

Unilateral Condylar Hyperplasia – A Case Report with a Mini Review of the Literature

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Abstract

Condylar hyperplasia (CH) is a rare disorder characterized by excessive growth of the mandibular condyle in the horizontal and/or vertical dimensions. It usually presents unilaterally and can result in facial asymmetry, malocclusion and temporomandibular joint disorders. The etio-pathogenesis of CH is uncertain and has been associated with hormonal factors, trauma, heredity, hypervascularity, aberrant growth factors, infection and neoplasia. A variety of specialized nuclear medicine studies (e.g. scintigraphy, PET-CT and SPECT) are utilized to determine if there is pathological growth activity in the suspected condyle. It is essential to identify the condition as mis-diagnosis can lead to unexpected adverse growth even after the surgical correction of facial asymmetry. The authors report a case in a 16-year-old male subject who developed facial asymmetry (due to unilateral active CH) while undergoing routine orthodontic treatment for maxillary dento-alveolar protrusion. The patient was treated by high condylectomy and has been followed up for 1 year without signs of active growth. This paper also provides a mini-review of the recent literature on the epidemiology, etiology, diagnosis, classification, and surgical treatments of CH.

Keywords: Condylar hyperplasia, scintigraphy, PET-CT, SPECT, epidemiology, etiology.

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INTRODUCTION

Condylar hyperplasia (CH) is a rare disorder characterized by excessive growth of the mandibular condyle in the horizontal and/or vertical dimensions. It usually presents unilaterally and results primarily in facial asymmetry of the lower third of the face. Malocclusion and temporomandibular joint disorders may also be present. The etio-pathogenesis of CH is poorly understood and has been associated with hormonal factors [higher insulin like growth factor expression in chondrocytes affected by CH], trauma, heredity, hypervascularity, aberrant growth factors, infection and neoplasia [1, 2]. Majority of the patients present during the teenage years with a predilection for the female sex [2-4]. Many diagnostic tools and criteria have been used to aid in the correct diagnosis of CH, which in turn is critical to determining the appropriate treatments and timing. With proper diagnosis, timing, and treatment, CH can be effectively treated with a high success rate. The authors report a case in a 16-year-old male subject who developed facial asymmetry [due to

unilateral active CH] while undergoing routine orthodontic treatment for maxillary dento-alveolar protrusion. The patient was treated by high condylectomy and has been followed up for 1 year without signs of active growth. This paper also provides a mini-review of the recent literature on the epidemiology, etiology, diagnosis, classification, and surgical treatments of CH.

CASE REPORT

A 16-year-old male patient presented to the Department of Maxillofacial Surgery at Jubilee Mission Medical College and Research Centre, Kerala. The patient gave a history of childhood trauma to the lower jaw. He also gave a history of developing lower facial asymmetry and worsening occlusion over a span of 12 months after starting routine orthodontic treatment for correction of maxillary dento-alveolar protrusion and anterior open bite [due to tongue thrusting] 2 years ago. Clinically, there was facial asymmetry of the lower face with a mandibular midline shift to the left (Fig 1 & 2).

Mouth opening was not limited; however, there was a mild deviation of the jaw to the left side on opening. Palpation of the temporomandibular joints did not reveal any tenderness or clicking. Intraorally, there was an asymmetric anterior open bite (more severe on the right side) with mandibular deviation to the left (Fig-3). A recent panoramic radiograph showed the right condyle with regular anatomy but a slightly elongated neck and body (Fig-4). Bone scintigraphy was performed, and increased activity in the right TMJ was found (Fig -5). Due to the patient's age, clinical findings, and anatomic features of the affected condyle, the condition was diagnosed as CH Type 1B according to the classification given by Welford *et al.*, [1]. Under general anesthesia, a pre-auricular incision was taken and layer wise dissection was done to expose the affected condyle. A high condylectomy (excision of 7mm of the affected condylar head) and contouring of the remaining condylar head was performed (Fig-6)

followed by which layer wise suturing was done [3, 5-7]. The patient was administered anti-biotics for a period of 24 hours post-operatively and was discharged after 3 days. There was no evidence of facial paresis post-operatively. The histo-pathological report confirmed the provisional diagnosis of condylar hyperplasia type 1. The histology of the affected condyle was similar to a normally growing condyle without any notable pathologic abnormalities. There was slight widening of the fibrocartilage on condyle and increased vascularity in proliferative zone (Fig 7) [1]. The patient was followed for a period of 1 year post-operatively during which worsening of facial asymmetry was not noted. Mouth opening and excursive movements were minimally impacted after the high condylectomy [6, 7]. The patient is currently being planned for orthognathic surgery after 18 years of age once skeletal maturity is obtained.



Fig-1: Frontal view showing facial asymmetry of the lower face with a mandibular midline shift to the left

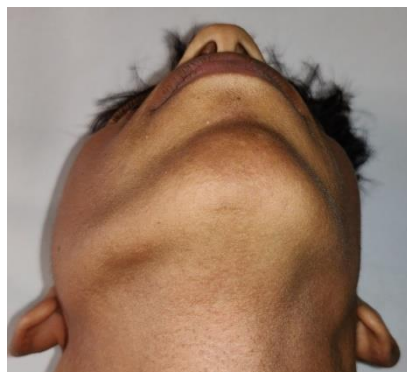


Fig-2: Submental view showing mandibular midline shift to the left



Fig-3: Asymmetric anterior open bite (more severe on the right side) and deviation of mandibular midline to the left



Fig-4: A recent panoramic radiograph showed the right condyle with regular anatomy but a slightly elongated neck and body



Fig-5: Bone scintigraphy revealed increased activity in the right TMJ

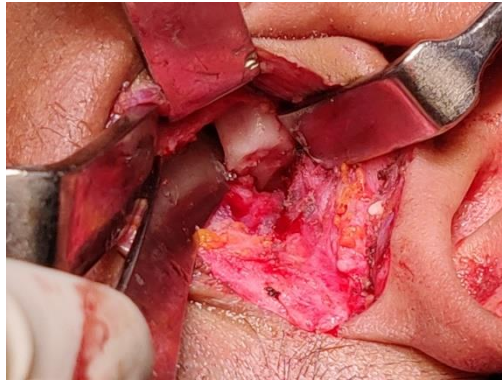


Fig-6: High condylectomy [excision of 7mm of the affected condylar head] and contouring of the remaining condylar head

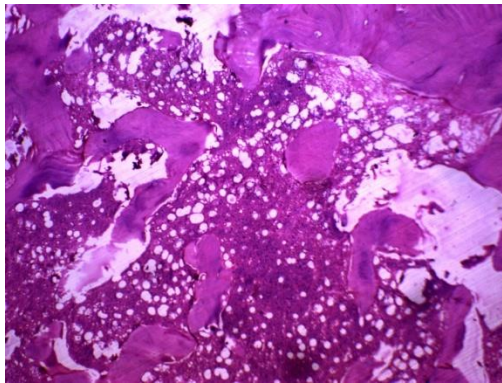


Fig-7: Histopathological examination revealed thickened irregular bony trabeculae, uninterrupted layer of undifferentiated mesenchymal cells, hypertrophic cartilage, islands of chondrocytes in subchondral trabecular bone and increased thickness of cartilaginous layer

Table-1: Obwegeser and Makek Classification of Condylar Hyperplasia 1986

Type	Clinical Findings
Type 1 (Hemimandibular Elongation)	<ul style="list-style-type: none"> • Chin deviation towards contralateral side • Midline shift to contralateral side • Lingual deviation of contralateral mandibular molars • Possible posterior crossbite • Excessive growth in the horizontal vector • Condyle often unaffected • Elongated mandibular ramus • Misshapen and slender condylar neck
Type 2 (Hemimandibular Hyperplasia)	<ul style="list-style-type: none"> • Sloping rima oris with minimal chin deviation • Supra-eruption of maxillary molars on affected side • Possible open bite • No midline shift • Excessive growth in the vertical vector • Enlarged and irregularly shaped condylar head • Neck of condyle can be thickened and/or elongated
Type III (Combination of Type I and Type II)	<ul style="list-style-type: none"> • Chin deviation towards contralateral side with a sloping rima oris • 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

Table-2: Various classifications of Condylar Hyperplasia

Author	Classification [CH = condylar hyperplasia]
Obwegeser and Makek 1986 [11]	Hemi-mandibular hyperplasia, Hemi-mandibular elongation and Hybrid hemi-mandibular hyperplasia
Nitzan <i>et al.</i> , 2008 [12]	Vertical CH, Horizontal CH and Combined CH
Wolford <i>et al.</i> , 2014 [1]	Type 1A [bilateral CH], Type 1B [unilateral CH], Type 2 [unilateral CH due to osteochondroma], Type 3 [unilateral CH due to benign tumors apart from osteochondroma] and Type 4 [unilateral CH due to malignant tumor]

Table-3: Wolford's Classification of Condylar Hyperplasia (CH) 2014

CH Type	Age at Onset	Clinical Findings	Imaging	Histology
CH Type 1 [similar to hemi-mandibular elongation]: Type 1A and Type 1B	Puberty	Type 1A [BL] or Type 1B [UL] accelerated growth; Self-limiting; Class 3 occlusion; Prognathic Mandible	UL/BL elongated condylar head, neck and body; Normal condylar head shape	Normally growing condyle; May show chondrocyte proliferation during initial and active phases, with normal bone after growth ceases
CH Type 2 [similar to hemi-mandibular hyperplasia]: Type 1A and Type 1B	2/3 rd of cases begin in 2 nd decade	UL vertical elongation of face and jaws; Non-self-limiting; Ipsilateral posterior open bite; Occlusal cant occasionally	Unilateral vertical enlarged condylar head, neck, ramus and body; Type 2A: Enlargement without horizontal exophytic growth of the condyle; Type 2B: Enlargement with exophytic growth of the condyle	Osteochondroma; Cartilaginous cap similar to that seen in a normal growth cartilage; Endochondral ossification; Cartilaginous islands in the sub-cortical bone; Thickened irregular bony trabeculae
CH Type 3	No specific age	UL facial enlargement	Varies from normal anatomy of condyle; usually presenting as condylar enlargement	Benign tumors, e.g., osteoma, neurofibroma, giant cell tumor, fibrous dysplasia, chondroma, chondroblastoma and arterio-venous malformation
CH Type 4	No specific age	UL facial enlargement	Varies from normal anatomy of condyle; usually presenting as condylar enlargement with lytic lesions	Malignant tumors, e.g., chondrosarcoma, multiple myeloma, osteosarcoma, metastatic lesion and Ewing's sarcoma

Table-4: Slootweg and Miller histopathological classification of Condylar Hyperplasia [21]

Type of CH	Slootweg and Miller Classifications
Type 1	A broad proliferation zone with an underlying thick layer of hyaline growth cartilage and bone that contained numerous cartilage islands.
Type 2	A patchier distribution of proliferation zones with a smaller number of cartilage islands.
Type 3	Irregular-shaped masses of cartilage found in the bone of the condylar neck or encroaching onto the superficial articular layer. Type 3 displayed great distortion compared to the histological findings of normal condyles.
Type 4	A burned-out appearance of the condyle due to a very cell-poor fibrocartilaginous layer covering the subchondral bone plate. Type 4 CH did not demonstrate a proliferation layer of the hyaline growth cartilage like that seen in the other types.

DISCUSSION

Historical Review and Classifications

Asymmetric conditions attributed to condylar hyperplasia of the mandible were first reported by Adams in 1836. Lohrann in 1918 reported about condylar hyperplasia followed by Guroca and Meisels in 1926 [8]. In 1946, Rushton reviewed 29 reported unilateral cases [9]. In 1951, Gottlieb described condylar hyperplasia as an osteoma causing unilateral deformity and prognathic deviation subsequent to a bilateral disproportion in the size of the condyles. In 1960, Rowe characterized unilateral condylar hyperplasia as being associated with elongation of the condylar neck, bowing of the inferior body of the mandible, lateral crossbite, and contralateral concavity of the lateral aspect of the ramus [10]. By 1968, a total of 150 cases had been reported in the literature, most of which were isolated [9]. Due to the variations in locations of excessive growth, multiple classification systems have been developed to better characterize the pathology (Table-2). In 1986, Obwegeser and Makek developed a classification system based on the asymmetry and predominant growth vector (Table-1). In 2008, Nitzan developed a classification system based on the predominant growth vector wherein he described CH as a unilateral disorder in which the pathology occurs at the head of the condyle, creating facial asymmetry in the vertical or horizontal direction or a combination of both. In 2014, Wolford developed an updated classification system that they considered more inclusive of pathologies causing CH (Table 3). Their report classifies CH into four different categories based on clinical, imaging, growth, and histological characteristics. This system was developed to classify CH into more specific types in order to provide optimal treatment to patients based on their specific disease characteristics [1-3, 11, 12]. Classification systems have also been created based on histological findings in CH patients. Slootweg and Müller were among the first to create a histological classification system based on a study they conducted in 1986, in which they classified 22 patients into four categories based on histological findings in various layers of hyperplastic condyles (Table 4). Specifically, they analyzed the fibrous articular layer, the undifferentiated mesenchymal layer, the transitional layer and the hypertrophic cartilage layer and characterized each layer based on histological findings [2].

Clinical Presentation and Etio-pathogenesis

CH can occur at any age and can continue past the growth period. It is usually unilateral and begins during the second decade of life around the pubertal growth phase and can continue into the middle or late twenties. It occurs predominantly in females (64%) but it is not clear whether this is a true predilection, or whether women are more likely to seek referral [4, 2]. Unilateral condylar hyperplasia is characterized by ipsilateral fullness of the lower third of the face with contralateral flatness and deviation of the chin away

from the affected side. The precise changes in facial appearance and occlusion, however, vary because abnormal growth can be vertical or horizontal or a combination of them both [3]. In vertical cases, there is down-growth of the ipsilateral mandibular condyle with minimal deviation of the chin or occlusal midline, and substantial sloping of the ipsilateral mandibular occlusal plane. The entire hemimandible looks enlarged in three dimensions, from ipsilateral condyle to symphysis. Initially, it causes an ipsilateral open bite, but gradual compensatory growth of the maxillary and mandibular dentoalveolar complexes results in an occlusal cant. Ipsilaterally, the mandibular body is bowed and the angle rounded; contralaterally it looks flattened. The inferior alveolar bundle remains in its position close to the lower border of the mandible because of overgrowth of the dentoalveolar segment. The whole face appears rotated [3]. The horizontal form presents with deviation of the chin and mandibular occlusal midline to the contralateral side, with a contralateral crossbite. The ipsilateral mandibular molars usually tip to maintain occlusion. The combined form presents with excess growth in both planes and clinical features of the vertical and horizontal types. The horizontal form seems to be more common than the vertical form, but estimates of relative incidence vary widely. In all cases, the increased functional load may cause contralateral temporomandibular dysfunction with associated pain and clicking [3]. Current research has yet to define an exact etiology for CH. Some researchers have supported the 'local circulatory theory', which claims that the abnormal growth of the condyle is caused by an increased number of capillaries in the posterior superior anatomy of the condyles. Possible etiologies include endocrine distortions (e.g., insulin-like growth factors [IGFs]), metabolic hyperactivity, trauma, TMJ loading, arthrosis, inflammation/infection of the temporomandibular joint (TMJ)/middle ear, osteomyelitis and genetics. Typical mandibular condyle soft tissue histology includes four layers: fibrous articular layer, undifferentiated mesenchymal layer, transitional layer, and hypertrophic cartilage layer. Active CH has been found to display a broader mesenchymal layer than that in the normal condyle. Wolford and LeBanc have suggested that insufficient bone plate closure when cartilage from the proliferative layer is replaced by bone around age 20 years, e.g., as also seen in chondromas, osteochondromas, etc., could be a possible cause [2, 3].

Diagnosis

Various methods are available for the diagnosis of CH. Correct diagnosis of CH is essential when deciding how to treat the condition. To prevent post-surgical reversion, accurate diagnosis of CH activity is also of utmost importance. Diagnostic methods such as clinical examination, radiographs, and nuclear imaging can be used to determine the type of CH as well as its activity. Clinical diagnosis has been described as the diagnostic gold standard [2, 3, 13-15]. Nuclear imaging is capable of providing physiological

details of CH using radionuclide-labeled tracers. Examples of different types of nuclear imaging include planar scintigraphy, single-photon emission computed tomography (SPECT) and PET. SPECT and planar scintigraphy utilize the radionuclide technetium-99m labelled methylene diphosphonate (99mTc-MDP), while PET utilizes the radionuclide (18F)-fluoride. Prior to the development of 99m Tc-MDP, (18F)-fluoride was the standard radionuclide tracer for SPECT. Planar scintigraphy produces a two-dimensional image, as opposed to SPECT and PET, which produce three-dimensional images [2, 3, 13-15]. Bone scintigraphy has high sensitivity and low specificity for bone metabolism, meaning that it can identify when a change in bone metabolism is present but is limited in its ability to differentiate among various conditions (e.g., bone healing, growth, infection, arthritic changes, or tumors). Generally, when condyles are being evaluated with bone scintigraphy, a difference in uptake levels of less than 10% indicates either normal condyles or individuals without progressive asymmetry. A meta-analysis by Saridin *et al.*, found that the SPECT technique of bone scintigraphy had a significantly higher sensitivity (0.90) in detecting unilateral CH than the planar technique (0.71) ($p = 0.04$). However, no difference in specificity was found between these two techniques. PET has been described as having better spatial resolution than SPECT. Further research is needed to establish a more formalized method for scintigraphy analysis. The current literature provides various methods such as comparing right and left condylar activity in the form of a percentage or ratio and comparing condylar activity to a different bony landmark such as the lumbar vertebrae. An attempt was made to relate SPECT findings to histopathological differences in CH, in which SPECT was found insufficiently sensitive to detect histopathological differences [2, 3, 13-15].

Identifying Condylar Hyperplasia Activity

Active CH growth can usually be determined by worsening functional and esthetic changes with serial assessments (preferably at 6- to 12-month intervals) consisting of clinical evaluation (surgeon's, orthodontist's, patient's report), photograph records, dental model analysis with orthodontically trimmed models or models mounted in centric relation, and radiographic evaluation by superimposition. Radiographic evaluation includes:

- Lateral cephalometric radiographs; during pubertal growth, the normal yearly growth rate of the mandible measuring from condyion to point B is 1.6 mm for girls and 2.2 mm for boys.
- Frontal cephalometric radiographs (particularly helpful in unilateral CH cases).
- Lateral cephalometric tomograms / OPG that include the TMJ, the mandibular ramus, the body, and posterior teeth to analyze the amount of condylar growth over time for each side.
- Bone scintigraphy or PET/CT scan are used to evaluate the metabolic activity of the bone.
- 3D-CT reconstruction of the facial skeleton to study the changes in bony morphology of the maxilla-mandibular structures.

When all the information, photographs, study models, radiographs, and bone scans are correlated over time, some indication of the activity can be made [16].

Differential diagnosis

The differential diagnosis for CH includes the following [3, 17]: maxillary hypoplasia, mandibular prognathism without mandibular condylar hyperplasia, dislocation of condyles anterior to the articular eminence, dental interferences, habitual posturing causing anterior positioning of the mandible, acromegaly, macroglossia, contralateral condylar resorption and congenital facial asymmetry unrelated to the temporomandibular joint.

Treatment Plan

Once a detailed diagnosis of CH has been obtained, a treatment plan must be established. Treatment is primarily surgical and often accompanied by orthodontics to correct occlusion. There is some controversy as to the ideal treatment option and time to treat. Treatment plans must consider the degree of asymmetry, resulting malocclusion, patient's motivation, psychological condition and condylar growth activity. Treatments to correct these problems can be approached jointly or separately. Usually, the selected strategy is dependent on growth activity and the patient's age. As always, the patient's demands and expectations are other important considerations [2]. Condylar surgery is a biologically-driven approach that aims to arrest progression by removing the affected tissue. "Condylectomy / low condylectomy" is used to describe the removal of the entire condyle down to the mandibular notch and is reserved for CH Type 2 [1, 16]. "Condylar reduction / high condylectomy" involves the removal of an average of 3-5 mm (1.5 to 7mm range) of condylar tissue to arrest further growth and is primarily used in the management of CH type 1 according to the classification by Wolford *et al.*, [1, 3, 16, 17]. The treatment plan for the different types of condylar hyperplasia is as follows [1, 3, 5-7, 16].

CH type 1 Patients under 18 years of age with SPECT positive scans are treated as follows:

- High condylectomy [with / without disc repositioning] + compensatory orthodontics with
- Surgical cosmetic camouflage after 18 years of age if discrepancy is less than 5mm in vertical / horizontal dimensions
- Orthognathic surgery with / without surgical cosmetic camouflage after 18 years of age if discrepancy is more than 5mm in vertical / horizontal dimensions

CH type 1 Patients above 18 years of age with SPECT positive scans are treated as follows:

- High condylectomy [with / without disc repositioning] + compensatory orthodontics with
- Surgical cosmetic camouflage if discrepancy is less than 5mm in vertical / horizontal dimensions
- Orthognathic surgery with / without surgical cosmetic camouflage if discrepancy is more than 5mm in vertical / horizontal dimensions

CH type 1 Patients above 18 years of age with SPECT negative scans are treated as follows:

- Compensatory orthodontics with
- Surgical cosmetic camouflage if discrepancy is less than 5mm in vertical / horizontal dimensions
- Orthognathic surgery with / without surgical cosmetic camouflage if discrepancy is more than 5mm in vertical / horizontal dimensions

CH type 2 Patients of any age with SPECT positive scans are treated as follows:

- Low condylectomy [with / without disc repositioning] + compensatory orthodontics with
- Surgical cosmetic camouflage after 18 years of age if discrepancy is less than 5mm in vertical / horizontal dimensions
- Orthognathic surgery with / without surgical cosmetic camouflage after 18 years of age if discrepancy is more than 5mm in vertical / horizontal dimensions

CH type 3 and 4 Patients of any age treated as follows:

- Resection of affected ramus-condyle unit with adequate margins
- Reconstruction of the ramus-condyle unit with a vascularized free flap especially if additional chemo-radiation therapy as indicated

TMJ Function after the Condylectomy

Joint function is relevant in every aspect of orofacial stability. From a morphological point of view, a condyle with hyperplasia undergoes significant modifications including the condyle, the articular fossa and coronoid process being integrally affected by the disease and determining the growth of the entire mandible structure. From the functional point of view, the mandibular dynamic is maintained with no significant changes when the high condylectomy is performed [6, 7, 9, 18]. Brusati *et al.*, [19] performed a condylar osteotomy in conjunction with orthognathic surgery in 15 patients; in every case intermaxillary fixation was used for 10 days and joint function was assessed after 4.5 years, showing a mouth opening over 40 mm and lateralities with differences of 0.5 to 1 mm in 14 of the 15 patients. There were no significant differences between the pre- and post-operative stages in either the objective or subjective evaluations, indicating excellent function in 53.3% and good function in 40% of the subjects. Saridin [20] observed

that patients undergoing a condylectomy for condylar hyperplasia with an average follow-up of 4.6 years presented no differences in disc displacement or myofascial pain compared to patients without condylar hyperplasia; however, the patients who underwent surgery had higher rates of TMJ osteoarthritis, which could be linked either to the nature of the disease or to the surgical procedure. Nevertheless, this clinical condition did not affect the daily activities of the patients studied. Saridin also reported that there were no depression-type changes in the patients after the condylectomy, which means the surgery carries no risk of causing depression in the postoperative stage [20]. In the same direction, Lippold [5] followed 6 patients with condylectomy for 2 years without functional changes or complications in daily life. Similarly, Olate *et al.*, [6] followed up 14 patients post-operatively and proved that mouth opening (over 35 mm) and lateral excursions (average 9 mm for the both right and left side) were normal and without statistical differences between the right or left side. In a study by Wolford *et al.*, [18], CH type 2 patients who were treated with low condylectomy were followed up post-operatively to assess TMJ function. At longest follow-up, there was a non-significant decrease (2.3 mm) in maximum incisal opening, but excursive movements decreased significantly an average of 2.5 mm (right) and 2.2 mm (left). There was a statistically significant improvement in pain, jaw function, diet and disability. A stable Class I skeletal and occlusal relation was maintained in 34 of the 37 patients (92%). Two patients developed relatively minor post-surgery malocclusions that were managed with orthodontics. Thus, it can be concluded that the condylectomy (high and low sub-types) is a safe and effective procedure with low morbidity for patients.

CONCLUSION

CH is defined in terms of presentation and consequences. In conclusion, treatment relies on careful assessment, including the patient's concerns, and confirmation of the presence of active growth with SPECT. Active disease should be treated by condylar reduction followed by monitoring. Inactive disease or residual asymmetry can be corrected according to conventional orthognathic principles. The rarity of the condition means that there is little level I evidence, and further research is needed.

Conflict of Interest: None

Informed Consent

The patient's written consent for publication has been obtained and is available on request.

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