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# Expert Opinions

## Phantosmia and Migraine With and Without Headache

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**Phantosmia is a rare migraine aura. We present two cases of phantosmias occurring before migraine headaches and also without headaches. To our knowledge, these are the third and fourth cases of phantosmias ever reported due to migraine aura without headache.**

**Key words: phantosmia, olfactory hallucination, migraine, headache**

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### CASE HISTORIES

**Case 1.—**This is a 53-year-old female with a 9-month history of smelling a dirty dog smell as often as 2–3 times a day or up to 4 days without the smell with an average duration of 5 minutes (range 30 seconds to 1 hour) followed by a headache about every 2 weeks but most of the time without an associated headache. She described a bifrontal throbbing headache with an intensity of 6/10 associated with nausea, light and noise sensitivity but no vomiting. She always takes ibuprofen with relief in 1 hour. During the episodes, there is no alteration of consciousness. She has no triggers. She denied depression, anxiety, or increased stress.

Prior to 9 months ago, she had occasional headaches since her 20s described as bitemporal aching with an intensity of 7/10 associated with light and noise sensitivity but no nausea or aura. She would take acetaminophen with relief in 1–2 hours. Stress was a trigger.

She saw a cardiologist about 1 month after onset and had a CT of the brain without contrast on 1/19/15 that was normal. She saw two ENT physicians who found normal exams.

Past medical history of hyperlipidemia on pravastatin. Family history: sister has migraine. Neurological examination was normal.

MRI of the brain with and without contrast was normal. EEG was normal. A complete blood count, chemistry profile, thyroid functions, erythrocyte sedimentation rate, antinuclear antibody, rheumatoid arthritis factor, and Sjogren's antibodies were negative.

She declined a trial of migraine preventive medication.

**Case 2.—**This is a 69-year-old female with a history of headaches since age 35 that have been occurring about 6 days per month the prior 12 weeks since starting onabotulinum toxin A 2 years previously and before about 4–6 days per week since age 45–50 years. She described a top of the head and/or then left or right sided throbbing with an intensity of 7–10/10 associated with nausea, light and noise sensitivity but no vomiting or visual,

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sensory, or language aura. She took naratriptan with relief in about 3–4 hours. Change of weather was a trigger.

For the past 20 years, once every 1–2 weeks, she would smell something burning like a campfire 90% of the time and like burning rubber 10% of the time lasting 15 minutes to 2 hours. Three times, first about 18 years ago and last about 1 month ago, she had a menthol smell lasting about 1 hour to 2 days. The smell would precede a typical headache 1 hour to a few hours later. She also reported occasional episodes of the abnormal smell occurring without a headache. Since starting onabotulinum toxin A injections, the frequency of olfactory hallucinations decreased to every few months. When she went 10 months between injections, the hallucinations increased after 5 months to about twice a month.

Propranolol, venlafaxine, divalproex, and verapamil were not effective for prevention. MRI of the brain in 2015 showed non-specific white matter abnormalities.

Past medical history of hyperlidemia and fibromyalgia. Recent nuclear cardiac stress test negative. Neurological examination was normal.

Questions. What is the diagnosis? How common is this disorder in adults and children? What might be the mechanism? How do you distinguish phantosmias due to migraine from other disorders? What is the treatment?

*What Is the Diagnosis?*.—Case 1 presents with a phantosmia of a dirty dog lasting 30 seconds to 1 hour with an average of 5 minutes sometimes followed by a migraine headache and often occurring without a headache. MRI of the brain and EEG were normal.

Case 2 presents with a 34 year history of migraine without aura and a 20 year history of a burning smell lasting 15–120 minutes and rarely a menthol smell before a migraine headache. She also reported occasional episodes of the burning smell occurring without a headache. MRI of the brain showed only non-specific white matter abnormalities. The frequency of the episodes of phantosmias significantly decreased after treatment of chronic migraine with onabotulinum toxin A injections.

Alterations in the sense of smell can be broadly classified into quantitative dysfunction (hyperosmia, hyposmia, and anosmia) and qualitative dysfunction (parosmia and phantosmia).[1,2] A distortion of the perceived odor is termed parosmia or troposmia, while the perception of an odor when there is no odorant stimulus present in the environment is termed phantosmia, cacosmia, or olfactory hallucination.[3,4] Phantosmia typically lasts longer than a few seconds, while olfactory hallucination usually lasts only a few seconds.[4,5]

The occurrence of abnormal perception of odor has long been described in ancient times as early as 131 A.D. by Aretaeus, a Cappodocian: "a heavy smell sometimes preceded the accession of a paroxysm."[6] In migraine, the majority of patients have normal olfactory function.[7] During a migraine episode, olfactory acuity may be impaired with a minority exhibiting microsmia or hyposmia during acute attacks and decreased olfactory sensitivity.[7,8] However, a heightened sense of smell/olfactory hypersensitivity has also been described during both a migraine attack and between attacks in up to 46 and 35%, respectively.[9–11] These patients tend to have greater frequency of migraines, odor-induced migraines and visual hypersensitivity. About 50% of migraineurs report that odors can also trigger their migraine attacks and olfactory hallucinations sometimes develop during migraine.[9,12–15] There is suggestion of the role of the piriform cortex and antero-superior temporal gyrus in olfactory hypersensitivity in migraine, as well as dysfunction in central olfactory processing.[8,16,17]

The description of phantosmia in both cases meets the criteria of aura, 5–60 minutes, followed by headache and is consistent with similar case series and not consistent with epilepsy as discussed below.[18] Case 2 did have some episodes of phantosmia lasting more than 1 hour which is not uncommon for various migraine auras and phantosmia (discussed below).[19] Interestingly, the International Classification of Headache Disorders (ICHD) does not yet recognize the presence of olfactory aura as a migrainous aura.[20]

*How Common Is This Disorder in Adults and Children?*.—Phantosmia has been found to occur in 6% of community dwelling adults and up to 12% in a tertiary otolaryngology clinic.[21,22] The majority had concomitant anosmia/hyposmia, and most phantosmias presented with no history of upper respiratory tract infection, head trauma or aging (idiopathic).[1,4,22] A typical history begins in a woman between the ages of 15–30 years old, who notices odor perception that is not appreciated by others. Odors last 5–20 minutes each time and resolve spontaneously. Recurrent episodes may occur more frequently and may last longer subsequently. They may be perceived arising from one or both nostrils, and resolve with sleep, Valsalva maneuver, or occlusion or instrumentation of the nostril.[4]

The first case of olfactory hallucination in migraine was described in 1982.[12] The late Oliver Sacks also reported seeing several migraine patients with hallucinations of smell associated with forced reminiscence and feelings of déjà vu.[23] Since then, multiple case reports have highlighted this phenomenon with migraines.[13,20,24–27] Estimated prevalence ranged from 0.1% in consecutive migraine patients to 1% in a sample of 200 patients with vascular headache attending a neurologic institute and up to 10.9% in a headache clinic among consecutive female migraineurs, though high prevalence may be confounded by depression.[14,28,29] More recently, case series from a single headache center found a prevalence of 0.66% (14/2110 patients) in primary headache disorders, including migraine (84.6%), cluster headache (7.7%), new daily persistent headache (2.6%), hemicrania continua (2.6%), and chronic daily headache (2.6%).[30] In pediatric primary headaches, 2.5% (21/839 patients) reported experiencing phantosmias, all of whom had migraine.[31] Among migraineurs in children, 3.9% had phantosmia.

Olfactory hallucinations have also been described in patients with schizophrenia, schizoaffective disorder, and bipolar disorder type 1, with an estimated prevalence of 17.3–20, 22.8, and 8.1%, respectively.[32,33] This varies across countries from 3 to 27%.[34] Deviant olfactory experiences in an initially nonpsychotic group of college students was interestingly found to predict the development of clinical psychosis at a 10-year reevaluation.[33] Olfactory hallucination has also been described in depression (19–33%) and in up to 10% of nondemented Parkinson's disease patients, though prevalence is less (5%) in those without accompanying visual and auditory hallucination.[35,36] In temporal lobe epilepsy, estimated prevalence ranges from 0.6 to 30%.[35,37–40] A higher prevalence has been reported in those with head injury and those with decreased smell and taste acuity in up to 40–60% of patients.[35]

*What Might Be the Mechanism?*.—The mechanism behind phantosmia has not been clearly elucidated. Theories of abnormal peripheral as well as central mechanisms have been postulated.[2,4,5]

Proponents of the peripheral theory highlight phantosmia being worse in the nostril with the poorer olfactory ability, the fact that phantosmia can be eliminated by occluding air flow and anesthetizing the olfactory mucosa in the affected nostril. Histopathological studies also demonstrated decreased number of neurons, greater ratio of immature to mature neurons and disordered growth of olfactory axons.[41,42] A combination of peripheral hypersensitivity, endogenous loss of inhibitory GABAergic neurons, and possibly estrogen may play a role in the pathogenesis of phantosmia.[2,35] Ultimately, abnormal signals arise from primary olfactory neurons, which then trigger a central process.[4,41]

Arguments for a central cause include a hyperfunctioning central area which could generate odor perception.[4,41] Olfactory aura that sometimes accompany seizures in epilepsies suggests a central origin as well. These seizures typically arise from the mesial temporal lobe, particularly the uncal region with the entorhinal cortex, and possibly the amygdala and olfactory bulb if unpleasant, and from the insular cortex if pleasant.[40,43] The persistence of symptoms despite excision of olfactory epithelium – similar to a "phantom limb" phenomenon – as well as functional imaging studies which demonstrated increased activity in the contralateral frontal, insular, and temporal region, which subsequently decreased after excision of the olfactory epithelium, suggests involvement of central processes in phantosmia.[4] Phantosmia is also

**Table 1.—Comparison of Features of Phantosmias in Different Syndromes (Adapted From Ref. 30)**

| | Duration | Identifiability | Source | Insight | Quality of Phantosmia |
|---|---|---|---|---|---|
| Migraine | 5–60 min | Usually precise | Extrinsic | + | Unpleasant > pleasant |
| Epilepsy | Seconds to minutes | Usually vague | Extrinsic | + | Unpleasant > pleasant |
| Depression | Continuous | Variable | Intrinsic | − | Unpleasant |
| Schizophrenia | Variable | Variable | Extrinsic | − | Unpleasant |

associated with severe depression, with speculation of abnormal functionality in the orbitofrontal cortex affecting olfactory processing, reduced grey-matter volume in anterior insular, anterior cingulate cortex, hippocampus, and left orbitofrontal cortex.[44]

Phantosmia may also be accompanied by primary headache disorders similar to the typical aura described in migraine with aura. In a case series and literature review of olfactory hallucinations in primary headache disorders, the authors described the majority (64%) having olfactory hallucinations lasting 5–60 minutes that occurred before, during and within an hour of headache onset.[30] A third of patients had another aura fulfilling criteria for migraine with typical aura. Olfactory aura may occur simultaneously or follow the visual aura prior to or during the headache phase. Similarly, in Mainardi's case series of 11 patients with olfactory aura, the majority of auras lasted less than 10 minutes (range 3 minutes to 24 hours) and occurred before or with the onset of headache.[45] Hence the authors proposed that it be classified as a distinct form of migraine aura as it fulfills most criteria of an aura. If so, the underlying electrophysiological substrate of the aura of migraine is the phenomenon of cortical spreading depression which propagates through the deep temporal structures and orbitofrontal cortex and generates the olfactory symptoms as it spreads. This was also proposed by Morrison, who described similar symptoms with transient mood symptoms during migraine attacks.[29] As odors tend to be recognizable, the authors suggest possible involvement of the piriform cortex, an olfactory association area.[30] At the same time, there have been reports showing sites of interaction between the olfactory and the trigeminal systems.[46] The overlap of the neural circuits of the olfactory system and trigeminovascular system involved in the pathogenesis of migraine suggests migraine and trigeminal activation as one of the causes of olfactory hallucination.[31,47] Those with aura and migraine usually exhibited both normal EEGs and CT brain scans;[48] however, migraine patients can also exhibit paroxysmal EEG and other neuronal abnormalities suggesting an underlying neuronal component in the generation of these symptoms.[49–55] There may also be genetic influences at play with aura susceptibility genes in migraine, predisposing some patients to having aura and some without.[56] Another proposed mechanism includes increased dopaminergic periglomerular cells in olfactory bulbs as dopamine may have a role in premonitory symptoms in migraines.[57,58]

*How Do You Distinguish Olfactory Hallucinations Due to Migraine From Hallucinations Due to Other Disorders?*.—Olfactory hallucinations have been described in psychotic disorders, depression, olfactory reference syndrome, epilepsy (temporal lobe, uncinate, or orbitofrontal seizures), dementia, Parkinson's disease, drugs and drug withdrawal states (including alcohol), migraines, aneurysms, and arterial-venous malformations, tumors, and head injury.[2,4,33,57,59–71] Olfactory hallucination has also been reported without a known cause, or with a remote history of sino-nasal disease.[35] Features that distinguish olfactory hallucinations due to migraine from hallucinations due to other disorders are summarized in Table 1.

In psychotic disorders like schizophrenia, olfactory hallucinations are rarely the dominant

symptoms of the illness, and usually pales in comparison to other forms of visual, auditory, or somatic hallucination and front rank symptoms of schizophrenia.[3,34,72] Hallucinations are typically perceived as extrinsic in nature, ie, arising externally and being caused or forced upon them by another person or agency.[3,65] The perceived odors are odd in content, such as smells of holiness or of space aliens, and usually do not result in patients attempting to remove the odor, though they may complain to the police.[72] Patients often retain insight into their symptoms.[3] This is in contrast to olfactory reference syndrome (ORS), a non-psychotic disorder characterized by perceived dominant foul odors arising from himself (intrinsic) and resulting in overwhelming contrite response and attempts to rid themselves of the odor with excessive washing of hands, change of clothes, and resultant avoidance of other people.[72] There may be associated secondary low mood, though milder than in those with depression. ORS may also be associated with depression, personality disorders, schizophrenia, hypochondriasis, alcohol and drug abuse, obsessive compulsive disorder, body-dysmorphic disorder, brain damage, and dementia.[3,73] Patients with ORS tend to be young males, with referential ideas, and most do improve with neuroleptics, antidepressants, and psychotherapy.[73] In depression with olfactory hallucinations, there usually is associated retardation, depressed mood, and morbid thoughts.[72] There is a correlation between phantosmia and degree of depression.[44] Unlike those with depression, ORS patients tend to be more self-critical.[3] Hence, origin of the hallucination, whether being extrinsic or intrinsic, its dominance with respect to other psychiatric symptoms, associated depression or psychotic symptoms as well as the patient's reaction to the hallucination may help to distinguish between the different psychiatric disorders.[3]

In primary headache disorders, the olfactory aura is usually temporally related to the headache phase, with the headaches meeting criteria for migraine.[30] However, a recent Italian case series of 11 patients reported 2 patients who had olfactory aura without the headache phase at times, presenting as an isolated symptom akin to visual aura without migraine.[45] Hallucinations tend to be perceived as extrinsic, highly specific in nature with odors being identifiable. Typically an unpleasant burning smell is noted by the patient.[26,30,45] Other smells that have been described by patients include certain food smells, decomposition, and chemical smells. Some may have more than 1 smell type or smells that evolved over time. Phantosmias can have sudden or gradual onset, last minutes to hours, and tend to respond to migraine prophylaxis medications including non-anticonvulsants in addition to anticonvulsants.[27,30,31,45] Some patients had concomitant typical visual and sensory auras occurring simultaneously.[31] Imaging and EEG were negative in these patients.[30,45] Phantosmia also occurred about a decade later than the mean onset of headache, 32–34 years compared to 18–21 years of age.[30,45]

In epilepsy, olfactory hallucinations tend to be crude and unrecognizable, unpleasant, and lasting seconds to minutes instead.[40,43,74,75] Olfactory aura may also be combined with other auras, in particular sensation of epigastric rising, nausea and fear. They are linked temporally with epileptic symptoms, and are seldom in isolation.[3,67] The majority have a structural lesion in the mesial temporal region.[40]

In Parkinson's disease patients, phantosmias were generally rare and infrequent (1–2 times/month), lasting seconds to minutes and not frightening to the patient as they have good insight. Most had associated hyposmia.[36]

Lastly, there is a group of patients with olfactory hallucination without clinical motor activity and no clear cause found.[1,4,35] The phantosmia tends to be spontaneous, worse after coughing, laughing, and shouting. They typically last 5 seconds to 20 minutes, are unpleasant (putrid/chemical) in nature and episodes increase in intensity, duration, and frequency over time. Valsalva maneuvers, naps/sleep as short as 20 minutes, nasal plugging, and topical tetrahydrocannabinol (THC) transiently relieves symptoms.

*What Is the Treatment?*.—The presence of olfactory dysfunction is not without significant impact to the patient. A large study involving 750 Parkinson's disease patients with olfactory dysfunction showed

a high proportion reporting altered quality of life (68%), changes in appetite or body weight (46%), and adverse influences on daily living or psychological well-being (56%).[76] In the United Kingdom, British sufferers of olfactory disorders suffer significant physical, social, psychological, and emotional impacts with higher rates of weight gain, isolation, depression, stress, and resignation to their disability.[77]

Treatment ranges from reassurance with no active therapy and watchful waiting, to topical and systemic medications, anesthesia to parts of the nose and rarely surgical excision of olfactory neurons.[4,5] Unfortunately, mixed outcomes in treatment have been reported, mostly in case reports and small series of patients, highlighting lack of good evidence on treatment options in this condition.

Analgesics were not found to be helpful for phantosmia in children, though sleep helped to minimize or terminate it.[31] Administration of topical nasal saline drops as needed in the head down and forward (Mecca) position, oxymetazoline HCl, and topical cocaine HCl blocks air flow and anesthetizes the olfactory cleft with improvement in symptoms.[1,4] However, olfactory cocainization was shown to be an ineffective long-term solution for phantosmia with resolution and improvement being transient and unsustained and retreatment necessary in 2 weeks to 4 months.[78]

In primary headache disorders, greater than 75% responded with initiation of prophylaxis therapies for migraine.[30,45] Medications used included topiramate, nortriptyline, amitriptyline, flunarizine, propranolol, lamotrigine, verapamil, Petasites hybridus root extract and magnesium oxide. Literature review of cases reported improvements with indomethacin, gabapentin, phenytoin, sodium valproate, oral alpha-lipoic acid and supraorbital and occipital neurostimulation.[5,27,30,79] Surgical therapies tried included bifrontal craniotomy to remove olfactory bulbs and endoscopic trans/intranasal procedure to excise the olfactory epithelium.[4,5] The majority of patients with olfactory hallucinations with no clear cause found improvement over time without specific treatments.[22] Case 2 had a significant reduction in the frequency of episodes of phantosmias with onabotulinum toxin A injections.

## CONCLUSION

Phantosmia is not an uncommon presentation of a neurological, psychiatry, or local ear, nose and throat (ENT) cause. Though not commonly appreciated and highlighted, they cause considerable impact on sufferers. These patient vignettes highlight phantosmia as a rare aura in migraine and, to our knowledge, the third and fourth cases of phantosmia ever reported occurring without headache. It needs to be emphasized that while symptoms fulfill criteria for migraine and that there are proponents of incorporating olfactory aura as a migraine aura in the ICHD criteria, due diligence must be taken to ensure that secondary causes are excluded, especially on initial presentation of onset in this age group. Patients who present with olfactory hallucinations need careful neurological and mental state examination especially to exclude partial seizures when the olfactory symptoms last seconds to minutes. Treatment options remain anecdotal, though prophylaxis for background migraine seems to be a good way to move forward if patients have frequent or bothersome symptoms.

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