REVIEW

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Movement disorder emergencies in the elderly: Recognizing and treating an often-iatrogenic problem

ABSTRACT

Movement disorder emergencies in the elderly—such as rigidity, dystonia, hyperkinetic movements, and psychiatric disturbances—are challenging to manage. Many cases are iatrogenic. In theory, some cases could be avoided by anticipating them and by avoiding polypharmacy and potentially dangerous drug interactions.

KEY POINTS

Supportive measures must be taken immediately to maintain the functions of vital organs.

Serotonin syndrome, which can cause rigidity or stiffness, can be prevented by avoiding multidrug regimens.

Withdrawing or decreasing the dose of dopaminergic drugs in patients with Parkinson disease can cause parkinsonism-hyperpyrexia syndrome, a condition similar to neuroleptic malignant syndrome.

Metoclopramide (Reglan) accounts for nearly one-third of all drug-induced movement disorders. The entire spectrum of drug-induced movement disorders, ranging from subtle to life-threatening, can ensue from its use.

Complications of Parkinson disease include hallucinations, dementia, depression, psychosis, and sleep disorders.

*Dr. Tousi has disclosed that he has received honoraria from UCB for teaching and speaking.

ALTHOUGH WE TEND to think of movement disorders as chronic conditions, some of them can present as true emergencies in which failure to diagnose the condition and treat it promptly can result in significant sickness or even death.

Many cases are iatrogenic, occurring in patients with Parkinson disease or those taking antipsychotic or antidepressant medications when their regimen is started or altered. Elderly patients are particularly at risk, as they take more drugs and have less physiologic reserve.

Movement disorder emergencies in elderly patients can be difficult to diagnose and treat, since many patients are taking more than one medication: polypharmacy raises the possibility of interactions, and different drugs can cause different movement disorder syndromes. Moreover, because so many patients are at risk—for example, more than 1 million people in the United States now have Parkinson disease, and the number is growing—it is important for physicians who take care of the elderly to be more informed about these disorders, especially the presenting symptoms.

SCOPE OF THIS ARTICLE

Movement disorder emergencies can be classified into four main categories (**TABLE 1**):

- Disorders presenting with rigidity or stiffness
- Disorders presenting with dystonia
- Disorders presenting with hyperkinetic movements

TABLE 1

Movement disorder emergencies in the elderly

Disorders presenting with rigidity or stiffness
Serotonin syndrome
Neuroleptic malignant syndrome
Parkinsonism-hyperpyrexia syndrome
Akinetic syndrome after failure of a deep brain stimulator

Disorders presenting with dystonia
Acute dystonic reaction
Laryngeal dystonia accompanied by multiple system atrophy
Sudden withdrawal of baclofen (Kemstro, Lioresal)

Disorders presenting with hyperkinetic movements
Acute hemichorea and hemiballism
Severe parkinsonian dyskinesia
Drug-induced myoclonus
Drug-induced akathisia

Disorders with psychiatric presentations
Hallucinations and psychosis in Parkinson disease

- Disorders presenting with psychiatric disturbances.

Of these, the scenarios most likely to require emergency evaluation in the elderly are acute hypokinetic and hyperkinetic syndromes and psychiatric presentations. This article discusses movement disorder emergencies in the elderly, focusing on the more common disorders with common presentations.

Any drug that enhances serotonin can cause serotonin syndrome

DISORDERS PRESENTING WITH RIGIDITY OR STIFFNESS

Serotonin syndrome

Serotonin syndrome can occur in a patient recently exposured to a serotonergic drug or, more commonly, to two or more drugs.[3] Any drug that enhances serotonergic neurotransmission can cause serotonin syndrome (TABLE 2), especially in the elderly, who may not be able to tolerate serotonergic hyperstimulation.

Chief among the offenders are the selective serotonin-reuptake inhibitors (SSRIs), either alone or in combination. This syndrome occurs in 14% to 16% of patients who overdose on SSRIs.[1] Examples of combinations that can lead to serotonin syndrome are an SSRI plus any of the following:

- An anxiolytic such as buspirone (BuSpar; this combination is popular for the treatment of depression and anxiety)
- A tricyclic agent such as imipramine (Tofranil)
- A serotonin and norepinephrine reuptake inhibitor such as venlafaxine (Effexor).

In addition, antiparkinson drugs such as levodopa and selegiline (Eldepryl) enhance serotonin release.

Signs and symptoms. Serotonin syndrome is characterized by:

- Severe rigidity
- Dysautonomia
- Change in mental status.

Other clinical findings include fever, gastrointestinal disturbances, and motor restlessness. Clonus is the most important finding in establishing the diagnosis.[2]

Some features, such as shivering, tremor, and jaw quivering, differentiate serotonin syndrome from neuroleptic malignant syndrome (see below; TABLE 3). In addition, signs of neuroleptic malignant syndrome evolve over several days, whereas serotonin syndrome has a rapid onset. Hyperactive bowel sounds, diaphoresis, and neuromuscular abnormalities distinguish serotonin syndrome from anticholinergic toxicity.

The syndrome may initially go unrecognized and can be mistaken for viral illness or anxiety.[4] Manifestations range from mild to life-threatening; initially, it may present with akathisia and tremor. The symptoms progress rapidly over hours and can range from myoclonus, hyperreflexia, and seizures to severe forms of rhabdomyolysis, renal failure, and respiratory failure. The hyperreflexia and clonus seen in moderate cases may be considerably greater in the lower extremities than in the upper extremities.[5]

No laboratory test confirms the diagnosis, but tremor, clonus, or akathisia without additional extrapyramidal signs should lead to the diagnosis if the patient was taking a serotonergic medication.[5] The onset of symptoms is usually rapid. The majority of patients present within 6 hours after initial use of the medication, an overdose, or a change in dosing.[5]

Treatment. The first steps are to stop the serotonergic medication and to hydrate and cool the patient to counteract the hyperpyrexic state. Benzodiazepine drugs are important in

TABLE 2

Drugs and drug interactions associated with the serotonin syndrome

Drugs associated with the serotonin syndrome

Selective serotonin-reuptake inhibitors
- Sertraline (Zoloft)
- Fluoxetine (Prozac, Sarafem)
- Fluvoxamine (Luvox)
- Paroxetine (Paxil)
- Citalopram (Celexa)

Other antidepressant drugs
- Trazodone (Desyrel)
- Nefazodone (Serzone)
- Buspirone (BuSpar)
- Clomipramine (Anafranil)
- Venlafaxine (Effexor)
- Mirtazapine (Remeron)

Monoamine oxidase inhibitors
- Phenelzine (Nardil)
- Moclobemide (Manerix)
- Clorgiline
- Isocarboxazid (Marplan)

Anticonvulsants
- Valproic acid (Depakote)

Analgesics
- Meperidine (Demerol)
- Fentanyl (Actiq, Duragesic, Sublimaze)
- Tramadol (Ultram)
- Pentazocine (Talwin)

Antiemetic agents
- Ondansetron (Zofran)
- Granisetron (Kytril)
- Metoclopramide (Reglan)

Antimigraine drugs
- Sumatriptan (Imitrex)

Bariatric medications
- Sibutramine (Meridia)
- Dexfenfluramine (Redux)
- Fenfluramine (Pondimin)

Antibiotics and antivirals
- Linezolid (Zyvox), a monoamine oxidase inhibitor
- Ritonavir (Norvir), through inhibition of cytochrome P-450 enzyme isoform 3A4
- Imipramine (Tofranil)

Over-the-counter cough and cold remedies
- Dextromethorphan

Drugs of abuse
- Methylenedioxymethamphetamine (MDMA, or "ecstasy")
- Lysergic acid diethylamide (LSD)
- 5-methoxy diisopropyltryptamine ("foxy methoxy")
- Syrian rue (contains harmine and harmaline, both monoamine oxidase inhibitors)

Dietary supplements and herbal products
- Tryptophan
- *Hypericum perforatum* (St. John's wort)
- *Panax ginseng* (ginseng)
- Lithium (Eskalith)

Drug interactions associated with severe serotonin syndrome

Phenelzine and meperidine
Tranylcypromine and imipramine
Phenelzine and selective serotonin-reuptake inhibitors
Paroxetine and buspirone
Linezolid and citalopram
Moclobemide and selective serotonin-reuptake inhibitors
Tramadol, venlafaxine, and mirtazapine

FROM BOYER E, SHANNON S. SEROTONIN SYNDROME. N ENGL J MED 2005; 352:1112–1120.

Computer-based ordering systems can help one avoid drug interactions

controlling agitation, regardless of its severity.[5] Propranolol (Inderal) is not recommended, as it may cause hypotension and shock in patients with autonomic instability.[5]

Patients with moderate cases may additionally benefit from cyproheptadine (Periactin), an antihistamine that antagonizes serotonin. The initial dose is 4 to 8 mg orally, with a repeat dose after 2 hours.[6] Whether to continue this treatment depends on the response after two doses.

If medications must be given parenterally, physicians can consider chlorpromazine (Thorazine) 50 to 100 mg intramuscularly.[5]

Vital signs should be monitored. In severe cases, intensive care may be required with immediate sedation, neuromuscular paralysis, and intubation.

In most cases, patients improve rapidly.

Comment. Serotonin syndrome can be avoided by educating physicians and by modifying prescribing practices.[5] Avoiding multidrug regimens is critical to preventing serotonin syndrome. Computer-based ordering systems and personal digital assistants can help one avoid drug interactions.[5]

Neuroleptic malignant syndrome

This syndrome is an infrequent but potentially lethal complication associated with therapy

TABLE 3

Serotonin syndrome vs neuroleptic malignant syndrome

FEATURES	SEROTONIN SYNDROME	NEUROLEPTIC MALIGNANT SYNDROME
History		
Medication history	Serotonergic drugs	Dopamine antagonist
Onset of symptoms	Minutes to hours	Days to week
Physical examination		
Vital signs	Elevated temperature, pulse, respirations, blood pressure	Elevated temperature, pulse, respirations, blood pressure
Pupils	Mydriasis	Normal
Mucosa	Sialorrhea	Sialorrhea
Bowel sounds	Hyperactive	Normal or decreased
Neuromuscular tone	Increased, predominantly in lower extremities	"Lead-pipe" rigidity in all muscles
Reflexes	Hyperreflexia	Bradyreflexia
Mental status	Agitation, coma	From alert to stupor, mutism, coma
Laboratory values		
Elevated aminotransferases	Rare	Common
Rhabdomyolysis	Rare	Common

Neuroleptic malignant syndrome evolves over several days, whereas serotonin syndrome has a rapid onset

with antipsychotic drugs such as haloperidol (Haldol) and lithium (Eskalith) and with other medications with dopamine type-2 receptor antagonist activity such as metoclopramide (Reglan) and prochlorperazine (Compazine). It has become rare since the introduction of atypical antipsychotics and now occurs in 0.2% of patients receiving atypical antipsychotics.[7] Its pathogenesis is not fully understood.

This syndrome occurs mainly in young or middle-aged patients receiving doses of neuroleptics within the usual therapeutic range, but it also appears to occur in elderly patients who receive higher doses.[8] Although most cases develop in the first 2 weeks of treatment, it can develop at any time during therapy.

Signs and symptoms. Four features characterize neuroleptic malignant syndrome[9]:

- Muscle rigidity—generalized ("lead-pipe") muscular rigidity is accompanied by bradykinesia or akinesia.
- Autonomic dysregulation, with tachycardia, tachypnea, alterations in blood pressure, excessive sweating, and incontinence.
- Hyperthermia—fever can begin hours to days after initiating or increasing the dose of a dopamine antagonist.
- Altered sensorium, ranging from confusion to disorientation and coma.

Symptoms of neuroleptic malignant syndrome typically evolve over several days, in contrast to the rapid onset of the serotonin syndrome. Knowing the precipitating drug also helps distinguish the syndromes: dopamine antagonists produce bradykinesia, whereas serotonin agonists produce hyperkinesia.[5]

Laboratory abnormalities include elevated serum creatine kinase concentrations and white blood cell counts. Renal function should be assessed when renal failure and rhabdomyolysis are suspected.

Treatment involves stopping the causative medication, cooling the patient, and supporting vital functions.

In mild cases (eg, low-grade fever) benzodiazepines such as lorazepam (Ativan) can stabilize the condition. In moderate cases (eg, more significant rigidity), dopaminergic ago-

nists such as bromocriptine (Parlodel) can be given, although there is no strong clinical evidence for their use. Bromocriptine is usually started at 2.5 mg three times a day and gradually increased in dose if tolerated.

In severe cases, muscle rigidity can be reduced with dantrolene (Dantrium), a muscle relaxant. Dantrolene is started at 1 mg/kg intravenously every 6 hours and gradually increased up to 10 mg/kg total per day.

Some patients remain rigid and febrile up to 4 weeks after the causative agent has been withdrawn. Therefore, these treatments can be continued for a few weeks. After the patient has recovered fully, if it is necessary to resume antipsychotic therapy, an atypical antipsychotic such as quetiapine (Seroquel) can be started after 2 weeks.[8]

Comment. Although uncommon, neuroleptic malignant syndrome is the most serious adverse effect of neuroleptic drugs, and it is potentially fatal. When neuroleptic malignant syndrome is suspected, treatment should be prompt, and the neuroleptic medication should be immediately stopped.

Parkinsonism-hyperpyrexia syndrome

Withdrawing or decreasing the dose of dopaminergic medications in patients with Parkinson disease can cause parkinsonism-hyperpyrexia syndrome, a condition that is similar to neuroleptic malignant syndrome. It can also arise after sudden withdrawal of amantadine (Symmetrel) or anticholinergics. In view of this concern, adjustments to antiparkinson drugs may need to be more gradual in some elderly patients.

Patients present with fever, rigidity, and autonomic instability and are at risk of aspiration pneumonia.

Treatment includes resuming dopaminergic therapy and giving supportive care.

Apomorphine (Apokyn), a dopaminergic agonist, was used in a 71-year-old female parkinsonian patient who developed parkinsonism-hyperpyrexia syndrome after abrupt reduction of chronic levodopa treatment.[10] The symptoms resolved within 24 hours of the addition of apomorphine to her previous levodopa therapy. If the patient is taking apomorphine for the first time, the injections should be given in low doses, 0.2 mL subcutaneously. Apomorphine can induce vomiting, and if this occurs an antiemetic such as trimethobenzamide (Tigan) should be given before subsequent injections. In the elderly, caution is advised as apomorphine may cause severe orthostasis.

Methylprednisolone (Solu-Medrol) pulse therapy has been shown to shorten the duration of this syndrome in a randomized, controlled study.[11]

Akinetic syndrome after failure of deep brain stimulator

Deep brain stimulation involves surgical placement of a pacemaker with electrodes in specific areas of the brain. It is used to control Parkinson disease, tremor, and, less commonly, dystonia, and a number of other uses are under investigation. Continuous electrical stimulation of different nuclei in the brain has been shown to alleviate some symptoms of Parkinson disease (eg, rigidity) and to enable some patients to decrease the dose of their antiparkinson medications.

Several cases have been reported of sudden, unexpected reappearance of freezing, gait disturbance, or severe akinesia in Parkinson disease patients whose stimulators had been turned off inadvertently (eg, by a magnet in a dicating machine that was placed too close to the stimulator) and who presented to an emergency room.[12]

Adjustments to Parkison medications may need to be more gradual in some elderly patients

Treatment is easy if this diagnosis is considered. Checking the neurostimulator and switching it to "on" are all that is needed. Since patients and their caregivers are trained how to check and turn on the stimulator, the role of the geriatrician is simply to remind the caregiver of this possibility.

FDA warning. The US Food and Drug Administration has issued a warning against use of shortwave or microwave diathermy for patients with deep brain stimulation or other implanted leads (**www.fda.gov/cdrh/safety/121902.html**), stating: "There are three types of diathermy equipment used by physicians, dentists, physical therapists, chiropractors, sports therapists, and others: radio frequency (shortwave) diathermy, microwave diathermy, and ultrasound diathermy. Shortwave and microwave diathermy, in both heating and nonheating modes, can result in

serious injury or death to patients with implanted devices with leads. This kind of interaction is not expected with ultrasound diathermy. Electrocautery devices are not included in this notification."

If a patient has an implanted deep brain stimulator, magnetic resonance imaging should be done only if absolutely needed and then only if the guidelines are followed.

DISORDERS PRESENTING WITH DYSTONIA

Acute dystonic reaction

Medications are a common cause of acute focal dystonia. The symptoms, which can be life-threatening, usually occur within 24 hours after taking the medication.[12] The most common offenders are neuroleptic drugs and antiemetic drugs with dopamine-blocking activity (eg, metoclopramide), although in older patients, they are more likely to cause tardive dyskinesia and parkinsonism.[13,14]

Metoclopramide accounts for nearly one-third of all drug-induced movement disorders, and this adverse effect is a common reason for malpractice suits. The entire spectrum of drug-induced movement disorders, ranging from subtle to life-threatening, can ensue from its use; akathisia and dystonia are generally seen early in the course of metoclopramide-induced movement disorders, whereas tardive dyskinesia and parkinsonism seem to be more prevalent in long-term users.[15]

Nocturnal stridor has a poor prognosis, with an increased risk of sudden death

Treatment includes stopping the precipitating medication and reversing dystonia with anticholinergic medications such as benztropine (Cogentin). Anticholinergic therapy is given intravenously or intramuscularly followed by oral therapy for few days, as the acute dystonic reaction may recur after the effect of parenteral medication wears off.

Intravenous diphenhydramine (Benadryl), an antihistamine with additional anticholinergic effects, can abort dystonia in a few minutes.[16]

Laryngeal dystonia accompanied by multiple system atrophy

Multiple system atrophy, a Parkinson-plus syndrome, is characterized by parkinsonism (mostly with poor response to levodopa) and early onset of dysfunction of the autonomic nervous system, urinary tract, cerebellum, and corticospinal tract (hyperreflexia).[17]

In the course of the disease, about one-third of patients develop respiratory stridor due to abnormal movements of the vocal cords.[18] Nocturnal stridor portends a poor prognosis,[19] with an increased risk of sudden death. Geriatricians should be aware of these symptoms, as these patients may seek care because of hoarseness or difficulty swallowing.

Treatment. Laryngeal dystonia can be improved with continuous positive airway pressure. In some cases, tracheostomy may be needed.[19]

Sudden withdrawal of baclofen

Baclofen (Lioresal), a treatment for spasticity and dystonia, is delivered via a pump through a catheter into an intrathecal space. The pump needs to be refilled every 3 to 6 months. Sudden discontinuation of medication caused by a dislodged catheter tip or forgetting to refill the pump provokes withdrawal symptoms. Patients with this life-threatening syndrome can present with rigidity, fever, change in mental status, and worsening dystonic symptoms.

Treatment involves high doses of baclofen (up to 120 mg/day in divided doses).[6]

DISORDERS PRESENTING WITH HYPERKINETIC MOVEMENTS

Chorea, ballism (ballismus), and athetosis constitute a range of involuntary, hyperkinetic movement disorders. Chorea consists of involuntary, continuous, sudden, brief, unsustained, irregular movements that flow from one part of the body to another. Hemiballism presents as forceful flinging movements of the limbs or high-amplitude chorea that affects one side of the body.

Acute hemichorea and hemiballism

Acute hemichorea and hemiballism commonly result from infarction or hemorrhage of the basal ganglia.[20] Computed tomography and especially magnetic resonance imaging can show the lesions in patients with ballism. Stroke-induced ballism is usually self-limited and resolves after a few weeks. Acute hemiballism generally evolves to hemichorea or

hemiathetosis in a few days, which requires only protective measures.

Treatment. Mild cases do not need treatment but severe cases call for medical therapy. Antidopaminergics are the drugs of choice. A dopamine depletor such as reserpine (Serpasil) 0.1 mg once or twice daily or dopamine receptor blockers such as neuroleptics are considered.[16] The combination of a benzodiazepine plus an antipsychotic such as olanzapine (Zyprexa) has been suggested.[6]

Severe parkinsonian dyskinesia

Dyskinesia is common in Parkinson disease, and patients may present to an emergency room with severe levodopa-induced dyskinesia. Dyskinesia can be exhausting if prolonged and severe. Elevated levels of creatine kinase raise the concern of rhabdomyolysis. In rare cases, the patient develops respiratory dyskinesia when respiratory muscles such as those in the diaphragm become involved.[21]

The risk of levodopa-induced dyskinesia increases with disease severity and higher levodopa doses. Using a dopamine agonist as initial therapy delays the onset of levodopa-induced dyskinesia in early Parkinson disease. However, Factor and Molho,[21] in a case series, reported that adding dopamine agonists to the regimen was a precipitating factor; another was infection.

Treatment. A reasonable approach to treating peak-dose dyskinesia is to lower the doses of dopaminergic medications.

A mild sedative such as lorazepam, alprazolam (Xanax, Niravam), or clonazepam (Klonopin) may reduce the severity of dyskinesia.[21] These drugs are particularly useful if the dyskinesia is worse at night, and they can be used in the emergency department while waiting for the effect of the dopaminergic medications to wear off.

Amantadine ameliorates levodopa-induced peak-dose dyskinesia without worsening parkinsonian symptoms in some patients.[22]

Drug-induced myoclonus

Myoclonus is sudden, jerky, brief involuntary movement of the face, limbs, or trunk. Unlike tics, myoclonus cannot be controlled by the patient.

Myoclonus has various pathophysiologic mechanisms. Most myoclonic emergencies are epileptic myoclonic seizures, which are beyond the scope of this article. Often, myoclonus is caused by opiate overdose or withdrawal. It can also be a side effect of SSRIs, tricyclic antidepressants, lithium, amantadine, and rarely, antibiotics such as imipenem (Primaxin).[23]

Treatment. Opiate-induced myoclonus may respond to naloxone (Narcan), whereas opiate withdrawal responds to benzodiazepines.[6]

Acute akathisia

Acute akathisia occurs in susceptible patients after exposure to dopamine receptor blockers or dopamine depletors. It is characterized by subjective restless feelings accompanied by objective restless movements. The course is usually self-limited after the causative medication is discontinued.

Treatment. Symptomatic treatment may be needed in most cases for several days. Anticholinergics are effective. Additionally, vitamin B_6, mianserine, propranolol, and mirtazapine (Remeron) in a low dose (15 mg/day) have been shown to be effective[16,24,25]

DISORDERS WITH PSYCHIATRIC PRESENTATIONS

Hallucinations and psychosis in Parkinson disease

Neuropsychiatric or behavioral complications of Parkinson disease include hallucinations, dementia, depression, psychosis, and sleep disorders.[21,26] Psychosis is the leading reason for nursing home placement in advanced cases.[27] Psychosis can present as hallucinations or a paranoid delusional state in association with clear sensorium.[28] However, hallucinations accounted for only 3% of emergency admissions to the hospital for Parkinson disease patients in one series.[29]

Risk factors for hallucinations in parkinsonian patients include dementia, long-term therapy with dopaminergic drugs, long duration of disease, advanced age, anticholinergic drugs, and sleep disorders. Severe cognitive impairment or dementia is a major and independent predictive factor for visual hallucinations.[30]

Give patients their usual Parkinson drugs before and after surgery

Most hallucinations are visual; auditory, tactile, and olfactory hallucinations are rare.[30]

Treatment initially should be the same as in any patient with delirium. The systemic disorders that can aggravate or cause hallucinations such as electrolyte abnormalities, urinary or respiratory infection, and systemic illness should be ruled out.

The next step is to reduce or discontinue the adjunctive drugs that have the least antiparkinsonian effect and the greatest potential of inducing hallucination or psychosis. Examples of such medications include histamine-2 antagonists (eg, cimetidine [Tagamet], amantadine, selegeline, and anticholinergics). Selegeline can be discontinued abruptly because it has a long duration of action in the brain, but amantadine and anticholinergics should be tapered. Dopamine agonists can be discontinued. Levodopa can be reduced until the side effects begin to subside without significant worsening of motor symptoms.

If all the above adjustments fail, an antipsychotic medication can be considered.[26] Clozapine (Clozaril) has the best result and is nearly free of extrapyramidal side effects but can cause agranulocytosis, which requires frequent blood counts. The Parkinson Study Group suggested that clozapine, at daily doses of 50 mg or less, is safe and significantly improves drug-induced psychosis without worsening parkinsonism.[31] Clozapine may be impractical for elderly patients due to its side effect profile.

In elderly patients, suspect that any new symptom is medication-related

Quetiapine is a good alternative to clozapine and is less likely to worsen parkinsonian symptoms than other atypical antipsychotics.[32] Olanzapine and risperidone (Risperdal) are reported to worsen parkinsonian symptoms.[33] Not enough data have been published about the efficacy of the newer medications such as ziprasidone (Geodon) and aripiprazole (Abilify) to advocate their routine clinical use.

Rivastigmine (Exelon) was reported to improve hallucinations, sleep disturbance, and caregiver distress in addition to enhancing cognitive performance in advanced Parkinson disease in a small study.[34] Burn and colleagues[35] reported that rivastigmine was beneficial in patients with dementia associated with Parkinson disease, with or without hallucinations. Efficacy measures were cognitive scales, activities of daily living, behavioral symptoms, and executive and attentional functions. The differences in these measures between rivastigmine and placebo recipients tended to be larger in patients with visual hallucinations than in those without hallucinations. The study was not designed to assess the effect of treatment on psychosis or hallucination.

WHEN PATIENTS WITH MOVEMENT DISORDERS NEED SURGERY

Some of these syndromes can be prevented, especially in patients who are known to have movement disorders and are undergoing surgery.

One problem is stopping oral dopaminergic drugs before the operation. Parkinson disease patients on dopaminergic drugs can develop parkinsonism-hyperpyrexia syndrome or akinetic crisis if the drug is stopped suddenly. Restarting dopaminergic therapy and supportive measures are the main treatments. Patients who have Parkinson disease should receive their usual dose of levodopa, dopamine agonist, or amantadine up until the time of surgery and then again as soon as they awaken in the recovery room.[36] That goal can be achieved more easily now that these drugs come in transdermal patches and long-acting formulas.[37] Droperidol (Inapsine) and metoclopramide worsen parkinsonism and should be avoided.

Myoclonus is the most common movement disorder seen in the postoperative period. In fact, myoclonic shivering is common as patients awaken from general anesthesia.[36] The anesthetic agents etomidate (Amidate) and enflurane (Ethrane) and the opioids fentanyl (Actiq, Duralgesic, Sublimaze) and meperidine (Demerol) can cause myoclonus.[38]

Occasionally, a patient in the recovery room suddenly develops a neurologic deficit that is inconsistent with the history and physical findings. Psychogenic movement disorders should be considered in the differential diagnosis. Reassurance and occasionally psychiatric intervention are required in these cases.[36]

IN THE ELDERLY, GO EASY

Polypharmacy is a huge issue in the elderly. Some of the principles in prescribing medications in the elderly can be helpful in preventing movement disorder emergencies:

- Assess the current regimen, including over-the-counter drugs, before prescribing a new drug.
- Begin with a low dose and increase as necessary. "Start low, go slow."
- Consider the possibility that any new symptoms can be a drug side effect or due to withdrawal of a drug.
- Discuss with the patient or caregiver what kind of side effect to expect and advise him or her to report serious ones.

REFERENCES

1. **Isbister GK, Bowe SJ, Dawson A, Whyte IM.** Relative toxicity of selective serotonin reuptake inhibitors (SSRIs) in overdose. J Toxicol Clin Toxicol 2004; 42:277–285.
2. **Dunkley EJ, Isbister GK, Sibbritt D, Dawson AH, Whyte IM.** The Hunter Serotonin Toxicity Criteria: simple and accurate diagnostic decision rules for serotonin toxicity. QJM 2003; 96:635–642.
3. **Mason PJ, Morris VA, Balcezak TJ.** Serotonin syndrome. Presentation of 2 cases and review of the literature. Medicine (Baltimore) 2000; 79:201–209.
4. **LoCurto MJ.** The serotonin syndrome. Emerg Med Clin North Am 1997; 15(3):665–675.
5. **Boyer E, Shannon S.** The serotonin syndrome. N Engl J Med 2005; 352:1112–1120.
6. **Kipps CM, Fung VS, Grattan-Smith P, de Moore GM, Morris JG.** Movement disorder emergencies. Mov Disord 2005; 20:322–334.
7. **Shalev A, Munitz H.** The neuroleptic malignant syndrome: agent and host interaction. Acta Psychiatr Scand 1986; 73:337–347.
8. **Rosebush PI, Stewart TD, Gelenberg AJ.** Twenty neuroleptic rechallenges after neuroleptic malignant syndrome in 15 patients. J Clin Psychiatry 1989; 50:295–298.
9. **Adityanjee, Singh S, Singh G, Ong S.** Spectrum concept of neuroleptic malignant syndrome. Br J Psychiatry 1988; 153:107–111.
10. **Bonuccelli U, Piccini P, Corsini GU, Muratorio A.** Apomorphine in malignant syndrome due to levodopa withdrawal. Ital J Neurol Sci 1992; 13:169–170.
11. **Sato Y, Asoh T, Metoki N, et al.** Efficacy of methylprednisolone pulse therapy on neuroleptic malignant syndrome in Parkinson's disease. J Neurol Neurosurg Psychiatry 2004; 74:574–576.
12. **Hariz MI, Johansson F.** Hardware failure in parkinsonian patients with chronic subthalamic nucleus stimulation is a medical emergency. Mov Disord 2001; 16:166–168.
13. **Pollera CF, Cognetli F, Nardi M, Mozza D.** Sudden death after acute dystonic reaction to high-dose metoclopramide. Lancet 1984; 2:460–461.
14. **Bateman DN, Rawlins MD, Simpson JM.** Extrapyramidal reactions with metoclopramide. Br Med J 1985; 291:930–932.
15. **Pasricha PJ, Pehlivanov N, Sugumar A, Jankovic J.** Drug insight: from disturbed motility to disordered movement—a review of the clinical benefits and medicolegal risks of metoclopramide. Nat Clin Pract Gastroenterol Hepatol 2006; 3:138–148.
16. **Hu S, Frucht S.** Emergency treatment of movement disorders. Curr Treat Options Neurol 2007; 9:103–114.
17. **Tousi B, Schuele SU, Subramanian T.** A 46-year-old woman with rigidity and frequent falls. Cleve Clin J Med 2005; 72:57–63.
18. **Merlo IM, Occhini A, Pacchetti C, Alfonsi E.** Not paralysis, but dystonia causes stridor in multiple system atrophy. Neurology 2002; 58:649–652.
19. **Silber MH, Levine S.** Stridor and death in multiple system atrophy. Mov Disord 2000; 15:699–704.
20. **Bhatia KP, Marsden CD.** The behavioural and motor consequences of focal lesions of the basal ganglia in man. Brain 1994; 117:859–876.
21. **Factor SA, Molho ES.** Emergency department presentations of patients with Parkinson's disease. Am J Emerg Med 2000; 18:209–215.
22. **Verhagen Metman L, Del Dotto P, van den Munckhof P, Fang J, Mouradian MM, Chase TN.** Amantadine as a treatment for dyskinesia and motor fluctuations in Parkinson's disease. Neurology 1998; 50:1323–1326.
23. **Frucht S, Eidelberg D.** Imipenem-induced myoclonus. Mov Disord 1997; 12:621–622.
24. **Miodownik C, Lerner V, Statsenko N, et al.** Vitamin B6 versus mianserin and placebo in acute neuroleptic-induced akathisia: a randomized, double-blind, controlled study. Clin Neuropharmacol 2006; 29:68–72.
25. **Poyurovsky M, Pashinian A, Weizman R, et al.** Low-dose mirtazapine: a new option in the treatment of antipsychotic-induced akathisia. A randomized, double-blind, placebo- and propranolol-controlled trial. Biol Psychiatry 2006; 59:1071–1077.
26. **Tousi B, Subramanian T.** Hallucinations in Parkinson's disease: approach and management. Clin Geriatr 2004: 12:19–24.
27. **Goetz CG, Stebbins GT.** Risk factors for nursing home placement in advanced Parkinson's disease. Neurology 1993; 43:2227–2229.
28. **Factor SA, Molho ES, Podskalny GD, Brown D.** Parkinson's disease: drug-induced psychiatric states. Adv Neurol 1995; 65:115–138.
29. **Woodford H, Walker R.** Emergency hospital admissions in idiopathic Parkinson's disease. Mov Disord 2005; 20:1104–1108.
30. **Tousi B, Frankel M.** Olfactory and visual hallucinations in Parkinson's disease. Parkinsonism Relat Disord 2004; 10:253–254.
31. **The Parkinson Study Group.** Low-dose clozapine for the treatment of drug-induced psychosis in Parkinson's disease. N Engl J Med 1999; 340:757–763.
32. **Merims D, Balas M, Pertez C, Shabtai H, Giladi N.** Rater-blinded, prospective comparison: quetiapine versus clozapine for Parkinson's disease psychosis. Clin Neuropharmacol 2006; 29:331–337.
33. **Goetz CG, Blasucci LM, Leurgans S, Pappert EJ.** Olanzapine and clozapine: comparative effects on motor function in hallucinating PD patients. Neurology 2000; 55:789–794.
34. **Reading PJ, Luce AK, McKeith IG.** Rivastigmine in the treatment of parkinsonian psychosis and cognitive impairment: preliminary findings from an open trial. Mov Disord 2001; 16:1171–1174.
35. **Burn D, Emre M, McKeith I, et al.** Effects of rivastigmine in patients with and without visual hallucinations in dementia associated with Parkinson's disease. Mov Disord 2006; 21:1899–1907.
36. **Frucht SJ.** Movement disorder emergencies in the perioperative period. Neurol Clin 2004; 22:379–387.
37. **Korczyn AD, Reichmann H, Boroojerdi B, et al.** Rotigotin transdermal system for perioperative administration. J Neural Transm 2007; 114:219–221.
38. **Gordon MF.** Toxin and drug-induced myoclonus. Adv Neurol 2002; 89:49–76.

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