True Epilepsy Unmasked in a Case of Apparent Conversion Disorder

Sir: The prevalence of psychogenic nonepileptic seizures is estimated to be between 2 and 33 per 100,000 persons.[1] We describe a patient in whom a difficult case of apparent conversion but true epilepsy was unmasked by video-electroencephalogram (video-EEG) monitoring.

Case report. Ms. A, a 40-year-old white woman, presented to the neurology clinic with a 2-year history of intermittent spells of disorientation and memory loss. She described these spells as a wavelike sensation starting in her stomach and rising up into her chest, after which she would "blank out" for about a minute or so and then for the next 15 to 20 minutes would ask questions like "Where am I?" "What day is it?" "Am I married?" and "Do I have children?" During these periods, she gradually became more responsive and eventually would become increasingly responsive until the disorientation abated completely. Most of these episodes would occur in her house in the presence of her husband and occasionally in front of her children. She would have no recollection of the event. At no point during these episodes would she lose consciousness or have fecal or urinary incontinence or tongue biting. She denied seeing an aura or any knowledge of exacerbating or relieving factors. These spells could occur as frequently as 10 times in a month, but sometimes a couple of weeks would pass without any episode.

Since her first spell 2 years before the admission described in the current report, she had been seen by her primary care physician, neurologist, psychiatrist, and psychologist several times. Occasional complete neurologic examinations revealed no focal findings. Results of a computed tomography scan and magnetic resonance imaging of the head as well as an EEG showed no evidence of intracranial pathology or any epileptogenic activity. EEG testing was repeated several times over the 2-year period and revealed no seizure activity. Her general blood chemistry findings and thyroid profile had also been within normal limits.

There was no history of head trauma, febrile seizures, meningitis, or encephalitis and no family history of seizures. Substance use history was nonsignificant. Her medical history was significant for hypertension, which was under good control with amlodipine, 5 mg once daily. She had also been diagnosed with depression for the same period of time and had undergone a trial of venlafaxine, which produced minimal benefits. At the present admission, the depression was under fair control with paroxetine, 60 mg daily.

On presentation to our clinic, Ms. A was admitted for video-EEG monitoring, which recorded 1 of her spells. When correlated with the EEG findings, the video-EEG findings revealed left hemispheric slowing with an epileptiform discharge that gradually spread to the bilateral hemispheres. She was then started on lamotrigine treatment for seizure prevention, which led to significant improvement within a few weeks.

Around 10% to 20% of patients are found to have both epilepsy and psychogenic nonepileptic seizures.[2] Although video-EEG is not widely accepted by psychiatrists, it is considered to be a standard of care for making a diagnosis of psychogenic nonepileptic seizures.[2] Obtaining a definite diagnosis and separating seizure disorder from psychiatric disorder is critical, as the treatment for each is different and an early intervention for seizure disorder is essential.[3] Failure to treat a true seizure disorder can lead to devastating physical and psychological complications.

Drs. Sharma and Sorrell report no financial affiliation relevant to the subject of this letter.

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Mirtazapine Therapy for Dysgeusia in an Elderly Patient

Sir: Taste disturbances are common among the elderly and can present in up to 11% of elderly persons taking multiple medications.[1] Dysgeusia was present in 38% of 196 cases of taste complaints in one study.[2] Changes in physiology, systemic medical illnesses, infections, dental problems, and multiple medications have been shown to cause taste disturbances. Phantogeusia, illusion of taste, is a form of dysgeusia and frequently presents as metallic taste.[3]

Metallic taste is often benign and usually does not necessitate discontinuation of medications.[4] In the elderly, metallic taste can contribute to loss of appetite and weight loss. This associated weight loss may warrant evaluation and management of dysgeusia. Unintentional weight loss is common in the elderly and is a predictor for decline in activities of daily living, higher rates of institutionalization, and mortality.[5] Depression is by far the most common cause of unintentional weight loss, followed by cancer, cardiac disorders, and benign gastrointestinal diseases.

Medications that cause gastrointestinal side effects can also contribute to unintentional weight loss.[6] Several medications have been implicated in the causation of metallic taste. Gastrointestinal tract–related adverse reactions occur with all fluoroquinolone antibiotics.[7] Furthermore, gastrointestinal disorders are the most common adverse reactions observed with use of levofloxacin (5.1%).[8,9] These reactions include diarrhea, nausea, flatulence, abdominal pain, dyspepsia, taste perversion, and vomiting. Chronic medical conditions such as chronic renal failure, antihypertensive medications such as valsartan, and gout treatments such as allopurinol are known to cause taste disturbances.

We report an 84-year-old woman who developed metallic taste with use of levofloxacin prescribed for an ear infection that persisted even after discontinuation of levo-

floxacin. Metallic taste resolved after her antidepressant medication was switched from fluoxetine to mirtazapine.

Case report. Ms. A, an 84-year-old woman, was seen in a geriatric clinic with a presenting complaint of unintentional weight loss of 20 lb over a period of 1 month prior to the assessment. Her medical conditions included hypertension, recent right otitis media, chronic renal insufficiency with current creatinine clearance of 14 mL/min, history of chronic obstructive pulmonary disease, history of gout (currently stable), umbilical hernia, and depression. She had quit smoking 20 years earlier. Her medications included fluoxetine, 20 mg/day; valsartan, 80 mg/day; atenolol, 25 mg every other day; amlodipine, 10 mg/day; allopurinol, 200 mg/day; and aspirin, 325 mg/day.

To treat the otitis media, she was prescribed levofloxacin, 500 mg once a day, for 10 days starting a month earlier. She developed nausea, metallic taste, and vomiting within 2 days of starting levofloxacin. Nausea and vomiting resolved spontaneously, but metallic taste persisted over 2 weeks after the last dose of levofloxacin. Her main concern was that the food tasted like bile, causing loss of appetite. She did not have dysphagia or anosmia.

Review of her depression revealed that it was poorly controlled despite her being on 20 mg/day of fluoxetine for over 2 years. She reported off and on depression, loss of interests, insomnia, decreased energy, fatigue, and weight loss during the previous 2 years. She denied any thoughts of hopelessness, worthlessness, or death wishes. She reported enjoying her time with family and weekly visits to church. A geriatric psychiatrist was consulted for the management of depression. Fluoxetine was stopped, and mirtazapine was chosen on the basis of the constellation of depression symptoms, which included loss of appetite and insomnia. The mirtazapine dose was adjusted according to the renal clearance (a 30% decrease in mirtazapine clearance is noted in patients with creatinine clearance from 11 to 39 mL/min/1.73 m^2; see reference 10). Mirtazapine was started at 7.5 mg each night and was increased to 15 mg per night after a week.

Ms. A was seen again after a month. She reported complete resolution of metallic taste within the first week of starting mirtazapine. This change was followed by increased appetite. She gained 12 lb in the interim period. Her sleep improved considerably, and she reported feeling rested upon awakening. She reported a decrease in depressive symptoms and was continued on mirtazapine treatment.

In this case, the patient had some factors that are known to cause taste disturbances. These factors include a history of chronic renal failure and current treatment for hypertension and gout. The temporal proximity of levofloxacin treatment and onset of dysgeusia increases the likelihood that the two events are related. Taste disturbance is a known side effect of levofloxacin, but it is considered a rare side effect.[11] The metallic taste usually resolves after stopping the medicine. In this case, the metallic taste persisted even after levofloxacin was stopped. Exactly how levofloxacin causes taste disturbance is not known.

Taste disturbances can be caused by numerous methods. Drugs can affect peripheral receptors, chemosensory neural pathways, and even the brain.[12] Some drugs are excreted in the saliva, causing taste disturbances, whereas other drugs (e.g., anticholinergics and antidepressants) can cause dryness of the mouth and in turn cause taste disturbance since there is not enough saliva to carry the tastants. Other ways that drugs can cause taste disturbances include directly affecting the taste receptor cells by passing through blood,[13] altering the taste cell turnover by inhibiting protein formation,[14] inhibiting calcium channel activity,[15] causing effects on sodium channels (e.g., lithium,[16] which can also cause taste disturbance through its inhibitory effect on norepinephrine[17]), and inhibiting receptor-coupled events (e.g., dihydropyridine).[18] Drugs can also cause taste disturbances secondary to the sulfahydryl group that they contain (e.g., antithyroid drugs and acetylcholinesterase inhibitors).[19] A few drugs induce taste disturbance by causing glossitis and by inhibiting receptor turnover (e.g., metronidazole).[20]

Mirtazapine is a noradrenergic and specific serotonergic antidepressant that blocks the α_2 receptors and selectively antagonizes serotonin-2 (5-HT_2) and 5-HT_3 receptors. This selective antagonizing activity at the 5-HT_3 receptor gives it its antiemetic property. The drug is administered orally and is well absorbed in the gastrointestinal tract—it follows linear kinetics. Its half-life is 20 to 40 hours, and a steady state is achieved in 5 days. Mirtazapine is metabolized via the cytochrome P450 pathway by the liver and is excreted in the urine. Renal and hepatic impairments can diminish the clearance of the drug. The clearance of mirtazapine is reduced by approximately 30% in patients with moderate renal impairment (creatinine clearance = 11–39 mL/min/1.73 m^2) and by approximately 50% in patients with severe renal impairment (creatinine clearance less than 10 mL/min/1.73 m^2).[10]

Mirtazapine is used in the treatment of depression ranging from mild to severe. It is also known to help in treatment of anxiety and insomnia. It may be the preferred antidepressant in anorexic elderly individuals with insomnia.[21] The most common side effects of mirtazapine are dose-dependent drowsiness (54%), dry mouth (25%), increased appetite (17%), weight gain (12%), and dizziness (7%).[10] These side effects tend to diminish with time. Further systematic study is needed to evaluate the use of mirtazapine in the treatment of dysgeusia.

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