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Original article

Non-sinonasal-related olfactory dysfunction: A cohort of 496 patients

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ABSTRACT

Introduction and aim: There is a high prevalence of olfactory dysfunction in the general population. Several causes of olfactory dysfunction have been reported and this disorder is classically divided into sinonasal and non-sinonasal-related olfactory dysfunction. The aims of this study were firstly, to evaluate the frequency of the various aetiologies of olfactory dysfunction in a population of patients with non-sinonasal-related olfactory dysfunction and secondly, to evaluate the degree of olfactory impairment associated with these various aetiologies.
Material and methods: We retrospectively reviewed a cohort of 496 patients with non-sinonasal-related olfactory dysfunction. The aetiology of the olfactory dysfunction was recorded for each patient. The aetiology was determined by a complete clinical assessment, including medical history, complete otorhinolaryngological examination, psychophysical testing of olfactory function, recording of olfactory event-related potentials and brain magnetic resonance imaging. Six groups of patients were defined on the basis of the aetiology of the disease and orthonasal and retronasal psychophysical olfactory performances were evaluated in each group.
Results: Post-infectious and post-traumatic aetiologies were the most common causes, representing 37.9% and 33.1% of patients, respectively, followed by idiopathic (16.3%), congenital (5.9%), toxic (3.4%) and neurological (3.4%) olfactory dysfunction. Anosmia was significantly more frequent in congenital (93.1%) and post-traumatic (62.8%) olfactory dysfunction, whereas hyposmia was more frequent in the post-infectious group (59.6%). Orthonasal and retronasal olfactory function tests were significantly correlated in all groups except for the congenital group.
Conclusions: The data of this study confirm that the most common causes of non-sinonasal-related olfactory dysfunction are post-infectious and post-traumatic. Post-infectious olfactory dysfunction is mainly observed in middle-aged women and is mainly associated with hyposmia, whereas post-traumatic olfactory dysfunction is mainly observed in young men and is associated with a high rate of anosmia.

1. Introduction

The prevalence of olfactory dysfunction in the population remains a subject of controversy. Although some authors have reported a prevalence of 1 to 3% [1,2], a more recent study reported a high prevalence of olfactory dysfunction, affecting almost 20% of the general population, with anosmia and hyposmia rates of 4.7% and 16%, respectively [3].

Olfactory dysfunction can be due to a large number of aetiologies and can affect all levels of the olfactory system, from the nasal fossae to central olfactory pathways. The most common causes of olfactory dysfunction are inflammatory lesions of the nasal sinuses, post-infectious disease and post-traumatic disease [4–6], as these three aetiologies account for two-thirds of all patients with olfactory dysfunction. However, several other diseases can also affect smell, such as a benign or malignant tumour (hamartoma, esthesioneuroblastoma, meningioma, etc), neurological disease (Parkinson's disease, Alzheimer's diseases, multiple sclerosis), metabolic or endocrine disorders, exposure to toxins (medications, neurotoxic drugs, chemical agents such as benzene, formaldehyde or sulphuric acid), and anosmia can also be congenital (either isolated or part of a more complex syndrome). In many

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cases, no cause can be formally identified and olfactory dysfunction is then considered to be idiopathic.

Olfactory disorders due to sinonasal disease are common and well known to clinicians. Medical and surgical treatment generally allows resolution of olfactory complaints.

In contrast, non-sinonasal-related olfactory dysfunction is less common and less well known to clinicians. However, clinicians must be aware of these various diseases in order to establish an aetiological diagnosis, which determines the patient's prognosis [7]. In some cases (neurological disease, metabolic disorder), the diagnosis also allows treatment of the underlying disease. This is particularly important, as it has now been clearly demonstrated that olfactory disorders severely alter the patient's quality of life, and can be responsible for social disability, anxiety, depression as well as household accidents [8,9]. In the absence of effective treatment for most these diseases, the patient must therefore be given detailed information about the disease and the prognosis for recovery.

The objective of this study was to evaluate the various aetiologies in a population of patients with non-sinonasal-related olfactory dysfunction, the frequencies of these various aetiologies, the clinical characteristics and the degree of olfactory impairment associated with each aetiology.

2. Material and methods

2.1. Patients

This study was conducted on patients attending our centre with olfactory disorders as the main complaint between 2004 and 2011. This population comprised 496 patients in whom olfactory dysfunction was not related to sinonasal disease (allergy, acute or chronic sinusitis, nasal sinus polyposis, benign or malignant tumours of the nasal fossae and paranasal sinuses). The aetiology of olfactory dysfunction had been previously established on the basis of medical history, complete ENT examination, psychophysical assessment of smell, olfactory event-related potentials and brain magnetic resonance imaging (comprising detailed examination of the olfactory bulbs). Six aetiological categories were defined:

- olfactory dysfunction was considered to be toxic when it occurred following exposure to various medicinal products and industrial chemicals;
- the diagnosis of congenital anosmia was proposed when the patient had no olfactory memory and on the basis of imaging (aplasia or hypoplasia of the olfactory bulbs, decreased depth of the olfactory sulcus);
- neurological causes concerned patients with central nervous system degenerative disease, sequelae of stroke or inflammatory disease or intracranial tumour;
- the diagnosis of post-traumatic olfactory dysfunction was based on a clinical history of head injury chronologically related to onset of olfactory dysfunction and on the basis of imaging (fragmented and smaller olfactory bulbs, basal frontal contusions);
- post-infectious olfactory dysfunction was diagnosed when olfactory dysfunction occurred no more than six weeks after an upper respiratory tract infection;
- finally, olfactory dysfunction was considered to be idiopathic when no cause could be identified after a thorough assessment.

This cohort of patients was therefore divided into six distinct groups: toxic, congenital, idiopathic, neurological, post-traumatic and post-infectious groups and the proportion of each aetiology within the cohort was recorded.

2.2. Psychophysical tests

The patient's orthonasal and retronasal psychophysical olfactory performances were evaluated in each group. Orthonasal psychophysical performances were measured by the "Sniffin' sticks test" method [10]. In this test, felt-tip pens impregnated with various odorant substances are presented in front of the patient's nose. This test comprises three aspects: determination of the detection threshold (T for threshold), odour discrimination (D) and odour identification (I). Each subcategory is scored from 1 to 16, with a maximum total score of 48 (TDI score). A total score less than 31 in subjects between the ages of 16 and 35 years, less than 28 in subjects between the ages of 26 and 55 years and less than 19 in subjects over the age of 55 years is considered to indicate hyposmia, and a total score less than 15.5 is considered to indicate anosmia [11]. Retronasal psychophysical performances (R) were evaluated by application of 20 standardized odorant powders on the mobile part of the patient's tongue, and the patient was asked to identify the odour from a multiple choice of four proposals. The maximum total retronasal score is 20; a score less than 16–18 is suggestive of hyposmia [12].

2.3. Statistics

Statistical analysis was performed with SPSS 17.0 software (SPSS Inc, Chicago, Ill). Data were tested by ANOVA with Bonferroni correction for multiple comparisons or nonparametric statistical tests, including Kruskal–Wallis, Mann–Whitney and Chi^2 tests. A P value ≤ 0.05 was considered to be significant. Correlations were determined by Spearman's coefficient.

3. Results

In this cohort of 496 patients, olfactory dysfunction was post-infectious in 188 cases (37.9%), post-traumatic in 164 cases (33.1%), idiopathic in 81 cases (16.3%), congenital in 29 cases (5.9%), toxic in 17 cases (3.4%) and neurological in 17 cases (3.4%) (Table 1).

The mean age of the patients was 56 years in the post-infectious group, 44 years in the post-traumatic group, 55 years in the idiopathic group, 28 years in the congenital group, 58 years in toxic group, and 67 years in the neurological group (Table 1). Patients with congenital olfactory dysfunction were significantly younger than patients of the other groups ($P < 0.001$). Patients with post-traumatic olfactory dysfunction were also significantly younger ($P = 0.002$ versus the toxic group, $P < 0.001$ versus the post-infectious, idiopathic and neurological groups). In contrast, patients with neurological olfactory dysfunction were significantly older than the patients of all other groups, except for the toxic group ($P < 0.001$ versus the post-traumatic and congenital groups, $P = 0.017$ versus the idiopathic and post-infectious groups).

This cohort comprised 275 females (55.4%) and 221 males (44.6%). A significantly higher proportion of females compared to males was observed in the post-infectious group (74.4% females vs. 26.6% males) ($\chi^2 = 34.3$, $P < 0.001$). In contrast, a significantly higher proportion of males compared to females was observed in the post-traumatic group (39.8% females vs. 61.2% males) ($\chi^2 = 8.8$, $P = 0.003$). No significant gender differences were observed in the toxic (35.3% of females vs. 64.7% of males), neurological (58.8% females vs. 41.2% males), congenital (62.1% females vs. 37.9% males) and idiopathic groups (55.1% females vs. 44.9% males) (Table 1, Fig. 1).

Psychophysical scores were indicative of anosmia in 267 patients and indicative of hyposmia in 229 patients [11]. The post-infectious olfactory dysfunction group comprised a significantly higher proportion of patients with hyposmia ($n = 112$)