UNILATERAL MANDIBULAR CONDYLAR HYPERPLASIA - A RARE ENTITY

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ABSTRACT

True unilateral condylar hyperplasia (CH) of mandible including its variants is an uncommon maxillofacial deformity. It represents unilateral abnormal growth of the mandible including its body and ramus. Discrepancy in the growth of the mandible causing vertical facial asymmetry, creating aesthetic problem resulting in malocclusion as well as psychological issues requiring bimaxillary surgical correction. In this report we discuss the use of imaging in the diagnosis of these pathologies and investigate its variants.

KEY WORDS: Condylar Hyperplasia (CH), Condylar Hyperplasia Variants, Facial Asymmetry.

INTRODUCTION

In 1986, Obwegeser and Makek described condylar hyperplasia (CH) along with its variants as a developmental deformity of unknown etiology affecting the mandible unilaterally. They classified it into 3 categories: hemimandibular hyperplasia, causing asymmetry in the vertical plane; hemimandibular elongation, resulting in asymmetry in the transverse plane; and a combination of the 2 entities categorized as a hybrid variant. 1 The etiology of the disease is still controversial and not well understood. Variety of studies has assumed lowered genetic control over the formation and development of bilateral structures of the face or environmental influences and trauma during development, response to infection or to abnormal loading to be the cause.2,3 Diagnosis of these lesions often poses a challenge to maxillofacial surgeons. 3-D CT scan plays a valuable role in the diagnosis and type of this condition. Here we are presenting a case of unilateral hemimandibular hyperplasia of Hybrid variant.

CASE

The present case is about a 33 years old male patient, referred by maxillofacial surgeon for 3-D CT scan face in our Radiology department. Patient has complaint of gradually developing asymmetry of the right side of the face for past 1 year. His history revealed developing asymmetry of the right side of the face which he had noticed from a self-photograph. Mandibular deviation toward the left side and overgrowth were noticed 1 year before and progressed slowly until it reached present proportion. He also developed mild pain in the right temporomandibular joint (TMJ) region while opening the mouth for past 3 months. There was no history of trauma, any systemic diseases, infection, or surgery of the face and jaws. He has also family history of same complaint with his elder brother and mother, but no clinical or imaging record provided.

Extra oral examination revealed facial asymmetry due to downward displacement of the entire right mandible and increase in the vertical height of the middle and lower facial thirds on the right side. There was a mild deviation of chin to the left side and slight downward tilt in lip line toward the right side (Figure 1). There was mild clicking heard during movement of the right TMJ.

Computed tomography with 3-D reformation was performed which apparently showed discrepancy in the size of both condylar heads with elongation of the right condylar neck as well as elongation and widening of mandibular rami (Figure 2). Clinical and radiographic findings were consistent with a diagnosis of unilateral Condylar hyperplasia (CH) of hybrid variant on right side.

DISCUSSION

Mandibular condylar hyperplasia (CH) is a non-neoplastic rare malformation that changes morphology and size of mandibular condyles.4 It was first described by Robert Adams in 1836 as a condition that causes overdevelopment of the mandible, creating functional and aesthetic problems.
problems. The excessive unilateral growth of the mandible may be associated with asymmetry, occlusal disturbance, and joint dysfunction. In 1986, Obwegeser and Makek classified the asymmetry associated with CH into 3 categories: hemimandibular hyperplasia, causing asymmetry in the vertical plane; hemimandibular elongation, resulting in asymmetry in the transverse plane; and a combination of the 2 entities called as hybrid variant.

The aetiology and pathogenesis of condylar hyperplasia is unknown. A plethora of presumed causes has been proposed in the course of time. Some of them include previous trauma, true neoplasia, hormonal disturbances, partial hemihyperplasty, arthritis, osteoarthromatosis, local circulatory disturbances, and neurotrophic disturbances. Other condition which can cause challenges in diagnosing this condition includes hemifacial hyperplasia and synovial chondromatosis. In the former condition, the associated soft tissues and teeth also will be enlarged and in the latter, there will be precocious swelling with pain and limitation of joint function. Condylar hyperplasia usually occurs after puberty and is completed by 18 to 25 years. Prominent clinical features of condylar hyperplasia include an enlarged mandible, elongated condylar neck, outward bowing and downward growth of the body, and ramus of the mandible on the affected side, causing fullness of the face on that side and flattening of the face on the contralateral side. The prominence of the chin is shifted to the unaffected side. An open bite might exist on the abnormal side. This, depending on one hand, on the rate of increasing enlarge-ment of condyle and, on the other hand, on the downward growth of the maxillary alveoli and teeth, can result in deep overbite or even class III occlusion. The mouth can be opened without restriction. At an early stage occlusal contact is maintained by increased vertical height of the dentoalveolar structures in both the upper and lower jaws. This results in slanting of the occlusal plane towards the affected side. When the downward growth of mandible continues further it exceeds the dento-alveolar growth potential and produces an open bite in the premolar and molar region.

Radiographically, the condyle may appear relatively normal but symmetrically enlarged, or it may be altered in shape. (e.g., concave, spherical, elongated,ivaluated) or irregular (irregular outline): it may appear more radiopaque because of additional bone present. A morphologic variation of elongation of the condylar head and neck may be seen. The ramus and mandibular body on the affected side also may be enlarged. Condylectomy on the affected side is the accepted method of treatment. It gives the best possible result with little post-operative discomfort to the patient.

Unilateral condylar hyperplasia is one of the rare condition and challenging for both orthodontists and oral and maxillofacial surgeons. Its various types and variants can be assessed by imaging including photographs, posteroanterior cephalograms and 3D computed tomography (CT) scans. Early diagnosis by 3D computed tomography (CT) scans is mandatory in order to obtain optimal functional and aesthetic results.

REFERENCE
5 Adams R. The disease in the temporomandibular articulation or joint of the lower jaw. In A Treatise on Rheumatic Gout or Chronic Rheumatic Arthritis of All the Joints, 2nd ed. London: Churchill, 1873:271.

CASE REPORT

Complete heart block also known as third-degree atrioventricular block (AV block) is a condition in which there is no conduction of the impulse produce in the sinoatrial node (SA node) in the atrium to the ventricle. Complete heart block may be congenital or acquired. There are certain conditions which can lead to third-degree heart block, commonest being the coronary ischemia. Initially there may be first degree atrioventricular block (AV block), second degree atrioventricular block (AV block) bundle branch block or bifascicular block ultimately leading to complete heart block. In most cases third-degree AV block presents with acute myocardial infarction. 12, 23 A 45 years male with no known comorbidities and without any risk factors for coronary heart disease coming with a short duration of complaints of dizziness and vertigo was diagnosed as a case of complete heart block without any functional and anatomical impairment in conduction system.

KEY WORDS: Complete Heart Block, Atrioventricular Block (AV block), Sinoatrial Node (SA Node), Coronary Ischemia, Bifascicular Block, Myocardial Infarction, Conduction System.

INTRODUCTION

Complete heart block is a condition in which there is inability in propagation of impulse from Sinoatrial node (SA node) to Atrioventricular node (AV node). It has been identified that many patients with third-degree heart block endure from a bilateral bundle branch block. Pathophysiology and clinical aspects of heart block have shown that myocardial infarction is the common cause of complete heart block. 4 Myocardial ischemia can produce broad range of conduction disturbances involving both the atrioventricular and infra nodal structures. Complete heart block is more common with inferior/posterior infarctions. Complete heart block occurring in association with anterior or myocardial infarction implies extensive myocardial damage and has a worse prognosis.

Complete heart block complicating either inferior or anterior wall myocardial infarction is independently associated with mortality and in hospital complications. We report an uncommon pattern of complete heart block in which myocardial ischemia is absent and any other causes are not present. 7

CASE

A 45 years old male presented to emergency department with complaints of dizziness and vertigo for two hours. There were no risk factors for coronary artery disease. He was alert, alert, oriented with time and person. His pulse rate was 38beats/min, blood pressure was 130/80mmHg, respiratory rate was 15beats/min and oxygen saturation was 97% on room air. There was no raised jugular venous pulsation. He had normal respiratory, cardiac, abdominal and neurologic examination. Serum electrolytes, creatinine, complete blood count, blood culture and sensitivity and chest x-ray were normal. Echocardiography showed no evidence of coronary artery disease. Patient’s escape heart rate stayed 35beats/min whenever rate of temporary pacemaker was decreased to 30beats/min (Figure 3).

All the possible causes for complete heart block were investigated. Coronary angiography was performed which showed insignificant coronary artery disease with preserved systolic function. Patient stayed in the hospital for 3 days.

Anti arrhythmics and anti coagulants were given to the patient. Patient’s escape heart rate stayed 38beats/min whatever rate of temporary pacemaker was decreased to 30beats/min (Figure 3).