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Subtle Olfactory Impairment in Azoospermia: Investigating the Association with Male Hormones

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ABSTRACT

Background: Anosmia accompanies hypogonadism cases, which develop as a result of the deficiency of hormones such as follicule-stimulating hormone (FSH), luteinizing hormone (LH), GnRH. Although the relationship of female sex hormone change with sinonasal functions has been widely discussed in the literature, the number of studies examining males is limited.

Methods: The study group was composed of 30 patients who presented to the infertility unit and were randomized with their consent. The control group consists of 22 patients having 20 million sperms per 1 mL in spermiogram without any sinonasal disease and previous surgical history. Testosterone, FSH, LH, and prolactin levels were measured for all the patients. SNOT-22 questionnaire was administered to assess the sinonasal functions, and Connecticut (CCCRC) smell test was conducted to assess the MCT and sinonasal function for all the patients.

Results: In the present study, 52 (30 patients in the azoospermia group and 22 patients in the control group) male patients were involved. When compared to the control group, the azoospermia group had significantly lower scores in smell identification and a lower mean smell score ($P = .006$ and $P = .021$, respectively). Statistically significant and mid-level relationship was found between LH and smell identification score in the azoospermia group ($r = 0.458$; $P = .016$).

Conclusion: The sinonasal functions of the azoospermia group followed up with the diagnosis of infertility were negatively affected. Since there was a statistically significant relationship between LH and sinonasal function and the number of studies carried out on male sex hormones in literature is limited, more comprehensive studies are needed.

Keywords Anosmia, sinus pathology, smell, smell disorder

Introduction

Azoospermia, which refers to the absence of sperm in the ejaculate, is observed in 1% of all males and 10-15% of males having infertility problems.[1] In cases such as cystic fibrosis (CF), Young syndrome, and immotile cilia syndrome, infertility and upper airway pathologies might accompany due to ciliary clearance dysfunction.[2] A mutation in Ka1 (Xp22.3) localized in the long arm of Chromosome X causes a degradation in GnRH secretion from the hypothalamus. Hypogonadotropic hypogonadism, anosmia, and male infertility develop as a clinical reflection.[3] Other than genetic pathologies, the relationships between infertility and sinonasal disorders were not sufficiently studied in the literature.

Nasal mucociliary clearance is an important defense mechanism of nasal respiratory epithelium. It is assumed that hormonal changes affect nasal mucosa activity. It was reported that variations in the sex hormones in females were one of the factors influencing the activity of nasal mucosa.[4] However, in the literature, there are very few studies examining the effects of male sex hormones on the nasal mucosa.[5] For this reason, hormone levels and sinonasal functions (SNOT-22, smell tests, and mucociliary clearance time) of patients followed-up due to infertility were compared to the control group.

Methods

The present prospective randomized study involved the patients, who were followed up with the diagnosis of azoospermia in the infertility branch of the Antalya Training and Research Hospital of Obstetrics and Gynecology between June 2021 and June 2022. Ethics committee approval was obtained from the Clinical Research Ethics Committee of Antalya

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Training and Research Hospital before the study (05/06/2021-Decision Nr. 6/1). All the patients were informed about the study and their written consents were obtained. The patients with CF, Kartagener, Young syndrome, and defined genetic disorder, sinonasal surgery and trauma history, and comorbid sinonasal disorder (polyp, allergic rhinitis, and malignancy), with a diagnosed endocrinological disease (such as a thyroid disorder) and using medication (such as topical sprays, antihistaminic, steroid, and antidepressant) were not involved. Individuals with established genetic etiologies of azoospermia (e.g., Klinefelter syndrome, Y-chromosome microdeletions) or anatomical obstructive causes were excluded from the study; therefore, the underlying etiologies of the azoospermia cases in our cohort remain undetermined. The control group consists of patients having 20 million sperms per 1 mL in spermiogram and no comorbid sinonasal disease or previous surgical history. There were 22 volunteer participants in the control group.

Ear-nose-throat examinations and endoscopic nasal examinations of all the participants were recorded, as well as their detailed histories. The Turkish version of SNOT-22 was used for all participants in order to assess the sinonasal quality of life.[6]

Smell Test

Sinonasal function changes in the azoospermia and control groups were investigated using the Connecticut (CCCRC) smell test kit. Smell threshold, smell identification, and mean total scores of patients were calculated for each test material. Connecticut (CCCRC) smell test is a method, which reflects the sinonasal function and has a validated reliability for Turkish society. Diluted butanol solutions were prepared for the butanol threshold test and aligned between 0 and 9; 0 representing the highest concentration and 9 representing the highest dilution rate. The strongest concentrate (Bottle 0) contained 4% butanol diluted with distilled water. In the following bottles (bottles 1-9), the ratio was 1:3 butanol/distilled water. The patients were provided with two bottles, which had the same appearance. However, one of them had distilled water and the other one contained diluted butanol concentrate. The patients were instructed to close one of their nostrils and place the tip of the bottle 3 cm below the other nostril. After each patient accurately identified the same butanol concentration consequently, the score was recorded for that nostril. Then, the other nostril was tested separately, and the final score was calculated as the average of the scores of the two nostrils. Scores range between 0 and 9 points, but the scores of 7 points or higher were recorded as 7 points per CCCRC test. As the identification test, the smells of CCCRC, which were reported to be suitable for use in Turkish society by Veyselleret al,[7] were used. Volunteers were given Vicks, soap, peanut butter, naphthalin, cacao, coffee, cinnamon, and baby powder in opaque bottles. The ability to identify Vicks indicates a strong trigeminal nerve function; its material was easily identified by all the participants and the scores were not included in the final score. The average of two nostrils was calculated and all the scores between 0 and 7 were recorded. To determine the smell capacity of each patient, CCCRC mean total smell score is calculated by averaging the butanol threshold score and the smell identification score.[8]

Measurement of Mucociliary Clearance Time

In the control and azoospermia groups, the participants were asked to sit straight in the polyclinic between 9 and 11 AM (23˚C, 60% humidity) and 1 × 1 cm 5 mg saccharin was placed on the lower conca medial of patients' right nasal passage by using a forceps. Time measurement was performed using a chronometer. During this period, patients were asked to not sneeze, sniff, or wipe their noses. The chronometer was stopped when patients felt a taste change and the time was recorded.

Laboratory Analysis

After 3-day sexual fasting, sexual fasting, the seminal fluids were taken from the participants and spermiogram was performed in the andrology laboratory. The inclusion criterion for the control group was 2 million sperms in 1 mL sample and the participants were divided into two groups as azoospermia and healthy. Peripheric blood samples were taken from all the participants and testosterone, follicule-stimulating hormone (FSH), luteinizing hormone (LH), and prolactin levels were measured in the biochemistry laboratory of our hospital by using the chemiluminescence immune-assay method with Beckman Coulter Dxl 800 device. The reference ranges for the hormonal parameters evaluated in the study were as follows: Testosterone 1.75-7.81 µg/L, LH 1.24-8.62 IU/L, FSH 1.27-19.26 IU/L, and Prolactin 2.64-13.13 ng/mL.

Statistical Analysis

Categorical variables are expressed as frequency (n) and percentage (%), whereas continuous variables are expressed as mean, standard deviation (SD), median, and interquartile range (IQR: 25-75, percentiles) values. Normal distribution assumption was tested using Shapiro–Wilk's test. The relationship between categorical variables was analyzed using Pearson's chi-square test and Fisher's exact test. The analysis of the difference between continuous variables of two independent groups was performed using the Mann–Whitney *U*-test and the independent *t*-test. The relationship between hormones and scores among the azoospermia patients was determined using Spearman's correlation test. All the analyses were performed using IBM SPSS 23.0 package software (SPSS Inc.; Chicago, IL, USA).The statistical significance level was set at $P < .05$.

Results

In the present study, 52 male volunteers (30 in the azoospermia group and 22 in the control group) were included in total; the mean age was 30.88 ± 6.53 years and 71.2% of participants were married. Of the patients, 25% had comorbidities (diabetes, hypertension, duodena lulcer, Meniere, anchylosing spondylitis) followed-up previously, 11.5% had medication use (oral diabetic, nonsteroid inflammatory medication, proton pump inhibitor), 51.9% had previous surgery history (varicocele surgery, inguinal hernia, appendectomy, tonsillectomy, and vocal cord polyp surgery), and 15.4% had previous varicocele history. The percentages of active smokers were 53% in the azoospermia group and 36% in the control group.

Although the previous surgery history was more prevalent among patients with azoospermia, the difference was not statistically significant ($P = .173$). The percentage of patients with varicocele history was higher in the azoospermia group (26.7%

and 0%; $P = .015$). The azoospermia group had higher median LH and FSH values ($P = .001$ and $P = .019$) (Table 1).

Comparing the scores of patients by the presence of azoospermia, it was determined that smell identification and mean smell score of azoospermia patients were significantly lower ($P = 0.006$ and $P = 0.021$) (Table 2).

It was found that there was a statistically significant and medium-level relationship between LH and smell identification score of azoospermia patients ($r = 0.458$; $P = .016$; Figure 1).

Moreover, although a weak relationship was found between prolactin hormone and smell identification, the relationship was found to be statistically non-significant ($P = .064$; Figure 2). Analysis of the patient cohort showed that LH levels were below the lower reference limit in 2 patients, and Testosterone levels were below the lower limit in 2 patients. No patients exhibited values below the lower limit for FSH or prolactin, and none of the hormone measurements exceeded the upper reference limits.

The correlations between hormones and sinonasal scores of azoospermia patients are presented in Table 3. Comparing the scores of azoospermia patients by smoking status, it was determined that there was no relationship between smoking and scores ($P > .05$) (Table 4).

In the present study, it was found that the smell identification and mean smell scores of azoospermia patients were statistically significantly lower in comparison to the control group, whereas there was no difference between the groups in terms of mean MCT and SNOT-22 scores. In the literature, the authors couldn't find a study examining the relationship between azoospermia and sinonasal function. Moreover, given the results achieved in the present study, there was a correlation between male LH hormone and smell scores. In literature, although the relationship between female sex hormones and sinonasal functions has been emphasized in many studies, the number of studies examining male sex hormones and sinonasal functions is limited.[5] For this reason, the present study aims to discuss the results achieved in comparison to the literature.

Azoospermia can be classified into three groups as hypothalamic–pituitary dysfunction, primary quantitative spermatogenic disorders, and urogenital tract obstructions.[9]

Table 1. Patient Characteristics

		Azoospermia		
Variables	**All Patients (n = 52)**	**No (n = 22)**	**Yes (n = 30)**	***P***
Age (years)				
Mean ± SD	30.88 ± 6.53	30.5 ± 5.29	31.17 ± 7.39	.720[1]
Median (IQR)	30 (26-36)	28.5 (26-35)	32 (26-36)	
Marital status, n (%)				
Single	15 (28.8)	11 (50)	4 (13.3)	**.004[3]**
Married	37 (71.2)	11 (50)	26 (86.7)	
Comorbidity, n (%)	13 (25)	6 (27.3)	7 (23.3)	.746[3]
Medication use, n (%)	6 (11.5)	4 (18.2)	2 (6.7)	.382[4]
Smoking,n (%)	26 (50)	8 (36.4)	18 (60)	.173[3]
Previous surgery, n (%)	27 (51.9)	9 (40.9)	18 (60)	.173[3]
Varicocele history, n (%)	8 (15.4)	0 (0)	8 (26.7)	**.015[4]**
LH				
Mean ± SD	5.07 ± 5.03	3.22 ± 1.43	6.37 ± 6.17	**.001[2]**
Median (IQR)	4.08 (2.88-5.58)	2.89 (2.24-4.12)	4.98 (3.59-6.17)	
Prolactin				
Mean ± SD	7.72 ± 2.38	7.78 ± 2.36	7.67 ± 2.44	.876[1]
Median (IQR)	7.39 (5.98-9.37)	7.39 (5.98-10.17)	7.45 (5.83-9.37)	
Testosterone				
Mean ± SD	3.65 ± 1.37	3.8 ± 1.4	3.54 ± 1.36	.558[2]
Median (IQR)	3.4 (2.69-4.81)	3.4 (2.69-4.96)	3.36 (2.71-4.07)	
FSH				
Mean ± SD	5.24 ± 3.46	3.92 ± 2.15	6.13 ± 3.91	**.019[2]**
Median (IQR)	4.33 (3.22-6.4)	3.7 (2.13-5.03)	4.7 (3.68-7.28)	

LH, luteinizing hormone.
[1]Independent *t*-test, [2]Mann-Whitney *U*-test, [3]Pearson chi-square test, [4]Fisher's exact test.

Table 2. Comparison of the Scores by the Groups

	Azoospermia			
Variables	**All Patients (n = 52)**	**Yok (n = 22)**	**Var (n = 30)**	***P***
Butanolsmell threshold				
Mean ± SD	5.85 ± 1.14	5.95 ± 1.21	5.77 ± 1.1	.450
Median (IQR)	6 (5-7)	6 (5-7)	6 (5-7)	
Smell identification				
Mean ± SD	5.75 ± 1.06	6.23 ± 1.02	5.4 ± 0.97	**.006**
Median (IQR)	5 (5-7)	7 (5-7)	5 (5-6)	
Mean smell score				
Mean ± SD	5.8 ± 0.88	6.09 ± 0.92	5.58 ± 0.79	**.021**
Median (IQR)	6 (5-6.5)	6 (5.5-7)	5.5 (5-6)	
MCT				
Mean ± SD	10.6 ± 5.96	9.64 ± 5.21	11.3 ± 6.45	.377
Median (IQR)	9.5 (6-13)	8 (6-13)	10 (6-13)	
SNOT-22				
Mean ± SD	30.38 ± 19.73	31.86 ± 22.33	29.3 ± 17.91	.810
Median (IQR)	26 (13.5-44.5)	27 (17-45)	26 (12-44)	

Mann–Whitney *U*-test.

Hypogonadotropic hypogonadism, hyperprolactinemia, and endogen androgen surplus are the reasons for spermatogenic failure. Some syndromes, in which sinonasal disorders and spermatogenic disorders have been observed together, have been reported in the literature.

Primary ciliary dyskinesia is an autosomal recessive disease characterized by dysmotility, immotility, or absence of cilia. The failure in ciliary movement causes mucociliary clearance and results in clinically recurrent or persistent sino-respiratory infections and infertility.[10] Young syndrome and Kartagener syndrome are rare hereditary diseases. The authors related their finding to the strong similarity of arrangement and morphology of ciliary columnar epithelium in efferent seminiferous tubules to the nasal and respiratory epithelia.[11] It suggests that the comorbidity of sinonasal disorders and azoospermia might also be sporadic, besides the genetic causes. In literature, the changes in female sex hormones in nasal mucosa after a menstrual cycle, pregnancy, and menopause have been histochemically reported.[12] In the present study, MCT was within the normal limits in azoospermia and control groups and there was no statistically significant difference between the two groups. Moreover, it was also determined that MCT had no correlation with LH, FSH, testosterone, and prolactin levels in the present study. The saccharin test provides a practical screening measure of mucociliary clearance; however, its reproducibility is limited, and gold-standard techniques such as radioisotopic mucociliary clearance measurement and high-speed video microscopy offer more objective and sensitive assessment of ciliary function. To the best of our knowledge,

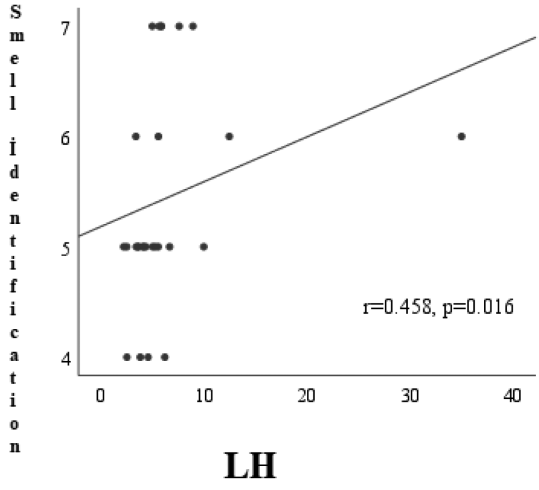

Figure 1. The correlation between luteinizing hormone (LH) and smell identification among azoospermia patients.

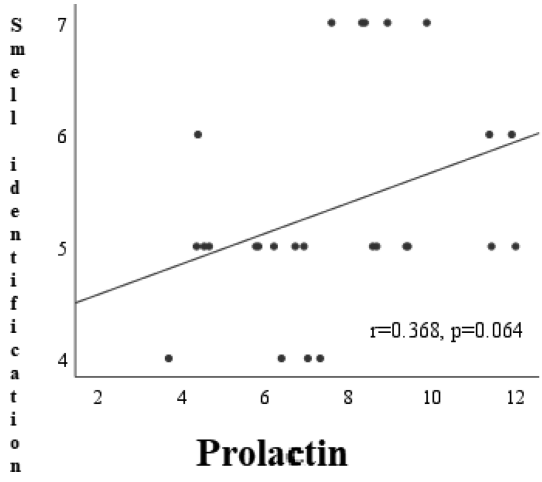

Figure 2. The correlation between prolactin and smell identification among azoospermia patients.

Table 3. Correlations Between Hormones and Scores Among Azoospermia Patients

	LH		Prolactin		Testosterone		FSH	
Variables	**r**	***P***	**R**	***P***	**R**	***P***	**r**	***P***
Butanol smell threshold	−0.063	.754	−0.104	.612	−0.007	.971	−0.231	.238
Smell identification	0.458	**.016**	0.368	.064	0.242	.215	0.123	.533
Mean smell score	0.246	.216	0.092	.656	0.127	.520	−0.121	.540
MCT	−0.230	.248	−0.295	.143	−0.187	.340	−0.044	.825
SNOT-22	0.169	.401	−0.182	.373	0.168	.392	−0.200	.308

Spearman's correlation test.
FSH, follicle-stimulating hormone; LH, luteinizing hormone.

the present study is the first study in the literature examining male hormones and MCT.

Smoking is considered to be one of the most important factors causing male infertility and it also has negative effects on the respiratory tract. In their study, Kılıç et al[7] measured the mucociliary clearance among passive smoke exposure by using a saccharine test and they reported that, in comparison to the non-smoker group, they had longer test times. In their meta-analysis, Ajmani et al[13] reported that smokers had significantly higher olfactory dysfunction possibility in comparison to those, who haven't smoked before. In the present study, there was no statistically significant difference by smoking status. Furthermore, there was no statistically significant difference between smokers and non-smokers in the azoospermia group in SNOT-22, MCT, and smell tests. In particular, the negative effect observed in the smell test in the azoospermia group suggested that it might be because of the hormonal change, independently from the smoking status.

Table 4. Comparison Between Scores of Azoospermia Patients by Smoking Status

	Smoking		
Variables	**No (n = 12)**	**Yes (n = 18)**	**P**
Smell threshold			
Mean ± SD	5.75 ± 1.14	5.78 ± 1.11	.950
Median (IQR)	6(5-7)	6(5-7)	
Smell identification			
Mean ± SD	5.5 ± 0.9	5.33 ± 1.03	.573
Median (IQR)	5(5-6)	5(5-6)	
Mean smell score			
Mean ± SD	5.62 ± 0.61	5.56 ± 0.91	.728
Median (IQR)	5.75(5-6)	5.5(5-6)	
MSC			
Mean ± SD	10.42 ± 5.53	11.89 ± 7.09	.733
Median (IQR)	10.5(5.5-13)	10(8-14)	
SNOT-22			
Mean ± SD	26 ± 14.67	31.5 ± 19.87	.692
Median (IQR)	25 (12-37.5)	26 (19-45)	

Mann-Whitney *U*-test.

SNOT-22 is considered as the reference survey used in measuring the quality of life in sinonasal disorders and especially in chronic rhinosinusitis. High scores indicate poor prognosis. While SNOT-22 scores were observed to increase during 1-year follow-up of postmenopausal patients and mucociliary clearance times prolonged, the correlation with changes in male sex hormones has not been examined.[14] Given the results achieved in the present study, no statistically significant difference was found between azoospermia and control groups. The SNOT-22 questionnairemay be subjectto self-reportbiasand can be influencedbynon-nasalfactorssuch as fatigue, mood, and over all health status, which limits it sensitivity for detecting isolated olfactory impairment. Moreover, there was also no statistically significant difference between hormone levels of azoospermia patients (testosterone, LH, FSH, and prolactin) and SNOT-22 scores.

Smell identification is the most refined sense based on chemoreception. The recipient organ, the olfactory epithelium, consists of specialized neurons localized at a very peripheric position.Nasal epithelium is a perfect example for hormone-related neuroplasticity.[4] Kallmann syndrome is related with hypogonadotropic hypogonadism and anosmia. Among these patients, the olfactory bulbus is either not developed sufficiently or absent. During embryonic life, there is a close topographic relationship between peripheric olfactory system and neuroendocrine GnRH cells.[3] In the male reproduction system, there is a classic endocrine feedback mechanism. Hypothalamic GnRH stimulates LH and FSH stimulating the testicles. LH also stimulates the secretion of testosterone by Leydig cells. Follicle-stimulating hormone and testosterone are the main hormones directing the spermatogenesis**.** In general, there is a reverse relationship between FSH and spermatogonia levels. Follicule-stimulating hormone (FSH) level increase when there is no or significantly decreased level of spermatogonium, whereas FSH level is within the normal limits in case of a normal number of spermatogonia.[15] In literature, the number of studies on male hormone levels and smell is limited. In the present study, the butanol threshold value was used for smell analysis, whereas subjective assessment of smell was performed using CCCRC olfactory test. Given the results achieved, it was determined that, when compared to the control group, the azoospermia group had significantly lower scores in smell identification and mean score ($P = .006$, $P = .021$).In their case study published in the year 1994, Diez et al[16] examined an azoospermia patient with isolated FSH deficiency and the authors reported that the patient had a normal sense of smell. Indeed,

all of the patients reported until now with LH deficiency, hypogonadism and infertility have no clinically smell abnormalities, where as before or after treatment.[17,18] In the present study, no correlation was found between FSH level and smell threshold and smell identification levels of patients in the azoospermia group ($r = -.231$, $P = .238$, $r = 0.123$, $P = .533$). Besides that, while there was a statistically significant and medium-level relationship between LH and smell identification in the azoospermia group, there was a weak relationship between prolactin hormone and smell identification and the difference was not statistically significant. It should be kept in mind that correlations may not imply causation.

Although the CCCRC test is validated for use in our population, it is not considered a global gold-standard olfactory assessment; therefore, future studies should incorporate internationally standardized tests such as UPSIT or Sniffin' Sticks (TDI) to more comprehensively characterize and confirm subtle olfactory deficits in azoospermic patients.

Gamsakhurdashvili et al[19] (2021) evaluated 60 healthy individuals to investigate the relationship between olfactory function and sex hormones. They measured salivary levels of 17-β estradiol, progesterone, and testosterone and assessed olfactory performance using the Düsseldorf Odor Discrimination Test. Although no significant association was found between hormone levels and odor discrimination, free-cycling women demonstrated higher performance compared to the other groups.

Olfactory function is also influenced by thyroid hormones. In a prospective study conducted by Deniz et al[20] in 2016, 32 patients with primary hypothyroidism were compared with 31 healthy controls, and the authors demonstrated that smell threshold, smell discrimination, and smell identification scores were significantly reduced in hypothyroid individuals. Following L-thyroxine treatment and restoration of euthyroid status, a marked improvement was observed in all smell test parameters. These findings suggest that thyroid hormone deficiency may impair smell function through both peripheral effects on the olfactory epithelium and central mechanisms, and that hormonal correction can largely reverse this dysfunction. In our study, patients with a previously diagnosed endocrinological disease who were receiving treatment were excluded; however, thyroid function test results were not evaluated separately for all patients within the scope of the study. Further large-scale clinical studies are recommended to better elucidate this relationship.

The effects of sex hormones on the nasal mucosa have generally been presented in rat studies. Pietras and Moulton reported that super physiological testosterone levels improved the sense of smell among the female rats with gonadectomy, but the male rat castration didn't have an effect.[21] The first study examining the male sex hormones and sinonasal functions was carried out by Kırgezenet al. In their study, Kırgezenet al[5] reported that low testosterone levels negatively affected the smell thresholds and smell identification scores of males. However, due to the limited sample size and insufficient hormonal stratification in the study by Kirgezent et al (2021), their findings should be interpreted with caution when compared across different patient groups. In the present study, it was determined that there was no statistical correlation between testosterone levels and sinonasal functions of azoospermia patients.

Conclusion

While some syndromes incorporating sinonasal disorders and spermatogenic disorders at the same time have been widely discussed in the literature, the studies examining the sporadic cases are limited. In the present study, no difference was found between MCT and SNOT-22 scores of azoospermia patients, whereas sinonasal functions were negatively affected in the azoospermia group. In the group of azoospermia patients followed up due to infertility, there was a statistically significant and mid-level relationship between LH and smell identification. Since the number of studies examining the male sex hormones and the sinonasal functions is limited in the literature, it is thought that more comprehensive studies examining this subject are needed.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of Antalya Training and Research Hospital (Approval No: 6/1, Date: 05/06/2021).

Informed Consent: Verbal and written informed consents was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

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Declaration of Interests: The authors have no conflicts of interest to declare.

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