WITHDRAWN:
Relationship Between Maxillary Sinus Hypoplasia and Maxillary Occlusal Cant: A Cone Beam CT Study

Noura A. Alsufyani

alsufyan@ualberta.ca

King Saud University

Michael P. Major

University of Alberta

Paul W. Major

University of Alberta

Research Article

Keywords: Maxilla, Maxillary sinus, Facial asymmetry, Maxillofacial Development

Posted Date: June 18th, 2021

DOI: https://doi.org/10.21203/rs.3.rs-599073/v1

Additional Declarations: No competing interests reported.

Version of Record: A version of this preprint was published at BioMed Research International on February 18th, 2022. See the published version at https://doi.org/10.1155/2022/4651514.

EDITORIAL NOTE:

The full text of this preprint has been withdrawn by the authors while they make corrections to the work. Therefore, the authors do not wish this work to be cited as a reference. Questions should be directed to the corresponding author.

Abstract

Background: maxillary sinus hypoplasia (MSH) has been reported to cause a cant in the orbital plane. No similar reports exist about the possible impact on the maxilla. The aim of this study was to assess if MSH is associated with maxillary occlusal plane (MOP) cant, and if dental or mandibular factors influenced the existence of the MOP cant.

Methods: 80 cone beam CT images of subjects with MSH were analyzed for type of MSH, degree of MOP cant, open or cross bite, mandibular asymmetry, and degenerative joint disease of the temporomandibular joints. The subjects were compared with a control group matched by age and gender.

Results: The degree of MOP cant (range 0-5.1°) was not statistically significantly different in unilateral vs bilateral MSH, or between the different types of MSH. The frequency of open bite, crossbite, mandibular asymmetry, or degenerative joint disease in unilateral vs bilateral MSH, or between the different types of MSH was not statistically significantly different. Between the case and control, there was no statistically significant difference in the degree of MOP cant (1.3±1.1° vs 1.1±0.9°, respectively), or frequency of dental and mandibular factors. There was low positive linear correlation between MOP cant and mandibular symmetry in MSH subjects (Spearman's rho= 0.23) and controls (Spearman's rho= 0.187).

Conclusions: The data suggests a view of the alveolar bone as the adaptable skeletal unit to ensure and bridge the functionality between the nasomaxillary complex and TMJ system – two systems with very different function, and therefore largely independent "matrix units".

Background

The maxillary sinus is one of four paranasal sinuses that are air-filled extensions of the nasal cavity. The maxillary sinus reaches final pneumatization inferiorly with the eruption of maxillary teeth by the age of 15–18 years.[1] At a prevalence of 1.5–10%, the process by which abnormal pneumatization would cause maxillary sinus hypoplasia (MSH) is not known and poorly described.[2–4]

The presence of MSH has been shown to cause asymmetries in its superior and medial neighbors; the orbit and nasal cavity, respectively. Inferior positioning of orbital floor or bowing of the lateral wall of the nasal cavity ipsilateral to the MSH have been documented.[2, 3, 5–8] Canting of the orbital floor was even proposed as an added feature to classify MSH.[7]

From the third week of intra-uterine life until 18 years of age, the craniofacial complex growth is affected by genetic and complex external and internal environmental factors which guide or alter the pattern of growth.[9, 10] The developmental complexity is understandable given the co-existence of dental structures, temporomandibular joints, osseous and soft tissues of the maxilla, mandible, and cranial base.

Asymmetries of the inferior neighbor, i.e. the alveolar process of maxilla, due to MSH have not been thoroughly studied. Selcuk et al found that 25.8% of MSH was associated with elevation of the canine

fossa.[11] Out of 495 clinical asymmetry patients, 36% of asymmetries were found in the midface and 74% were in the chin.[10] However, there are no studies highlighting the possible association of MSH with dento-alveolar cant of the maxilla, mandibular or TMJ asymmetry.

In the presence of MSH, would an asymmetry in the alveolar process of the ipsilateral maxilla exist? If so, would the mandible also show an asymmetry?

The primary objective of this study was to assess whether MSH is associated with maxillary occlusal plane (MOP) cant. Secondary objectives were to assess if dental or mandibular factors influenced the existence of the maxillary occlusal plane cant.

Methods

Cone beam CT data

The study received approval (Pro00089618) from the Health Research Ethics Board- Health Panel, University of Alberta, Canada. It was based on retrospective maxillofacial CBCT images that included the maxillary sinuses and was used in a previous study analyzing MSH and associated nasal anomalies.[8]

The CBCT scans included subjects referred to dental imaging clinic to rule out pathology in the TMJs. Scans excluded: subjects younger than 18 (to ensure full development of maxillary sinus), edentulous jaws (to eliminate sampling bias due to sinus pneumatization), incomplete image of the maxillary sinuses, poor scan quality or motion artifact, evident history of trauma or surgery to the maxilla or maxillary sinuses, congenital disease or syndrome affecting the maxillofacial complex.

To standardize the orientation of the CBCT volume, the following planes were used:

Horizontal plane X: connecting R and L Porion is the 0 plane

Sagittal plane Z: connecting R and L Porion with Right Orbitale is made parallel to 0 plane

Coronal plane Y: mid-sagittal plane (bisecting crista galli and fronto-nasal suture) is perpendicular to the above planes.

MSH analysis

The classification of MSH by Bolger[2] was used by the principal investigator (oral & maxillofacial radiologist) by primarily assessing the coronal section, confirmed by axial and sagittal sections, and comparing right and left sides. Two variables were collected:

- Unilateral vs bilateral MSH
- MSH type: Type I hypoplasia: mild hypoplasia, normal uncinate process, and a patent infundibular tract. Type II: sinus hypoplasia, hypoplastic uncinate process, and an ill-defined or absent

infundibular passage. Type III is characterized by profound sinus hypoplasia, “cleft-like”, and an absent uncinate process.[2]

MOP Cant analysis

Intermolar line connecting buccal most point of the furcation between the mesiobuccal and distobuccal roots of #16 and #26 (or #17 and #27) was used.

Zero cant is when the angle formed between intermolar line and mid-sagittal plane is 90˚. Any deviation is measured as possible cant. Using five cases (not included in this study), intra-examiner reliability in identifying furcation points (measured by intra-class correlation coefficient *ICC* and maximum error in mm at cut off of 1.5mm = 3 pixels) and MOP cant were measured three times, one week apart by the PI.

Dental and mandibular analysis

1. Mandibular asymmetry: Subjectively recorded as present/absent and objectively measured as the distance (mm) of lingual foramen to the mid-sagittal plane.

2. Anterior open bite: present/absent

3. Crossbite: present/absent

4. Degenerative joint disease: present/absent.

Statistical analysis

To adequately assess the possible association of MSH with MOP cant, dental and mandibular factors, a control group, i.e. subjects without maxillary sinus hypoplasia, matched for age and gender were analyzed for the same variables.

To assess differences in MOP cant among the MSH, dental and mandibular variables, Chi-squared or Fisher’s Exact, and Kruskal Wallis tests were used for categorical and numerical data, respectively.

To assess differences in MOP cant among the dental and mandibular variables between MSH subjects and their matched controls, McNemar or Fisher’s Exact tests were used for categorical data and Wilcoxon signed – rank test for numerical data. Linear correlation between MOP cant and mandibular asymmetry was tested using Spearman rho, in both groups. SPSS (Version 25.0, IBM Corp., Armonk, New York, USA) was used for all tests.

Results

The intra-examiner reliability was excellent for marking the maxillary furcation for the right and left maxillary molar (ICC range 0.97–100). Mean error ranged from 0.08 to 0.20 mm for the X, Y, Z coordinates for maxillary furcation with maximum examiner error ranging from 0.10 to 0.65 mm. The

intra-examiner reliability was excellent (ICC 0.90) for MOP cant with mean error of 0.4 ± 0.2 degrees and maximum error of 0.8˚.

The sample consisted of 80 subjects with MSH and 80 matched controls (total n = 160). There were 100 females and 60 males, mean age 36.3 ± 13.4 years. The data was not normally distributed and non-parametric tests were used.

Within the MSH group, most were unilateral (82.5%) and type I (70%). The degree of MOP cant was not statistically significantly different in unilateral vs bilateral MSH, or between the different types of MSH (Type I, II, or III hypoplasia), Table 1. The frequency of open bite, crossbite, mandibular asymmetry, or degenerative joint disease in unilateral vs bilateral MSH, or between the different types of MSH was not statistically significantly different, Table 1.

Table 1
Difference in MOP cant, dental and mandibular factors among MSH factors within MSH subjects (n = 80)

	Unilateral **66 (82.5%)**	**Bilateral** **14 (17.5%)**	**P value**	**Type I** **56 (70%)**	**Type II** **20 (25%)**	**Type III** **4 (5%)**	**P value**
MOP cant mean ± SD° (range)	1.32 ± 1.2 (0-5.1)	1.25 ± 0.92 (0.2–3.1)	0.80*	1.2 ± 1 (0-5.1)	1.7 ± 1.4 (0–5)	1.1 ± 1.7 (0-3.7)	0.21*
Mandibular asymmetry > 5mm N(%)	16 (20%)	0	0.06	11 (13.8%)	5 (6.3%)	0	0.5
TMJ-DJD N(%)	19 (23.8%)	2 (2.5%)	0.26	15 (18.8%)	6 (7.5%)	0	0.4
Open bite N(%)	11(13.8%)	1 (1.3%)	0.36	6 (7.5%)	4 (5.0%)	2 (2.5%)	0.08
Cross bite N(%)	7 (8.8%)	2 (2.5%)	0.69	5 (6.3%)	4 (5.0%)	0	0.3
*Kruskal-Wallis test Chi-squared test Fisher's exact test TMJ DJD: temporomandibular joint-degenerative joint disease							

Between the MSH subjects and their matching controls (total n = 160), there was no statistically significant difference in the frequency of dental and mandibular factors, Table 2. There was non-significant, low positive linear correlation between MOP cant and mandibular symmetry in MSH subjects (Spearman's rho = 0.23, p-value 0.04) and controls (Spearman's rho = 0.187, p-value 0.09), Fig. 1.

Table 2
Difference in MOP cant, dental and mandibular factors between MSH subjects and their controls

	Case n = 80	Control n = 80	P value
MOP cant mean ± SD° (range)	1.3 ± 1.1 (0-5.1)	1.1 ± 0.9 (0-4.2)	0.58*
Mandibular Asymmetry mean ± SD mm (range)	1.6 ± 1.7 (0–7)	1.5 ± 1.7 (0–6.4)	0.90*
TMJ-DJD N (%)	Absent 59 (73.8%) present 21 (26.3%)	Absent 63 (78.8%) present 17 (21.3%)	0.61
Open bite N (%)	Absent 68 (85%) present 12 (15%)	Absent 71 (88.8%) present 9 (11.3%)	0.65
Cross bite N (%)	Absent 71 (88.8%) present 9 (11.3%)	Absent 79 (98.8%) Present 1 (1.3%)	0.88
*Wilcoxon Signed Rank Test Chi-squared test McNemar test TMJ DJD: temporomandibular joint-degenerative joint disease			

Discussion

The method used to assess the MOP cant was similar to the work done by Kheir et al[9] and was found to be reliable with clinically acceptable measurement error; less than 1 mm.

In the MSH group, the sinus hypoplasia was mostly unilateral and mild (type I). Although the population represents subjects referred to rule-out pathology in the TMJs, degenerative joint disease was found in only 21 (26.3%). Similarly, mandibular and dental factors were not commonly present; mandibular asymmetry 16 (20%), open bite 12 (15%), and crossbite nine (11.3%). Their frequency was not statistically

significant between unilateral and bilateral MSH nor between the different types of MSH. As such mandibular asymmetry, open bite, and crossbite were not considered as possible covariates for the analysis of MOP cant. The mean MOP cant was small, not exceeding 2°, across all types of MSH as well as unilateral versus bilateral MSH. The maximum MOP cant was 5.1°. Padwa et al found that cants greater than 4° were detected clinically by both untrained and trained observers.[12] Olivares et al agree with this finding and furthermore showed that although orthodontists had better detection occlusal cant of 2° compared to general dentists and laypersons, all examiners evaluated them as esthetically acceptable.[13] Thus, although some occlusal cants within MSH group can be detected clinically, their existence was not affected by the fact that the MSH was unilateral or severe, for example.

In the control group, the extent of mandibular asymmetry, the presence of TMJ-DJD, open bite, and crossbite were not statistically significantly different than the MSH group; were not a common occurrence. Moreover, the linear relationship between mandibular asymmetry in mm and MOP cant was very low and not significant. As such mandibular asymmetry, open bite, and crossbite were not considered as possible covariates for the analysis of MOP cant. The mean MOP cant was not statistically significantly different between the MSH group and their matched controls. In both groups, the MOP cant did not exceed 2°.

Although there are reports of canine fossa elevation in cases with MSH, the possible impact on dental occlusal plane was not assessed.[11, 14]

If MSH was found to alter the position of the inferior orbital wall and lateral wall of nasal cavity, why wasn't such impact significant in the alveolar process of maxilla? There are two unique facts about the inferior neighbor of the maxillary sinus; the TMJs and teeth. Based on Moss' functional matrix theory, the development and change in size, shape, and position of the skeletal unit responds to the morphogenetic primary demand of the non-skeletal tissues i.e. the functional matrices.[15] As such, the development of the TMJ is largely dictated by the surrounding non-skeletal tissues, mainly muscles, to reach a state of equilibrium and to ensure that the osseous tissues are not under pressure.[16] To maintain the joint in a musculo-skeletally stable position, the TMJs influence the position of posterior and anterior teeth. The role of posterior teeth is to maintain vertical dimension of occlusion and the anterior teeth are crucial for anterior guidance (movement) of the mandible.[16, 17] The location of teeth during eruption and post-eruption is also largely guided by buccal and lingual muscular forces, from tongue and cheeks. Therefore, the functional matrix i.e. muscles and teeth influence how the TMJs, alveolar process of maxilla and mandible respond.[16–18]

In addition to Moss' theory, Wolff's law explains that remodeling of bone responds to loading. Cyclic loading exists in the maxilla and mandible through the occlusal forces generated and maintained by the teeth, so the metabolism of bone adapts to the loads exerted by teeth.[19] Collectively, the functional demands and physiologic response described by Moss and Wolff may explain why the maxilla and MOP did not significantly cant in the presence of MSH.

An example is shown in in Fig. 2, a type III (severe) MSH with evident depression in the anterior wall of sinus however the left maxillary premolar maintains the occlusal plane symmetry and the underlying alveolar process is well-formed due to the occlusal forces exerted by the posterior maxillary teeth, and as such did not follow the MSH.

Although MOP cant did not show strong correlation with mandibular deviation or TM-DJD, cases with a more significant MOP cant (4.2° in control case and 5°in MSH case) demonstrated unilateral condylar hypoplasia, Fig. 3. This may indicate condyle growth has a larger influence on occlusal cant than MSH.

To take into consideration the complex factors, larger sample size and advanced 3D analysis of the mandible would be needed to test such influence. However, the data suggests a view of the alveolar bone as the adaptable skeletal unit to ensure and bridge the functionality between the nasomaxillary complex and TMJ system – two systems with very different function, and therefore largely independent “matrix units.” The high degree of plasticity of alveolar bone demonstrated during orthodontic treatment is strong pieces of collaborative evidence.

The main shortcoming of this study is related to the sample size. The complexity of craniofacial-mandibular growth would require larger sample size to allow for robust statistical analyses. Three-dimensional analysis of the osseous and dental structures would also be required to better measure changes rather than linear and angular measures.

Conclusion

The presence of MSH did not statistically significantly cause a cant in the MOP and was not associated with mandibular asymmetry, open bite or crossbite. These findings suggest the alveolar complex is able to functionally compensate for a diminished volume of underlying maxillary basal bone caused by MSH. Instead, the influence of the TMJs, the cyclic forces exerted by mastication, the need for functional occlusion, and the maintenance of musculoskeletal stability, may surpasses the influence of maxillary sinus size on lower face development including the occlusal plane.

Abbreviations

MSH
maxillary sinus hypoplasia
MOP
maxillary occlusal plane
TMJ
temporomandibular joint
DJD
degenerative joint disease
CBCT

cone beam computed tomography
ICC
intra-class correlation coefficient

Declarations

Ethics approval and consent to participate: The study received approval from the Health Research Ethics Board- Health Panel, University of Alberta, Canada [Pro00089618]

Consent for publication: Not applicable

Availability of data and material: The data underlying this article cannot be shared publicly due to ethical reasons. After obtaining necessary institutional ethical approval, the data can be shared on reasonable request to the corresponding author.

Competing interests: The authors declare that they have no competing interests

Funding: Not applicable

Authors' contributions: NA contributed to the conception, design of the work, acquisition, analysis, and interpretation of data, and drafted the work. MM and PM contributed to analysis, and interpretation of data, and substantively revised the work. All authors read and approved the final manuscript and are personally accountable for their contributions.

Acknowledgments: Not applicable

References

1. Daniels JS, Ali I, Al Bakri IM, Sumangala B. Pleomorphic adenoma of the palate in children and adolescents: A report of 2 cases and review of the literature. J Oral Maxillofac Surg. 2007;65:541-549.

2. Bolger WE, Woodruff Jr. WW, Morehead J, Parsons DS. Maxillary sinus hypoplasia: Classification and description of associated uncinate process hypoplasia. Otolaryngol Head Neck Surg. 1990;103:759-765.

3. Price DL, Friedman O. Facial asymmetry in maxillary sinus hypoplasia. Int J Pediatr Otorhinolaryngol. 2007;71:1627-1630.

4. Sánchez Fernández JM, Anta Escuredo JA, Sánchez Del Rey A, Santaolalla Montoya F. Morphometric study of the paranasal sinuses in normal and pathological conditions. Acta Oto-Laryngol. 2000;120:273-278.

5. Wake M, Shankar L, Hawke M, Takeno S. Maxillary sinus hypoplasia, embryology, and radiology. Arch Otolaryngol Head Neck Surg. 1993;119:1353-1357.

6. Erdem T, Aktas D, Erdem G, Mimam MC, Ozturan O. Maxillary sinus hypoplasia. Rhinology. 2002;40:150-153.

7. Sirikçi A, Bayazit Y, Gumusburun E, Bayram M, Kanlikana M. A new approach to the classification of maxillary sinus hypoplasia with relevant clinical implications. Surg Radiol Anat. 2001;22:243-247.

8. Alsufyani N, El-Hakim H, Major P. Prevalence of maxillary sinus hypoplasia and association with variations in the sinonasal complex: A cone beam CT study. Clin Oral Investig. 2021; doi: 10.1007/s00784-021-03854-3.

9. Kheir NA, Kau CH. The use of three-dimensional imaging to evaluate the effect of conventional orthodontic approach in treating a subject with facial asymmetry. Ann Maxillofac Surg. 2016;6:105-112.

10. Severt TR, Proffit WR. The prevalence of facial asymmetry in the dentofacial deformities population at the university of north carolina. Int J Adult Orthodon Orthognath Surg. 1997;12:171-176.

11. Selcuk A, Ozcan KM, Akdogan O, Bilal N, Dere H. Variations of maxillary sinus and accompanying anatomical and pathological structures. J Craniofac Surg. 2008;19:159-164.

12. Padwa BL, Kaiser MO, Kaban LB. Occlusal cant in the frontal plane as a reflection of facial asymmetry. J Oral Maxillofac Surg. 1997;55:811-6

13. Olivares A, Vicente A, Jacobo C, Molina SM, Rodríguez A, Bravo LA. Canting of the occlusal plane: Perceptions of dental professionals and laypersons. Med Oral Patol Oral Cir Bucal. 2013;18:e516-20

14. Geraghty JJ, Dolan KD. Computed tomography of the hypoplastic maxillary sinus. Ann Otol Rhinol Laryngol. 1989;98:916-918.

15. Moss ML. The functional matrix hypothesis revisited. 1. the role of mechanotransduction. Am J Orthod Dentofacial Orthop. 1997;112:8-11

16. Okeson J. Management of Temporomandibular Disorders and Occlusion. 8th ed. St. Louis, Missouri: Elsevier Mosby; 2019.

17. Björk A, Skieller V. Facial development and tooth eruption. an implant study at the age of puberty. Am J Orthod. 1972;62:339-383.

18. Moss ML, Salentijn L. The primary role of functional matrices in facial growth. Am J Orthod. 1969;55:566-577.

19. Frost HM. A 2003 update of bone physiology and wolff's law for clinicians. Angle Orthod. 2004;74:3-15.

Figures

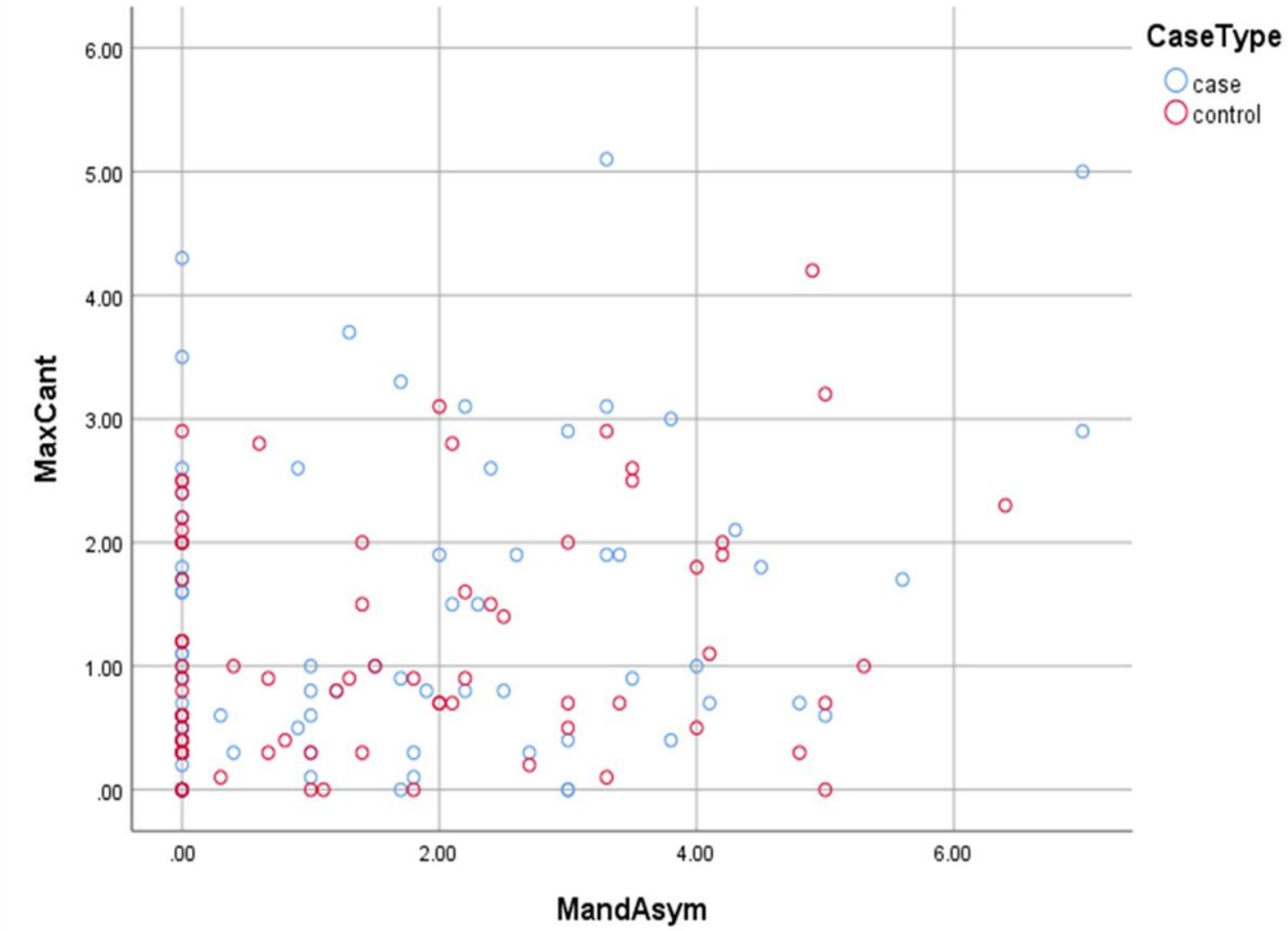

Figure 1

Scatter plot of linear correlation between mandibular asymmetry (mm) and MOP cant (°)

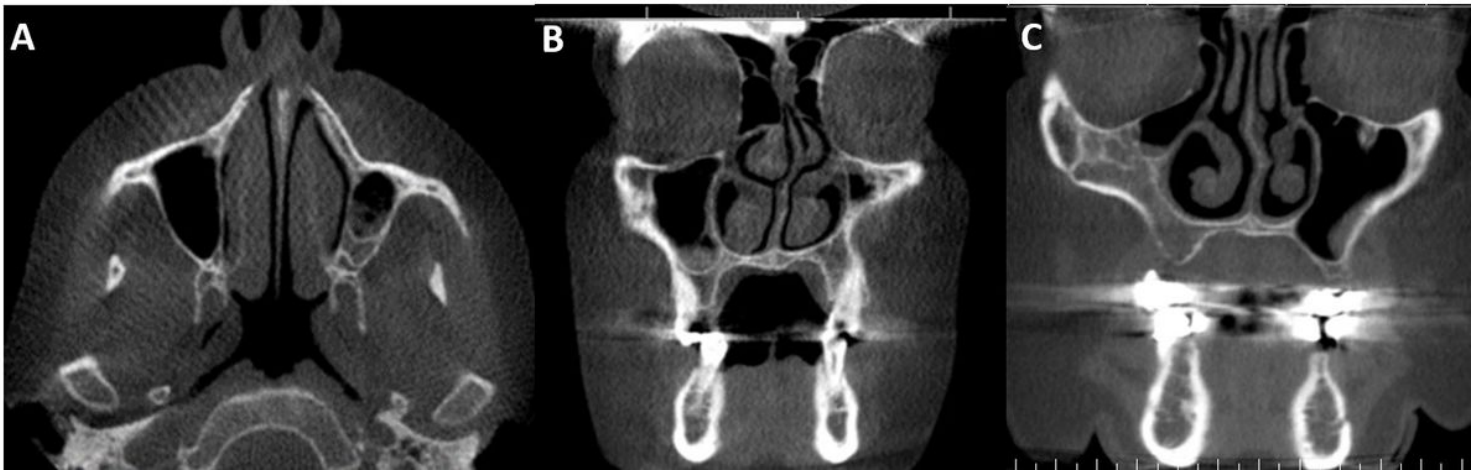

Figure 2

MSH type III with 0-degree maxillary occlusal plane cant. A) axial, and B) coronal cone beam CT images left MSH (same subject) showing symmetry of premolars on the occlusal plane. C) coronal cone beam CT image of right MSH showing well-formed alveolar process irrespective of the sinus hypoplasia.

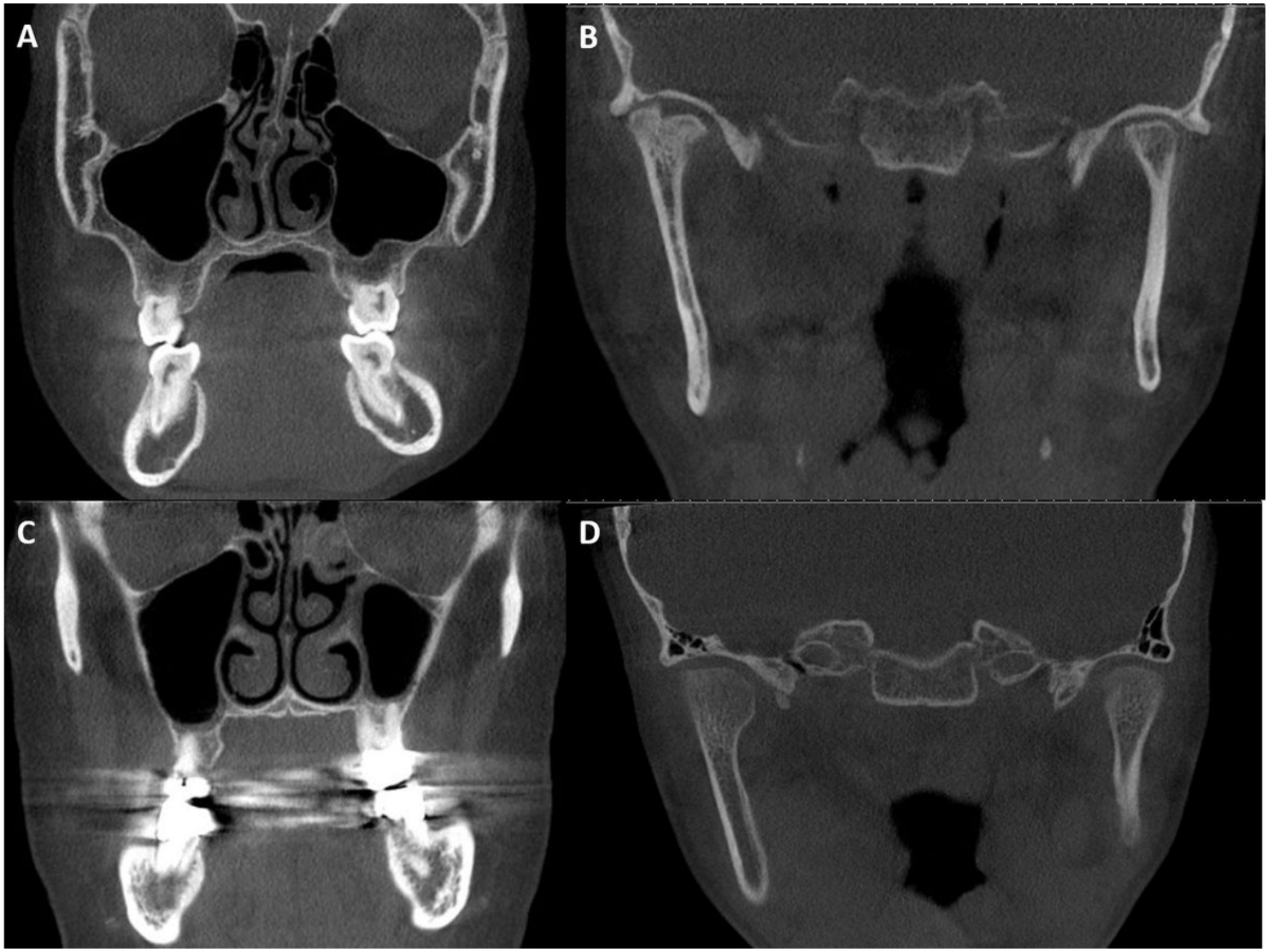

Figure 3

Large maxillary occlusal plane cant over 4°. Coronal cone beam CT images of A) and B) of Control case, C) and D) of MSH case, showing left condylar hypoplasia.