

# Dysosmia and Dysgeusia: A Patient's Nightmare and an Opportunity for Learning

A case of severe smell and taste disturbance resulting in weight loss and impaired quality of life offers an opportunity to clarify terms and understand treatment options.

By Ronald Devere, MD

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**A** patient who recently presented with the most impaired quality of life that I have seen since I established my Taste and Smell Disorders Clinic in 1996 offers an opportunity to review the challenges of diagnosing and managing parosmia and parageusia. Before beginning, it is worthwhile to define *parosmia* and *parageusia* and the related phenomena of *phantosmia* and *phantageusia*.

Parosmia is a form of dysosmia that refers to a usually very unpleasant odor triggered by any or specific environmental odor. Phantosmia is a form of dysosmia that is usually unpleasant and occurs spontaneously without a trigger. Parageusia is a form of dysgeusia that is usually unpleasant and triggered by any or specific tastes. Phantageusia is a form of dysgeusia that is usually unpleasant and occurs spontaneously without a trigger.

## Case Presentation

My patient is a 73-year-old male who about a year ago developed parosmia that could be triggered by

any odor. He said the triggered smell was like feces and would last five to 10 minutes and recur frequently.

At about the same time he developed parageusia whenever he put any food or drink in his mouth and began to chew. The taste he described was a "horrible," sour, metallic taste. Over the next three months this horrible smell and taste continued to be more frequent, and he lost 80 pounds. He required a feeding gastrostomy tube because he couldn't even sip water without getting these symptoms. All food and medications were given through his gastrostomy tube.

His past history revealed excellent health without any recent history of head trauma, sinusitis, or upper respiratory tract infection. He was treated for long-standing hypertension with lisinopril.

He was extensively tested by his family physician and otorhinolaryngologist to include nasal endoscopy; MRI of the brain, including medial temporal lobes; CT scan of sinuses; Upper GI; and standard lab work, which included thyroid func-

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tion, Vitamin B12 and folate level, sed rate, ANA, protein electrophoresis, and zinc blood level. Every one of these tests was normal or nonspecific in results. He went through a hip replacement surgery three months after his smell and taste problems, and his symptoms did not worsen. He had no appetite and lost total interest in eating. He became very depressed and didn't know how long he could live in this condition with a feeding tube and no enjoyment in life.

His general medical and neurological exam was completely normal, except that he looked emaciated and was very depressed. His mouth, tongue, gums, and palate all looked normal, and he had adequate saliva.

### **Evaluation**

I tested his smell using the University of Pennsylvania Smell Identification Test. He scored 18/40, which placed him in the moderate microsmia range. He was administered the Taste Strip Test, which evaluates sweet, sour, bitter and salt at different concentrations. He scored 4/16, which is moderately abnormal. A normal score is 9/16. It was surprising to find that none of the odors in the UPSIT smell test or the tastes in the taste strip test triggered any bad tastes or odours. During my evaluation, I gave him samples of MSG, spicy salts, and chili powder to see if he could "taste" them. He actually thought they were "tasty" without triggering any of his symptoms.

I believed he had moderate anosmia, parosmia, hypogeusia, and parageusia of undetermined cause. I decided to try to treat his parosmia and parageusia, which was potentially treatable and was the main reason for his inability to eat, depression, Peg tube, and horrible quality of life.

### **Treatment**

I put him on zinc gluconate tablets via gastrostomy tube 40mg TID to see if this would improve his parageusia as described by Heckmann, et al. in 2005.<sup>1</sup> He previously was on a short course of zinc sulphate early on in his disorder, which was not effective. Why Heckman's publication chose zinc

gluconate is not clear. To help his parosmia, I told him to put 5-10cc of normal saline in a syringe and, in the head down position, to gently drop this amount into each nostril. When he raised up, he was not to sniff so that the saline would stay in the high nasal cavity to try to block any outside odor. He was to do this four to five times per day for a week to see if it helped. I also gave him a prescription for gabapentin to try to reduce his bad smells and tastes. This treatment had been successful in six of my past cases of dysosmia and two cases of dysgeusia. He started at 300mg at bedtime and over the next four days increased the dose to three times a day by gastrostomy tube.

The patient called me one week later and he noted that his parosmia was decreasing: it was of shorter duration and less intense and not triggered by all environmental smells. His parageusia was unchanged, and he still required all feeding by tube. Three weeks later his parosmia was 90 percent gone and the parageusia was 50 percent less (less intense and shorter duration). He was now taking his pills by mouth and was able to eat vegetables, some soups, and fruit, although chicken or beef still triggered parageusia. I increased his gabapentin to 1200mg per day. He also discovered that if he ate very spicy French fries before he ate his regular food his symptoms were much less. In the last 30 days he has gained 10 pounds and no longer has a feeding tube. His depression markedly improved. He is still on gabapentin and zinc gluconate tablets. He no longer uses saline nasal drops.

### **Discussion**

This case was unusual in my experience because of the occurrence of parosmia and parageusia at the same time, both contributing to weight loss and inability to eat and requiring the need of a feeding gastrostomy tube for survival. The exact cause is unclear. Reviewing some of the literature on the subject of causes, natural history, and treatment of dysosmia and dysgeusia, there are very few "large" studies. Most are case reports and many of the treatments are anecdotal. Bonfils<sup>2</sup>

studied 56 patients with parosmia. The duration of their parosmia ranged from three months to 22 years with an average of 55 months. All patients reported olfactory dysfunction. Seventy-five percent had diminished smell, and 25 percent had total smell loss. All cases described their parosmia as foul, rotten, sewage, or burnt smell. Eighteen percent of the patients were unable to identify an odor that triggered the parosmia.

Eighty-two percent of the studied subjects were able to identify a trigger, which included gasoline (30 percent), tobacco (28 percent), coffee (28 percent), perfumes (22 percent), fruits (mainly citrus 15 percent) and chocolate (14 percent). Ninety percent of the patients had trouble identifying flavors.

The causes of parosmia in this large series were upper respiratory tract infection (43 percent), chronic paranasal sinus disease (12 percent), head trauma (10 percent), toxic chemical exposure (seven percent), nasal surgery (two percent), and idiopathic (26 percent). The temporal relationship between olfactory dysfunction and development of parosmia is not simple. In 57 percent of cases they occurred simultaneously. In the remaining 43 percent, parosmia developed after olfactory loss. This ranged from three months (34 percent) to after three months (nine percent). The mean time was 1.5 months after olfactory loss.

There are two theories regarding causes of parosmia: Peripheral and Central. In the peripheral theory, evidence suggests that abnormal olfactory neurons are unable to form a complete picture of the odorant. This goes along with the clinical feature in this study that all the parosmic patients have an intensity odor loss.

Leopold<sup>3</sup> states that the peripheral theory is supported by the histology of the olfactory organ in individual patients, which shows a decreased number of neurons, more immature than mature neurons, and distorted growth of olfactory axons.

For patients who develop immediate parosmia with olfactory loss, ephaptic transmission between disconnected axons and others that innervate the olfactory bulb might result in a distorted signal in response to an odorant.

A central theory of parosmia is still viable that states that the integrative or interpretive centers in the brain form parosmia. Leopold<sup>3</sup> stated in his paper that the support for a central theory of parosmia development is that olfactory auras can accompany seizures and that excising the olfactory epithelium in some of his patients still leaves a feeling of the “bad” smell coming, but it never occurs.

The fact that gabapentin or other antiseizure medications can improve parosmia, and that they act peripherally *and* centrally, supports both of these theories.

### **Treatment of Dysosmia**

Patients need to be reassured that their condition does not represent a progressive disorder and in time will eventually disappear. Since the majority of dysosmia patients have a smell loss, they need to be counseled about safety issues like smoke and carbon-monoxide detectors, not to eat open foods not date labeled, and have family members monitor their perfume and deodorant use.

There appears to be no particular reference about using normal saline in the nose for parosmia. Leopold<sup>3</sup> mentions this in his article and states it is effective in 50 percent of his patients. I find a similar experience. The treatment is done by taking 10cc of normal saline and putting it in each nostril in the head-down position. After 20 seconds, the person is to sit up and let the saline block the nasal upper passage where the olfactory organ resides. This is recommended to do three to four times a day. Its main purpose is for the saline to block odors from coming in contact with the olfactory organ.

The use of anticonvulsants in dysosmia is mostly anecdotal without a published series. Dr. Leopold mentions its use but does not describe any details. I have used gabapentin to treat eight patients with dysosmia including this case. Six had parosmia and two had phantosmia. There was a 90 percent improvement in five out of six parosmia patients and one with phantosmia with 900-2000mg daily in three divided doses. I only use

gabapentin in cases that do not or incompletely respond to the normal saline nasal drops. The majority of my patients received gabapentin for six months or longer, because when the dose was reduced earlier than six months, the symptoms returned. Only two of my patients are completely off gabapentin without symptom recurrence, probably due to the spontaneous recovery of their symptoms. I have tried zonisamide in one case of parosmia at 100mg/day with 75 percent improvement. None of these patients had any significant side effects from these medications. It is important that the doses of each drug be increased slowly every week to get to the appropriate dose levels mentioned above. Because of the severity of this case being reported, the decision was made to increase his dose much more quickly—in less than a week.

Leopold<sup>3</sup> described his first experience, in 1988, excising the olfactory epithelium by nasal endoscopy in intractable phantosmia. His patient recovered completely from dysosmia (phantosmia) and had some residual smell loss. He has described 18 of these procedures in 10 cases over a 13-year period. His criteria for surgery were intractable phantosmia preferably in a unilateral nostril and eliminated temporarily preoperatively with intranasal cocaine. All cases except one made a complete recovery from their phantosmia. The intent of the surgery was to cut all the olfactory axons and destroy all connections between the nasal cavity nerves and olfactory bulb.

It is not clear why he only chose phantosmia cases, not parosmia. Despite this, I was contemplating this surgery for my patient if he didn't improve, although he may have still been plagued by his severe parosmia. Follow-up smell tests in Leopold's patients over 11 years showed no change in five of 10, improvement in two of 10, and decrease in three of 10, compared to preoperative level. Histological changes as previously mentioned in his cases showed peripheral nerve damage with large fascicles lacking neurons. The big puzzle in this treatment is why olfactory function returns after cutting all the olfactory nerves.

### Treatment of Dysgeusia

You may question whether my patient really had dysgeusia. Couldn't putting food in his mouth allow the food molecules to travel retronasally to the olfactory organ and produce a very altered flavor? The patient told me that when food entered the mouth and just touched his tongue, he developed the parosmia, and his taste testing was very abnormal, leading me to believe he had primary parosmia.

In my review of dysgeusia, I couldn't find any reported large series other than by Heckmann.<sup>1</sup> In their 116 cases of dysgeusia, 50 were idiopathic and the remainder were due to allergy to dental material, poor oral and dental hygiene, poorly controlled diabetes, decreased saliva due to some medication or diseases of the salivary gland, low zinc, low thyroid and side effects from many medications.<sup>4</sup>

There are many anecdotal reports of treatment of dysgeusia suggesting improvement and worth a try. I have used these treatments in some of my patients with varied success.

1. *Cepacol Lozenges with Benzocaine*. Patients should take lozenges before mealtime. May help parosmia.

2. *Xylocaine 0.5-1.0% mouth gel*. Apply twice a day.

3. *Gabapentin (Neurontin)*. Anticonvulsant. This category of medications likely works by altering or blocking abnormal electrical discharges arising from the peripheral damaged smell or taste organ as well as altered central brain connections. Begin 300mg at bedtime and increase slowly over seven to 10 days to 900-1200mg in divided doses. I have had success in four patients when options one and two (above) have failed. I believe this was successful in the current reported case.

4. *Zonisamide (Zonegran)*. Anticonvulsant. Start at 50mg in a.m. daily and after one week increase to 100mg per day. This agent has been helpful in some of my cases of dysosmia or dysgeusia.

5. *Zinc Gluconate 140mg/day*. This intervention has been moderately effective, with improved taste, mood and dysgeusia in 50 percent of patients.<sup>1</sup>

Heckman randomized 50 patients with idiopathic dysgeusia to 140mg zinc gluconate and placebo. They rated response to a taste test and self rated the dysgeusia and reported no side effects from treatment. No significant increase in zinc was found. This is probably because zinc is a trace element and is rapidly transferred into cells. Higher doses above 140mg/day have been known to cause anemia, leukopenia and GI symptoms.<sup>7</sup> Zinc's value has been reported to help regenerate taste bud cells and influence the activity of carbonic anhydrase in saliva, which is important in breaking down foods in our mouth.

*6. Ice cube stimulation.* The patient should put one small ice cube in the mouth for one minute just before meals. Fujiyama<sup>5</sup> described an elderly patient who lost the ability to sense sweetness. Whenever she ate foods that were very sweet she developed a bad sour taste. Her taste test showed high threshold for saltiness. The author decided to put an ice cube in her mouth for one minute which lowered the oral temperature by 5 degrees. They retested her taste capabilities and her saltiness recognition improved. She was told to place an ice cube in her mouth before every meal. After a month the patient reported to her physicians that she could recognize sweet again and lowered her threshold for all other tastants.

There has been evidence in the literature that the gustatory nerve fibres are sensitive to temperature changes by thermosensitive ion channels. A thermosensitive channel called TRPM5 is present in taste bud cells and can confer a steep temperature dependence on the processing of taste perception. Therefore, the authors say, the demonstrated recovery in this patient of her taste sensitivity may be caused by interaction between taste and cold signals. The patient reported complete recovery of her sweet taste after the cold treatment and previous sour taste (dysgeusia) was eliminated. The authors speculate that cold treatment may improve circulation in the tongue and that subsequently taste sensitivity recovers. More studies are needed, however, this intervention should be tried in some of our patients.

*6. Artificial saliva.* If there is insufficient saliva, try artificial saliva before each meal.

*7. Mirtazapine 15mg at bedtime.* Kalpana, et al.<sup>6</sup> reported a case of an elderly woman who developed otitis media. She was given antibiotic levofloxacin 500mg a day. After 10 days she developed a spontaneous metallic taste. Her food tasted like bile, causing loss of appetite and weight loss. This dysgeusia continued three weeks after her antibiotic was stopped. She had no smell problems but smell was not tested. She had a long history of depression and was on fluoxetine for many years. A psychiatrist saw her and changed fluoxetine to Mirtazapine. Patient reported complete resolution of her dysgeusia in four to five days after starting Mirtazapine. Mirtazapine is a noradrenergic and specific serotonergic antidepressant. How and why it worked in this case is not clear. More studies need to be done.

Most of the treatments mentioned for dysosmia and dysgeusia have not been scientifically studied to show their benefits. However the symptoms and impaired quality of life these disorders produce in our patients should prompt us to try these treatments singly or in combination. The majority are very safe, and patients, like mine, are very grateful. ■

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