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Association between adenotonsillar hypertrophy and dentofacial characteristics of children seeking for orthodontic treatment: A cross-sectional study

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Institutional Review Board Statement: All procedures performed in studies involving human participants

were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Statement: As a retrospective study using routinely collected data from healthcare activities, this study was approved by the Ethics Committee of School & Hospital of Stomatology, Wuhan University (No. 2020-B02) to be conducted without patients' informed consent.

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Abstract

Objective: To compare the dentofacial characteristics of children with and without adenoid and/or tonsillar hypertrophy.

Methods: A consecutive sample of orthodontic patients aged 6-12 that took pre-treatment lateral cephalograms were included in this study. Those with history of previous orthodontic treatment, adenoidectomy or tonsillectomy, or craniofacial anomalies were excluded. The diagnosis of adenoid and tonsillar hypertrophy was based on Fujioka's and Baroni's methods, according to which the subjects were divided into four groups: the adenoid hypertrophy only (AHO) group; tonsillar hypertrophy only (THO) group; combined adenoid and tonsillar hypertrophy (AH+TH) group; and no adenoid or tonsillar hypertrophy (NH) group. Cephalograms were used for skeletal and dental measurement. Data were analyzed using one-way ANOVA, LSD post-hoc tests and Chi-square test.

Results: A total of 598 patients were included. Compared with the NH group, the THO group had significantly larger SNB angle ($P<0.001$), as well as significantly smaller ANB angle ($P<0.001$) and Wits value ($P=0.001$). The U1-L1 angle of AHO group was significantly smaller than that in the NH group ($P=0.035$). The proportion of adenoid hypertrophy in Class II patients was significantly higher than that in Class III patients ($P=0.001$). The proportion of tonsillar hypertrophy in Class III patients was significantly higher than that in Class I patients ($P<0.001$) and Class II patients ($P<0.001$).

Conclusion: Over 80% of children seeking orthodontic treatment had either adenoid or tonsillar hypertrophy. Children with adenoid hypertrophy tend to have skeletal Class II malocclusion, while those with tonsillar hypertrophy tend to have skeletal Class III malocclusion.

Keywords: adenoid hypertrophy; tonsillar hypertrophy; dentofacial morphology; malocclusion

Introduction

As part of the Walderyer's ring, adenoids and tonsils serve as the first-line defence mechanism against microorganisms and antigenic substances during childhood. Infectious and multiple non-infectious causes such as allergies may lead to adenoid or tonsil hypertrophy, resulting in nasopharynx or oropharynx obstruction, respectively (1). It is generally believed that adenoids and tonsils reach their maximal size by age six, and then gradually regress during adolescence (2). However, a longitudinal observational study reported that no significant decrease was observed in the size of adenoids and tonsils from childhood to adolescence (3).

Adenotonsillar hypertrophy is a major cause of upper airway obstruction and the resultant obstructive sleep apnea (OSA) in children (4). Therefore, adenotonsillectomy is the first line treatment for OSA in children with adenotonsillar hypertrophy according to relevant clinical practice guidelines (4). Untreated pediatric OSA is associated with impaired neurocognitive function, impaired growth, cardiovascular dysfunction, behavioral problems, as well as impaired dentofacial development (5-7). The reported prevalence of OSA ranged widely from 0.1% to 13% depending on the surveyed population and diagnostic criteria (8). In addition to sleep medicine and ENT specialists, dental professionals also play a role in the screening of OSA due to the certain dentofacial characteristics of OSA cases (9, 10).

The common diagnostic methods for adenoid or tonsil hypertrophy include nasopharyngoscopy, oral examination and cephalometric analysis. While nasopharyngeal endoscopy is the gold standard for diagnosing adenoid hypertrophy, lateral cephalogram as the most common examination method in orthodontic clinic exhibits very good diagnostic accuracy for the diagnosis of adenoid hypertrophy (11). In a randomly chosen representative sample, the prevalence of AH was 34.46%; however, in convenience samples, the prevalence ranged from 42 to 70% (12). The prevalence of tonsil hypertrophy was found to be 11% in school children (13). Besides, according to our previous study, the prevalence of tonsil hypertrophy in malocclusion children as high as 66.3%, and the prevalence of tonsil hypertrophy in skeletal class Ⅲ patients was significantly higher than that in patients with skeletal class Ⅰ and Ⅱ malocclusion. (14).

Orthodontic research has been focused on the association between adenotonsillar hypertrophy and dentofacial morphology (15-18). During growth and development, nasal breathing pattern can promote normal dentofacial growth of children (19). Linder-

Aronson et al. (15, 16) hypothesized that the establishment of nasal respiration in children with severe nasopharyngeal obstruction can be eliminated as a factor in determining the mandibular growth direction. Besides, plenty of studies have found that children with mouth breathing regardless of the etiology of obstruction are more likely to have a narrower maxillary arch, retruded chin, steep mandibular plane, vertical growth pattern and a tendency toward Class II malocclusion (20-23).

There is research futher investigating the cephalometric pattern of mouth breathing children with distinct obstructive tissues (adenoids or tonsils) (24). It found that mouth breathing children with isolated hypertrophy of the palatine tonsils presented with a more forward and upward mandible compared with children obstructed only by the enlarged adenoid (24) .However, this study had relatively few measurement values. Besides, there was no new evidence to confirm these findings.

Therefore, the objective of this study was to further investigate the association between upper airway obstruction sites and dentofacial development by comparing the dentofacial characteristics (e.g. ANB, SN-MP and U1-SN) of children with or without adenoid and/or tonsil hypertrophy with a relatively large sample.

Materials and methods

This cross-sectional study was approved by the Ethics Committee of the School of Stomatology, Wuhan University (No. 2020-B02). Subjects were selected from consecutive patients attending Department of Orthodontics, Hospital of Stomatology, Wuhan University seeking for orthodontic treatment during January to August, 2019. The inclusion criteria were children aged 6-12 taking lateral cephalograms. Exclusion criteria were: (a) poor quality cephalometric image inadequate for identification of upper airway and dentofacial structures; (b) history of previous orthodontic treatment; (c) history of adenoidectomy or tonsillectomy; (d) acute adenoiditis or tonsilities; (e) craniofacial anomalies such as the presence of cleft lip and palate.

All lateral cephalograms were taken in natural head position and intercuspal position with the same cephalostat (OP200DSoredex, Instrumentarium, Finland). Cephalometric measurements were performed by a well-trained investigator (T.Z) using Dolphin-3D (version 11.7; Dolphin Imaging, Chatsworth, Calif). The selected cephalometric landmarks and measurements are summarized in **Table 1**.

The diagnosis of adenoid hypertrophy was made based on Fujioka's method (25). The presence of tonsillar hypertrophy were determined according to the criteria of

Baroni et al (26). The reference points and lines used on the lateral cephalogram for adenoid and tonsil mearsurements are shown in **Figure 1**. Line segment **A** indicated the size of the adenoid; line segement **N** indicated the size of nasopharyngeal space; line segment **T** indicated the size of the tonsils and line segment **O** indicated the size of oropharyngeal space. The percentage of adenoid or tonsillar obstruction in the pharyngeal airway space was calculated mathematically as follows: (A/N)×100 % and (T/O) × 100%. If the percentage was greater than 50%, adenoid and/or tonsillar hypertrophy was determined.

Based on the obstruction sites, subjects were divided into four groups: the adenoid hypertrophy only (AHO) group (**Figure 2a**); tonsillar hypertrophy only group (THO) (**Figure 2b**); combined adenoid and tonsillar hypertrophy (AH+TH) group (**Figure 2c**); and no adenoid or tonsillar hypertrophy (NH) group (**Figure 2d**).

The SPSS 25.0 software (version 25.0; IBM, Armonk, NY) was used for data analyses. Differences among groups were analyzed with a one-way ANOVA and LSD post hoc test. A Bonferroni corrected chi-square test was used to analyze the difference between adenoid and tonsillar hypertrophy proportion in different types of malocclusion.

Results

After the application of eligibility criteria, 336 of the total 934 children were excluded and the remaining 598 subjects were included. Among the 598 subjects, 88 were classified to the AHO group (42M, 46F; 10.75±1.34 years old), 124 to the THO group (68M, 56F; 10.45±1.74 years old), 274 to the AH+TH group (126M, 148F; 10.26±1.84 years), and 112 to the NH group (40M, 72F; 11.07±1.25 years). A total of 81.3% children had either adenoid or tonsillar hypertrophy (387/598), more specifically, 60.5% (362/598) had adenoid hypertrophy and 66.6% (398/598) had tonsillar hypertrophy.

The descriptive analysis of all cephalometric variables is presented in **Table 2**. Using a one-way ANOVA analysis, significant differences among the four experimental groups were found with the variables U1-L1 angle (P=0.007); L1-MP angle (P=0.008), SNB angle (P<0.001), ANB angle (P<0.001), NSAr angle (P=0.019) and Wits value (P<0.001). No significant differences were found for U1-SN, SNA, SArGo, ArGoMe, NGoAr, NGoMe, SUM and SN-MP among the four groups.

Using pair-wise comparison (**Table 3**), the SNB angle in the THO group was significantly larger than the other three groups (P<0.001). The ANB angle, L1-MP and Wits appraisal were significantly smaller than the other three groups. The NSAr angle in THO group was significantly smaller than that in the other three groups. The U1-L1 angle in the AHO group was significantly smaller than that in the other three groups. No significant differences were found in dentofacial morphology between the AHO and NH group subjects except for the incisor inclination (U1-L1 angle). These results suggested that subjects with tonsillar hypertrophy tend to have Class III malocclusion with mandibular protrusion, while subjects with adenoid hypertrophy tend to have larger incisor labial inclination.

When subjects were divided into three groups according to the ANB angle, the proportion of skeletal Class I ($0 \leqslant ANB \leqslant 4$), Class II (ANB>4) and Class III (ANB<0) malocclusion in the overall sample was found to be 37.0% (221/598), 50.7% (303/598) and 12.4% (74/598), respectively. The proportion of adenoid or tonsillar hypertrophy in different sagittal malocclusion classification were shown in **Table 4.** Analysis using the Chi-square test indicated significant differences among the three groups for the percentage of adenoid or tonsillar hypertrophy (P=0.004; P<0.001). According to the Bonferroni Chi-sqaure test, the proportion of adenoid hypertrophy in Class II patients was significantly higher than the Class III patients (X^2=10.2, P=0.001). The proportion of tonsillar hypertrophy in Class III patients was significantly higher than the Class I (X^2=36.903, P<0.001) and Class II patients (X^2=35.431, P<0.001).

Discussion

In the current study, 60.5% of the 598 children between 6-12 years old were diagnosed with adenoid hypertrophy. This is higher than that the 34.36% reported by Feres et al. in a randomized sample of adolescent children (12). Our study shows that the ratio of adenoid hypertrophy in skeletal Class II patients was significantly higher than Class I and Class III. Besides, the mean value of SNB in the AHO group was lower than that of the other three groups, and the mean values of ANB and Wits were higher than those of the other three groups. These results were consistent with the systematic review published by Flores etal. in 2013 which reported that most of the children with adenoid hypertrophy showed the trend of Class II malocclusion (20). However, the study conducted by Feres et al drawn opposite conclusions that specific dentofacial

patterns, such as Class II and hyperdivergency, might not be associated with adenoid hypertrophy (27).

The effect of adenoid hypertrophy on the inclination of children's anterior teeth is controversial (28, 29). We found that the interincisal angle (U1-L1) in the AHO group was significantly lower than the other three groups, and the upper central incisor lip inclination (U1-SN) and the lower central incisor lip inclination (L1-MP) in the AHO group were higher than those in the other three groups. It is suggested that adenoid hypertrophy may only cause labial inclination of upper and lower anterior teeth. However, Zettergren-Wijk L et al. (16) reported that the lingual inclination of the upper incisor was obvious in children with adenoid hypertrophy, and the inclination of the upper anterior teeth returned to normal 5 years after adenoidectomy. The reasons for the different results may be related to many factors, such as lip muscle tension, the course of adenoid hypertrophy and so on. Therefore, more studies are needed to gain a further insight.

Moreover, according to our results, the SNB of the THO group was larger than the other three groups; the proportion of tonsillar hypertrophy in Class III patients was significantly higher than that in Class I patients and Class II patients. These findings corroborate the results of Franco et al. (24) that children with isolated hypertrophy of the palatine tonsils presented with a more forward and upward mandible compared with children obstructed only by the enlarged adenoid.

In the current study, the average values of SNB, ANB, Wits and U1L1 in AH+TH group were all between the AHO and THO groups, suggesting that the dental and craniofacial development of children with adenoid and tonsillar hypertrophy might be the combined effect of both adenoid and tonsillar hypertrophy. As part of the pharyngeal lymphatic ring, adenoid and tonsil often influence each other. Therefore, it is difficult to accurately judge whether children's dentofacial developmental abnormalities are caused by adenoid or tonsillar hypertrophy, or which factors play a major role.

No significant differences were found between adenoid or tonsillar hypertrophy with the vertical skeletal pattern in our study. This was contrary to the findings presented in previous studies (20, 30-32). These investigators reported that most of the patients with mouth breathing caused by adenoid hypertrophy are characterized by steepening of mandibular plane and palatal plane, vertical growth pattern, increase of lower facial height, decrease of posterior facial height. These manifestations are mainly

caused by changes in facial muscles, jaw position and function during oral breathing (33) , such as lip opening and low tongue position. Long-term adaptive changes in the oral cavity will lead to changes in the morphology and position of craniofacial bones and teeth (34).

According to the literature, tonsillar hypertrophy may lead to airway obstruction in the oropharynx, and may push the tongue forward to open the oropharynx, which over time can lead to the habit of mandibular protrusion, anterior teeth cross bite, Class III malocclusion (24, 26, 35). Iwasaki et al. (36) analyzed the relationship between upper airway obstruction sites and dentofacial morphology by hydrodynamics. It was found that there was a significant correlation between tonsillar size and tongue position protrusion and lower incisors protrusion in patients with III malocclusion. In patients with III malocclusion, there was a significant relationship between tongue position and mandibular protrusion. One possible mechanism is that children with tonsillar hypertrophy may push the tongue forward, which may lead to mandibular protrusion (36).

These findings suggest that orthodontists should have more concerns on children's upper airway condition when dealing with children who seeking orthodontics. The presence of certain craniofacial abnormality may serve as a significant predisposing factor in individuals who exhibit of adenotonsillar hypertrophy (37). Children with these features could be candidates for early intervention to prevent the potential negative impacts on craniofacial development. Conversely, certain craniofacial morphologies can increase a child's risk for having OSA. For example, long and narrow faces, narrow and deep palate, steep mandibular plane angle, mandibular retrognathia, and midface deficiency may predispose a child to developing OSA (38). Besides, it is found that rapid maxillary expansion (RME), a well-known orthodontic treatment option for patients with a narrow maxilla, can significantly reduce the size of both adenoid and palatine tonsils (39) .

There were several limitations in the current study. Firstly, the evaluation of adenoid or tonsil was based on the lateral cephalogram of a given moment, which can not reflect the airway condition of the whole dentofacial growth period. Secondly, the sample was selected from those children who attended to our department seeking for orthodontic treatment. Thus, the results of our study may not be applicable to the general population, and this could be the reason for the higher percentage of adenoid and/or tonsil hypertrophy compared with previous relevant studies (12). Thirdly, this is

a cross-sectional study, which means the causal relationship between adenotonsillar hypertrophy and maxillofacial development could not be investigated, the results of this study only indicate their correlation.

Our results demonstrated the need for futher investigating dentofaocial morphological differences among children with mouth breathing or OSA based on obstruction sites. However, well-designed cohort studies are needed to confirm the results and further exploring the causal relationship of upper airway obstruction and detofacial morphology.

Conclusions

Over 80% of children seeking orthodontic treatment had either adenoid or tonsillar hypertrophy. Children with adenoid hypertrophy tend to have skeletal Class II malocclusion, while children with tonsillar hypertrophy tend to have skeletal Class III malocclusion.

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Table 1. Cephalometric landmarks used in the study

Landmarks / Value	Definition
N	Most posterior point on the Curve at the Bridge of the Nose
S	Midpoint of the sella turcica
Ba	Most inferior-posterior point on margin of the foramen magnum
Ar	Point of intersection of the inferior cranial base surface and the averaged posterior surfaces of the mandibular condyles
Go	Most posterior-inferior point on the outline of the mandible angle
Po	Most superior point of the outline of the external auditory meatus
Or	Deepest point on the infra-orbital margin
Me	Most inferior point on the outer inferior margin of the mandible
Gn	Most anterior-inferior point on the outline of the bony chin
Pog	Most anterior point on the contour of the bony chin
ANS	Anterior tip of the median palate
PNS	Most posterior point on the bony hard palate
A	Subspinale, most posterior point on the anterior contour of the upper alveolar process
B	Supramental, most posterior point on the anterior contour of the lower alveolar process
PM	Point on the anterior border of the symphysis between point B and Po where the curvature changes from concave to convex
NSAr	Saddle angle
SArGo	Articular angle
ArGoMe	Gonial angle
SUM	Sum angle
NGoAr	Upper gonial angle
NGoMe	Lower gonial angle
U1-SN	Upper incisors inclination
L1-GoMe	Lower incisors inclination
U1-L1	Inter-incisal angle
SNA	Sella-nasion-subspinale
SNB	Sella-nasion-supramental
ANB	Subspinale-nasion-supramental
SNGoMe	Mandibular plane angle
Wits	Wits value

Table 2. Comparison of cephalometric analysis among adenoid hypertrophy only group (AHO), tonsillar hypertrophy only group (THO), combined adenoid and tonsillar hypertrophy group (AH+TH), and no adenoid or tonsillar hypertrophy group (NH).

	AHO（n=88）		THO（n=124）		AH+TH（n=274）		NH（n=112）		P value *
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
SNA	81.29	3.54	81.18	4.40	81.19	3.91	81.66	3.52	0.734
SNB	76.74	3.40	78.72	4.22	77.09	3.64	76.91	3.63	**0.000**
ANB	4.56	2.31	2.25	3.36	4.07	2.89	4.74	2.60	**0.000**
SNGoMe	36.39	6.31	36.37	7.32	36.33	5.78	35.60	6.49	0.734
U1	105.30	8.76	102.96	8.64	102.35	9.47	102.65	8.53	0.064
L1	92.87	8.27	89.15	8.01	91.89	9.15	91.46	7.95	**0.008**
U1L1	125.48	12.35	131.69	12.44	128.83	13.50	129.36	12.04	**0.007**
NSAr	122.54	4.81	122.15	5.40	123.71	4.92	122.75	4.92	**0.019**
SArGo	150.67	6.66	150.52	6.53	150.26	6.28	151.96	6.10	0.121
ArGoMe	123.12	7.76	123.41	6.73	122.68	6.93	121.50	6.86	0.181
Sum	396.38	6.28	396.13	6.14	396.67	5.71	396.23	5.96	0.829
NGoAr	46.89	4.17	47.13	3.89	46.86	4.18	46.02	3.69	0.169
NGoMe	76.27	5.57	76.31	4.93	75.81	4.51	75.45	4.99	0.482
Wits	0.68	3.05	-1.34	4.82	-0.43	3.45	0.42	3.93	**0.000**

* One-way ANOVA analysis; values in bold indicate statistical significance

Table 3. The P values of pair-wise comparison of cephalometric analysis among the four groups

	AHO/THO	AHO/AH+TH	AHO/NH	THO/AH+TH	THO/NH	AH+TH/NH
SNB	**0.000**	0.444	0.739	**0.000**	**0.000**	0.680
ANB	**0.000**	0.157	0.656	**0.000**	**0.000**	**0.035**
L1-MP	**0.002**	0.352	0.249	**0.003**	**0.040**	0.655
U1-L1	**0.001**	**0.034**	**0.035**	**0.040**	0.166	0.712
NSAr	0.575	0.058	0.769	**0.004**	0.358	0.089
Wits	**0.000**	**0.020**	0.644	**0.031**	**0.001**	0.054

Results of one-way ANOVA analysis with LSD post hoc test; values in bold indicate statistical significance

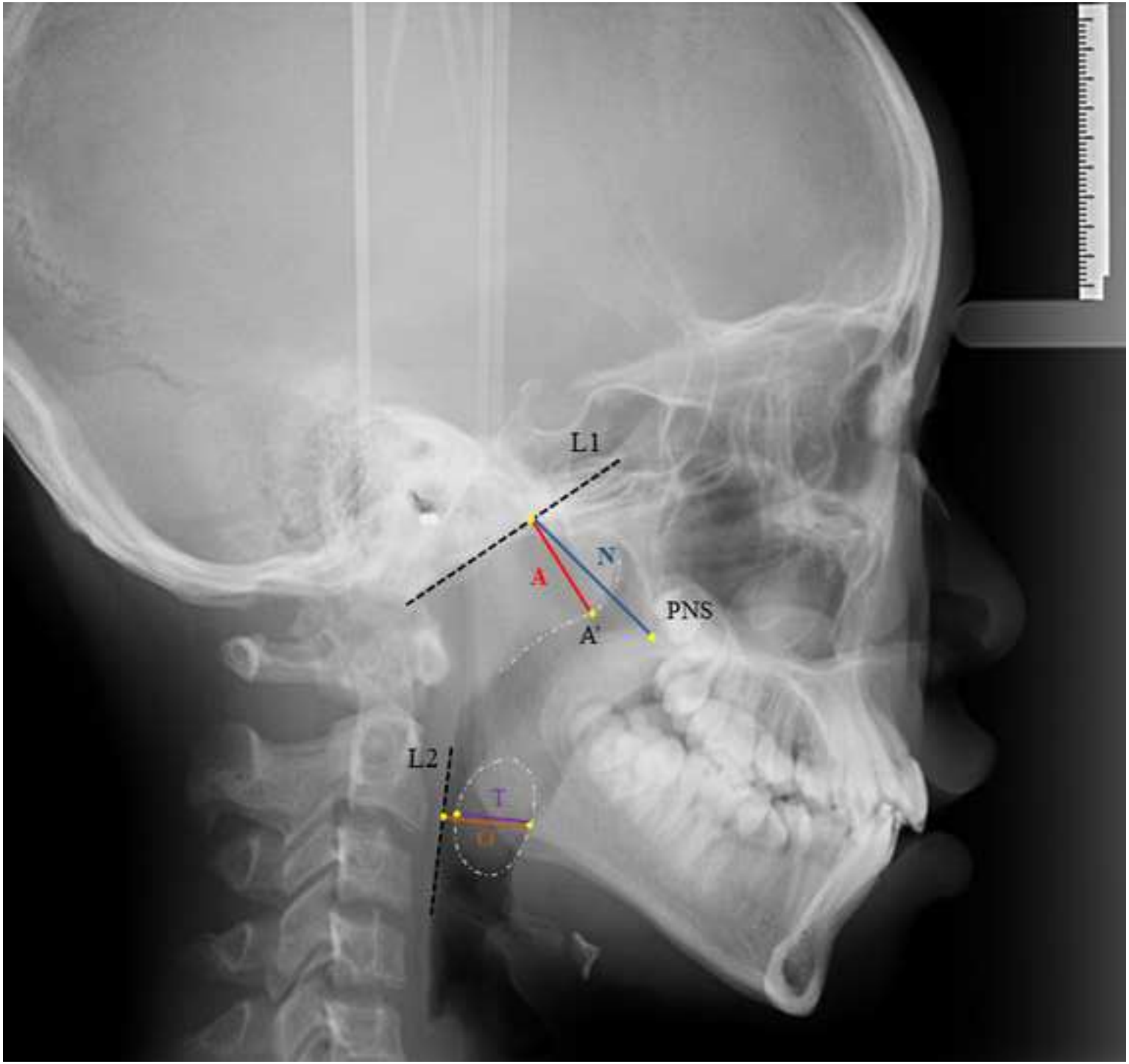
Table 4. The proportion of adenoid or tonsillar hypertrophy in different sagittal malocclusion classification

Malocclusion type	Adenoid hypertrophy	Tonsillar hypertrophy
Skeletal Class I	128/221(57.9%)	145/221（65.6%）
Skeletal Class II	200/303 (65.8%)	188/303（62.0%）
Skeletal Class III	34/74 (45.9%)	65/74（87.8%）
$\chi 2$	11.0	17.9
P value	0.004	<0.001

Figure 1. Reference points and lines on the lateral cephalogram for adenoids and tonsils measurement. *L1 is drawn along the straight part of the anterior margin of the basiocciput; PNS is the posterior superior edge of the hard palate; A' is the maximal convexity along inferior margin of adenoid; L2 is the line tangent to the posterior wall of the oropharynx; To is the nearest point of the tonsils to the L2; L3 is the line perpendicular to L2 passing through To; Op is the intersection of the line L3 and L2. Oa is intersection of the line L2 and the anterior wall of the oropharynx. Line segment A is measured along the line perpendicular from point A' to its intersection (point D) with L1; line segment N is distance between point A' and D. Line segment T is distance between point To and Oa; line segment O is distance between point Oa and Op.*

Figure 2. Adenoid hypertrophy only (a); tonsillar hypertrophy only (b); combined adenoid and tonsillar hypertrophy (c); No adenoid or tonsillar hypertrophy (d).

Figure



Figure