EurAsian Journal of Oral and Maxillofacial Surgery
Official Publication of AÇBİD
(Association of Oral and Maxillofacial Surgery Society)

January 2022 Volume 1 Number 4
e-ISSN: 2687-5497

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RESEARCH ARTICLE

The Effect of Single Dose Teriparatide Administration on the Early Healing Period of Bone Defects Created in the Rat Mandibular

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Abstract

Introduction

Bone grafts are widely used in the repair of bone tissue. In recent years, studies on the use of biomaterials have became popular in order to increase the efficiency of bone grafts. In this study, the effects of local teriparatide used in different doses on bone healing were evaluated.

Materials - Methods

In this study, 30 male Spraque-Dawley rats were used and they were divided into 5 groups, 6 each. A critical sized defect of 5 mm in diameter was created in the mandible. Defects in Group 1 were left empty. Particulate autograft was applied to the defects in Group 2. Group 3 was applied allograft. In Groups 4 and 5, 20 µg and 40 µg teriparatide were administered with the allograft, respectively. At the postoperative 4th week, the anesthesia dose was doubled and euthanasia was performed. The samples were taken out in one piece and evaluated histomorphometrically.

Results

While the amount of newly formed bone tissue (0.28±0.01 mm2) and the number of osteoblasts (41.67±1.56) were significantly lower in Group 1, these values were found in Group 5 (0.79±0.01 mm2), (77.72±3.06) is significantly higher. The findings in Group 4, in which teriparatide was used (0.72±0.01 mm2, 72±4.26), were significantly higher than Group 3 and Group 2. The amount of new bone formation (0.48 mm2) and the number of osteoblasts (54.17±1.99) were significantly higher in Group 3 than Group 2 (0.39±0.01 mm2, 50±1.4).

Conclusion

As a result, it was observed that teriparatide, which was used locally in two different doses, significantly increased bone healing.

Keywords: bone healing, teriparatide, rat mandible

Introduction

Bone tissue is the only tissue that heals without scarring. Organic components make up 35% of the bone matrix. Its intermediates consist of Glycosaminoglycans, Glycoproteins. Bone glycoproteins are responsible for initiating matrix calcification.[1]

Since the matrix of the bone tissue is hard, it does not allow diffusion, so nutrition is through channels. These rings form a regular circular structure, 3-7 µm thick and usually around the bone canal. The nutrient transfer from the innermost layer of the bone tissue to the outermost layer is achieved with these lamellae. It consists of intertwined circular structures around the Haversian canal with a diameter of 20-100 µm and containing vascular structure.[2,3]

Bone formation occurs by two different mechanisms, intramembranous and chondral ossification. While flat bones ossify intramembranous, short and long bones are formed by chondral ossification.[4,5] As a result of the deterioration of the anatomical integrity of the bone tissue, bone healing occurs with a series of complex electrical, chemical and mechanical mechanisms. Bone healing consists of 3 stages.[6]

In the inflammatory phase, the occurrence of vascular endothelial damage causes platelet aggregation and secretion of vascular endothelial growth factor in the region. While the hematoma formed in the region contributes to the stabilization of the fracture line with the pressure it creates, it also initiates the necessary mechanism for healing thanks to the thrombocyte and macrophages in it. The hematoma is organized after 48 hours. The fibrin network formed is an

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important factor for healing thanks to the periosteum and endosteum-derived precursor cells and the framework it forms.[6]

One of the factors that act as a chemotactic agent for monocytes and macrophages is ESM. This environment is well suited for the function of polymorphous core leukocytes and macrophages. Since the anastomosis is disturbed between the fracture ends, circulation in this region is disturbed and necrotic areas are formed. Osteocytes undergo lysis in the region. This helps the capillary fibroblasts to form the intermediate and collagen by feeding. There is a sudden increase in the number of osteoblasts and chondroblasts coming from the endosteum and periosteum, and osteogenesis begins in the later stages.[6]

The repair phase begins after the fracture occurs, but manifests itself 7-12 days later. In areas close to the fracture line, blood vessels and osteogenic cells are densely located. As it moves away from the fracture ends, vascularization decreases and chondroblasts begin to appear. Gradual differentiation of the cells, accumulation of the substances they secrete and callus formation begin with the formation of ESM.[6]

The task of the callus is to stabilize the fracture site. The callus is soft at first and is not visible on the radiograph. Chondrocytes in the areas of cartilage tissue transform into hypertrophic chondrocytes and cartilage matrix secretion begins. Vascular development begins in the empty lacunae.[6]

Without calcification, there is no vascularization. Bone cells begin to migrate to the region with vascularization. With the formation of internal and external callus, the bone ends become much more stable. The formation of hard callus means that most of the healing has taken place at the fracture site.[6]

In remodeling, the remodeling of bone tissue takes place according to Wolff's law. As I said at the beginning of my presentation, bone healing is the only tissue that heals without scarring. Various graft materials are used to support bone formation in bone defects larger than the critical size defect. Autograft, allograft, xenograft and alloplastic bone are examples of these graft materials.[6]

Bone graft materials affect new bone formation at the defect site in many ways. Osteogenesis is the formation of bone in the recipient area by cellular elements in the graft material. Osteoinduction is the stimulation of pluripotent cells to transform into osteoblasts or chondroblasts by the growth factors in the graft material, and Osteoconduction is the progression of the vascular structures in the recipient area using the graft material as a skeleton and moving into it. Autografts are accepted as the gold standard because they have all of the bone graft acceptance mechanisms mentioned in graft materials. Besides, the unpredictable resorption of autografts is a serious disadvantage and many biomaterials or agents are used with graft materials to support bone formation, including autografts.[7]

Many biomaterials such as CMP, Collagen, fibroblastic growth factor and hyeloronic acid have been developed to be used in the repair of bone defects, and studies on this subject continue.[8] In case of intermittent administration of teriparatide, an anabolic change begins in the bone tissue by affecting the osteoblastic activity.[9] Conversely, if the frequency of administration increases, in other words, if it is given continuously, not intermittently, it will affect the osteoclastic activity and change in the direction of resorption in the bone tissue. It increases bone resorption by affecting balance.[10]

Materials and Methods

30 male Spraque-Dawley rats with complete skeletal development were used in the study.

The block autograft obtained during defect creation in the rat mandible was ground with a bone grinder and applied in the form of particles. Bovine demineralized freeze-dried bone particle graft was used as allograft.

Table 1. Distribution of Study Groups

GROUPS	N	KULLANILAN MATERYAL
Group 1	1	Defect left blank (negative control group)
Group 2	2	Autograft
Group 3	3	Allograft
Group 4	4	Allograft + 20µg Teriparatide
Group 5	5	Allograft + 40µg Teriparatide

Figure 1: Teriparatide agent (Forsteo, Eli Lilly and Company, France) used in many defect areas

In order to provide general anesthesia, 90 mg/kg Ketamine HCl (Alfamine Ege-Vet, Turkey) and Xylazine HCl2 (Alfazyne Ege-Vet, Turkey) were injected intraperitoneally to all animals. The depth of general anesthesia was controlled by pedal reflex. After providing anesthesia, the right half of the mandible was preferred as the operation site in all animals. The surgical site

was wiped using iodine solution and shaved.

In order to reduce the force on the mandible during the surgical procedure and to ensure its stabilization, the rats were fixed on the foam block with a 10 cc injector tip from the nape of the neck. By palpating the lower edge of the mandible, an 11 mm long longitudinal incision was made approximately 3 mm above and parallel to the lower edge of the mandible. The lateral aspect of the ramus was exposed so that the posterior edge of the mandible, its inferior edge, and the sigmoid notch were visible.

The region where the defect will be created; It was determined to be 3 mm away from the inferior and posterior margins of the mandible. A bicortical critical size defect of 5 mm in diameter was created in the determined area. After the defect was created, material was applied to the defect area according to the groups or the defect area was left empty.

In this study, the same amount of allograft was used to fill the defect area in 3 groups, groups 3-4-5, and only allograft was applied in group 3. In group 4-5, after allograft was applied, tp was applied to the defect area by dropping 20 mkg for group 4 and 40 mkg for group 5 in one go.

Figure 2: Particulate BLC placed in the surgical field.

Muscle and skin tissues were sutured separately with 4/0 vicryl using the continuous locking suture technique. For infection and pain control, intramuscular 5mg/kg/day amikacin sulfate (Amikozit, Eczacıbaşı, Turkey) and intraperitoneal ketoprofen (Profenid ampul, Senıfi Aventis İstanbul, Turkey) were administered on the day of surgery and for 2 days postoperatively.

At the end of the postoperative 4th week, the anesthesia dose was doubled and the experimental animals were sacrificed. Mandible samples were taken by stripping the muscle and dermal tissue.

Bone tissue samples fixed in 10% buffered formaldehyde for histomorphometric analysis were cleaned of excess parts before analysis. After fixation, all the material was decalcified in the prepared solution by taking 1 scale from 50% formic acid and 20% sodium citrate solutions.

After the decalcification process was completed, the bone tissue samples were embedded in paraffin blocks after washing in running tap water, passing through increasing degrees of ethanol series (50%-99%) and xylene series, followed by melted paraffin infiltration at 62°. 5-7 µm thick sections from paraffin blocks were placed on slides with a microtome (Leica RM2245).

These sections were stained with the hematoxylin–eosin staining method and evaluated. Stained sections were examined and photographed using a Nikon Ci-S light microscope, Nikon DS-Fi3 camera and NIS-Elements D image analysis system (Nikon Corporation, Tokyo, Japan).

Osteoblast counts in all samples included in the study were calculated by taking the average of 3 different unit areas (200 µm). All measurements and analyzes were performed twice by a single pathologist.

Results

No complications such as infection or fracture were observed during or after the experiments that would cause the subjects to be excluded from the study.

At the end of the 4th postoperative week, proliferation in the bone tissue was observed in all groups by histopathological analysis.

High vascularization was detected in all groups in the newly formed granulation tissue in the defect area.

Table 2. Comparison of the amount of bone tissue area formed between the groups

Groups	N	Ort±Ss	Min-Max (Median)	p
Group 1	6	0,28±0,01 mm^2	0,27-0,29 (0,28)	0,001
Group 2	6	0,39±0,01 $mm^{2\,b}$	0,38-0,39 (0,39)	
Group 3	6	0,48±0 $mm^{2\,c}$	0,47-0,48 (0,48)	
Group 4	6	0,72±0,01 $mm^{2\,d}$	0,7-0,73 (0,72)	
Group 5	6	0,79±0,01 $mm^{2\,e}$	0,78-0,8 (0,79)	

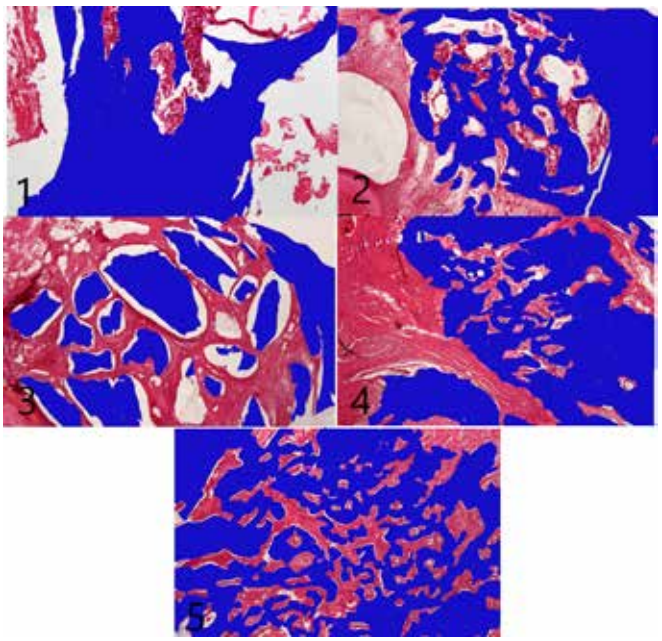

Figure 3: The histomorphometric view of the defect area at the postoperative 4th week of the groups. The blue area shows the newly formed bone tissue.

Table 3. Comparison of osteoblast counts between groups

Grups	N	Ort±Ss	Min-Max (Median)	p
Group 1	6	41,67±1,56 [a]	39-43 (42)	0,001
Group 2	6	50±1,4 [b]	48,67-52,33 (49,67)	
Group 3	6	54,17±1,99 [c]	51,67-57,67 (53,84)	
Group 4	6	72±4,26 [d]	66,67-76,67 (73)	
Group 5	6	77,72±3,06 [e]	74,67-81,67 (76,67)	

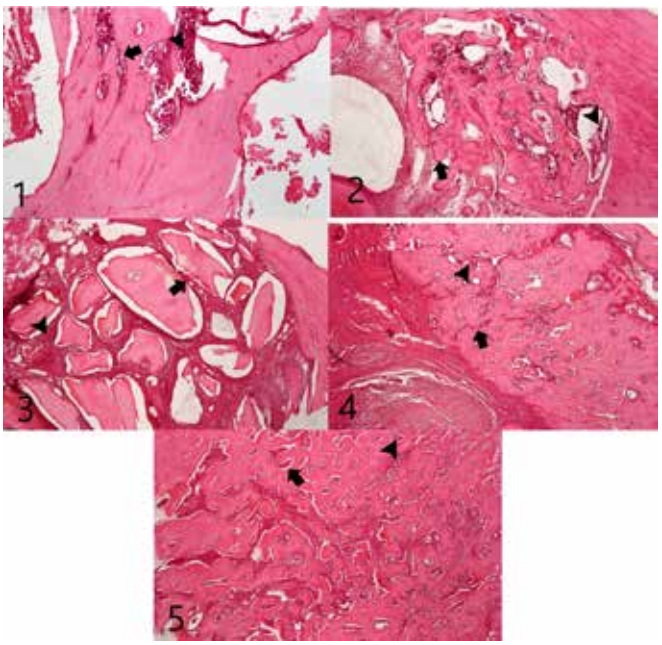

Figure 4: The histomorphometric view of the defect area at the postoperative 4th week of the groups. Black arrow indicates osteoblast cells, black arrowhead shows organized collagen tissue.

Discussion

Today, as a result of the use of bone grafts, completely satisfactory results cannot be obtained in the regeneration of bone tissue. For this reason, bone tissue regeneration still constitutes an important clinical problem in the field of oral and maxillofacial surgery. In recent years, it can be stated that studies on the use of biomaterials have accelerated with the aim of increasing the efficiency of bone grafts used.[3]

The main task of an ideal graft material/biomaterial is to play a role in the healing of the defect area in the applied area as soon as possible and by guiding the newly formed tissues. It has been observed in studies that the osteogenic potential of bone cells in the iliac crest is less than the osteogenic potential of bone cells in the mandible.[3]

The effects of external factors affecting bone metabolism, such as bisphosphonate-induced osteonecrosis or osteoradionecrosis, on osteogenic cells originating from the femur and mandible differ from each other. Hormones that regulate calcium metabolism, such as calcitonin and PTH, are known to directly and indirectly affect bone tissue in terms of strength, integrity, and functionality. The minimum size defect that the body cannot repair on its own is defined as CBD.[12]

Apart from these studies, it is seen in the literature that defects with a diameter of 5 mm in the rat madibula are considered to be CBD. Another factor that determines the contribution of applied materials on bone healing is time. reported that the findings in the week could be evaluated as significant. The fact that the number of osteoblasts and the amount of newly formed bone tissue area of Group 1, which is the defect group that was left blank in this study, were significantly lower than the other groups, which is consistent with the literature and is an expected finding.

In Group-2, the number of osteoblasts and the amount of newly formed bone tissue area were found to be significantly lower than the data of other study groups. On the other hand, it was observed that the number of osteoblasts and the amount of newly formed bone tissue area of Group 3 were significantly lower than only Group 4 and Group 5 data. Autograft has been accepted as the gold standard in the literature.

Zandi et al.13 and Koca et al.10 reported that low dose use had a positive effect on bone tissue in their studies. It has been stated that it has an anabolic effect on the bone tissue if it is used intermittently, and a catabolic effect if it is used continuously. It was observed that the amount of newly formed bone in the group administered continuously was significantly less than the group given intermittently.

As a result of the study, it was shown that teriparatidine plays a role in the increase of both newly formed bone tissue and total bone tissue volume. This issue is important because the extent to which topically applied teriparatidine enters the systemic circulation is not fully known. It was stated that the amount of newly formed bone tissue per day did not differ.

New bone formation was evaluated in the 4th and 8th weeks postoperatively, and it was reported that the amount of new

bone area in the teriparatide groups was significantly higher in all time periods. The number of osteoblasts and the amount of newly formed bone tissue area of Group-4 and Group 5, which are the groups in which teriparatide was used, were significantly higher than the other groups.

Yu et al. reported in their study that teriparatidine accelerates and supports bone tissue healing by increasing vascularity and reducing fibrotic tissue formation.[14]

One of the studies in which teriparatidine was applied locally in the rat mandible was done by Koca et al. The researchers created a 3 mm diameter defect in the rat mandible and applied different doses of teriparatidine, 20 µg and 40 µg, locally and did not use any graft material or ground material together. evaluated the effect of teriparatidine on bone healing and masseter muscle at 1 week. Researchers reported that bone healing was statistically significantly higher in the groups using teriparatide compared to the null defect group, but there was no difference between the groups using teriparatide.[15]

Koca et al. created a 3 mm diameter defect in the mandible of the rat and applied teriparatidine locally at different doses, 20 µg and 40 µg, without using any graft material or ground material.[15]

Shiraki et al. evaluated the effects of a single-dose injection of teriparatidine on bone turnover markers and calcium mechanisms in postmenopausal female patients.[16]

Conclusion

There are 2 main factors that determine the efficacy of teriparatidine: the dose of use and the frequency of administration. Bone defects show different healing stages in different time periods. Therefore, the effect of locally applied teriparatide on bone healing can be evaluated in more than one different and critical time period.

Source of Finance

None declared.

Conflicting Interests

None declared.

Authorship of Contributions

Muhammed Fatih Çiçek , Cansu Gül Efeoğlu Koca

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Official publication of AÇBİD (Association of Oral and Maxillofacial Surgery Society)

CASE REPORT

Lip Repositioning as an Alternative Treatment of Gummy Smile

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Abstract

Objective

Excessive gingival display (EGD) may compromise esthetics during smile. As the etiology is multi-factorial, various treatment options have been described for the management of EGD. The aim of this report was to present the management of four patients with EGD by lip repositioning technique as a minimally invasive treatment modality.

Case

Four female patients referred to our department with a chief complaint of unaesthetic appearance during smiling. Clinical examination revealed EGD as a result of vertical maxillary excess and hypermobile upper lip. Lip repositioning surgery was performed in order to restrict the pull of the elevator lip muscles. No complications or recurrence were observed at 6-month follow-up and all patients were satisfied with the outcomes of the procedure.

Conclusion

As a safe and relatively simple procedure, lip repositioning may provide satisfactory outcomes for patients with excessive gingival display.

Keywords: excessive gingival display, gummy smile, hypermobile lip, lip repositioning, smile esthetics

Introduction

Excessive gingival display (EGD), commonly termed gummy smile, is a condition characterized by overexposure of the maxillary gingiva during smiling. According to Tijan et.al; smile line has been graded depending on the exposure of tooth and gingiva[1]. A display of marginal gingiva more than 3 mm is defined as a very high smile line which is also called as gummy smile[2].

Etiology of gummy smile may vary including vertical excess of the maxilla, delayed tooth eruption, compensatory eruption of the maxillary teeth, incompetent lips or hyperfunctionality of upper lip muscles[3]. Genetic factors have also been reported to play a role in gummy smile[4]. As a result of multifactorial etiology, correction of gummy smile may be performed using various techniques including orthognatic surgery, orthodontic intrusion, surgical crown lengthening, detachment of lip elevator muscles, botulinum toxin injections and surgical lip repositioning [5,6].

Lip repositioning was first described in 1973 for the correction of gummy smile cases caused by the hypermobility of the upper lip [7]. It was performed by removing a band of mucosa including the frenulum of the upper lip from the buccal vestibule to apical to the mucogingival junction as a partial thickness flap. Since the introduction, several modifications of the technique have been developed including preservation of the frenulum to decrease the morbidity.

Indications of lip repositioning surgery include gummy smile, thin upper lip and smile line asymmetry while it is contraindicated in case of an attached gingiva less than 3 mm in the anterior maxilla due to the difficulties in the flap design and stabilization or a gingival display more than 6 mm caused by skeletal factors [8].

Lip repositioning surgery is gaining popularity among patients and surgeons. The purpose of this study to present the management of four patients with excessive gingival display by lip repositioning technique as a minimally invasive treatment modality.

Case Presentation

Four female patients referred to our department with a chief complaint of unaesthetic appearance during smiling. The patients' medical history were insignificant with no contraindication for surgery. Clinical examination revealed 3 to 6 mm of maxillary gingival display during full smile and normal height and width-to-height ratio of maxillary anterior teeth therefore diagnosis of moderate vertical maxillary excess was made (Figure 1). All patients refused the orthognathic surgery

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and preferred lip repositioning as a less invasive procedure. Informed consents were obtained prior to the procedure.

Figure 1. Preoperative view of one of the patients: A very high smile line with the display of marginal gingiva more than 3 mm was observed.

Surgical Technique

Local anesthetic was administered in the vestibular mucosa and lip between the maxillary first molars. The incision outlines were marked with a sterile surgical marking pen on the dried mucosa.

Following a partial thickness incision at the mucogingival junction from the right first molar to the left first molar, a second parallel incision was made at approximately 6 to 10 mm distance from the first incision. The incisions were connected at each first molar creating an elliptical outline of the incisions. The epithelial layer was removed, leaving the underlying connective tissue exposed (Figure 2). The amount of tissue to be removed was decided approximately two times of gingival exposure in length. Electrocoagulation was used to control bleeding.

Figure 2. A 6 to 10 mm width elliptical epithelial layer was removed and the underlying connective tissue was exposed.

Prior to surgery, images were obtained with patients in active smile, and the amount of gingival display in active smile was evaluated six months after the surgery. No intra- or post-operative complications were observed and all patients were satisfied with the outcomes of the treatment (Figure 3).

Figure 3. A remarkable decrease in gingival exposure was observed at 6-months follow-up of the patient.

Discussion

Lip repositioning surgery is an innovative technique to correct excessive gingival display creating an attractive smile which will hopefully improve quality of life for gummy smile patients. Investigations have shown that the amount of gingival display in an attractive smile varies from 1 mm to 3 mm[9]. The spesific etiologies of gummy smile require different treatments which may include botulinum toxin injections, orthodontic treatment, crown lengthening, and so forth[2]. The current literature consists of many case reports suggesting different treatment modalities for correction of gummy smile according to their etiology.

In the presented cases, lip repositioning were preferred since the underlying etiology for gummy smile were hypermobility of the upper lip elevator muscle. The procedure has been reported to be related with minimal postoperative side effects including bruising, discomfort, swelling of the upper lip, mucocele formation [10]. Similarly, our patients did not experience any severe symptoms following the procedure.

Conclusion

As a safe and relatively simple procedure, lip repositioning may provide satisfactory outcomes for patients with excessive gingival display. However, careful examination of the patient and etiology of EGD is mandatory for proper indication and achieving successful results.

Acknowledgement

There was no grant support for this study. All authors declare that ther is no conflict of interest related to this work.

This report was presented as a oral presentation at the ACBID 13th International Congress with EACMFS Endorsement held in Antalya, Turkey, in 2019.

Source of Finance

Conflict of Interest

All authors disclose any financial and personal relationships

with other people or organizations that could inappropriately influence (bias) their work.

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CASE REPORT

Bad Split During Bilateral Sagittal Split Osteotomy of Mandible: Case Report

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Abstract

Bilateral sagittal split osteotomy (BSSO) is a well-defined process that moves the mandible in three directions of space and moves it into the correct position. BSSO has been described in the literature as a safe procedure. However, it includes some intraoperative and postoperative complications. The most common of these is the bad split. The aim of this article is to present bad splits cases and treatment methods. In our center had made 102 BSSO cases in 2012-2019 and 6 of these include bad split. 5 of these buccal plate fracture on proximal segment, one of these distal segment fracture include condyler process. All cases had managed intraoperatively with screw osteosynthesis and no need for additional precaution like rigit intermaxillary fixation or a prolonged stay. All patient were followed for 1 week, 1month, 6 month and 1 year. None of patient had showed poor function or temporomandibular dysorder postoperatively.

Even if a bad split consist of during surgery, no influence final result or postoperative course.
Consequently, bad split is not avoidable all time. When treated well the chances of functional success are good.

Keywords: Orthognatic surgery, Bilateral Sagittal Split Osteotomy, Bad Split

Introduction

Orthognathic surgery is a commonly performed maxillofacial procedure with a documented safety record[1].

Bilateral sagittal split osteotomy (BSSO) of the mandible is one of the most common operative techniques used in orthognathic surgery. Since its initial description by Trauner and Obwegeser more than 50 years ago, various modifications have been advocated by Dal Pont, Hunsuck, and Epker to decrease the incidence of its complications [2].

Particularly in elective orthognathic surgery, it is important that surgeons inform their patients about the risk of these complications and attempt to minimize these risks [3].

BSSO has included intraoperative and postoperative complications. Excessive bleading, insturment fracture, foreign body, soft tissue injury, nerve exposure and nerve injury, dental complications and bad split are included intraoperative complications. On the other hand, sensory disturbance, temporomandibular joint disorder, bone necrosis, skletal relaps, postoperative swelling, malocclusion, infection, psycholoical depression, respiratory diffuculty, neck pain, gastrointestinal disease are included postoperative complications [4,5].

One of the common operative complications during BSSO is a bad split. This unwanted fracture is normally located in either the distal (lingual plate) or the proximal cortical plate (buccal plate) of the mandible, and more rarely affects the coronoid process or the condylar neck [6]. The incidence of bad splits varies between 0,9 and 20%. Nevertheless, risk factors should be identified and reduced so far as possible, particularly because it is an elective operation [7].

The role of impacted third molars in unfavorable splits is controversial. Third molar extraction decision depend on surgeon experience and choosing method, the site, angulation, relative height and root form of the third molar and its morphological relation to the neurovascular bundle. If the surgeon has large experience, extraction third molar surgery and the same time BSSO has some adventages. Removing third molar during BSSO allows better operative wiew and reduces the risk of injury to the inferior alveolar nerve. Besides, other time surgery may cause occur bone loss from osteotomi line. But in not well defined cases, this operation prosidure not always safe and may cause occur neurosensorial injury [8].

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On the other hand, many study say that, tooth extraction should be done at least 9 to 12 months before surgery to allow for complete socket bone fill and maturation [3].

During the osteotomy can use chisels and separators. Mensing et al. says that using chisels in separation for fragment is safer than using separators for avoid neurovascular bundle injury[6].

Mandibular morphology has been reported to influence both the difficulty of the procedure and the risk of bad splits. Anglulation in bone on the osteotomy line may cause increase the risk of this complication [5].

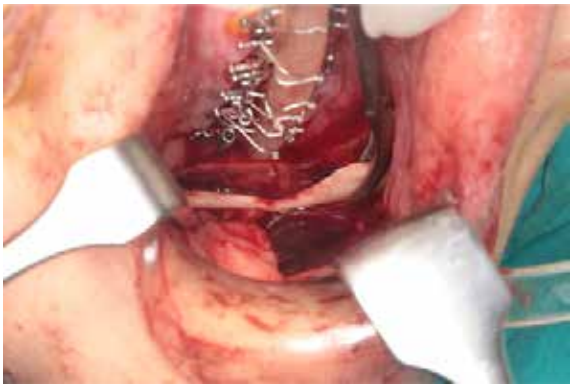
Some authors found that older age was a risk factor for bad splits. Other authors, however, reported that younger patients have an increased risk for bad splits.The higher incidence of bad splits in younger patients may be due to larger number in that age group [3,5,9].

Consequently, there is no consensus in the literature as to what combination of factors predisposes to a bad split.
When bad split occur, some concequenses may cause. These are mechanical instability, disturbance in body union, bone sequestiration, infection, temporomandibular joint disfunciton syndrome, neurovaskular damage and increase relaps risk [10,11].

When bad split occurs, the first rule is careful inspection on area. For inspections, periosteum dissections are needed. As a consequenses of dissection may occur intraoperative swelling and affect proxsimal segment. Excesive swelling withn joint an increase vertical joint space and in postoperative process can cause temporomandibular dysfuncion [7].

Case Series
The various types of bad split may require different salvage approaches [7].

Case 1: A 24- years old female patient was referred our clinic by orthodontic clinic for orthognatic surgery.



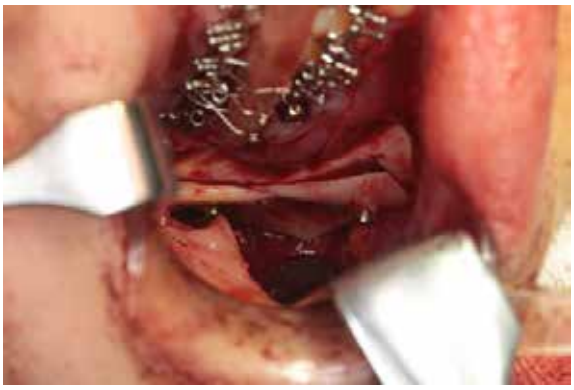


Fig1: Inferiorborderofmandiblewasattacheddistalsegment.

Corpus mandible was so concave and during medial split, inferior border of mandible wasattached distal segment. After carefully inspactation, team had seen that distal segmentinclude neurovascular bundle. Then, team decided that operation should be complete in thiscircumsentes.

Case 2: A 27- years old female patient was referred our clinic with a complain of prognathicmandible. She was found suitable for orthognathic surgery after radiographic and clinicalexaminations.

Fig 3: Corpus mandible was so thin. During osteotomy when using chisels proximal segmentbuccal wall was broken.

Fig 4: Broken and removed bone part.

According to operation's plan, mandible set back procude should had been complete. Segments of bone were close thanks to this procedur. And this bone part had not connect periosteum. Team had decided prefer remove this part of bone and finish mandibular set backoperation. Besides, this decision can protect for bone part from sequestration

In this case, for mandibular set back operation, none ed brokenpart osteo syntesis.

In the literature, proximal segment repatation is easly than other bad split's types. The difficulty of proximal segment fracture reduction depends on the fractured segment size and anatomical location [7].

When performing BSSO, high lingula position, high osteotomy line on horizontal split, excessive force for using chisel can cause bad splits on distal segment. In this area bad splitcan be occur a vertical line of lingual cortex of mandible, distal segment can be include coronoid process and distal segment

can be include coronoid process and joint area. It has been proposed that when third molar area is thin than desiare, bad split can be occur in lingual cortex of mandible. Third molar extraction complate 9-12 month before surgery can be protect for this type of split.

Case 3: A-28 years old male patient was referred to our clinic for orthognathic surgery.

High lingula position and high osteotomy line on horizontal split, incomplete osteotomy lineon assending ramus area can be cause bad split influence condyle and coronoid process region.

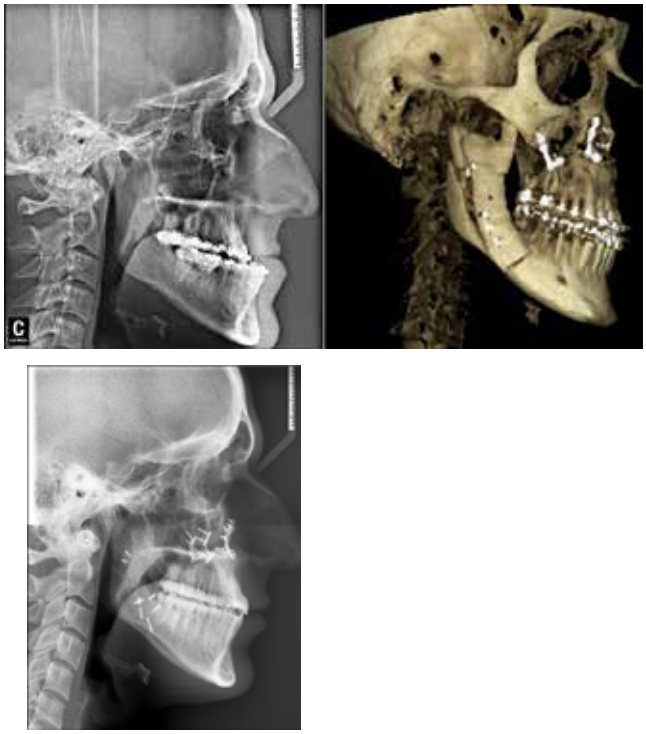


Fig 5: During BSSO, many area had been bad split. On the surgery, this complication had treated with three screws.

High lingula position was cause high osteotomy line on horizontal split. During osteotomy, bad split was occur condylar process. Dissection was complete this region then condyl was stabilized proximal segment with two screws.Then new horizontal osteotomy line was created. During the medial osteotomy buccal cortex of mandible was broken because of mandible corpus was so concave. It was seen that broken bone part was connected to periosteum then team had decided reunion this part with one screw.

Lingual segment fractures may be challencing to repair. Because, this part of bone is not seen easly therefore surgeon must be complete osteotomy for his experience and this area is not stabilized with ease.

Discussion

BSSRO has some intraoperative and postoperative complications. One of the most common of these is the bad split.

In our clinic, complete a total of 102 bilateral sagittal split osteotomies, these of six bad splitsoccurred, including five buccal plate fractures, one distal segment fractures on condylar process. All bad splits were managed intraoperatively without the need for specific additional measures, such as rigid intermaxillary fixation or a prolonged stay. All the fragments were immediately stabilized using screws osteosynthesis. All cases showed good and functional occlusion 6 months postoperatively.

Even if a bad split occurs, this has no influence on the postoperative course or the end result. All bad splits could be easily repaired by additional osteosynthesis measures resulting inenoughrigid skeletal fixation, not require post operative intermaxillaryfixation.

As a final remark, the occurrence of bad splits cannot always be avoided. When treated well the chances of functional success are good [2].

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Source of Finance
Not applicable

Conflicting Interests
Not applicable

Authorship of Contributions
SDS wrote the article.
TB, YF and. GK designed the research and performed surgery.
MFŞ is our language supervisior.
TY is collected the references and cited them.

EurAsian Journal of Oral and Maxillofacial Surgery
Official publication of AÇBİD (Association of Oral and Maxillofacial Surgery Society)

CASE REPORT

Management of Complex Odontoma in Posterior Maxilla: Case Report

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Abstract

Objective

Odontomas are hamartomas which consist of different types of dental tissue (enamel, cementum, dentin). They are slow-growing benign tumors with unknown etiology. Thus, they are detected in routine radiographs and have non-aggressive behaviour.

Cases

A 13-year-old girl was referred to our department for the evaluation of well-defined radiopaque mass associated with impacted upper right second molar adjacent to maxillary sinus. After clinical and radiological examination, surgery was performed. The mass was removed with associated tooth. Removed mass was sent to histopathological examination and reported as complex odontoma.

Conclusion

Odontomas are usually asymptomatic and are detected during routine radiographic examinations. The treatment of choice is surgical removal, usually performed by ostectomies to expose the tumor. However, close follow up and frequent control is important for the successful treatment.

Keywords: Odontoma, impacted tooth, radiopaque mass

Introduction

Odontomas are developmental disorders of dental organs and may relate to permanent tooth as well as retained decidious tooth. Odontomas can cause impaction, malformation, aplasia and devitalization of adjacent teeth. They are slow growing, benign mixed odontojenic non-aggressive tumors; mostly show no symptoms without exposed the oral cavity[1,2]. Their etiology is not known exactly although trauma, genetic mutation in the tooth germ and infection have been asserted[3,4]. Odontomas have no sex predilection. They can come about any age but highest prevalance is in first twenty years of the life[5].

WHO 2017[5], has divided odontomas into two categories as compound and complex type. The radiopaque mass in compound odontoma composed of many tooth-like component, on the other hand complex odontoma composed of disorganized dental tissue[5]. Compound odontoma mostly occur in the anterior upper jaw, whereas complex odontoma are found often in the posterior lower jaw[6]. We document a case report of female patient with complex odontoma.

Case Report

A 13-year-old apparently healthy female patient was referred to our department with the radiopaque lesion over the upper right second molar tooth. Patient had no complaint about such lesion. It is not exposed to oral cavity. Surrounding mucosa was not ulcerated and had no history of infection. Both intraoral examination and extraoral examination was normal. To define the exact position of the lesion, and its proximity to the nearby anatomical vital structures, Cone Beam Computer Tomography (CBCT) was requested. CBCT revealed dense, disorganized, radiopaque mass limited by corticated border, positioned distal to upper first right molar tooth and over the impacted upper right second molar (Figure 1). Taking into considering these findings, preliminary diagnosis was complex odontoma.

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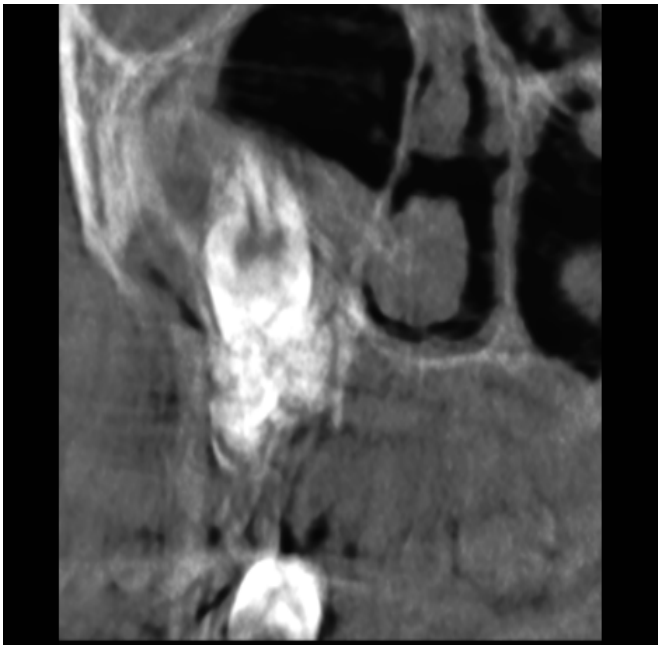

Figure 1. Disorganized radiopaque mass (blue arrow) and proximity of tooth for the sinus floor (red arrow)

Surgical consent was taken from the patient's parent. After local anesthesia was performed, surgical access to the lesion was accomplished via the intraoral vestibular sulculer route. Buccal bone guttering was performed to reach the lesion. The lesion was removed. The associated impacted tooth was extracted (Figure 2).

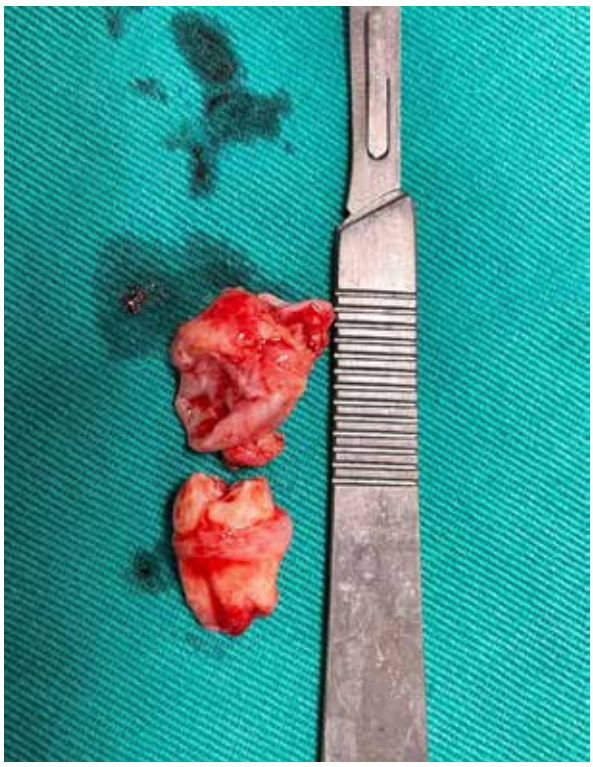

Figure 2. Removed lesion (red arrow) and tooth (yellow arrow)

Orthodontic traction was not the treatment of choice for associated tooth due to both its mobility and its absence of occlusion. The surgical region was curetted and irrigated with saline. The flap was replaced with 4-0 cutting vicryl suture. The specimen showed a yellowish calcified mass with an irregular surface also showing differences from normal tooth structure. The retrieved specimen was sent for histopathological examination. It was reported as a complex odontoma. The postoperative period was uneventful. Recurrence was not detected during six months follow-up (Figure 3).

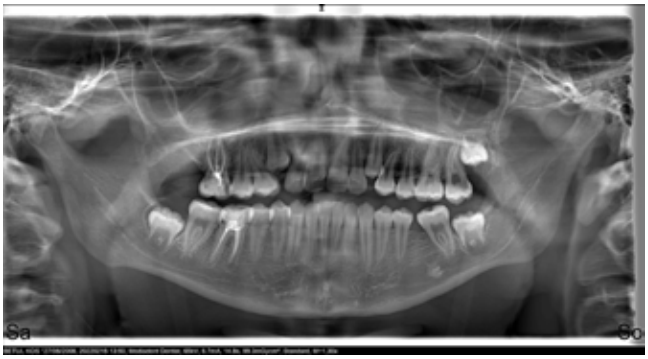

Figure 3. Six months follow up

Discussion

Odontomas are the most prevalent developmental mixed odontogenic tumors. They are often discovered as radiographic findings. Although odontomas develop in the tooth-bearing area, compound odontoma is mostly located in anterior upper jaw, whereas complex odontoma occurs into posterior lower jaw[5].

Treatment of the odontomas is surgical therapy. After the enucleation of the odontoma, the recurrence rate is very low. Evaluation of the root development of the associated impacted tooth is important for either orthodontic traction or extraction. Because of both mobility of the impacted tooth and the absence of its occlusion, we decided to the extraction of the tooth. The unique clinical and radiographic findings of odontoma help clinicians predict an accurate diagnosis. If the odontoma is discovered earlier, the surgical burden both for clinician and patient decreases dramatically 8formerly named ameloblastic fibro-odontoma (AFO.

Acknowledgement

Source of Finance

There is no financial source

Conflict of Interest

There is no conflict of interest between authors and all authors contributed to study.

Written consent was obtained from the patient's parent for publication.

Authorship of Contributions

All authors contributed to study.

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Running Head: Complex Odontoma

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EurAsian Journal of Oral and Maxillofacial Surgery
Official publication of AÇBİD (Association of Oral and Maxillofacial Surgery Society)

CASE REPORT

Conservative Treatment of Mandibular Condyle Fracture in a Patient With Wegener's Granulomatosis

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Abstract

Introduction

Wegener's granulomatosis (WG) is a multisystem disease with significant morbidity and mortality, which limits invasive procedures both concerning the disease and drugs.

Case Report

The patient who applied to our clinic with a history of trauma was a 71-year-old woman. The patient had a non-displaced fracture in the left mandibular condyle at the intracapsular level and clinically vertical loss in the relevant region. A total prosthesis was applied to the patient to increase the vertical dimension in the fracture area.

Conclusion

In this case, we describe the successful management of unilateral condyle fracture with a conservative method.

Keywords: Conservative Treatment, Mandibular condyle fracture, Wegener's granulomatosis

Introduction

Wegener's granulomatosis (WG) is an idiopathic, systemic inflammatory disease characterized by necrotizing granulomatous inflammation and pauci-immune small vessel vasculitis of the upper and lower respiratory tract and kidneys. The clinical apperations of WG are usually limited to the lower and/or upper respiratory area. The lungs are commonly involved and renal involvement may also be observed. Although rare, cardiac involvement may include [1, 2].

Strawberry gingivitis is one of the signs of WG and this feature, thought to be an early manifestation is extremely rare but characteristic. The disease may run a course that might vary from indolence to one of rapid progression leading [3]. Less common sites of involvement include the skin, central nervous system, salivary gland, breast, eye and orbit, spleen, gastrointestinal system, the thyroid gland, pituitary gland, and urogenital system [1, 4].

WG has initial presenting symptoms including salivary gland enlargement oral and/or nasal ulcers. Oral manifestations of WG include delayed healing of extraction wounds, osteomyelitis, resorption, and osteonecrosis. WG is rare, the mean age at diagnosis is 55 years affects both genders, equally [1 5].

Case Report

The patient, who applied to our clinic with a history of trauma to the jaw area due to falling on a hard floor, was a 71-year-old woman. Her medical history was significant for heart rhythm disorder and WG. She was taking medications to manage these conditions, including methylprednisolone and methotrexate. In his clinical examination, widespread ecchymosis spreading to the extraoral neck planes, and limitation in mouth opening was observed (Figure 1). Radiographic examination revealed a non-displaced fracture in the left mandibular condyle at the intracapsular level and clinically vertical loss in the relevant region (Figure 2,3). A total prosthesis was applied to the patient to increase the vertical dimension in the fracture area (Figure 4). Fracture treatment was carried out by preventing complications that may occur in invasive procedures with conservative treatment (Figure 5).

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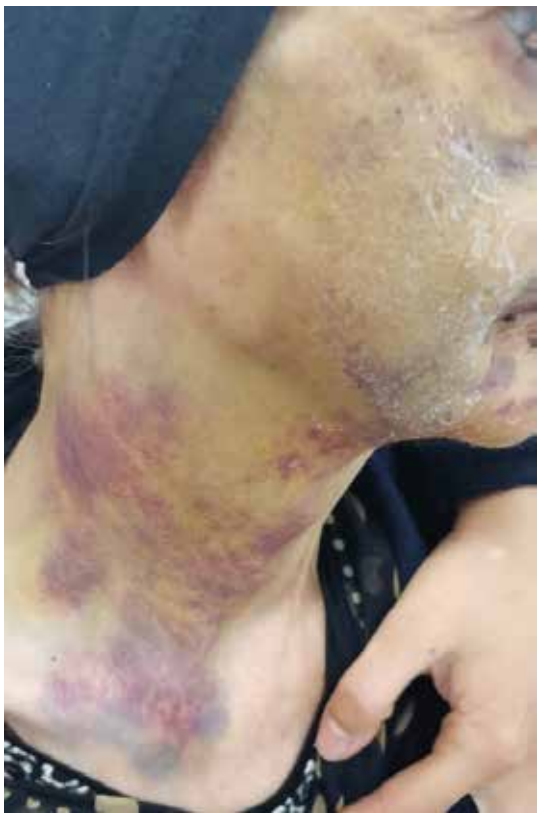

Figure 1: The view of facial and neck ecchymosis of patient

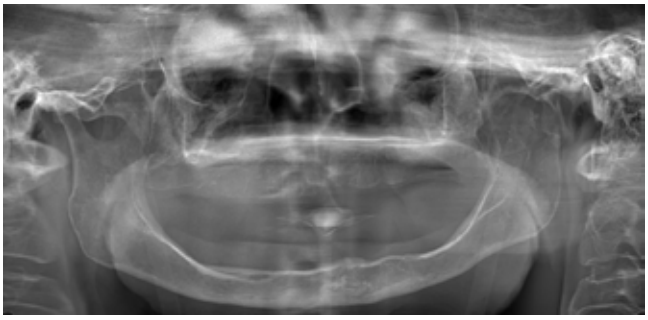

Figure 2: Preoperative panoramic view

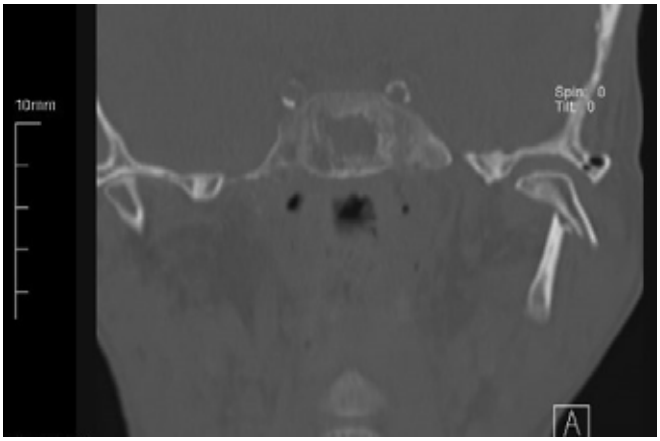

Figure 3: Preoperative Computed Tomography view

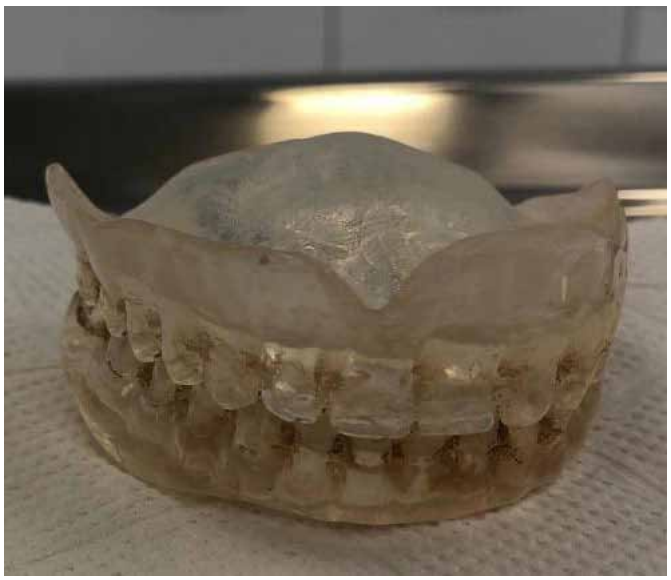

Figure 4: View of custom made total prosthesis of patient

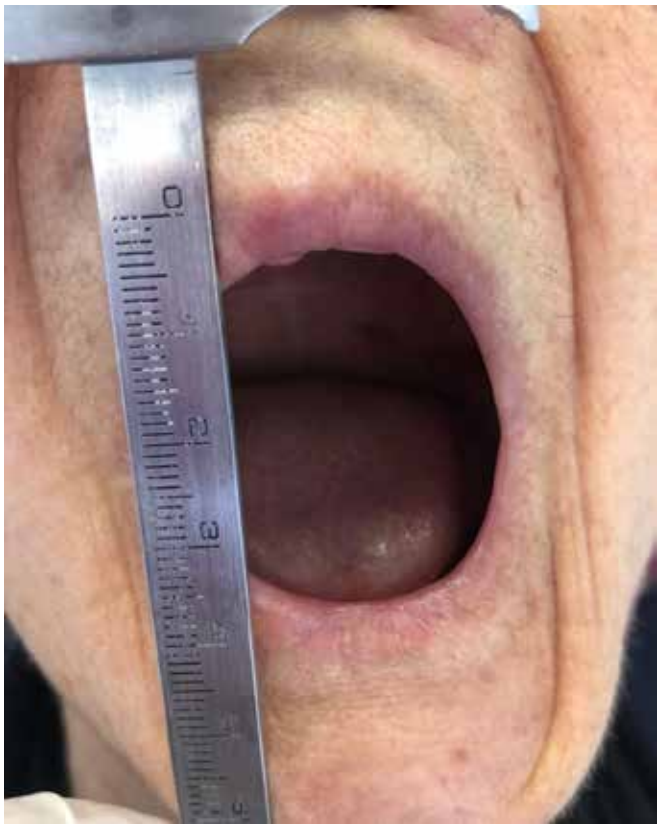

Figure 5: Postoperativ mouth opening mesurement

Conclusion

Closed reduction applied with the functional therapy method is a relatively sure treatment. There is no injury to the blood vessels and nerves during the treatment, and no complications such as infection or scarring occur after surgery[6]. Where condyle displacement is minimal and ramus height is close to normal, closed treatment can be applied[7 8]. Functional

treatment is indicated for intracapsular and extracapsular fractures in adults without serious condylar dislocation[7]. A conservative non-surgical approach such as a removable prosthesis becomes the treatment of choice[9].

In our cases, the patient was under the treatment of WG for 10 years. WG is a multisystem disease with significant morbidity and mortality, which limits invasive procedures both about the disease and drugs[1]. Immunosuppressive therapies that are applied in the management of WG will also be a risk factor for surgical approaches[1]. Our patient uses methylprednisolone and methotrexate. With this immunosuppressive drugs can cause metabolic disorders, including electrolyte disturbances and diabetes[10].

Bacteremia arising from invasive dental procedures represents a significant potential risk in the immunocompromised patient[10]. Caution should be exercised in invasive dental management decisions the patient may also encounter infections caused by the herpes simplex virus and Candida albicans, which usually originate in the oral cavity [10]. Therefore, the dental care provider may prefer closed reduction as a treatment option for WG patients on immunosuppressive therapy. Moreover, removable prosthesis hygiene habits should be given to the patient.

The choice of surgical and non-surgical treatment for condylar process fractures is controversial[11]. This clinical report describes the prosthodontic treatment of a unilateral condyle fracture.

In our case, we performed the treatment of unilateral condyle fracture with a conservative method. We did not encounter any problems in the follow-up of the patient. We presented our case report to increase awareness of WG and dental management strategies.

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Acknowledgement

Source of Finance

There is no financial source

Conflict of Interest

There is no conflict of interest between authors and all authors contributed to study.
Written consent was obtained from the patient's parent for publication.

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Running Head: Condyle Fracture in a Patient With Wegener's Granulomatosis

EurAsian Journal of Oral and Maxillofacial Surgery
Official publication of AÇBİD (Association of Oral and Maxillofacial Surgery Society)

REVIEW ARTICLE

Condylar Hyperplasia: Case Report and Literature Review

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Abstract

Introduction

The condition characterized through excessive growth of the mandible on the condyle and neck is called condylar hyperplasia (CH).It is considered as an uncommon malformation. Although the reason of this condition is not known exactly, some etiological factors have been suggested.These factors are; endocrinal disorders, trauma and local circulatory disorders. When CH is evaluated clinically, some symptoms such as facial asymmetry, prognathism, crossbite and open bite appear. While the acceleration of growth in one of the two developing condyles in adolescence period may cause this condition, another reason is enlargement of the condyle after stopping of skeletal growth. CH should be differentiated from structures such as osteochondroma and osteoma. During this distinction, the method such as scintigraphy is used in addition to histopathological and radiographic evaluations.

Materials and Methods

An observational study was used to create this report. Journal of Oral and Maxillofacial Surgery, International Journal of Oral and Maxillofacial Surgery, and Journal of Craniomaxillofacial Surgery publications that were available through to the PubMed database were included in the study.

Results

CH consists of a comprehensive examination and diagnostic methods for correct treatment. Patients often apply for treatment because of facial asymmetry and related aesthetic problems. Additional research is needed to compose a more standardized approach to diagnose CH activity

Keywords: Condylar hyperplasia, Facial asymmetry, Condylectomy

Introduction

The American Dental Association conducted a study to classify Temporamandibular Joint Diseases in 1986 (Table I).[1]

Disorders of chewing muscles	TMJ Disorders	Chronic Mandibular Hypomobility	Developmental Disorders
Protective co-contraction	irregularity in the condyle-disc complex	Ankylosis	Congenital and developmental bone disorders
Local Muscular pain	1. Disk displacement	1. Fibrous	1. Agenesis
Myofascial pain	2. Reduced disc dislocation	2. Bone	2. Hypoplasia
Myospazm	3. Disc dislocation without reduction	Muscle Contactures	3. Hyperplazi
Myositis and others	Structural incompatibility of joint surfaces	1. Miyostatik	4. Neoplazi
	1. Shape Changes	2. Myofibrotic	Congenital and developmental muscle disorders
	2. Adhesions	Chronoid impedance	1. Hypotrophy
	TMJ's inflammatory diseases		2. Hypertrophy
	1. Synovitis/capsulitis		3. Neoplasia
	2. Retrodisc		
	3. Arthritis		
	a. Osteoarthritis		
	b. Polyatrite		

Table 1: Classification of TMJ Disorders

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CH with mandibular asymmetry was first described in 1836 as a result of rheumatoid arthritis.[2] It was defined as a malformation independent of rheumatoid arthritis by Langenbeckin the next period, in 1853.[3]

When we scan the literature, CH cases are mostly seen bilaterally, and unilateral cases are seen less frequently.[4] The fact that bilateral cases are more common in males has led to the thought that this condition is inherited due to x-relatedness.[5] Since unilateral cases showed heterogeneous distribution in terms of gender, the cases were interpreted as multifactorial.[6] However, there are also studies in the literature showing that unilateral cases are seen 64% more in women.Therefore, it has been suggested that being a female is a risk factor.[7]

CH is a condition that usually appears between the ages of 10 and 30, appears in most cases at the end of pubertal growth. However, it has been suggested in some publications that cases can also be seen after adolescence period.[8] In addition, there are studies stating that CH can be seen at any age.[9]

It was also thought that the affected aspect may change depending on gender in the case of CH, and studies have been found showing that it is more frequently affected respectively on the right and left sides of women and men.[9] However, the absence of a significant difference in another study on which side was more common suggested that the right or left sides were equally affected.[7]

Classification

Depending on the authors, different CH kinds may be considered different.[10] Based on clinical features and origin, distinct forms of CH can be differentiated and classified.[11] A variety of classification schemes have been researched to better understand the pathophysiology and variations in the overgrowth locations. Two hemimandibular disorders, hemimandibular hyperplasia and hemimandibular elongation, were explicitly explained by Obwegeser and Makek in 1986.[12] A classification system was developed by Obwegeser and Makek using asymmetry and the totally dominant growth vector (Figure 1).

In their article, the writers divided CH in three types. They defined type 1 as excessive development in the horizontal direction and hemimandibular elongation. It was believed that Type 1 CH, which does not present asymmetry, is characterized by chin deviation to the unaffected side. Overgrowth is the origin of the elongation at the mandibular midline to the contralateral side. As a result, the lingual side of the contralateral mandibular molars frequently tend to stay in optimal occlusion with the maxillary molars. A crossbite may happen if the opposite molars are still unable to adapt to growth. The neck is typically impacted, but not the condyle. Hemimandibular hyperplasia connected to vertical overgrowth was classified as type 2 CH. Overall, there would be little jaw deviation. The maxillary molars on the affected side follow the downward growth of the jaw as compensatory for the downward overgrowth of the mandible. To keep the occlusion, the ipsilateral maxillary alveolar bone enlarges excessively. On the affected side, an open bite develops if the maxillary molars are still unable to keep up with excessive downward growth. The condyle frequently seems larger in type 2 CH, and the head is generally asymmetrical or malformed. Moreover, it has been claimed that the condyle neck enlarged and/or extended. With Types 1 and 2, Type 3 CH.[12,13]

Wolford et al. developed a classification scheme by taking into consideration the disorders that lead to CH. They categorized CH into four groups based on histology, clinical, imaging, and growth characteristics. In order to give patient care based on unique disease criteria, this system was developed to categorize CH into more distinct types[11] (Fig. 2).

Type	Clinical findings	Histological findings
Type I (Hemimandibular Elongation)	-Chin deviation towards contralateral side -Lingual deviation of contralateral mandibular molars -Possible posterior crossbite	-Excessive growth in the horizontal vector -Elongated mandibular ramus -Misshapen and slender condylar neck
Type II (Hemimandibular Hyperplasia)	-Sloping rimaoris with minimal chin deviation -Supraeruption of maxillary molars on affected side -Possible open bite No midline shift	-Excessive growth in the vertical vector -Enlarged and often irregularly shaped condylar head -Neck of condyle can be thickend and /or elongated
Type III (Combination of Type I and Type II)	-Chin deviation towards contralateral side with a sloping rimaoris -Midline shift -Possible open bite and/or cross bite	-Excessive growth in vertical and horizontal vectors -Enlarged condylar head,neck and ramus -Irregularly shaped condylar head, neck and/or ramus

Figure 1: Obwegener Classification

Type	Clinical finding	Histological finding
Type lA	-Bilateral mandibular elongation -No midline deviation -Prognathism and Class lll occlusion -Accelerated and prolonged growth	-Excessive growth in the horizontal vector -Condyle often unaffected -Bilateral elongated mandibular head, neck and ramus -Misshapen and slender condylar neck
Type 1B	-Unilateral mandibular elongation -Chin deviation towars contralateral side -Midline shift to contralateral side -Lingual deviation of contralateral mandibular molars -Possible posterior crossbite -İpsilateral class lll occlusion	-Excessive growth in the horizontal vector -Condyle often unaffected -Elongated mandibular head, neck and ramus -Misshapen and slender condylar neck
Type llA	-Unilateral vertical elongation of face -Sloping rimaoris with minimal chin deviation -Supraeruption of maxillary molars on effected site -Possible open bite -No midline shift	-Excessive growth in the vertical vector -Condylar enlargement without horizontal exophytic growth off condyle --Enlarged and often irregularly shaped condylar head -Neck of condyle can be thickend and /or elongated
Type ll B	-Unilateral vertical elongation of face -Sloping rimaoris with minimal chin deviation -Supraeruption of maxillary molars on effected site -Possible open bite -No midline shift	-Excessive growth in the vertical vector -Condylar enlargement without horizontal exophytic growth off condyle -Enlarged and often irregularly shaped condylar head -Neck of condyle can be thickend and /or elongated
Type lll	Unilateral facial enlargement	-Caused by bening tumor growth -Osteomas, neufibromas, fibrousdysplasia,giant cell tumor, chondroma, chondroblastoma, etc
Type lV	Unilateral facial enlargement	-Caused by malignant tumor growth -Caused bychondrosarcoma, multiplemyeloma, osteosarcoma, Ewing sarcoma and metastatic lesions

Figure 2: Classification of Wolford et al.

Type 1 and Type 2 CH may match the classification developed by Obwegeser and Makek in this approach.[12]

Type 1 is separated into 1 A and 1 B and is characterized by a rapid and protracted growth that produces condylar and mandibular extension. Mandibular elongation is categorized as bilateral in CH Type 1A and unilateral in CH Type 1B. CH Type 2 is defined by a vertical overgrowth of the mandible and unilateral expansion of the condyle carried on by an osteochondroma. CH Types 2A and 2B have been categorized by Wolford et al. Vertical elongation of the condyle head and neck leads in type 2A. In Type 2B, the condyle experiences horizontal exophytic tumor formation in addition to vertical head and neck enlargement. CH Type 3 including but not limited to fibrous dysplasia, osteomas, neurofibromas, and other benign tumors that cause CH, causing unilateral facial enlargement. Type 4 CH results from malignant tumors originating from the condyle, causing enlargement and facial asymmetry.chondrosarcoma, multiple myeloma, osteosarcoma, and Ewing's sarcoma are included in some tumors attributed to type 4 CH.[14]

Moreover, categorizations based on histological findings in CH patients have been developed. In a results released in 1986, Slootweg and Müller classified 22 patients into four groups based on histological findings in the various levels of hyperplastic condyles. They were the first to develop a histological classification system in accordance with this research. They analyzed the fibrous joint layer, undifferentiated mesenchymal layer, transitional layer, and hypertrophic cartilage layer in particularthen they categorized each layer in compliance with histological findings.A large proliferation zone, multiple cartilage islands, and a thick layer of hyaline growth cartilage are evident in CH Type 1's underlying bone layer. The location of proliferation zones in CH Type 2 is depicted as having less cartilage islands. CH Type 3 is distinguished by cartilage masses with irregular shapes that are found in the condylar neck bone or the area surrounding the superficial joint layer. When comparing to the histological results of normal condyles, Type 3 exhibits significant degeneration. A multicellular weak fibro-cartilaginous layer covering the subchondral bone plate is a feature of type 4 CH. Slootweg and Müller also pointed out that unlike other forms of hyaline growth cartilage, Type 4 CH does not have a proliferation layer.[15]

Reasons

Its pathogenesis and etiology are not clearly realized. Possible etiologies include hormonal changes (such as insulin-like growth factors [IGFs]), metabolic hyperactivity, trauma, arthrosis, heredity, circulatory issues, and exposure to abnormal force.[16,17]

CH may form as a result of condylar cartilage, according to these theories. A layer of fibro cartilage covers the condyle head's surface. Damage to this delicate layer of cartilage, which served as an important center for growth, may result in the condyle developing in a dysmorphic form.[18]

The fibrous joint layer, the undifferentiated mesenchymal layer, the transitional layer, and the hypertrophic cartilage layer are the four layers that make up the typical mandibular condyle soft tissue histology.[19] The mesenchymal layer in the active CH is assumed to be broader than in the normal condyle. Studies indicate that the number of chondrocytes impacted by CH increases dramatically when IGF-1 and IGF-1 receptor (IGF-1R) are expressed.[20]

Additionally, research indicates that as compared to normal condylar cartilage, CH exhibits much higher levels of collagen type II A1 gene expression.[20] These findings suggest that IGF-1 affects the chondrogenesis of the mandibular condyle, although it is uncertain how much of an impact it has and how it works.[10]

In a study, Saridin et al. hypothesized a link between the PET test, blood flow, and CH.[21] In contrast, another study found no association between blood flow and CH.[22]

Additionally, research link the hormone estrogen and its high prevalence in women.[7] This link is explained by estrogen hormone's control over bone formation and its presence in articular cartilage and growth centers.[23,24]

Clinical Studies

In the case of CH, the mandible is moved in the direction of the unaffected condyle, causing facial asymmetry. Crossbite and mandibular prognathism follow this. Additionally, noises, joint pain, and a limited mouth opening are all visible.[16,17,25]

When the literature is scanned, facial asymmetry is the most prevalent patient complaint, while discomfort and dysfunction are less frequent.[9]

If CH develops after adolescence, the posterior open bite problem is faced. If CH develops throughout adolescence, there is no open bite in the posterior with compensation in the teeth.[8]

Enlargement on the same side of the face and a flattened appearance on the other are the primary clinical features of unilateral CH.[26]

Differential diagnosis

In order to choose the right treatments and timing, an accurate diagnosis is essential.[10] Giant cell tumor, fibroosteoma, myxoma, fibrous dysplasia, fibrosarcoma, chondrosarcoma, and osteochondroma should all be taken into consideration in the differential diagnosis of CH.[9] CH may cause osteoma, osteochondroma, and resorption of the contralateral condyle.[27] Condylar osteomas are incredibly uncommon. Contrary to CH, which is radiopaque, condylar osteomas can be identified from it radiographically because they frequently provide a mixed radiolucent-radiopaque appearance.[28]

CT imaging can be used to discriminate between condylar osteochondromas. Contrary to the homogeneous enlargement of the condyle head that is typical of condylar hyperplasia, condylar osteochondroma tends to show growth that is morphologically distinct from the normal condyle on coronal and sagittal CT scans.[29]

Diagnosis

Different techniques are employed to diagnose CH. Correct CH diagnosis is crucial when deciding how to treat the ailment. It's crucial to correctly diagnose CH activity in order to avoid a recurrence after surgery.[30]

CH can be diagnosed using diagnostic techniques such clinical examinations, radiography, and nuclear imaging. The gold standard has been identified as clinical diagnosis.[21]

These aberrations may show up clinically both separately and together. The proper identification of CH has been aided by the use of numerous diagnostic criteria and techniques. CH can be effectively treated with high success rateswith the right diagnosis, timing, and treatment.In addition to histopathological, clinical and radiographic evaluation, scintigraphy is also used in the diagnosis of CH.[17,25]

Radiology

Condylar hyperplasia is indicated by clinical signs such malocclusion and facial asymmetry, but radiological analysis is necessary for a conclusive diagnosis. Computed tomography (CT), magnetic resonance imaging (MR), arthrography, and direct radiography are imaging techniques routinely utilized in the temporomandibular joint.[18]

Despite the fact that CH cases have been investigated since 1899, the measurement method and the definition of the term "hyperplasia" remain unclear.[31] Through Levandoski analysis, Kubota et al. assessed how the coronoid and condyle apex related to the Gonion level.[32] Using the deepest point of the sigmoid notch as a reference, Tavassol et al. compared the coronoid and condyle.[33]

Higashi evaluated the relationship between the condyle and coronoid crests while measuring in the panoramic image; if there is a 4 mm difference in favor of the coronoid, coronoid hyperplasia would be mentioned; as a result, he claimed that the patient did not need to have a restriction in mouth opening.6 To track growth, cephalometric images taken every 6 to 12 months and scintigraphic analyses with "99m technetium pyrophosphate" are utilized.[17,25,34]

Single photon emission computed tomography (SPECT) can be used to assess the sequence activity of CH.[35] Quantitative comparisons are made between the side that appears normal on SPECT and the side with the unilateral hyperplastic condyle.[30] Between normal condyles, an activity differential of 0–5 percent is typically seen. CH is suspected in a condyle with increased activity if the difference in activity between the two condyles is greater than 10%.[21,36]

Condyle assessment is done in three dimensions using computed tomography images.[2] By removing the zygomatic arch's superposition in panoramic radiographs with the use of cone-beam computed tomography, whose indication area is expanding daily in dentistry, CH cases are given a clearer inspection.[37]

Additionally, temporomandibular joint (TMJ) illnesses such midface pain and limited mouth opening might mimic the symptoms of CH patients. To prevent incorrect diagnosis and treatment, the examination in this situation should focus less on the TMJ region and more anteriorly on the coronoid process and zygomatic arch. This is confirmed by radiographic results.[33]

Treatment

Orthognathic surgery, condillectomy along with orthodontic treatment, or high condylectomy alone are all possible treatments for CH.[38] Surgery is the main CH therapy. Additionally, orthodontic therapy can be necessary. The best course of action depends on the patient's age, the degree of hyperplasia, and whether condylar development persists.[12]

A treatment strategy should be created following a thorough CH diagnosis. To rectify the occlusion, the primary mode of treatment is surgery, which is frequently supplemented with orthodontics. The best course of treatment and the length of the course of treatment are topics of discussion. The level of asymmetry, the condition of the malocclusion, and the rate of condylar growth should all be taken into account when creating treatment plans.The solutions to these issues can be thought of together. The chosen approach typically depends on the patient's age and development activity. The patient's wants and expectations are additional crucial factors, as is always the case. In a study by Naini et al., it was discovered that the patient's desire for surgical intervention grows in direct proportion to the degree of asymmetry. It's interesting that they said laypeople could detect asymmetries as tiny as 5 mm.[39]

The simplest, least intrusive, and most sophisticated CH therapy methods should be chosen. Mandibular ramus osteotomy of the afflicted condyles is one of the most straightforward treatments that may be carried out. Over a ten-year span, Motamedi performed ramus osteotomies on 13 CH patients. Of these 13 patients, seven underwent bilateral osteotomies alone, six underwent unilateral osteotomies, and in two of those situations, Le Fort I procedures were added. The study shown that unilateral ramus osteotomies on the afflicted side can be used to successfully treat patients with unilateral CH.However, it should be emphasized that unilateral osteotomies may result in excessive rotation of the unaffected condyle in individuals with severe prognathic profiles. Bilateral osteotomies did not demonstrate any advantage over unilateral procedures. In the investigations that were conducted, combining osteotomies with Le Fort I was proven to be helpful in resolving occlusal discrepancies.[40]

High condylectomies are recommended for treating CH in many research and case reports. An examination of 22 patients who underwent high condylectomies and underwent a 4-year follow-up revealed that this technique is a practical, beneficial, and reliable surgical choice for patients.[15]

High condylectomy has been recommended as a suitable treatment for unilateral CH by Lippold et al. In their research, the afflicted condyle's top pole was removed, and this procedure appeared to successfully inhibit CH growth.[41]

The condylectomy and orthognathic surgery combination is the most involved procedure for CH. High condylectomy and orthognathic surgery were employed to treat a group of patients in a well-known study by Wolford et al. in 2002, and it was found that this combination of procedures was quite successful in addressing both functional and aesthetically unappealing CH-related issues.11Another study that supports this assertion found that in 30 out of 36 patients, combined condylectomy and orthognathic surgery was beneficial from a functional and cosmetic standpoint. In the same study, it was asserted that orthognathic surgery without any condylar intervention would result in issues down the road because condylar growth might not take place. This viewpoint contends that growth may proceed following orthognathic surgery without the need to repair the hyperplastic condyle.[42]

On the other hand, if excessive condyle growth has halted, orthognathic surgery alone might be a suitable treatment. 44 CH patients were surgically treated in research that was undertaken when it was confirmed that excessive condyle growth had halted. Evaluation was accomplished by capturing numerous spect pictures for a certain amount of time in order to compare the quantity of active growth in each condyle, where condyle growth ceased. Timing and patient preferences are critical in choosing the best CH treatment strategy for all surgical treatments.[43]

There is very little research on orthodontic care for people with CH, and what there is typically consists of case studies. It was stated that mild to severe CH can only be treated surgically in a report of five instances conducted by Rajkumar et al., and that orthodontic treatment would not be sufficient on its own.[44]

According to several research, orthodontic therapy is required to maintain the occlusion following surgery. In a case requiring two-stage surgery, Xavier et al. categorized their methods as orthodontic treatment and orthognathic surgery followed by condylectomy.[45]

Each of these studies' findings only explains the therapy applied and which technique gave these patients' relevant outcomes. A trustworthy suggestion for treatment cannot be made because there is no comparable literature available. It is crucial to ensure that the growth has stopped before beginning orthodontic treatment if braces are chosen. To avoid the possibility of the deformity returning, corrective osteotomy ought to be used after bony growth has stopped.[30,46,47]

The age of the patient, the state of condylar growth, and the severity of the issue are all taken into account by the surgeon and orthodontist when creating the treatment plan.[12,48]

Surgical operations involving the maxilla and mandible are planned if bone growth is finished, and a suitable occlusion and skeletal connection are attempted. High condylectomy is favored in older patients, albeit, if condylar development is still present. As a result, identifying whether the growth persists in the condyle is important for developing a therapeutic strategy.[2] It is possible to encounter some serious complications in condylectomy cases. These serious complications are as follows; open bite, shifting of the mandible towards the operated side during mouth opening, TMJ ankylosis, failure to regain normal healthy functions, and inability to perform lateral movements because the lateral pterygoid muscle cannot reattach to the operated condyle.[49-51]

Result

CH includes a thorough assessment and diagnostic techniques for the right kind of care.Patients frequently request therapy due to facial asymmetry and associated cosmetic issues.
To create a more uniform method of diagnosing CH activity, more study is required.
Clinicians can employ diagnostic techniques like clinical examination, radiography, and scintigraphy while planning surgeries. The most effective treatment options need to be determined by longer follow-up research. In order to diagnose CH earlier and offer better treatment options, more research is required to understand its underlying causes.

Our Clinical Case

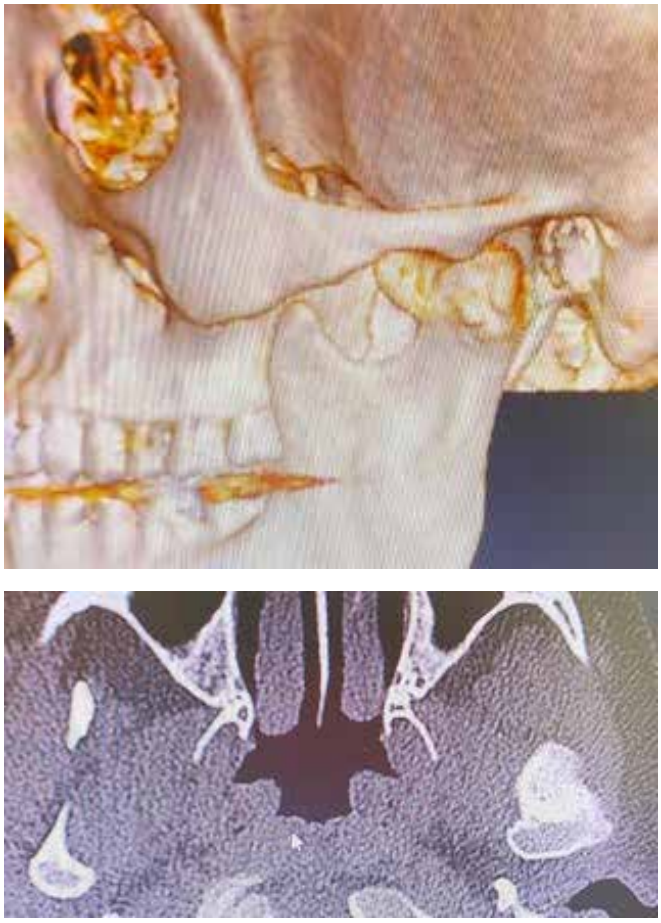

Figure 3 and 4: Images of the patient with CH

A 33-year-old female patient applied to our clinic due to the asymmetry complaint that she noticed one year ago.In the anamnesis taken from the patient, it was learned that she was systemically healthy.The patient stated that she had difficulty in eating due to the incomplete closure of her teeth. When clinical evaluation was applied, it was seen that there was laterognathia on the right side. After the panoramic and CT evaluation, it was decided to perform condillectomy under general anesthesia (Figure 3). Before the procedure, the patient was asked to sign the consent form by explaining the possible complications. A preauricular incision was made under general anesthesia and the condyle was reached after dissection. No appliance was placed in the area after condillectomy. The area was closed primarily and the removed tissue was sent for pathological evaluation. At the end of the pathology, it was learned that she had osteochondroma. In the evaluation applied immediately after the procedure, it was observed that there was no neurological injury. It was determined that the patient's occlusion problems were removed and the difficulty in eating was eliminated, and the asymmetry complaint decreased.

Source of Finance

none

Conflict of Interest

none

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