Introduction

The mandible is the largest, strongest and lowest bone in the face. It has a horizontally curved body that is convex forwards, and two broad rami, that ascend posteriorly. The body of the mandible supports the mandibular teeth within the alveolar process. The rami bear the coronoid and condylar processes. The coronoid process projects upwards and slightly forwards as a triangular plate of bone. Its posterior border bounds the mandibular notch, and its anterior border continues into that of the ramus. Coronoid process projects upwards and slightly forwards as a triangular plate of bone. Its posterior border bounds the mandibular notch, and its anterior border continues into that of the ramus. Coronoid process is large and project above the level of condyle at birth and gradually with the growth of the neck of the mandible, condyles are at higher level in adulthood. Bilateral elongation of the coronoids of the mandible made of histologically normal bone goes more in favour of it being hyperplasic. This leads to a progressive, painless difficulty in opening the mouth, due to contact of coronoid process with the temporal surface of the zygomatic bone or medial surface of the zygomatic arch [1].

Case Report

Routine preclinical educational teaching of osteology specimens revealed a mandible with unusually long coronoid processes on both sides. Age and sex determination indicated it to be that of male in late adulthood. The length of the coronoid was taken from the line tangent to the deepest part of mandibular notch to the apex which measured 2.4 cm on the right and 2.6 cm on the left side. Rest of the mandible did not show any unusual feature.

Discussion

Coronoid processes project above the level of condyles at the time of birth. With growth of neck of the mandible it comes to lie at lower level in adults. Coronoids projecting much above the level of condyles in a mandible of late adulthood is being reported here. Abnormal elongation of the coronoid process, formed of histologically normal bone without any synovial tissue around it, is suggestive of hyperplasia [1]. Bilateral hyperplasia of the coronoid processes of the mandible is quite infrequent and affects mostly males between the ages of 14 and 16 with male and female ratio of 5:1 [3]. It leads to restricted mouth opening caused by impingement of the process on the medial and anterior surfaces of the zygomatic arch [3].

Craniofacial development is an extraordinarily complex process which requires the integration of multiple specialized tissues, such as the surface ectoderm, neural crest, mesoderm, and pharyngeal endoderm. Development of the lower jaw occurs mainly between the fourth and
eighth weeks of gestation, from the paired mandibular primordia (mandibular prominences). All of these prominences are produced by the proliferation of the neural crest cells that migrate into the arches from the neural crest during the fourth week of gestation. Neural crest cells of the mandibular primordia come mainly from the region of the anterior rhombencephalon (hindbrain) and give rise to the connective tissue components, including cartilage, bone and ligaments in the facial and oral regions [4]. Factors causing disturbance in formation of lower jaw can lead to modification in morphology of mandible. The etiology of elongation is not yet elucidated, but several theories have been postulated, including hyperactivity of the temporal muscle that caused reactive elongation of the coronoid process, dysfunction of the temporo-mandibular joint caused by chronic disc displacement, which would be related with cases of unilateral hyperplasia and is mentioned as one of the causes of Jacob's disease. Other causes may include endocrine stimuli, traumatism and even genetic and family factors [1]. In one of the studies ankylosing spondylosis has also been said to cause mandibular elongation (Bechterew disease) [5].

Evolutionary change in the mandibular morphology can also be one of the factors leading to elongation of coronoid processes and can be explained by studying the developmental units of mandible which include: the number of stem cells in preskeletal condensations, the time of initiation of condensation formation, the fraction of cells that is mitotically active within a condensation, the rate of division of these cells, and their rate of cell death. These units and their derivative structures are discussed in terms of types of tissue differentiation (chondrogenesis, osteogenesis, primary/secondary osteogenesis, intramembranous/ endochondral ossification) and growth properties of major morphological regions of the mandible. Variations in these five units provide the developmental basis for ontogenetic and phylogenetic modification of mandibular morphology [6].

The resulting excess growth of the coronoids results in impingement on the zygomatic processes leading to mandibular hypomobility. The diagnosis can be made via a good radiography. The treatment is surgery by intraoral approach with removal of coronoid process on both sides. Early post-operative rehabilitation has to be done with physiotherapeutic techniques.

Conclusion

Coronoid process hyperplasia as one the causes of mandibular hypo mobility is largely under diagnosed as it is a very rare entity, but a thorough clinical and radiological examination can help to rationalize the line of management and the ultimate clinical outcome.
References


