Smell and Taste Corner

Apparently Minor Head Trauma Can Lead to Anosmia: A Case Report

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Keywords

Apparently mild head trauma · Functional anosmia · Phantosmia

Abstract

We report the case of a 49-year-old female patient who suffered from anosmia following an apparently mild head trauma when bumping into a door at her home. She reported no other accompanying symptoms after the injury that day. Olfactory function was completely lost, which was noted the day after the trauma. Gustatory function remained normal. Magnetic resonance imaging indicated lesions/bleeding in the right frontal lobe and in the area of the olfactory sulcus/bulb. The present case indicates that in case of apparently mild head trauma with anosmia, an MRI scan of the head should be performed because of suspect brain damage. This case also points to the deeper question how to gauge severity of head trauma.

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Introduction

A variety of factors can cause olfactory dysfunction; e.g., head trauma (HT), upper respiratory tract infection, or chronic rhinosinusitis [1]. Haxel et al. [2] estimated the overall prevalence of olfactory dysfunction after HT at 13%. If HT was classified according to severity, in the reviewed clinical studies, the reported cases of olfactory dysfunction with “mild” HT was 20% [3], 23% [4], and 26% [5]; with “moderate” HT: 37% [3] and 68% [6]; with “moderate to

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severe” HT: 49% [4] and 56% [7]; and with severe HT: 33% [3] and 61% [6]. These patients often have problems with attention, memory, or problem solving, and they show changes in behavior and immune system [8], which seriously affects their private and professional lives. Here, we report the case of a 49-year-old female patient with an apparently mild head injury presenting with olfactory loss and a phantom odor.

Case Presentation

A 49-year-old healthy woman had an apparently mild forehead bump against the door in her home environment about 4 months ago. She reported no other accompanying symptoms after the injury that day. However, the next day she no longer had a sense of smell, and food tasted bland. She could still taste sweet, sour, salty, and bitter, and she perceived food texture, spiciness, and its temperature. In addition, she perceived some nasal trigeminal sensations as cold air, the cooling of peppermint.

One week after the incident, she reported to have vague olfactory percepts to be present now and then. She exhibited daily and incense-like phantom odor. The patient did not report parosmia. She did not regularly take medication, denied drug abuse/smoking, and reported to occasionally drink alcoholic beverages. She had no history of previous trauma or environmental or occupational exposure to potentially toxic/harmful substances suspect to affect the sense of smell. One month after her head injury, she received a magnetic resonance imaging (MRI) scan (Fig. 1) showing local edema in the right frontal lobe with cerebral contusion and laceration. Neither MRI nor nasal endoscopy showed nasal pathology. Orthonasal olfactory function was tested using the “Sniffin’ Sticks” test battery [9], her transition dyspnea index score was 6 (sum of scores from Threshold (1), Discrimination (2), Identification (3)). The patient recognized 4/5 taste sprays [10], indicating normogeusia. Finally, the patient received the initial diagnosis of posttraumatic functional anosmia with phantosmia and normogeusia.

Two months after the incident, the patient returned for review of her case. Her olfactory test score remained unchanged. Nasal trigeminal testing (lateralization test: 17/20) [11] indicated normal function. Results from recordings of EEG-derived chemosensory event-related potentials [12] showed absence of responses to phenyl ethanol and hydrogen sulfide, which supported our initial diagnosis of functional anosmia.

Discussion

What damage is most likely to cause complete olfactory loss? Olfactory loss can be brought about by rupture of the olfactory nerves, although convincing evidence for this shearing in humans is missing [13]. Due to its exposed position, the olfactory bulb appears to be vulnerable in terms of contusion, hematoma compression, and fracture of the cribriform plate [14]. Lötsch et al. [15] combined morphological information from 27 olfactory-eloquent brain regions acquired with four different brain imaging techniques in 127 anosmic patients. Their results showed that lesions in the right olfactory bulb region were much more frequent in anosmia than in patients with a preserved sense of smell. This compares to the present case where HT is likely to having caused the loss of smell.

With regard to the site of the trauma, Sumner [16] suggested that olfactory dysfunction occurs more frequently after occipital than after frontal impact (28 vs. 5%). Another work showed that forehead injury, as the occipital injury, can also be associated with complete olfactory loss [17]. MRI-based studies in HT patients with olfactory loss found that 88% of the anosmic patients had injuries of the olfactory bulbs and tracts, 60% within the sub-frontal region, and 32% within the temporal lobes [18]. According to the literature mentioned above and the MRI findings (Fig. 1) demonstrating local edema and bleeding in the right lateral orbitofrontal cortex and right olfactory bulb, it appears likely that the lesions of the injured parts are associated with olfactory dysfunction.

Fig. 1. Head MRI demonstrating the local edema and bleeding in the right lateral orbitofrontal cortex (black arrows) and right olfactory bulb (white arrow).

Numerous studies show that the severity of the trauma is associated with the likelihood and degree of olfactory loss. In the retrospective analysis, Haxel et al. [2] showed that the overall prevalence of olfactory dysfunction after HT was about 12.8%. Those with loss of consciousness for more than 1 h, high degree of HT, and skull base fractures were most likely to have olfactory dysfunction. However, Doty et al. [19] found that there is no significant association between olfactory function and HT severity (see also [16]). On the other hand, it has been reported that mild HT may even lead to complete loss of the sense of smell. For example, De Kruijk et al. [5] studied 61 males and 50 females with apparently mild HT. In line with the currently described case, their results showed that 5 patients (4%) were diagnosed as anosmic. Considering the results of the present case, on the one hand it indicated that severity of HT may not always predict the presence of olfactory loss. On the other hand, sudden post-traumatic anosmia, as seen in the present case, may prompt the presence of major brain lesions with intracranial hemorrhage, which should prompt more thorough examination of the patient despite the absence of major clinical symptoms.

Another consequence of HT is phantosmia, which is the perception of an odor that is not actually there. Until now, the pathophysiological mechanisms of phantosmia are poorly understood. Leopold et al. [20] suggested that it may relate to causes at the level of the olfactory mucosa or at higher-order structures within the olfactory system. A previous study reported phantosmia to occur after HT [21]. In this case, the phantosmia appeared after HT, and head MRI (Fig. 1) showed the local edema and bleeding in the right lateral orbitofrontal cortex and right olfactory bulb. This seems to indicate that damage to the olfactory bulbs and/or the orbitofrontal cortex is involved in the generation of phantom odor. Both structures are critical in the processing of olfactory information [22].

During routine clinical assessment, HT is classified as mild, moderate or severe, based on the Glasgow Coma Score (GCS) at first examination [23]. HT patients show symptoms as headache, neck pain, nausea, dizziness, vomiting, and amnesia; however, these symptoms were not present in our patient who only reported anosmia with phantosmia. In addition, MRI indicated lesions/bleeding in the right lateral orbitofrontal cortex and right olfactory bulb. This case indicates that anosmia in HT patients without major other symptoms should trigger a more thorough clinical examination including MRI or computed tomography scans of the head and also points to the deeper question how to gauge severity of HT independent of the initial assessment of trauma severity.

Statement of Ethics

All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments. The study was approved by the Ethics Committee of the Medical Faculty of the Technical University of Dresden (EK441102016). Informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

Prof. Thomas Hummel serves as a review board member to this journal. All other authors declare that they have no conflicts of interest.

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Author Contributions

Thomas Hummel, Yunpeng Zang, Antje Hähner, Theresa Lakner: data acquisition. Yunpeng Zang, Thomas Hummel: analysis of the data. Yunpeng Zang, Antje Hähner, Theresa Lakner, Simona Negoias, Thomas Hummel: writing of the manuscript.

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