as 1,200 to 1,500 mg have been used in some patients(15).

Side effects of levodopa therapy include nausea, vomiting, orthostatic hypotension, cardiac dysrhythmias, vivid dreams, as well as confusion and nightmares. This drug combination should be used cautiously in patients with narrow-angle glaucoma, malignant melanoma, and in those with angina or dysrhythmias.

Therapeutic issues associated with Parkinson’s disease and levodopa treatment include the end of dose effect commonly referred to the “wearing off” and the “on-off” phenomenon, also referred to as the “all or nothing” effect. “Wearing off” is experienced as the disease progresses and the patient receives benefit for shorter period of times from each dose of drug. Levodopa needs to enter the neuron and be metabolized to dopamine where it is then stored in presynaptic vesicles. With disease progression, fewer neurons are available to provide storage sites and a more constant delivery of drug to the brain is needed to compensate for the loss of neurotransmitter storage. Two therapeutic strategies used to treat “wearing off” include decreasing the interval between doses or to use a sustained release product.

The “on” in the “on-off” phenomenon refers to the normal or dyskinetic movements when levodopa therapy action is at its peak and the “off” period, also called “freezing,” is when bradykinesia or akinesia presents due to diminished or absent drug action. Similar strategies used to treat the end of dose effect may be used to treat the “on-off” phenomenon (more frequent dosing, use of a sustained release product, adding selegiline). Additional benefit may be experienced if dietary protein is limited, since excess protein can compete for levodopa absorption. Likewise, decreased stomach emptying leads to delayed drug absorption and the use of antacids or having the patient chew the tablet may increase absorption rate. Unfortunately, not all patients respond to the above strategies. In one study the “on-off” effect was weakly correlated to the plasma levodopa levels, which may explain why some patients don’t respond as expected to changes in therapy(16).

Utilization of the “drug holiday” was once a strategy employed to increase the effective response to anti-Parkinson’s therapy in patients whose response declined over time. During the holiday, the patient was discontinued on all anti-Parkinson’s drug therapy to modify the dopamine receptors in the hopes of increasing their sensitivity and response upon drug reinitiation. Patients often experienced severe and potentially life-threatening complications such as neuroleptic malignant syndrome during the holiday and this strategy is no longer practiced.

**Ten Months Later:** December 12, 1999, Mr. Nigra reports to you over the phone that he is experiencing increased tremor, muscle stiffness and rigidity. He says, “I’m not able to move at my normal pace, either.” The above signs and symptoms should be recognized as a decrease in drug effectiveness or due to disease progression. Also, he has recently been diagnosed with diabetic gastropathy and prescribed metoclopramide by his primary care physician. It is your job to call the physicians with recommendations. What might they be?

The discussion with the neurologist is initiated and the logical conclusion is that carbidopa/levodopa needs to be added or substituted to his current regimen. After an exam by the physician carbidopa/levodopa 25/100 tid is added to Mr. Nigra’s drug regimen on January 2, 1999. You consult with Dr. Dura-Mater during the visit. Given that Mr. Nigra is older and also on selegiline therapy the students recommend a slower dose titration. It is agreed that one tablet daily will be given for week one, bid for the second week, then tid thereafter. The primary care physician is contacted and educated on the potential drug/disease interaction with metoclopramide due to its dopaminergic blocking properties in the striatum. It is decided to use cisapride, because its mechanism of action does not affect dopamine or other brain neurotransmitters. You get a call from Mr. Nigra’s certified diabetes educator wondering why cisapride was used in place of metoclopramide. You explain the rational and she is appreciative of your explanation and expertise. Once again the students are asked to counsel the patient on the new medications and to monitor his therapy.

Many of the same points made with selegiline are reinforced with the addition of levodopa. It is particularly important to report and to monitor for abnormal muscle movements. The students are then asked if they have any further questions of the neurologist. One question that arises relates to the length of time of selegiline therapy. With a quick call to the physician it is discovered that it is his standard practice to discontinue the medication one year after starting levodopa therapy. This information is noted and indeed the selegiline is discontinued as planned.

**Anticholinergic “AntiTurkey Dinner”Agents**

Anticholinergic agents restore neuronal function by blocking cholinergic receptors thereby rebalancing the cholinergic and dopaminergic systems in the basal ganglia(17). Prior to levodopa therapy, anticholinergics were the preferred therapy. They tend to be most effective for tremor and rigidity while offering little benefit for bradykinesia. Various agents are available with little therapeutic advantage of one over the others, although diphenhydramine tends to be quite sedating. The main disadvantage of the anticholinergic drugs are their adverse effects that include dry mouth, urinary retention, blurred vision, sinus tachycardia, and constipation. Central anticholinergic side effects include confusion, decreased memory, delirium and psychosis, with the elderly being particularly sensitive to these effects.

The concept of “anti-turkey dinner” is then introduced. When a large meal is eaten such as the turkey feast on...
Thanksgiving Day our bodies are predominately under cholinergic activity. The digestive system is working at maximal efficiency, with adequate saliva production and proper gastrointestinal motility. Pupils are normal sized or smaller than normal (miosis) and the heart rate slows. Anticholinergics may have the opposite effects and I refer to this as “anti-turkey dinner” effects. Xerostomia, mydriasis resulting in blurred vision, increased heart rate, and constipation may occur. Explanation of the “anti-turkey dinner” effects is an effective method of helping the students recognize the classic side effects of anticholinergic drugs (see Table II).

<table>
<thead>
<tr>
<th>Turkey dinner</th>
<th>Anti-turkey dinner</th>
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<tr>
<td>Miosis</td>
<td>Mydriasis</td>
</tr>
<tr>
<td>Salivation</td>
<td>Xerostomia</td>
</tr>
<tr>
<td>GI activity increased</td>
<td>GI activity decreased (constipation)</td>
</tr>
<tr>
<td>Normal urination</td>
<td>Urinary retention</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>Tachycardia</td>
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In general anticholinergics should be avoided or used cautiously in patients with cardiac disease, gastrointestinal or genitourinary obstruction, and in patients with narrow-angle glaucoma. Additionally, concurrent use with CNS depressants or alcohol should be discouraged, and driving should be postponed until individual patient response is known.

Three Years Later: March 27, 2002, Mr. Nigra says that the drugs he is taking are not working quite as long as they used to and that he has been experiencing insomnia that is responding well to diphenhydramine. From Mr. Nigra’s description in the later part of the previous sentence, the students should recognize that Mr. Nigra is experiencing the “wearing off” phenomenon. Constipation is also periodically bothersome. Dr. Dura-Mater increases his carbidopa/levodopa to 25/100mg qid. Once again counseling is provided and issues concerning the diphenhydramine are addressed. You discover that he has had a transurethral transection of his prostate six months previously, so you are not as concerned about urinary retention. However, the central anticholinergic effects are discussed and you both agree that occasional use is appropriate as long as additional side effects aren’t experienced. The students note to monitor his insomnia more vigilantly with the increase in the carbidopa/levodopa dose. A docusate/casamthral product is suggested and prescribed for the constipation. Mr. Nigra is counseled on the proper use of the laxative and other nonpharmacologic strategies are offered. I emphasize to the students that both the insomnia, constipation and “wearing off” can be indicators of disease progression. With the recent change in his drug regimen, Mr. Nigra’s end of dose effect becomes less of a problem and he is able to continue working at the elementary school.

Dopamine Reuptake Blocker
The antiviral agent amantadine, is a dopamine reuptake blocker that may also increase dopamine storage and release. It has been shown to have modest efficacy in patients with Parkinson’s patients with akinesia and rigidity. Unfortunately, patients quickly develop tolerance to its effects and it is rarely used for more than a few months. The initial starting dose is 100 mg bid, although patients with renal compromise require downward adjustment. Amantadine can be used as a single agent or in conjunction with carbidopa/levodopa(17). Side effects of amantadine therapy include dry mouth, constipation, urinary retention, hallucinations, insomnia, and psychosis. An unusual, but benign and reversible side effect of amantadine is the appearance of a reddish blue rash known as levento reticularis.

Direct Dopamine Agonists
The dopamine agonists directly stimulate postsynaptic receptors. Bromocriptine is a D2 agonist and D3 antagonist while pergolide is a D, and D2 agonist. These classic ergoline dopamine agonists are commonly prescribed when the clinical response to carbidopa/levodopa therapy declines. Newer, nonergoline dopamine agonists include ropinirole and pramipexole and may be used as monotherapy or as adjuncts to carbidopa/levodopa therapy. Ropinirole and pramipexole are potent D2 agonists, with pramipexole also having alpha-2 adrenergic receptor antagonist activity and a greater propensity to cause orthostatic hypotension(18).

A new approach advocated and practiced by some neurologists is to initiate therapy with the newer dopamine agonists. It has been shown that the dopamine agonists infrequently result in the motor fluctuations and dyskinesias commonly experienced with levodopa therapy(18). In general, the dopamine agonists need to be gradually titrated and changes should be based on patient response and tolerance. When adding a dopamine agonist, the dose of levodopa can often be reduced by 20 percent. Side effects of the direct acting dopamine agonists include nausea, postural hypotension, dizziness, dry mouth and constipation. CNS side effects include confusion, hallucinations, somnolence, and vivid dreams or nightmares. The development of pulmonary or retroperitoneal fibrosis is related to the presence of the ergot structure in bromocriptine and pergolide, and these adverse events have not been noted with the use of the nonergoline drugs(18). Maximal dosing is often limited by the development of side effects, which tend to resolve when the dosage is decreased or the drug discontinued.

Three Years Later: February 25, 2005, Once again, Mr. Nigra reports that his medicine is working as long as it once did (“wearing off”). Recently, his poor appetite has been accompanied by a 12 pound weight loss. He also expresses “hopelessness” about the future. His social worker calls you wondering if there is anything else can be done for her client. She reports that Mr. Nigra has quit his job and for the past three months has rarely left his house. What are you going to tell the social worker? What recommendations are you going to make to the physician? The previous description of weight loss, poor appetite, and feelings of hopelessness should be recognized as possible signs of depression. Discussions with the social worker include an overview of Parkinson’s disease and its progression. The therapies for Parkinson’s and depression are discussed as well. A Parkinson’s support group that is run by Neuron’s Associates is recommended and the social worker is encouraged to contact a local senior center that has other members with Parkinson’s disease.

Levodopa dosing issues are discussed with the students and the possibility of adding a dopamine agonist is enter-
tained as well as an antidepressant. The TCA and SSRI class of drugs are most familiar to students at this point in their careers. The side effect profile of the SSRIs and the anticholinergic side effects of the TCAs are highlighted. These medicines are discussed and the physician is called to update her on recent changes in Mr. Nigra’s health.

After a visit to the physician, Mr. Nigra’s “wearing off” is addressed and he receives a diagnosis of depression. He is instructed to take carbidopa/levodopa 10/100 tid, carbidopa/levodopa sustained release (CR) 50/200 bid, and paroxetine 10 mg qd. The physician did not want to add a dopamine agonist at this time, but may in the near future if symptoms progress. Once again the student is required to counsel and monitor the patient. Mr. Nigra is made aware that with increasing levodopa doses side effects need vigilant monitoring and that the paroxetine may help with insomnia and should be titrated to 20 mg over the next month. The potential but mild anticholinergic side effects of paroxetine are discussed with the patient also.

Adjunctive Therapies

Propranolol has been effective in controlling the tremor associated with Parkinson’s disease(19). Hypotension and the propensity for propranolol to cross the blood brain barrier causing depression or other psychiatric disorders should always be considered. Keratitis sicca is commonly encountered due to decreased blinking and/or the use of anticholinergic medicines. Various forms of artificial tears are available to alleviate the discomfort associated with this condition. Many patient’s with Parkinson’s also suffer form chronic constipation and the choice of a laxative is dependent upon factors such as severity, mobility, and clinical response to past use.

Two Years Later: March 1,2007, Mr. Nigra is tired of taking so many doses of medicine. He has also fallen a few times and is more forgetful and less able to recall words when speaking (early signs of dementia). You call the physician and recommend carbidopa/levodopa CR (50/200) tid and carbidopa/levodopa 10/100 in the morning before getting out of bed. The physician and patient are satisfied with the change. Six months later Mr. Nigra is requiring more assistance with his activities of daily living. He is also complaining of dry eyes, insomnia, and constipation. Recently he was treated for pneumonia. You were on vacation when he had pneumonia and you discover that he was started on pramipexole during your absence. The physician is contacted and the recommendation is made to use ropinirole, in place of pramipexole during your absence. The physician is contacted and the recommendation is made to use ropinirole, in place of pramipexole, due to its decreased incidence of postural hypotension. A 20 percent decrease in the dose of levodopa is also advised. Additionally, zolpidem is recommended for his insomnia, since diphenhydramine can contribute to constipation and contribute to a worsening of the dementia. The physician accepts your suggestions and starts Mr. Nigra on ropinirole 0.25 mg tid, and the early morning dose of levodopa is discontinued. Zolpidem, 5 mg at bedtime is prescribed prn. You then counsel him on his new therapy and continue to monitor. Counseling points for the ropinirole include: discussion of side effects including headache, vomiting and insomnia, and that slow upward dose titration may be needed depending on the clinical response. The doctor forgot to recommend a product for his dry eyes and you suggest an artificial tears product.

The Future

Catechol-O-Methyltransferase (COMT) is capable of metabolizing levodopa to 3-O-methyltyrosine, which competes with levodopa transport across the blood brain barrier. When dopa-decarboxylase activity is inhibited by carbidopa, levodopa is more likely to be metabolized by the COMT enzyme. By inhibiting COMT, more levodopa remains intact and less methyldopa is formed which decreases the competition for levodopa uptake into the brain(20). In early studies, COMT inhibitors have been shown to prolong the effects of levodopa and may prove to be effective adjuncts in the treatment of Parkinson’s disease(21). Posteroventral pallidotomy is effective in alleviating dyskinesias in some patients and neural transplantation of fetal cells and other surgeries may prove beneficial in the future.

Three Years Later: March 15,2010, Mr. Nigra is diagnosed with inoperable lung cancer and passes away two months later. You wonder if you shouldn’t have more aggressively encouraged smoking cessation in your patient.

CONCLUSION

Parkinson’s disease is a chronic, debilitating disease that is best managed by a dedicated team of health professionals that should include a neurologist, a pharmacist, and social worker. Since a cure is not presently available, Parkinson’s patients require lifelong drug therapy that can be expertly managed by a pharmacist. When prescribing Parkinson’s agents the age of the patient, presence of comorbidities, cognitive function, and physical disabilities must be taken into account. Response to treatment is highly variable and signs and symptoms related to disease progression can easily be mislabeled as drug side effects. The pharmacist has unique knowledge of pharmacotherapy and is in the ideal position to make positive contributions to Parkinson’s patients and the patients’ healthcare team.

References

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