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When recreation turns risky: the multifaceted negative impact of cocaine-induced nasal septal perforations

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Abstract

Purpose This study evaluates the impact of nasal septal perforation (NSP) due to recreational intranasal cocaine use on quality of life (QoL), comparing it with NSPs from other causes using both disease-specific and generic QoL instruments.

Methods A prospective observational study was conducted from 2017 to 2024 acrosstwo referral centres.

Results A total of 152 patients with NSP were included, of whom 93 (61.2%) were male and 57 (37.5%) cocaine-related NSP. Cocaine-induced perforations showedlarger antero-posterior (mean 27.1 mm, SD 12.2) and super-inferior (mean 18.6 mm, SD 7.5) diameters, as well as greater surface area compared to other aetiologies ($p < 0.001$ for all comparison). Nasal obstruction, rhinorrhoea and crusts were the most severe symptoms reported via visual analogue scale (VAS) scores in both groups.Cocaine-induced NSPs reported worse SNOT-22 (mean 53.9 vs. 42.8; $p = 0.004$), particularly in Emotion, Sleep and Nasal. SEPEQOL item #12 ("aesthetic changes inmy nose") was reported as problematic by 57.8% cocaine users, with 80.8% rating it as fairly bad to a severe issue. Mental health outcomes were worse in the cocaine group based on SF-36 scores ($p = 0.003$). NSP-specific questionnaires (NOSE-Perf and SEPEQOL) demonstrated a similar impact on QoL across both groups.

Conclusion NSP negatively impairs sinonasal symptoms and QoL. Cocaine-induced NSPs are associated with more severe anatomical damage and greater psychosocial burden. A comprehensive assessment addressing both physical and psychological aspects is essential for accurate diagnosis and effective management.

Keywords Nasal septal perforation · Cocaine · Quality of life · NOSE-Perf · SEPEQOL · SNOT-22

Introduction

Cocaine is one of the most consumed illicit drugs worldwide and its recreational use is related to the stimulating effect on the central nervous system. In Europe, surveys indicate almost 2.5 million young adults (15 to 34 years old) used cocaine in 2024. In Spain, according to the latest European Drug Report, the prevalence of consumption is 2.4%, reaching 3.1% in young adults from 15 to 34 years old [1].

Intranasal administration remains the most common route of cocaine intake, leading to a significant impact on the superior aerodigestive tract, especially the nasal cavity. Cocaine's vasoconstriction properties may result in ischemia, necrosis, and eventually nasal septal perforation (NSP). Systemic manifestations of cocaine use, such as dermatological, pulmonary, renal, and cardiovascular adverse effects, are also well described in the literature [2].

Some patients may develop cocaine-induced midline destructive lesions (CIMDL), a necrotizing inflammatory tissue response that can mimic granulomatosis with polyangiitis (GPA), Eosinophilic granulomatosis with polyangiitis (EGPA), or malignant neoplasms [3, 4]. CIMDL should be suspected when at least two of the following criteria are present in the context of cocaine use: NSP, palatal

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perforation, or deconstruction of the lateral nasal wall [2, 5]. When cocaine is adulterated with levamisole, a vasculopathy may occur, typically presenting with skin manifestations, fever, and arthralgia. ENT involvement has been reported in approximately 15.1% of such cases [6, 7].

A detailed medical history along with a complete physical examination including assessment of the nasal pyramid, nasal endoscopy, and sinus CT scan is recommended in NSP due to cocaine. Based on the prevalence of identified lesions in patients with CIMDL according to location, Nitro et al. proposed a 4 grades classification system. This ranges from isolated NSP, observed in 99.2% of cases, to more extensive involvement of the orbit, lamina papyracea, or skull base seen in 7.9% of cases [8].

In addition, general blood tests including anti-neutrophil cytoplasmatic antibodies (ANCA) and/or biopsy are recommended when vasculitis is suspected, or to rule out malignancy or concomitant infections. Urinalysis and/or hair drug testing is also advised in case of suspected ongoing cocaine use or when findings are inconsistent with the reported history [9–11].

Patients with cocaine-induced NSP require a comprehensive evaluation, including assessment of quality of life (QoL) through patient-reported outcome measures (PROMs). This approach helps to accurately capture the broader impact of the condition on each individual beyond the anatomical defect, and supports the development of multidisciplinary, patient-centred care strategies.

This study aims to test the hypothesis that NSPs due to cocaine consumption significantly impair QoL compared to other aetiologies using PROMs.

Materials and methods

Study design

A prospective observational study was conducted from 2017 to 2024 in two referral centres (Hospital Clinic and Centro Médico Teknon, Barcelona, Spain). We selected surgical candidates to ensure homogeneity in disease severity and minimize confounding factors due to varying perforation sizes or stages. The study was approved by the hospital's Drug Research Ethics Committee (CEIm) under number HCB/2017/0268 and all patients gave their consent.

Population selection

Patients over 18 years old with symptomatic NSP unresponsive to medical treatment and deemed suitable for surgical reconstruction were included in this study. The aetiology of NSP was recorded and categorized into two groups: (1) recreational cocaine use and (2) other causes, including post-sinonasal surgery, rhinotillexomania, trauma, use of intranasal vasoconstrictors, and idiopathic origins. Recreational cocaine use was assessed either by interview, urine test and/or hair analysis.

Exclusion criteria included the presence of other pathology of the paranasal sinuses or nasal cavity, such as chronic rhinosinusitis, a history of neoplasia, prior head and neck radiotherapy, or pre-existing inflammatory diseases with sinonasal involvement such as GPA. Total or subtotal NSPs not tributary to surgical closure were not included.

Outcomes and assessments

1- Nasal examination

NSP dimensions were measured in millimetres (mm) using a surgical ruler under endoscopic examination. The length was recorded as anteroposterior (AP) distance and the height as super-inferior (SI) distance. The approximate area was calculated using the elliptical area formula (½ height ×½ length × π). Perforations were classified by horizontal length according to the Bansberg et al. classification: small (1–5 mm), medium (6–15 mm) and large (>15 mm) [12].

In cases of suspected vasculitis, infections, malignancy, or when the evaluator identified clinical discrepancies, a biopsy was performed. ANCA testing was included in the blood panel for patients presenting with vasculitis-like lesions [13, 14].

A Sinus CT-scan was performed with the main purposes of classifying the NSP's location, assessing the remnant of the osseocartilaginous support, identifying additional other CIMDL, and excluding chronic rhinosinusitis. The nasopalatine canal (also known as incisive canal) was identified on sagittal plane. NSPs were classified as anterior when their posterior border was located anterior to the canal.

CIMDL lesions were classified according to the grading system proposed by Nitro et al., based on lesion prevalence and anatomical involvement [8].

- Grade 1: Involvement limited to the nasal septum.
- Grade 2a: Extension to the inferolateral district (inferior turbinate and maxillary sinus medial wall).
- Grade 2b: Involvement of the palate.
- Grade 3: Additional destruction of ethmoid bone, middle turbinate and superior turbinate.
- Grade 4: Extension to the neurocranium (papyracea, orbit or skull base) involvement.

2- Sinonasal symptoms

Patients completed a visual analogue score (VAS) assessment ranging from 0 to 100 mm; where 0 indicated no discomfort and 100 represented the worst imaginable discomfort. The VAS was used to evaluate 7 sinonasal symptoms: nasal obstruction, crusting, epistaxis, rhinorrhoea, nasal whistling, loss of smell, and facial pain. A global VAS score was posteriorly calculated by averaging the scores of these 7 symptoms, referred to as the Total 7 Symptom Score (T7SS).

3- Quality of life

Patients completed both generic and disease-specific questionnaires. The generic Short-Forms 36 (SF-36) was administered alongside the following specific instruments: Sinonasal Outcomes Test 22 (SNOT-22), Nasal Obstruction System Evaluation (NOSE-Perf) and/or Septal Perforation Quality of Life (SEPEQOL). All questionnaires were provided in paper format and filled out autonomously by the patients during their initial consultation.

– SNOT-22

The Spanish version of SNOT-22 contained 22 questions grouped into 5 domains by Khan: nasal, ear/facial, sleep, function, and emotion [14]. Each question was scored from 0 to 5 on a Likert scale, where 0 was 'no problem' and 5 was 'the most serious possible problem'. The SNOT-22 with a total score of 0 to 110 points was classified into 4 categories: no alteration (0–10), mild (11–20), moderate (21–50) and severe (more than 50) [15].

– NOSE-Perf

The Spanish version of NOSE-Perf questionnaire was used [16]. However, it was not administered to all patients due to its recent validation, which may induce a selection bias in the analysis. Seven specific questions about NSP were measured with a 5-point Likert added to NOSE questionnaire [17]. Questions were expressed from 0 to 4 and total NOSE-Perf score ranges from 0 to 48 points, being 48 the worst result [18].

– SEPEQOL

The Spanish version of SEPEQOL was used [19]. It contained 12 specific questions about NSP were measured with a 5-point Likert expressed from 0 to 4. The total score ranges from 0 to 48 points, being 48 the worst result.

– SF-36 questionnaire

The Spanish version of SF-36 was used [20]. It contained 36 questions grouped into 8 dimensions: physical function, role physical, bodily pain, general health, vitality, social function, role emotional, and mental health. These domains were summarized by physical and mental aspects. SF-36 items and summaries were scored from 0 (worse QoL) to 100 (better QoL) [21].

Statistical analysis

Qualitative variables were expressed in frequencies and percentages. The Chi-square test and Fisher's exact test were used to compare categorical variables. The normality of the continuous variables was evaluated using the Shapiro-Wilk test. The mean and standard deviation or median and interquartile range (IQR) of the continuous variables were described. Levene's test assessed the homogeneity of variance. Continuous variables were compared between 2 groups using Student's t-test or Mann-Whitney.

To explore the association between clinical and anatomical variables and patient-reported quality of life, a linear regression model was constructed using the total SNOT-22 score as the dependent variable. Univariable analyses were initially performed for each variable of interest, including aetiology (intranasal recreational cocaine use vs. other), age, sex, perforation size (Bansberg classification), perforation position (anterior vs. posterior), and the presence of psychiatric comorbidity.

Two multivariable linear regression models were subsequently developed. Model A included aetiology, age, sex, perforation size, and perforation position as covariates. Model B incorporated psychiatric comorbidity as an additional covariate, to account for its potential influence on quality of life.

Regression coefficients (β), 95% confidence intervals (CI), and p-values were reported for all models. A two-tailed p-value < 0.05 was considered statistically significant. All statistical analyses were performed using Rstudio v2024.12.0+467 and STATA v.16.1 software (StataCorp, TX, USA).

Results

A total of 152 patients with NSP were included, 57 cases (37.5%) were related to recreational intranasal cocaine use. Among them, 54 patients (94.7%) were classified in the cocaine group based on self-reported current or past use. However, 3 patients (5.3%) who initially denied cocaine consumption were found to have active use confirmed by positive urine test conducted after obtained informed consent.

Within the cocaine-related group, 15 patients underwent urine drug testing. Of these, 8 tested positive for active cocaine consumption: 4 cases associated the presence of other substances (all 4 with benzodiazepines and 2 with cannabis). One patient with a negative urine test underwent subsequent hair analysis, which was also negative. Another patient, without any prior toxicological testing, also had a negative hair test.

Only 3 patients had cocaine-induced vasculitis with positive ANCA in the blood analysis, requiring a multidisciplinary approach.

By combining findings from the nasal examination and CT imaging, patients in the cocaine-related NSP group were classified according to Niro et al. classification: a total of 49 patients (86.0%) were classified as Grade 1, 4 patients (7.0%) as grade 2 A, 3 patients (5.3%) as grade 3, and 1 patient (1.7%) as grade 2B.

Compared to the other aetiologies, patients with cocaine-related NSPs were younger, with a mean age of 43.5 years (SD 10.3; range 18.2–64.9) ($p<0.001$). Moreover, psychiatric comorbidity was significantly more prevalent among patients with cocaine-induced NSP compared to those with other aetiologies (70.6% vs. 42.5%, $p=0.002$).

Additionally, they had larger NSPs' diameters (AP and SI) and overall area compared to other aetiologies ($p<0.001$, $p<0.001$ and $p<0.001$ respectively). The mean AP diameter was 27.1 mm (SD 12.2, range 6–55), the main SI diameter was 18.6 (SD 7.5, range 10–35), and the main perforation area was 444.1 mm^2(SD 325.3, range 14.1–1295.9.1.9). According to the Bansberg et al. classification, 82.5% of patients in the cocaine group were categorized as having large perforations (> 15 mm). (Table 1) (Fig. 1).

Table 1 Comparison between cocaine nasal septal perforation and others by age, sex, position and measures

Variables	Intranasal cocaine recreational use $N=57$	Other causes $N=95$	p-value
Age, mean (SD)	43.5 (10.3)	50.3 (13.7)	**<0.001**
Sex, n (%)			
• Female	27 (47.4)	32 (33.7)	0.133
• Men	30 (52.6)	63 (66.3)	
NSP position, n (%)			
• Anterior	25 (43.9)	63 (66.3)	**0.011**
• Posterior	32 (56.1)	32 (33.7)	
NSP size*, n (%)			
• <5 mm	0 (0.0)	4 (4.2)	**0.001**
• 6–15 mm	10 (17.5)	39 (41.1)	
• >15 mm	47 (82.5)	52 (54.7)	
Measures, mean (SD)			
• Length (mm)	27.1 (12.2)	18.9 (8.2)	**<0.001**
• Height (mm)	18.6 (7.5)	14.0 (5.6)	**<0.001**
• Elliptica area (mm^2)	444.1 (325.3)	231.7 (181.7)	**<0.001**

Abbreviations: *NSP* nasal septal perforation. *SD* Standard Deviation. Statistical significant differences ($p<0.05$) in bold. *Size classified as Bansberg et al. [12]

Nasal obstruction, rhinorrhoea, and crusts were the most bothersome symptoms reported in both groups, as measured by VAS. Patients in the cocaine group reported worse rhinorrhoea, with a moderate mean VAS of 53.9 (SD 35.5) ($p=0.007$). In contrast, they reported less epistaxis compared to other aetiology, with a mean VAS of 12.4 (SD 22.7) ($p<0.001$). (Table 2)

In terms of PROMs, patients with cocaine-induced NSPs reported a greater QoL impairment, as reflected by higher SNOT-22 scores ($n=56$. Mean 53.9, SD 23.4, range 7–108) compared to those with other aetiologies ($n=95$. Mean 42.8, SD 22.0, range 3–101) ($p=0.004$). (Table 2) (Fig. 2).

The more affected domains of SNOT-22 reported by the cocaine recreational use group were Emotion, Sleep, and Nasal. Significant differences with other aetiologies were reported in the Nasal ($p=0.003$), Ear/Facial ($p=0.026$), Function ($p=0.042$), and Emotion ($p<0.001$) domains. (Table 2) (Fig. 2).

In univariable analysis, recreational intranasal cocaine use was significantly associated with worse quality of life, as reflected by higher SNOT-22 total scores (β = 11.1, 95% CI: 3.6–18.6; $p=0.004$). In the multivariable model adjusted for age, sex, perforation size (Bansberg classification), and perforation position (Model A), the association between cocaine use and SNOT-22 remained statistically significant (β = 9.6, 95% CI: 1.5–17.7; $p=0.021$). However, when psychiatric comorbidity was included in the model (Model B), the association between cocaine use and SNOT-22 scores was no longer statistically significant (β = 4.3, 95% CI: −4.8 to 13.4; $p=0.350$), while psychiatric comorbidity itself was independently associated with worse quality of life (β = 11.8, 95% CI: 3.1–20.5; $p=0.008$). (Table 3)

Using the NOSE-Perf questionnaire, patients with cocaine-induced NSPs reported similar scores ($n=35$. Mean 26.6, SD 10.3, range 0–48) as other aetiologies ($n=51$. Mean 24.3, SD 9.5, range 0–47). (Table 2)

Cocaine-induced NSP's showed similar SEPEQOL scores ($n=46$. Mean 25.6, SD 10.8, range 0–44) as the other aetiologies group ($n=65$. Mean 23.1, SD 9.53, range 5–44). (Table 2) Notably, in SEPEQOL, item #12 [aesthetic changes in my nose] was reported as a problem by 26 patients (57.8%) in the cocaine group. Among them, 21 patients (80.8%) rated this issue as a fairly bad to severe problem. However, only 7 patients (12.3%) in this group were found to have a visible nasal pyramid defect on physical examination.

Consistent with SNOT-22, worse mental outcomes were reported in the recreational cocaine use group by SF-36 ($p=0.003$). (Table 2)

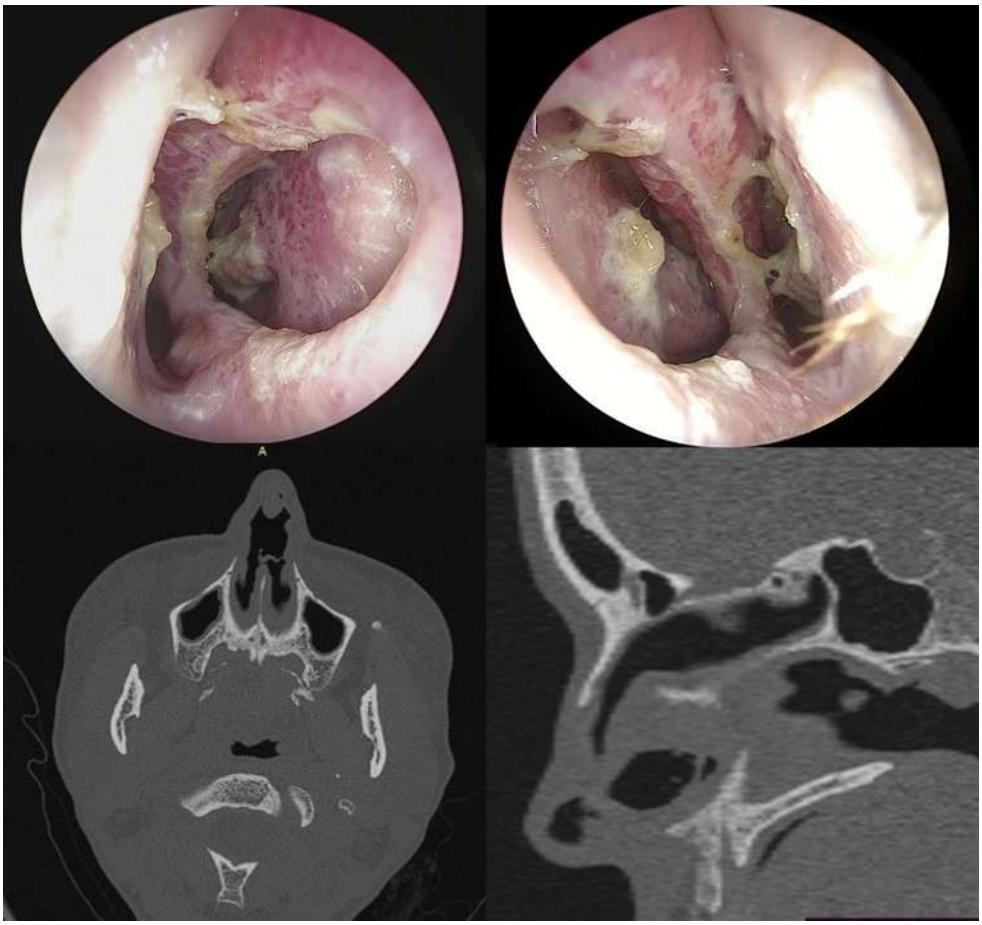

Fig. 1 Nasal septal perforation endoscopic (A: right nostril, B: left nostril) and CT scan view (C: axial, D: sagittal)

Discussion

To the best of our knowledge, this is the first study specifically focused on NSP induced by recreational cocaine use. The most relevant findings of this study are: (1) Cocaine-induced NSPs affect a significantly younger population compared to other aetiologies. (2) These perforations are typically larger in both height and length. (3) The predominant symptoms in cocaine-induced NSPs are nasal obstruction, rhinorrhoea, and crusts. (4) Patients with cocaine-induced NSPs report a greater negative impact on QoL, including both nasal and psychological dimensions, compared to those from other aetiologies. (5) More than a half of patients report aesthetic changes.

NSP resulting from recreational cocaine use not only causes nasal symptoms and potential nasal deformity but also negatively impacts self-esteem and overall QoL.

Cocaine use is strongly associated with emotional disturbances, including anxiety, depression, paranoia, and impaired emotion regulation. Acute use often triggers paranoid ideation and agitation, while chronic exposure is linked to persistent mood disorders and empathy deficits [23]. Neuroimaging studies show cocaine users have hyperactive limbic responses to negative emotions and reduced prefrontal regulation, contributing to emotional dysregulation and impulsivity [24, 25]. Additionally, reduced emotional empathy has been documented in chronic users, affecting social functioning [26].

Beyond medical or surgical management, it is essential to address the underlying addiction through psychological or psychiatric support to prevent further damage. Achieving full recovery requires a comprehensive, multidisciplinary approach that prioritizes physical and mental health, ultimately promoting better long-term outcomes.

In the largest NSP case series to date (n = 202), recreational cocaine use caused only 4.9% of NSP [27], while Khong et al. reported a prevalence of 22.5% in their cohort [28]. In contrast, our study observed a significantly higher rate, with cocaine-related NSPs comprising 37.3% of all cases. This discrepancy may be attributed to the accessibility to the public healthcare system in Spain, which provides care regardless of socioeconomic status. In addition, patients with cocaine-induced NSP demonstrated greater psychiatric comorbidity and psychological impact, highlighting the importance of systematic counselling and support. Instruments such as SNOT-22 not only quantify nasal

Table 2 Comparison between cocaine nasal septal perforations and others by visual analogue scale and quality of life questionnaires

Variables	Intranasal cocaine recreational use $N=57$	Other causes $N=95$	p-value
VAS, mean (SD)			
• Nasal obstruction	40.8 (18.9)	43.8 (19.2)	0.419
• Rhinorrhoea	56.6 (31.7)	59.3 (26.8)	0.766
• Loss of smell	53.9 (35.5)	39.2 (32.9)	**0.007**
• Whistling	38.6 (34.1)	30.9 (32.2)	0.184
• Facial pain	37.9 (29.6)	37.8 (35.0)	0.730
• Crusts	33.7 (36.4)	26.6 (31.1)	0.250
• Epistaxis	51.2 (35.7)	58.2 (34.3)	0.370
	12.4 (22.7)	39.8 (34.8)	**<0.001**
SNOT-22, mean (SD)			
• Nasal domain	53.9 (23.4)	42.8 (22.0)	**0.004**
• Ear/Facial domain	20.6 (8.4)	16.3 (8.3)	**0.003**
• Sleep domain	6.1 (5.1)	4.3 (4.7)	**0.026**
• Function domain	10.4 (6.2)	9.9 (6.0)	0.694
• Emotion domain	7.6 (5.2)	5.8 (4.6)	**0.042**
	9.3 (5.1)	6.4 (4.9)	**0.001**
NOSE-Perf, mean (SD)	26.6 (10.3)	24.3 (10.0)	0.299
SEPEQOL, mean (SD)	25.6 (10.8)	23.1 (9.5)	0.210
SF-36, mean (SD)			
• Physical summary	44.7 (10.4)	45.2 (10.6)	0.720
• Mental summary	36.1 (12.5)	43.1 (14.2)	**0.003**

Abbreviations: *SD* Standard Deviation. *VAS* Visual Analogue Scale. Statistical significant differences ($p<0.05$) in bold. SNOT-22 domains classified by Khan et al. [22]

symptoms but also address psychological domains, offering a comprehensive view of patient well-being.

After adjusting for psychiatric comorbidity, the association between recreational cocaine use and total SNOT-22 scores was attenuated and no longer statistically significant. This finding should be interpreted with caution, as psychiatric comorbidity may not simply confound the relationship but instead act as an intermediate variable in the causal pathway linking cocaine exposure to impaired quality of life. In our cohort, psychiatric comorbidity was significantly more prevalent among patients with cocaine-induced perforations (70.6%) compared to those with other aetiologies (42.5%, $p=0.002$), and was independently associated with worse SNOT-22 scores. These findings suggest that part of the negative impact of cocaine on patient-reported outcomes may be mediated through its psychiatric consequences. Adjusting for such a mediator in multivariable models may underestimate the total burden of disease attributable to cocaine. This highlights the importance of comprehensive, multidisciplinary management that includes mental health support for patients affected by NSP.

The aetiologic diagnosis of NSP requires a thorough clinical history conducted in a trusting and non-judgmental environment that fosters a strong doctor-patient relationship. In most cases, patients will disclose cocaine use when asked directly. However, if cocaine consumption is denied but remains strongly suspected, several diagnostic tests can be employed (blood, saliva, urine and hair analysis). Cocaine can be detected in blood or saliva for up to 48 h after use. Urine testing allows detection for approximately 3 days following occasional use and up to 14 days in frequent users. If cocaine use is still suspected despite negative findings from these tests, hair analysis offers a reliable alternative, capable of identifying long-term use and providing a timeline of drug exposure. Hair grows at an average rate of approximately 0.35 mm per day, enabling retrospective assessment of cocaine consumption patterns [10, 29].

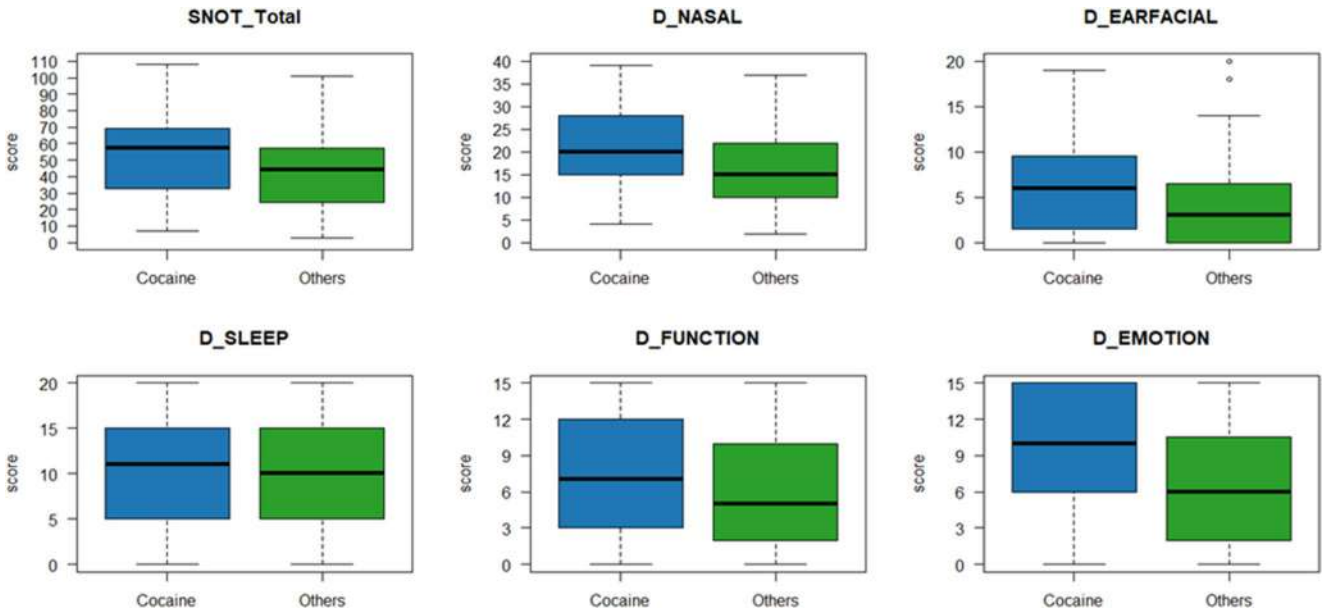

Fig. 2 Box plots of the total score of the SNOT-22 and by Khan et al. Domains. [22]

Table 3 Association between clinical variables and SNOT-22 total score: univariable and multivariable linear regression models

Variable	Univariable β (95% CI)	*p*-value	Multivariable A β (95% CI)*	*p*-value	Multivariable B β (95% CI)**	*p*-value
Intranasal cocaine recreational use (vs. other causes)	11.1 (3.6–18.6)	**0.004**	9.6 (1.5–17.8)	**0.021**	4.3 (−4.8–13.4)	0.350
Age (per year)	0.0 (−0.3–0.3)	0.930	0.1 (−0.2–0.4)	0.610	0.0 (−0.4–0.3)	0.848
Female sex (vs. male)	3.8 (−3.8–11.5)	0.324	2.0 (−5.5–9.6)	0.599	3.8 (−4.5–12.1)	0.368
NSP size* **						
• <5 mm						
• 6–15 mm	16.1 (−7.4–39.6)	0.179	12.7 (−10.7–36.0)	0.285	5.6 (−17.9–29.0)	0.640
• >15 mm	21.1 (−2.0–44.2)	0.073	12.3 (−11.2–35.9)	0.303	6.6 (−17.0–30.2)	0.580
Posterior position (vs. anterior)	9.0 (1.6–16.4)	**0.018**	6.4 (−2.0–14.7)	0.133	7.4 (−1.5–16.3)	0.104
Psychiatric comorbidity	15.5 (7.7–23.2)	**<0.001**	-	-	11.8 (3.1–20.5)	**0.008**

Abbreviations: *NSP* nasal septal perforation. *SD* Standard Deviation. *CI* Confidence Interval. Statistical significant differences ($p<0.05$) in bold

*Multivariable A: adjusted for age, sex, perforation size and position. **Multivariable B: additionally adjusted for phychiatric comorbidity. ***Size classified as Bansberg et al. [12]

Self-reported cocaine use may be affected by recall bias or social desirability, potentially underestimating true prevalence. Toxicological validation was not performed in all cases. A detailed physical examination, including assessment of the nasal pyramid, sinonasal cavity and adjacent anatomical structures such as oral cavity, should be mandatory in all patients with cocaine-induced NSP to exclude any synchronous lesions, such as saddle nose deformity or palate perforation [3, 4]. While the Nitro et al. classification, based on lesion prevalence, offers useful framework for standardizing the CMIDL presentations, our findings revealed a higher prevalence of ethmoidal and middle-superior turbinate involvement (Grade 3) compared to palatal lesions (Grade 2B) within our study population [8].

Moreover, CIMDL has been characterized within a limited subgroup of cocaine users. It is important to consider the differential diagnosis of CIMDL, which include: (1) infections such as tuberculosis, tertiary syphilis and mucormycosis, (2) neoplasms (hematologic neoplasms), (3) systemic diseases, and (4) chemical exposures [7, 10, 30, 31].

In addition, various immune-mediated disorders can cause midline destructive lesions, including GPA, rheumatoid arthritis, and mid-facial granuloma among others. A multidisciplinary approach involving internal medicine, anatomopathologist, and ENT is crucial for stablishing a definitive diagnosis and guiding appropriate management.

The main limitations of this study are: (1) Only symptomatic NSP patients who were candidates for surgical closure are included in the analysis potentially limiting generalizability. (2) At the time this study was conducted, validated patient-reported outcome measures such as the NOSE-Perf or SEPEQOL scales had not yet been published. As a result, they were not administered to all patients and may impact subgroup comparisons; (3) Patients with autoimmune diseases (GPA or EGPA) were excluded. However, it is worth noting that the vast majority of other studies did not include this subgroup either. We acknowledge that these conditions are often present with multiple sinonasal manifestations, which may amplify sinonasal symptoms severity and have a more pronounced negative impact on QoL. (4) The relatively small number of patients who underwent objective toxicological testing (such as urine or hair analysis) represents a limitation of the study, potentially leading to an underestimation of the true prevalence of cocaine use within the cohort.

Although the anatomical location of the perforation may influence functional outcomes, its classification varies across research groups and is often closely correlated with perforation size, complicating its analysis as an independent variable. Therefore, we consider that this anatomical characteristic should be addressed as a primary focus in future studies, ideally using standardized anatomical criteria.

This study provides a foundation for understanding the population affected by cocaine-induced NSP and promotes the standardization of QoL questionnaires including extra-nasal domains as SNOT-22 among professionals to establish patient-centred care in NSP. Further research, including patients across all grades of CMIDL, is needed to fully characterize this population and its clinical spectrum. In addition, this study highlights the importance of holistic assessment in patients with cocaine-related NSP who suffer greater psychological affectation compared to other aetiologies.

Conclusion

Cocaine-induced NSP is associated with greater negative impact on overall QoL, with psychological domains being particularly affected. As patients with psychiatric comorbidity experience significantly worse quality of life regardless the aetiology, routine assessment of QoL and mental health

in patients with NSP should be standard care to guide both surgical and psychological interventions. These aspects should be systematically assessed using validated QoL instruments. A holistic, multidisciplinary approach is essential to establish an accurate diagnosis prior to initiating any medical or surgical treatment. Future research should aim to evaluate QoL outcomes in NSP of varying aetiologies, as well as to assess treatment efficacy across different patient subgroups.

Declarations

Conflict of interest The authors declare no conflict of interest.

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