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Olfactory dysfunction has been reported to affect more than 200,000 patients a year in the USA. A total of 79,000 patients per year are treated for olfactory dysfunction in German hospitals.[1].

Damm M, et al (2004) surveid and obtained comparable epidemiological data and treatment information on olfactory dysfunction in German speaking countries. They sent questionnaires to all otorhinolaryngology departments in Germany, Austria and Switzerland; 52% of hospitals completed the survey. An average of 46 patients with olfactory dysfunction were treated per hospital every month. Hyp- and anosmia were most commonly caused by inflammatory diseases of the nose/paranasal sinuses (53%), respiratory dysfunction (19%), or postviral conditions (11%). Steroids were used most frequently for pharmacological treatment (topically 82%; orally 65%). A compilation of previous and new studies of olfactory disorders suggests associated complaints of poor quality of life, depression, and various specific consequences.

Epidemiological studies show that loss in odor sensitivity is common in both general and clinical populations, whereas dysosmia is less common in general populations but frequent in clinical populations. The most common etiologies are post-upper respiratory infection, nasal/sinus disease and head trauma [8]. Approximately one third of the clinics used B vitamins, or zinc; 80% of the hospitals performed surgery to treat underlying diseases. Acupuncture and smell training was used by approximately 20%.

A viral upper respiratory infection is one of the most commonly identified causes of olfactory loss, accounting for 11% to 40% of patients in most series [4]. Postviral olfactory disorders usually occur after an upper respiratory tract infection (URTI) associated with a common cold or influenza [4]. Postviral olfactory disorder occurs most commonly in middle-aged women between the fourth and eighth decade of life[4] and is most prevalent in spring and summer. . A comparison of the monthly frequency of the disorder with the incidence of isolation of various viruses suggest that the influenza virus, respiratory syncytial virus, and herpes virus are not causative viruses, while parainfluenza virus type 3 is most likely to be a causative virus. This conclusion is supported by immunological study and analysis of symptoms of the cold.

Sugiura M, et al (1998) in their epidemiological study suggested that all patients with postviral olfactory disorder had increased serum antibody titre for parainfluenza virus type 3. No prominent or specific symptoms of common cold were identified as a potential trigger of this syndrome in patient questionnaires [3].

Suzuki M,et al, (2007) investigated the causative viruses in patients with PVOD For this reason nasal discharge was collected from 24 patients with PVOD. They investigated the presence of 10 viruses in nasal discharge and examined the time course, with regard to changes in olfactory dysfunction and nasal obstruction in patients with PVOD, using questionnaires, acoustic rhinometry, and olfactory tests[5]. Rhinoviruses were detected in 10 patients by electrophoresis. Rhinoviruses were also confirmed in four patients by nucleotide sequences.

Viral serotypes were identified to be human rhinovirus (HRV)-40, HRV-75, HRV-78, and HRV-80. One of the four patients complained of anosmia, whereas another complained of dysosmia. Olfactory testing did not show significant improvement at 4, 8, 11, and 24 weeks after the first visit in the four patients, although results of acoustic rhinometry significantly improved.

Two of the four patients complained of olfactory dysfunction even 6 months after the first visit. Coronavirus and parainfluenza virus were detected in one patient each, and Epstein-Barr viruses were detected in three patients[5]

Wang JH, et al (2007) assayed the nasal cavity mucosae of PVOD patients for the presence or persistence of PIV3. For this reason they assessed 25 patients (5 men, 20 women), ranging in age from 31 to 85 (mean, 51) years, diagnosed with PVOD and 22 controls (18 men, 4 women) diagnosed with nasal septal deviation between July 2005 and August 2006. Inferior turbinate epithelial cells were collected using a Rhino-probe mucosal curette, and PIV3 was assayed by seminested reverse-transcription polymerase chain reaction[6].

PVOD occurred most frequently between May and July. Hyposmia was observed in 60% of patients and anosmia in 40%. The most common clinical symptoms were rhinorrhea, sore throat, nasal obstruction, fever, myalgia, cough, and hoarseness. Patients usually visited the outpatient clinic within 3 months after the onset of olfactory dysfunction. Twenty-two of 25 (88.0%) epithelial samples from PVOD patients were positive for PIV3 compared with 2 of 22 (9.1%) epithelial samples from controls The high detection rate of PIV3 in the turbinate epithelial cells of PVOD patients suggests that PIV3 may be the causative virus of PVOD [6].

Commercial preparations of intranasal zinc gluconate gel are marketed as a remedy for the common cold. However, intranasal zinc has been reported as a cause of anosmia in humans and animals. Seventeen patients presenting with anosmia after the use of intranasal zinc gluconate are described [7].

Alexander TH, Davidson TM.(2006) conducted a retrospective case series of the above patients presenting to a nasal dysfunction clinic and conducted complete history and physical examination on all patients, including nasal endoscopy. All patients underwent detailed odor threshold and identification testing.

Threshold and identification testing revealed impaired olfaction in all patients. Inflammatory and traumatic causes of anosmia were excluded based on history, physical examination, and imaging. All patients diagnosed with zinc-induced anosmia or hyposmia reported sniffing deeply when applying the gel. This was followed by an immediate sensation of burning lasting minutes to hours. Loss of sense of smell was then perceived within 48 hours. Seven of 17 patients never developed symptoms of an upper respiratory infection. The zinc-induced anosmia syndrome, characterized by squirt, sniff, burn, and anosmia, occurs after the exposure of olfactory epithelium to zinc cation. It can be distinguished from postviral anosmia based on history[7].

Given the ubiquitous nature of upper respiratory infections, it is not clear what predisposes some patients to develop this complication. Studies have demonstrated degenerative changes within the olfactory epithelium, the severity of which seems to correlate with the severity of olfactory loss. The exact location of the damage in post-URTI is not yet known even though from biopsies a direct damage of the olfactory receptor cells is very likely. Nevertheless, central mechanisms cannot completely be ruled out[4].

The diagnosis is made according to the history, clinical examination and olfactory testing. Affected patients usually recall the acute URTI and a close temporal connection should be present to establish the diagnosis. Although no available therapy has proved effective even though specific olfactory training might be promising. Long-term follow-up data have found that approximately two thirds of these patients eventually experience a significant improvement in their olfactory function[2]. Spontaneous recovery might occur within 2 years[4].

Recent studies illuminate the difficulties that patients with olfactory disorder face in daily life, which underlines the need to understand its prevalence, and to diagnose and treat these patients. Any olfactory disorder must be characterized and described the associated complaints.

Procedures to diagnose olfactory disorders and to identify etiologies are available. Depending on etiology, certain types of treatment are often successful, such as endoscopic sinus surgery and corticosteroid administration in nasal/sinus disease. In post-upper respiratory infection and head trauma, spontaneous recovery is fairly high. In any case, it is advisable to counsel the patient with regards to strategies to cope with olfactory disorders [8].

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