

Magnetic Resonance Imaging Findings in the Evaluation of Traumatic Anosmia

Jeffrey B. Wise, MD; Gul Moonis, MD; Natasha Mirza, MD

Objectives: Head trauma is a common cause of anosmia, but diagnosis is typically late, owing to more life-threatening sequelae of the injury. Herein, we describe our workup for a case of traumatic anosmia and the magnetic resonance imaging (MRI) findings both at the time of injury and at the 18-month follow-up.

Methods: We present a case report and a review of the literature.

Results: A 33-year-old woman presented to our institution with a chief complaint of loss of smell and taste following an occipital blow to her head that occurred when she was hit by a car while riding a bicycle. We present the findings of MRI performed at the time of the injury and at the 18-month follow-up. We describe the clinical progression of her disease, from symptoms of parosmic and phantosmic episodes accompanied by dysgeusia to total anosmia at the 18-month follow-up.

Conclusions: We advocate the use of MRI, coupled with otolaryngology consultation and formal olfactory testing, in the diagnosis, management, and counseling of patients with anosmia sustained from head injury.

Key Words: anosmia, magnetic resonance imaging, trauma.

INTRODUCTION

Head trauma is a common cause of anosmia, but diagnosis is typically late, owing to the attention given to more life-threatening sequelae of the injury. In fact, studies have cited olfactory dysfunction as occurring in 5% to 10% of all head traumas.^{1,2} Histopathologic changes in posttraumatic anosmia have been described, as well as basic pathophysiologic mechanisms for this type of injury, the most important of which involves the shearing of olfactory fila at their site of penetration of the cribriform plate from the nose.³ Trauma severity and site of injury, specifically, lateral and occipital blows, are associated with a higher degree and incidence of anosmia. Prior studies have noted the rate of at least partial recovery of function on objective testing in patients who have head injuries as being in the range of 30% to 40% of patients, although far fewer patients demonstrate true subjective improvement.⁴ Conventional tests of smell and taste function, such as the University of Pennsylvania Smell Identification Test, are helpful in obtaining a diagnosis of olfactory loss, although these tests are limited by their inability to offer insight into the mechanism of injury (ie, nasal obstruction versus central brain trauma). Magnetic resonance imaging (MRI) with and without contrast exhibits char-

acteristic findings for patients in whom anosmia develops as a result of head injury, and additionally provides information regarding pathophysiology. Herein, we describe our workup for a case of traumatic anosmia and the MRI findings both at the time of injury and at the 18-month follow-up. We advocate the use of MRI, coupled with otolaryngology consultation and formal olfactory testing, as a means to diagnose and manage cases of anosmia caused by head injury and for use in counseling patients with this disorder.

CASE REPORT

A 33-year-old woman presented to the University of Pennsylvania Smell and Taste Center with a chief complaint of loss of smell and taste following an occipital blow she received to her head when she was hit by a car while riding a bicycle. At the time of the accident, she did not lose consciousness, but she reported a loss of smell within a few hours of her injury. In the subsequent weeks, she noticed parosmic and phantosmic episodes accompanied by dysgeusia. These unpleasant sensations occasionally were induced by specific odorants and at other times arose spontaneously. She denied a history of sinonasal disease, prior facial trauma, or tobacco use prior to her injury. Her only medications were oral contracep-

From the Departments of Otorhinolaryngology–Head and Neck Surgery (Wise, Mirza) and Radiology (Moonis), University of Pennsylvania, Philadelphia, Pennsylvania.

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Correspondence: Jeffrey B. Wise, MD, Dept of Otorhinolaryngology–Head and Neck Surgery, Hospital of the University of Pennsylvania, 5-Ravdin, 3400 Spruce St, Philadelphia, PA 19104.

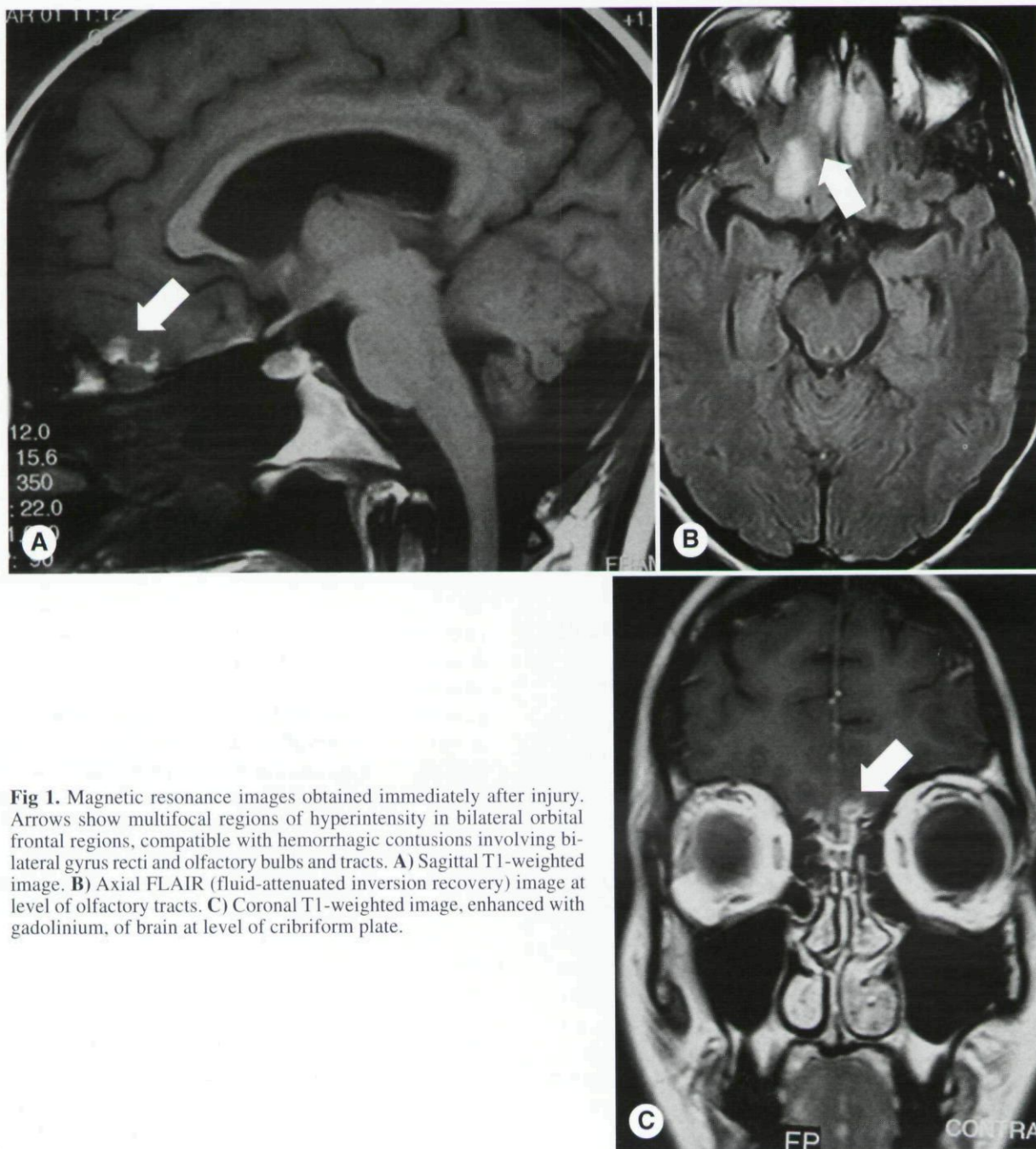


Fig 1. Magnetic resonance images obtained immediately after injury. Arrows show multifocal regions of hyperintensity in bilateral orbital frontal regions, compatible with hemorrhagic contusions involving bilateral gyrus recti and olfactory bulbs and tracts. **A)** Sagittal T1-weighted image. **B)** Axial FLAIR (fluid-attenuated inversion recovery) image at level of olfactory tracts. **C)** Coronal T1-weighted image, enhanced with gadolinium, of brain at level of cribriform plate.

tive pills and acetaminophen used occasionally. A complete head and neck examination was performed and showed no abnormalities. Nasal examination and endoscopy demonstrated a mild left-sided septal deviation with no evidence of mucopus or polyps present. The olfactory clefts were clear on both sides.

An MRI scan of the brain was performed in the emergency department within 4 hours of the injury. Sagittal T1-weighted imaging followed by axial FLAIR (fluid-attenuated inversion recovery) imaging revealed multifocal regions of hyperintensity in the bilateral orbital frontal regions (Fig 1). These regions also demonstrated T1 hyperintensity compa-

tible with hemorrhagic contusion. Specifically, there was bilateral involvement of the gyrus recti and the olfactory bulbs and tracts. No additional cranial abnormalities were noted.

The results of the University of Pennsylvania Smell Identification Test, a 40-item standardized forced-choice microencapsulated test, were indicative of total bilateral anosmia (score 13/40). Performance on the 12-item Odor Memory Test was essentially at chance level (left 4/12, right 5/12). Objective testing of taste function was then performed. The percent correct identification of whole-mouth suprathreshold sweet, sour, bitter, and salty tastant concentrations

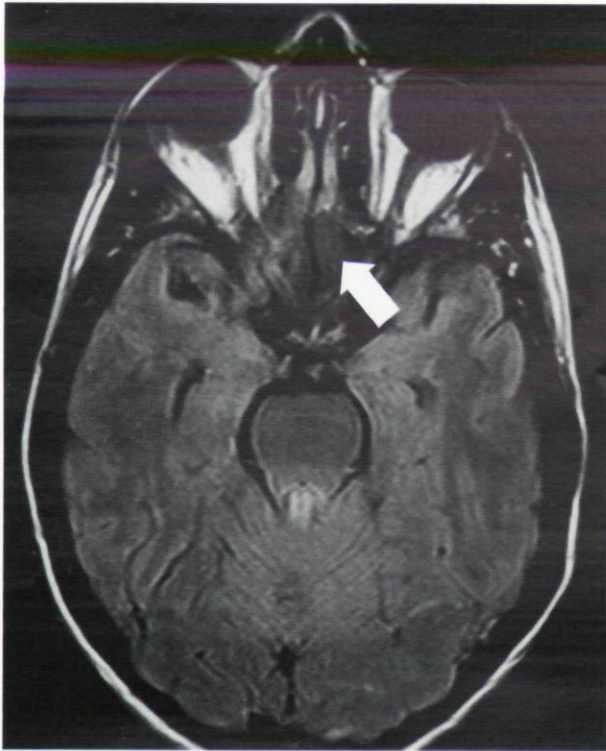


Fig 2. Follow-up axial FLAIR magnetic resonance imaging, performed at 18 months, demonstrates bilateral interval resolution of abnormal signal (hemorrhagic contusions) in orbital frontal regions, and encephalomalacic change in olfactory apparatus and gyrus rectus (arrow).

was within normal limits (40/40). Her score on the Beck Depression Inventory-II (score 3) was unremarkable.

At 18 months' follow-up, the patient reported no return of olfactory function. In fact, along with the development of total anosmia, her phantosmic and parosmic episodes also disappeared. The findings on physical examination remained unchanged. An MRI scan performed at the 18-month follow-up was notable for interval resolution of abnormal signal in the orbital frontal regions. There was gliosis and malacic change noted in the olfactory apparatus and gyrus recti as a result of the prior trauma (Fig 2).

DISCUSSION

Patients who lose their sense of smell are often confronted with a multitude of pragmatic and lifestyle challenges, ranging from employment disability (eg, natural gas workers, cooks, firefighters) to a failure to appreciate pleasing odors and tastes. In their series of 750 patients who presented with smell and taste disorders, Deems et al cited that the majority of patients who presented with complaints of loss of taste actually suffered from olfactory function, "reflecting decreased effectiveness of retronasal stimulation of the receptors by flavor molecules arising from food during deglutition."^{5(p526)} Furthermore,

28.5% of the patients in their group who had smell and taste disorders scored in the range of mild to severe depression as measured by the Beck Depression Inventory.⁵

Although no universal consensus has been reached, it is generally agreed upon that total anosmia occurs in 5% to 10% of head traumas, and that as many as 30% to 40% of head trauma victims experience at least partial loss of smell function (hyposmia).⁶ Although histopathologic evaluation of human olfactory tissue following head trauma has been limited, 3 primary mechanisms of injury have been proposed: 1) shearing of the olfactory fibers from the skull base at the cribriform plate, 2) conductive loss of olfaction via nasal distortion or obstruction, and 3) injury to central olfactory tracts from brain trauma.⁷

Approximately one third of patients with traumatic anosmia recover at least partial function of their sense of smell within a time period of 1 year of their injury. Animal studies support the notion that neuronal olfactory tracts are capable of regeneration, as evidenced by morphological and electrophysiological examination. Kern et al,³ in an immunocytochemical and histopathologic analysis of the olfactory bulbs and tracts of a patient with posttraumatic olfactory dysfunction, postulated that some degree of neuronal regeneration is possible in humans; however, they report that fibrosis and scarring of the cribriform region often limits the reconnection between the axons of regenerating neurons and the olfactory bulb.

Dysosmia, defined as the distortion or absence of smell, has been observed in a high proportion of patients with head injury. This disorder of smell can be temporary or permanent. Doty et al⁴ noted in a series of 268 patients with head trauma that more than one third had experienced at least some degree of dysosmia after sustaining their injuries. The patient complaint of dysgeusia in our case presentation most likely manifested from dysosmia, rather than taste dysfunction per se. This was verified by her perfect score on suprathreshold taste testing (40/40). As described, her dysgeusic and dysosmic episodes gradually disappeared and were replaced by total bilateral anosmia. This clinical course, along with the experience of Doty et al, suggests that dysosmia more likely represents a neurodegenerative process, rather than a regenerative one. Kern et al³ reported a patient who experienced dysosmic episodes following a 2-month period of total anosmia, which was interpreted to represent at least partial regeneration of olfactory tracts. By correlating this time course to that of previously studied animal models, Kern et al gave at least indirect evidence that some functional reconnection to the olfactory bulb can occur.

In the radiographic evaluation of head injuries that

cause anosmia, the inferior frontal lobe appears to be a common site for cortical contusion. This injury is due to the direct force of impact during rapid deceleration of cortical surfaces with edges of the inner table of the skull along the floor of the anterior cranial fossa. Because gray matter is much more vascular than white matter, contusions are much more likely to be hemorrhagic than diffuse axonal injury. Because of the higher resolution of MRI images and the existence of special susceptibility sequences sensitive to the presence of blood products, MRI is the preferred imaging technique for evaluating hemorrhagic contusions and diffuse axonal injury. Additional areas of abnormality that can be missed on computed tomographic images, including nonhemorrhagic foci of injury, will be better detected on MRI scans. However, computed tomographic scanning remains superior to MRI in the assessment of bony injury. In the acute setting after hemorrhagic contusion and shearing injury to the inferior frontal lobe and olfactory apparatus, one observes hyperintensity on T1-weighted images (indicating hemorrhage) and T2-weighted images (indicating edema). In the chronic setting, there is resolution of hemorrhage and edema, but residual gliosis may be seen manifested by focal T2 hyperintensity and loss of brain substance or atrophy. There may be residual hemosiderin deposition, best seen as signal dropout on gradient echo images. Additionally, the dura may adhere to the underlying brain by fibroglial scarring, which may be a source of seizure in posttraumatic cases.⁸

Magnetic resonance imaging has demonstrated abnormalities in patients with posttraumatic olfactory dysfunction at a very high rate (88%), predominantly in the olfactory bulbs and tracts and the inferior frontal lobes.^{9,10} However, those studies were performed in the late stage of posttraumatic olfactory dysfunction

(>3 months after injury). As discussed, posttraumatic anosmia is most often seen in the setting of frontal contact injury. Fractures of the skull or face are commonly associated with bilateral posttraumatic anosmia, and are shown in MRI and computed tomographic studies. Yousem et al^{9,10} found that the volume of the olfactory bulb and tracts was smaller in patients with posttraumatic anosmia than in patients with residual smell function. They hypothesized that the source of olfactory deficit after trauma may be related to shearing of olfactory nerves at the cribriform plate. Direct injury to the olfactory bulb or tract, intracerebral hematoma compressing these structures, and injury to the septal nuclei have also been implicated in posttraumatic olfactory deficits.¹¹⁻¹³ The location and imaging appearance of MRI abnormalities in our case are characteristic of hemorrhagic contusions involving the olfactory apparatus and the inferior frontal cortex. Additionally, it is conceivable that shearing of the olfactory nerve fibers at the site of their passage through the cribriform plate may have been contributory to this patient's symptoms.

In the setting of head trauma, smell dysfunction is often underdiagnosed because of the presence of or search for more life-threatening injuries. We propose that conventional MRI, performed both at the time of injury and in follow-up, may alert the clinician to the possibility of olfactory dysfunction, thus allowing for early consultation by an otolaryngologist and early use of objective smell testing. Furthermore, it has been shown that MRI findings obtained at the time of injury and in follow-up allow insight into the cause and progression of smell dysfunction, thus serving to guide patient counseling and management of this common yet troublesome sequela of head injury.

REFERENCES

1. Sumner D. Post-traumatic anosmia. *Brain* 1964;87:107-20.
2. Yousem DM, Geckle RJ, Bilker WB, Kroger H, Doty RL. Posttraumatic smell loss: relationship of psychophysical tests and volumes of the olfactory bulbs and tracts and the temporal lobes. *Acad Radiol* 1999;6:264-72.
3. Kern RC, Quinn B, Rosseau G, Farbman AI. Post-traumatic olfactory dysfunction. *Laryngoscope* 2000;110:2106-9.
4. Doty RL, Yousem DM, Pham LT, Kreshak AA, Geckle R, Lee WW. Olfactory dysfunction in patients with head trauma. *Arch Neurol* 1997;54:1131-40.
5. Deems DA, Doty RL, Settle RG, et al. Smell and taste disorders, a study of 750 patients from the University of Pennsylvania Smell and Taste Center. *Arch Otolaryngol Head Neck Surg* 1991;117:519-28.
6. Costanzo RM, Becker DP. Smell and taste disorders in head injury and neurosurgery patients. In: Meiselman HL, Rivlin RS, eds. *Clinical measurement of taste and smell*. New York, NY: Macmillan, 1986:565-78.
7. Costanzo R, Zasler ND. Head trauma. In: Getchell TV, Doty R, Bartoshuk L, Snow J, eds. *Smell and taste in health and disease*. New York, NY: Raven Press, 1991:711-30.
8. Grossman RI, Yousem DM. *Neuroradiology — the requisites*. St Louis, Mo: Mosby, 1994:149-70.
9. Yousem DM, Geckle RJ, Bilker WB, McKeown DA, Doty RL. Posttraumatic olfactory dysfunction: MR and clinical evaluation. *AJNR Am J Neuroradiol* 1996;17:1171-9.
10. Yousem DM, Oguz KK, Li C. Imaging of the olfactory system. *Semin Ultrasound CT MR* 2001;22:456-72.
11. Levin HS, High WM, Eisenberg HM. Impairment of olfactory recognition after closed head injury. *Brain* 1985;108:579-91.
12. Potter H, Butters N. An assessment of olfactory deficits in patients with damage to prefrontal cortex. *Neuropsychologia* 1980;18:621-8.
13. Zusho H. Posttraumatic anosmia. *Arch Otolaryngol* 1982;108:90-2.

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