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Case Report

Need for treatment in all skeletal class III cases – a dilemma? a case report

Monika Mahajan *

Assistant Professor Department of Orthodontics & Dentofacial Orthopedics Himachal Pradesh Government Dental College & Hospital Shimla
**Corresponding author E-mail: monika.hemender@gmail.com*

Abstract

Skeletal malocclusion affects dental and facial tissues. A complicating factor for diagnosis and treatment of skeletal class III malocclusion is its multifactorial etiology. Genetics play an important role in determining the facial morphology of an individual. Prediction of a skeletal class III based on morphology can play an important step in orthodontic diagnosis and treatment planning. This case report further supports the significant role of genetics in skeletal class III malocclusion. As seen in our case the skeletal class III if left untreated does not detiorate but rather shows a decrease in ANB ie in anterioposterior discrepancy. Hence the need for treatment should be analysed thoroughly in skeletal class III patients.

Keywords: *Class III malocclusion; Genetics; Multifactorial etiology; Skeletal malocclusion; Treatment need.*

1. Introduction

Skeletal malocclusion may occur due to disturbance in the development of maxillary and or mandible leading to various types of anomalies both dentally and skeletally. According to (Angle 1899), class III malocclusion is defined as a condition when mandibular 1st molar is positioned mesially relative to the 1st molar of maxilla[1]. The prevalence of class III malocclusion varies among different ethnic groups with a higher frequency reported in Asian population2, 3,4. Sanborn recorded that in skeletal class III malocclusion, 45.2% occurred due to mandibular protrusion, 33.0% due to maxillary retrusion, 9.5% with a combination of both and 9.5% with normal relationship. Etiology of class III malocclusion is multifactorial with genetics and environment playing a role in various combinations. Most true class III cases show a strong hereditary component as also seen in the Hapsburg family5, 6, 7. The pattern of inheritance in their family indicated that mandibularprognathism is not a mandelian disorder but a multifactorial genetic disorder[1]. The diagnosis of true class III malocclusion can be made after making dental assessment by checking for class III malocclusion relationship and negative overjet along with functional assessment by seeing no shift from CR to CO on mouth closure. Studying cephalometric parameters and familial pattern also helps in making final diagnosis.

Treatment according to (Haitaoet al. 2013) should be undertaken as soon as the detection of the abnormality is made in the developing class III cases [8]. According to(Dietrich 1970) the class III skeletal discrepancy worsened with age whereas (Guyer 1986) found that class III features present in different age groups of the sample may worsen with age, but they usually do not begin class III development later in life9, 10. It has been observed that untreated class III subjects maintained the morphological characteristics of mandibular prognathism even after pubertal growth peek[11].

We present here a case report where class III skeletal features were seen in 11-year-old girl along with similar features in her elder sister and mother who was initially unaware of their condition. This study showed that class III inheritance has a strong genetic influence and no treatment undertaken did not worsen their class III features.

2. Case report

A girl 11years 7months of age came to the Department of Orthodontics and Dentofacial Orthopedics, HPGDC Shimla, with the chief complaint of irregular placement of upper /lower front teeth. The extraoral examination revealed a deficiency in a maxillary region, protruding chin making the profile concave. On intraoral examination she had been full complement of teeth. The molar relationships with both sides were developing class III with mandibular molar being 2 mm ahead of mesiobuccal cusp tips of maxillary molar. The overjet was reduced to 1mm. The mouth closure from CR to CO showed no shift of mandible (Fig 1).

The lateral cephalogram showed pt. A within normal range but pt. B was increased. The SNA was 81^0 and SNB was 83^0, hence ANB came out to be -2^0The Wits appraisal was -2mm (Fig 2).

OPG revealed presence of 3rd molars (Fig 3). All these findings led to concluding that it was a true class III case.

She was accompanied by her mother who gave consent for being examined .She was 42 years of age.On extra oral examination, a concave profile was also seen in her with more of protruding chin and a normal maxilla. Intraorally she had full complement of teeth with molars in class I relationship with reduced overjet(Fig 4) .

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Fig. 1: Extraoral & Intraoral Photographs of the Patent.



Fig. 2: lateral Cephalogram of the Patient.

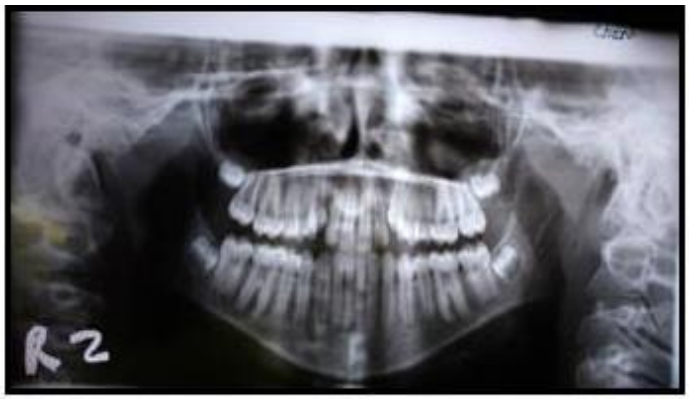


Fig. 3: OPG of the Patient.

The lateral cephalogram revealed increased SNB 84^0 but a normal SNA of 85^0. The ANB was $1^0$and Wits appraisal was 0mm (Fig 5).

The OPG showed mandibular right 3rd molar fully erupted (Fig 6).She was asked to bring her husband and other daughter also to

the department to find a possible genetic link for class III malocclusion.



Fig. 4: Extraoral & Intraoral Photographs of the Mother of Patient.



Fig. 5: Lateral Cephalogram of the Mother of Patient.



Fig. 6: OPG of the Mother of Patient.

The father of the patient did not reveal any class III features whereas the elder daughter , 16 years of age showed similar concave profile The protrusion of her chin was very prominent , about which she was getting conscious recently. Intraorally the molars were in class III relationship (Fig 7).

The lateral cephalogram also showed a skeletal class III pattern with normal maxilla and prognathic mandible with cephalometric readings of SNA as 79^0 and SNB as 81^0 making ANB of -2^0. The Wits appraisal was -2 mm (Fig 8).

The OPG showed the presence of all 3rd molars (Fig 9).



Fig. 7: Extraoral & Intraoral Photographs of the Sister of Patient.



Fig. 8: Lateral Cephalogram of the Sister of Patient.



Fig. 9: OPG of the Sister of Patient.

The aim of this case report was to enhance the role of genetics in skeletalclassIII malocclusion and to review the need for treatment in these patients.

3. Discussion

Skeletal malocclusion occurs due to aberrations in maxillary and mandibular growth during fetal development accentuated by intrinsic and extrinsic factors. True class III is one such skeletal malocclusion, which may be due to maxillary growth deficiency, or mandibular prognathism or various combinations of above two bringing about changes in the positioning, alignment and health of primary and or permanent dentition. The prevalence of class III malocclusion due to ethnicity ranges over from1% to 10%, sex and age12.

Etiology of class III malocclusion is due to interaction of various environmental and genetic factors. (Jacobson et al.1974) studied that growth and size of mandible are affected by hereditysalso heredity also by (McGuigan 1966) in the study on Hapsburg family 13, 14. Family 13, 14. Are some environmental factors like enlarged tonsil, mouth breathing habit, posture, trauma and disease, pharyngeal dimensions dimensions, may also contribute to class III malocclusion 1.

According to (Harris & Johnson 1991) heritability of craniofacial features is higher in comparison to that of dental features15. The inheritance pattern in Hapsburg jaw shows class III being a multifactorial genetic disorder1. (Sthohmayer 1937and Woff 1993)

supported the inheritance of phenotypic features in mandibular prognathism16, 17. (prognathism16, 17. 1961) reported it reported in mandibular prognathism 31% of the sample had their father affected, in 18% their mother was affected as is seen in our case and in 40% cases both the parents were affected18.

The heritability of craniofacial features is also seen in siblings as shown in studies of (Horowitz et al 1960 and Fernex 1967) with the transmission seen more from mothers to sons than from mothers to daughters whereas in our case inheritance has been shown from mother to her two daughters19, 20. Human studies support an autosomal dominant mode of inheritance in two studies of class III phenotype21, 22.(Dietrich 1970) reported that discrepancy worsened with age whereas (Rakosi 1981 and Schilli 1981) concluded that with increasing age the skeletal class III decreased from 18% to 3% 9, 23.(Mitani 23.(Mitani 1993)also reported that morphological characteristics of mandibular prognathism seen before pubertal growth peak did not fundamentally change showing that the skeletal features have not worsened with age 24. As can be seen in our case where the patient is 11 years of age presents with true skeletal class III features. Her elder sister 16 years of age who had not undergone any treatment also had skeletal class III features with almost similar hard tissue readings. However, her mother at the age of 42 years showed a decrease if not increase in class III skeletal features. She had never undergone any orthodontic treatment and was unaware of any such skeletal malocclusion. It can be seen that class III cases if left untreated do not further deteorate but rather an improvement is seen with growing age, further masking the skeletal deformity.

The improved Annotation of genetic and physical maps offer great future potential for identifying genes associated with this trait. Class III cases are difficult to diagnose and treat due to its etiological diversity. Inspite of many treatment options, some patients fail to show an improvement while some show relapse whereas others not receiving any treatment showed no further deterioration of skeletal deformity. Within the dentofacial complex, a compensatory mechanism exists, which tries to preserve a proportional and harmonious facial pattern. As seen in our case the skeletal class III if left untreated does not detiorate but rather shows a decrease in ANB ieanterioposterior discrepancy. Hence the need for treatment should be analysed thoroughly in skeletal class III patients.

4. Conclusion

By knowing the role of genetics in skeletal class III malocclusion, we have better understanding regarding its pathophysiology, enabling us for better therapeutic approach. Hence by including the parents and siblings of affected child with class III malocclusion a better correlation and analysis can be made leading to better prognosis of the case. More studies should be promoted to identify etiological risk factors in order to assist in better diagnosis and management. in spite of many treatment options some patients not receiving any treatment showed no further deterioration of skeletal deformity

References

[1] Profitt WR, Fields HW. Contemporary orthodontics, 4th edition, St.Louis. The CV. Mosby Co; 2000.

[2] Iwagaki H: Hereditary influence of malocclusion. Am J Orthodontics. 1938; 24:328-36. https://doi.org/10.1016/S0096-6347(38)90140-1.

[3] Ishii H, Morita S, Takeuchi T, Nakamura S: Treatment effect of combined maxillary protraction and chincup appliance in severe class III cases. Am J Orthod Dentofacial Orthop. 1987; 92: 304-12. https://doi.org/10.1016/0889-5406(87)90331-3.

[4] Allwright WC, Bundred WH: A survey of handicapping dentofacial anomalies among Chinese in Hong Kong. Internat DJ.1964; 14: 5 05-19.

[5] Litton SF, Ackermann LV, IssacsonRJ,Shapiro BL: Agenetic study of class 3 malocclusion. J Ortod.1970; 58: 565-77. https://doi.org/10.1016/0002-9416(70)90145-4.

[6] Harris JE, Kowalski CJ, WatnickSS:Genetic factors in the hape of craniofacial complex. Angle Orthod.1973;43:107-11

[7] Jacobson A, Evans WG,Preston CB, Sadowsky PL: Mandibular prognathism .Am J Orthod .1974;66:140-71. https://doi.org/10.1016/0002-9416(74)90233-4.

[8] Haitao Li, Ahmed Mosoud, Lawrence R, Voss. Hybrid Hyrax / quad- helix appliance in the phase I treatment of pseudo- class III malocclusion. Journal of the World Federation of Orthodontists. 2013; 2 (2): e107. https://doi.org/10.1016/j.ejwf.2013.02.003.

[9] Dietrich UC: Morphological variability of skeletal class 3 relationships as revealed by cephalometric analysis. Rep CongrEurOrthod Soc.1970; 131-43.

[10] Guyer EC, Ellis EE 3rd, McNamara JA Jr., Behrents RG: components of class III malocclusion in juveniles and adolescents. Angle Orthod.1986; 56: 7-30.

[11] Ellis E 3rd, McNamara JA. Components of adult class III malocclusion.J Oral Maxillofac Surg.1984; 42: 295-305. https://doi.org/10.1016/0278-2391(84)90109-5.

[12] Emrich RE, Brodie AG, Blayney JR. prevalence of Class 1,Class 2, and Class 3 malocclusions (Angle) in an urban population. An epidemiological study. J Dent Res. 1965; 44: 947-53. https://doi.org/10.1177/00220345650440053301.

[13] Jacobson A, Evans WG, Preston CB, Sadowsky PL: Mandibular prognathism. Am J Orthod 1974; 66:140-71? https://doi.org/10.1016/0002-9416(74)90233-4.

[14] McGuigan DG: The Hapsburgs. London, WH Allen, 1966.

[15] Harris EF,Johnson MG. Heritability of craniometrics and occlusal variables: a longitudinal sib analysis. Am J Orthod Dentofacial Orthop.1991; 99 (3):258-268. https://doi.org/10.1016/0889-5406(91)70007-J.

[16] Strohmayer W. Die Vereburg des Hapsburg Familientypus. Nova Acta Leopoldina.1937; 5: 219-296.

[17] Wolff G, Wienker TF, Sander H. On the genetics of mandibular prognathism: analysis of large European noble families. J Med Genet.1993; 30: 112-116. https://doi.org/10.1136/jmg.30.2.112.

[18] Suzuki S. Studies on the so called reverse occlusion. J Nihon UnivSch Dent.1961; 3:51-58.

[19] Horowitz S. Osborne R. De George F. A cephalometric study of craniofacial variation in adults twins. Angle Orthod.1960; 30:1-5.

[20] Fernex E, Hauenstein P, Roche M.Heredity and craniofacial morphology.Transactions of the European Orthodontic society.1967:239-257.x

[21] El-Gheriani AA, Maher BS, El- Gheriani AS, ScioteJJ.Segregation analysis of mandibular prognathism in Libua. J Dent Res.2003 Jul; 82 (7): 523-7. https://doi.org/10.1177/154405910308200707.

[22] Cruz et al. Major gene and multifactorial inheritance of mandibular prognathism. Am J Med Genet A. 2008 Jan 1; 146 a (1):71-7.

[23] Rakosi T, Schilli W. ClassIII anomalies: a coordinated approach to skeletal, dental, and soft tissues problems. J Oral Surg.1981; 39:860-70.

[24] MitaniH,SatoK,Sugawara J: Growth of mandibular prognathism after pubertal growth peak. Am J Orthod Dentofacial Orthop.1993; 104: 330-36. https://doi.org/10.1016/S0889-5406(05)81329-0.