The Association Between Obstructive Sleep Apnea and Periodontitis in Chinese Male Adults: A Cross-Sectional Study

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Abstract

Background: Obstructive sleep apnea (OSA) is characterized by complete or partial upper airway obstruction during sleep. Periodontitis is an infectious and inflammatory disease of periodontal tissues. Recent studies suggested that both diseases could alter the host response synergistically by sharing same inflammatory pathways. The study aimed to investigate the association between OSA and periodontitis in Chinese male adults.

Methods: This was a cross-sectional study of 93 male adults recruited from a dormitory compound and examined between June and September 2019. All participants were diagnosed OSA using the apnea–hypopnea index (AHI) with a portable, overnight polysomnography (PSG). Periodontal examinations were conducted the same day before PSG measuring: 1) mean probing depth (PD); 2) mean clinical attachment level (CAL); and 3) percentage of sites with bleeding on probing (BOP). An objective nasal airway resistance assessment was also practiced before PSG to reflect on the mouth breathing during sleep.

Results: In all, 43.0% participants had periodontitis, 20.4% had OSA and 32.5% of those diagnosed with periodontitis were in combination with OSA. OSA was positively associated with periodontitis (odds ratio (OR) = 3.77, 95% confidence interval (CI) = 1.29 ~ 11.07). The OSA group showed a significantly higher BOP (p = 0.034) and CAL (p = 0.046), but there was no statistically significant difference of PD (p = 0.090) between the two groups. Correlation analysis showed a low but positive correlation between OSA severity and periodontitis severity classifications and periodontal parameters. Further regression analysis identified Lowest oxygen saturation (SaO_2) (OR = 0.894, 95% CI = 0.842 ~ 0.949) to be significantly associated with the prevalence of periodontitis.

Conclusions: A significant association was observed between OSA and periodontitis. And increasingly severe OSA might increase the severity of periodontitis. Hypoxia parameters might have a predicting effect of periodontitis, suggesting hypoxia related systematic inflammation may be the possible explanation of the association.

1 Introduction

Obstructive sleep apnea (OSA) is a common disease with an estimated prevalence of 3% to 7% worldwide, and is the most common type of sleep disordered breathing.[1] OSA is characterized by periodic and repetitive partial or complete collapse of the upper airway during sleep, which could result in apnea and/or hypopnea. Consequently, that status causes hypoxia, hypercapnia, sleep fragmentation, increased sympathetic activity and altered immunity.[2, 3] Recent studies revealed more molecular changes in OSA patients including gene-related local and systemic inflammation towards oxidative stress and intermittent hypoxia.[4]

Periodontitis is becoming a disease of high prevalence worldwide. Global epidemiologic data shows that severe periodontitis was the sixth most prevalent condition of all disease conditions and that it affected

10.8% of the population or 743 million people aged from 15 to 99 worldwide.[5] Periodontitis is a chronic infectious and inflammatory disease caused by bacterial pathogens that could initiate the inflammatory response of the host.[6] The synergetic effect of both bacterial toxins and host immune response result in the destruction of the periodontal tissues, clinically manifested as loss of alveolar bone, progressive periodontal attachment lost, pocket formation and, ultimately, tooth loss.[7]

It's reported the resultant inflammatory response of OSA could enhance the inflammation status of currently existing diseases, or initiate other inflammatory diseases in people with environmental, behavioral or genetic risk factors[8], for example, periodontitis. Since subjects with OSA are observed to have elevated levels of inflammatory cytokines or mediators including C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), intrerleukin-1B (IL-1B), interleukin-6 (IL-6)[9], which also play key roles in the pathogenesis of periodontitis[7], a potential association between OSA and periodontitis is highly suggested.

Gunaratnam et al firstly investigated the association between OSA and periodontitis, finding the prevalence of periodontitis in the OSA group (N = 66) is four times that of historical controls.[10] But Seo et al (N = 687)[11], Loke et al (N = 100)[12], Keller et al (N = 21963)[13] then reached conflicting conclusions, making whether OSA is significantly associated with periodontitis still controversial. The probable reason is current studies set historical-based or population-based controls, which involved with certain intervals between the diagnosis of OSA and periodontal examinations. While both diseases are highly sensitive to inflammatory status of the body,[8, 14] alternations during intervals such as clinical treatment, environmental exposure and habit could affect the relevance.

In the cross-sectional study, we aimed to determine whether the association between OSA and periodontitis exists and, if possible, to further investigate the association between the severity of OSA and that of periodontitis in Chinese male adults.

2 Materials And Methods

2.1 Participants

The cross-sectional study was approved by the Ethics Committee of Air Force General Hospital, China, and all work was carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki). It was conducted in an establishment's male dormitory compound under full-close management in Beijing during June to August, 2019.

The calculation of sample size was performed according to the method reported by Dupont et al.[15] Based on previous publications[10-13], the Odds Ratio (OR) of OSA in periodontitis is approximately 2. So, we set twofold increase in the prevalence of periodontitis in OSA group compared with control group as clinical important, and the sample size of 90 reached study power >90% with a two-sided significance test α = 0.05. Due to the relevance of smoking and periodontitis[16], the inclusion criteria were: 1) Age > 18 years old; 2) male; 3) self-reported without alcohol abuse; 4) self-reported never smoking or having

quitted smoking for more than 6 months. Considering the complication of the study, we initially recruited 135 participants from all residents at the dormitory compound using simple random sampling method.

All participants signed informed consent after elucidating study details. The exclusion criteria are set as follows: 1) lack complete medical records; 2) have systematic diseases that could affect OSA and periodontitis; 3) have specific use of medication that could affect OSA and periodontitis; 4) currently diagnosed and/or under treatment of OSA or periodontitis; 5) with genetic susceptibility; 6) have special diet preference. Among the originally included 135 participants, 119 underwent medical check within one year and provided with complete medical history. The elimination reasons were: 1) Blood-system diseases (n = 1); 2) Uncontrolled severe hypertension, SBP/DBP > 180/110mmHg (n = 1); 3) Diabetes mellitus (n = 1); 4) Medication history of phenytoin, calcium channel blocker, cyclosporin (n = 2); 5) Currently under treatment of OSA (n = 1) or periodontitis (n = 2).

Before further examinations, participants were measured body mass index (BMI), completed a questionnaire related to sleep factors and life habits and passed the pre- polysomnography (PSG) check. Two participants voluntarily quitted the periodontal examination and the following nasal airway resistance (NAR) assessment, and one failed to record PSG data due to connect failure, leaving a total of 93 completed the whole study. (**Fig. 1**)

All participants were then divided into OSA group and non-OSA group according to the PSG diagnosis, with a final total 19 in the OSA group and 74 in the non-OSA group. Additional participants demographics are presented in Table 1. Almost all participants could be considered normal range (59.1% had BMI from 18.5 ~ 24.9 kg/m^2) or pre-obese (39.8% had BMI from 25 ~ 29.9 kg/m^2) according to the WHO criteria for obesity.[17] A higher proportion of pre-obese participants were discovered in the OSA group, and t-test also indicated significantly higher AHI ($p < 0.001$) than the non-OSA group. Other demographic characteristics including age ($p = 0.505$) and alcohol use were basically matched between the two groups. (**Table 1**)

2.2 Diagnosis of OSA

All participant underwent overnight PSG with portable in-lab PSG devices (SOMNOscreen^{TM} plus, SOMNOmedics GmbH, Randersacker, Germany) in the dormitory compound.

Before the PSG, each participant had heart rate and blood pressure recorded. Accordingly, electrocardiogram, central electroencephalogram, bilateral electrooculograms, submental electromyogram, bilateral anterior tibialis electromyogram was taken. Chest and abdominal movement were traced by respiratory effort bands. Body position recorded by pressure sensors. Oronasal airflow was measured by oronasal thermal sensors. And oxygen saturation (SaO_2) was measured by a pulse oximeter. In the morning, heart rate and blood pressure were recorded again within one hour after the rising.

Sleep stages, respiratory events, arousals and body movements were manually scored by one experienced sleep technician according to the manual of the American Academy of Sleep Medicine (AASM).[18] To score a respiratory event as an apnea, the followings should be met: 1) There was a drop in the peak signal excursion by ≥ 90% of pre-event baseline using the oronasal thermal sensor. 2) The duration of the ≥ 90% drop in sensor signal was ≥10 seconds. A hypopnea was scored when: 1) The peak signal excursions dropped by ≥ 30% of pre-event baseline using nasal pressure sensor. 2) The duration of the ≥ 30% drop in signal excursions was ≥ 10 seconds. 3) There was ≥ 3% oxygen desaturation from pre-event baseline or the event is associated with an arousal. If a portion of a respiratory event that would meet both the criteria for a hypopnea and an apnea, the entire event should be scored as an apnea. The apnea–hypopnea index (AHI) is defined as apneas plus hypopneas per hour of sleep. In adults, OSA is diagnosed when AHI score is ≥ 5. OSA severity classification is based on AHI scores, as mild OSA is diagnosed as an AHI of at least 5 to 15, moderate OSA as AHI >15 to 30, and severe OSA as AHI >30.[18] The lowest SaO_2 and oxygen desaturation index (ODI) were also recorded as sensitive markers of intermittent hypoxia.[19] ODI is the average number of desaturation episodes per hour, where desaturation is defined as a decrease in the mean SaO_2 of ≥4% that lasts for at least 10 seconds over the last 120 seconds.[18]

2.3 Periodontal Examination

The periodontal examination was practiced for each participant by one experienced dentist in the same day immediately before PSG. The examiner was full blinded to the participants' correlative clinical data in the examination phase and masked to the patient's OSA conditions in the data collection phase.

Based on these available data and experience from clinical practice[20], the following "Ramfjord Teeth" were examined to indicate the full-mouth periodontal conditions: #3 (maxillary right first molar), #9 (maxillary left central incisor), #12 (maxillary left first bicuspid), #19 (mandibular left first molar), #25 (mandibular right central incisor), #28 (mandibular right first bicuspid).[21] If a Ramfjord tooth was missing, a substitute tooth (#2, #8, #13, #18, #24, #29) was selected as Fleiss et al suggested.[22]

Periodontal parameters including: 1) probing depth (PD), 2) clinical attachment level (CAL), 3) bleeding on probing (BOP) were recorded of six points (mesio-buccal, mid- buccal, disto-buccal, disto-lingual, mid-lingual, mesio-lingual) each tooth in special designed periodontal charts. All measurements were obtained using a Williams style periodontal probe (Hu-Friedy, Inc., Chicago, IL, USA) and lengths were rounded up or down to the nearest millimeter. CAL is determined as follows: 1) If the gingival margin is located on the anatomic crown, CAL is PD minors the distance from the gingival margin to the cemento-enamel junction (CEJ). 2) If gingival margin coincides with the CEJ, CAL equals to PD. 3) If the gingival margin is located apical to the CEJ, CAL is PD plus the distance between the CEJ and the gingival margin. BOP was recorded as either present or absent within 30 seconds of probing.[23]

In diagnosis of periodontitis, we used the clinical case definitions proposed by the Centers for Disease Control and Prevention (CDC), in collaboration with the American Academy of Periodontology (AAP)

working group for use in population-based surveillance of periodontitis. Periodontitis is defined as two or more interproximal sites with CAL ≥ 3mm (not on same tooth) and two or more sites interproximal sites with PD ≥ 4mm (not on same tooth) or one site with PD ≥ 5mm. And periodontitis severity classification is defined as: 1) severe periodontitis, when two or more interproximal sites with CAL ≥ 6 mm (not on same tooth) and one or more interproximal site(s) with PD ≥ 5mm; 2) moderate periodontitis, when two or more interproximal sites with CAL ≥ 4 mm (not on same tooth) or two or more interproximal sites with PD ≥ 5 mm (not on same tooth); 3) mild periodontitis, when the patient could not be diagnosed as neither moderate nor severe periodontitis.[24]

To evaluate the severity of periodontitis more accurately, the mean PD, mean CAL and percentage of BOP sites were calculated for each participant.

2.4 Nasal Airway Resistance

It's suggested that OSA patients may also have a tendency of mouth breathing[25, 26], and a former cross-sectional study concluded a possible association between mouth breathing and gingivitis in children.[27] Then we attempted to record the mouth breathing rate of the study sample. However, surveying mouth breathing by questionnaires could be greatly affected by self-knowledge biases. So, we used NAR as a proxy, as NAR was proved to be an accurate predictor for mouth breathing and subjects with normal nasal airway resistance breathe almost completely through the nasal airway during sleep.[28]

To objectively assess the NAR, both active anterior rhinomanometry (Rhinomanometer NR6; GM Instruments Ltd., Irvine, UK) and rhinospirometry (Rhinospirometer NV2; GM Instruments Ltd., Irvine, UK) were performed on each participant after the periodontal examination and before the PSG. NAR was indicated by recording both nasal pressure and inspiratory nasal airflow to reflect the resistant status of the nasal airway when breathing.

The measuring procedures were conducted in accordance with the guidance of the International Committee on Rhinomanometry Standards.[29] For participants, measurements were obtained in a sitting position, after an adaptation time of 20-30 minutes. Each participant was guided to gently clear the nostrils by blowing, and a cotton swab was used to clear the left secretions.

Rhinomanometry was conducted at first. The pressure nozzle attached by an elastic and fitting plug was placed into one nostril and sealed with adhesive tape. Then, a respiratory face mask with a flowmeter was used, thus providing with a transnasal pressure gradient between two sides. The values of pressure gradient (ΔP) and transnasal airflow (V) were monitored by one experienced rhinomanometry technician. Participants were instructed to breathe normally until at least four consecutive inspirations were recorded. The resistance value of the other side of nasal airway was obtained afterwards using the same method.

Rhinospirometry was conducted immediately after the rhinomanometry. The participant was instructed to place the nasal cannulae of the rhinospirometer in seal with the alar rim without deforming the nostril, so

as to avoid any leakage. Then, the participant was told to inhale through the nose and exhale through the mouth in normal breathing rate. The volume of inspired air for each nasal passage was recorded simultaneously in a continuous 20 seconds. The measurements were repeated on two further occasions, and mean volumes were calculated.

Before analyzation, the data was examined to eliminate false breaths. Rhinomanometry measurement was expressed by the formula $R = \Delta p/V$, where we picked measurements at $\Delta p = 150$ Pa according to the recommendation of Standardization Committee on Objective Assessment of the Nasal Airway.[29] Rhinospirometry measurement was presented by the mean volume of left and right nasal passage in the 3 successive measurements.

2.5 Statistical Analysis

All statistical analysis was conducted using IBM SPSS Statistics for MacOS, version 26.0 (IBM Corp., Armonk, N.Y., USA), and a two-sided p-value of <0.05 was considered to be statistically significant. A normality test was practiced on all variances we collected, showing that AHI, PD, BOP, and CAL was basically in line with normal distribution.

To investigate the association between OSA and periodontitis, we set OSA as the risk factor and periodontitis as the outcome. Due to the low prevalence of OSA in our study and the lack of moderate/severe OSA patients, we simplified the OSA classification into two dichotomous variables (Yes/No). Then, periodontal parameters including PD, BOP and CAL were firstly tested between OSA group and Non-OSA group. The differences between the mean values of PD, BOP and CAL were evaluated using the t-test. Then, the χ^2 test was applied to measure the difference in prevalence of periodontitis between variables. And a logistic regression model was used to practice multivariate analysis of periodontitis and exclude confounding factors such as age, BMI and alcohol use.

To identify the possible association between the severity of OSA and that of periodontitis, we took AHI to estimate the severity of OSA according to the recommendation of AASM.[18] Spearman rank correlations were calculated for ordinal variables, and Pearson correlation analysis were used in continuous variances in normal distribution.

Then, to test the possible mechanism that periodontitis was with increased prevalence and severity in OSA patients, we set a binary logistic regression model with five independent variables to calculate ORs and the 95% confidence interval (CI) predicting periodontitis,[30] where AHI, ODI, lowest SaO_2, NAR (indicated by rhinomanometry and rhinospirometry measurements) were included as covariates.

3 Results

3.1 Association Between OSA and Periodontitis

Among the 93 participants, 43.0% participants had periodontitis, 20.4% had OSA and 32.5% of those diagnosed with periodontitis were in combination with OSA. The OSA group shows significantly higher BOP (p=0.034) and CAL (p=0.046) and a marginally higher PD (p=0.090) than the control group. (**Fig. 2**) In addition, when comparing the prevalence of periodontitis between the two OSA groups, the χ^2 test demonstrated a statistically significant difference (OR = 3.77, 95% CI = 1.29 ~ 11.07, p = 0.012). (**Table 2**)

To exclude confounding variables, the age was divided into two groups (24 ~ 29 years and 30 ~ 35 years). Then, age groups, BMI classifications and alcohol use were analyzed as categorical independent variables in a binary logistic regression model predicting periodontitis. The result showed that OSA was independently associated with periodontitis (OR=3.691, 95% CI = 1.256 ~ 10.845, p = 0.018).

3.2 Association between the severity of OSA and that of periodontitis

We analyzed the correlations among AHI absolute and periodontitis classification (none, mild, moderate, severe) and periodontal parameters (PD, BOP, CAL). Spearman analysis indicated that periodontitis classifications was positively related to AHI with an eligible coefficient (r = 0.297, p=0.004). Similarly, small or weak correlations were also found between AHI absolute and BOP (r=0.214, p=0.04) and CAL (r=0.225, p=0.03) using Pearson correlation analysis. Noneligible or marginal correlation was observed between AHI absolute and PD (r = 0.196, p=0.09).

3.3 Logistic Regression Model Testing Risk Factors of Periodontitis

Given the fact that there were 40 participants who have periodontitis, the regression model was efficient to test maximum five predictors of periodontitis in the study sample. In the former statistical analysis, other demographic variables considered as confounding factors such as age and alcohol use were adjusted. Thus, the binary logistic regression model included the continuous variables of hypoxia indicators (AHI, ODI, Lowest SaO_2, NAR) and the binary variables of periodontitis (Yes/No). The Hosmer-Lemeshow goodness-of-fit test showed p = 0.374, which indicated the model has good prediction effect.

As shown in **Table 3**, AHI (OR = 1.184, 95% CI = 0.987 ~ 1.419, p = 0.126) might have a weak association with periodontitis. And the Lowest SaO_2 showed a statistically significant predicting effect for periodontitis (OR = 0.894, 95% CI = 0.842 ~ 0.949, P = 0.002), indicating a negative relation between Lowest SaO_2 and diagnosis of periodontitis.

4 Discussion

The findings of this study were in accordance with previous studies which indicated a significant association between OSA and periodontitis.[10, 11, 13] In other studies, the association measured by OR was relatively weak (less than 2)[11, 13]. In the current study, the OR for OSA and prevalence of periodontitis was stronger (3.69), but with a wider 95% confidence interval. Furthermore, coupled with the weak but positive correlation between OSA severity and periodontitis severity, we supposed that the

association between OSA and periodontitis, as well as the association between the severity of OSA and periodontal status, if any, was weak in this specific study population.

To explain the higher prevalence of periodontitis observed among the OSA patients, Gunaratnam et al firstly suggested the reason might be both diseases sharing several overlapping risk factors.[10] However, we tested and excluded the overlapping risk factors (systematic diseases, smoking, age, BMI and alcohol use) for both two diseases, finding these factors alone were not sufficient enough to explain the elevated periodontitis prevalence in OSA patients. Seo et al then considered mouth breathing as the risk factor for periodontitis[11], as continuous mouth breathing airflow might cause drying of the mouth, which could reduce the self-cleaning ability of oral cavity, lead to formation of bacterial plaque and ultimately cause gingivitis.[6, 31] Thus, we objectively assessed nasal airway resistance to reflect mouth breathing condition during sleep, but no statistically significant association was found. We further investigated several hypoxia parameters and identified Lowest SaO_2, which indicates the intermittent hypoxia status during apnea or hypopnea[32], could effectively predict the prevalence of periodontitis. Previous studies had confirmed that intermittent hypoxia in OSA could selectively activate inflammatory pathways.[8] Plus, the intermittent episodes of hypoxia, particularly the associated episodes of intermittent reoxygenation are important mediators of systematic inflammatory status. Therefore, we suggested that OSA might increase the prevalence and severity of periodontitis by contributing to increase systematic inflammation as a result of intermittent hypoxia.

Considering the sample selection process, previous studies recruited patients from hospitals and clinics[10, 12, 33], or from health database[11, 13]. In this study, we recruited participants from one dormitory compound under full-close management. The distribution of age and BMI is relatively concentrated. And all residents of the dormitory were employed by one establishment, thus had similar occupations, socioeconomic conditions, education experience and life habits. Besides, we set relatively strict inclusion criteria to exclude possible confounders including smoking status, poorly controlled systematic diseases, or any other interfering conditions, thus including a study sample with the least number of patient-related confounders. Therefore, quite homogeneous samples were finally included in the study, which was ideal for investigate possible influencing factors.

This preliminary study had several important limitations. Firstly, the sample size was relatively small in this study compared with previous studies using historical-based or population-based controls. While our findings showed a statistically significant association between OSA and periodontitis, this association measured by OR was weak and with a wide confidence interval. Secondly, the prevalence of moderate and severe OSA recorded in this study was much lower than the prevalence found in the Chinese population as a whole. The narrow distribution of AHI absolute may possibly result in underestimating or ignoring the prediction effect of OSA severity for periodontitis severity. Thirdly, as we included quite homogenous samples in the study, the prevalence of both OSA and periodontitis could’t reflect the genuine distribution of the diseases in the general population. Moreover, considering the study sample mainly consisted of young Chinese male adults with weight of normal range or pre-obese, it might not be

appropriate to extrapolate the conclusions to other population groups with different gender, age, race and body weight.

Thus, adequate consideration should be given when extending the results to the general population. And future studies should evaluate larger and broader study participants in the general population, in order to improve the popularization of the results.

Nevertheless, if in conclusion, a significant association was observed between OSA and periodontitis in this typical homogenous study sample. And increasingly severe OSA might increase the severity of periodontitis. We also discovered that hypoxia parameters might have a strong predicting effect of periodontitis, suggesting hypoxia related systematic inflammation might be the possible explanation of the association.

5 List Of Abbreviations

AAP	American Academy of Periodontology
AASM	American Academy of Sleep Medicine
AHI	apnea–hypopnea index
BMI	body mass index
BOP	bleeding on probing
CAL	clinical attachment level
CDC	Centers for Disease Control and Prevention
CEJ	cemento-enamel junction
CI	confidence interval
CRP	C-reactive protein
IL-1B	intrerleukin-1B
IL-6	intrerleukin-6
NAR	nasal airway resistance
ODI	oxygen desaturation index
OR	odds ratio
OSA	obstructive sleep apnea
PD	probing depth
PSG	polysomnography
SaO_2	oxygen saturation
TNF-α	tumor necrosis factor-alpha

6 Declarations

6.1 Ethics approval and consent to participate

The study was approved by the Ethics Committee of Air Force General Hospital, China and was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Written informed consent was obtained from all individual participants included in the study.

6.2 Consent for publication

And all participants consented to publish their data and examination results.

6.3 Availability of data and materials

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

6.4 Competing interests

The authors declare that they have no competing interests.

6.5 Funding

No funding was received.

6.6 Authors' contributions

Y. L. Chen, H. Gao and X. M. Gao conceived and designed the study strategy. Y. L. Chen conducted the periodontal examination. Y Duan, X.X. Han and Y. Li practiced medical checks, PSG and data collection. Y. L. Chen wrote the manuscript and prepared the tables and figures. H. Gao and X. M. Gao reviewed and edited the manuscript. H. Gao and X. M. Gao were responsible for study supervision. All authors reviewed the manuscript.

6.7 Acknowledgements

Not applicable.

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Tables

Table 1

Demographic characteristics of the study sample grouped by OSA

Demographic characteristics	All Participants (n = 93)	OSA Yes (n = 19)	OSA No (n = 74)	p Value
Age (years) 24 ~ 29 30 ~ 35 Male	28.8 ± 3.0 52 (55.9) 41 (44.1) 93 (100)	28.4 ± 3.2 11 (57.9) 8 (42.1) 19 (20.4)	28.9 ± 2.9 41 (55.4) 33 (44.6) 74 (79.6)	0.505
BMI (kg/m^2) <18.5 18.5 ~ 24.9 25.0 ~ 29.9 Alcohol Use Never Former Current	24.2 ± 2.4 1 (1.1) 55 (59.1) 37 (39.8) 31 (33.3) 7 (7.5) 55 (59.1)	26.1 ± 2.3 0 (0) 5 (26.3) 14 (73.7) 6 (31.6) 1 (5.3) 12 (63.2)	23.7 ± 2.2 1 (1.4) 50 (67.6) 23 (31.1) 25 (33.8) 6 (8.1) 43 (58.1)	< 0.001
Values are shown as mean ± SD or n (%).				

Table 2

Periodontal parameters of study participants stratified by OSA

Periodontal Parameters	All Participants (n = 93)	OSA Yes (n = 19)	OSA No (n = 74)	p Value
Periodontitis Yes No Periodontal Parameters	40 (43.0) 53 (57.0)	13 (68.4) 6 (31.6)	27 (36.5) 47 (63.5)	0.012*
PD (mm) <3 >3	3.0 ± 0.4 53 (57.0) 40 (43.0)	3.1 ± 0.4 6 (31.6) 13 (68.4)	2.9 ± 0.4 47 (63.5) 27 (36.5)	0.090
BOP (%)	47.5 ± 11.0	52.2 ± 10.1	46.2 ± 11.0	0.034
CAL (mm)	2.14 ± 0.57	2.37 ± 0.58	2.08 ± 0.55	0.046
Values are given as mean ± SD or n (%).				
*χ^2 test showed OR = 3.77, 95% CI = 1.29 ~ 11.07				

Table 3

Logistic Analysis for Hypoxia Indicators Predicting Periodontitis Prevalence

Predictors	OR	95% CI	P Value
ODI NAR	0.960	0.813–1.133	0.684
Rhinomanometry Measurements Rhinospirometry Measurements	2.530 0.969	0.758–8.449 0.676–1.391	0.205 0.887
AHI Lowest SaO_2	1.184 0.894	0.987–1.419 0.842–0.949	0.126 0.002*
*Statistically significant			

Figures

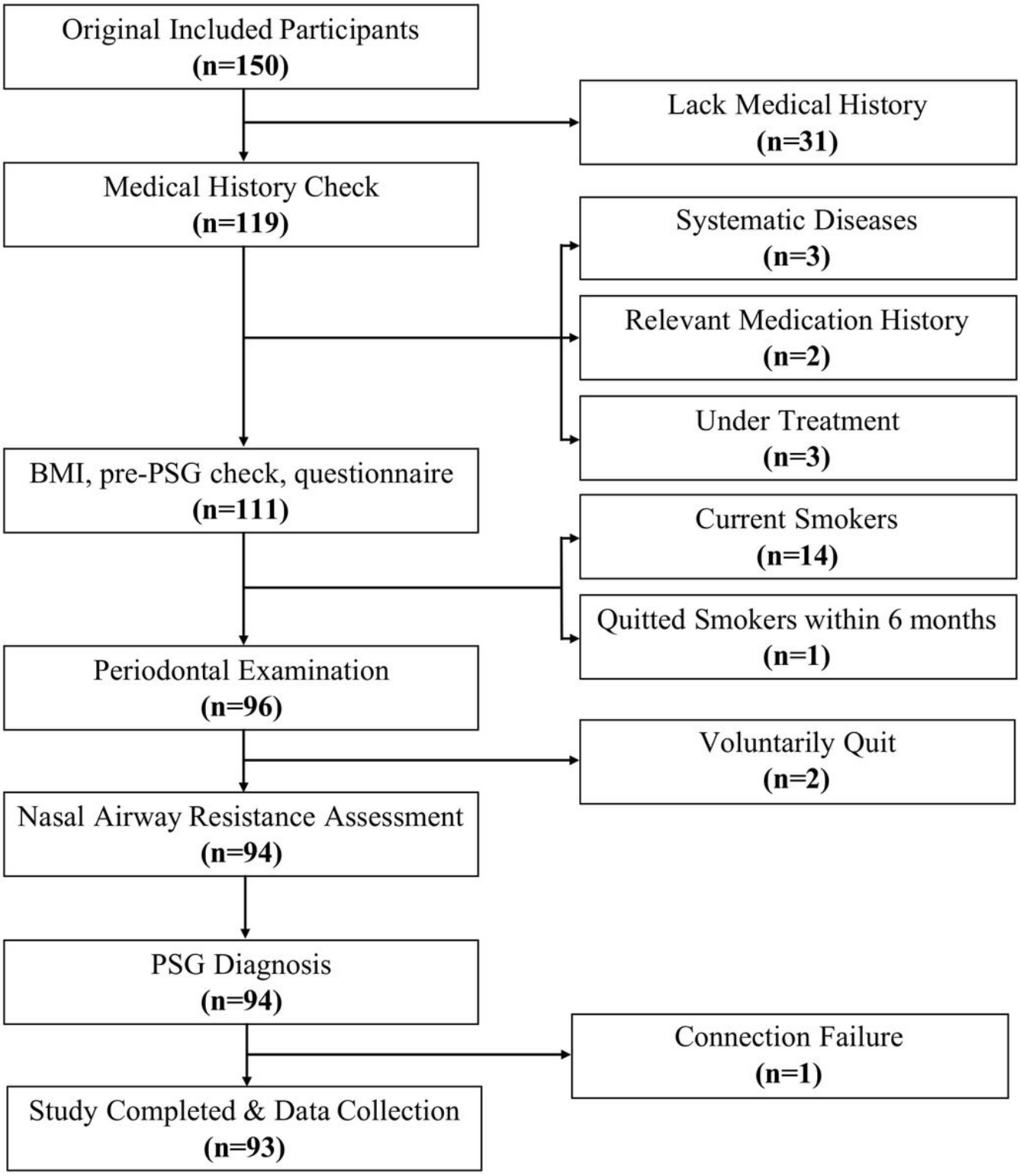

Figure 1

Process flow illustration

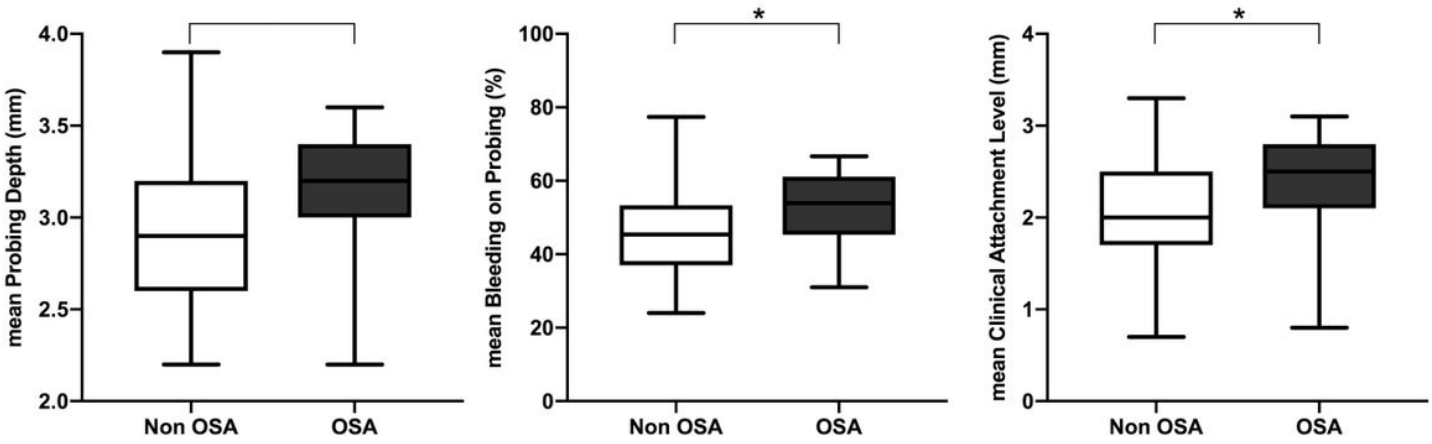

Figure 2

Distribution of three periodontal parameters in OSA and non-OSA group in 93 Chinese male adults * Statistical significance ($p<0.05$)