Nasal septal perforation has a negative impact on olfactory and trigeminal function

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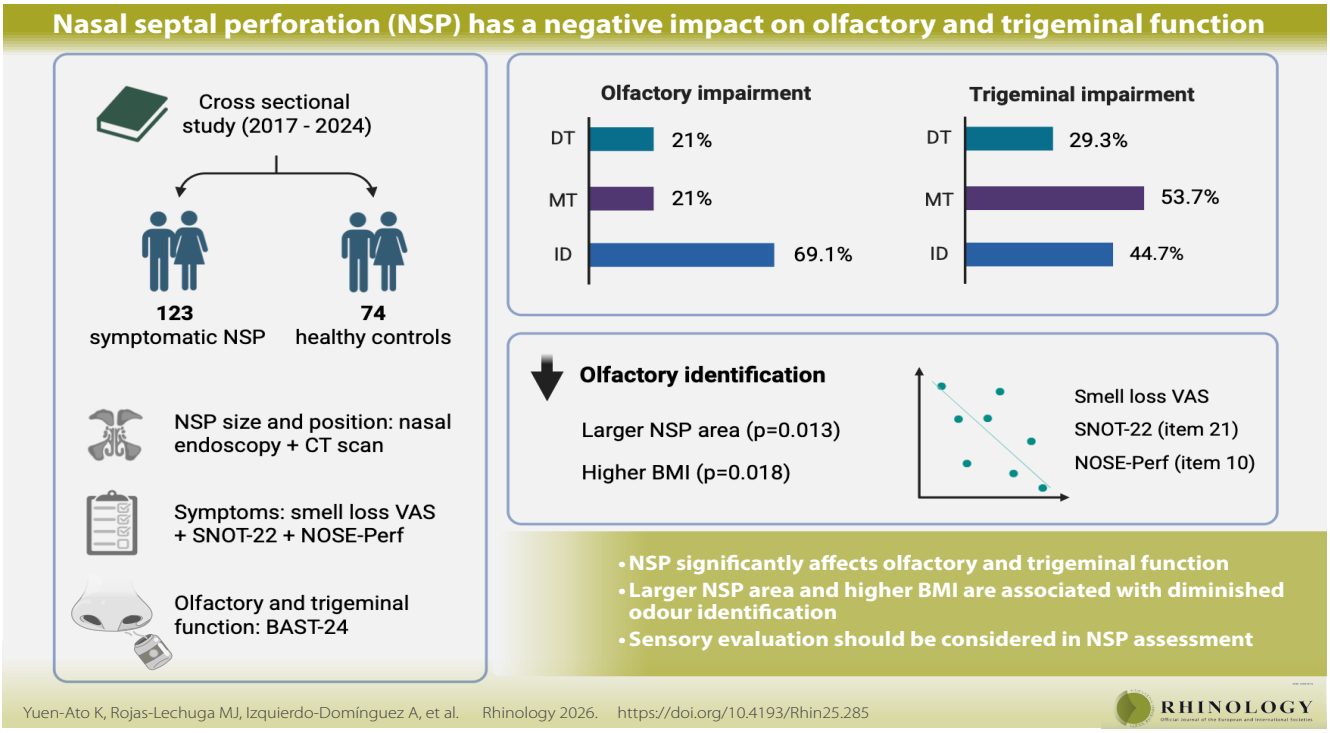

Abstract

Background: Nasal septal perforation (NSP) is commonly associated with nasal obstruction, crusting and epistaxis. Research on its impact on olfactory function remains limited. This study aims to evaluate the sense of smell in patients with NSP and identify factors associated with the olfactory dysfunction.

Methodology: Cross-sectional study of patients with symptomatic NSP and a control group was conducted at Clinic and Teknon hospitals in Barcelona from 2017 to 2024. Data collected included sex, age, comorbidities, body mass index (BMI), NSP aetiology, SNOT-22 and NOSE-Perf questionnaires. Olfactory function was assessed using visual analogue scale (VAS) and the Barcelona Smell Test 24 (BAST-24). NSP size and position were determined through nasal endoscopy and sinus computed tomography scan.

Results: 123 patients with symptomatic NSP and 74 healthy controls were included. Intranasal cocaine use was the most common aetiology. Olfactory dysfunction was observed in 26 patients (21.1%) for detection, 26 (21.1%) for memory, and 85 (69.1%) for identification, significantly different from control group. Patients with olfactory dysfunction presented trigeminal detection and identification impairment in 34% and 51%, respectively. In multivariate analysis, significant differences were found in olfactory identification based on NSP area and BMI, corrected by aetiology. A moderate correlation was found between olfactory identification with VAS, SNOT-22 item 21 and NOSE-Perf item 10. Olfactory identification was significantly correlated with trigeminal scores.

Conclusions: NSP significantly affects olfactory and trigeminal function. Lower identification outcomes were associated with larger NSP area and higher BMI, independently of aetiology.

Key words: anosmia, nasal septal perforation, olfaction, smell loss

Introduction

Nasal septal perforation (NSP) is defined as a defect involving the mucosa, submucosa, perichondrium, and/or the osseocartilaginous framework of the nasal septum, resulting in a communication between the two nasal cavities [1]. NSP prevalence is estimated to range between 0.9 and 2.5% [2,3].

The aetiology of NSP is highly heterogeneous. It can be caused by surgical procedures, intranasal cocaine use, nasal trauma, septal cauterization, occupational exposure, inflammatory, infectious and others. In adults, NSP is more frequently associated with nasal surgery and intranasal cocaine use [4,5].

NSP symptoms are variable and include epistaxis (58%), crusting (43%), nasal obstruction (39%), facial pain (17%) and whistling (10%) [4-6]. The presence of NSP disrupts the normal lamellar airflow, leading to turbulent airflow, impairing the air-conditioning function of nasal cavity and epithelial damage, ciliary loss, respiratory metaplasia and low-grade of chondritis. These changes result in dryness, crusting, bleeding and nasal obstruction. Likewise, NSP can cause chronic inflammation or recurrent infections, which, when combined with repeated local trauma, can perpetuate symptoms [6-8].

Symptom severity often correlates with size and position of the NSP [7-10]. Anterior perforations tend to be more symptomatic due to higher resistance and greater pressure differential, causing increased air shunt through the defect. In contrast, posterior or superior NSP are frequently asymptomatic [6,7,11]. Computational fluid dynamic studies have shown that symptomatic patients exhibit higher wall shear stress and heat flux along the posterior margin of the perforation, which may contribute to crusting and bleeding [9,12,13]. The study of all these factors allows better understanding of the development of NSP symptoms.

Despite extensive research on NSP symptomatology, few studies have specifically assessed olfactory function. Most studies characterize smell loss as a binary condition (present/absent) [14,15], uses Likert or visual analogue scale (VAS) or address it as part of broader symptom questionnaires [7,10,16]. These tools may not accurately capture the complexity of olfactory dysfunction, especially when total scores are influenced by comorbid conditions affecting other symptom domains. To the best of our knowledge, only one study has assessed olfactory function using validated psychometric test [17].

The method employed to evaluate the olfactory function is of vital importance for achieving an accurate diagnosis. Prior research has shown that patients do not accurately self-assess their sense of smell when compared to psychometric testing [18]. Therefore, incorporating both patient-reported outcomes (PROMs) alongside psychometric assessment is strongly recommended [19].

The aim of this study is to assess the olfactory function in patients with symptomatic NSP using the Barcelona Smell Test-24 (BAST-24) and evaluate the factors associated with the development of smell loss.

Materials and methods

Study design

Cross-sectional study involving patients diagnosed with symptomatic septal perforation and a control group. Participants were evaluated in two referral centers (Centro Médico Teknon and Hospital Clinic, Barcelona, Spain) from January 2017 to April 2024. The present study received approval from the clinical research ethics committee (HCB/2017/0268). Informed consent was obtained from all the enrolled participants.

Study population

Patients over 18 years of age diagnosed with symptomatic NSP were selected. NSP associated with inflammatory or infectious conditions were excluded, as well as other pathologies that could alter olfaction, such as neurodegenerative diseases, head and neck radiotherapy, chronic rhinosinusitis or head trauma. A control group was recruited and matched 2:1 by age and gender consisting of healthy volunteers over 18 years of age without sinonasal disease.

Data collection

Demographic data, comorbidities, smoking status and body mass index (BMI) were recorded. Smoking status was categorized as active or non-smoker (former and never smokers). BMI was calculated as weight (kg)/height^2 (m^2) and categorized according to World Health Organization cut-off values: overweight (≥25kg/m^2) and obesity (≥30 kg/m^2). NSP aetiology was categorized as: post sinonasal surgery (endoscopic sinus surgery, septorhinoplasty or turbinate surgery), intranasal cocaine use, intranasal vasoconstrictors use, rhinotillexomania, traumatic or idiopathic.

NSP measurements

NSP dimensions were measured by nasal endoscopy in millimetres (mm), obtaining length as anteroposterior distance (AP) and height as superoinferior distance (SI). Elliptical area (mm^2) was calculated as ½ length x ½ height x π. All patients underwent sinus computed tomography (CT) scan as part of the standardized diagnostic protocol, to characterize the NSP and to exclude other concomitant sinonasal conditions. Radiological evaluation on the sagittal plane of sinus CT scans included the distance from the nasal floor to the inferior edge of the NSP and distance from the incisive canal to the anterior and posterior edges of the NSP. Perforations were classified as anterior if its posterior edge was located anterior to the incisive canal (Figure 1).

Olfactory epithelium boundaries were defined as the anterior

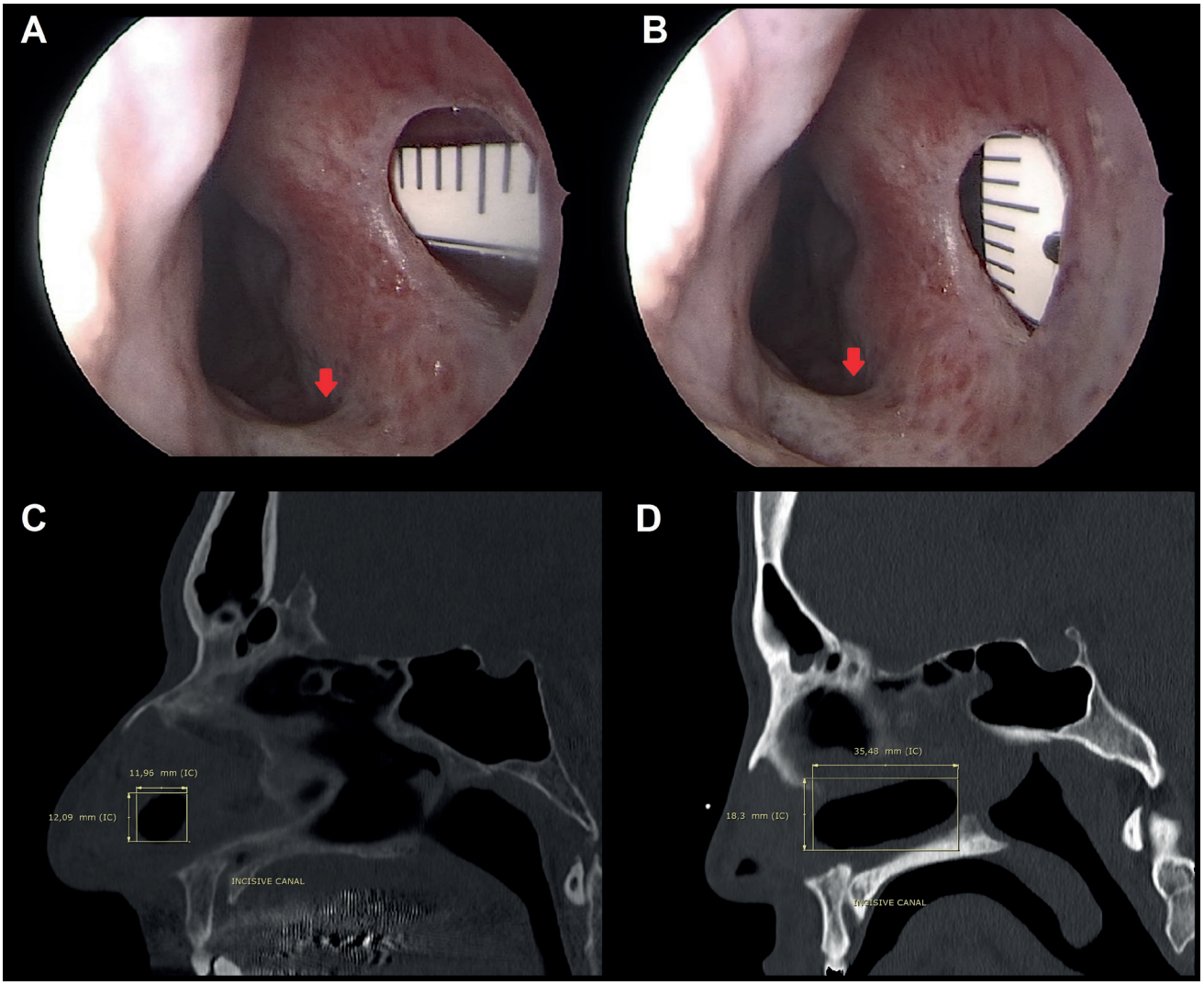

Figure 1. Assessment of NSP measurements by nasal endoscopy and sinus CT scan. A) Endoscopic assessment of anteroposterior distance (length). B) Endoscopic assessment of superoinferior distance (height). NSP position is determined based on the location of the NSP posterior edge relative to the incisive canal (red arrow). C) Sagittal plane of sinus CT scan showing an anterior NSP: the posterior edge is located anterior to the incisive canal. D) Sagittal plane of sinus CT scan showing a posterior NSP: the posterior edge is located posterior to the incisive canal.

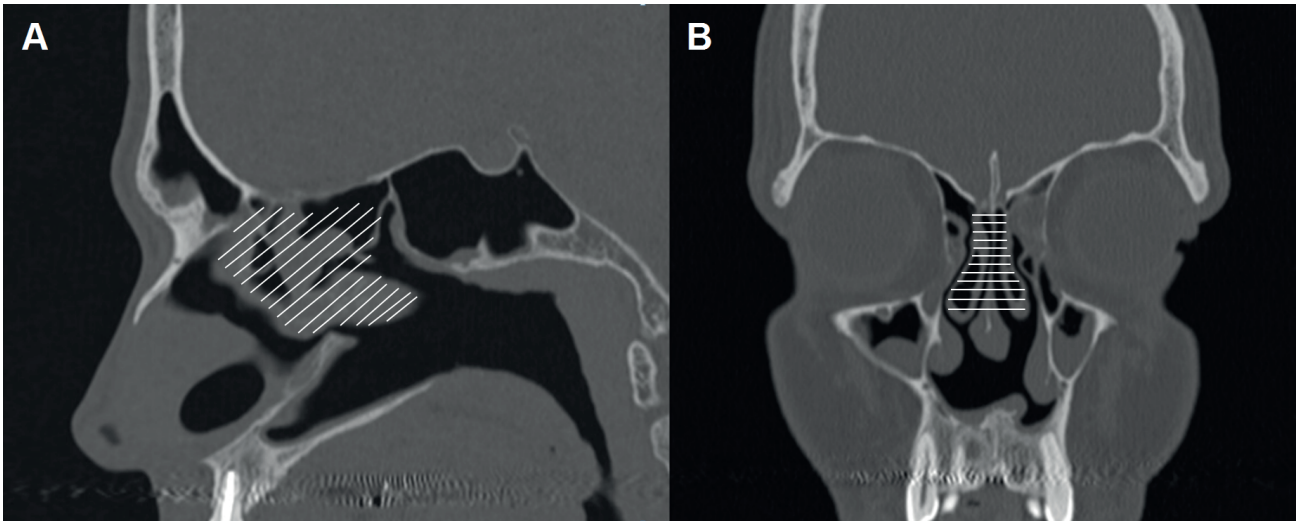

Figure 2. Radiological image of a patient with NSP with an illustrative representation of the olfactory epithelium in a (A) sagittal view and (B) coronal view of the CT scan.

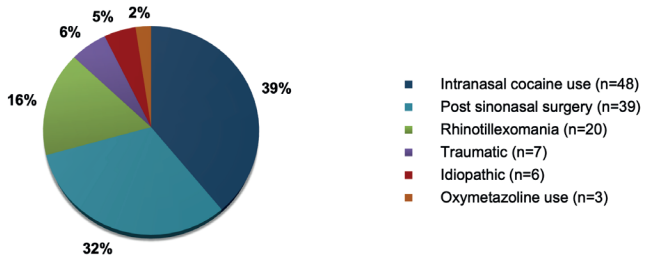

Figure 3. Distribution of aetiologies for nasal septal perforation. The chart illustrates the percentage and number of cases (n) associated with each aetiology.

attachment of the middle turbinate anteriorly, anterior wall of the sphenoid sinus posteriorly, nasal septum medially and middle and superior turbinates laterally [20]. To assess involvement of the olfactory epithelium, we calculated the distance from the incisive canal to the posterior edge of the NSP, as well as the total height (SI diameter + distance from the nasal floor to the inferior edge of the NSP). Perforations located posteriorly with a total height ≥20mm were considered to involve the olfactory epithelium (Figure 2). The cut-off point was estimated based on published measurements [21].

Symptoms assessment

VAS from 0 to 10 cm was used to evaluate symptom severity, where 0 indicated no discomfort and 10 represented the worst imaginable discomfort. Symptoms assessed included: nasal obstruction, rhinorrhoea, loss of smell, facial pain, nasal whistling, crusting and epistaxis. Patients also completed the validated Spanish versions of the Sino-Nasal Outcome Test-22 (SNOT-22) and NOSE-Perf questionnaires (22,23). Total SNOT-22 scores range from 0 to 110 and total NOSE-Perf scores from 0 to 48 points.

Olfactory assessment

Smell loss VAS

Patients were asked to rate their olfaction in a scale from 0 (no impairment) to 10cm (complete loss of smell). Olfaction was rated before performing the smell test.

QoL questionnaires

Smell-related items from the SNOT-22 and NOSE-Perf were evaluated independently. Specifically, SNOT-22 item 21 addresses loss of smell/taste, NOSE-Perf item 10 evaluates decreased sense of smell and item 11 refers to foul or odd smell in the nose.

Barcelona Smell Test-24

Olfactory testing was performed by using the Barcelona Smell Test-24 (BAST-24), in which 20 odorants for the olfactory nerve (first cranial nerve) and 4 odorants for the trigeminal nerve (fifth cranial nerve) were presented using semi–solid-state odorants contained in hermetic glass jars.

Testing comprised of three components:

1. Detection (DT): "Can you smell anything?" (Yes/No)
2. Memory/recognition (MT): "Do you remember having smelt it before?" (Yes/No)
3. Identification (IT): "Which of these 4 odorants is correct?" (Forced-choice)

The score was calculated independently for detection, memory, and identification as percentages. Total score was ranged from 0 to 20 (0-100%) for olfactory nerve odorants and 0 to 4 (0-100%) for trigeminal odorants. Cut-off values were based on validated study in healthy Spanish population [24]. Scores for the 1st cranial nerve were 99.2% for detection, 54.7% for memory, and 72.2% for forced-choice identification, and for the 5th cranial nerve, scores were 98.3%, 59.3% and 42.6% respectively.

Statistical analysis

The mean and standard deviation of the continuous variables were calculated. Qualitative variables were expressed as frequencies and percentages. The normality of the continuous variables was evaluated using the Shapiro-Wilk test. Homogeneity of variance was assessed using Levene's test. The Chi-square test and Fisher's exact test were used to compare categorical variables. Continuous variables were compared between the 2 groups using Student's t-test or Mann-Whitney U test. When multiple comparisons were performed, Bonferroni's correction was applied. NOSE-Perf was incorporated into the study after its Spanish validation. Consequently, data were unavailable for 23 symptomatic NSP patients recruited prior to its implementation. Analysis involving NOSE-Perf was performed using a complete-case approach, and baseline characteristics were compared between NSP patients with and without available NOSE-Perf data to assess potential selection bias. Univariate and multivariate linear regression analysis were performed to evaluate the association between the BAST identification score and variables that may contribute to olfactory dysfunction adjusting for confounders. Subgroup analyses were conducted to assess the independent effects of intranasal cocaine use and smoking status by comparing BAST-24 olfactory and trigeminal scores between cocaine-related and non-cocaine related NSP, and between smokers and non-smokers. A contingency table was performed to assess olfactory and trigeminal evaluation. Pearson or spearman correlations coefficients were calculated to evaluate associations between BAST-24 identification score and VAS for loss of smell, NOSE-perf question 10 and SNOT-22 question 21. A strong correlation was considered for coefficients 0.8-0.9, moderate for 0.4-0.6 and weak for 0.1-0.3 values. All statistical tests were two-tailed. Alpha was set at 0.05 for significance. All statistical analysis were performed using STATA v.16.1 software (StataCorp, TX, USA).

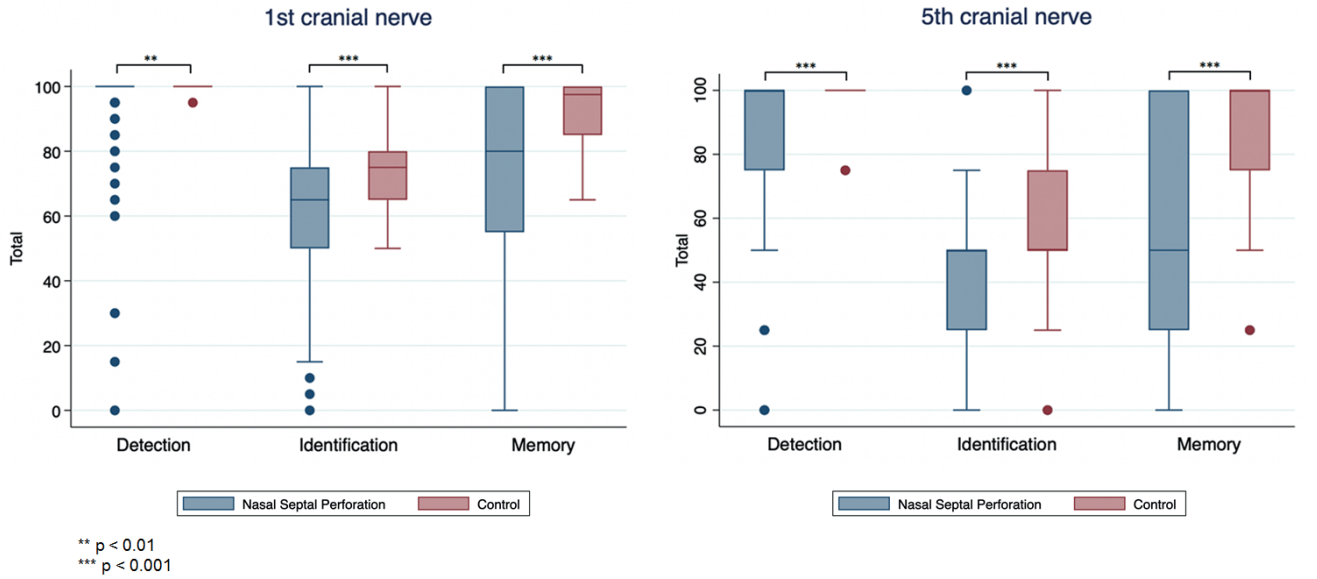

Figure 4. Box plots of Barcelona Smell Test (BAST-24) olfactory and trigeminal scores for nasal septal perforation and control group.

Results

General results

A total of 123 patients with symptomatic NSP were evaluated, with a mean age of 48.8 years (range 18-76), of these, 80 (65%) were male. Forty-five patients (36.9%) were active smokers and 39.5% (n=47) had normal weight according to BMI. Control group comprised 74 individuals (mean age of 47.3 years, range 20-75), with 41 (55%) being male. No differences were observed in age, sex or BMI between groups ($p > 0.05$).

The most common aetiology of NSP was intranasal cocaine use followed by post sinonasal surgery (Figure 3). No differences were observed in aetiology based on sex (p=0.110).

Endoscopic measurements revealed a mean NSP length of 21.5mm (SD=10.4), mean height of 15.4mm (SD=6.7), and a total area of 300.9 mm2 (SD=261.3). Nineteen patients (15.4%) had subtotal or total perforation exceeding 30 mm. NSP related to intranasal cocaine use had larger areas, 406 mm2 (SD=320.4), compared to those caused by rhinotillexomania, 158.8 mm2 (SD=85) (post hoc Bonferroni p=0.005).
Based on NSP position, 41.5% (n=51) were classified as anterior and 58.5% (n=72) as posterior. Among posterior NSP with a total height ≥20mm, 33% (n=29) of cases involved the olfactory epithelium.

The most frequently reported symptoms included nasal obstruction, crusting and rhinorrhoea. No sex-related differences were observed in any QoL instrument. All patients completed the Spanish version of SNOT-22 questionnaire. NOSE-Perf data were collected in 174 patients due to its late validation. No significant differences in baseline characteristics were observed between patients with and without available NOSE-Perf data. Mean total scores for symptomatic NSP were 45.9 (SD=23.4) for SNOT-22 and 25.4 (SD=10.5) for NOSE-Perf. In contrast, the control group reported scores of 2.4 (SD=3.7) and 1.2 (SD=1.7), respectively ($p < 0.001$). Detailed results are presented in Table 1.

Olfactory outcomes

The mean VAS score for loss of smell in symptomatic NSP was 3.6 (range 0-10), indicating moderate severity.

Analysis of specific smell-related items from each QoL questionnaires (Table 1), revealed that patients with symptomatic NSP had significantly higher scores. More than a very mild problem was reported by 80 patients (65.7%) for SNOT-22 item 21 (loss of smell/taste), 47 patients (64.3%) for NOSE-Perf item 10 (decreased sense of smell), and 35 patients (47.9%) for NOSE-Perf item 11 (foul or odd smell in the nose).

In olfactory testing using BAST-24, 26 patients (21.1%) presented impairment in detection, 26 (21.1%) in memory and 85 (69.1%) in identification for the first cranial nerve. Regarding trigeminal sensitivity, 36 patients (29.3%) presented impairment in detection, 66 (53.7%) in memory and 55 (44.7%) in identification. Notably, 50.6% of patients exhibited impairment in both olfactory and trigeminal identification and 34.1% had combined olfactory identification and trigeminal detection impairment. Significant differences were observed in olfactory and trigeminal testing between symptomatic NSP and control group (Figure 4).

To isolate the individual effects of intranasal cocaine use and smoking status, a subgroup analysis was performed by categorizing aetiology into cocaine-related vs. other NSP aetiologies,

Table 1. Patient's demographic data, nasal septal perforation characteristics, and clinical and olfactory outcomes according to VAS, QoL questionnaires and Barcelona Smell Test (BAST-24).

Characteristics	NSP n=123	Control n=74	p-Value
Age, mean (SD)	48.8 (12.6)	47.3 (14.7)	0.391
Sex, n (%)			
Males	80 (65)	41 (55)	0.195
Females	43 (35)	33 (45)	
VAS (0-10), mean (SD)			
Nasal obstruction	5.8 (2.8)	0.9 (1.7)	< 0.001
Crusting	5.4 (3.5)	0.4 (1.3)	< 0.001
Rhinorrhoea	4.2 (3.4)	0.7 (1.6)	< 0.001
Whistling	3.7 (3.4)	0.1 (0.3)	< 0.001
Loss of smell	3.6 (3.4)	0.1 (0.5)	< 0.001
Facial pain	2.8 (3.3)	0.1 (0.8)	< 0.001
Epistaxis	2.7 (3.3)	0.1 (0.5)	< 0.001
QoL questionnaires, mean (SD)			
SNOT-22 (0-110)	45.9 (23.4)	2.4 (3.7)	< 0.001
NOSE-Perf (0-48)	25.4 (10.5)	1.2 (1.7)	< 0.001
VAS smell loss (0-10), mean (SD)	3.6 (3.4)	0.1 (0.4)	< 0.001
SNOT-22 item 21 (0-5), median (IQR)	2 (3)	0 (0)	< 0.001
NOSE-Perf item 10 (0-4), median (IQR)	1 (3)	0 (0)	< 0.001
NOSE-Perf item 11 (0-4), median (IQR)	0 (3)	0 (0)	< 0.001
BAST-24 olfactory, mean (SD)			
Detection	95 (15.7)	99.9 (0.6)	< 0.01
Memory	73.4 (27.5)	93.2 (8.9)	< 0.001
Identification	62.7 (18.7)	73.5 (11.5)	< 0.001
BAST-24 trigeminal, impaired, n (%)			
Detection	83.3 (29.8)	98.9 (4.9)	< 0.001
Memory	53.7 (37.8)	84.1 (22.9)	< 0.001
Identification	38.2 (26.3)	54.4 (26)	< 0.001

revealing lower trigeminal scores in patients with intranasal cocaine use. Similarly, a subgroup analysis of smokers vs. non-smokers showed lower trigeminal detection scores in the smoking group (Figure S1).

In the univariate analysis of BAST-24 identification scores (Table 2), significant differences were found by NSP area (p=0.007) and obesity according to BMI (p=0.014). NSP identification score by aetiologies showed better identification scores for rhinotillexomania comparing with intranasal cocaine use (p=0.021). No significant differences were found in BAST-24 scores when comparing NSP with olfactory-epithelium involving vs. non-involving areas (p=0.476).

In multivariate analysis (Table 2), lower identification scores were associated with NSP area (p=0.013) and obesity (p=0.018). Neither intranasal cocaine use nor smoking showed statistically significant associations after adjusting for these variables.

Moderate negative correlations were found between BAST-24 identification and loss of smell VAS (rho=-0.43, p<0.001), SNOT-22 item 21 (rho=-0.40, p<0.001) and NOSE-Perf item 10 (rho=-0.45, p<0.001). Positive moderate correlation was found between BAST-24 olfactory identification and BAST-24 trigeminal detection (rho=0.47, p<0.001) and identification score (rho=0.35, p<0.001).

Discussion

The main findings in this study are: First, over 60% of NSP patients experience olfactory dysfunction. Second, larger NSP area and higher BMI are associated with lower identification scores, independently of aetiology. Third, half of patients with olfactory dysfunction also exhibit trigeminal impairment.

Smell loss was reported in 69% of symptomatic patients. We hypothesized that this may result from altered nasal airflow dynamics, decreased airflow to the olfactory cleft, and impaired conditioning of inspired air, consistent with computational models showing restoration of airflow after surgical repair [25]. Additionally, crusting and rhinorrhoea may further obstruct olfactory pathways.

In our sample, the mean VAS score for smell loss (3.6) aligns with previous studies [26,27], suggesting moderate QoL impact. This was corroborated by QoL questionnaires, with mean scores

Table 2. Univariate and multivariate models for Barcelona Smell Test (BAST-24) identification scores.

Variables	Univariate model		Multivariate model	
	Coefficient (95% CI)	p-Value	Coefficient (95% CI)	p-Value
Age	-0.04 (-0.30, 0.23)	0.795		
Female	1.78 (-5.26, 8.81)	0.618		
Diabetes mellitus	3.97 (-10.50, 18.44)	0.588		
Body Mass Index				
Normal weight (reference)				
Overweight	-6.60 (-14.33, 1.12)	0.093	-7.52 (-15.29, 0.26)	0.058
Obesity	-11.11 (-19.95, -2.27)	0.014	-10.79 (-19.67,-1.90)	**0.018**
Active smokers	-2.30 (-9.29, 4.68)	0.515	4.78 (-3.81, 13.37)	0.273
Intranasal cocaine use	-6.96 (-13.73, -0.12)	0.044	-6.63 (-15.3, 2.04)	0.132
Nasal septal perforation area	-0.01 (-0.02, -0.004)	0.007	-0.02 (-0.03, -0.003)	**0.013**
Nasal septal perforation affecting olfactory epithelium	-3.10 (-11.73, 5.52)	0.476		

of 45.9 for SNOT-22 and 25.4 for NOSE-Perf. SNOT-22 has been reported to be significantly higher in patients with NSP and CRS compared to controls [16,26]. Moreover, Leong et al. [16] reported a mean SNOT score of 50.2, like Khong et al. [7] who described that incorporating NSP-specific symptoms such as crusting, bleeding and whistling, revealed significant changes with position and size variations. In this regard, a specific questionnaire for NSP was developed [10]. Our mean NOSE-Perf score is in line with previous reports, ranging from 23.7 to 25.7 [23,28-30].

Smell-related specific questions revealed that most patients rated their loss of smell as "no problem" to "mild problem" for both SNOT-22 item 21 and NOSE-Perf item 10. The response average score for the last item in our sample was in line as other studies [29,31], while the first item showed more variability [16,26,32]. This discrepancy can be explained by the fact that this question evaluates both taste and smell together, leading to significant variability in the definition and perception of taste among patients.

Qualitative olfactory dysfunction was also reported. This may be related to crust formation, accumulation of secretions and possible recurrent infections. Previous studies reported foul smell in 26-31% [33] and cacosmia in up to 50% of cases [34]. In our study, qualitative alterations were measured using item 11 of NOSE-Perf, yielding a mean of 1.1 (SD=1.3), consistent with previous findings [23,29].

Psychometric testing (BAST-24) revealed impairment in detection (21.1%), olfactory memory (21.1%), and identification (69.1%), with significantly lower scores in patients with greater NSP areas and higher BMI. The only study that used a psychometric test was conducted by Altun et al., [17] where 42 patients were evaluated after NSP repair using Sniffin' Sticks at one, three and six months after surgery, reporting a baseline TDI score of 25 with significant postoperative improvement, particularly in threshold scores. Notably, their cohort included younger patients (mean age 32.5 years) with smaller perforations (1.8 ± 3 cm).

Our second finding indicates that lower identification outcomes were associated with larger NSP area and higher BMI, independently of aetiology.

The most common aetiology in our cohort was intranasal cocaine use. Although two systematic reviews [35,36] identify iatrogenic and traumatic causes as most common, Spanish studies confirm a higher incidence of cocaine-related NSP (19-23.3%) [26,37]. This condition can lead to more extensive damage to the septum and nasal structures making surgical repair more challenging. As a referral centre, this could explain the higher incidence in our population. It should be considered that etiological classifications can be inconsistent between studies leading to variable results.

Several questionnaires have been used to assess the impact of size and position on NSP symptoms. Gökgöz et al. [8] reported significant differences in NOSE scores based on position, with anterior and middle NSP more symptomatic than posterior, without size-related differences. In their study, position was defined by the anterior perforation edge using the inferior and middle turbinate heads as references. The NOSE scale is used to assess nasal obstruction; however, it does not evaluate the entire clinical spectrum of NSP. In another study, Khong et al. [7] reported greater NSP-specific symptom improvement in larger perforations, such as crusting, epistaxis and whistling. Computational

fluid dynamic studies corroborated these findings, demonstrating disturbances in warming and airflow patterns with high wall shear stress levels particularly at the posterior margin of the NSP [9,12,13]. Beckmann et al. [38] further showed symptom improvement after posterior septectomy. It is important to recognize that there is not a standardized method for measuring NSP or determining its position; landmarks have included the inferior turbinate [9], the columella [7] and the extent of involvement of cartilaginous or bony septum [39]. Our methodology used the incisive canal, assessed both endoscopically and radiographically, as a reproducible anatomical reference.

BMI was also associated with impaired olfaction. Recent systematic reviews reported lower threshold and discrimination scores assessed by Sniffin' Sticks with increasing weight, without differences in identification across weight groups [40,41]. Olfactory improvement has also been reported after bariatric surgery with variations in odour subtest between studies [42,43]. These changes have been associated with metabolic and hormonal dysfunctions, such as hyperglycemia, hyperinsulinemia, insulin resistance, ghrelin and IGF-1 deficiency, increased leptin, among others, which modulate functions in both central and peripheral olfactory pathways [44].

Our third result highlights the relation between olfactory and trigeminal systems. Patients with olfactory dysfunction presented trigeminal detection and identification impairment, and over one-third presented impairment in both cranial nerves, reinforcing the need to assess both pathways. The trigeminal system plays a crucial role in odour perception. Olfactory activation can enhance the intensity of trigeminal sensations, inversely; the presence of a trigeminal component in an odorant can reduce its olfactory perception [45]. Trigeminal activation also influences the perception of nasal airflow and mediates respiratory reflexes [46]. In anosmic patients, reduced trigeminal sensitivity has been reported by lower scores in lateralization task [47], higher thresholds of intranasal irritants [48] and diminished electrophysiological responses [49]. This highlights the cooperative nature and importance of both systems for effective sensory processing.

To date, no studies have specifically addressed trigeminal sensitivity in NSP. Scheibe et al. [50] evaluated olfactory and trigeminal function in patients undergoing septoplasty, and reported reduced trigeminal sensitivity compared to healthy controls prior to surgery, with no significant postoperatively changes, suggesting that diminished sensitivity to nasal airflow could contribute to the perception of impaired nasal breathing. These results support the need for systematic research on trigeminal function in NSP. They may also help explain symptoms frequently reported and support the need for a comprehensive trigeminal assessment prior to surgical decision-making, to accurately identify patients who are most likely to benefit from intervention and thereby optimize therapeutic outcomes.

Our study provides clinically meaningful insights. The high proportion of patients with olfactory dysfunction emphasizes the role of sensory impairment as an integral component of the clinical presentation, reinforcing the need to incorporate olfactory and trigeminal testing into the standard diagnostic evaluation. Furthermore, identifying variables associated with poorer olfactory performance adds predictive value and enables more precise risk stratification. These findings support future investigations into the impact of surgical repair or olfactory training aimed at enhancing nasal sensory function.

Taking all findings into consideration, we hypothesized that smell loss in NSP could be multifactorial. Significant changes in odour identification in larger areas could be related to greater changes in nasal airflow and air conditioning in addition to damage of the olfactory epithelium regardless of the NSP aetiology. Moreover, chronic local inflammation and distorted transport of odorants due to altered ciliary function and mucus properties could act as contributing factors.

Strengths of our study include the use of a validated psychometric test to measure olfactory function and trigeminal assessment along with the inclusion of a robust sample with diverse aetiologies and a control group. Moreover, the analysis of tomographic images allows determining the exact position, height, and area of the NSP.

Limitations involve the exclusion of asymptomatic patients and lack of smell threshold test. The BAST-24 is not regarded as the standard method for assessing intranasal trigeminal sensitivity. Odours that strongly co-activate trigeminal fibres inevitably also stimulate olfactory receptors at lower thresholds, introducing a potential confounding bias between olfactory and trigeminal responses. Therefore, conclusions regarding trigeminal sensitivity should be interpreted with caution. Additionally, the BAST-24 is limited to Spanish-speaking populations, which may affect generalizability to other cultural contexts. Furthermore, absence of longitudinal follow-up limits the evaluation of any changes on olfaction over time or after surgical intervention.

Conclusion

This study highlights the significant association between NSP and olfactory dysfunction, emphasizing the potential impact on both olfactory and trigeminal systems. Larger perforation areas and elevated BMI contribute to diminished odour identification that could be related to changes in nasal airflow. Future research should explore longitudinal changes and intervention outcomes

to further understand sensory impairments in NSP.

Author contributions

KYA, MJRL: concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript; AID, JM, IA: concept and design, revision of the manuscript, supervision and final approval.

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None.

Conflict of interest

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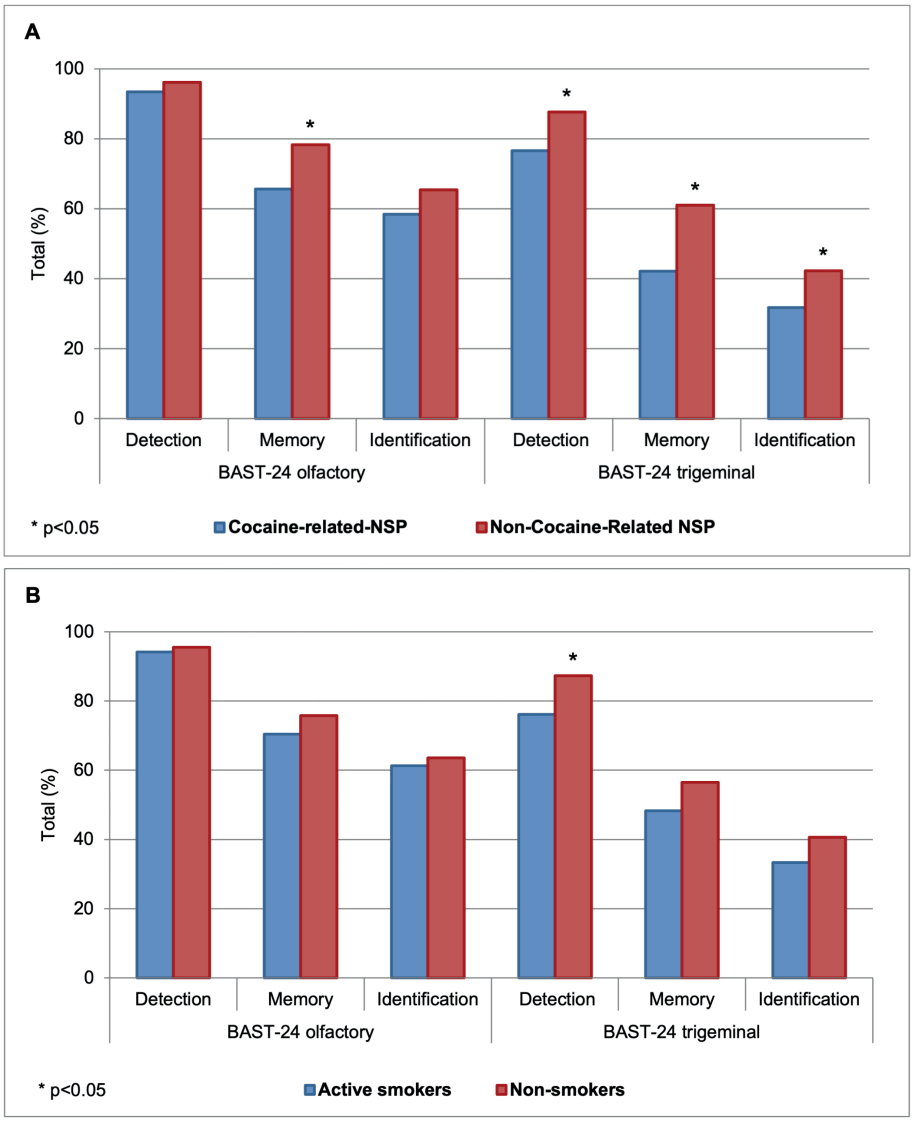

Figure S1. Subgroup analysis of olfactory and trigeminal function scores by NSP aetiology and smoking status. A) Comparison between cocaine-related and non-cocaine-related NSP. B) Comparison between active smokers and non-smokers.