Influence of mandibular incisor agenesis and growth pattern on symphysis characteristics: a retrospective cephalometric study

Saori Yoshida¹⁾, Shin Ota²⁾, Sakurako Kobayashi^{1,2)}

1) Department of Orthodontics and Dentofacial Orthopedics, Graduate School of Life Dentistry at Niigata, The Nippon Dental University, Niigata, Japan

2) Department of Orthodontics, The Nippon Dental University School of Life Dentistry at Niigata, Japan

Corresponding author: Saori Yoshida

Department of Orthodontics and Dentofacial Orthopedics, Graduate School of Life Dentistry at Niigata, The Nippon Dental University, 1-8 Hamaura-cho, Chuo-ku, Niigata 951-8580, Japan

1

E-mail: choco0822@ngt.ndu.ac.jp

Declarations

Acknowledgments

Not applicable.

Conflict of interest

The authors declare that they have no conflict of interest.

Authors contribution

SY coordinated the research project, contributed to the data acquisition and analysis, participated in the interpretation of the results, drafted and revised the manuscript. SO contributed to the data acquisition and analysis. SK coordinated the research project and revised the manuscript. All authors read and approved the final manuscript.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Abstract

Objective: This study was performed to investigate the effects of mandibular incisor (MnI) agenesis and divergent malocclusion type on mandibular symphysis inclination and morphology.

Methods: A total of 162 selected patients were divided into two groups: one group consisted of patients with one or two congenitally missing MnIs, and another group comprised patients without tooth agenesis. Patients in each group were categorized into three divergent malocclusion groups (hypodivergent, normodivergent and hyperdivergent) according to the Frankfort mandibular plane angle, with 27 patients per group. Lateral cephalograms were used to evaluate mandibular symphysis inclination and morphology. Two-way analysis of variance, simple main effect analysis, and Tukey's test were used for statistical comparisons.

Results: The agenesis group demonstrated a significantly greater retroclination of the mandibular symphysis than the non-agenesis group in the normodivergent group. In the hypodivergent and normodivergent groups, the agenesis group showed a significantly smaller area of the alveolar bone with thinner width and shorter height than the non-agenesis group.

Conclusion: For the Japanese orthodontic patients, MnI agenesis caused a significantly great retroclination of the mandibular symphysis in patients with normodivergent malocclusion and significantly small area of the alveolar bone with thin width and short height in patients with hypo- and normodivergent malocclusions.

Keywords; Mandibular symphysis, Mandibular incisor agenesis**,** Divergent malocclusion type**,** Japanese orthodontic subjects

Introduction

The mandibular symphysis is "orthodontically defined as the area that covers the mandibular symphyseal region on the lateral cephalogram"[1] and serves as a primary component for facial profile esthetic and the determination of mandibular incisor (MnI) positioning [2,3]. The mandible derives from the appearance of Meckel cartilage at 6 weeks of fetal life and consists of two halves that are connected by a fibrous cartilage called the mandibular symphysis at birth [4]. However, the mandibular symphysis fuses before or almost at the time occlusion begins [5]. Alveolar bone in the mandibular symphysis can grow vertically at a rate of 0.7-0.9 mm per year during childhood and puberty [6].

Some studies have shown that hyperdivergent malocclusion causes greater retroclination of the alveolar bone [3,7], with a narrower and longer mandibular symphysis, than hypodivergent and normodivergent malocclusions [7-10]. Another study suggested that alveolar bone height is not associated with divergent malocclusion type [11]. Some researchers have reported that skeletal class III malocclusion involves greater retroclination of the alveolar bone and a larger area of the mandibular symphysis with thinner width and longer height than the skeletal class I and II malocclusions [7,8,12], while others have reported no significant differences in mandibular symphysis width and height among sagittal malocclusion types [13]. These conflicting results may be attributed to differences in patient selection according to either sagittal or vertical skeletal malocclusion, measurement of the mandibular symphysis separated into alveolar or basal bone, and ethnic characteristics [14,15].

Previous studies have revealed that MnI agenesis induces great retroclination [1,16,17], and small width [17,18], height [19], and area of the mandibular symphysis [1]. Their studies have not categorized patients according to divergent malocclusion type. To our knowledge, there have been no studies concerning the effects of MnI agenesis and divergent malocclusion type on the mandibular symphysis. This study was performed to investigate the effects of MnI agenesis and divergent malocclusion type on mandibular symphysis inclination and morphology. The null hypothesis tested was that neither MnI agenesis nor divergent malocclusion type would affect mandibular symphysis inclination and morphology.

Materials and methods

This study protocol was approved by the Research Ethics Committee of our institution (ECNG-R-457).

Patients

Sample size was determined based on a priori power analysis for two-way analysis of variance (ANOVA) at an effect size of 0.25 (Cohen's medium effect size), an alpha error probability of 0.05, a power of 0.80, two degrees of freedom, and six groups [20]. Power analysis showed that 158 patients were required; the sample size in each group was set at 27 patients.

A total of 902 Japanese patients (557 female patients and 345 male patients) were retrospectively selected from the files of orthodontic patients who had attended the Nippon Dental University Niigata Hospital. Patient selection was based on the following criteria: (1) available lateral cephalograms and panoramic radiographs, (2) full eruption of all maxillary and mandibular permanent teeth up to the second molars

(excluding congenitally missing MnIs), (3) no prior orthodontic treatment, (4) no congenital disease, (5) no previous extraction of any permanent teeth, and (6) little or no MnI crowding. The patients selected were divided into two groups: one group consisted of 107 patients with one or two congenitally missing MnIs (group A), and another group comprised 795 patients without tooth agenesis (group C). Third molars were excluded from consideration in this study.

In each of groups A and C, patients were categorized into three divergent malocclusion groups according to the Frankfort mandibular plane angle (FMA): hypodivergent group (<20.6° for female patients and <18.3° for male patients), normodivergent group (20.7-31.5° for female patients and 18.4-26.2° for male patients), and hyperdivergent group (>31.6° for female patients and >26.3° for male patients). The categories for these subgroups were based on the Japanese FMA criteria (26.1°±5.4° for female patients and 22.3°±3.9° for male patients) [21]. A total of 162 patients, 27 per group, were selected using the random numbers table. Figure 1 shows the classification of patients. Table 1 shows that the distribution of each congenitally missing mandibular incisors pattern and mean patient age in each group.

ANB angle in the divergent malocclusion group

The patients in the six divergent malocclusion groups (three groups each in groups A and C) were classified into three sagittal skeletal malocclusion types based on the ANB angles $(3.3°±1.8°$ for female patients and $2.8°±2.0°$ for male patients): skeletal Class I (1.5–5.1° for female patients and 0.8-4.8° for male patients), skeletal Class II (>5.1° for female patients and >4.8° for male patients), and skeletal Class III (<1.5° for female patients and <0.8° for male patients) [21].

Tooth agenesis

The diagnosis of tooth agenesis was performed using panoramic radiographs

acquired at the age of ≥14 years. If patients underwent the first orthodontic examination before 14 years of age, only panoramic radiographs acquired at the age of ≥14 years were used. A tooth was regarded as agenesis when no mineralization of the tooth crown was recognized on panoramic radiographs. The age of 14 years was adopted to avoid inaccurate diagnosis due to delayed tooth calcification, based on the findings by Garn and Lewis [22] that the upper age limit for agenesis in third molars, which were the last permanent teeth to calcify, was 14 years.

Cephalometric analysis

Lateral cephalograms were acquired using the same equipment (CX-150 SK, Asahi Roentgen, Kyoto, Japan) with a magnification factor of 1.1 at standard settings; these images were used to evaluate mandibular symphysis inclination and morphology. The calibration ruler of included in each cephalogram was used and adjusted to standardize the enlargement of 10%. After calibration, all cephalograms were traced and measured by one investigator (S.Y.). Six reference points and one reference line were configured, and five angular, five linear, and three area measurements were made on each tracing paper (Figure 2, Table 2). Angular, linear, and area measurements were made to with a computer system including an ImageJ analysis software (version 1.52, NIH, MD, USA) to the nearest 0.1°, 0.1 mm, and 0.1 mm², respectively.

Statistical Analysis

Statistical analyses were performed using BellCurve for Excel software (version 3.20, SSRI, Tokyo, Japan). The means and standard deviations were calculated for each measurement in each group. Two-way ANOVA, simple main effect analysis, and Tukey's test were performed to analyze the effects of MnI agenesis and divergent malocclusion type on mandibular symphysis inclination and morphology. Two-way

ANOVA was used to test the effects of these two factors on mean patient age among the six divergent malocclusion groups (three groups each in groups A and C), and mean ANB angles among these six groups in each sagittal skeletal malocclusion type (Class I, II, and III). The χ^2 test were performed to determine any significant differences in the distribution of sex and the number of patients with three sagittal skeletal malocclusion types among these six groups. These parametric tests were performed after testing the normality of the distribution and variance homogeneity. The Kruskal-Wallis test was used to examine whether the number of patients significantly differed according to the pattern of congenitally missing MnIs among the three divergent malocclusion groups in group A. The level of statistical significance for all analyses was set at P<0.05.

Measurement error

To assess measurement errors, 40 randomly selected cephalograms were remeasured by the same investigator (S.Y.) at 3 months after the first measurements. Random errors, evaluated with the Dahlberg formula [23], were found to be less than 0.9° for angular measurements, less than 0.3 mm for linear measurements, and less than 1.7 mm² for area measurements, which were unlikely to substantially influence the results in this study. Paired t-tests indicated no systematic measurement errors.

Results

The x^2 test showed no significant sex differences in numbers of patient among the six groups (three groups each in groups A and C) (Figure 1). Two-way ANOVA indicated that the main effects of MnI agenesis and divergent malocclusion type on mean patient age were not significant and that there was no significant interaction between these two factors after assessment of data normality and variance homogeneity using the Shapiro-Wilk and Levene's tests, respectively (Table 1). The Kruskal-Wallis test demonstrated no significant difference in the number of patients with each pattern of congenitally missing MnIs among the three divergent malocclusion groups in group A (Table 1). These results suggested that patient selection in this study was suitable for statistical comparisons.

The Shapiro-Wilk and Levene's tests confirmed the data normality and variance homogeneity for measurements of mandibular symphysis inclination and morphology, respectively.

Two-way ANOVA showed significant interactions between MnI agenesis and divergent malocclusion type in IdB-MP angle (alveolar bone inclination) and L1-MP angle (MnI inclination); no significant interactions were detected with respect to other measurements (Table 3). Two-way ANOVA also showed significant differences in IdmMe-MP angle (mandibular symphysis inclination), Id-MP dimension (mandibular symphysis height), Id-B dimension (alveolar bone height), B-th dimension (alveolar bone width), and AB area (alveolar bone area); there were no significant differences in Id-B-Me angle (alveolar bone to basal bone inclination), B-MP dimension (basal bone height), and BB area (basal bone area) between groups A and C and/or among the divergent malocclusion groups (Table 3). Additionally, significant differences in BMe-MP angle (basal bone inclination), and MS area (mandibular symphysis area), and in Pog-th dimension (basal bone width) were observed between groups A and C and among the divergent malocclusion groups, respectively (Table 3).

Mandibular symphysis inclination

Simple main effect analyses and post hoc Tukey's tests verified significant differences in the IdmMe-MP angle between groups A and C in the hypo- and

normodivergent groups; they also demonstrated significant differences in the BMe-MP, IdB-MP, and L1-MP angles between groups A and C in the normodivergent group. Furthermore, group A exhibited a significant difference in the IdB-MP angle between the hypo- and normodivergent groups; group A also demonstrated significant differences in the L1-MP angle between the hypo- and normodivergent groups and between the hypo- and hyperdivergent groups (Table 3). Additionally, group C exhibited a significant difference in the IdmMe-MP angle between the hypo- and hyperdivergent groups; group C also demonstrated significant differences in the IdB-MP and L1-MP angles between the hypo- and hyperdivergent groups and between the normo- and hyperdivergent groups (Table 3).

Mandibular symphysis morphology

 Post hoc Tukey's tests revealed significant differences in the Id-MP, Id-B, and B-th dimensions between groups A and C in the hypo- and normodivergent groups; they also showed significant differences in the AB and MS areas between groups A and C in the three divergent malocclusion groups (Table 3). These tests also showed that in group A, there were significant differences in the Id-MP dimension between the hypoand hyperdivergent groups and between the normo- and hyperdivergent groups; significant differences in the Id-B dimension among the three divergent malocclusion groups; and significant differences in the Pog-th dimension and AB area between the hypo- and hyperdivergent groups (Table 3). Moreover, group C exhibited significant differences in the Id-MP dimension between the hypo- and normodivergent groups and between the hypo- and hyperdivergent groups; significant differences in the Id-B dimension among the three divergent malocclusion groups; significant differences in the B-th dimension and AB area between the hypo- and hyperdivergent groups; and significant differences in the Pog-th dimension between the hypo- and hyperdivergent groups and between the normo- and hyperdivergent groups (Table 3).

ANB angle in the divergent malocclusion group

Table 4 shows the numbers of patients with skeletal Class I, II, and III malocclusions in each of the six groups, along with their mean ANB angles. The χ^2 test showed no significant difference in the numbers of patients according to sagittal skeletal malocclusion type among the six divergent malocclusion groups (Table 4). In each sagittal skeletal malocclusion type, the Shapiro–Wilk and Levene's tests confirmed the data normality and variance homogeneity for the ANB angles, respectively. Twoway ANOVA showed that MnI agenesis and divergent malocclusion type had no significant effect on the mean ANB angle in any sagittal skeletal malocclusion type, with no significant interaction (Table 4).

Discussion

The null hypothesis, that neither MnI agenesis nor divergent malocclusion type would affect mandibular symphysis inclination and morphology, was rejected. Our findings may suggest that the effect of divergent malocclusion type on mandibular symphysis inclination and morphology should be considered when planning orthodontic treatment in patients with MnI agenesis.

Mandibular symphysis inclination

In the normodivergent group, the agenesis group demonstrated significantly greater retroclination of the mandibular symphysis than the non-agenesis group, but not in the hypo- and hyperdivergent groups. Our finding in the normodivergent group was partially supported by the results of previous studies that patients with MnI agenesis [1] and with severe tooth agenesis (including MnIs) [16] had significantly greater

retroclination of the MnIs than the non-agenesis group. In contrast to our study, the previous studies did not classify patients according to divergent malocclusion type [1,16]. It was speculated that mandibular symphysis retroclination in the normodivergent group might be caused by retroclination of the MnIs, as was evidenced by the L1-MP angle in this study. This speculation could be warranted by the results of correlation analyses, which showed significantly positive correlations of the L1-MP angle with the IdmMe-MP, BMe-MP, and IdB-MP angles in this study. Our results were supported by Khateeb et al [12] and Yamada et al [24], who found that when the MnIs inclined lingually, the associated mandibular symphysis also inclined lingually. This MnI retroclination might be due to a disturbance in tongue-lip pressure balance and a lack of lingual support as a consequence of MnI agenesis [1].

Ozdemir et al [25] demonstrated that patients with hypodivergent malocclusion had a thicker cortical bone than patients with normodivergent malocclusion. Irrespective of agenesis or non-agenesis of the MnIs, the thick cortical bone may restrict the retroclination of the MnIs and symphysis in patients with hypodivergent malocclusion, thus causing no significant difference in mandibular symphysis inclination between groups A and C in this study. Patients with hyperdivergent malocclusion had difficulty in closing their lips without the excessive use of perioral muscles [26,27]. This weak lip closure may induce low lip pressure and limit the retroclination of the MnIs and symphysis in patients with hyperdivergent malocclusion who exhibit MnI agenesis, thus bringing no significant difference in mandibular symphysis inclination between groups A and C in this study.

In the agenesis group, the patients with normodivergent malocclusion exhibited significantly greater alveolar bone retroclination than patients with hypodivergent malocclusion. This finding was attributed to the fact that in the normodivergent group,

the thin cortical bone might cause the great lingual inclination of the MnIs due to the lip pressure [25], which was proved by the L1-MP angle in this study.

The hyperdivergent group exhibited significantly greater retroclination of the alveolar bone than the hypo- and normodivergent groups of the non-agenesis group. Our results were partially in agreement with findings by Jain et al [7] and Mangla et al [9] that greater retroclination of the mandibular symphysis was present in patients with hyperdivergent malocclusion than in patients with hypodivergent malocclusion. Anwar et al [28] reported that the retroclination and extrusion of the MnIs served as a compensatory factor for an increased vertical skeletal relationship. Therefore, in our patients with hyperdivergent malocclusion, alveolar bone retroclination might have been induced by dental compensation for MnI retroclination.

Mandibular symphysis morphology

In the hypo- and normodivergent groups, the agenesis group showed a significantly smaller area of alveolar bone thinner width and shorter height than the non-agenesis group. Our findings were consistent with those of Bertl et al [29], who reported that the mandibular alveolar bone was narrow and short in patients with tooth agenesis compared with that in controls. However, Bertl et al [29] did not classify patients according to divergent malocclusion type. From a genetic perspective, Msx1 homeodomain deficiency causes tooth agenesis and mandibular alveolar bone deficiency [30], resulting in a switch from hyperdivergent malocclusion to normo- or hypodivergent malocclusion [31]. Therefore, tooth agenesis might coincide with the small area of alveolar bone with thin width and height in patients with normo- and hypodivergent malocclusions.

In our hyperdivergent group, the agenesis group had a significantly smaller area of alveolar bone than the non-agenesis group, although neither alveolar bone height nor

thickness exhibited significant differences. These results might support the previous finding that area measurements of partial skeletal structure were useful for identifying the effects of tooth agenesis [32,33].

Irrespective of the presence or absence of MnIs, patients with hyperdivergent malocclusion exhibited a significantly larger area of alveolar bone with longer height and thinner width of basal bone than patients with hypodivergent malocclusion. It was considered that in our patients with hyperdivergent malocclusion, the increase in FMA caused dentoalveolar compensation and mandibular symphysis remodeling, resulting in decreased basal bone width and increased alveolar bone height and area. This consideration was corroborated by the findings of previous studies [8,34-36]. Hong et al [34] and Guerino et al [35] showed that a thin mandibular symphysis was attributed to overeruption of the tooth and alveolar bone in patients with hyperdivergent malocclusion to compensate the vertical skeletal dimension. Berlanga et al [8] reported that the extent of mandibular symphysis remodeling was determined by an increase in the FMA. Frost [36] demonstrated that "bone remodeling maintained bone mass by resorption and formation drifts". The mandibular symphysis remodeling for maintaining bone mass might be evidenced by our results that the increasing alveolar bone brought led to decreasing basal bone width with the increasing FMA, thus avoiding significant differences in the MS area among the three divergent malocclusion groups.

The alveolar bone width was significantly smaller in patients with hyperdivergent malocclusion than in patients with hypodivergent malocclusion in the non-agenesis group; this difference was not present in the agenesis group. This finding indicated that for the mandibular symphysis, the alveolar bone width was predominantly affected by MnI agenesis rather than the divergent malocclusion type. MnI agenesis

might limit morphological changes in alveolar bone of the mandibular symphysis caused by increased FMA.

ANB angle in the divergent malocclusion group

Previous studies have not shown consistent findings regarding the association between mandibular symphysis morphology and the divergent malocclusion type; they did not mention the ANB angle in the patients with different divergent facial types [7,11,35]. One of the possible reasons for the contrary results might be due to the concomitant changes in mandibular symphysis morphology caused by different FMA and ANB angles. In this study, 27 patients in each divergent malocclusion group were classified into the skeletal Class I, II and III according to ANB angle. Two-way ANOVA showed no significant differences in mean ANB angles among the three divergent malocclusion groups or between groups A and C in each of the skeletal Class I, II, and III (Table 4), suggesting that by chance, this comparative study of divergent malocclusion types was not affected by ANB angle.

Limitations

One of the limitations of this study was that the molar anteroposterior relationship for the six groups was not considered. Andrews [37] reported that mandibular central incisors exhibited signs of positional compensation in untreated Class II orthodontic patients with the molar anteroposterior discrepancy of more than 3.0mm. Evaluating the molar anteroposterior relationship might have provided additional insight, as the skeletal status may not fully reflect the molar anteroposterior discrepancy severity and its consequent overjet. Another limitation is about the mandibular plane set as the reference line. Mandibular plane is one of the major measurements in cephalometric analysis and has been set as the reference line for evaluating mandibular symphysis morphology in previous studies [1,7], but this reference line

may change depending on different divergent malocclusion types. For example, the hypodivergent malocclusion patients with a flat mandibular plane angle would have a shorter reference line than the hyperdivergent malocclusion patients with steep mandibular plane angle. The hypodivergent malocclusion patients may have had longer B-th and Pog-th distances. This limitation would not change our findings that the patients with hypodivergent malocclusion tended to have thicker alveolar bone and basal bone width than the patients with hyperdivergent malocclusion, however it is thought that a measurement method that can correct this factor is necessary in the future. Measurement of the molar anteroposterior relationship on a model [37] and precision imaging by CBCT with the mandibular plane oriented horizontally parallel [38], is necessary to improve the evidence.

Conclusion

For the Japanese orthodontic patients, MnI agenesis caused a significantly great retroclination of the mandibular symphysis in patients with normodivergent malocclusion and significantly small area of the alveolar bone with thin width and short height in patients with hypo- and normodivergent malocclusions.

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Figure legends

Fig. 1 Flow chart showing the numbers of patients, with their sex. M, male; F, female

Fig. 2 Reference points and line used. Id, infradentale; LId, lingual infradentale; Idm, midpoint of infradentale and lingual infradentale; B, point B; Pog, pogonion; Me, menton; MP, mandibular plane (a tangent to the lower border of the mandible through the Me)

Figures

Figure.1

Tables

Table 1. Distribution of each congenitally missing mandibular incisors pattern and mean patient age in each group

n indicates number of patients; y: years; m: months.

Table 3. Mandibular incisor and symphysis inclination and morphology measurements

SD indicates standard deviation; *NS:* not significant.

	Hypodivergent group				Normodivergent group			Hyperdivergent group		
Skeletal Class I	n	Mean	SD	n	Mean	SD	n	Mean	SD	
Group A	15	2.6	0.7	13	3.1	1.2	13	3.3	1.4	
Group C	15	3.1	1.1	14	3.1	1.2	12	3.8	1.3	
Skeletal Class II	n	Mean	SD	n	Mean	SD.	n	Mean	SD	
Group A	4	5.8	0.5	8	6.4	1.0	10	6.8	1.3	
Group C	6	6.2	1.1	9	6.2	0.7	11	7.1	1.3	
Skeletal Class III	n	Mean	SD	n	Mean	SD	n	Mean	SD	
Group A	8	-1.5	1.7	6	-1.4	2.1	4	-0.9	2.1	
Group C	6	-1.2	2.1	4	-0.2	1.2	4	-1.1	2.2	

Table 4. Numbers of patients and mean ANB angles

n indicates number of patients; *SD:* standard deviation.