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ORIGINAL RESEARCH

Asymmetry of glenoid fossa as differential diagnosis for hemimandibular elongation

Asimetría de cavidad glenoidea como diagnóstico diferencial de la elongación hemimandibular

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ABSTRACT

Facial asymmetry is one of the main findings during clinical practice with a prevalence between 21-85%; this causes both functional and aesthetic problems, and is manifested by an inconsistency in size, shape, or position of craniofacial structures on both sides of the mid-sagittal plane. Its etiology is attributed to hereditary and/or environmental factors that may be expressed during the fetal, childhood and/or pubertal stage, including unilateral condylar hyperactivity, functional disharmony of the masticatory muscles, dominance of one cerebral hemisphere, plagiocephaly, unilateral craniosynostosis, among others. The time of evolution prior to its detection contributes to the level of expression of the asymmetry. By means of a literature review, the proposal of a differential diagram and the presentation of a case report which includes facial analysis, cephalometric analysis, 3D tomographic reconstruction and findings of nuclear medicine, differential diagrams are suggested as well as a new classification of facial asymmetry. The differential diagnosis between asymmetry of the glenoid fossa and hemimandibular elongation is established, which requires a careful correlation of clinical findings and imaging tests, since both have similar clinical characteristics, but differ in their therapeutic approach.

RESUMEN

La asimetría facial es uno de los principales hallazgos durante la práctica clínica con una prevalencia entre el 21-85%; ésta causa problemas tanto funcionales como estéticos, y se manifiesta por la inconsistencia en tamaño, forma o disposición de las estructuras craneofaciales en ambos lados del plano medio sagital. Su etiología se atribuye a factores hereditarios y/o ambientales que se pueden expresar durante el periodo fetal, infantil y/o puberal, incluyendo la hiperactividad condilar unilateral, desarmonía funcional de los músculos de la masticación, dominancia de algún hemisferio cerebral, plagiocefalia, craneosinostosis unilateral, entre otros. Donde el tiempo de evolución previo a su detección contribuye con el nivel de expresión de la asimetría. Por medio de la revisión de literatura, la propuesta de un diagrama diferencial y la presentación de un caso clínico que incluye análisis facial, análisis cefalométrico, reconstrucción tomográfica 3D y hallazgos de medicina nuclear. Se sugieren diagramas diferenciales y una nueva clasificación de asimetría facial. Estableciendo el diagnóstico diferencial entre asimetría de la fosa glenoidea y elongación hemimandibular, que exige una cuidadosa correlación de los hallazgos clínicos e imagenológicos, ya que ambos presentan características clínicas similares, pero difieren en su enfoque terapéutico.

Key words: Facial asymmetry, glenoid cavity, condylar hyperplasia, hemimandibular elongation.
Palabras clave: Asimetría facial, cavidad glenoidea, hiperplasia condilar, elongación hemimandibular.

INTRODUCTION

Facial asymmetry is one of the main findings during clinical practice with a prevalence between 21-85%;[1] this causes both functional and aesthetic problems[2] and manifests itself by an inconsistency in size, shape or position of craniofacial structures on both sides of the midsagittal plane.[3]

Its etiology is attributed to hereditary and/or environmental factors that may be expressed during the fetal, infantile period, and/or pubertal stage and may include unilateral condylar hyperactivity,[3] functional disharmony of the mastication muscles, dominance of one cerebral hemisphere,[4] plagiocephaly, unilateral craniosynostosis and other disorders associated with chromosomal genetic and multifactorial anomalies such as 13q deletion, Williams syndrome, among others that have oral manifestations.[5] Time of evolution prior to its detection contributes to the level of expression of the asymmetry.[3]

In some cases asymmetries may be mild and hardly perceptible, hence they may not require any type of surgical treatment and the facial and

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skeletal imbalances may be masked through dental compensation, soft tissue compensation or a change head posture.[1]

Severt and Proffit[6] found in a group of patients with facial asymmetry that only 5% of these involved the upper facial third; 36%, the middle third and 75%, the lower third with lateral deviation of the chin.

Alterations in the upper facial third involve the development of the skull, which is believed to be apparently symmetrical; but the presence of an anatomical difference between the right and left sides may be an indicator of some acquired genetic or congenital pathological condition, so a boundary between what is considered to be a non-perceptible asymmetry and a pathological one must be established.[7] Craniofacial architecture develops thanks to the interaction between the different bone structures that compose it, which will be modulated by the role of the organs they harbor.[8,9] A clear example of this is how some asymmetries in the base of the skull develop due to its relationship with neural structures such as the brain;[7] Serjsen et al. (1997),[10] found that growth of the base of the skull between 4 and 5 years of age is more intense and decreases with age until growth finishes.

Embryologically neural crest cells (NCC), considered specific migratory cells, whose origin is located in the dorsal part of the neural tube during development, subsequent to their induction, they de-laminate and migrate to different regions of the embryo, where they differentiate into a wide range of cell types, including peripheral neurons, enteric cells, melanocytes and smooth muscle, among others.

In the cranial region,[11] they contribute in large part to the formation of cartilage and bone. Facial NCC cooperate extensively in the development of the skeleton of the frontonasal and membranous bones of the skull, while more posterior cranial NCC fill the pharyngeal arches where they form the jaw, the middle ear, the hyoid bone and cartilage.[11]

Although the initial patterns of segmentation and migration of NCC are fairly preserved among species, the great diversity of craniofacial morphology in vertebrates indicates that cranial subpopulations of NCC are able to generate specific skeletal structures during the complex interaction that occurs between their intrinsic genetic program and extrinsic environmental signals that they may be exposed to during craniofacial morphogenesis.[11]

Because of this, birth defects are associated with craniofacial malformations. It is increasingly evident that these anomalies may be attributed to defects in the generation, proliferation, migration and differentiation of NCC produced by alterations in the regulation of genes that are crucial for shaping the neural cranial crest by altering the signaling pathways that regulate tissue interactions during development.

On the other hand, alterations of the middle third compromise the mandibular fossa or glenoid fossa, considered as a structural component of bone in the connection of the mandible to the skull, forming the most active functional craniofacial complex: the temporomandibular joint.[12] However, information reported in the literature on the importance of the position of the glenoid fossa and its interrelationship with facial structures in the development of some type of malocclusion is very limited.[13]

It has been suggested that the spatial correlation between anatomical structures might determine craniofacial conformation,[14] which proposes that the type of articulation that exists between the temporal, occipital and parietal bones is a reflection of forces generated during chewing that are distributed through the skull. This indicates that the mandible and the temporal bones affect their position and movement on a reciprocal basis behaving as a unit.[13-15]

Changes in the position of the glenoid fossa during growth may influence the development of a malocclusion and a facial asymmetry as an expression of the morphological and functional alteration; likewise, the position of the glenoid cavity may be determined by the role of the mandibular condyle as well as by dental position occlusion as a possible modulator of the continuous remodeling of the morphology of the joint.[16-18]

The most common types of facial asymmetry are those that affect the lower third of the face and the occlusion. They are characterized by changes in the three planes of space with or without lateral deviation of the chin. According to their etiology and time of evolution, they may be considered mild, moderate, or severe.[19]

Among the possible causes there are:

1. Unilateral condylar hyperplasia.
2. Asymmetric mandibular prognathism.
3. Laterognathia (chronic or congenital muscular torticollis).
4. Functional laterognathia.
5. Craniofacial syndromes (hemifacial microsomia, craniosynostosis, facial clefts among others).
6. Facial trauma (fracture).
7. Infections (otitis media, varicella zoster virus).
8. Tumors (chondroblastoma).
9. Condylar hypoplasia.[19]