Prevalence and risk factors of silent obstructive sleep apnea in patients with dentofacial deformities

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Abstract

Objective

Prevalence of silent obstructive sleep apnea (OSA) in patients with dentofacial deformities is unknown, although OSA is severe risk of airway obstruction in perioperative orthognathic surgery or complication after surgery. The aim of this study was to investigate prevalence and risk factors of silent OSA in patients with dentofacial deformities.

Methods

We analyzed 72 patients (24 male, 48 female) with dentofacial deformities without previous OSA symptoms. Polysomnography was performed before orthognathic surgery. Prevalence and risk factors of silent OSA were statistically analyzed as related to Apnea hypopnea index (AHI).

Results

Mean AHI was 1.6 (range: 0-12.1) /h. Three patients of 72 patients (4.1%) were diagnosed silent OSA. AHI during REM sleep phase 3.7 (0-32.3) was higher than AHI during NREM sleep phase 1.0 (0-9.7). AHI of male patients was higher than that of female. AHI was increased according to high BMI. AHI was higher in deep bite than open bite, edge to edge bite and nomal bite. AHI of mandibular asymmetry cases were higher than that of symmetry cases.

Conclusions

The prevalence of silent OSA was 4.1%. Obesity, male, deep bite, mandibular asymmetry and REM sleep phase were risk factors of silent OSA.

Introduction

Dentofacial deformity is a condition involving maxillofacial abnormalities and skeletal malocclusion with a variety of acquired or congenital causes, including abnormal growth and development of the jaw, trauma, and postoperative tumor resection. Regardless of the presence of subjective symptoms, it is likely to cause problems with oral functions such as mastication, swallowing, articulation, and breathing. Sleep plays an important role in the maintenance of physiological and psychological functions. Obstructive sleep apnea (OSA) is the most common type of sleep disorder and is characterised by repeated episodes of complete or partial obstructions of the upper airway during sleep [1]. OSA is thought to be associated with traffic accidents due to excessive daytime sleepiness and the development of cerebral and cardiovascular diseases [2]. The risk of OSA due to anatomical stenosis of the upper respiratory tract is

high in patients with micromandible [3]. Respiratory is necessary to consider not only the morphology but also the function as well as the mastication and articulation. In the perioperative period of orthognathic surgery, airway management is one of the most important perioperative management, and there is a risk of airway obstruction due to the effects of postoperative swelling, bleeding, trismus, and intermaxillary elastic traction. Emergency tracheal intubation or tracheostomy after orthognathic surgery have been reported [4]. A more recent study reports that unstable sleep, called REM rebound, increases with changes in sleep after general anesthesia, and that airway risk is particularly high in perioperative management for OSA patients [5]. It is well known that OSA may be improved in patients with micromandible by mandibular advancement [6,7]. In recent years, sleep surgery by maxillomandibular advancement (MMA) for OSA has developed [8]. However, the mandibular body must be moved to the posterior position by orthognathic surgery in mandibular prognathism, so it is undeniable that the pharyngeal cavity is constricted and causes OSA. Although the risk of OSA after orthognathic surgery in mandibular prognathism is thought to be low because mandibular prognathism has a wider airway than that of normal occlusion without dentofacial deformities [9]. Incidence of OSA after mandibular setback in mandibular prognathism was reported however the status of OSA in mandibular prognathism and the risk of mandibular setback has not been fully investigated [10]. Therefore, considering the safety of orthognathic surgery, evaluation of OSA status and perioperative airway in patients with dentofacial deformities is extremely important. These days, three-dimensional image examination has progressed and many simulations of occlusal and facial morphology have been reported in orthognathic surgery, but airway changes in orthognathic surgery have been evaluated using standard X-ray photographs of the head[11]. However, it is not always said that functional airway evaluation is performed because it is evaluated based on the images at the temporary points taken. However, OSA in patients with dentofacial deformities have been evaluated before and after surgery using images such as CT images and cephalometric X-ray photographs postoperatively[12]. Although a functional evaluation has been reported after a long period of time[13], OSA evaluation using polysomnography and perioperative functional evaluation in mandibular prognathism have hardly been performed[14]. There was no report for prevalence and factors of silent OSA in patients with dentofacial deformities without obstructive sleep apnea symptoms using polysomnography, althogh prevalence of symptomtmatic OSA on dentofacial deformities and maxillo-mandibular advancement for OSA treatment were much reported.

The aim of this study was to evaluate prevalence and factors related to silent OSA in patients with dentofacial deformities.

Material And Methods

Paticipants

Seventy-two patients (24 male, 48 female) with dentofacial deformities who were performed orthognathic surgery at Department of Oral and Maxillofacial Surgery, Kanazawa University Hospital, Kanazawa, Japan from 2016 to 2020 were included in this study. After providing informed consent, participants were

enrolled based on the following inclusion criteria: (1) over 15 years old male or female; (2) skeletal class or III dentofacial deformites with or without mandibular asymmetry; (3) without OSA symptoms evaluated by less than 10 of Epworth Sleepiness Scale (ESS) score. Exclusion criteria were as follows: (1) more than two missing posterior teeth, excluding third molars or the use of a removable prosthesis; (2) sleep disorder (e.g. sleep apnea syndrome, narcolepsy); (3) congenital disease including cleft palates; (4) use of medication with possible effects on sleep or motor behavior; (5) alcohol or drug abuse; and (6) major neurological or psychiatric disorders. This study complied with the principles stated in the Declaration of Helsinki Ethical Principles for Medical Research Involving Human Subjects, adopted by the 18th World Medical Assembly. The requirement for IRB has been approved by Kanazawa University Hospital Research Ethical Committee (Ref. No.1765-1).

Silent obstructive sleep apnea evaluation using polysomnography

We defined silent obstructive sleep apnea (OSA) as present in patients who were undiagnosed at initial surgical consultation but were then confirmed by an attended PSG and evaluation by a pulmonologist to have symptomatic OSA. Prior to orthognathic surgery, interviews with Epworth Sleepiness Scale (ESS) and polysomnography were performed, Polysomnography was performed before orthognathic surgery (Embla N7000 with MDrive, CHEST, Japan) by two sleep technicians during over night evaluation and diagnosed OSA by a pulmonologist. The sleep quality and respiratory status were evaluated by sleeping hours, sleeping efficiency, rapid eye movement (REM) sleep phase, non-rapid eye movement (NREM), apnea-hypopnea index (AHI), oxygen desaturation index (ODI), Lowest peripheral oxygen saturation (lowest SPO2), average SPO2 and cumulative percentage time at SpO2 below 90 % during the measurement (CT90). Sleeping efficiency was calculated (REM sleep time + NREM sleep time) / total sleep time.

Risk factors related to silent obstructive sleep apnea

ESS, sleep time, sleep efficiency, REM sleep rate, sleep phase (REM/NREM) were evaluated as the sleep factors, age, gender and body mass index (BMI) were evaluated as physical factors, overjet, overbite, SNA angle, SNB angle, ANB angle, SN-MP angle, GZN angle, Go-angle and mandibular deviation using cephalometric analysis were evaluated as the morphological factors.

Statistical analysis

Statistical relationship between risk factors related to silent OSA in patients with dentofacial deformities and apnea hypopnea index values was analyzed by Linear regression. Comparison between two factors was analyzed by Mann-Whitney test, among 3 or more factors was analyzed by one-way ANOVA test. The level of signicance was set at $p < 0.05$. All statistical analysis were performed using GraphPad Prism, version 7 (GraphPad Software, San Diego, CA, USA).

Results

Prevalence of silent obstructive sleep apnea

The mean and standard deviation average SPO2 was 97.0 ± 0.8 % (range: 94.2–98.3%). Lowest SPO2 was 92.4 ± 2.6% (range: 82.9–96.0). CT90(min) was 0.014 ± 0.07 (range: 0-0.5). ODI was 1.3 ± 2.7 (range:0-21.6) and AHI was 1.6 ± 1.9 (0-12.1). Three patients (two male and one female) with skeletal class 3 dentofacial deformity of 72 patients (4.1%) were diagnosed silent OSA (AHI: 12.1, 8.5, 6.4, Age: 31, 25, 54, BMI: 33, 20, 31) (Table.1).

Risk factors related to silent obstructive sleep apnea

REM sleep phase, BMI, Gender, overbite, mandibular deviation were related to prevalence of OSA (Table 2). AHI during REM sleep phase 3.7 ± 5.2 (0-32.3) was statistically higher than AHI during NREM sleep phase 1.0 ± 1.6 (0-9.7) (Fig. 2). AHI of male patients 2.6 ± 2.7 (0-12.1) was higher than that of female 1.0 ± 1.1(0-6.4) (Fig. 3). AHI was increased according to high BMI (Fig. 4). AHI was higher in deep bite 3.2 ± 3.7 (0.2–12.1) than open bite 1.1 ± 1.0 (0-3.4), edge to edge bite 1.0 ± 1.2 (0-4.4) and nomal bite 1.6 ± 1.5 (0.2–6.4) (Fig. 5). AHI of mandibular asymmetry cases 2.0 ± 2.3 (0-12.1) were higher than that of symmetry cases 1.1 ± 1.3 (0-6.4) (Fig. 6).

Discussion

We defined silent OSA is first observed $5 \leq$ AHI by polysomnography even if patient ESS under 10. The Epworth Sleepiness Scale (ESS) is established as self-reporting questionnaire used by the clinical assessment an individual's level of daytime sleepiness [15]. In clinical practice, ESS scores of 11–24 represent increasing levels of 'excessive daytime sleepiness' to recommend for OSA examination. The diagnosis for OSA is defined as follows: normal, AHI < 5; mild sleep apnea, $5 \leq$ AHI < 15; moderate sleep apnea, $15 \leq$ AHI < 30; severe sleep apnea, AHI $\geq$ 30 [16]. In this study, 3 patients of 72 patients (4.1%) were diagnosed mild silent OSA although they had no symptom of OSA before polysomnography evaluation. Posnick et al reported 23%(60 of 262)had silent OSA with dentofacial deformities exceeded that estimated in the general population by PSG confirmed. This prevalence rate of 23 % was remarkable higher than our results of 4.1% although these two studies were performed both dentofacial deformities. Prevalence of OSA was reported in the range of 3 to 7% for adult men and 2 to 5% for adult women [17–20] in Asia, however, in the range of 13–33% in adult men and 6–19% for adult women, in Europe and North America[21]. LI et al [22] evaluated the possible differences between Asian and White patients with OSA and reported that the Asian patients were less obese and had maxillomandibular protrusion, narrower cranial base angle, larger posterior airway space, and more superiorly positioned hyoid bone compared with the White patients. Mandibular protrusion has a wider airway than that of normal occlusion without dentofacial deformities [9]. For that reason, silent OSA in our study targeted Japanese was low rate may be due to difference of ethnicity between Asian and White patients. Ethnicity was one of the risk factors of OSA was reported[23].

Further in previous research, male gender, obesity, age, and craniomaxillofacial morphology are considered to be predictable factors for development of OSA too[23]. Regarding gender, OSA was more prevalent in male than female regardless of ethnicity[17–21]. These facts supported our result of OSA in male was higher than in female even if participants were Asian in this study. Anatomical differences or sex hormone are cause for OSA differences between male and female was reported. Pharyngeal airway anatomy or tissue characteristics between males and females has fundamental difference and males are more susceptible than females to load-induced hypoventilation because of the increased airway collapse [24]. A low testosterone level results in a higher OSA risk [25] and Estrogen protects against OSA by exerting antidepressant and sleep effects[26]. A reduced estrogen level could affect the level of serotonin, which controls the tongue and palate muscle tone. In women who are pregnant or with menopause, the OSA risk is higher[27]. Regarding obesity, It was also greater in obese men and women compared with overweight men and women[21]. According to World Health Organization definitions, BMI ≥ 30 kg/m2 can be defined as obesity, and BMI 25–29.9 kg/m2 can be defined as overweight [28]. Two of three patients with silent OSA of BMI in our study were over 30 kg/m2 was convincing results.

. Regarding age, The prevalence of OSA in the elderly population was remarkably high was reported. This was 88% in men and 66% in female aged 65–69 years old [29], 90% in men and 78% in female aged 60–85 years old [21]. The reason is why there is no significant relationships with age in our study may be our participants were almost under 50 years old and young. Regarding morphology, another previous study [30] suggested that skeletal conditions such as short mandibular body and mandibular retrusion, size of the tongue, position of the hyoid bone, and shape of the airway are factors that contribute greatly to a further increase in the severity of OSA syndrome in males. On the other hand, mandibular protrusion has a wider airway than that of normal occlusion without dentofacial deformities [9]. We speculated before study that large ANB angle and small SN-MP angle will be the factors related to prevalence of silent OSA. However, overbite and mandibular asymmetry were related to prevalence of silent OSA. Although mandibular retrusion was included in our study, patients with symptomatic OSA were excluded was may be the reason different factors had in silent OSA. However, large overbite generally indicate short face pattern and asymmetry was reported as factor related to OSA in previous study. Our results was should be considered to risk factors of silent OSA.

Guillminault et al[31]and RILEY et al[32] reported on female patients who developed OSAS after bilateral mandibular osteotomies for treatment of skeletal Class III malocclusion. Two other patients with relatively high values of 3%ODI (26.0 and 19.9 at T1) were mildly obese (BMIs of 26.2 kg/m2 and 27.3 kg/m2) and their amounts of mandibular setback were comparatively large (10.4 mm and 13.0 mm) [33]. A large amount of mandibular setback might inhibit biological adaption and cause sleep-disordered breathing, and it might be better to consider maxillary advance or another technique that does not reduce the airway for patients with skeletal class III malocclusions who have large anteroposterior discrepancy and/or maxillary hypoplasia[34]. A systematic review suggested that although the development of postsurgical OSA has been reported, there is no clear evidence that confirms a direct cause-and-effect

relationship between mandibular setback surgery and OSA development[16]. On the other hand, maxillomandibular advancement (MMA) is the most effective surgical treatment for OSA, with results that are comparable to the efficacy of therapeutic use of continuous positive airway pressure (CPAP) [35,36]. Another results showed a high prevalence of acute sleep disturbance. Patients with acute sleep disruption had low postoperative sleep efficiency and impaired functional ability three months after surgery[37]. In this way, maxillamandibular morphology is related to risk of OSA and orthognathic surgery caused perioperative acute respiratory complication or postoperative OSA and improvement OSA by MMA. Preoperative diagnosis for silent OSA is very important alike symptomatic OSA in treatment for dentofacial deformities.

Conclusions

We require attention to OSA in dentofacial deformities even if without OSA symptoms in particular with obesity, male, deep bite, mandibular asymmetry and REM sleep phase.

Declarations

Ethics approval and consent to participate

The requirement for IRB has been approved by Kanazawa University Hospital Research Ethical Committee (Ref. No.1765-1). All patients are informed research purpose and agree to use their clinical data for this study. Informed consent for participation in the study was obtained from their parent or guardian if participants are children (under 16 years old).

Author contributions

K. O and N. I designed most of the experiments and wrote the main manuscript text and prepared all figures and tables. K. M and H. Y are the primary person responsible for carrying out all experimental procedures. T. M analyzed the data. S. K and K. T are the person who made the final approval of the article.

Conflict of interest

The authors declare that they have no conflicts of interest.

Role of the funding source

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Tables

Due to technical limitations, table 1, 2 is only available as a download in the Supplemental Files section.

Figures

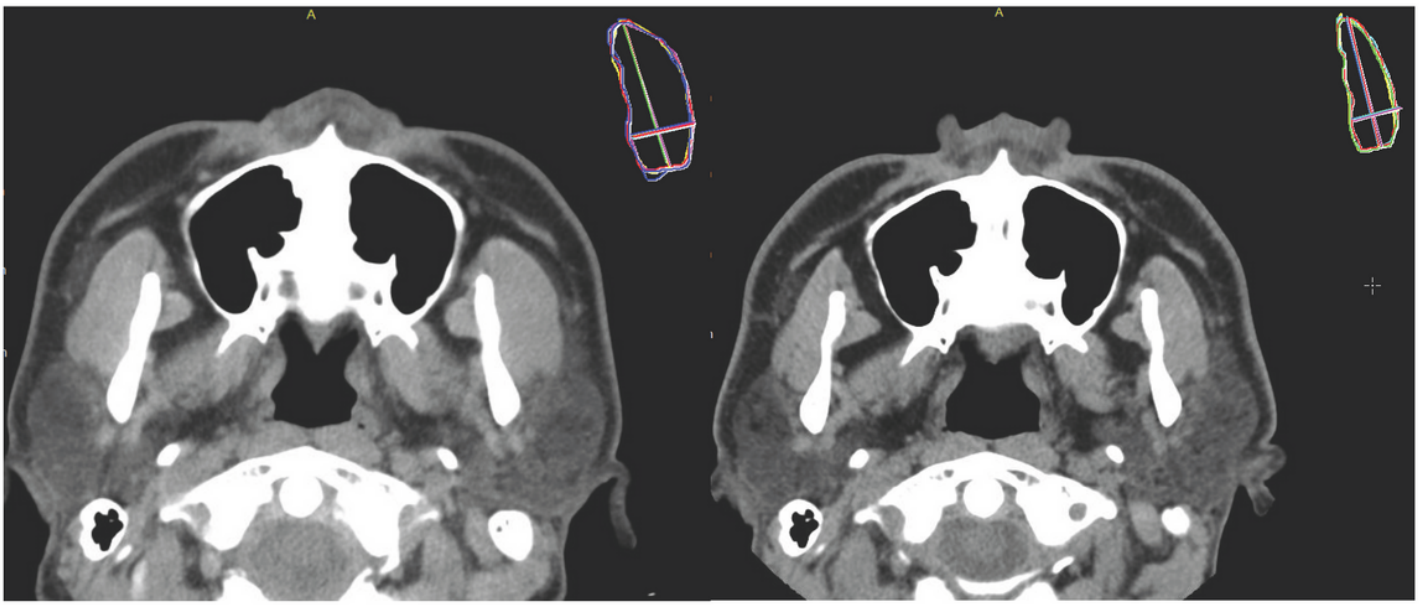

Figure 1

Measurements of cross sectional masseter muscle using CT images

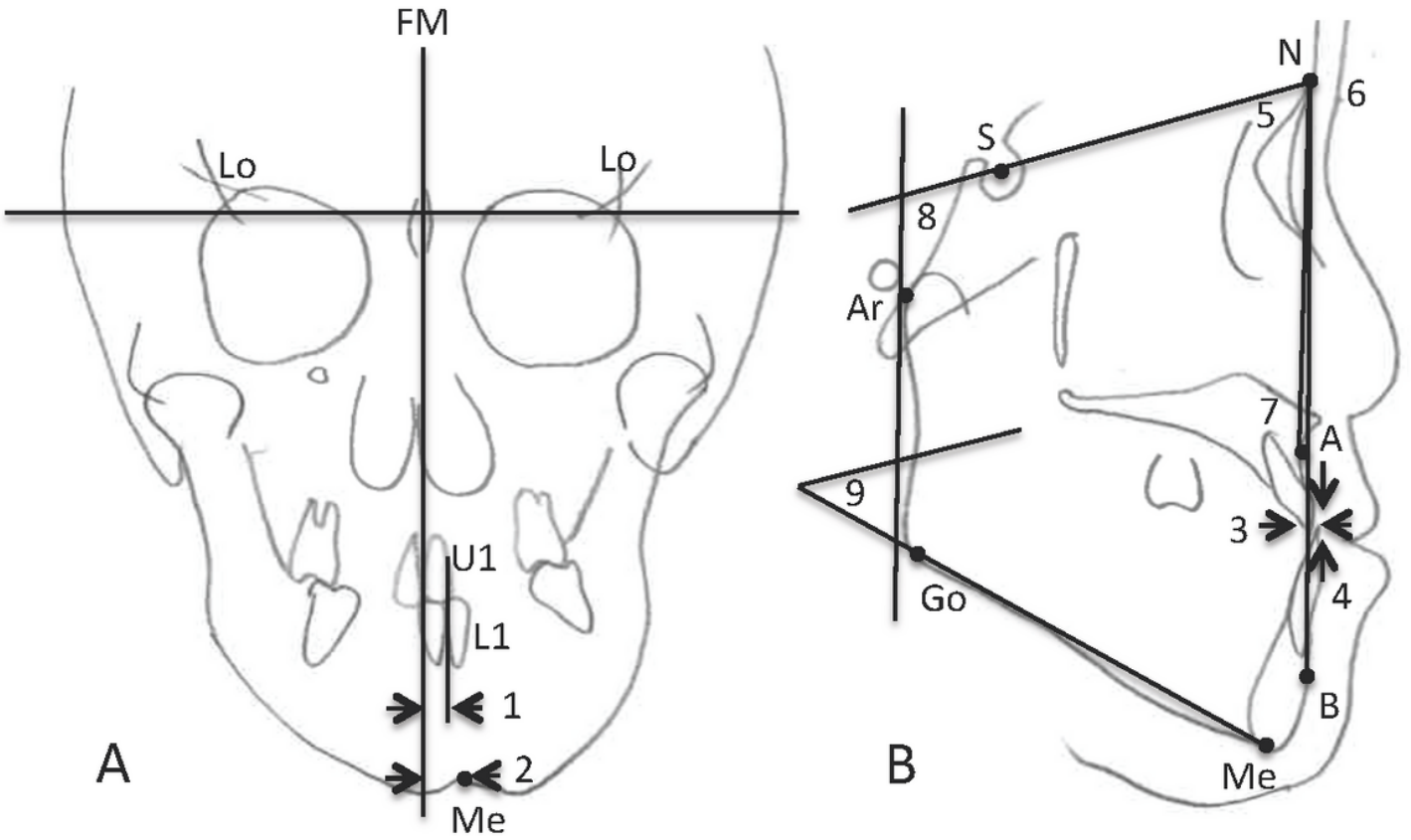

Figure 2

Cephalometric measurements A: Anteroposterior cephalometric measurements. 1;DFFM at Menton (mm), 2;U1-L1 deviation (mm) B: Lateral cephalometric measurements 3;overjet (mm) , 4;overbite (mm), S;SNA angle (□), 6;SNB angle (*), 7;ANB angle (□), 8;GZN angle (□), 9;SN-MP angle (□) Cephalometric landmarks: menton (Me), midpoint of the upper incisor edge (U1), midpoint of the lower incisor edge (L1), sella (S), nasion(N), point A (A), point B (B), gonion (Go), articulare (Ar) FM: facial midline, DFFM :Deviation from the facial midline

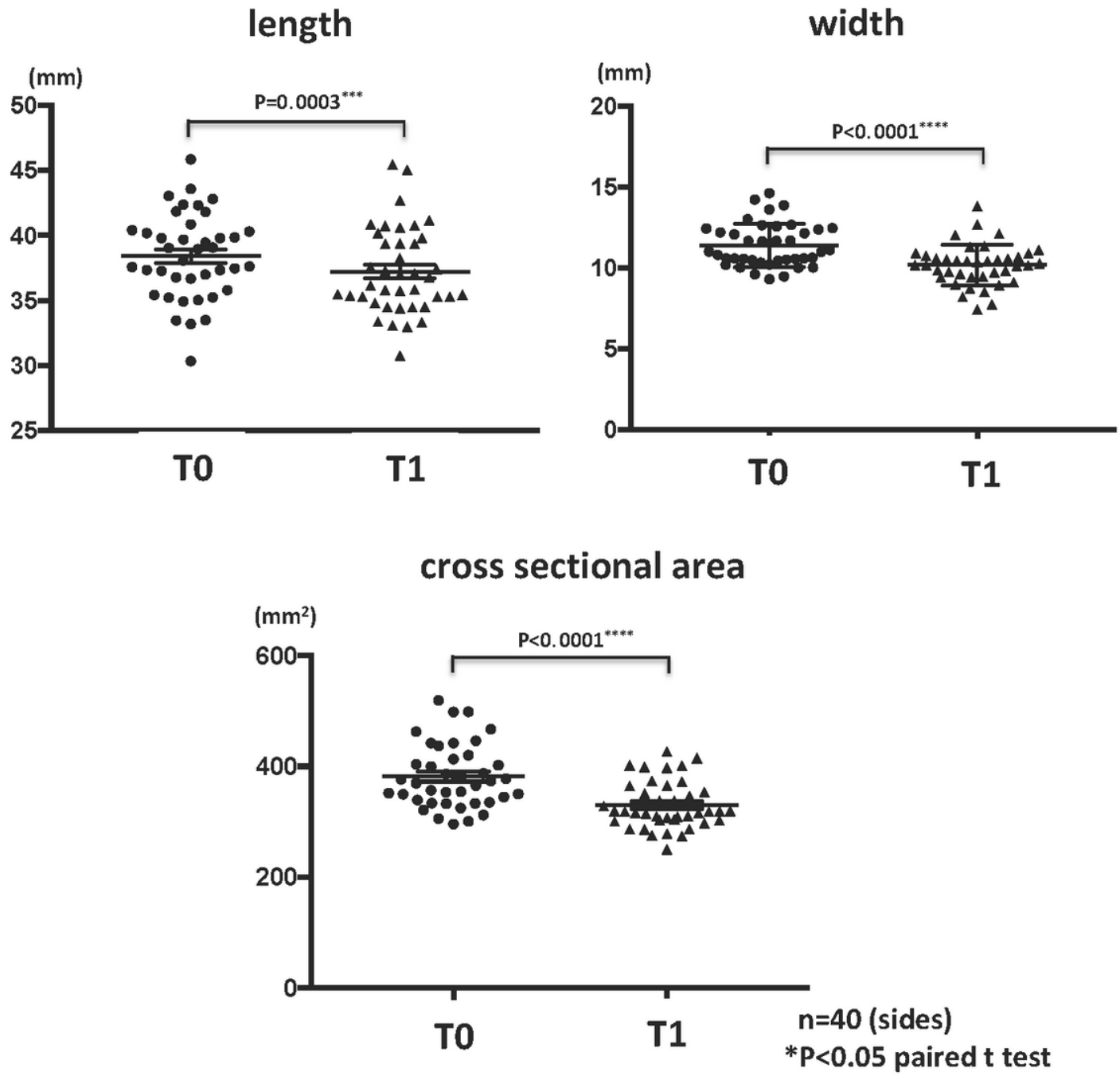

Figure 3

Comparison of masseter muscle length, width and area between before and after preoperative orthodonthic treatment

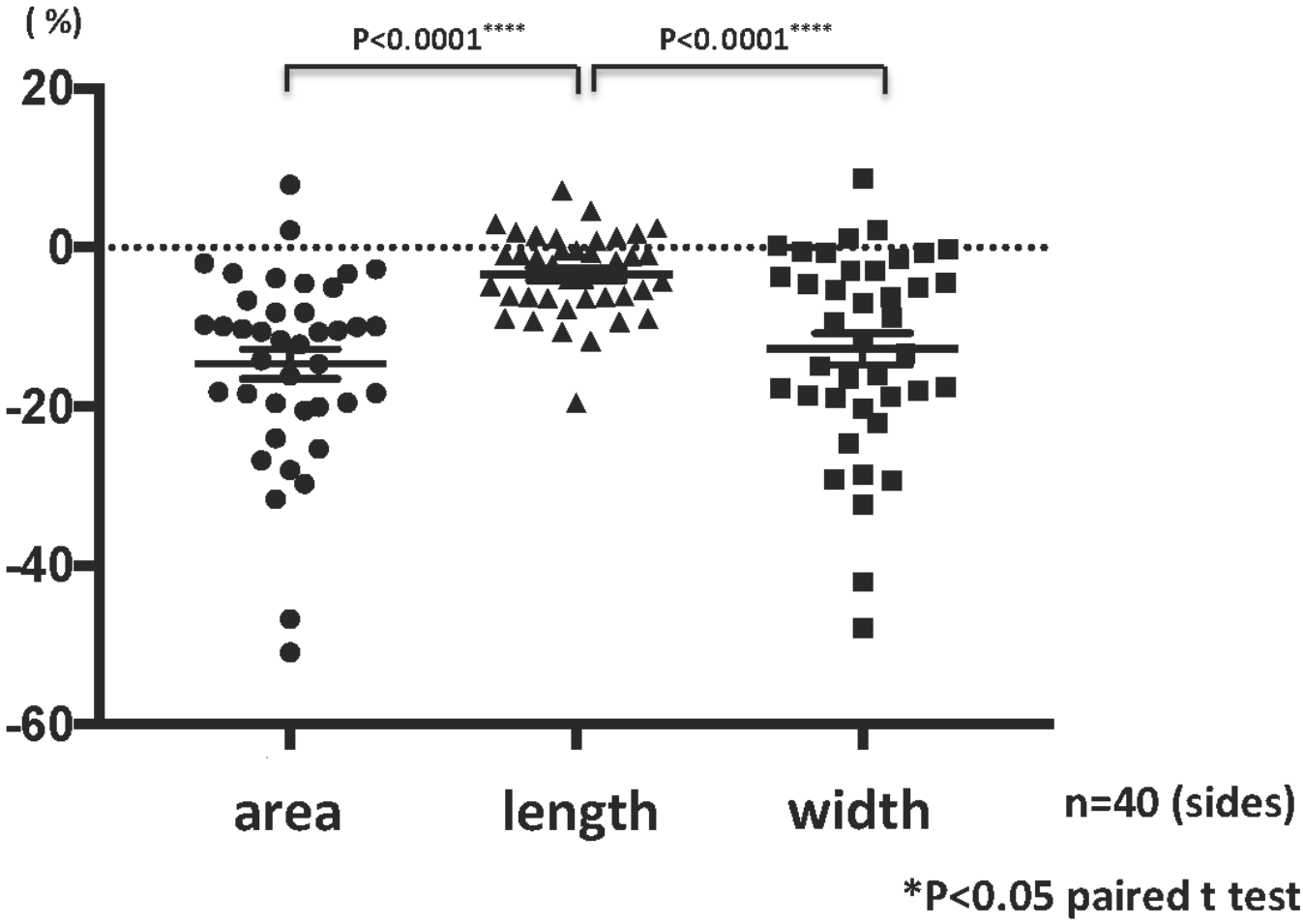

Figure 4

Change rate of masseter muscle length, width and area after preoperative orthodontic treatment

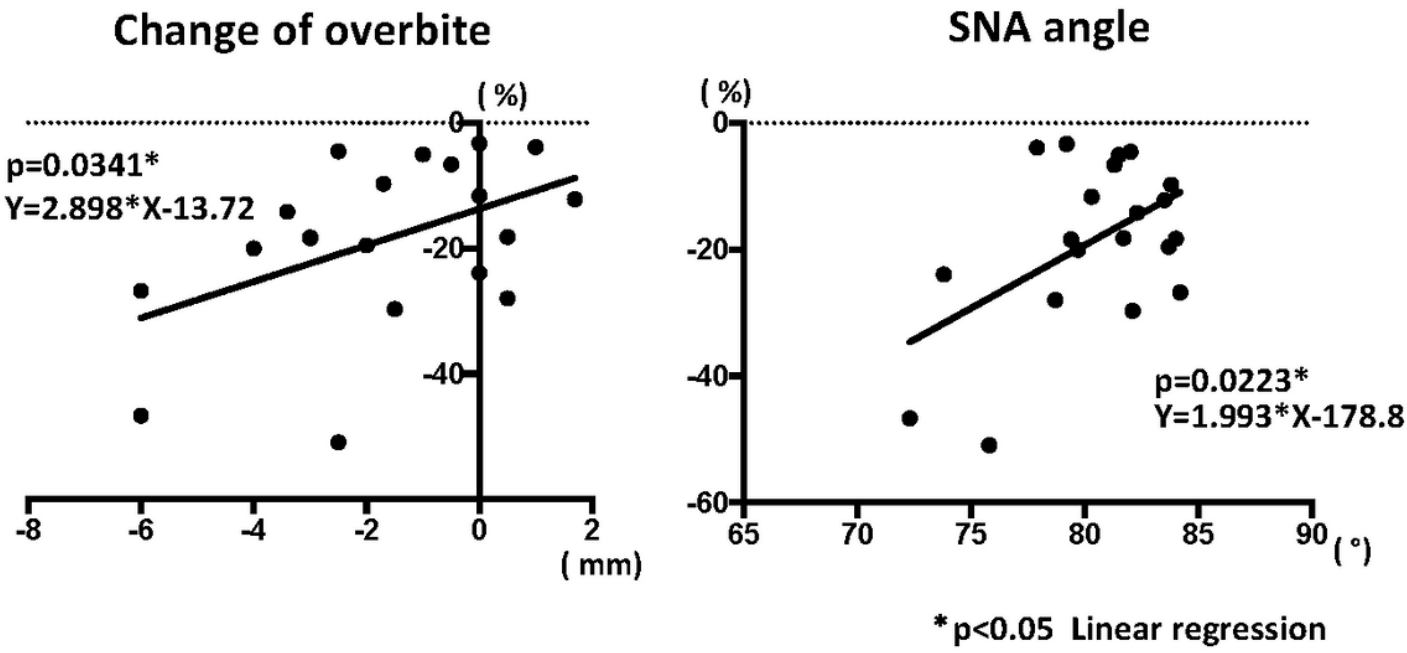

Figure 5

Relations between changes of cross sectional masseter muscle area and changes of overbite and SNA angle

Supplementary Files

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