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

ELİF ESRA ÖZMEN[1], Doğan DOLANMAZ[2]

[1]Karamanoğlu Mehmetbey University, Ahmet Keleşoğlu Faculty of Dentistry, Oral and Maxillofacial Surgery Department, Karaman
[2]Bezmiâlem Vakif University, Oral, Dental and Maxillofacial Surgery Department

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

Corresponding Author: Elif Esra ÖZMEN
Assistant Professor
Address: Karamanoğlu Mehmetbey University, Ahmet Keleşoğlu Faculty of Dentistry, Oral and Maxillofacial Surgery Department, KARAMAN
Mobile: +90(507) 1524213
E-mail: elifesraozmen89@gmail.com

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

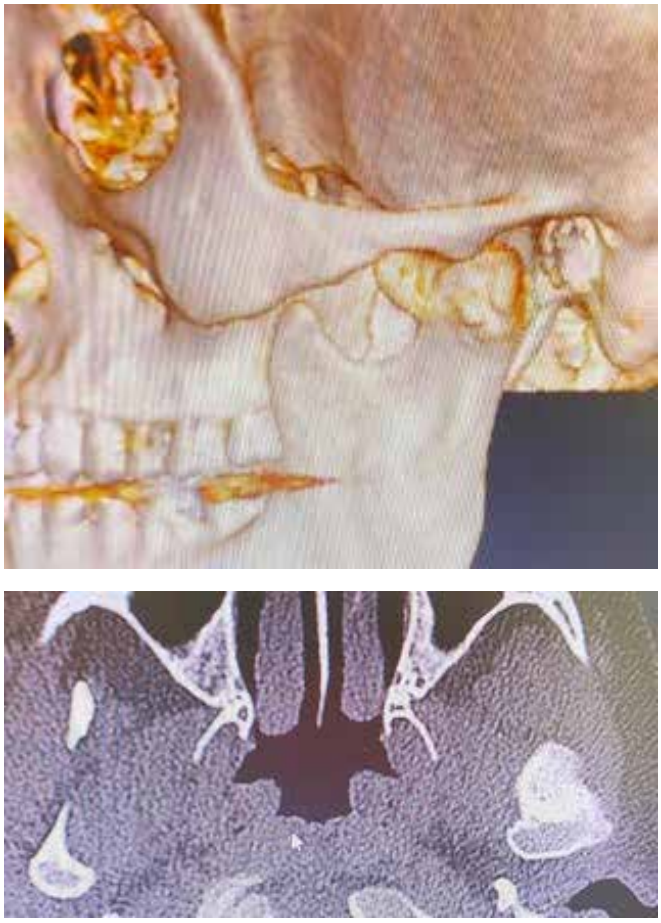

Figure 3 and 4: Images of the patient with CH

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

References

1. Güreser G, Temporomandıbular joınt dısorders. The Eur J of Med. 2003;6(2):37–45.
2. Giray B, Aktaş A. Yetişkin hastada kondiler hiperplazi. J Hacettepe Faculty of Dent. 2008;32 (3):45-50.
3. Hernáandez-Alfaro F, Escuder O. Joint formation between an osteochondroma of the coronoid process and the zygomatic arch (Jacob disease): report of case and review of literature.J of oral maxillofacial surg.2000;58(2):227-32.
4. Costa YM, Porporatti AL, Stuginski-Barbosa J. Coronoid process hyperplasia: an unusual cause of mandibular hypomobility. Braz Dent J. 2012;23(3):252–5.
5. McLoughlin P, Hopper C. Hyperplasia of the mandibular coronoid process: an analysis of 31 cases and a review of the literature.J of oral and maxillofacial surg.1995;53(3):250-5 .
6. Kim S, Lee J, Kim H. Mouth opening limitation caused by coronoid hyperplasia: a report of four cases. J of the Korean Association of Oral and Maxillofacial Surg 2014; 40(6): 301-7.
7. Raijmakers PG, Karssemakers LHE, Tuinzing DB. Female predominance and effect of gender on unilateral condylar hyperplasia: A review and meta-analysis. J Oral Maxillofac Surg. 2012;70(1).
8. Shetty S.Case Report: unilateral condylar hyperplasia. F1000Research 2021;10:46.
9. Nitzan D, Katsnelson A, Bermanis I. The clinical characteristics of condylar hyperplasia: experience with 61 patients.Joms.2007;66(2):312-8.
10. Almeida L, Zacharias J, Pierce S. Condylar hyperplasia: An updated review of the literature. Korean J of Orthodontics 2015; 45(6): 333-40.
11. Wolford L, Movahed R. A classification system for conditions causing condylar hyperplasia. J of oral and maxillofacial surg.2014;72(3):567-95.
12. Obwegeser H, Makek M. Hemimandibular hyperplasia-hemimandibular elongation.J of maxillofacial surg.1986;14:183-208.
13. Bruce R, Hayward J. Condylar hyperplasia and mandibular asymmetry: a review.J Oral Surg.1968;24(4):281-90.
14. Larry M.Wolford, PushkarMehra. Efficacy of high condylectomy for management of condylar hyperplasia. Am J Orthod Dentofac Orthop. 2002;121(2):136–51.
15. Slootweg P, Müller H. Condylar hyperplasia. A clinico-pathological analysis of 22 cases. J maxillofacial surg.1986;14:209-214.
16. Muñoz MF, Monje F, Goizueta C, Rodríguez-Campo F. Active condylar hyperplasia treated by high condylectomy: Report of case. J Oral Maxillofac Surg. 1999;57(12):1455–9.
17. Gray R, Sloan P, Quayle A. Histopathological and scintigraphic features of condylar hyperplasia. Int j oral and maxillofacial surg.1990;19(2):65-71.
18. Tatlı D, Keles B.Unilateral kondiler hiperplazinin konik ışınlı bilgisayarlı tomografi ile değerlendirilmesi:iki olgu sunumu ve literatür derlemesi. J Dent Fac Ataturk Univ.

2010;3:198-204
19. Hansson T, Öberg T, Carlsson GE, Kopp S. Thickness of the soft tissue layers and the articular disk in the temporomandibular joint. Acta Odontol Scand. 1977;35(1):77–83.
20. Chen Y, Ke J, Long X, Meng Q. Insulin-like growth factor-1 boosts the developing process of condylar hyperplasia by stimulating chondrocytes proliferation.Osteoarthritis and cartilage. 2012;20(4):279-87.
21. Saridin CP, Raijmakers PGHM, Kloet RW, Tuinzing DB, Becking AG, Lammertsma AA. No Signs of Metabolic Hyperactivity in Patients With Unilateral Condylar Hyperactivity: An In Vivo Positron Emission Tomography Study. J Oral Maxillofac Surg. 2009;67(3):576–81.
22. Li QF, Rabie ABM. A new approach to control condylar growth by regulating angiogenesis. Arch Oral Biol. 2007;52(11):1009–17.
23. Talwar RM, Wong BS, Svoboda K, Harper RP. Effects of Estrogen on Chondrocyte Proliferation and Collagen Synthesis in Skeletally Mature Articular Cartilage. J Oral Maxillofac Surg. 2006;64(4):600–9.
24. Yu S, Xing X, Liang S, Ma Z, Li F, Wang M, et al. Locally synthesized estrogen plays an important role in the development of TMD. Med Hypotheses. 2009;72(6):720–2.
25. Hodder SC, Rees JIS, Oliver TB. SPECT bone scintigraphy in the diagnosis and management of mandibular condylar hyperplasia. Br J Oral Maxillofac Surg. 2000;38(2):87–93.
26. Lopez B, Corral S. Condylar hyperplasia: characteristics, manifestations, diagnosis and treatment. a topic review. Rev Fac Odontol Univ Antioquia. 2015;26(2):425–46.
27. Tripathi T, Srivastava D. Differential Diagnosis and Treatment of Condylar Hyperplasia. J Clin Orthod. 2019.
28. Valente L, Tieghi R, Mandrioli S, Galiè M. Mandibular Condyle Osteoma. Ann Maxillofac Surg. 2019;9(2):434.
29. Ito FA, De Andrade CR, Vargas PA, Jorge J, Lopes MA. Primary tuberculosis of the oral cavity. Oral Dis. 2005;11(1):50–3.
30. Pripatnanont P, Vittayakittipong P, Markmanee U. The use of SPECT to evaluate growth cessation of the mandible in unilateral condylar hyperplasia. Int J Oral Maxillofac Surg. 2005;34(4):364–8.
31. Bilgili E. Investigation of Unilateral Mandibular Coronoid Hyperplasia Cases Using Cone Beam Computed Tomography. Van Med J. 2017.
32. Kubota Y, Takenoshita Y, Takamori K. Levandoski panographic analysis in the diagnosis of hyperplasia of the coronoid process. Br J Oral Maxillofac Surg. 1999;37(5):409–11.
33. Tavassol F, Spalthoff S, Essig H.Elongated coronoid process: CT-based quantitative analysis of the coronoid process and review of literature. Int J Oral Maxillofac Surg. 2012;41(3):331–8.
34. Kaban LB, Cisneros GJ, Heyman S. Assessment of mandibular growth by skeletal scintigraphy. J Oral Maxillofac Surg. 1982;40(1):18–22.
35. Alyamani A, Abuzinada S. Management of patients with condylar hyperplasia: A diverse experience with 18 patients. Ann Maxillofac Surg. 2012;2(1):17.
36. Yang Z, Reed T, Longino BH. Bone Scintigraphy SPECT/CT Evaluation of Mandibular Condylar Hyperplasia. J Nucl Med Technol. 2016;44(1):49–51.
37. Sreeramaneni SK, Chakravarthi PS, Krishna Prasad L. Jacob's disease: report of a rare case and literature review. Int J Oral Maxillofac Surg. 2011;40(7):753–7.
38. S Sembronio, A Tel, F Costa MR. An updated protocol for the treatment of condylar hyperplasia: computer-guided proportional condylectomy. J Oral Maxillofac. 2019.
39. B.Naini F, A.Donaldson AN. Assessing the influence of asymmeftry affecting the mandible and chin point on perceived attractiveness in the orthognathic patient, clinician, and layperson. J Oral Maxillofac Surg. 2012;70(1):192–206.
40. Motamedi MHK. Treatment of condylar hyperplasia of the mandible using unilateral ramus osteotomies. J Oral Maxillofac Surg. 1996;54(10):1161–9.
41. Lippold C, Kruse-Losler B, Danesh G. Treatment of hemimandibular hyperplasia: The biological basis of condylectomy. Br J Oral Maxillofac Surg. 2007;45(5):353–60.
42. Villanueva-Alcojol L, Monje F, González-García R. Hyperplasia of the Mandibular Condyle: Clinical, Histopathologic, and Treatment Considerations in a Series of 36 Patients. J Oral Maxillofac Surg. 2011;69(2):447–55.
43. Yamashita Y, Nakamura Y, Shimada T. Asymmetry of the lips of orthognathic surgery patients. Am J Orthod Dentofac Orthop. 2009;136(4):559–63.
44. Gc R, Muralidoss H, Ramaiah S. Conservative management of unilateral condylar hyperplasia. Oral Maxillofac Surg. 2012;16:201-5.
45. Xavier SP, de Santana Santos T, Silva ER. Two-Stage Treatment of Facial Asymmetry Caused by Unilateral Condylar Hyperplasia. Braz Dent J. 2014;25(3):257–60.
46. Marchetti C, Cocchi R, Gentile L. Hemimandibular hyperplasia: treatment strategies. J Craniofac Surg. 2000;11(1):46–53.
47. Deleurant Y, Zimmermann A,Peltomäki T. Hemimandibular elongation: treatment and long-term follow-up. Orthodontics & Craniofacial Research.2008;11(3):172-9.
48. Matteson SR, Proffit WR, Terry BC. Bone scanning with99mtechnetium phosphate to assess condylar hyperplasia: Report of two cases. Oral Surgery, Oral Med Oral Pathol. 1985;60(4):356–67.
49. Sugawara Y, Hirabayashi S-I, Susami T. The Treatment of Hemimandibular Hyperplasia Preserving Enlarged Condylar Head. Cleft Palate-Craniofacial J. 2002;39(6):646–54.
50. Olate S, Netto HD, Rodriguez-Chessa J. Mandible condylar hyperplasia: a review of diagnosis and treatment protocol. Int J Clin Exp Med. 2013.
51. Cervelli V, Bottini DJ, Arpino A. Hypercondylia: Problems in diagnosis and therapeutic indications. J Craniofac Surg. 2008;19(2):406–10.