Headache-Associated Phantosmia as a Harbinger of Lewy Body Dementia

Tara C. Carlisle, M.D., Ph.D., Marius Birlea, M.D., Diego Restrepo, Ph.D., Christopher M. Filley, M.D.

Olfactory hallucinations (OHs), called phantosmias, can occur in many neurological, psychiatric, and medical conditions, but no widely used standardized approach exists to comprehensively assess qualitative olfactory dysfunction in the clinical setting. In addition, medical professionals and patients and their family members may not recognize phantosmia as a potential neurological problem. Given the many possible etiologies for symptomatic phantosmia, it is important to recognize this unusual condition and elicit a meaningful history to explore its potential underlying cause. We describe a 77-year-old man with a 2-year history of headaches accompanied by smelling a foul odor, and we then discuss the differential diagnosis for new-onset, persistent phantosmia. The patient in this unusual case ultimately manifested features consistent with Lewy body dementia (LBD), highlighting the varied clinical presentations that are possible with this neurodegenerative disorder. We discuss the possible pathophysiology of phantosmia in Lewy body disorders, including a proposed mechanism for OHs arising prior to the typical well-formed hallucinations in LBD.

CASE REPORT

A 77-year-old left-handed man with the factor V Leiden mutation, hyperlipidemia, and sensorineural hearing loss presented to a headache clinic with dull and vise-like daily headaches that were typically followed by smelling a foul odor within the hour. The headache was predominantly located posteriorly, with intermittent frontal involvement and extension above both ears. He was often awakened by the headache, which would then lessen during daytime. During an episode, he would appear distraught but not confused.

He reported headache episodes without olfactory symptoms starting 2 years ago, and onset of accompanying smell in the prior 14 months. He was initially managed by an otolaryngologist for abnormal smell sensation. He also reported recurrent falls starting about 5 years prior. His initial fall was with head strike and loss of consciousness, for which he did not seek medical care, but his ambulation had improved over the ensuing 18 months and he did not experience additional falls.

His evaluation prompted concern for phantosmia, likely representing the aura of a primary headache disorder versus epilepsy. Posttraumatic headache was also considered, although the temporal correlation of his falls to his initial headaches was not consistent given that headache onset occurred years after his fall with traumatic brain injury (TBI). Lamotrigine was initiated as a preventative treatment for headache and for the potentially underlying epilepsy. The plan of care included admission to the epilepsy monitoring unit (EMU) for spell capture and, after his family reported cognitive changes, referral to the behavioral neurology clinic (BNC).

In the BNC, the patient reported worsening short-term memory, difficulty with name recall, slowed responses, and vision changes. His family noticed additional word-finding problems, worsening handwriting and spelling, and misplacement of items. These changes were present for at least 3 years, resulting in the inability to manage his business, but had worsened significantly over the last 6 months. Multiple mistakes were made in personal finances, including inappropriately sharing sensitive information. His family expressed increasing concerns about his ability to drive safely, noting out-of-character incidents of aggressive driving and attempts to take the wheel as a passenger. The patient recently misidentified a stranger as a close relative and misinterpreted his hotel as a clinic.

Notably, behavior changes were observed prior to his falls, headaches, and cognitive changes. New marital discord started about 6 years before his evaluation, driven by his suspicions that his wife was unfaithful. He later became increasingly concerned about strangers trespassing on his property, leading to him regularly carrying a firearm.

Current medications included those for vascular risk factors and lamotrigine (75 mg, twice daily). He was a remote former smoker, but he had no recent alcohol use and no prior illicit drug use. Family history was unremarkable. His neurologic examination disclosed mild parkinsonism, including bradykinesia with decrement and a slowed, stooped

walk with reduced arm swing, but without tremor. Cognitive evaluation with the Montreal Cognitive Assessment yielded a score of 14/30 (higher scores indicate better performance), with the most profound deficits in visuospatial/executive function, language, abstraction, delayed recall, and orientation.

Serum tests for reversible causes of dementia (e.g., thyroid stimulating hormone, rapid plasma reagin, vitamin B_{12}) were unremarkable. Brain magnetic resonance imaging (MRI) performed a year prior showed old left lentiform nucleus and external capsule lacunes, prominent vascular spaces in the basal ganglia, and atrophy with predominance in the posterior regions. A routine electroencephalogram disclosed no epileptiform activity or other abnormalities. His EMU stay revealed two events of mild phantosmia without electrographic correlates.

The patient met clinical criteria for dementia given his progressive cognitive decline that interfered with daily activities. The additional clinical history and examination were most concerning for LBD and, more specifically, dementia with Lewy bodies (DLB). Despite multiple vascular risk factors, his imaging was not consistent with vascular dementia. The differential diagnosis also included cognitive impairment due to ongoing subclinical seizures, because an unrevealing EMU stay does not conclusively rule out epilepsy. Other neurodegenerative disorders were considered, but the patient did not clearly meet the clinical criteria for behavioral variant frontotemporal dementia, despite the early marital discord (1), and there were no other features to suggest other Parkinson-plus syndromes such as supranuclear palsy (2) or corticobasal syndrome (3). He initially met criteria for probable Alzheimer's disease, except for the exclusionary criterion of DLB core features (4).

The DLB clinical criteria require dementia (5), the initial deficits of which most notably manifest in altered executive function, visuospatial abilities, attention/processing speed, and (in some cases) language and memory (6). Core clinical features include prominent cognitive fluctuations, well-formed visual hallucinations (VHs), rapid eye movement sleep behavior disorder, and parkinsonism (Table 1) (5). DLB is distinguished from Parkinson's disease with dementia (PDD) by the "1-year rule": a diagnosis of PDD must include a clear Parkinson's disease diagnosis for at least 1 year before the onset of dementia (5). Notably, LBD is an umbrella term that includes both DLB and PDD. An unusual feature of the patient described in this case is the appearance of early paranoid delusions about infidelity, because delusions typically arise later in the course of LBD (6).

The most novel feature of the patient described in this case is that the inaugural and most prominent hallucination was olfactory. One year after he was first seen in the BNC, he described a well-formed VH of "gentlemen" sharing his room whom he could physically guide with touch, consistent with tactile hallucinations previously described in DLB (7). Given his dementia, cognitive fluctuations, VHs, and parkinsonism, he met the clinical criteria for probable DLB without biomarkers (Table 1) (5). Over time, the patient's Lewy Body Composite Risk Score (LBCRS; range 0–10) (8) was five (Table 2). An LBCRS of three or more indicates probable DLB and assists in distinguishing DLB from Alzheimer's disease with a sensitivity of 94.2% and a specificity of 78.2%, and from any other dementia with a sensitivity of 97.9% and a specificity of 86.1% (8). His TBI was thought to be unrelated to his later diagnosis of DLB, because the current literature does not support a strong association (9–12), but his falls likely represented unrecognized parkinsonism during his early disease course.

TABLE 1. Diagnostic criteria for dementia with Lewy bodies

Level of certainty	Criteria
Possible	• One core clinical feature[a] with no indicative biomarker[b]
	• No core clinical features[a] with one or more indicative biomarkers[b]
Probable	• Two or more core clinical features[a] with or without indicative biomarkers[b]
	• One core clinical feature[a] with one or more indicative biomarkers[b]

[a] Core clinical features include prominent cognitive fluctuations, well-formed visual hallucinations, rapid eye movement sleep behavior disorder, and parkinsonism.

[b] Indicative biomarkers include reduced dopamine transporter uptake in basal ganglia by single-photon emission computed tomography or positron emission tomography scans, low cardiac uptake by ^{123}iodine-meta-iodobenzylguanidine myocardial scintigraphy, and confirmation of rapid eye movement sleep without atonia by polysomnography.

His headaches were initially prominent despite uptitration of lamotrigine but became less frequent and then ceased over months, at which time lamotrigine was titrated off without headache recurrence. His phantosmia remained persistent. He also developed features consistent with olfactory reference syndrome (ORS), which is categorized under other specific obsessive-compulsive and related disorders (13); he would wake up perceiving a foul odor emanating from his own body that prompted nightly showers. ORS is rare, generally has an early age of onset (average=21 years), and is often associated with other comorbid psychiatric diagnoses (14). ORS was described previously in Parkinson's disease (15), but the prevalence of ORS in LBD is unknown.

DISCUSSION

Olfaction requires a functional olfactory neuroepithelium, olfactory bulb, and olfactory nerve (16). The primary olfactory regions (i.e., anterior olfactory nucleus, tenia tecta, olfactory tubercle, entorhinal cortex, piriform cortex, and amygdala) and secondary olfactory regions (i.e., thalamus, hypothalamus, and orbitofrontal cortex), with additional connections to the hippocampus and insular cortex, are all required for the emotional response to and the perception, identification, discrimination, and modality-specific memory consolidation of olfactory inputs (16). Olfactory function can be localized to the left or right cerebral hemisphere on

TABLE 2. Lewy Body Composite Risk Score[a]

Does the patient…	Yes	No
Have slowness in initiating and maintaining movement or have frequent hesitations or pauses during movement?	X	
Have rigidity (with or without cogwheeling) on passive range of motion in any of the four extremities?	X	
Have a loss of postural stability (balance) with or without frequent falls?	X	
Have a tremor at rest in any of the four extremities or head?		X
Have excessive daytime sleepiness and/or seem drowsy and lethargic when awake?		X
Have episodes of illogical thinking or incoherent, random thoughts?	X	
Have frequent staring spells or periods of blank looks?		X
Have visual hallucinations (see things not really there)?	X	
Appear to act out his/her dreams (kick, punch, thrash, shout or scream)?		X
Have orthostatic hypotension or other signs of autonomic insufficiency?		X

[a] The Lewy Body Composite Risk Score is composed of 10 dichotomous history and examination features (present at least three times over the prior 6 months) that can easily be assessed in the clinical setting. Scores range from 0 to 10, with a score of three or greater indicating probable dementia with Lewy bodies. Reprinted with permission. Copyright 2015. Lewy Body Composite Risk Score: Rapid Method to Improve the Clinical Detection of Lewy Body Dementia is a copyrighted instrument of James E. Galvin. All rights reserved.

the basis of lesional and functional brain imaging studies, but some authors have hypothesized a dominant role for the right temporal lobe in light of patients with epilepsy who underwent unilateral temporal lobectomy (16).

OHs are a type of qualitative olfactory dysfunction and involve a perception of a smell in the absence of an odor stimulant (16). This phenomenon, also known as phantosmia, contrasts with parosmia (also called troposmia), another qualitative olfactory dysfunction that refers to a distorted smell perception in the presence of an odor stimulant (16, 17). Hyposmia (reduced smell) and anosmia (lost smell) are quantitative olfactory dysfunctions (16).

Many nonneurological and neurological conditions are associated with qualitative or quantitative olfactory dysfunction; however, less is known about qualitative olfactory dysfunction (16, 18). Phantosmia is associated with a wide range of conditions; therefore, the differential diagnosis for new-onset phantosmia is broad (Table 3) (19–25). Phantosmia has been described in primary headache disorders (26–28), focal epilepsy (16, 29), multiple sclerosis (30), Parkinson's disease (16, 19, 31), and following TBI (19, 32). Phantosmia has also been described in multiple nonneurologic medical conditions, including psychiatric disorders (33–35), post upper respiratory tract infections (19), sinonasal disease (19, 36, 37), and gastroesophageal reflux disease (38). Phantosmia is also correlated with some prescription medications (38). Idiopathic phantosmia is common but does not clearly predict the development of serious health conditions or neurodegenerative disorders when assessed longitudinally (39).

For quantitative olfactory dysfunction (i.e., hyposmia and anosmia), standardized clinical assessments for smell identification can be made, but no standardized approach exists for the clinical assessment of qualitative olfactory dysfunction. A detailed clinical interview performed by an experienced clinician is the most frequent approach (17), but standardized questionnaires have been proposed (32, 37, 38). Given the absence of standardized assessments, phantosmia is predicted to be underassessed and underidentified, despite an estimated prevalence of 4.9% (40) to 6.5% (32) in older adults. However, only a small portion (11.1%) of individuals with phantosmia discuss alterations in smell with a medical provider (32). Furthermore, the current literature regarding the duration, quality (pleasant, unpleasant, neutral), and other features (e.g., vague vs. precise; intrinsic vs. extrinsic) of the perceived odors associated with qualitative olfactory dysfunction and possible specific associations with the various etiologies is limited to case reports and case series (26, 31, 34, 41). Phantosmia is associated with parosmia (40), female sex (32, 40), younger age (32), low socioeconomic status (32), vascular risk factors (40), fair or poor health status (32), and brain-derived neurotrophic factor allele status (40). Given the association of phantosmia with COVID-19 infections (42), phantosmia prevalence is likely to increase.

There are multiple proposed mechanisms for phantosmia that depend on the clinical context (Table 3) (26, 29), including both peripheral and central etiologies (43). Regarding phantosmia in synucleinopathies, olfactory bulb alpha-synuclein has high specificity and sensitivity for multiple Lewy body disorders, including LBD and PD (44), but there is actually increased dopaminergic signaling within the olfactory bulb and anterior olfactory nucleus in Parkinson's disease (31). Thus, it has been proposed that hyperexcitable dopaminergic activity in the setting of olfactory pathway denervation may be responsible for phantosmia in Parkinson's disease (31). However, reduced thalamocortical cholinergic signaling resulting in enhanced cortico-thalamocortical and cortico-cortical signaling has been implicated in VH pathogenesis in DLB (45); therefore, it is appealing to hypothesize that disrupted cholinergic signaling to the piriformis (29) may play a role in OHs accompanying DLB. Regardless of the neurotransmitter system involved, it has been proposed that incomplete loss of olfactory sensory neurons or the disproportionate loss of inhibitory neurons may be responsible for overactivity that leads to parosmia and phantosmia (17).

The type and frequency of hallucinations in DLB include the more common VHs, which are followed by auditory hallucinations, OHs, and finally tactile hallucinations; notably, OHs are present in only 6.6% of patients (46). OHs are rare in neurodegenerative disorders and are most often accompanied by other types of hallucinations or delusions (47).

TABLE 3. Differential diagnosis, positive clinical and/or diagnostic findings, and proposed mechanism(s) of various phantosmia presentations[a]

Associated diagnosis	Positive clinical and/or diagnostic findings	Proposed mechanism(s)
Neurologic		
Trauma	Evidence of trauma (hemorrhage or volume loss) on imaging (16)	Damage to olfactory neurons, olfactory bulb, or primary olfactory regions (32)
Epilepsy	EEG epileptiform activity; focal lesions on imaging (16)	Seizure focus within mesial temporal lobe structure (e.g., amygdala) (16) or piriform cortex (29)
Multiple sclerosis	Demyelination on imaging (30)	Gray matter atrophy and/or demyelination of the central olfactory system (30)
Primary headache disorders	Meets ICHD-3 criteria for aura (20)	Cortical spreading depression affecting primary, secondary, or association olfactory areas (26)
Parkinson's disease	Reduced striatum uptake on SPECT (31)	Multiple[b]
Psychiatric		
Schizophrenia spectrum disorders	Most often associated with other sensory hallucinations (21)	Dopamine dysregulation in the olfactory tubercle (35) and multiple structural and functional abnormalities of the olfactory system (22)
Depression	Depression severity correlates with severity of olfactory dysfunction (34)	Reduced volumes of the olfactory bulb, primary/secondary olfactory regions, and association cortices (33, 34)
Eating disorders	Additional gustatory dysfunction is common (23)	Alteration in the olfactory microbiome[c] or disrupted cell regeneration of the olfactory epithelium (23)
Medical		
Sinonasal disease	—	Microbe-associated odor stimulus (37),[c] conductive olfactory loss (36), inflammatory damage to olfactory receptor neurons (24), or olfactory bulb atrophy (24)
Post upper respiratory infection	Close temporal relationship to URI (25)	Viral damage to the neuroepithelium and central olfactory pathways (25)
Gastroesophageal reflux disease	—	Reflux-associated odor stimulus (38)[c]
Medications	—	Medication-associated odor stimulus (38)[c]
Radiation therapy	Temporal correlation to radiation therapy (41)	X-ray-induced odor stimulus[c] vs. activation of the olfactory pathway (41)

[a] ICHD-3, International Classification of Headache Disorders, 3rd edition; EEG, electroencephalography; SPECT, single-photon emission computed tomography; URI, upper respiratory infection.
[b] See the Discussion for proposed mechanisms of phantosmia in synucleinopathies.
[c] This phenomenon may not represent true phantosmia given the potential odor stimulus; therefore, it may be closer to parosmia.

Isolated OHs have been described in a subset of patients with Parkinson's disease without cognitive impairment (48). However, VHs more commonly occur first in Parkinson's disease and are usually followed by nonvisual hallucinations over time (49). To our knowledge, there are no reported cases of isolated OHs in DLB. Additionally, there is no literature on the prevalence of OHs at the time of DLB diagnosis, suggesting that OH is either a rare or an underassessed symptom of early DLB.

If the inaugural phantosmia in our patient was secondary to DLB neurodegeneration, it is intriguing to speculate about why the olfactory system was affected before the visual and sensory systems. Differences in temporal symptom onset between Parkinson's disease and DLB may be related to the differential and sequential anatomy affected by synuclein pathology (50). Specifically, it has been proposed that DLB synuclein pathology begins in the olfactory bulb and propagates to nearby limbic regions, followed by higher associative neocortices in a rostrocaudal fashion, as opposed to the caudorostral Braak staging model for Parkinson's disease (50). With a rostrocaudal model in mind, the piriform cortex would be affected early in DLB (50). Given the well-known gradient of inhibitory interneurons conferring a smaller population of inhibitory interneurons in the anterior piriformis (51), the rostrocaudal spread of synuclein pathology may lead to an even more disproportionate loss of inhibitory neurons in the anterior piriformis, leading to overactivity and, ultimately, to phantosmia. Along this line, the later spread of synuclein to the thalamus (50) may be responsible for the later onset of VHs via alteration of thalamocortical connections to the temporo-occipital visual areas (45).

Given the unusual presentation of symptoms and wide range of differential diagnoses for phantosmia, there was some delay in diagnosis even after the patient received specialized care in the BNC. Incorrect initial diagnoses are frequently made among patients with LBD; some reports document an 18-month average delay in the diagnosis of this

disease (52). However, recognizing OHs as a potential early symptom of DLB may lead to earlier diagnoses in future cases.

AUTHOR AND ARTICLE INFORMATION

Departments of Neurology, Behavioral Neurology (Carlisle, Filley) and Headache (Birlea) sections, Psychiatry (Filley), and Cell and Developmental Biology (Restrepo), University of Colorado School of Medicine, Aurora; Marcus Institute for Brain Health, Aurora (Filley).

Send correspondence to Dr. Carlisle (tara.carlisle@cuanschutz.edu).

This work was supported in part by the U.S. National Institute on Deafness and Other Communication Disorders (project 5R01DC000566).

We acknowledge the patient and his family, who made this case report possible.

The authors report no financial relationships with commercial interests.

Received November 8, 2021; revisions received December 17, 2021, and March 9, 2022; accepted March 21, 2022; published online August 22, 2022.

J Neuropsychiatry Clin Neurosci 2023; 35:92–97; doi: 10.1176/appi.neuropsych.21110265

REFERENCES

1. Seeley WW: Behavioral variant frontotemporal dementia. Continuum 2019; 25:76–100
2. Höglinger GU, Respondek G, Stamelou M, et al: Clinical diagnosis of progressive supranuclear palsy: the Movement Disorder Society criteria. Mov Disord 2017; 32:853–864
3. Armstrong MJ, Litvan I, Lang AE, et al: Criteria for the diagnosis of corticobasal degeneration. Neurology 2013; 80:496–503
4. McKhann GM, Knopman DS, Chertkow H, et al: The diagnosis of dementia due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimers Dement 2011; 7:263–269
5. McKeith IG, Boeve BF, Dickson DW, et al: Diagnosis and management of dementia with Lewy bodies: fourth consensus report of the DLB Consortium. Neurology 2017; 89:88–100
6. Gomperts SN: Lewy body dementias: dementia with Lewy bodies and Parkinson disease dementia. Continuum 2016; 22:435–463
7. Ukai K: Tactile hallucinations in dementia with Lewy bodies. Psychogeriatrics 2019; 19:435–439
8. Galvin JE: Improving the clinical detection of Lewy body dementia with the Lewy Body Composite Risk Score. Alzheimers Dement (Amst) 2015; 1:316–324
9. Nguyen TP, Schaffert J, LoBue C, et al: Traumatic brain injury and age of onset of dementia with Lewy bodies. J Alzheimers Dis 2018; 66:717–723
10. Crane PK, Gibbons LE, Dams-O'Connor K, et al: Association of traumatic brain injury with late-life neurodegenerative conditions and neuropathologic findings. JAMA Neurol 2016; 73:1062–1069
11. Hasan S, Mielke MM, Turcano P, et al: Traumatic brain injury preceding clinically diagnosed alpha-synucleinopathies: a case-control study. Neurology 2020; 94:e764–e773
12. LoBue C, Cullum CM, Didehbani N, et al: Neurodegenerative dementias after traumatic brain injury. J Neuropsychiatry Clin Neurosci 2018; 30:7–13
13. Diagnostic and Statistical Manual of Mental Disorders, 5th ed. Arlington, VA, American Psychiatric Association, 2013
14. Krooks JA, Weatherall AG, Holland PJ: Review of epidemiology, clinical presentation, diagnosis, and treatment of common primary psychiatric causes of cutaneous disease. J Dermatolog Treat 2018; 29:418–427
15. Moroy A, Bellivier F, Fenelon G: Olfactory reference syndrome: an unusual delusion in a patient with Parkinson's disease. J Neuropsychiatry Clin Neurosci 2012; 24:E2
16. Ciurleo R, De Salvo S, Bonanno L, et al: Parosmia and neurological disorders: a neglected association. Front Neurol 2020; 11:543275
17. Leopold D: Distortion of olfactory perception: diagnosis and treatment. Chem Senses 2002; 27:611–615
18. Philpott C, Dixon J, Boak D: Qualitative olfactory disorders: patient experiences and self-management. Allergy Rhinol 2021; 12: 21526567211004251
19. Landis BN, Frasnelli J, Croy I, et al: Evaluating the clinical usefulness of structured questions in parosmia assessment. Laryngoscope 2010; 120:1707–1713
20. Headache Classification Committee of the International Headache Society: The International Classification of Headache Disorders, 3rd edition. Cephalalgia 2018; 38:1–211
21. Lim A, Hoek HW, Deen ML, et al: Prevalence and classification of hallucinations in multiple sensory modalities in schizophrenia spectrum disorders. Schizophr Res 2016; 176: 493–499
22. Turetsky BI, Hahn C-G, Borgmann-Winter K, et al: Scents and nonsense: olfactory dysfunction in schizophrenia. Schizophr Bull 2009; 35:1117–1131
23. Leland EM, Xie DX, Kamath V, et al: Psychophysical chemosensory dysfunction in eating disorders: a qualitative systematic review. Eat Weight Disord 2022; 27:429–447
24. Rombaux P, Huart C, Levie P, et al: Olfaction in chronic rhinosinusitis. Curr Allergy Asthma Rep 2016; 16:41
25. Rombaux P, Martinage S, Huart C, et al: Post-infectious olfactory loss: a cohort study and update. B-ENT 2009; 5:89–95
26. Coleman ER, Grosberg BM, Robbins MS: Olfactory hallucinations in primary headache disorders: case series and literature review. Cephalalgia 2011; 31:1477–1489
27. Wolberg FL, Ziegler DK: Olfactory hallucination in migraine. Arch Neurol 1982; 39:382
28. Ahmed M, Donaldson S, Akor F, et al: Olfactory hallucination in childhood primary headaches: case series. Cephalalgia 2015; 35: 234–239
29. Vaughan DN, Jackson GD: The piriform cortex and human focal epilepsy. Front Neurol 2014; 5:259
30. Fleiner F, Dahlslett SB, Schmidt F, et al: Olfactory and gustatory function in patients with multiple sclerosis. Am J Rhinol Allergy 2010; 24:e93–e97
31. Landis BN, Burkhard PR: Phantosmias and Parkinson disease. Arch Neurol 2008; 65:1237–1239
32. Bainbridge KE, Byrd-Clark D, Leopold D: Factors associated with phantom odor perception among US adults: findings from the National Health and Nutrition Examination Survey. JAMA Otolaryngol Head Neck Surg 2018; 144:807–814
33. Athanassi A, Dorado Doncel R, Bath KG, et al: Relationship between depression and olfactory sensory function: a review. Chem Senses 2021; 46:bjab044
34. Croy I, Yarina S, Hummel T: Enhanced parosmia and phantosmia in patients with severe depression. Psychol Med 2013; 43: 2460–2464
35. Kopala LC, Good KP, Honer WG: Olfactory hallucinations and olfactory identification ability in patients with schizophrenia and other psychiatric disorders. Schizophr Res 1994; 12: 205–211
36. Seiden AM, Duncan HJ: The diagnosis of a conductive olfactory loss. Laryngoscope 2001; 111:9–14
37. Nordin S, Murphy C, Davidson TM, et al: Prevalence and assessment of qualitative olfactory dysfunction in different age groups. Laryngoscope 1996; 106:739–744
38. Bainbridge KE, Byrd-Clark D: Prescription medication use and phantom odor perception among US adults. Chemosens Percept 2020; 13:152–158
39. Landis BN, Reden J, Haehner A: Idiopathic phantosmia: outcome and clinical significance. ORL J Otorhinolaryngol Relat Spec 2010; 72:252–255

40. Sjölund S, Larsson M, Olofsson JK, et al: Phantom smells: prevalence and correlates in a population-based sample of older adults. Chem Senses 2017; 42:309–318
41. Hara N, Isobe A, Yamada K, et al: Unusual visual and olfactory perceptions during radiotherapy sessions: an investigation of the organs responsible. J Radiat Res 2021; 62:718–725
42. Lechien JR, Chiesa-Estomba CM, De Siati DR, et al: Olfactory and gustatory dysfunctions as a clinical presentation of mild-to-moderate forms of the coronavirus disease (COVID-19): a multicenter European study. Eur Arch Otorhinolaryngol 2020; 277: 2251–2261
43. Frasnelli J, Landis BN, Heilmann S, et al: Clinical presentation of qualitative olfactory dysfunction. Eur Arch Otorhinolaryngol 2004; 261:411–415
44. Beach TG, White CL III, Hladik CL, et al: Olfactory bulb alpha-synucleinopathy has high specificity and sensitivity for Lewy body disorders. Acta Neuropathol 2009; 117:169–174
45. Esmaeeli S, Murphy K, Swords GM, et al: Visual hallucinations, thalamocortical physiology and Lewy body disease: a review. Neurosci Biobehav Rev 2019; 103:337–351
46. Simard M, van Reekum R, Cohen T: A review of the cognitive and behavioral symptoms in dementia with Lewy bodies. J Neuropsychiatry Clin Neurosci 2000; 12:425–450
47. Schutte MJL, Linszen MMJ, Marschall TM, et al: Hallucinations and other psychotic experiences across diagnoses: a comparison of phenomenological features. Psychiatry Res 2020; 292:113314
48. Bannier S, Berdagué JL, Rieu I, et al: Prevalence and phenomenology of olfactory hallucinations in Parkinson's disease. J Neurol Neurosurg Psychiatry 2012; 83:1019–1021
49. Goetz CG, Stebbins GT, Ouyang B: Visual plus nonvisual hallucinations in Parkinson's disease: development and evolution over 10 years. Mov Disord 2011; 26:2196–2200
50. Cersosimo MG: Propagation of alpha-synuclein pathology from the olfactory bulb: possible role in the pathogenesis of dementia with Lewy bodies. Cell Tissue Res 2018; 373:233–243
51. Luna VM, Pettit DL: Asymmetric rostro-caudal inhibition in the primary olfactory cortex. Nat Neurosci 2010; 13:533–535
52. Galvin JE, Duda JE, Kaufer DI, et al: Lewy body dementia: the caregiver experience of clinical care. Parkinsonism Relat Disord 2010; 16:388–392