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Chemosensory Impairment after Traumatic Brain Injury: Assessment and Management

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Introduction

Chemosensory impairment is common after head trauma, and conversely, head trauma is a common cause found in patients seeking evaluation of chemosensory disturbances. While chemosensory disturbances are not considered as significant a disability as disturbances of audition or vision, they add to the burden of disability and compromise quality of life for patients with brain injury. Physicians tasked with the acute and rehabilitative care of patients with brain injury should be aware of the pathophysiologic mechanisms, assessment, and management of chemosensory disorders in this patient population.

Olfactory dysfunction is common following traumatic brain injury, occurring in approximately 20% of patients, depending on the mechanism of injury[1]. Associations have been found between the degree of injury, and duration of post-traumatic amnesia[2]. In contrast, gustatory disturbances are infrequent, occurring in less than 1% of cases. Disturbances experienced by patients may be complete (anosmia, aguesia) or partial (hyposmia, hypoguesia) sensory loss, sensory distortion (dysosmia, dysguesia), or the presence of phantom sensations.

Pathophysiology of Post-traumatic Chemosensory Dysfunction

A variety of mechanisms exist whereby head injury or its treatment may lead to chemosensory dysfunction. Multiple causes may coexist, further complicating evaluation, treatment, and determination of prognosis for recovery.

Olfactory disturbances may be categorized as conductive or neurosensory. In the former, pathologies within the sinonasal tract impair odorant access to the olfactory receptors within the superior nasal cavity. Due to their prominence and thin structure, the nasal bones are the most commonly fractured bones of the maxillofacial skeleton. Fractures causing septal deviation may lead to nasal blockage and altered olfaction. Nasal fractures are also involved in more severe midfacial fractures, such as naso-orbital ethmoid or LeFort fractures seen in high impact injuries. Other than mechanical blockage caused by altered bony and cartilaginous anatomy, such injuries inevitably cause mucosal disruption, which may damage the olfactory neuroepithelium or lead to mucosal scarring that may impair odorant

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access. Injury to the olfactory epithelium may also occur secondary to neurosurgical or maxillofacial surgical procedures or life support intervenions including placement of nasotracheal or nasogastric tubes involved in the treatment of craniomaxillofacial injuries,.

Neurosensory deficits may be caused by injuries to any portion of the olfactory pathways, from the superior nasal cavity to the cortical processing centers in the frontal and temporal lobes. The olfactory neurons are particularly susceptible to injury as they traverse the cribriform plate to synapse in the olfactory bulb. Direct injury may occur with fractures involving the anterior cranial base, as seen with high impact injuries or projectiles striking the central midface. However far more common is neuronal injury from shear forces generated by rapid deceleration, with “coup-contracoup” forces causing movement of the brain, mobile within the cerebrospinal fluid, with respect to the calvarium. This mechanism of injury was demonstrated by visualization of severed olfactory nerve fibers at the cribriform plate using electron microscopy in patients with post-traumatic anosmia[3]. Forces sufficient to damage olfactory neurons may be generated with relatively mild injuries, as evidenced by multiple patients seen at our center suffering complete bilateral anosmia after ground level falls. Deficits in the olfactory cortical centers may occur with contusion or intraparenchymal hemorrhage. Injury to the olfactory bulbs or orbitofrontal poles may result from the same coup-contracoup forces that may shear the olfactory neurons. Penetrating projectiles or depressed skull fractures also pose risk to cortical centers. However due to the extensive and bilateral projections of the olfactory pathways, direct cortical injury is anunlikely cause of complete anosmia. Further, cortical injuries are more commonly associated with impairment of odorant recognition, rather than detection[4].

In analogy to olfactory dysfunction, one may also consider gustatory losses as either conductive or neurosensory. The conductive medium allowing substances to reach taste receptors of the tongue and oropharyngeal mucosa is saliva. Although tramatic injuries to the major and minor salivary would be an unlikely cause of gustatory disturbance, various medications used in the management of patients with brain injury may impact saliva production and gustation. These include antidepressants (tricyclics, selective serotonin reuptake inhibitors), anticonvulsants (carbemazepine, phenyoin), antipsychotics (clozapine, resperidone, lithium), antispasmodics/antichlinergics (baclofen, oxbutynin), and narcotic analgesics.

Neurosensory deficits due to peripheral injuries are feasible, but unlikely due to the redundant and bilateral nature of taste innervation to the tongue, carried by cranial nerves VII, IX, and X, and to the deeper, more protected course of these nerves. Tastes fibers to the anterior two thirds of the tongue, carried by the facial nerve, may be injured in temporal bone fractures. Whereas the more common longitudinal fractures (70–90%) result in facial nerve deficits in only 10–20% of cases, the less common transverse fractures (10–20%) result in facial nerve injury in almost 50% of cases[5]. Injury to cranial nerves IX and X is unlikely as both have only a brief course through the wide jugular foramen, making direct injury from skull base fractures implausible. Although the cortical processing centers for taste are not well characterized, taste disturbances from forebrain and basal ganglia lesions have been reported.

Lastly, the impact of olfactory dysfunction on patients’ taste must be considered. Due to the phenomenon of retronasal olfaction, whereby foods in the oral cavity release odorants that pass via the nasopharynx detected by the olfactory epithelium, olfactory dysfunction significantly impacts patients’ perceptions of taste. With the higher prevalence of traumatic injuries to the olfactory system than the gustatory system, it follows that patients with brain injury reporting taste disturbances more likely have olfactory deficits.

Assessment of Chemosensory Impairment

Patients with traumatic brain injury often have concurrent neurosurgical, orthopedic, or visceral injuries whose management leads to unavoidable delay in identification of chemosensory deficits. Thus chemosensory complaints may only arise when patients enter the rehabilitative phase of their management.

History should seek to elucidate the nature of the deficit, potential causes, and the impact of the chemosensory disturbance. The patient should be queried on the severity (partial or complete) and quality (distortion or presence of phantom sensations) of their olfactory or gustatory deficit. The time course may also be useful, as an immediate loss suggests direct effect of trauma, while delayed onset suggests a treatment effect such as due to medication, surgical intervention, or post-traumatic rhinosinusitis. The mechanism, direction, severity, and location of the traumatic injury should be explored. If the patient cannot provide details, family members, hospital personnel, and medical records may provide information about craniofacial lacerations or ecchymosis, epistaxis or cerebrospinal fluid rhinorrhea or otorrhea, which may suggest a mechanism. Operative reports and current and previous medication lists should be reviewed. The patient should be asked about the impact of the chemosensory deficit on daily activities. In particular, dietary intake, as severe chemosensory alterations may cause food aversions and malnutrition, while compensatory overuse of salt or sugar may hinder antihypertensive or diabetic management.

Examination of patients with chemosensory disturbances requires neurologic and otolaryngic evaluation. In the acute setting, the location and extent of craniofacial lacerations, ecchymosis, edema, or tenderness may help elucidate a causative mechanism. Patients with naso-orbital-ethmoid fractures, often associated with direct injury to the cribriform plate, present with widened intercanthal distance, or telecanthus. Nasal bone or septal fractures are readily detected by external inspection or anterior rhinoscopy with a speculum or otoscope. Nasal endoscopy with either rigid or flexible endoscope should be performed on all patients with chemosensory complaints. This provides the means whereby the olfactory cleft may be inspected for edema, ecchymosis, scar formation, or obstruction. In addition, the middle and superior turbinates are inspected for purulent secretions, edema, or polyposis suggesting rhinosinusitis contributing to chemosensory disturbance.

For gustatory complaints, the oral cavity is inspected for trauma to the tongue, deficits of cranial nerves V or XII, and the quantity and quality (viscosity, clarity, color) of saliva. The presence of pathologic post-nasal drainage, carious dentition, or cryptic tonsillitis should be noted, as these may lead to dysguesia. Facial nerve injury is suggested by the presence of ear canal laceration or bony step-offs, bloody or CSF otorrhea, hemotympanum, or Battle’s sign (ecchymosis over the mastoid region). Lastly, the integrity of cranial nerves IX and X may be verified by assessment of gag reflex, or in the case of cranial nerve X, endoscopic assessment of vocal fold function.

Radiologic testing is useful for determination of the pathogenic mechanism of chemosensory disturbances. In patients with brain injury, radiographs obtained during the acute or neurorehabilitative assessment should be reviewed for cortical injuries potentially contributing to chemosensory disturbances. Although standard head CT using 5mm or greater cuts provides a limited view of the sinuses and cribriform plate, two plane (axial and coronal) non-contrast high resolution CT (HRCT) of the maxillofacial region using 1mm or less cuts is optimal for detecting fractures through the thin bone of the anterior skull base and cribriform fossa, and can also demonstrate sinusitis or nasal airway obstruction from septal deviation or scarring. Similar two plane HRCT of the temporal bone may identify fractures through the course of the facial nerve, or in the vicinity of cranial nerves IX and X at the jugular foramen. MRI is more sensitive in detecting subtle cortical injuries, and has

been shown to have adequate resolution to detect changes in the olfactory bulbs in 88% of patients with post-traumatic olfactory deficits[6]. However as HRCT is capable of detecting bony abnormalities, obstructive lesions, or sinusitis, which represent the primary treatable causes of post-traumatic chemosensory disturbances, the additional cost of MRI is seldom justified unless indicated for concomitant neuropsychiatric deficits or to exclude neoplasm.

Clinical tests of chemosensory function are used to confirm patient complaints and to quantify sensory deficits. Objective testing is of greater importance when legal proceedings are involved, disability determination is required, or malingering is suspected. Tests of olfaction assess odor detection thresholds and/or odor identification, with deficits of the former more indicative of receptor neuron injury, and deficits of the latter more suggestive of cortical injury[7]. Popular tests used in clinical centers include the University of Pennsylvania Smell Identification Test (UPSIT)[8], a self-administered 40-item “scratch-and-sniff” test of odor identification, and the University of Connecticut Chemosensory Research Center test[9], which includes odor detection threshold and identification subtests. Test for rapid screening in the hospital setting include the commercially available three-item Pocket Smell Test and the alcohol sniff test[10], the latter requiring only a ruler and standard alcohol pad to assess smell threshold. Quantitative testing of gustation is more problematic, although multiple techniques are used in chemosensory centers[11–13]. The test used in our center involves application of salty, sweet, bitter, and sour solutions first selectively to the anterior two thirds or posterior one third of the tongue and then to the whole mouth, with assessment of detection and identification of these substances, to establish both sidedness and taste specificity of the deficit. This requires quantitative mixing of test solutions and is not readily applicable to the inpatient setting.

Management

For most post-traumatic chemosensory deficits, commonly neurosensory losses, there is no specific treatment. Spontaneous recovery may occur up to a year or more following injury in up to 30% of patients with post-traumatic olfactory deficits[1], presumably from resolution of neuronal or cortical edema, or regeneration of olfactory neurons at the cribriform plate, as demonstrated in animal models[14]. Neuronal regeneration may however lead to aberrant connectivity between olfactory neurons and second order neurons of the olfactory bulb, leading to debilitating dysosmia[15]. Conductive olfactory deficits from nasoseptal fractures, mucosal hematoma or sinusitis, may be amenable to surgical repair in the former case, and medical therapy or endoscopic sinus surgery in the latter cases. Fortunately, post traumatic gustatory dysfunction is rare compared to olfactory dysfunction, and when present spontaneously resolves more frequently[16].

Although seemingly minor compared with extensive neuropsychiatric deficits, chemosensory disturbances can have considerable impact on patients’ lives. Although the AMA Impairment Rating System assigns only 3% disability for complete loss of taste or smell, real world impact of such deficits varies greatly with patients’ vocational and avocational pursuits. Patients in occupations such as cooks, firefighters, plumbers, and cosmeticians may have significant difficulty resuming work after suffering post-traumatic anosmia. Home or workplace safety is also compromised, as patients with impaired olfaction have increased risk of delayed detection of fires, gas leaks, or spoiled foods[17]. Further, significant impact of olfactory impairment on quality of life and increased incidence of depression has been reported[18]. Even when specific treatment does not exist to restore chemosensory function, patient assessment may serve to validate patients’ complaints and concerns, and counseling may reduce their risk of hazardous exposures and mitigate the emotional burden of their deficit[19].

Conclusions

Chemosensory dysfunction is common following brain injury, with olfactory far more common that gustatory deficits. History, physical examination including nasal endoscopy, chemosensory testing, and radiologic imaging are useful in the assessment of these complaints. While the majority of cases represent neurosensory deficits with no specific treatment options, spontaneous recovery may occur within the first 6 months to a year following injury, and patients may benefit from counseling regarding safety issues and compensatory strategies.

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