An in Vitro Correlative Study of Sella Turcica Roofing and Dental Anomalies

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Abstract

Objective: The objectives of the present study were to find out the association between the Sella Turcica Bridging with dental anomalies and incidence of most common type of bridging in local population.

Material and Methods: For the study, 50 pretreatment lateral cephalometric radiographs showing complete Sella Turcica Bridging and 50 pretreatment lateral cephalogram without Sella Turcica Bridging were taken. After collection of the samples, retrospective study was performed with analysis of patient's records that included case history, orthodontic study models, orthopantomograms, intraoral periapical radiograph, occlusal radiograph, intraoral and extraoral photographs. The purpose of this analysis was to assess the associated dental anomalies.

Result: The results of the present study indicated that there was a significant association between the presence of Sella Turcica Bridging with dental anomalies

Conclusion: Incidence of dental anomalies was found to be higher in patients with Sella Turcica Bridging as compared to those without Sella Turcica Bridging. There was no significant difference in incidence of patients having Type A and Type B Sella Turcica Bridging.

Keywords: Sella Turcica Bridging, Neural Crestal Cells, Lateral Cephalogram, Pituitary Gland

INTRODUCTION

With the introduction of cephalometer by Broadbent in 1931 orthodontist were given a valuable tool for investigation of facial and cranial growth which helped the practicing orthodontist in clinical evaluation and treatment planning for patients. However, orthodontist also have the responsibility to carefully examine the cephalometric radiograph, not only to observe facial patterns or verify accuracy of tracing but also to determine if other abnormalities are present. We must not forget that the field of orthodontics is concerned with the health of entire individual.[1]

Most of these pathologic conditions, developmental abnormalities, or normal variants are associated with a significant problem in other system. Interestingly, some of these findings are detectable very early in life and often precede other signs or symptoms in syndromes. Therefore, in some cases, they could potentially be valuable for an early diagnosis. [2]

The sella turcica is an important anatomical structure for cephalometric assessment because of its central landmark. The sella turcica lies on the intracranial surface of the body of the sphenoid and consists of a central pituitary fossa. Two anterior and two posterior clinoid processes project over the pituitary fossa. Fusion of the posterior and anterior clinoid processes is known as a sella turcica bridge.

There are two types of bridging depending on their radiographic appearances. [3]

Formation and development of the sella turcica and teeth share, in common, the involvement of neural crest cells. In fact, the anterior part of the sella turcica is believed to develop mainly from neural crest cells and in tooth development; dental epithelial progenitor cells differentiate through sequential and reciprocal interaction with neural crest-derived mesenchyme. [4]

Recently some studies have been done to establish association of craniofacial skeletal anomalies with dental anomalies. This relationship may be based on the involvement of neural crest cells and/or homeobox or hox genes during the development stage. It appears that tooth formation and their eruption and sella turcica bridge calcification, as well as neck and shoulder skeletal development, are influenced by neural crest cells.[2]

So, the main purpose of this study is to elucidate relationship between Sella Turcica Bridging and dental anomalies in local population. Other purpose of this study is to find out most common type of Sella Turcica Bridging in local population.

MATERIAL AND METHODOS

In the present study, pretreatment cephalometric radiographs of 100 patients of local population of Karnataka; aged 7-30 years were taken. They were grouped in to two groups based on presence or absence of Sella Turcica Bridging.

Group 1: Fifty Subjects with Sella Turcica Bridging

Group 2: Fifty Subjects without Sella Turcica Bridging.

Armamentarium:

1. High quality radiographs which were taken by trained radiographic technicians in a standardized manner with clearest reproduction of sella turcica area.
2. High quality orthodontic study models
3. High quality orthopantomograms
4. High quality intraoral periapical radiographs
5. High quality occlusal radiographs
6. High quality intraoral and extraoral photographs
7. Case history records of patients

For the present study, 50 pretreatment lateral cephalometric radiographs showing complete Sella Turcica Bridging were retrieved from the 500 existing case records. Control group consisted of 50 pretreatment lateral cephalogram without Sella Turcica Bridging; retrieved from same case records by using simple random sampling. After collection of sample, retrospective study was performed with analysis of patient records that include case history, orthodontic study models, orthopantomograms, intraoral and extaoral photographs, intraoral periapical radiographs and occlusal radiogrophs. The purpose of this analysis was to assess any associated dental anomaly in patients with Sella Turcica Bridging and patients without Sella Turcica Bridging. Considering Shafer's classification morphological variations in size, shape, structure, number and eruption of teeth were analyzed. Correlation between Sella Turcica Bridging and dental anomalies was evaluated. [5]

To determine most common type of the Sella Turcica Bridging; two different morphological appearances of sella bridging (Type-A and Type-B) were used and compared with the current study.

Type-A: that manifest ribbon like fusion

Type-B: that manifest extension of anterior and/or posterior clinoid process, where these two meet either anteriorly, posteriorly or in the middle, with thinner fusion.[6] **(Photograph 1, 2)**

Statistical Method:

Chi-Square test and z-test were used in this study.

1. Chi-Square test:[7]

A chi-square test was used for:

To find out association of dental anomalies in the group with Sella Turcica Bridging and without Sella Turcica Bridging.

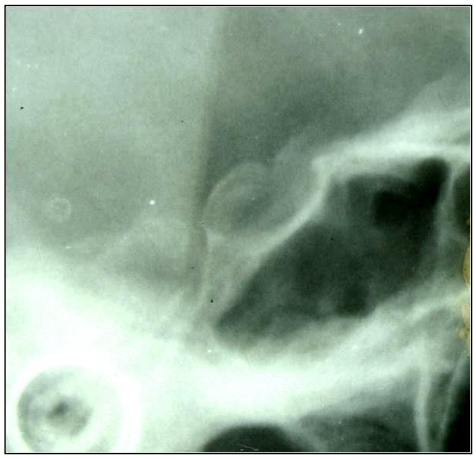
2.Z test:[8]

Z test was used to compare the different morphological variations of Sella Turcica Bridging i.e. **Type-A** and **Type-B**.

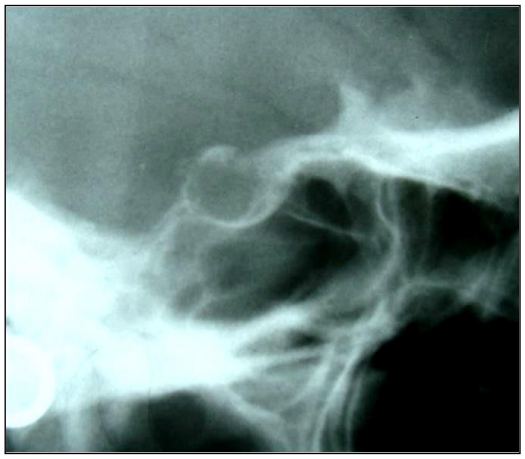
It is used to test significance of difference in means for large samples (>30)

Statistical software:

The Statistical software namely SPSS 11.0 and Systat 8.0 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**



Photograph: 1
Sella Turcica Bridging-**Type: A**
(Ribbon like fusion)



Photograph:2
Sella Turcica Bridging-**Type: B**
(Thinner fusion with extension of anterior and/or posterior clinoid process)

RESULTS

In the present study, association of Sella Turcica Bridging with dental anomalies was studied. The sample constituted 50 cases with Sella Turcica Bridging and 50 controls without Sella Turcica Bridging. The gender distribution in the present study was found to be 36 males and 64 females.

Results obtained in the study are as follows:

1. **Association of Sella Turcica Bridging and dental anomalies: (Table 1, Graph1)**

As the main objective of this study was to find out association between Sella Turcica Bridging and dental anomalies; chi square test was carried out. 90% cases with Sella Turcica Bridging showed presence of anomalies and 38% cases without Sella Turcica Bridging showed presence of anomalies. Presence of anomalies was found to be higher in patients with Sella Turcica Bridging compared to those without Sella Turcica Bridging. This association was statistically highly significant (P-value: <0.001).

2. **Distribution of type of bridging: (Table 2, Graph2)**

One of the objectives of this study was to find out most common type of Sella Turcica Bridging in local population. Z test was carried out to compare the proportions of patient with Type- A and Type-B Sella Turcica Bridging. 46 % cases had Type-A Sella Turcica Bridging and 54% cases had Type-B Sella Turcica Bridging. No statistically significant difference was found between the proportions of patients having Type A and Type B bridging in the study sample (P value: >0.05).

Table No. 1: Comparison of the presence of anomalies in cases with bridging and without bridging:

Anomaly	With Bridging		Without Bridging		Total	Chi-sq	P-Value
	N	%	N	%			
Present	45	90.00	19	38.00	64	29.340	<0.001**
Absent	5	10.00	31	62.00	36		
Total	50	100	50	100	100		

** Highly significant

Graph No. 1: Presence of anomalies in patient with bridging and without bridging:

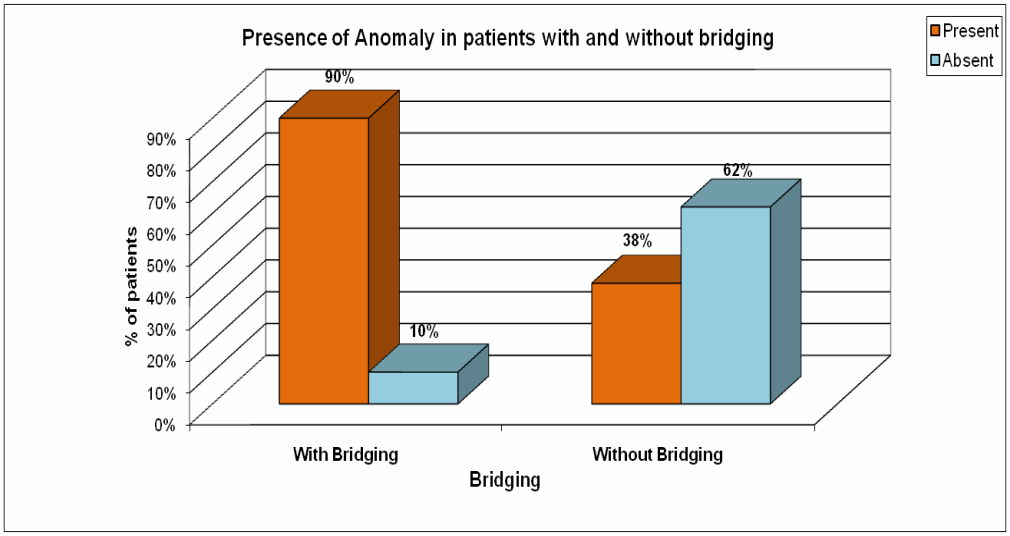
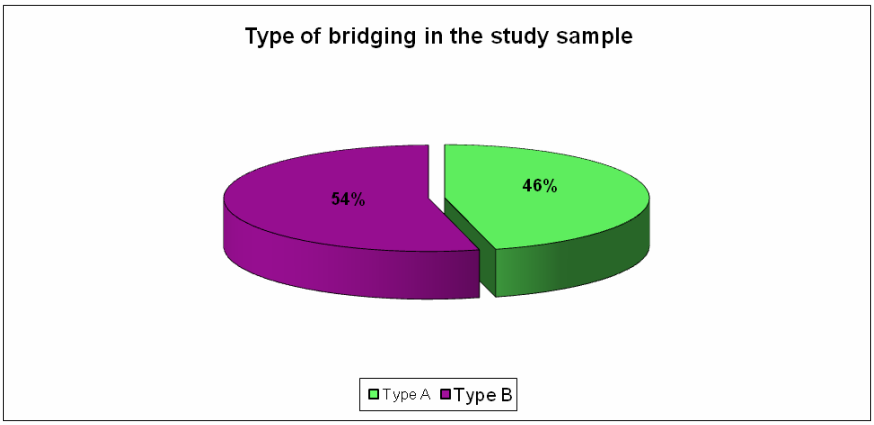


Table No. 2: Distribution of type of bridging in study sample:

Bridging Type	N	%	Z	P-Value
Type A	23	46.00	-0.800	0.422
Type B	27	54.00		
Total	**50**	**100**		

Graph No. 2: Distribution of type of bridging in study sample:



DISCUSSION

Calcification of diaphragma sellae, which radiologically has been described as 'roofing' or 'bridging' of the sella, in the absence of clinical signs or symptoms, is considered as a normal variant of the sella turcica.[9] Although many pathological conditions can be associated with this calcification. As far as the etiology is concerned, it has been suggested that an inter cliniod ligament is laid down in cartilage at an early stage of development and then ossifies in very early childhood. According to this theory, a sella turcica bridge should be considered as a developmental anomaly.[10,11] Moreover, as the area anterior to the sella turcica in the early embryonic period develops predominantly from neural crest cells, any structural deviations in the anterior wall are believed to be related to specific deviations in the facial skeleton. [12]

Hochstetter and Kier postulated that osseous interclinoid ligament was a developmental anomaly and they showed the existence of the foramen formed by this ligament in fetus and infant skull.[13] It was concluded that cartilagenous interclinoid taeina were extremely rare hence could not be regarded as routine occurrences.[14]

In the present study, association of Sella Turcica Bridging with dental anomalies was studied in 50 cases with bridging and 50 controls without bridging. The statistical analysis showed highly significant association between the presence of anomalies and the Sella Turcica Bridging ($P<0.001$). Presence of anomalies was found to be higher in patients with bridging as compared to those without bridging. In this study, incidence of dental anomalies was 90 per cent with Sella Turcica Bridging and 38 percent without Sella Turcica Bridging. This is in accordance with the studies done by Rosalia Leonardi, Ersilia Barbato, Maurizio Vichi and Mario Caltabiano.[4] This finding is also consistent with the study done by Sandham A, Horsewell B, Kjaer I, where they found that skeletal anomalies or normal variant seen in cephalometric radiograph are associated with dental anomalies[15,16,17]

One of the possible etiologies for the increased occurrence dental anomalies with Sella Turcica Bridging could be that the formation of Sella Turcica Bridging and the development of the teeth, share in common the involvement of neural crest cells. In fact, the anterior part of the sella turcica is believed to develop mainly from neural crest cells. In the initial period of embryogenesis, the primitive oral cavity is lined by oral ectoderm; most of the connective tissue cells underlying the oral ectoderm are of neural crest cells. These cells are thought to instruct or induce the overlying ectoderm to start tooth development.[18] Dental epithelial progenitor cells differentiate through sequential and reciprocal interaction with neural crest-derived mesenchyme. [19,20]

Molecular studies of odontogenesis, using the mouse tooth as a model, have shown that the tooth development is under strict genetic control, which determines tooth position, number, size, and shape .[21,22,23] The majority of cases involving hypodontia and oligodontia are due to genetic factors. Mutations of several genes are associated with syndromic tooth agenesis. To date, the familial and sporadic forms of tooth agenesis have been associated with mutations in MSX1 and PAX9.[24] Interestingly, MSX1- and PAX9-deficient mice exhibited several other craniofacial abnormalities.[25,26]

Knowledge of sella turcica morphology is of great importance for orthodontic diagnosis and treatment planning because orthodontists regularly analyze considerable number of profile radiographs. Orthodontists will be in many cases the first to register minor malformations of sella turcica. Insight into the sella turcica malformations and information about the etiological background of such malformations is very important.

As one of the objectives of this study was to find out the most common type of Sella Turcica Bridging in local population, the results showed no statistically significant difference between the proportions of patients having Type A and Type B bridging in the study samples ($P>0.05$). But there was an overall increased incidence of Type-B Sella Turcica Bridging (54%) when compared with Type-A (44%) sella turcica brigding. This finding is in accordance with the study done by R.M.Jones, A. Faqir, D.T. Millett, Jonas P. Becktor, Sanna Einnerson.[3]

From a clinical point of view, these skeletal anomalies and /or normal variants may be considered as risk factors that could enable the clinician to make an early diagnosis and thus treat the dental anomalies at an early stage. Because many of these skeletal anomalies and normal variant present early in life, their early detection can be used to forecast the presence of dental anomalies later in life, enabling the clinician to adopt preventive measures.

CONCLUSIONS

The conclusions from this study were:

1. Incidence of dental anomalies was found to be higher in patients with Sella Turcica Bridging as compared to those without Sella Turcica Bridging.
2. There was no significant difference in incidence of patients having Type A and Type B Sella Turcica Bridging.

REFERENCES

1. Stuart B, Fred A.L. Abnormalities found on cephalometric radiographs. Angle Orthod Oct1976. Vol 46. No 4:381-386.
2. Rosalia L,Ersilia B, Maurizio V, Mario C. Skeletal Anomalies and Normal Variants in Patients with Palatally Displaced Canines. Angle Orthod 2009. Vol 79. No 4:727–732.
3. Jones R.M., Faqir A., D. T. Millett D. T, Moos K. F, McHugh S. Bridging and Dimensions of Sella Turcica in Subjects Treated by Surgical-orthodontic Means or Orthodontics Only. Angle Orthod 2005. Vol75. No 5: 714–718.
4. Rosalia L, Esralia B, Maurizio V, Mario C. Sella turcica bridge in subject with dental anomalies.Eur J. orthod 2006. Vol 28:580-5853.
5. Shafer W.G, Hine M.K, Levy B.M. A textbook of oral pathology. 4th edition. Elsevier publication;1997.
6. Inger K, Karin B. B, Lisson J, Charlotte G, Russel B.G. Face, palate and craniofacial morphology in patients with solitary median maxillary central incisor. Eur J. orthod 2001. Vol 23: 63-73.
7. Park K. Park's textbook of preventive and social medicine. 18th edition. Banarasidas Bhanot publication; 2005.
8. Soben P. Essentials of preventive and community dentistry. 4th edition. Arya publication;2009.
9. Kantor M L, Nortan L A,. Normal radiographic anatomy and common anomalies seen in cephalometric films. Am J Orthod Dentofacial Orthop 1987. Vol: 91. 414-426
10. Lang J. Sructure and postnatal organisation of heterofore uninvestigated and infrequent association of sella turcica region. Acta Anatomica 1977. Vol: 99: 121-139
11. Inoue T, Rhoton AL, Barry M E. Surgical approaches to cavernous sinus: a microsurgical study. Neurosurgery 1990. Vol: 26. 903-932
12. Kjar I, Keeling J W, Reintoft I, Nolting D, Fischer Hansen B. Pituitary gland and sella turcica in human trisomy 21 fetuses related to axial skeletal development. American journal of Medical Genetics 1998. Vol : 80. 494-500
13. Kier E L. Embryology of normal optic canal and its anomalies. An anatomic and roentgenographic study. Invest. Radiol. 1966. Vol: 1. 346-362
14. Lang J. Skull base and related structures: Atlas of clinical anatomy. Stuttgart: Schattauer, 1995;175
15. Sandham A. Cervical vertebral anomalies in cleft lip and cleft palate . Cleft Palate J.1986. Vol:23, 206-214
16. Horsewell B B,The incidence and relationship of cervical spine anomalies in patient with cleft lip and cleft palate. J Oral Maxillofacial Surg. 1991. vol: 49. 693-697
17. Kjaer I, Fischer Hansen, Keeling J W, Reintoft I. Pituitary gland and axial skeletal malformations in human faetuses with spina bifida. Eur J Pediatr Surg. 1999. Vol:9: 354-358.)
18. Orban. Orban's oral histology and embryology. 12th edition. Elsevier publication;2008.
19. Miletich I, Sharpe P T neural crest contribution to mammalian tooth formation. Birth defect research. Part C, Embryo Today. 2004. Vol 72: 200-212
20. Mortoni T et al. in vitro differentiation of dental epithelial progenitor cells through epithelial mesenchymal interactions. Archives of oral biology2005. Vol: 50, 695- 705
21. Thesleff I. The genetic basis of normal and abnormal craniofacial development. Acta Odontologica Scandinavica 1998. Vol :56. 321-325.
22. Vastardis H. The genetics of human tooth agenesis: new discoveries of understanding human tooth anomalies. Am J Orthod Dentofacial Orthop 2000. Vol: 117. 650-656.
23. Peters H, Balling R. Teeth: where and how to make them. Trends in genetics. 1999. Vol 59. 59-65
24. Mostowska A,Kobielak A, Trzesiac W H. Molecular basis of nonsyndromic tooth agenesis: mutations of MSX1 and PAX 9 reflect their role in patterning human dentition. Eur J. Of Oral Sciences 2003. Vol 111. 365-370
25. Satokota I, Mass R. MSX1 deficient mice exhibit cleft palate and abnormalities of craniofacial and tooth development. Nature Genetics 1994. Vol: 6. 348-356
26. Peter H, Neubuser A, Kratochwil K, Balling R,. PAX9 deficient mice lack pharyngeal pouch derivatives and teeth and exhibit craniofacial and limb abnormalities. Genes And Development 1998. Vol : 12. 2735-2747.