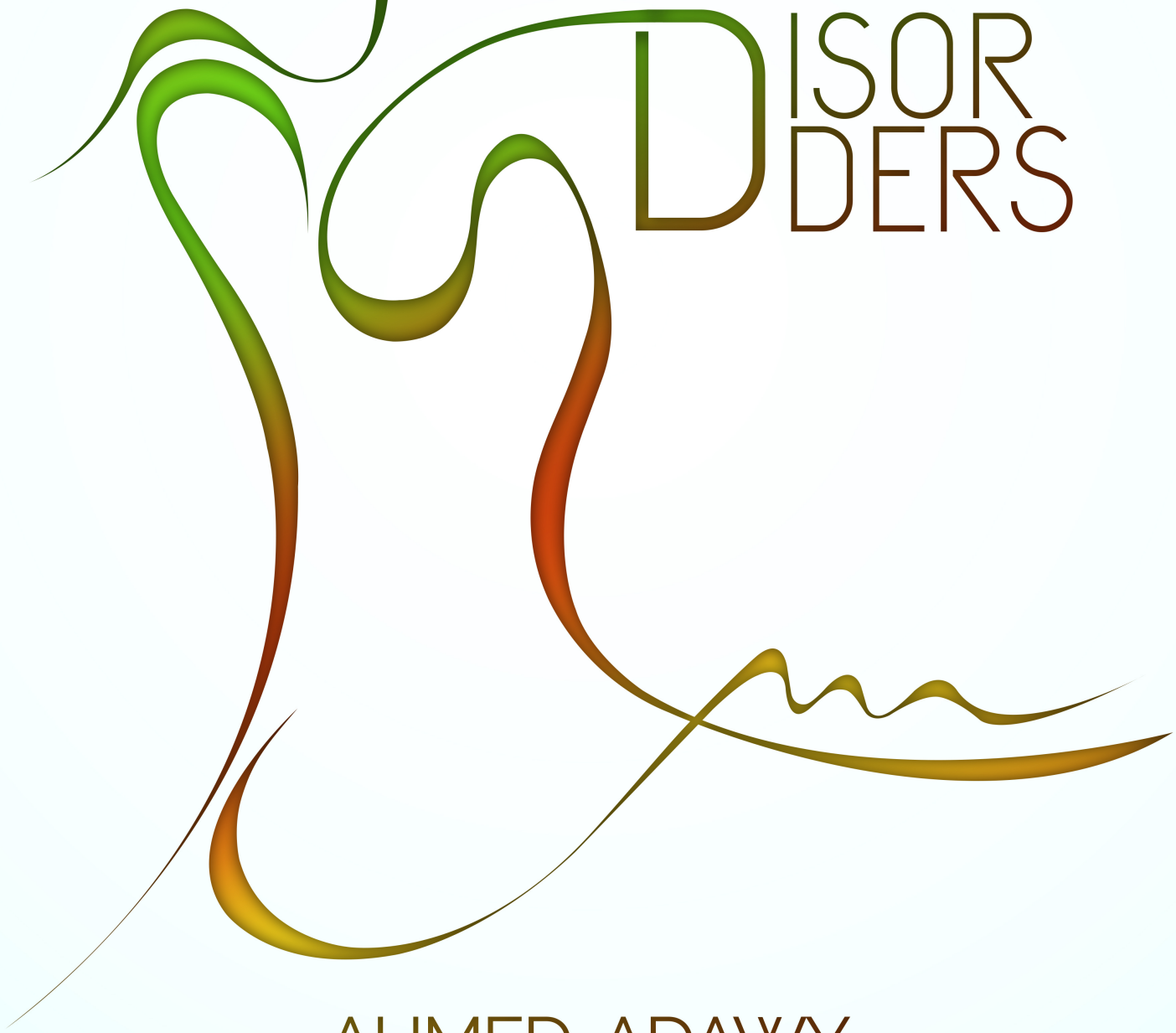
TEMPORO MANDIBULAR DISORDERS



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Ahmed M. Adawy, BDS, HDD, MDS.
Professor Emeritus, Dept. Oral & Maxillofacial Surg.
Former Dean, Faculty of Dental Medicine,
Al-Azhar University.

About the author

Ahmed M Adawy

Professor Emeritus, Dept. Oral and Maxillofacial Surgery, Faculty of Dental Medicine, Al-Azhar University.

Former Consultant Maxillofacial Surgeon, Al-Azhar University Hospitals.

Former Consultant Maxillofacial Surgeon, King Abdul-Aziz Hospital, Makkah, KSA.

Former Chairman, Dept. Oral and Maxillofacial Surgery, Faculty of Dental Medicine, Al-Azhar University.

Former Dean, Faculty of Dental Medicine, Al-Azhar University.

Former Editor in-chief, Al-Azhar Dental Journal.

Former Director, Permanent Scientific Committee for Professor and Assistant Professor Promotion in Dental Science, Al-Azhar University.

Preface

Despite extensive clinical research, temporomandibular disorders (TMD) remain a challenge. Controversies exist in the literature regarding the diagnosis and the management protocol for TMD. These disorders are patho-physiological conditions related to the masticatory muscles, temporomandibular joints or their associated structures and share main symptom expressions and clinical features. Symptoms commonly related to TMD are pain from the face and jaw area at rest or on function, jaw tiredness, TMJ sounds such as clicking or crepitation, jaw movement limitations and locking/catching or luxation of the mandible. Signs regarded as clinical indicators of TMD are tenderness upon palpation of the TMJs and the masticatory muscles, TMJ sounds and irregular paths of jaw movement, impaired jaw movement capacity and pain on jaw movement. Despite decades of research, a comprehensive etiological picture of TMD is still lacking. The variety of included conditions and the complexity of the masticatory system are reflected in the currently accepted multifactorial etiology. The balance between function and dysfunction or adaptation and maladaptation can be affected by a number of factors, such as external macro-trauma to the face, indirect trauma as in whiplash, or repeated micro-trauma, mostly related to oral parafunctions and psychosocial elements. These factors interact dynamically and can, in certain individual circumstances, act as predisposing or initiating elements, leading to the disturbed equilibrium and dysfunction of the masticatory system. Epidemiological studies have revealed that TMD symptoms and signs are very common in the population. It has been estimated that symptoms are reported by almost every third individual and 44–67% of the population present with clinical signs. Symptoms have more commonly been reported by younger and

middle-aged individuals than by children or elderly persons with higher female predominance. In general, the three most common temporomandibular disorders are myofascial pain, internal derangement and osteoarthrosis. In fact, more than 50% of TMD is myofascial pain. Internal derangement is used to describe a temporomandibular disorder where there is an abnormal position of the articular disc. Osteoarthrosis is a localized degenerative disorder mainly affecting the articular cartilage of the mandibular condyle. Less common disorders are joint hypermobility and ankylosis of the TMJ. Rare disorders are hyper- and hypoplasia of the condyle. Diagnosing TMD requires a focused history and physical examination. Radiographic studies can also be used as supplemental diagnostic tools. According to several investigations, the majority of TMD patients, irrespective of the specific TMD diagnosis, achieve good relief of their symptoms with conservative therapy. The majority of TMD patients can be treated by general dental practitioners, but some patients who do not respond to this form of management need to be referred to and treated at specialized TMD clinics, where a multidisciplinary approach and treatment modalities are sometimes necessary.

The main aim of the present book is to provide the readers with an easily accessible text to face the complex and controversial issue of TMJ disorders with greater competence. I sincerely hope that this book will help to better understand TMJ disorders, diagnosis and management, and providing much needed clarity in an area filled with a great deal of misinformation and confusion. The text has been divided into 8 chapters. At first a description of TMJ anatomy, as well as the dynamics of the joint was given. Information regarding etiology and classifications were then presented. In the following chapters, the common, less common, and rare disorders were considered in detail.

I have enjoyed the friendship with the staff members of our department who were willing to share so generously. I am grateful for all the updated information they

provide. A special measure of appreciation goes to young colleagues, residents, and students. They have been a resource of tremendous stimulus to me. My grandson Ziad Marei deserves special acknowledgment for designing the cover artwork of this book.

Ahmed M. Adawy

Dedication

In the memory of my beloved wife Ahlam, and to my daughters; Heba and Maha, for their inspiration, patience, and support.

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Chapter 1: Anatomy and biomechanics

Joints are the sites of articulation of two or more bones. Joints can be grouped by their structure into fibrous, cartilaginous, and synovial Joints. In the fibrous and cartilaginous type of joints, space between adjacent bones is filled with solid connective tissue either fibrous tissue or cartilage. In fibrous joints (synarthrosis), which are known as immovable joints, connective tissue fills the gaps between the bones and tightly fixes the bones with no joint cavity. One example of immovable joints is the skull sutures (Fig. 1). In cartilaginous joints (amphiarthrosis), which are known as partially movable joints, hyaline cartilaginous tissue is found between the articular surfaces. In the vertebrae, a compressible fibrocartilaginous pad connects two bones (Fig. 2). In synovial joints **(**called diarthrosis**),** articular capsule surrounds articular surfaces and articular cavities, holding the articular surfaces together (Fig. 3). This capsule structurally and functionally comprises two layers, which are an outer fibrous layer and inner synovial layer (synovial membrane). Fibrous layer is made of fibrous connective tissue and protects the joint from extraneous effects. Synovial layer lines the inner surface of the fibrous layer and is attached to it via loose connective tissue. The primary role of this membrane is to secrete synovial fluid. Synovial fluid is a clear, pale - yellow, viscous solution secreted by the synovial tissues and consists mainly of an ultrafiltrate of plasma enriched with a proteoglycan - containing hyaluronic acid synthesised by synovial cells. The high viscosity of this fluid is a result of the presence of sodium hyaluronate which provides lubrication.

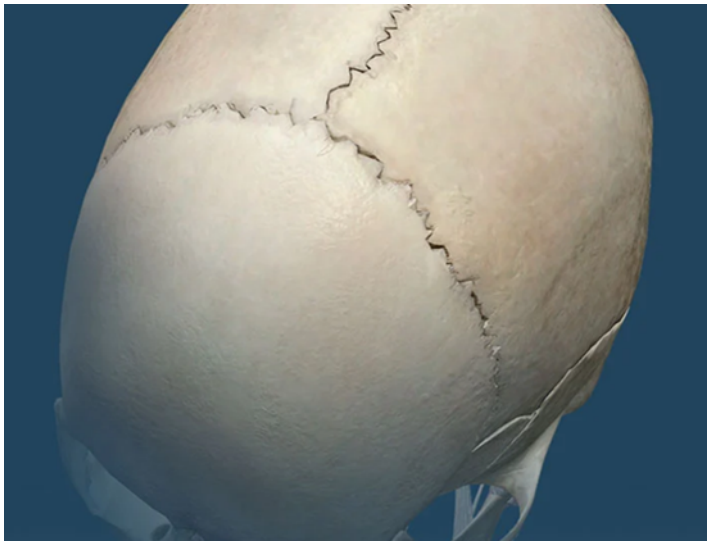

Fig. 1: Fibrous joint (no movement).

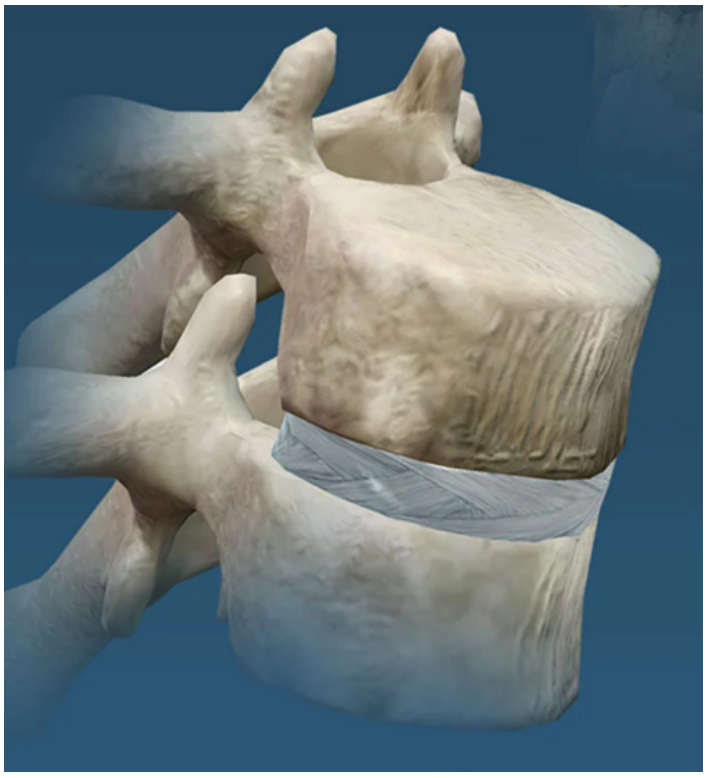

Fig. 2: Cartilaginous joint (some movement).

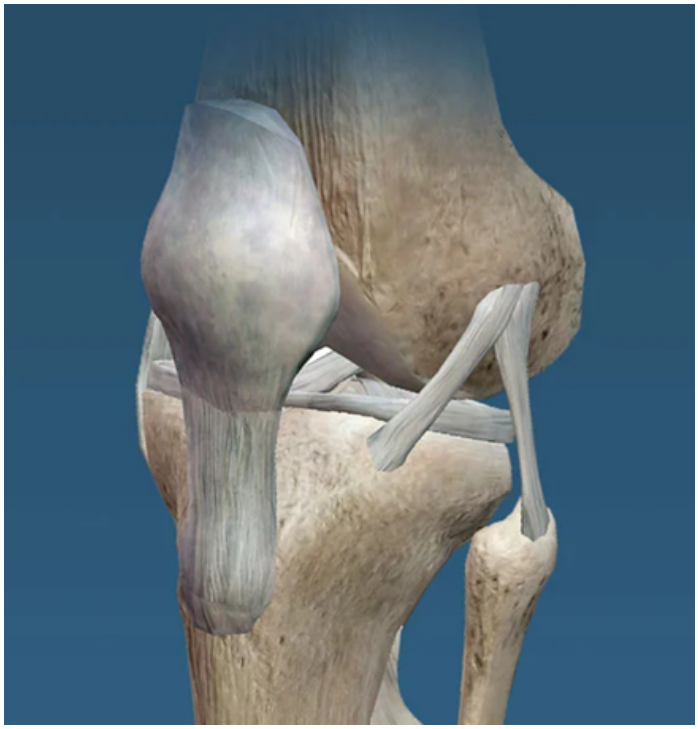

Fig.3: Synovial joints (full movement).

The temporomandibular joint (TMJ) is formed by the mandibular condyle fitting into the mandibular fossa of the temporal bone (Fig. 4). It is located anterior to the external acoustic meatus and posterosuperior to the masseter muscle. TMJ has some common features with the other joints in the human body. TMJ includes an articular disc that could be seen in some other joints in the human body. TMJ has bony articular surfaces, articular capsule, synovial membrane, and ligaments just like other joints. But TMJ has differences that make it unique among other joints in the body. TMJ of each side is connected with a single mandible that necessitates the harmonic and coordinated function of each TMJ. In general, all of the joints in the human body have a hyaline articular cartilage except sternoclavicular joint, acromioclavicular joint, and TMJ where the articular surfaces are covered by fibrocartilage. The TMJ is the only joint in the human body to have a rigid endpoint of closure with the dental arches on each jaw contacting each other that is called

occlusion. Moreover, it is the only joints able to move in six directions; anterior-posterior (forward and backward), lateral (side-to-side), vertical (up and down). In addition, the condylar cartilage is considered a growth center that significantly contributes to the overall growth of the mandible. The most important functions of the TMJ are mastication and speech. The TMJ is a ginglymoarthrodial joint, a term that is derived from ginglymus, meaning a hinge joint, allowing motion only backward and forward in one plane, and arthrodia, meaning a joint of which permits a gliding motion of the surfaces (1).

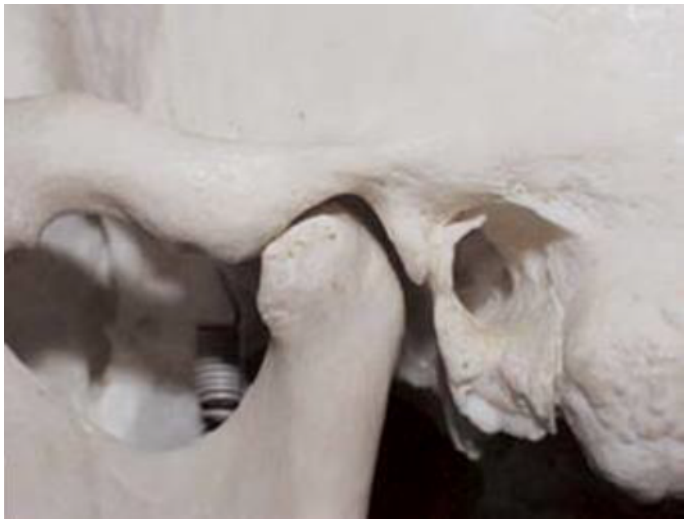

Fig. 4: Temporomandibular joint, lateral view.

A thorough knowledge of the anatomy and functional biomechanics of the TMJ is essential for the correct analysis of several of its disorders.

Anatomy of the temporomandibular joint:

The TMJ is composed of the following structures: 1. The mandibular condyle, 2. Articular (glenoid) fossa and eminence 3. The articular disc, 4. TMJ capsule, 5. Temporomandibular joint ligaments.

Mandibular condyle

The adult mandibular condyle is roughly ovoid in shape. It is approximately measuring about 15-20 mm mediolaterally and 8-10 mm anteroposteriorly (2). From anterior view, the condyle has a bipolar structure, comprising medial and lateral poles that give attachment to the disc. The medial pole is generally more prominent than the lateral (Fig. 5). From above, a line drawn through the centers of the poles of the condyle will usually extend medially and posteriorly toward the anterior border of the foramen magnum forming an angle ranging between 145° and 160°, (Fig.6), (3). The size and shape of the condyle varies greatly among different age groups and individuals and there are often differences between the right and left sides in an individual. The superior surface could be; flat, convex, round or angled. Morphologic changes may occur on the basis of simple developmental variability as well as remodeling of the condyle to accommodate developmental variations, malocclusion, trauma, and other developmental abnormalities.

Histologically, there are four distinct layers or zones described in the articular surface of the condyle and mandibular fossa. These layers are the articular zone, proliferative zone, cartilaginous zone and calcified zone (4).

1. Fibrous connective tissue layer forms the outer functional surface of the condyle head. It is suggested that it is less susceptible to the effect of ageing and breakdown over time. In addition, despite a poor blood supply, it has a better ability to repair, good adaptation to sliding movement and the ability to act as a shock absorber compared with hyaline cartilage (5).

2. Proliferative undifferentiated mesenchymal layer is mainly cellular and is the area in which undifferentiated germinative mesenchyme cells are found. This layer is responsible for the proliferation of the articular cartilage. Also, the proliferative layer is capable of regenerative activity and differentiation throughout life.

3. Cartilaginous layer contains collagen fibers arranged in a criss – cross pattern of bundles. This offers considerable resistance against compressive and lateral forces but becomes thinner with age.

4.The calcified zone is the deepest zone and is made up of chondrocytes, chondroblasts and osteoblasts. This is an active site for remodeling activity as bone growth proceeds.

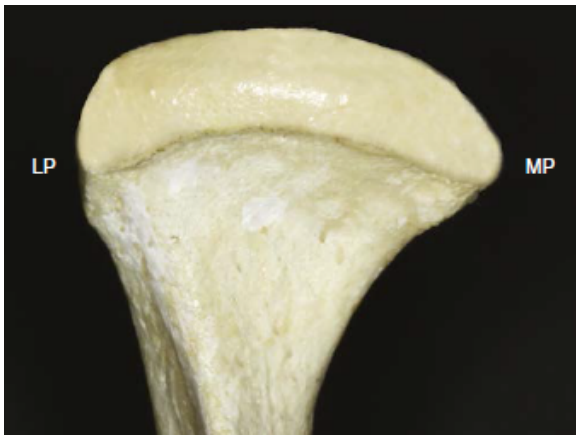

Fig. 5: The Condyle (Anterior View). The medial pole (MP) is more Prominent than the lateral pole (LP).

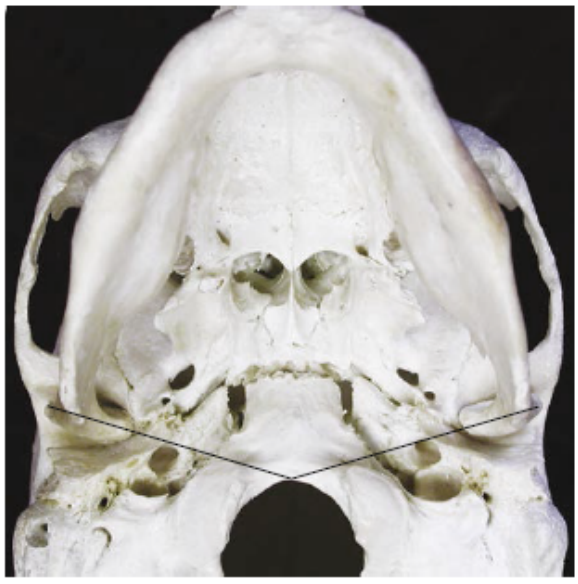

Fig. 6: Condylar axes intersect at the foramen magnum with an angle of 145° and 160°.

Articular (glenoid) fossa and eminence

The articular surface of the temporal bone consists, posteriorly, of the concavity of the glenoid fossa and, anteriorly, of the convexity of the articular eminence. It extends from the anterior margin of the squamotympanic fissure to the margin of the articular eminence. The glenoid fossa is located on the inferior aspect of the squamous part of temporal bone just anterior to the tympanic plate. The roof of the fossa is very thin, indicating that this part is not a load - bearing area. Anteriorly, however, the articular eminence is thicker and this area, together with the disc, may be the area that bears most of the load during function. The articular eminence has a convex structure and is found on the anterior of the mandibular fossa just at the lower edge of the zygomatic arch (6). While the degree of convexity of this prominence varies, it is important for the perpendicularity of the forward movement of the mandible.

Articular disc

The articular disc acts as a cushion to absorb stress in the TMJ and divides the TMJ into two separate and functionally different cavities. The disc is a biconcave oval dense fibrous structure, for the most part devoid of any blood vessels or nerve fibers and receives its nutrition from the synovial fluid (7). The extreme periphery of the disc, however, is slightly innervated. In the sagittal plane, it can be divided into three regions according to thickness (Fig.7). The anterior portion of the disc is approximately 2 mm thick, while the posterior portion is 3 mm thick. The intermediate portion of the disc is the thinnest part nearly 1 mm. In the normal joint, the articular surface of the condyle is located on the intermediate zone of the disc, bordered by the thicker anterior and posterior regions. From an anterior view, the disc is generally thicker medially than laterally. The articular disc is attached posteriorly to a region of loose connective tissue that is highly vascularized and

innervated. This tissue is known as the retrodiscal tissue or posterior attachment**.** Superiorly, it is bordered by a lamina of connective tissue that contains many elastic fibers, the superior retrodiscal lamina. The superior retrodiscal lamina attaches the articular disc posteriorly to the tympanic plate. At the lower border of the retrodiscal tissues is the inferior retrodiscal lamina, which attaches the inferior border of the posterior edge of the disc to the posterior margin of the articular surface of the condyle. The inferior retrodiscal lamina is composed chiefly of collagenous fibers, not elastic fibers like the superior retrodiscal lamina. The remaining body of the retrodiscal tissue is attached posteriorly to a large venous plexus, which fills with blood as the condyle moves forward (8). Anteriorly, the articular disc is attached superiorly and inferiorly to the capsular ligament, which surrounds most of the joint. The superior attachment is to the anterior margin of the articular surface of the temporal bone. The inferior attachment is to the anterior margin of the articular surface of the condyle. Between the attachments of the capsular ligament the disc is also attached by tendinous fibers to the superior lateral pterygoid muscle. The remaining large part of the muscle called as the inferior head of the lateral pterygoid muscle attaches to the pterygoid fovea of the mandible. The articular disc is attached to the capsular ligament not only anteriorly and posteriorly but also medially and laterally. This divides the joint into two distinct cavities. The upper or superior cavity is bordered by the mandibular fossa and the superior surface of the disc. The lower or inferior cavity is bordered by the mandibular condyle and the inferior surface of the disc. The internal surfaces of the cavities are surrounded by specialized endothelial cells that form a synovial lining.

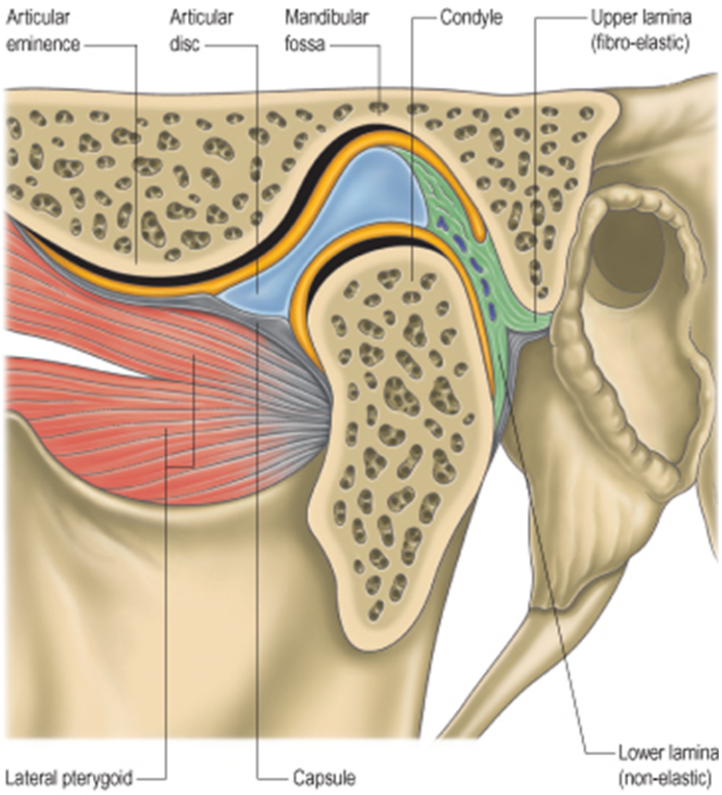

Fig. (7): Sagittal view of the TMJ; the articular disc interposes the condyle and the glenoid cavity.

TMJ capsule

The joint capsule is a fibroelastic, highly vascular and highly innervated dense connective tissue that completely surrounding the joint. It extends from the circumference of the cranial articular surface to the neck of the mandible. The lateral aspect of the capsule attaches to the zygomatic tubercle, the lateral rim of the glenoid fossa and the post-glenoid tubercle. Medially, the capsule attaches to the medial rim of the glenoid fossa. Posteriorly, the capsule attaches to the petrotympanic fissure and fuses with the superior stratum of the posterior bilaminar zone. Anteriorly, the capsule has an orifice through which the lateral pterygoid tendon passes. The outline of attachment on the mandibular neck lies a short distance below the edge of the

articular surface in front and a considerable distance below the articular margin behind. Laterally, it is attached to the lateral condylar pole but medially it dips below the medial pole. On the lateral part of the joint, the capsule is a well-defined structure that functionally limits the forward translation of the condyle. This capsule is reinforced more laterally by an external TMJ ligament, which also limits the distraction and the posterior movement of the condyle. Medially and laterally, the capsule blends with the condylodiscal ligaments. As the articular cavity is divided by the disc into two separate cavities, there are two synovial membranes: synovial membrane of the superior joint cavity and that of the inferior joint cavity. The former lines the fibrous membrane superior to the disc, and the latter lines the fibrous membrane inferior to the disc (9). The superior joint cavity contains an average of 1.2 ml, and the lower joint cavity contains 0.5–0.9 ml synovial fluid. The role of the synovial fluid is to nourish the avascular tissues in the joint, lubricate the articular surfaces, and clear the tissue debris caused by normal wear and tear of the articulating surfaces.

Temporomandibular joint ligaments

The structures that connect the bones are called ligaments, and they are divided into two groups, extracapsular ligaments and intracapsular ligaments depending on their relationship with the capsule. Ligaments of the TMJ consist of collagen connective tissue, which is not elastic. They are structures that maintain the position of the mandible and play a passive role in the restriction of the movements of the joint (2). TMJ has three ligaments other than the joint capsule. These are the temporomandibular ligament, stylomandibular ligament, and sphenomandibular ligament. In another way of classification, the ligaments of the TMJ are divided into functional ligaments and accessory ligaments. Functional ligaments comprise the temporomandibular ligament, capsular ligament, and collateral ligaments, while the

stylomandibular ligament and sphenomandibular ligament are counted as accessory ligaments.

Temporomandibular ligament (lateral ligament)

The temporomandibular ligament is attached to zygomatic arch and articular eminence superiorly. It narrows as it extends downward and posteriorly and adheres to the outer surface and posterior edge of the mandibular neck. It reinforces the capsule from the lateral side (Fig. 8). Temporomandibular ligament comprises two parts, outer oblique and inner horizontal. The outer oblique part begins from the external surface of the articular eminence and zygomatic process of the temporal bone and extends posteriorly and downward to the mandibular neck via the inferior part of the condyle. The outer oblique part restricts the movement of the opening of the jaw, which is performed in the rotational direction, by inhibiting the excessive forward movement of the condyle (2). The inner horizontal part is narrower and shorter, begins from the outer surface of articular eminence and zygomatic process of the temporal bone and extends horizontally toward the back, and is attached laterally to the condyle. It prevents the posterior movement of the condyle and articular disc, preventing the retrodiscal tissue from trauma (9).

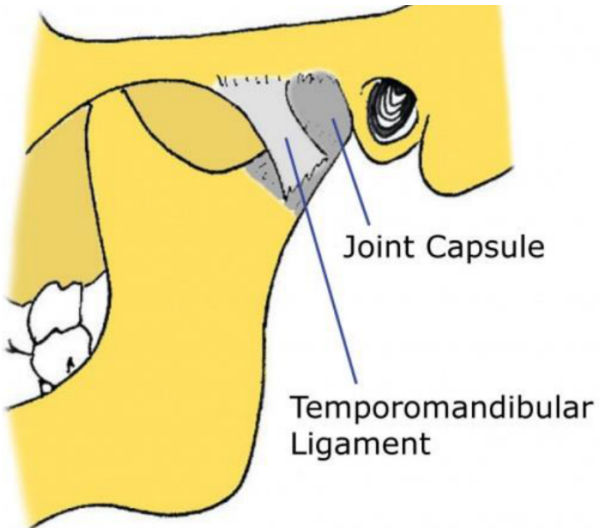

Fig. (8): Joint capsule and temporomandibular (lateral) ligament.

Stylomandibular ligament

Stylomandibular ligament is a segment of deep cervical fascia, thickened into a band shape. It extends from styloid process to the inferior of the posterior edge of the mandibular ramus and mandibular angle (Fig. 9). Stylomandibular ligament passes between the masseter muscle and medial pterygoid muscle throughout its course. Moreover, it is found between the parotid gland and submandibular gland (10). Stylomandibular ligament restricts the excessive protrusive movement of the mandible and supports the temporomandibular ligament (2).

Sphenomandibular Ligament

Sphenomandibular ligament is attached superiorly to the spine of the sphenoid bone that is lateral to the spinous foramen, expands while extending downward, and adheres to the lingula of the mandible (Fig.9). Its outer surface neighbors the lateral pterygoid muscle, and inner surface neighbors the medial pterygoid muscle (9,10). It is crossed by chorda tympani at the region close to its superior tip. Between the sphenomandibular ligament and neck of the mandible, maxillary artery, maxillary vein, and inferior alveolar neurovascular bundle pass. The main function of this ligament is to prevent the effect of an extra pressure during the opening and closing of the jaw on the inferior alveolar neurovascular bundle that passes through the mandibular canal. Moreover, this ligament also provides primary passive support to the mandible, and the extent of jaw opening (6).

Other ligaments related to the temporomandibular joint

Other ligaments related to TMJ have been defined in the literature including retinacular ligament, medial and lateral collateral ligaments, and tanaka ligament. Retinacular ligament is approximately 5 cm long and 2 cm wide, is between the parotid gland and mandibular ramus. Actually, it is defined as a thickening of the fascia of the masseter muscle. Its course begins in the superior by adhering to the

anterior and posterior surfaces of the articular eminence, cartilaginous part of the external acoustic meatus, posterior tip of the lateral pterygoid muscle, outer segment of the temporomandibular ligament, mandibular condyle, and retrodiscal tissue at the posterior side and becomes narrower as it descends and ends when it attaches to the fascia of the masseter muscle at the level of mandibular angle. Retinacular ligament conducts the strain generated during the contraction of the masseter muscle to the retrodiscal tissue and TMJ (11). Collateral intracapsular ligaments are the ligaments between lateral and medial borders of the disc and poles of the mandibular condyle. These are short collagen fibers that restrict rotational movements in lower joint space, and these ligaments ensure that the disc and condyle move together in protraction and retraction (12). Another intracapsular ligament of TMJ called the Tanaka ligament (13). This ligament attaches the articular disk to the wall of the mandibular fossa in the temporal bone.

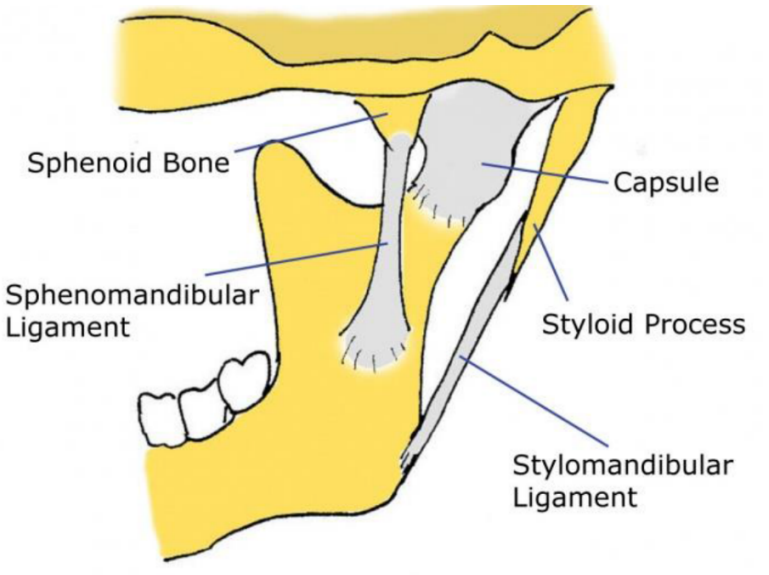

Fig. (9): The stylomandibular ligament and sphenomandibular ligament (Medial view)

Muscles associated with jaw function

Muscles of the masticatory system (Fig.10), are the masseter muscle, temporal muscle, pterygoid muscles (medial pterygoid muscle and lateral pterygoid muscle), suprahyoid muscles (digastric muscle, mylohyoid muscle, stylohyoid muscle), and some other head and neck muscles (14).

The masseter muscle is a rectangle-shaped muscle that mainly originates from the zygomatic arch and adheres to the lateral part of the inferior edge of the mandibula. It has three layers, superficial, middle, and deep, all acting to elevate (close) the mandible. It also contributes to the act of protrusion. superficial and deep fibers. While the superficial fibers extend downward and slightly to the posterior, deep fibers extend vertically. When the masseter muscle contracts, it lifts the mandibula upward and is a strong muscle that provides the required strength for efficient chewing. Its superficial branch also helps the protrusion of the mandible (15).

Temporal muscle is a fan-shaped, broad muscle. It originates from the temporal fossa and passes through the zygomatic arch and adheres to the coronoid process. Depending on the direction of the fibers, it is classified into three parts: anterior, medial, and posterior parts. When the temporal muscle contracts as a whole, the fibers act strongly on elevation as well as retrusion. Latera deviation of the jaw is provided by the ipsilateral posterior fibers and the contralateral middle fibers (2).

Medial pterygoid muscle is a thick quadrilateral muscle that originates from the inner surface of the lateral pterygoid plate and adheres to the pterygoid tuberosity at the medial surface of the mandibular angle. When it contracts, it protrudes the mandible and strongly assists in the elevation of mandible. Unilateral contraction of this muscle acts to laterally deviate the mandible to the opposite side (2).

Lateral pterygoid muscle has two heads as superior and inferior head with two different functions. The superior head is smaller than the inferior head. It starts from

the infratemporal surface of the sphenoid bone and adheres to the joint capsule, disc, and condyle. When this muscle contracts, it pulls the disc anteromedially. It is inactive during the downward movement of the mandible, which is the opening of the jaw. It is active when it contracts together with the elevator muscles, particularly when power is exerted as the teeth are closing, so it helps joint stability during closure (2). Inferior head starts from the outer surface of the lateral pterygoid plate and adheres to the condyle neck (6). When contracted bilaterally, condyles are pulled downward, toward articular eminence, and the mandible becomes protruded. Unilateral contraction causes the condyle to move mediotrusively and the mandible to move laterally in the opposite direction. When this muscle contracts together with the depressor muscles, the mandible moves downward, and condyles move forward and downward on the articular eminence; thus, lateral pterygoid muscle is highlighted as the only muscle that opens the jaw.

Suprahyoid muscles

Suprahyoid muscles are those above or superior to the hyoid bones of the neck. These are many muscles including the digastric, mylohyoid, geniohyoid and stylohyoid muscles. The two muscles in this group that mainly act in jaw function are the digastric and mylohyoid muscles. The digastric muscle, is divided by its central tendon into an anterior belly that extends forward to the mandible and a posterior belly that extends backward to the mastoid process of the temporal bone. They are connected by an intermediate tendon that runs through a fibrous sling on the hyoid bone. The mylohyoid muscle is a thin sheet of muscle arising on the inner aspect of the mandible from the whole length of the mylohyoid line. The two halves of this muscle meet in a median raphe, which inserts into the body of the hyoid bone. This muscle forms the floor of the mouth, separates the submandibular and sublingual regions, and is unattached posteriorly. The suprahyoid muscles elevate the hyoid bone when the mandible is fixed in place, and depress the mandible

together with the tongue and the floor of the mouth, when the infrahyoid muscles stabilize the hyoid bone. In addition, the posterior belly of the digastric is one of the retruding muscles of the mandible (16).

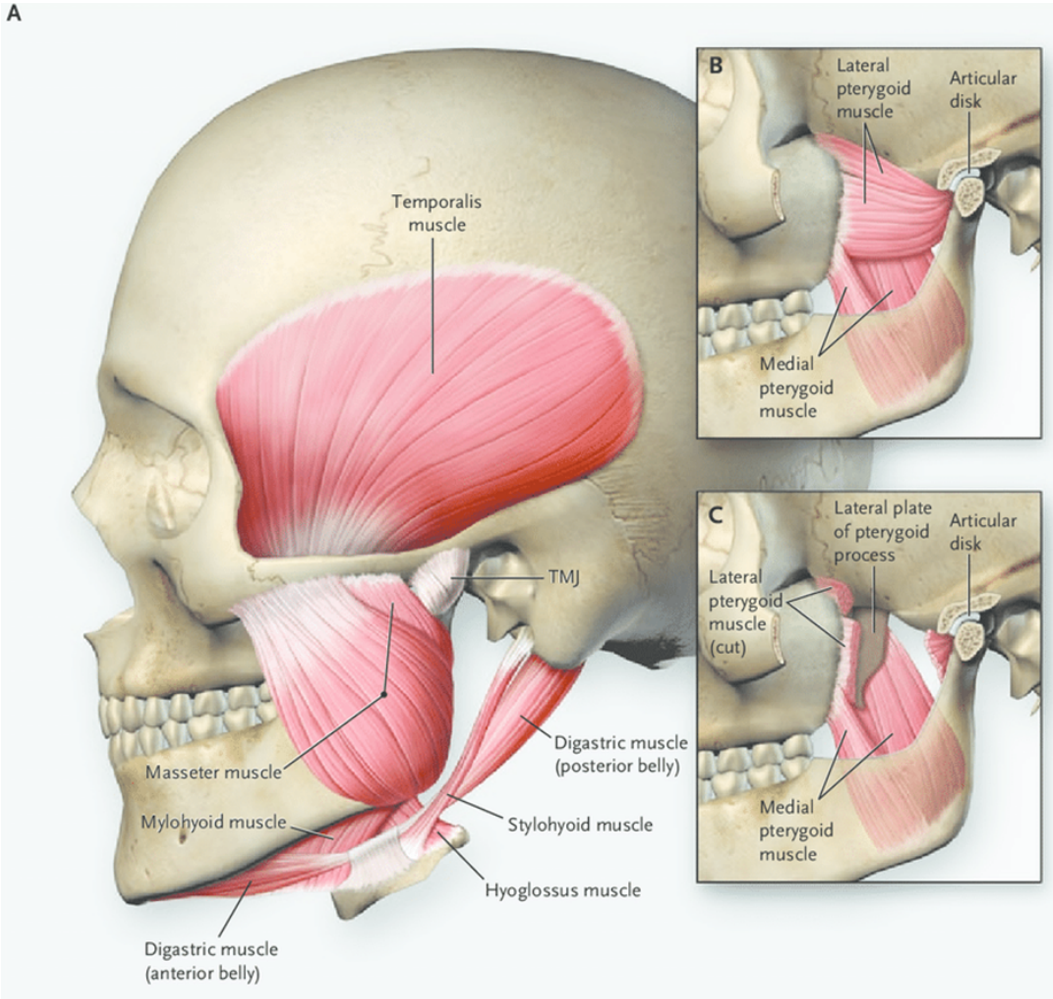

Fig. (10): Muscles associated with jaw function; temporalis, masseter, medial and lateral pterygoid, mylohyoid, anterior and posterior digastric, hyoglossus, and stylohyoid.

Blood supply and innervation of the temporomandibular joint

The blood supply to the TMJ is only responsible for superficial structures of the joint, and there is no blood supply inside the capsule. The avascular structures inside the capsule are supplied by the synovial fluid. The arteries that supply the TMJ mainly derive from the superficial temporal artery and maxillary artery, which are the terminal branches of the external carotid artery (Fig.11). The course of the

maxillary artery and its proximal branches is found just medial to the joint capsule. This part of the maxillary artery is named as the mandibular part of the artery and has four branches here: deep auricular artery, anterior tympanic artery, middle meningeal artery, and inferior alveolar artery. Its most important branch here is the middle meningeal artery which is just located in the medial part of the joint. The main artery of the joint is the articular branch of the superficial temporal artery. In addition to this artery, TMJ is supplied by the ascending pharyngeal branch of the external carotid artery; deep auricular, anterior tympanic, and middle temporal branches of the maxillary artery; and transverse facial branch of the superficial temporal artery. The veins of the TMJ are named the same as the arteries. A special venous structure called retroauricular plexus assists the drainage of the veins. Lymphatic vessels of the TMJ drain to superficial and deep parotid nodes (17).

The TMJ is mainly innervated by the auriculotemporal nerve and masseteric nerve, which are the branches of mandibular branch of the trigeminal nerve. Branches of facial nerve innervate the temporomandibular ligament. Articular branches of the otic ganglion found just below the oval foramen are responsible for the parasympathetic innervation of the synovial membrane and its synovial fluid secretion (6).

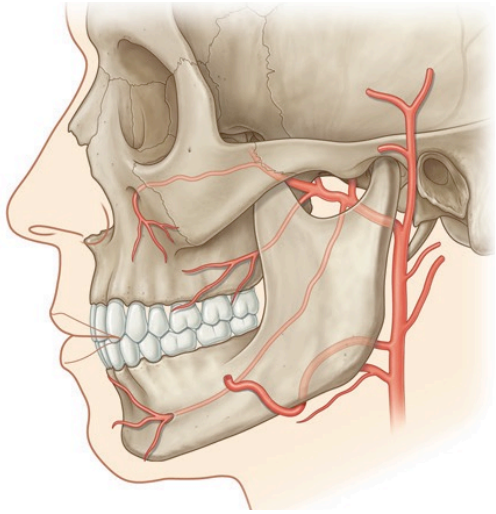

Fig. (11): Maxillary artery located just medial to the joint capsule.

Biomechanics

Anatomical descriptions of human joints should always be coupled with a detailed functional evaluation. The masticatory system is the functional and anatomic relationship among the teeth, jaws, TMJs, and muscle of mastication (18). There is a physiological harmony between these structures. In addition, the vessels and nervous structures that nourish and constrict these structures and the soft tissues control and coordinate all these structural components (19). The TMJ has a unique dynamic behavior, allowing movement of the lower jaw with six degrees of freedom. The biomechanics of the mandible include depression (opening), elevation (closing), protrusion, retrusion, and lateral excursion. Mandibular depression is measured as the space between the maxillary and mandibular incisors; normal range of motion can vary from 35 mm to 50 mm, depending on the size and shape of the mouth and teeth. Lateral excursion and protrusion motions are approximately 10 mm. A 4:1 ratio of depression to lateral excursion and protrusion is considered ideal.

Mandibular depression and elevation

Mandibular depression begins with the first 25 mm of opening that occurs primarily as a rotational motion of the condyle in the inferior joint space. This occurs initially between the articular disc and condyle, in the lower joint cavity. The disc and its attachment to the condyle are called the condyle-disc complex and is the joint system responsible for rotational movement in the TMJ. The rotational movement of the mandible can occur in all three planes, which are transverse, frontal and sagittal planes. In each plane, it occurs around an axis (2). In the masticatory system, rotation occurs when the mouth opens and closes around a fixed axis with no positional change of condyles. The rotation or hinge movement of the mandible around a transverse axis passing through the centers of the mandibular condyles, fig. (12), is probably the only pure rotational movement. When the jaw opens fully, both the

condyle and the disc translate anteriorly and inferiorly together as a unit against the slope of the articular eminence. Full opening of the mouth fig. (13), maximally stretches and pulls the disc anteriorly (8). The forward movement of the disc is permitted by the loose fibroelastic tissue of the superior retrodiscal lamina, which is stretchable to 7-10 mm. At this point, the forward translation is limited by tension in the superior retrodiscal lamina. The inferior head lateral pterygoid muscle provides a protracting force on the condyles and discs; the geniohyoid and digastric muscles produce a depressing and retracting force on the chin; and the mylohyoid muscle pulls downward on the body of the mandible to combine to produce the rotatory and translatory movements of the jaw that occur with mandibular depression. Elevation or mouth closing of the mouth is the reversal of the movements of opening. The mandible rotates upward and retrudes. Tension in the superior retrodiscal lamina starts to retract the disc. The condylar head translates posteriorly along with the disc, and then the condyle rotates anteriorly (20). At the end of the range of closing the mouth, the disc rotates slightly anterior as a result of the contraction of the superior head of the lateral pterygoid (21). The mouth closing is terminated when contact is made between the teeth.

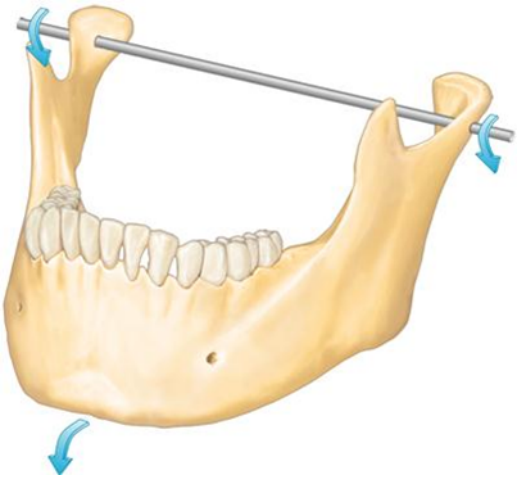

Fig. (12): Rotation movement of the mandible around a transverse horizontal axis passing through the centers of both mandibular condyles.

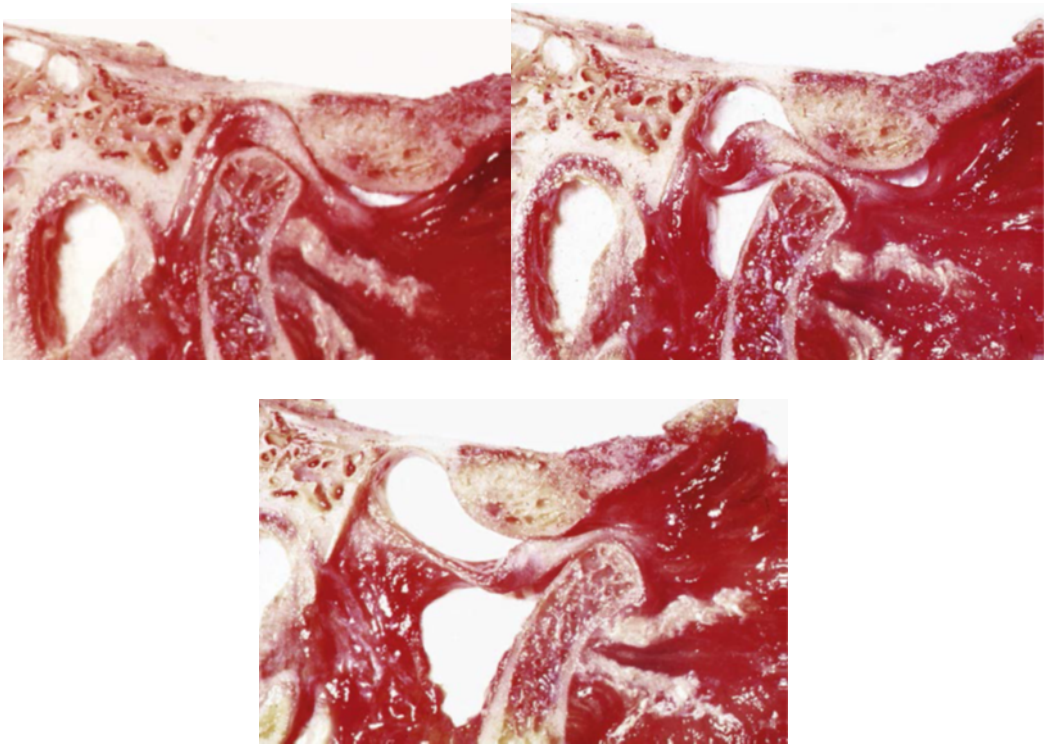

Fig. (13): Normal movement of the condyle and disc during mouth opening. Rotational movement predominately occurs in the lower joint space while translation predominately occurs in the superior joint space.

Protrusion and retrusion

The mandibular condyles and the articular disc translate together anteriorly during protrusion or posteriorly during retrusion (22). Protrusion of the mandible is created with symmetrical anterior translation of both condyle/disc complexes on the articular eminence, and the motion occurs at the superior joint space (1). Protrusion is created by contraction of the inferior head of the lateral pterygoid and holding action of the masseter and medial pterygoid muscles. The lateral pterygoid muscles pull the condyle and disc forward and down along the articular eminence while the elevator and depressor muscles maintain the mandibular position. Retrusion is the return to rest position from the protrusion position and is created by the contraction of the middle and posterior fibers of both temporalis muscles while the depressors and

elevators maintain a slight opening of the mouth. The posterior portion of the joint capsule limits the protrusion, while the retrusion is limited by anterior ligaments and muscle fibers of the superior head of the lateral pterygoid muscle, and the mass of the retrodiscal pad of the disc (21).

Lateral excursion

During lateral movements, the condyle on the working side rotates laterally around a vertical axis, fig. (13), and moves laterally usually in a slightly upward direction (23). This movement, consisting mainly of a rotation around a vertical axis and a predominantly lateral translation, was originally referred to as Bennett movement, and has been renamed mandibular lateral translation (24). Simultaneously, the balancing condyle slides medially and inferiorly on the distal aspect of the articular eminence.

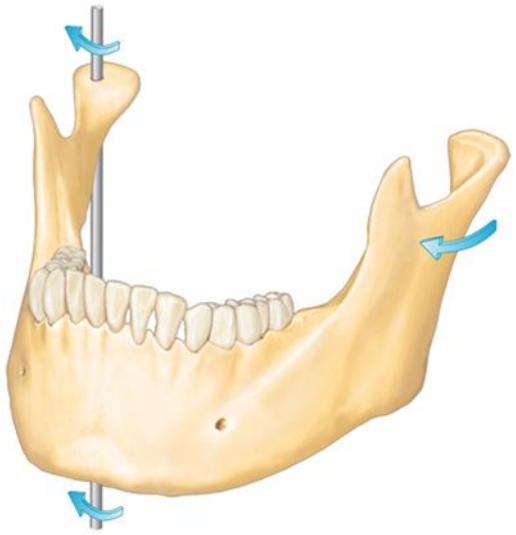

Fig. (13): Condylar rotation around the vertical axis.

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Chapter 2: Etiology and classifications

Etiology and classifications

Temporomandibular disorder (TMD) is a collective term that includes several clinical problems affecting the muscles of mastication, temporomandibular joints and other related structures (1). TMD problems are widespread, affecting 90% of the general population at one life stage or another (2), especially 20–40-year-old women. However, only 10% of affected individuals seek treatment for pain, and, less frequently, for articular noises. A recent systematic review concluded that the prevalence of TMD was 31% for adults and 11% for children and adolescence (3). Pain is the presenting symptom in the majority of patients. Initially it is provoked by loading or movement of the joint and relieved by rest, but as the disease progresses, the pain may be more prolonged and experienced at rest and may become severe enough to wake the patient at night. As the disease progresses and the ligaments and the disc become involved, mechanical changes may occur. Patients may develop painful or painless functional impairment due to restricted movements. Common physical signs include clicking, restriction of range of movement of the joint, joint crepitus, periarticular tenderness, deformity, and muscle weakness.

In the early 1930s, Costen (4) described a group of symptoms that linked with malfunction of the ear and temporomandibular joint (Costen syndrome: loss of hearing, dizziness, tinnitus, restriction of the jaw, preauricular pain, burning sensation of the tongue and globus hystericus). In more recent years, other etiologic factors, besides structural one, have been recognized. For example, trauma at both the macro and micro level (5), has been observed in the history of certain patients. The importance of psychologic factors in the onset, progression, treatment, and persistence of various TMD is well recognized as foundational knowledge in this field (6). More recently, the combination of a biologic and psychologic perspective

in etiologic discussions about TMD has been given the name "biopsychosocial" (7). Another approach to describing the complexity of etiologies is to invoke the word "multifactorial" (8), thereby indicating an awareness that many extrinsic factors in the environment, as well as various intrinsic factors within the patient, might be involved in the development of symptomatic TMD. These factors are classified as predisposing (structural, metabolic and/or psychological conditions), initiating (trauma or repetitive adverse loading of the masticatory system) and aggravating (parafunctional, hormonal or psychosocial factors).

Occlusion and Temporomandibular disorders.

The role of occlusion in the development of TMD is controversial. Some authors believe that occlusion is the primary factor in the onset of TMD symptoms, whereas others feel that occlusion has no role and that etiological factors are based more on behavioral, psychological and neurological problems. Moreover, there is no evidence to support an increased incidence of TMD in patients with malocclusion. In an attempt to assess the relationship between TMD and occlusion, Lipp (9) found that experimental, epidemiological and clinical studies failed to support a significant role of occlusion in the development of a TMD. The same author suggested that remodeling capacity of the articulatory system would allow accommodation to most occlusal function and dysfunction. Furthermore, controlled studies of occlusal factors and TMD show either no relationship, or at best only a weak correlation, between specific variables and TMD. In a series of studies by Seligman and Pullinger (10), an overjet of >4mm, unilateral posterior crossbite and retruded contact position, and intercuspal position slides of greater than 1.75mm were associated with TMDs, these associations were found to be statistically weak. They also found that no single occlusal factor was able to differentiate TMD patients from healthy subjects. The same authors concluded that many occlusal parameters, traditionally believed to be influential, contribute only in a minor manner to the development of

TMDs and that the occlusion cannot be considered to be the most important factor in the etiology of a TMD. In a recent systematic review, Manfredini et al. (11), reviewed the literature on the association between features of dental occlusion and TMDs. They concluded that, although there were a few papers that may have suggested a possible association, the existing evidence supports the absence of a disease specific association, and there is no ground to hypothesis a major role for dental occlusion in the pathophysiology of TMDs.

Trauma and TMDs

Trauma has been a controversial issue although it has been considered to be a major factor for the temporomandibular disorder. Trauma can be divided into two general types: macrotrauma and microtrauma. Macrotrauma is considered any sudden force that can result in structural alterations, such as a direct blow to the face. Whiplash injury, fig. (1), to the head or neck is often considered a significant risk factor for development of TMDs, and has been proposed to produce internal derangements of the TMJ (12). However, TMD pain after whiplash injury is rare, suggesting that whiplash injury alone is not a major risk factor for the development of TMD problems.

Fig. (1): Whiplash injury caused by car accident, the head jerks backward and then forward.

Endotracheal intubation has also been linked with TMJ dysfunction in a limited number of published case reports and systematic studies (13).

Microtrauma refers to any small force that is repeatedly applied to the structures over a long period of time. Para-functional behaviors such as bruxism (fig. 2) or clenching can produce microtrauma and are considered initiating factors for the development of TMD. The association between bruxism and TMD signs supports the theory that repetitive adverse loading of the masticatory system may cause functional disturbances (14). There are also researches based on the hypothesis that parafunctional habits are the source of internal derangements of TMDs and myofacial pain disorders (15). These findings showed that macro- and microtrauma can be considered as the major etiologic factors of TMD.

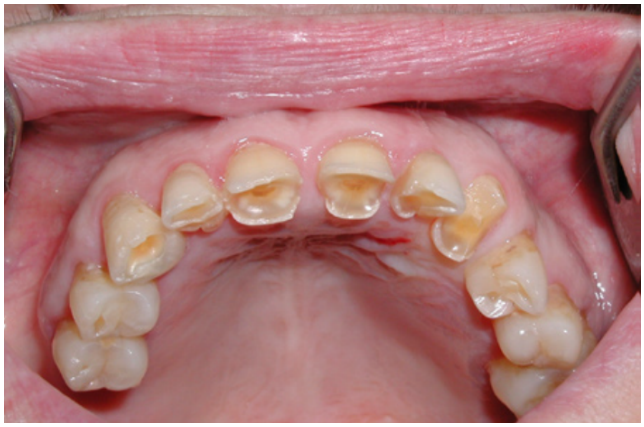

Fig. (2): Dental attrition induced by bruxism.

Psychopathology and TMDs

Several studies have demonstrated a link between TMDs and certain psychopathologies. Depression, anxiety, sleep disorders, somatization and irritability are some emotional features present in chronic TMD patients (16). These biopsychosocial factors may be involved in predisposition as well as progression of TMD. In a study by Vasudeva et al (17), significant differences in the anxiety levels

were found between the TMD and control subjects. Elevated anxiety states in an individual may increase the masticatory muscle activity which in turn may cause TMD. Moreover, a prospective cohort study (18), with more than 6,000 participants showed a twofold increase in TMD in persons with depression and a 1.8-fold increase in myofascial pain in persons with anxiety.

Condylar position and TMDs

Many authors suggest that the position of the condyles is critical to the equilibrium of the masticatory system and that occlusal factors may affect this position (19). Conversely, condylar displacement could also affect activity of the masticatory muscles, with the result that muscle spasm may be a source of pain. It is also suggested that muscle spasm might displace the condyle, and occlusal interferences may therefore be a result rather than a cause. More recently, however, condyle position has not been considered as the main cause of TMDs alone, but it may be effective together with other possible etiological factors synergistically (20).

Classifications

Over the years many classification schemes have been offered, each with various advantages and disadvantages. Some classifications have used etiologic factors to group disorders, whereas others have used common signs and symptoms. In 1956 Schwartz (21), introduced the term "temporomandibular joint pain-dysfunction syndrome" to distinguish the masticatory muscle disorders from organic disturbances of the joint proper. Another proposed classification system was offered by Stegenga et al (22). They divided their classification into inflammatory and noninflammatory articular disorders with another category that included non-articular disorders. The subcategories of osteoarthrosis and internal derangements were further divided according to staging over time. In 1986, the International Association for the Study of Pain (23), published a classification of pain conditions.

Of the 32 categories of pain disorders, category III was designated as "craniofacial pain of musculoskeletal origin." Within this category were two subcategories: temporomandibular pain and dysfunction syndrome and osteoarthritis of the temporomandibular joint. In 1990, the American Academy of Head, Neck, Facial Pain and TMJ Orthopedics (24), offered a classification with five TMD categories and two non-TMD categories. There were 19 subcategories under the main category of myofascial disorders, some of which were separated by the specific muscle or tendon involved. Another diagnostic classification of TMD is that introduced by the American Academy of Orofacial Pain (25) which, according to the presenting symptoms and clinical examination, classifies TMD into muscular disorders and articular disorders. The articular group includes disc disorders, inflammatory disorders, osteoarthritis, condylar dislocation, ankylosis, and fractures. The research diagnostic criteria suggested by Dworkin and LeResche (26), offer what appears to be reasonable diagnostic criteria, especially for research purposes. This classification not only provided very specific diagnostic criteria for eight TMD subgroups; it also recognized another level, or "axis," that must be considered when evaluating and managing TMD pain. Axis I of the RDC/TMD assesses the clinical characteristics of TMD, by means of palpation and physical measures of oral and facial tasks. Diagnoses are split into three categories: masticatory muscle disorders; disc displacements; or other degenerative joint conditions, such as arthralgia and osteoarthritis. Axis II provides a reliable, valid assessment of psychosocial factors, including: pain intensity, pain-related disability, depression, and nonspecific physical symptoms. This classification systems have been the most widely employed diagnostic protocol for TMD. Recently, a validated development of the RDC/TMD was published (27). The Diagnostic Criteria for TMD (DC/TMD) provides a comprehensive assessment of the most common TMD disorders, based on the biopsychosocial model of chronic pain. There are 12 most common diagnoses of

TMD described in Axis-I of the DC/TMD, which are divided into painful conditions (myalgia, local myalgia, myofascial pain, myofascial pain with referral, arthralgia, headache attributed to TMD) and non-painful conditions (disc displacement with reduction, disc displacement with reduction with intermittent locking, disc displacement without reduction with limited opening, disc displacement without reduction without limited opening, degenerative joint disease, subluxation). In some instances, however, multiple diagnoses are present at any timepoint in a single patient, and that diagnoses may change as the disease progresses or resolves. For example, a patient with complaints of joint clicking with pain in the TMJ and masseter muscle, and headache during mouth opening may be diagnosed with having local myalgia, arthralgia, disc displacement with reduction, and headache attributed to TMD. The classification of TMD also includes those that are less common, but clinically important diseases (28). Some of these less-common diagnoses include fractures of the TMJ, manifestations of systemic diseases, as well as rare conditions such as neoplasms and developmental disorders. Generally, the three most common temporomandibular disorders are myofascial pain syndrome, internal derangement and osteoarthrosis (29).

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Chapter 3: Myofascial pain syndrome

Myofascial pain syndrome (MPS) is a painful condition of myofascial trigger points in the skeletal muscle (1). Trigger points (TrPs) are defined as tender or hyper-irritable areas in muscles and/or their fascia. The condition is widespread, and some studies have cited myofascial pain conditions as the most common cause of pain, responsible for 85% of back pain and for 54.6% of chronic head and neck pain (2). MPS is primarily a muscle disorder resulting from oral parafunctional, habits such as clenching or bruxism that is sometimes related to psychogenic disorders such as headache, fibromyalgia, chronic back pain and irritable bowel syndrome with stress, anxiety and depression being the key features (3). Patient generally presents with diffuse pain that is cyclic and distributed in multiple sites in the head and neck. particularly the muscles of mastication (4). Pain is frequently worst in the morning and the patient will often report sore teeth from nocturnal clenching. There is often a history of stress and difficulty sleeping. Clinically, most of the patients will present with diffuse muscle tenderness and intermittent decreased range of mandibular movements with wear facets on the teeth. Symptoms may occur after muscle overuse or injury, but there are some patients with no precipitating factors. The onset of pain may be acute or insidious. Physical examination will reveal the presence of TrPs, which can be either active or latent (5). Active TrPs cause consistent pain in the affected muscle, whereas latent TrPs cause pain only when compressed or perturbed. MPS is typically diagnosed via physical examination, and the generally agreed upon diagnostic criteria include the presence of TrPs, pain upon palpation, a referred pain pattern, and a local twitch response (5). Examination of the masticatory musculature is usually accomplished by digital palpation. Areas of tenderness, trigger points and pain referral patterns should be noted which may be mapped out (Fig. 1) to visualize the site and extent of the pain (6).

Many theories of the pathophysiology of MPS have been proposed, such as local tissue damage leading to decreased pH, tissue hypoxia, and release of histamines and bradykinin (7).

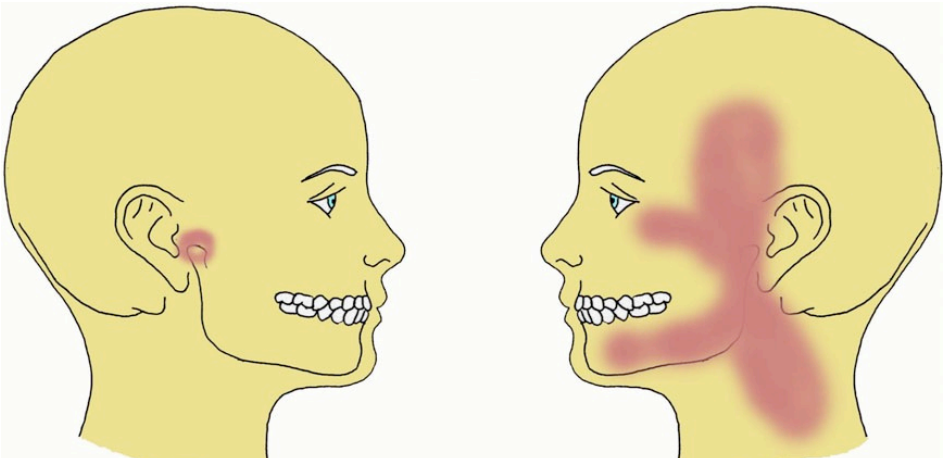

Fig. (1): Facial mapping is useful for showing the site and extent of the pain. Right diagram showing pain well localized to the TMJ and in left diagram more diffuse distribution of pain suggesting myofascial pain.

Treatment and management

The main goals of treatment are to reduce or eliminate pain and to restore normal mandibular function. Patient education and self-care should be formulated first. This includes the following; limitation of mandibular function, habit awareness and modification, a home exercise program and avoiding stress. Voluntary limitation of mandibular function (i.e., avoid excessive chewing and talking) is encouraged to promote rest. Hence, the patient is advised to maintain a soft diet and avoid foods where a considerable amount of chewing is involved. Furthermore, the patient should also be discouraged from wide yawning, chewing gum, and any other activities which would promote excessive mandibular function. Massage of the affected muscles with application of moist heat will help by promoting muscle relaxation (8). Patients should also be advised to identify the source(s) of stress, and try and change their lifestyle accordingly.

Pharmacotherapy

Several studies have examined different treatments for myofascial pain syndrome, including the use of Pharmacotherapy. Pharmacotherapy can be a valuable adjunctive aid to the relief of symptoms when they are prescribed as part of a comprehensive management program. Clinicians dealing with temporomandibular disorders should be well conversant with the different families of drugs which include non-steroidal anti-inflammatory drugs, opiates, anxiolytics, muscle relaxants, tranquillizers and antidepressants. The use of a local anesthetic applied topically to the TrPs is often chosen as this provides a pharmaceutical intervention without being invasive (9). Local anesthetics reduce patients' reported pain and discomfort and increase daily functionality. Commonly used anesthetics include lidocaine, tetracaine, and diclofenac sodium applied either as a cream or as a patch. Local anesthetics can also be injected at the trigger points to provide relief for patients with MPS. Nonsteroidal anti-inflammatory drugs (NSAIDs) are the most commonly used drugs for MPS as they are readily available and have a relatively mild side-effect profile. Their use is appealing because of their analgesic and anti-inflammatory properties. Although the role of NSAIDs in treating MPS is unclear, there is clear evidence that the analgesic properties of NSAIDs relieve pain in acute musculoskeletal disorders (10). Muscle relaxants are a group of drugs with varying pharmacology that act on the central nervous system to disrupt nociceptive pain. Tizanidine is a centrally acting alpha-2-adrenergic agonist, which decreases muscle spasticity. Studies have indicated that tizanidine significantly decreased pain intensity and disability from baseline (11), and suggested that tizanidine should be considered as a first-line agent for the treatment of MPS. There is an increasing role for antidepressants in the treatment of chronic pain. Tricyclic antidepressants (TCAs) are a class of medications that have been indicated for chronic pain, fibromyalgia, and neuropathic pain (12). Although their use is widespread, there are

limited studies specifically for the treatment of MPS. Botulinum type A toxin (BTX-A) is a potent neurotoxin that prevents muscle contraction. BTX-A injections into the TrPs block the release of excitatory acetylcholine at the neuromuscular junction resulting in temporary localized paralysis and potentially reduced patient pain. The literature suggests that BTX-A injection is a promising therapy to alleviate MPS (13).

Occlusal splint therapy

The most common form of treatment provided by dentists for temporomandibular disorders is occlusal splint therapy. This may be otherwise referred to as a bite raising appliance, occlusal appliance or bite guard (fig. 2). The idea is to protect the teeth from abnormally high loads in clenchers and grinders and also to reduce the maximum loads on the TMJ, particularly in patients with nocturnal clenching/grinding. By distracting the occlusion, maximum contraction of the masticatory muscles is also prevented which theoretically reduces muscle pain. Although the use of occlusal splint therapy has been clinically shown to alleviate symptoms of temporomandibular disorders in over 70 per cent of patients, the physiological basis of the treatment response has never been well understood. Although there are many occlusal splints designs available, the most effective splints are those that are custom made, safe, comfortable to wear and do not cause occlusal changes (14).

Fig. (2): Occlusal splint

Physiotherapy

The aim of physiotherapy is to restore normal mandibular function by a number of physical techniques that serve to relieve musculoskeletal pain and promote healing of tissues. Physiotherapy is especially useful in the management of myofascial pain and TMJ closed lock and is essential following TMJ surgery. Acupuncture is a system of medical treatment based on the principle of applying small needles or pressure to specific points in the body. Acupuncture is most commonly used in the treatment of chronic, non-cancer pain in adults. Acupuncture is mostly safe; local side effects can occur, but they typically resolve soon after removal of the acupuncture needle. Trigger point injections may employ dry needling, short- or long-acting anesthetics, or steroids. In a prospective, double-blinded, randomized controlled study (15), dry needling treatment appeared to be effective in relieving pain and in improving the quality of life of patients with myofacial pain.

Manual therapy is a commonly used treatment for MPS as it has been considered as one of the most effective techniques for the inactivation of musculoskeletal trigger points pain (16). There are various sources in the literature, which have specifically described effective modalities, including deep-pressure massage, and superficial heat. Although there are many techniques that have been described in treating MPS with effective results, there have been no controlled studies proving significant long-term effectiveness. Close co-operation with a physiotherapist who is well versed in the management of musculoskeletal disorders of the head and neck is essential, especially those who have a particular interest in TMJ disorders.

Ultrasound is a technique that has been proposed to treat myofascial pain by converting electrical energy to sound waves in order to provide heat energy to muscles. Multiple studies of ultrasound on MPS have demonstrated a statically significant improvement in pain and reduced the sensitivity of the trigger points (17). On the other hand, another study concluded no statistically significant reduction in

pain or analgesic usage between ultrasound plus massage, sham ultrasound plus massage, and exercise versus control (18).

Transcutaneous electric nerve stimulation (TENS) is a treatment modality that utilizes an electrical current to stimulate nerve fibers in order to provide pain relief. TENS is one of the passive therapies available for treating MPS. The therapy can be self-administered, is without significant side effects, and is cheap. The therapy reduces pain significantly better compared to placebo (19). The only limitation of using TENS alone is that its effect is not long-term.

Laser therapy; A form of light therapy known as low-level laser therapy has shown effectiveness in managing a variety of musculoskeletal pain disorders. The therapy has an analgesic and anti-inflammatory effect despite its unclear mechanism. A review article of eight studies concluded that laser therapy was superior in alleviating pain compared to placebo laser on MPS patients (20). Although laser therapy has shown some therapeutic promise, as a whole the body of evidence is mixed regarding the efficacy of this treatment strategy.

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Chapter 4: Disc displacement disorders

In normal joint the disc is bound to the medial and lateral condyle pole by discal ligaments. Therefore, translational movement in the healthy joint is possible only between the condyle-disc complex and the articular eminence. The only physiological movement between condyle and disc is rotation. The amount of rotation of the disc in normal circumstances depends on the shape of the disc, the degree of interarticular pressure and the synergic function of the upper head of the lateral pterygoid muscle and the upper layer of the bilaminar zone (1). In opening the mouth and moving the condyle-disk complex forward, the upper layer of the bilaminar zone is tensed and retracted the disc in the posterior direction. The interarticular pressure increasing during the mouth opening maintained the condyle below the thin intermediate zone of the disc and prevented the thicker anterior disc border from being pulled between the condyle and eminence. This force only works with the translatory movement of the condyle. There is no tension in the retrodiscal tissue during the closing of mouth. During closing the mouth, the upper head of the lateral pterygoid muscle is activated and pulls the disc forward, while the condyle-disc complex slides back and up. The moderate rotation of the disc, which occurs in the normal joint, allows the disc and condyle to remain in intimate contact during all movements and all mandibular positions. The normal relationship between condyle and disc in mandibular movements is also maintained due to the specific form of disc. The biconcave form of the disc and its thickening borders itself ensures the stability of the disc condition while the interarticular pressure increases during the opening of the mouth also helps to center the disc on the condyle. Medial and lateral discal ligaments support the maintenance of disc because they avoid any translatory movement between condyle and disc (1). If, however, the form of the disc is changed and the discal ligament is elongated, the translatory movement between the condyle

and the disc becomes possible. The amount of this movement depends on the change in the form of disc and the degree of elongation of discal ligament. Discal ligaments are not elastic and after elongation they retain that length. In such cases, the articular surface of the condyle no longer rests below the intermediate zone of the disc during closing mouths, but under the thinned posterior band of the disc or, even in the retrodiscal tissue. This condition is referred to as a functional derangement of the disc or disc displacement disorder.

Disc displacement (DD) is defined as the disc improperly positioned in relation to the condylar-fossa complex (2). A synonymous term to DD is internal derangement. Although the disc can be displaced in any direction; anterior, posterior, lateral, or medial (3), posterior and pure sideways displacements seem to be rare, whereas anterior displacement appears to be the most common. According to the classification of Research Diagnostic Criteria for TMD the three main types of internal TMJ derangement are: 1) disc displacement with reduction, 2) disc displacement without reduction with or 3) disc displacement without reduction and without limited mouth opening (4). In disc displacement with reduction (Fig. 1), when the mouth is closed, disc does not take a normal position between condyle and articular eminence, but is displaced anteromedially to the condylar head. During opening of the mouth, the disc reduces to approximately normal position on the condyle. It is commonly an asymptomatic condition. A characteristic sign of the disc displacement with reduction is clicking sound during the opening of the mouth, or when opening and closing the mouth. In some cases, this may be accompanied by increased muscular activity, pain, and limitation of mandibular movements. Disc displacement without reduction (Fig. 2) is a clinical condition in which the disc is dislocated, anteromedially to the condyle and does not return to normal position with condylar movement. Disc displacement without reduction is usually presented as a closed lock. Epidemiological studies confirm a high prevalence of disc disorders. It

has been reported that disc displacement accounts for 41.1%. of temporomandibular disorders (5).

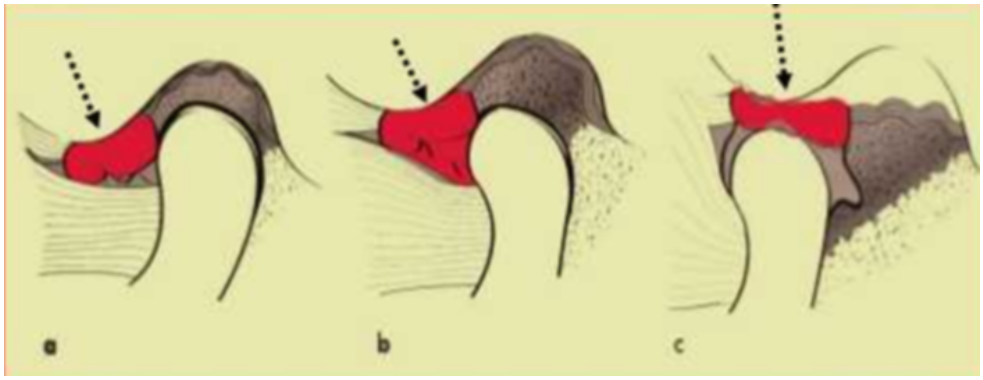

Fig. (1): Disc displacement with reduction; The disc recaptures its normal relationship with the condyle during mouth opening; a: closed position, b: partially open, c: fully open.

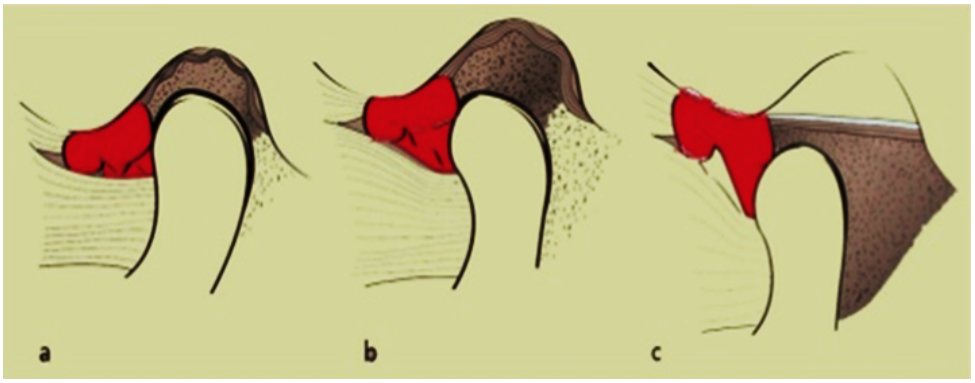

Fig. (2): Disc displacement without reduction; The disc failed to reduce or recapture the normal relationship with the condyle upon opening.

Etiology

The etiology of TMD remains controversial. Various etiological factors for disc displacement have been mentioned in the literature.

Trauma

Acute (macrotrauma) or chronic (microtrauma) injuries directed against the TMJ can be associated with most cases of disc displacement. Macrotrauma or direct

extrinsic trauma to any element of the masticatory system can initiate loss of structural integrity thus reducing the adaptive capacity and altering the function. Mandibular fractures or face and neck injuries may result in disc displacement (6). Prolonged opening of mouth, third molar extractions have been associated with TMDs (7). Moreover, any force that overloads the TMJ complex can cause damage to the joint structures or disrupt the normal functional relationship between condyle, disc and articular eminence, resulting in dysfunction or pain, or both.

Disc deformation

Once mechanical stresses exceed adaptive capability the TMJ progresses to dysfunctional remodeling (8). A disc with altered shape is more likely to be displaced.

Inclination of the articular eminence

The disc should rotate forward over the condyle to maintain the correct relationship during movements, and therefore it is suggested that a steep articular eminence may be a predisposing factor for disc displacement. Hall et al. (9), demonstrated association between the presence of a steep articular eminence and displaced disc. Further studies reported controversial results and thus association of steepness of articular eminence and presence of disc displacement has not been established. In a recent study, it is found that the disc position in disc displacement with/without reduction is not influenced by morphology of articular eminence, however, the articular eminence inclination and condylar excursion angle demonstrated influence on disc reduction (10). Even though the anatomical and structural factors influencing the onset of a disc displacement have not been clarified as yet, it is found that joints characterized by a condyle centered in a well-shaped fossa of normal dimensions are the most resistant to disc displacement.

The role of the lateral pterygoid muscle

Previous studies demonstrated a relationship between lateral pterygoid muscle (LPM) and TMJ internal derangement. It was believed that the disc could be positioned more anteriorly by traction of the LPM. Moreover, some authors suggested that spasm of the superior head of LPM is the cause of anterior disc displacement (11). Further, a hypo/hyperactivity of this muscle or a poor coordination of its two bellies are thought to be possible causes of functional imbalance of the TMJ. This theory, however, was disproved based on the assumption that only a small amount of muscle fibers of the LPM are able to transpose the joint capsule and attach directly into the disc (12). In a study with magnetic resonance imaging (MRI), evaluating the correlation between the LPM muscle attachment type and internal derangement of the TMJ, no statistically significant correlation was found (13).

Joint hypermobility

TMJ hypermobility, defined as condylar translation beyond the eminence at maximum mouth opening, has also been positively correlated with disc displacement with reduction. The theory that some types of TMD, and disc displacement in particular, could have a high prevalence in subjects with articular hypermobility was based on some early studies pointing out an association between TMD and the generalized joint hypermobility syndrome (14).

Disc displacement with reduction

Among the intra-articular disorders of the TMJ, disc displacement with reduction (DDWR) corresponds to 41% of TMD clinical diagnoses (15). The prevalence of DDWR is higher in female patients (16). This fact may derive from the influence of some female-specific characteristics such as greater joint laxity, and greater intra-articular pressure. There is an association between increased age and DDWR.

Clinically, DDWR is related to TMJ noise. The movement of the disc onto and off may result in a clicking, snapping, and/or popping sound known as opening and closing click (17). The disc-condyle complex derangement comes from the alteration of the relation of the disc on the mandibular condyle. Because the opening movement relocates the disc in the joint, the condition is termed DDWR.

It has been suggested that DDWR would be the first stage of disc displacement, possibly evolving to disc displacement without reduction (18). Nevertheless, such observation is not consistent to all conditions and DDWR can remain stable for years depending on adaptive physiological processes that may occur (19). The main disc adaptive physiological process is fibrosis of the retrodiscal bilaminar zone. It is possible that this retrodiscal fibrosis explains why most DDWR are painless.

Diagnosis of DDWR

Patients with DD often have musculoskeletal symptoms in the orofacial region and a thorough examination has to be conducted in order to identify patients with strict muscular diagnosis or when other differential diagnoses are suspected. Firstly, demographic data must be surveyed with respect to onset, duration, severity of symptoms, medical history, including past and present medication, heredity, social situation, general joint problems for example. The clinical evaluation must include palpation of masticatory muscles on both sides, in particular the masseter (Fig.3) and the temporal (Fig.4) muscles. Palpation of the TMJ from the lateral aspect at rest and during opening and closing movements, as well as protrusion and lateral excursions allows assessment of pain, clicking, crepitus, restricted or asymmetric mobility. Visual inspection of the mandibular movements during both opening and closing is of importance. Maximal interincisal opening (MIO), maximal protrusion and lateral excursion to both sides have to be measured with a millimeter-graded ruler (Fig.5). Examination of teeth, periodontal status and occlusion are also mandatory and should be performed with probes, palpation, percussion, and sensibility tests if

necessary. Signs of parafunctions, such as excessive attrition, tongue and buccal impressions, should be documented.

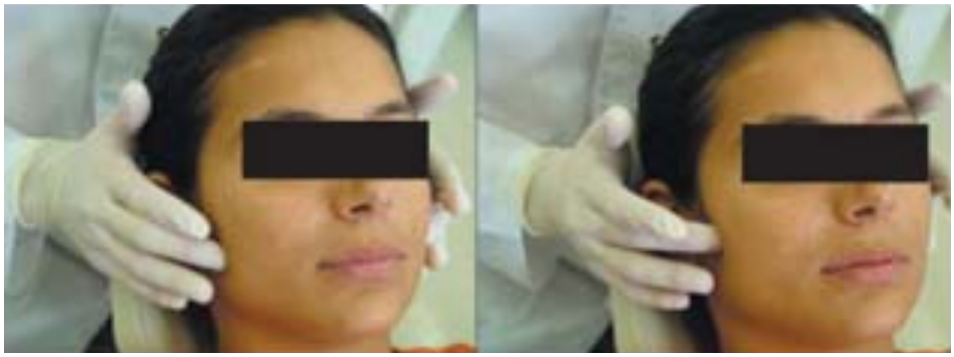

Fig. (3): Palpation of the superficial and deep masseter muscle.

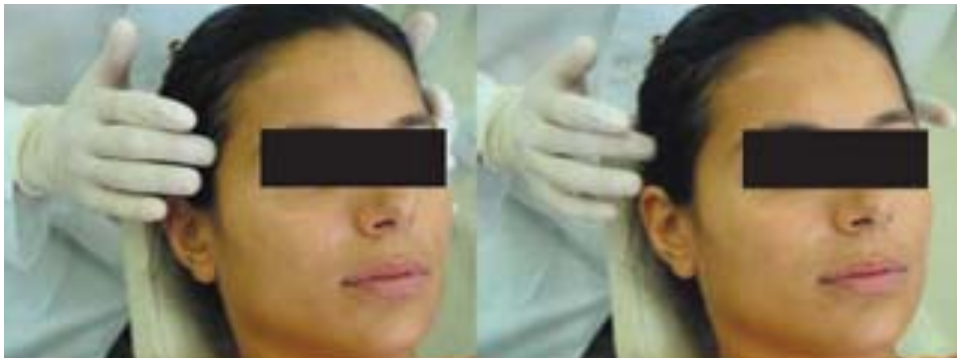

Fig. (4): Palpation of anterior and posterior temporalis muscle.

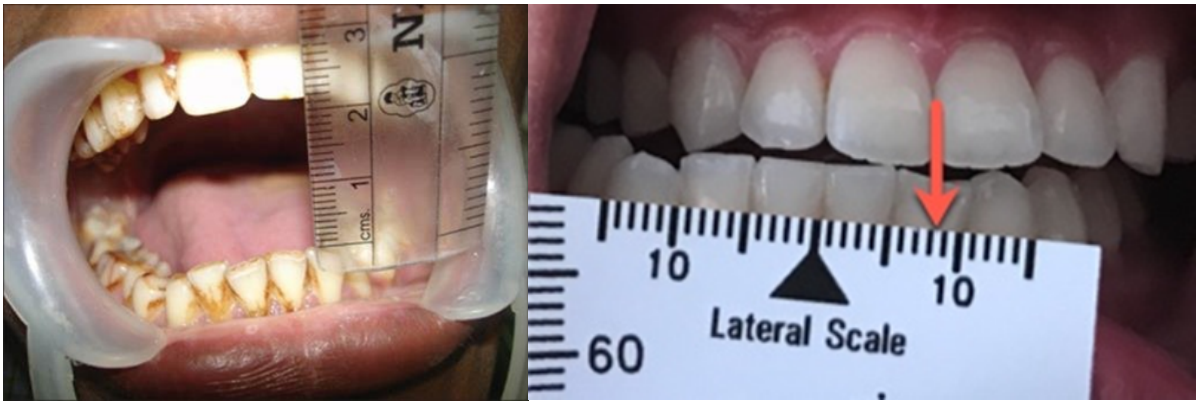

Fig. (5): Measurement of maximum active opening and lateral movement.

The Diagnostic Criteria for Temporomandibular Disorders (DC/ TMD) consists of guidelines and procedures that help the examiner to gain adequate inter-observer reliability by using diagnostic criteria for investigating muscle origin pain, disc displacement, and arthralgia and degenerative bone change of the TMJ (20). If the clinical examination indicates that further investigation is necessary imaging often starts with an orthopantomogram. This is a useful diagnostic and cost-effective entity that often can rule out other possible causes for the present symptoms, such as odontogenic infections, cysts, and coronoid hyperplasia. However, it is not an acceptable tool for diagnosing TMJ diseases, and other options have to be considered. Computed tomography (CT) or cone beam CT (CBCT) are the radiographic examinations of choice when possible hard tissue changes need evaluation. Magnetic resonance imaging MRI is often regarded as the gold standard for the evaluation of TMJ conditions. It is superior to CT/CBCT in diagnosing soft tissue changes and in depicting the disc status. MRI is also superior in diagnosing TMJ inflammatory states where joint effusion might be present. In addition, it allows the simultaneous evaluation of the morphology and position of the articular disc and bone structures of the TMJ (21). A multisection analysis of MRI images allows distinguishing the correct disc position from disc displacement and can improve the ability to distinguish between various stages of intra-articular derangement of TMJ (Figs. 6,7). A draw-back to MRI is the less accurate hard tissue diagnostics, such as fossa integrity and early local erosive changes.

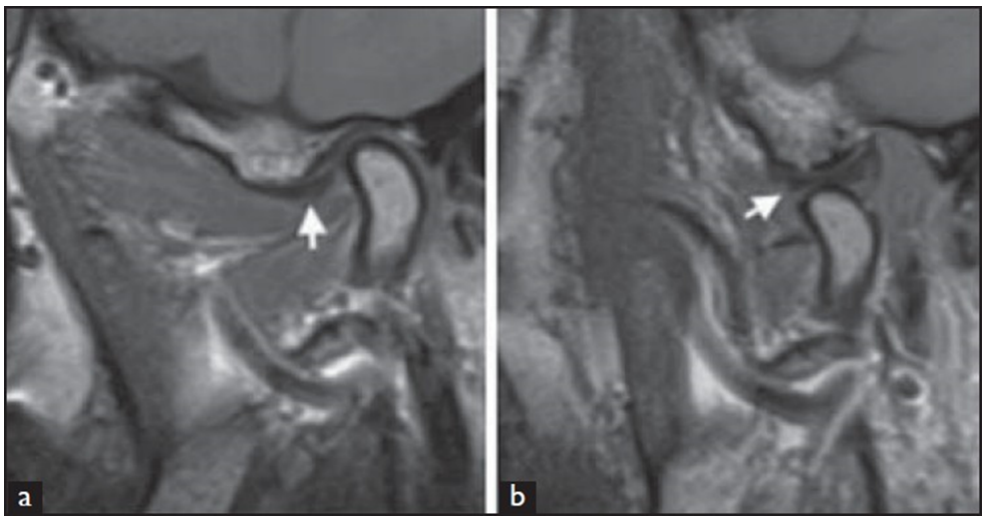

Fig. (6): T1-Weighted sagittal MRI (a) closed- and (b) open-mouth positions showing anterior disc displacement with reduction (arrows).

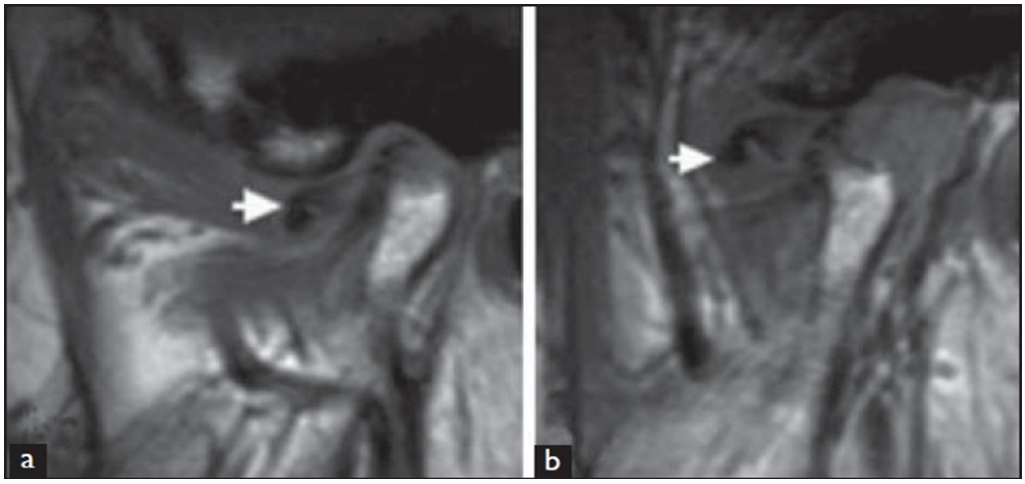

Fig. (7): T1-Weighted sagittal MRI (a) closed and (b) open-mouth positions showing anterior disc displacement without reduction (arrows).

Over the last decade, ultrasonography (US) of the TMJ has also been the focus of an increasing number of studies, which aimed to assess the diagnostic accuracy of US for TMJ disc displacement (22). US is a valuable imaging technique in assessing

TMJ disc position, however, the diagnostic value of high-resolution US depends strictly on the examiner's skills and on the equipment used.

Treatment of DDWR

Treatment of DDWR can be considered in three ways: non-invasive, minimally invasive and invasive management. According to Wilkes classification for internal derangement, early stages of internal derangement can be managed by non-invasive or minimally invasive methods; however advanced stages may require invasive/surgical approach (23). The principal treatment consists of removal of factors causing internal derangement, reduction of symptoms and promoting healing of the articular structures. Non-invasive methods include patient education, nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxants, mouth opening exercises and physiotherapy, softer diet, occlusal appliances, thermotherapy, bio-stimulating lasers and inter-maxillary fixation. Minimally-invasive methods include intra-articular injections, and arthrocentesis. Invasive/surgical methods include arthroplasty which includes reshaping of the articular surfaces, discectomy and implementation of autologous and alloplastic material and total joint replacement. However, in the absence of complaints, no treatment is recommended (19). Occlusal appliance/ splints are a standard method to treat disc displacement with reduction of the temporomandibular joint. Stabilization splint (Fig. 8), distraction splint and the anterior repositioning splint (Fig.9), are the most commonly used occlusal appliances to treat disc displacement with and without reduction. Huang, et al. (24), treated 59 patients with painless clicking with a mandibular stabilization occlusal splint (hard acrylic). After six months, there was an elimination of TMJ clicking in 71.2% of cases. In another study, Conti, et al. (25), assessed the effectiveness of the partial use of intraoral devices and counseling in the management of 60 patients with DDWR and arthralgia. When joint sounds (clicking) were investigated after 3 months, a decrease in frequency were observed. The basic aim of splint therapy is to

temporarily stabilize the mandible in an appropriate anterior position that allows the disc, if possible, to take a normal position on the condyle, and that the retrodiscal tissue releases the pressure and thus eliminates the pain and clicking sounds of the joint. Much more important is moving the condyle forward, which should enable the adaptive and regenerative processes in the retrodiscal tissue. On the other hand, Truelove, et al. (26), evaluated treatment outcomes in 200 patients with anterior disc displacement with reduction, arthralgia, and myalgia and divided them in different treatment groups (self-care, hard splints, soft-splints). Treatment outcomes were evaluated at 3 months and 12 months, which revealed no significant difference in all 3 groups. These investigators concluded that self-care and low-cost therapy are as effective as occlusal splint therapy. Splint therapy is however indicated for a short-term use only. Longer treatment duration may result in undesirable occlusal changes and dependency (27).

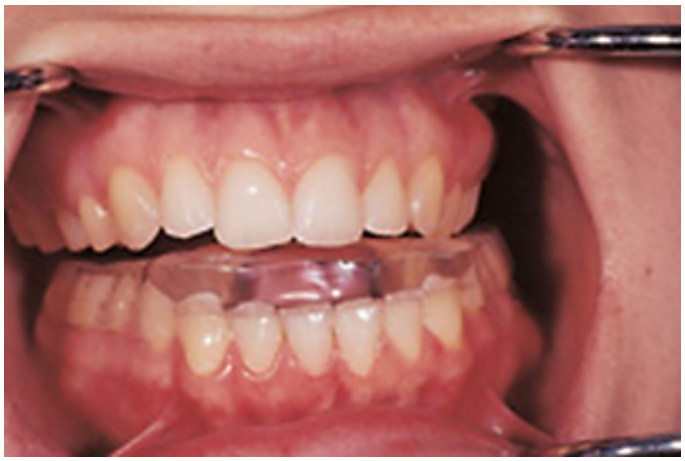

Fig. (8): Stabilization appliance allows freedom of movement for the teeth and the condyle, but is guided by the anterior teeth.

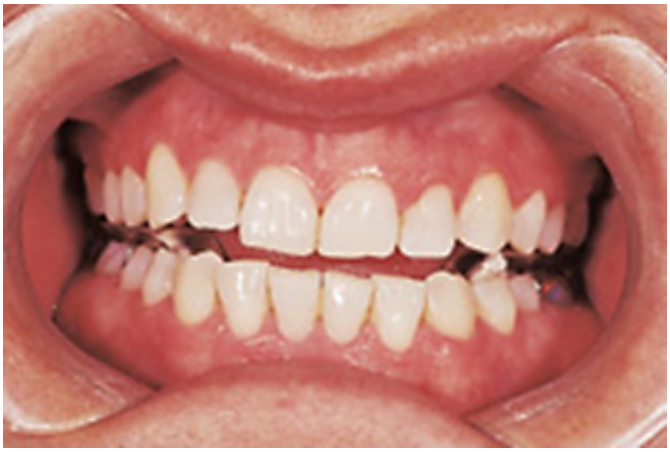

Fig. (9): An anterior repositioning appliance (nonpermissive) locks the teeth and mandible in a forward position.

Disc displacement without reduction

Among the different kinds of temporomandibular joint (TMJ) disorders, the displacement of the disc without reduction (DDWoR) has a prevalence of 35.7% (16). In this condition, either with the mouth open or closed, the disc remains anteriorly displaced in relation to the condyle (Fig. 2). The most prominent clinical sign of acute disc displacement without reduction is very limited mouth opening, maximum about 25–30 mm. When opening the middle line of the mandible, it suddenly turns to the affected side. The protrusion of the mandible is also limited and associated with the deflection of the mandible on the affected side (Fig.10). Since the displaced disc represents a mechanical barrier for each translatory motion of the condyle, the lateral movement of the mandible to the opposite side is also limited. Acute disc displacement without reduction, due to the accompanying inflammation of the capsule, retrodiscal connective tissue and the discal ligaments, is always followed by pain. The activity of master and temporalis muscles on the affected side is also disturbed. The spasm of these muscles increases the pain and still limits the opening of the mouth. Acute condition has no sounds, as its function

is practically blocked, but when the disc displacement becomes chronic, degenerative changes may occur on the articular surfaces of the joints causing the characteristic crepitations.

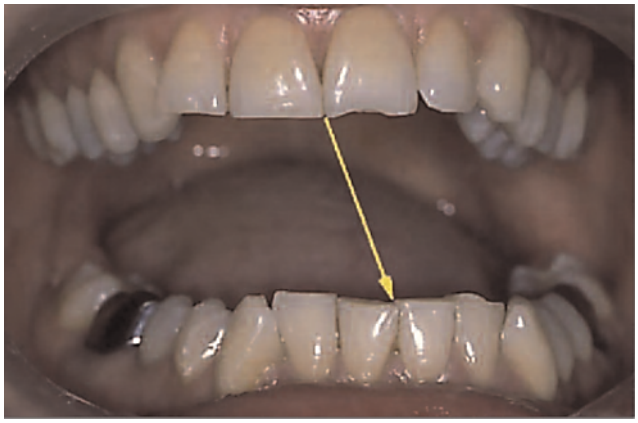

Fig. (10): Deviation and decrease in opening amplitude are characteristic of disc displacement without reduction (affected left side).

Treatment for anterior disc displacement without reduction

Mandibular manipulation

The aim of mandibular manipulation is to recapture a displaced disc. The common technique for mandibular manipulation is pulling the condyle of the affected side downward and forward in order to locate the condyle on the anteriorly displaced disc. Usually the operator's thumb is placed on occlusal surfaces of lower molars on the affected side and the rest of the fingers are covering the external surface of mandible (Fig.11). The other hand is placed in temporal area to stabilize patient's head. Foster, et al. (28), in their study concluded that mandibular manipulation under general anesthesia showed improvement in patients with disc displacement without reduction. It is difficult to predict the therapeutic effect of this method as it depends not on the duration of locking, but rather on the stage of internal derangement. Kurita, et al. (29), reported that in 9% of cases, closed locked disc could be reduced by manipulation technique and if the procedure is performed under general

anesthesia in the presence of muscle relaxants the percentage of successful outcome rises to 42%. After successful unlocking it is recommended that the patient is provided with an anterior repositioning splint in order to eliminate acoustic symptoms in TMJ disc. The literature on manual manipulation for TMD indicate that it is a viable, cost effective and reversible mode of conservative treatment.

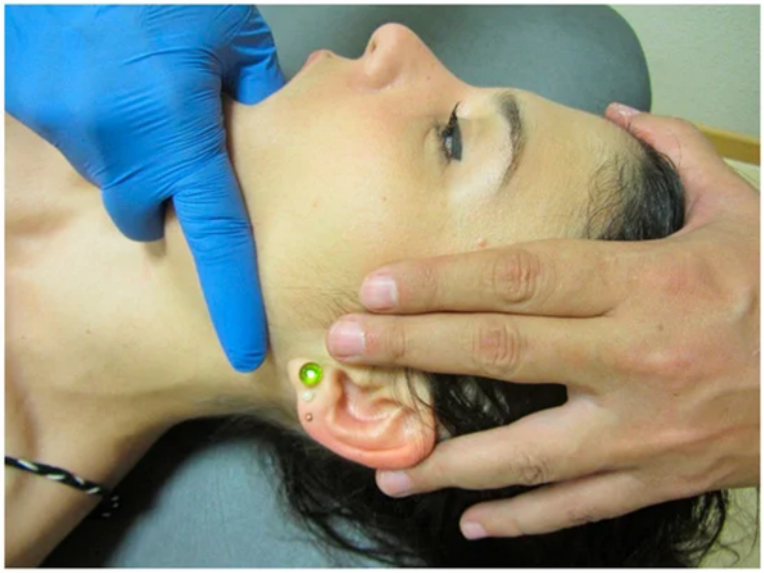

Fig. (11): Mandibular distraction mobilization technique.

Occlusal appliance

According to many authors, stabilization splint, leads to a significant reduction of symptoms of closed lock. Stiesch-Scholz, et al. (30), reported that stabilization splint therapy is an efficient method of closed lock treatment. In that study the significant improvement of clinical outcomes was present in 92.7% of patients. In another study, Schmitter, et al. (31), found that stabilization splint seems to be more effective than distraction splint in closed lock therapy. On the contrary, Minakuchi, et al. (32), compared the short-term effect of combined splint plus exercises (self-

care/medication/education) with education only, found no statistically significant differences in effect between the interventions on all measured outcomes.

Arthrocentesis

Sometimes the clinical symptoms compatible with DDWOR and joint pain did not respond to a conservative treatment for at least three months (occlusal splints, anti-inflammatory drugs, compresses, soft diet and physiotherapy). If these methods are not efficient, surgical procedures should be considered (33). There is a spectrum of surgical procedures currently used for the treatment of temporomandibular disorders ranging from temporomandibular joint arthrocentesis and arthroscopy to the more complex open joint surgical procedures, referred to as arthrotomy. Indications for surgery have been suggested to be either absolute or relative (34). Absolute indications are associated with trauma, ankylosis, congenital anomalies or organic pathology that requires excision. Relative indications, are subjectively determined by the surgeon and should not blindly include failure of conservative therapy as this may be based on inaccurate diagnosis and treatment.

Arthrocentesis of the temporomandibular joint refers to lavage of the upper joint space, hydraulic pressure and manipulation to release adhesions of the "anchored disc phenomenon" and improve motion. It was first described by Nitzan and colleagues in 1991 (35) to treat acute closed lock jaw. Arthrocentesis is a minimally invasive joint surgery and it is effective for decreasing pain, increasing maximal interincisal distance, eliminating joint effusion and improving the oral health related to the quality of life of the patients with TMJ disorders (36). Through arthrocentesis the microscopic tissue debris resulting from the breakdown of the articular surfaces and the pain mediators can be washed out, and normal lubricating properties of synovial membrane can also be stimulated. The technique (37), starts by drawing a straight line from the middle portion of the tragus to the outer canthus of the eye (Fig. 12) and two points were marked on this line for the insertion of the needles:

the first, the most posterior one, at a distance of 10 mm from the tragus and 2 mm below the reference line; the second was inserted 20 mm anterior to the tragus and 10 mm inferior to the reference line. Antisepsis was performed with 2% chlorhexidine solution that was used all over the face, mainly in the preauricular area and ear. The next step was the auriculotemporal nerve block, followed by the anaesthesia of the posterior deep temporal and masseter nerves with local anaesthetic without vasoconstrictor. Intra-venous sedation may be employed as an adjunctive measure for patient comfort. The patient was then requested to open the mouth to the maximum, and a sterile mouth opener was placed on the contralateral side of the procedure, allowing the displacement, down and forward, of the condyle, which enabled the access to the posterior recess of the superior compartment of the temporomandibular joint where the first needle (18G) was introduced. The needle was then connected with a 5mL syringe and 4 mL physiological solution, sodium chloride 0.9%, was administered in order to distend the joint space. A second needle was introduced into the distended compartment, at the point established before to enable a free outflow of the solution (Fig. 13). Through one needle, 100-300 ml of Ringer's lactate is injected during a 15 to 20-minute period under hydraulic pressure generated by placing the infusion bag at an elevation of above the level of the joint. The second needle acts as an outflow portal, which allows lavage of the joint cavity (Fig. 14). During the procedure, reestablishment of normal maximum mouth opening is determined by having the patient make repeated attempts to open the mouth.

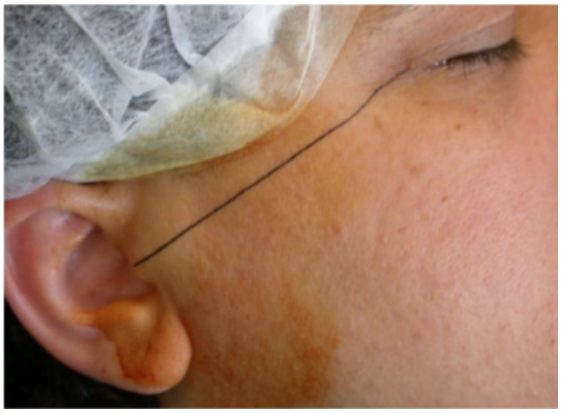

Fig. (12): Hollumuland-Helsing line (tragus-canthus line).

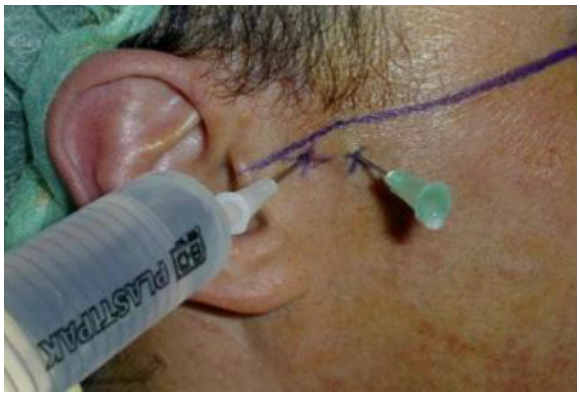

Fig. (13): The inlet and outlet needles inserted in the predetermined locations.

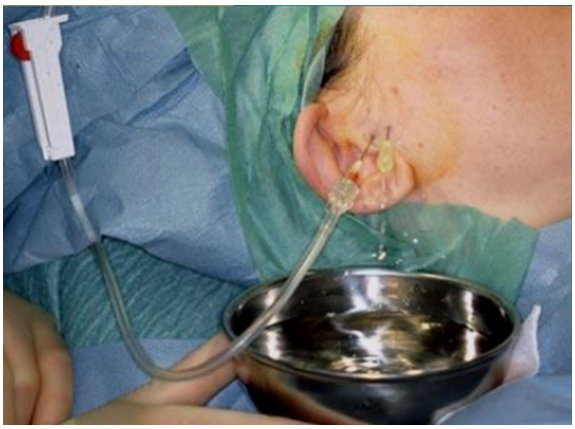

Fig. (14): Lavage of the upper joint space.

Following arthrocentesis different types of medications could be injected. These includes infiltrations of sodium hyaluronate (38), intra-articular analgesics (39), corticosteroids (40), and platelet rich plasm (41).

The minimally invasive character of the arthrocentesis produces less post-operative morbidity if compared with other surgical techniques for the TMJ. The main disadvantages in relation to the arthroscopy is that: it is not possible to visualize the intra-joint pathology; there are limitations for performing biopsies; and it is more difficult to eliminate adherences or adhesions in more advanced stages. Although the complications associated to the arthrocentesis are rare, the literature has reported some risks (42), such as: extravasation of liquid to the surrounding tissue, lesion of the facial nerve, optical lesion, pre-auricular hematoma, arteriovenous fistula, trans articular perforation, intracranial perforation, extradural hematoma and intra-articular problems.

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Chapter 5: Temporomandibular joint osteoarthrosis

The term “Osteoarthritis” is derived from the Greek word part osteo-, meaning “bone”, combined with arthritis: arthr-, meaning “joint”, and –itis. Strictly taken, this application is incorrect. Because the suffix “-itis” implies the presence of inflammation. Therefore, “osteoarthritis” should be considered misleading since inflammation is not a primary feature of the degenerative joint disease. Here the mechanical stress, avascular necrosis, posttraumatic sequelae, metabolic causes, etc. are the etiopathophysiological factors leading to the process of joint cartilage degenerative noninflammatory breakdown. The term “osteoarthrosis” is correct for degenerative joint disease. Inflammation may play only a secondary or concomitant role in this condition. This is in contrast to rheumatoid arthritis, which is primarily an inflammatory pathologic entity. In short, “osteoarthritis” means inflammation of the joint, while “osteoarthrosis” means degeneration of the joint (1). The diagnostic criteria used to differentiate between “osteoarthritis” and the biologically different “osteoarthrosis” is not effective due to their shared multiple clinical signs and symptoms (2). Gross morphologic abnormalities can be present in the absence of TMJ pain and dysfunction. Furthermore, the distinction between the diagnoses of “arthralgia” and “arthritis” is difficult since the signs and symptoms overlap. Arthralgia may be due to intra-articular pathology (e.g., arthritis or osteoarthrosis) or extra-articular factors (e.g., joint hypermobility or sensitization, peripheral or central) (2).

The term ‘‘osteoarthritis’’ has classically been defined as a low-inflammatory arthritic condition, either primary or secondary to trauma or other acute or chronic overload situations, characterized by erosion of articular cartilage that becomes soft, frayed, and thinned resulting in eburnation of subchondral bone and outgrowths of

marginal osteophytes. Pain and loss of joint function result. The term "osteoarthrosis", a synonym for osteoarthritis in the medical orthopedic literature, has recently come to be identified in the dental temporomandibular joint (TMJ) literature with any low-inflammatory arthritic condition that results in similar degenerative changes as in osteoarthritis. In the dental TMJ literature, however, the term osteoarthrosis is generally used to denote an abnormal osseous morphology of the mandibular condyle and the articular eminence (3).

Epidemiologic studies have indicated that the disease occurs in 8-16 % of the population and involvement may be unilateral or bilateral (4). Aging is one of the main risk factors for incidence of OA. Degenerative changes of TMJs have been shown to occur in as many as 80% of people between the ages of 60 and 89 years with a predilection for females after the age of 50 years. This may be due to estrogen receptor alpha polymorphism and increased pain susceptibility (5). OA has multiple risk factors' such as age, genetics, trauma, joint and muscle disorders, infection, generalized osteoarthritis, congenital and developmental abnormality and idiopathic degenerative process. Trauma includes past fractures, repetitive adverse loading, high-impact and torsional loads, external or overt jaw trauma and prolonged micro trauma. Joint and muscle disorders consist of joint instability, inadequate muscle strength/endurance, internal derangements, discectomy and ligament laxity (6). The natural course of TMJ osteoarthritis is usually favorable and consists of periods of remission and cartilage regeneration. The disease process takes about 5.5 years from initiation to the final burnout phase. It is divided into three slow progressive phases (7).

1. Early phase (2.5–4 years): During evolution of the disease, patient complaints of clicking sounds and intermittent locking.

2. Intermediate phase (6 months to 1 year): It is associated with TMJ destruction and patient develops spontaneous joint pain at rest or with function, limitation in opening of mouth and grating sounds.

3. Late phase (burnout phase) (6 months): There is no degenerative activity, and TMJ eventually stabilizes with time. Therefore, if invasive procedures can be postponed with medical management, patients will ultimately benefit from it. There is no joint pain but some patient may have limitation of mouth opening and grating sounds.

The clinical assessment consisted of a standardized evaluation of signs and symptoms of TMD, including mandibular range of motion, joint sounds, joint and muscle pain on palpation, and pain on unassisted or assisted mandibular opening. Mandibular range of motion was evaluated for maximum opening and lateral movements. Maximum opening was measured from central maxillary incisor to the opposing mandibular incisor on a millimeter ruler. Lateral movements were measured relative to the maxillary midline with the teeth slightly separated. The TMJs were auscultated with a stethoscope, with the subject performing three openings, and three lateral and protrusive movements. These were described as single and reciprocal clicks. TMJ pain on palpation was assessed through bilateral manual palpation of the lateral aspect of the condyle. The parameter of TMJ pain during unassisted mandibular opening was assessed by asking the patient to perform maximum voluntary jaw opening. Assisted opening was performed by the application of force to the lower and upper incisors with the middle finger and thumb. A positive pain score was recorded by the examiner if a patient experienced a distinctively painful sensation in the TMJ during the procedure. Pain of the muscles was assessed as positive or negative by a bilateral manual palpation technique. The following sites were palpated: the anterior, posterior, and middle temporalis, the tendon of temporalis, the superficial and deep masseters, the lateral pterygoid, and

the anterior and posterior digastric muscle. A diagnosis of myalgia was assigned if palpation produced a clear reaction from the patient, i.e., if the patient experienced a distinctly tender or painful sensation in two or more muscle sites with muscle palpation pain of two or more on a 0 to 3 severity scale (8).

The most common symptom of any TMJ arthritic condition is painful joints. The pain arises from the soft tissues around the affected joint that are under tension and the masticatory muscles that are in protective reflex co-contraction as the result of Hilton's Law (9). This orthopedic principle states that the nerves that innervate a joint also innervate the muscles that move that joint and the overlying skin. Pain may also arise from the subchondral bone that is undergoing destruction as the result of the arthritic process. Other common and significant signs and symptoms of TMJ arthritis are loss of joint function or late-stage ankylosis, joint instability, and facial deformity attributable to loss of posterior mandibular vertical dimension as pathologic osteolysis decreases the height of the condyle and condyloid process resulting in an apertognathia (10).

The diagnosis of TMJ OA is mainly based on medical history and clinical examination. There are no specific laboratory tests to make a definitive diagnosis of OA. Results of laboratory tests such as rheumatoid factor (RF), antinuclear antibody (ANA), and erythrocyte sedimentation rate (ESR) are normal and are, therefore, useful only to rule out other diagnoses (11). The diagnosis in late-stage arthritic TMJ disease is usually obvious, when the disease process manifests in other joints. The problem in diagnosis comes with the uncommon patient whose arthritic disease first manifests itself as TMJ pain and mandibular dysfunction. A history of joint overload because of habits (e.g., excessive gum chewing, unilateral chewing) or parafunction (e.g., bruxism, clenching) and clinical examination are important. Because of the lack of correlation between the signs and symptoms and the history and physical findings, however, the most helpful approach to diagnosis may be derived from

information provided by appropriate imaging. A radiographic diagnosis of OA was made if there was radiographic evidence of the following: erosion of the articular surface of the condyle, rough condylar surfaces with bilateral joint space narrowing, thickening of the subchondral bone, sclerosis areas (Fig. 1), subchondral cysts (Fig. 2), and bone outgrowths-osteophytes (Fig. 3). Specific changes in the architecture of the subchondral trabecular bone due to accelerated bone turnover can form subchondral cysts called pseudocysts or Ely's cysts (12).

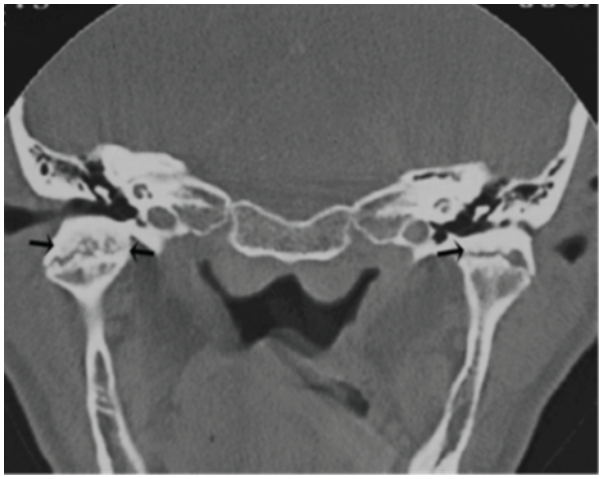

Fig. (1): Coronal CT image demonstrating bilateral joint space narrowing, rough condylar surfaces, and sclerosis of the subchondral bone (arrows).

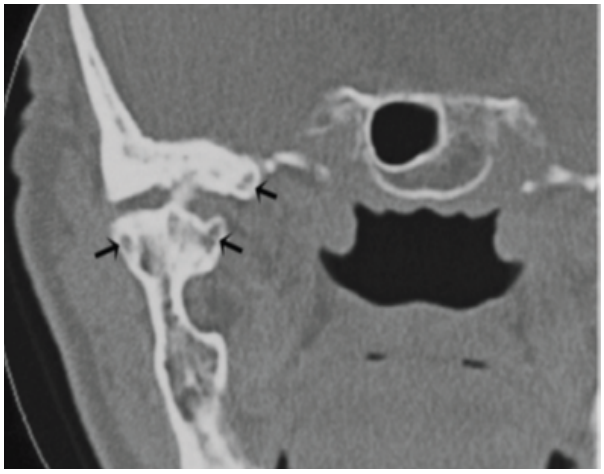

Fig. (2): Subchondral cysts called Ely's cysts (arrows).

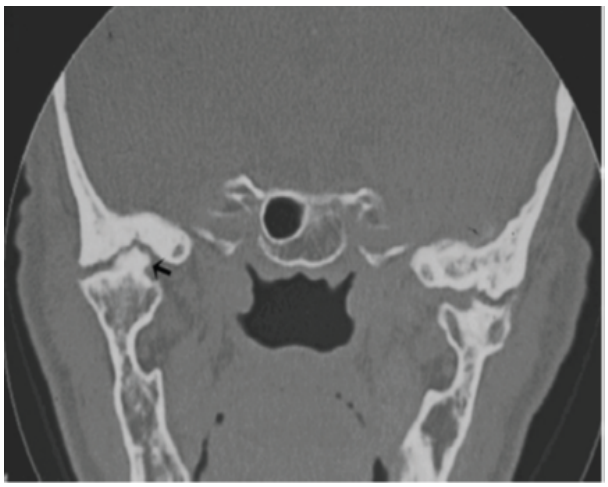

Fig. (3): Large bone outgrowth; osteophyte (arrow).

Management of TMJ osteoarthrosis may be divided into noninvasive, minimally invasive, and invasive or surgical modalities. Noninvasive modalities include topical and oral non-steroidal anti-inflammatory drugs, and muscle relaxants medications. The role of splint therapy in the treatment of TMJ osteoarthrosis remain debatable. Certain studies have found resolution of pain with the use of these appliances (13,14). Moreover, some studies provided evidence that pharmacotherapy increases benefits of splint therapy (15). On the contrary, other studies, found them ineffective (16).

Minimally invasive modalities included intra-articular injections of hyaluronic acid and corticosteroids, arthrocentesis, and arthroscopic surgery. The efficacy of hyaluronic acid (HA) injections was found comparable with corticosteroid injections or oral appliances (17). Nitzan and Price (18), presented a study of 36 patients who had not responded to nonsurgical management to determine the efficacy of arthrocentesis in restoring functional capacity to their osteoarthritic joints. They concluded that arthrocentesis is a rapid and safe procedure that may result in the osteoarthritic TMJ returning to a functional state. Arthrocentesis combined with HA, local anesthetics and corticosteroids have also been suggested. Arthroscopy is an important diagnostic and therapeutic modality in the treatment of TMJ disorders

being an alternative to arthrotomy (open TMJ surgery) and can be very effective in eliminating symptoms as pain, mandibular dysfunction, hypomobility, acute and chronic “closed lock” due to osteoarthritis and arthrosis with adhesive capsulitis, where nonsurgical treatment has been unsuccessful (19).

Despite the high success rate of conservative measures, there is still a group of patients whom an arthrotomy, the so-called invasive surgical modalities are necessary. Henny and Baldridge (20), described arthroplasty (high condylar shave) as a limited removal of the damaged articular surface of the condyle that maintains the height of the ramus, the articular disk, and the surrounding soft tissue, including the lateral pterygoid muscle attachment. Its use was advocated in severe, unremitting osteoarthritis pain. Reshaping the articular surfaces to eliminate osteophytes, erosions, and irregularities found in osteoarthritis refractory to other modalities of treatment was described by Dingman and Grabb (21). Severe inflammatory joint disease with condylar resorption in rheumatoid arthritis has been reported to have the best results with alloplastic reconstruction. Mercuri et al., (22), reported long-term outcome of 193 patients (mean follow up of 11.4 years), which showed a significant reduction in pain and an increase in mandibular function and range of motion after TMJ reconstruction using customized prosthesis. In another study Park et al. (23), described the outcomes of four patients receiving TMJ reconstruction using stock prosthesis involving, respectively, trauma, tumor, resorption, and ankylosis, and concluded that alloplastic prosthesis is a safe and effective management option for the reconstruction of TMJs.

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Chapter 6: Temporomandibular joint hypermobility

The normal range of jaw opening is between 40 and 50 mm. The initial 25 mm of opening is primarily achieved by rotation which occurs in the lower joint cavity between the mandibular condyle and the underneath surface of the disk. The remaining 15 to 25 mm is gained primarily through the forward gliding (anterior translation) motion that occurs in the upper joint cavity between the upper surface of the disk and the temporal bone of the skull. Most people who suffer from TMJ hypermobility open beyond 50mm (Fig.1). Radiographically, the condyle is found translating anterior to articular eminence (Fig.2). Hypermobility is characterized by early and/or excessive forward gliding (translating) of one or both TMJs. Hypermobility of the temporomandibular joint can be classified as a subluxation or a luxation. TMJ subluxation is a condition where the condyle translates anteriorly of the articular eminence during jaw opening and briefly catches in an open position before returning to the fossa spontaneously (1), or with manual self-manipulation by the patient. The Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) classification scheme (2), was expanded in 2014 to include less common, but clinically important disorders. According to these (DC/TMD), subluxation should have a positive history that the jaw has been caught in a wide-open position and the patient had to do a self-maneuver to be able to close the jaw (3). During TMJ luxation, the patient is unable to self-return to the fossa without the help of a clinician to maneuver the jaw back into a normal position. Thus, relocation of the condyle to its normal position occurs through self-manipulation in cases of subluxation, but not in luxation. Clinically, the patient will present with the jaw wide open, or protruded in bilateral luxation, or in lateral position to the nonaffected side in the case of a unilateral luxation (3). Bilateral luxation of the TMJ is most common with the mandible in a straight open position, whereas with a unilateral luxation, the

mandible is deviated to the opposite side (Figs.3&4), with a partially open mouth. Cardinal symptoms of TMJ luxation are impaired occlusion/inability to close the jaws, and pain (4). On clinical examination, an empty temporomandibular joint socket may be found.

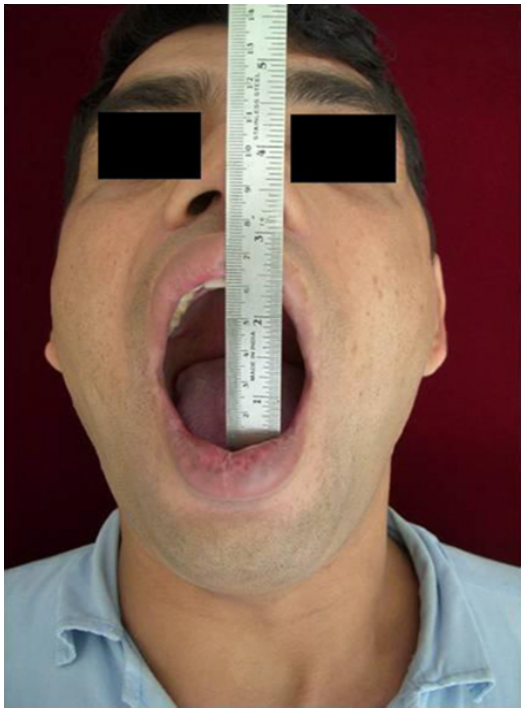

Fig. (1): Increased mouth opening beyond 50mm.

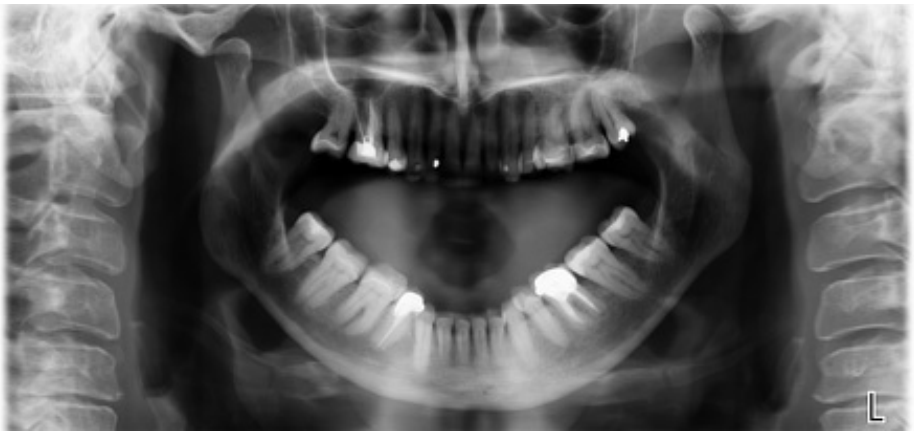

Fig. (2): Excessive forward translation of one both TMJs.

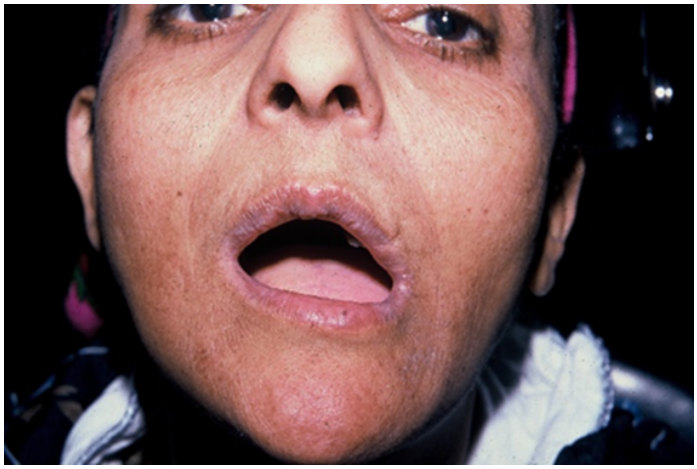

Fig. (3): Mandibular deviation in unilateral luxation (dislocation).

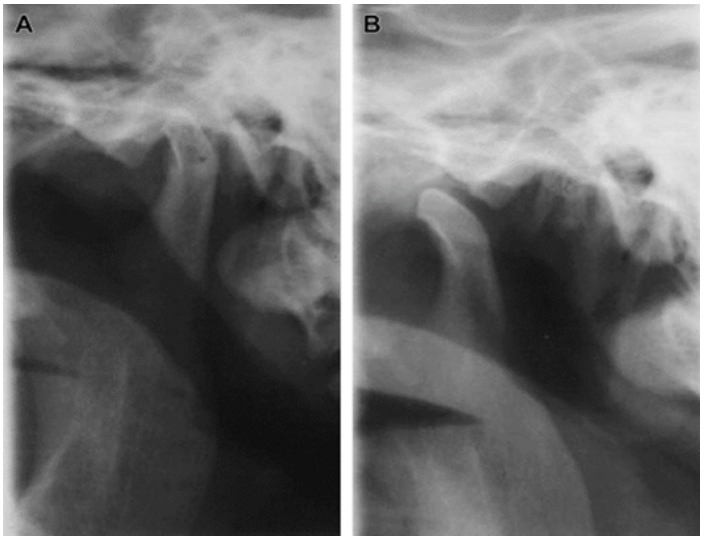

Fig. (4): Radiographic picture of unilateral luxation, A: Closed mouth, B: Mandibular condyle luxates into an anterior position of the eminence.

TMJ luxation can be acute or chronic. Acute TMJ luxation may occur as a result of external trauma, sudden wide mouth opening while yawning, taking a large bite, or laughing. In the clinical situation, TMJ luxation may occur after excessive mouth opening during dental treatment or other oro-pharyngeal procedures (5). It has been proposed that abnormalities in the stabilizing structures of the TMJ may be associated with luxation. The main factors for joint stability are the ligaments and

muscles together with the anatomy of the bony components of the joint. Moreover, luxation has been connected with anatomical variant where the articular eminence has a steep - short, posterior slope and longer anterior slope or an abnormal condylar shape (6). The condition may also occur as a result of injury, damage to the joint capsule. Factors such a generalized joint hypermobility (GJH), Ehler Danlos syndrome or Marfan syndrome have been linked with TMJ hypermobility. Generalized joint hypermobility (Fig.5), is a hereditary problem defined by the increase in range of motion in multiple joints. The Ehlers-Danlos syndromes (EDS) are a group of hereditary disorders that affect collagen and connective tissue structures throughout the body. Several subtypes have been identified; however, all are generally characterized by tissue fragility, skin hyperextensibility, and joint hypermobility (7). In patients with Ehlers-Danlos syndrome, hypermobility is common and associated with a weak capsule and ligament laxity. These patients are at risk for recurrent TMJ luxation. Pasinato et. al. (8), noted that the prevalence of GJH was higher in group with TMD. Nosouhian et. al. (9), observed correlation between the pain in TMJ and maximum mouth opening. Obtained results have led to the conclusion that the largest number of patients was at the age of 31-42 (70.99%), 37.67% of patients were at the age of 26 to 35 years old. Further, TMJH was observed much more frequently for women (78.2%) than men. In another survey Winocur et al. (10), the prevalence of general joint laxity and TMJH was estimated among adolescent girls. They concluded that the prevalence of generalized joint laxity was 43% and TMJH was recognized in 27.3%.

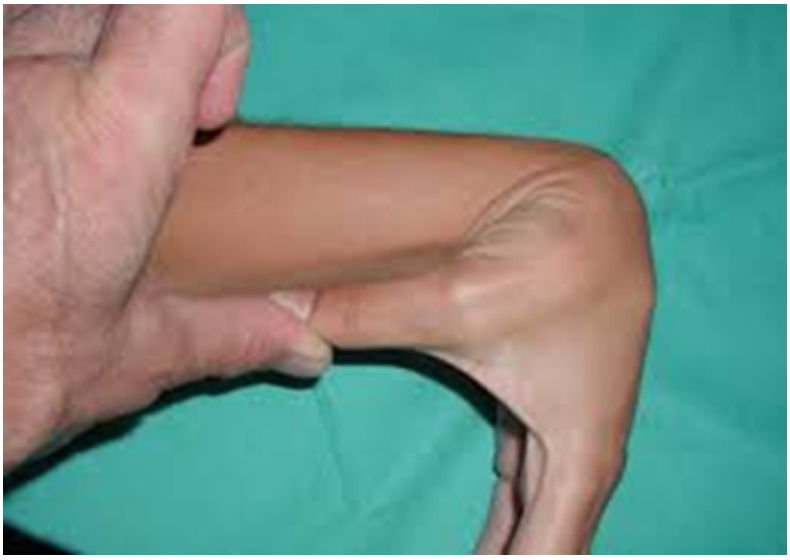

Fig. (5): Apposition of the thumb to the flexor aspect of the forearm in generalized joint hypermobility.

Management

Management of hypermobility remains a challenge, despite a large number of conservative and surgical techniques with variable results described in literature. Luxation (dislocation) of the TMJ can be classified as acute, chronic or recurrent. Most of the cases, however, are acute. When the TMJ condyle luxates into an anterior position of the eminence, a reflex is generated that sets the masticatory muscles into a spasm; this hinders the condyle from moving back to its normal position (11). These are associated with a significant negative effect on the quality of life of the affected patients and lead in the long term to subsequent damage to the temporomandibular joint (12). The standard treatment of acute TMJ dislocation is immediate (emergency) manual reduction of the mandible. Relocation of the condyle requires the condyle to move inferiorly until it bypasses the articular eminence, and then posteriorly back into the glenoid fossa (Fig.6). The earliest possible reduction is crucial since the longer the delay the more difficult it is to reduce the dislocation and the higher is the risk for recurrent dislocations (4).

Reduction of a dislocation of several-days duration is facilitated by injection of local anesthesia in the fossa and sedating the patient with diazepam. In resistant case the manipulation is carried out under general anaesthesia. In patients with persistent dislocations, reduction should be followed by immobilization to limit maximum opening of the mouth over a prolonged period of time (1 to 4 weeks) to prevent recurrence. Bandages, head-chin cap or an elastic intermaxillary fixation may be used for immobilization (13). After surgery, regardless of the technique used, patients should for some days only eat soft food and avoid opening the mouth widely.

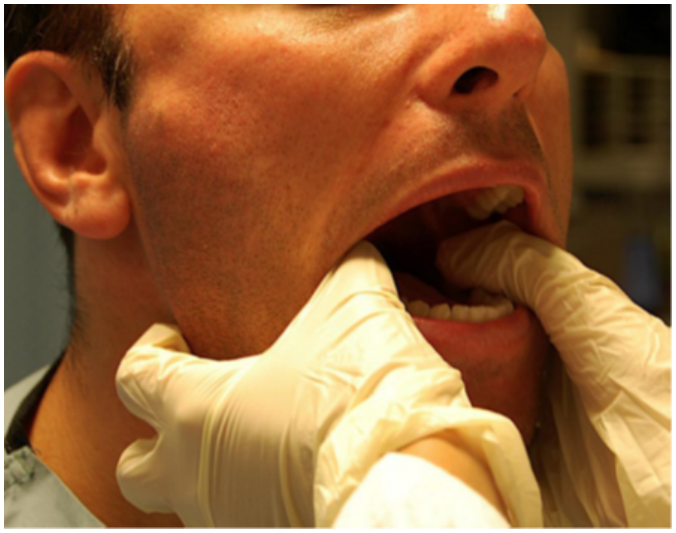

Fig. (6): Manual reduction of condylar dislocation.

Throughout history, many surgeons have described methods to treat recurrent TMJ dislocation. These include minimally invasive methods such as injection of sclerosing agents into the pericapsular tissues, eminectomy to remove obstruction to condylar movements, or various eminoplasty techniques to block the path of the condyle. These methods have been modified and adjusted by subsequent surgeons and academics, but to date no true gold standard treatment exists for recurrent TMJ dislocation. Goals of treatment of recurrent joint dislocation could be classified into

three categories. The first category was where management strategies could aim to either counteract joint hypermobility by inducing fibrosis in the superior joint space or pericapsular tissues. The second group of treatment modalities aims to remove barriers to condyle movement (such as with eminectomy) and the third group limit excess anterior condylar translation (such as with down fracture of the zygomatic arch and eminoplasty). Some authors suggested that conservative or minimally invasive methods should be tried before resorting to surgical management, while others suggested that the management of recurrent TMJ dislocation is primarily surgical. In general, minimally invasive methods were associated with a success rate of 80%, although some patients required multiple treatments.

Jaw exercises are one example of conservative treatment that aims to improve muscle strength and coordination after TMJ luxation. Other nonsurgical methods include intermaxillary fixation (14), prolotherapy, which is injection of sclerosing solutions (15), or autologous blood into the TMJ (16), and botulinum toxin injection into the masseter and pterygoid muscles (17). Some studies evaluated the effect of dextrose prolotherapy and reported that maximal mouth opening was significantly reduced with dextrose treatment (1). In the Daif study (5), autologous blood was injected in the superior joint space in one group and compared with a group of patients injected in both the superior joint space and the pericapsular tissues. The group injected in both areas had a more reduction in maximal mouth opening after 1 year with an 80% success rate compared to 60% in the superior joint space only group. Injections of autologous blood into the superior joint space and pericapsular tissue were also done in the Hegab study (18). Patients treated with autologous blood injection in combination with 4 weeks of IMF had significantly better outcome concerning reduced interincisal distance (average reduction 11 mm) and no recurrence of TMJ luxation, compared to autologous blood injection alone (8 mm reduction) and the intermaxillary fixation group (9 mm reduction) after 1 year. The

autologous blood injection alone group had the highest recurrence of luxation during the study period (8 of 16 patients). Hegab, performed repeated injections of autologous blood in the superior joint space and pericapsular tissues in patients with recurrent dislocations; this resulted in 6 recurrences of luxation in 16 patients after the first injection. After a second injection, there were still 2 recurrences, which were then finally successfully treated with a third injection. This indicates that repeated injections might be successful in recurrent TMJ luxation. According to Hasson et al. (16), blood injected into the superior joint space and pericapsular tissues causes scarring when fibrous tissue forms, restricting the mobility of the condyle and preventing TMJ luxation. Postoperative scarring may also be responsible for a substantial portion of the surgical benefit as evidence suggests that immobilization of a joint after an intra-articular surgical procedure results in fibrosis (19). Botulinum toxin injection into the masseter and pterygoid muscles is another proposed non-surgical technique (17).

Many surgical procedures have been advocated for treatment of hypermobility of the TMJ. The surgical options can be divided in two main techniques. The first option consists of creating a barrier to the extreme anterior slide of the condyle. It may be done through intentional down fracture of the zygomatic arch, also known as Dautrey's procedure (20), miniplating, bone grafting, and alloplastic materials attached to the articular eminence have also been described. In Dautrey's procedure (Fig.7), the zygomatic arch is cut downward and forward just in front of the articular eminence and then locked under the eminence, preventing excessive forward movement of the condyle. Soft tissue surgery for restricting condyle movement has been suggested, i.e., myotomy of the lateral pterygoids, lateral pterygoid muscle tendon scarification, scarification of the temporalis tendon (21), and capsule plication.

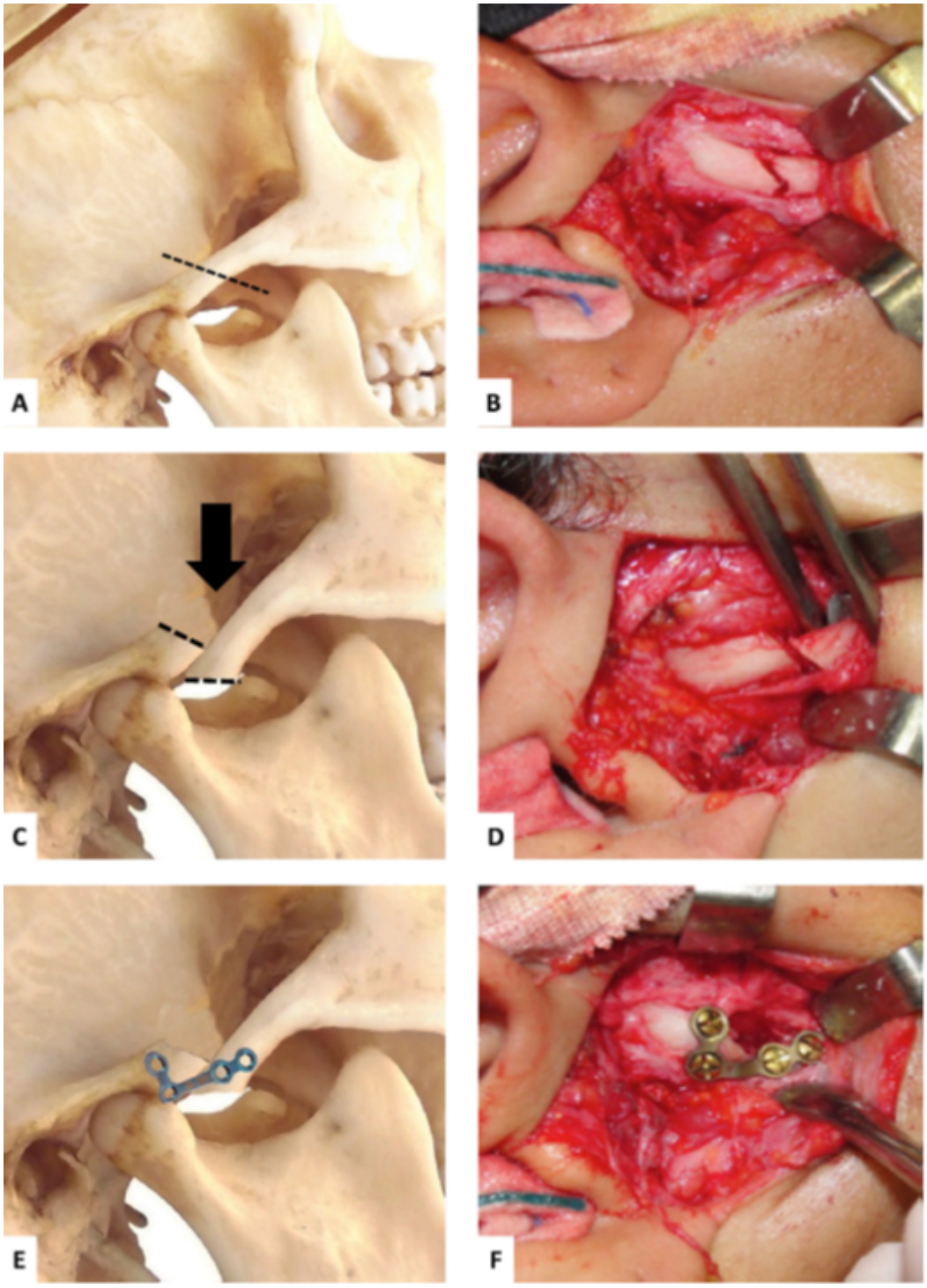

Fig. (7): Down fracture of the zygomatic arch, oblique osteotomy (A and B), caudal displacement of the distal segment of the zygomatic arch (C and D), and fixation with microplates (E and F).

The second option involves removing any barrier for spontaneous reduction which is accomplished best by eminectomy (Fig.8). The procedure prevents the condylar head from locking in the infratemporal fossa and allows it to return easily into the glenoid fossa when the mouth closes (22). Eminectomy was shown to be efficient in the treatment of chronic mandibular dislocations in relation to postoperative MMO, recurrence and articular function.

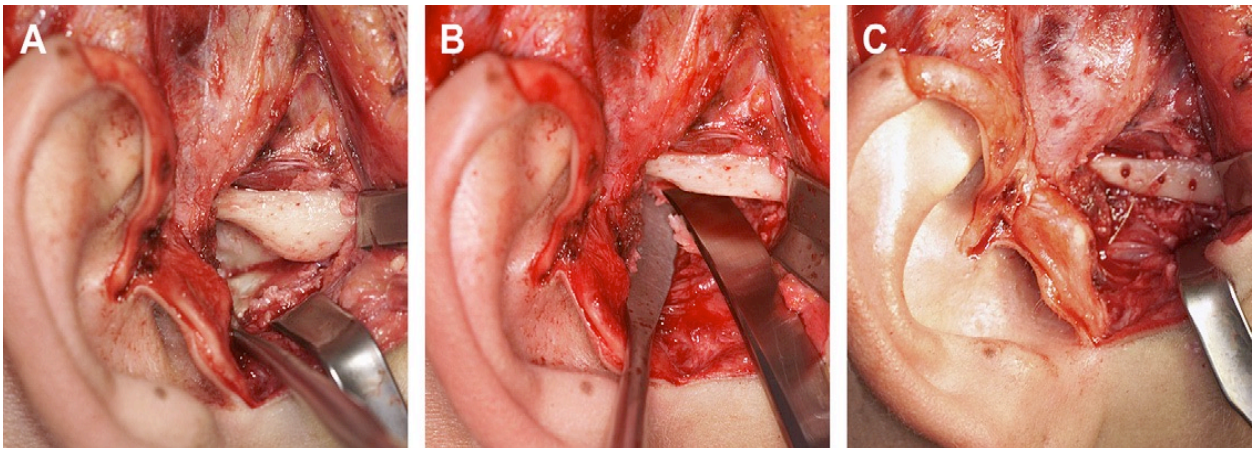

Fig. (8): Eminectomy, (A) The joint capsule is carefully preserved when exposing the lateral parts of the eminence. (B) Total eminectomy is performed with the aid of a chisel. (C) Following eminectomy, holes are drilled into the zygomatic arch and the joint capsule is reinserted.

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Chapter 7: Temporomandibular Joint ankylosis

Temporomandibular joint ankylosis is a condition characterized by fibrous, osseous or fibro-osseous fusion between the mandibular condyle and the roof of glenoid fossa in the temporal bone, resulting in partial or total loss of normal rotational and translational movement (1). Ankylosis of the TMJ is a serious and disabling condition. Impairment of speech, difficulty with mastication, rampant caries, poor oral hygiene, disturbances of facial and mandibular growth, and acute compromise of the airway invariably result in physical and psychological disability. This is particularly true of young children who are completely unable to open their mouth (2). Ankylosis is a Greek terminology meaning “stiff joint”. TMJ ankylosis may either be acquired or congenital. TMJ ankylosis is most commonly associated with trauma (13-100%), local or systemic infection (10-49%) or systemic diseases (10%), such as ankylosing spondylitis, rheumatoid arthritis and psoriasis (3). It has also been reported that TMJ ankylosis can also occur secondary to a TMJ surgical procedure (4). The pathogenesis of TMJ ankylosis of traumatic origin is that traumatic injury results in an intra-articular hematoma leading to fibrosis, excessive bone formation and hypomobility of the joint (5). Interesting to note that injuries leading to TMJ ankylosis may occur during forceps delivery at birth (6). Trauma to the condyle of the mandible may cause condylar fracture that can produce intra-articular ankylosis secondary to bony fusion of the condyle to the zygomatic process of temporal bone or the base of the skull. When a condylar fracture comes into close contact with the zygomatic arch it may cause an extra-articular type of ankylosis. Some studies have reported infection as the main cause of ankylosis in children. Infection of the TMJ may be a direct extension from other sites as occurring in otitis media, mumps, osteomyelitis of the zygomatic temporal bone, mastoiditis and soft tissue abscesses (5). Scarlet fever and typhoid may also be able to attack the joint directly through the bloodstream or cause otitis media or mastoiditis and affect the joint secondarily by direct extension (7).

Classification of TMJ ankylosis

Several classifications have been proposed in the classification of TMJ ankylosis in order to have a picture of surgical difficulty (4). It has been classified simply as true or false ankylosis. The true ankylosis is further classified by Sawhney (8), into four types, (Fig. 1).: In ankylosis type 1, fibrous adhesions are formed in/around the joint. In ankylosis type 2, there is a formation of a bony bridge between the condyle and glenoid fossa. In ankylosis type 3, there is a bony bridge between ramus and zygomatic arch on the outer aspect, whereas the articular fossa on the deeper aspect is intact with the articular disc, and in ankylosis type 4, there is a wide and thick bony bloc bridging across ramus and zygomatic arch. It is possible to detect an extension and a penetration into the middle cranial fossa.

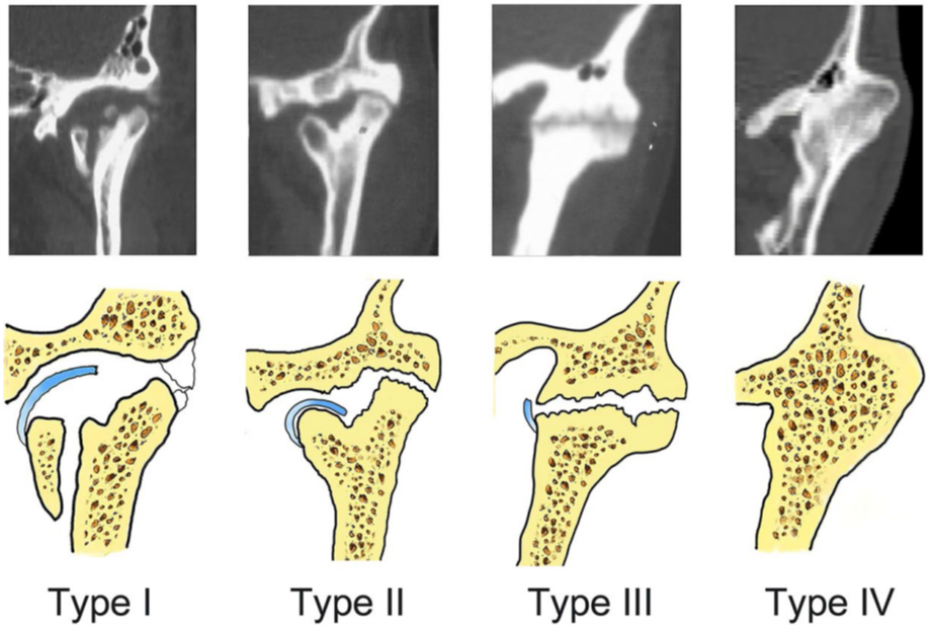

Fig. (1): Computed tomography images and representations of the four types of TMJ ankylosis. Type I is nonbony fibrous adhesions. Type II is lateral bony ankylosis of the joint. The medially displaced condyle, residual disc, and fossa form a pseudarthrosis. Type III is complete bony ankylosis of the entire joint with a radiolucent line inside the fusion area.
Type IV is extensive bony ankylosis.

Clinical findings

The clinical appearance of the patients with TMJ ankylosis depends largely upon the age of the patient at the time of affection and the duration of the ankylosis. The earlier the onset of the ankylosis, the more severe is the facial disfigurement due to affection of the condylar growth center. Also, the longer the duration of ankylosis, the severe is the deformity. Such deformity is due to destruction of the condylar growth center as well as loss of growth stimulated by mandibular function. Limited mouth opening is the common clinical feature of patients with TMJ ankylosis. Inter incisors opening is an indicator of the severity of the ankylosis, and clinically, complete ankylosis is defined as a condition when opening is less than 5 mm. In unilateral ankylosis there is the possibility of some degree opening of the mouth, while in bilateral ankylosis the movement of the mandible is minimum or completely impossible. The severity of this limitation depends on whether the ankylosis is fibrous or bony (9). Severity of facial deformities resulting from TMJ ankylosis depends on the joint involvement, age of onset and duration (7). The ankylosis that occurs during early age (childhood), in patients still growing and over a long duration, results in severe facial deformities (9). In a unilateral case of early onset, there will be asymmetrical face, with deviation of the jaw to the affected side, accentuated antegonial notch, flattening of the unaffected side and canting of the occlusal plane (Figs. 2,3). Malocclusion is characterized by a cross-bite and canting of the occlusion- up on the affected side (10). This occlusal cant is caused by compensatory growth of the maxilla on the unaffected side. In unilateral joint involvement, regardless of the extent, there is slight movement when the patient attempts to open the jaw. The vertical height of the ramus may be reduced on the affected side when measured from the angle of the mandible to the lower border of the zygomatic arch. Bilateral TMJ ankylosis of childhood result in a symmetrical face, but the mandible is micrognathic, giving rise to what is described as-bird face deformity, (Fig. 4), (10).

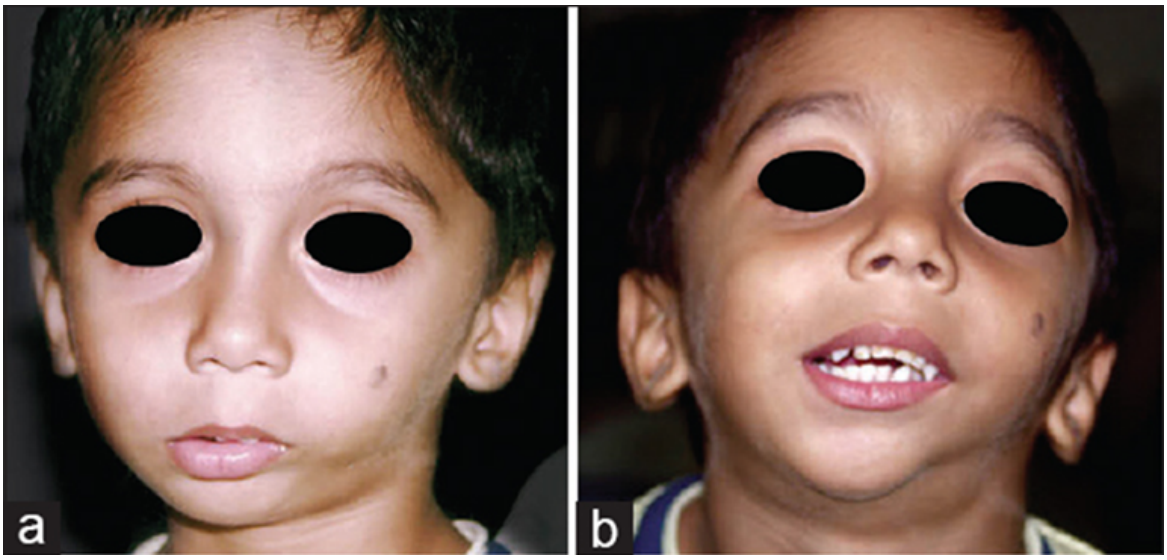

Fig. (2): Unilateral ankylosis of the right side; flattening left side of the face, deviation of chin to right side, and canting of the occlusal plane.

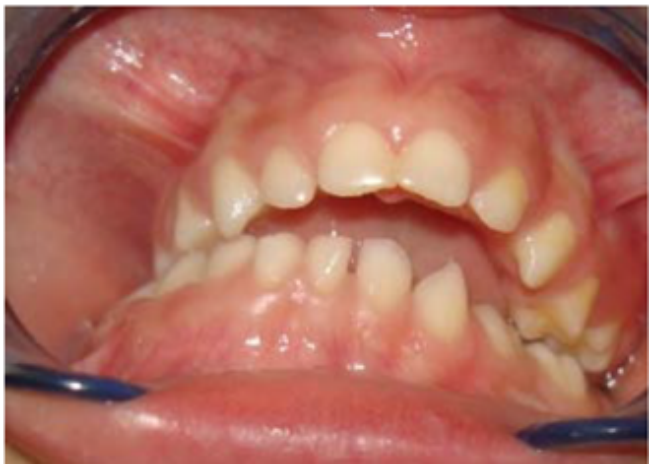

Fig. (3): Intraoral photograph shows midline deviation, mandibular shift, Occlusal cant and increased overjet.

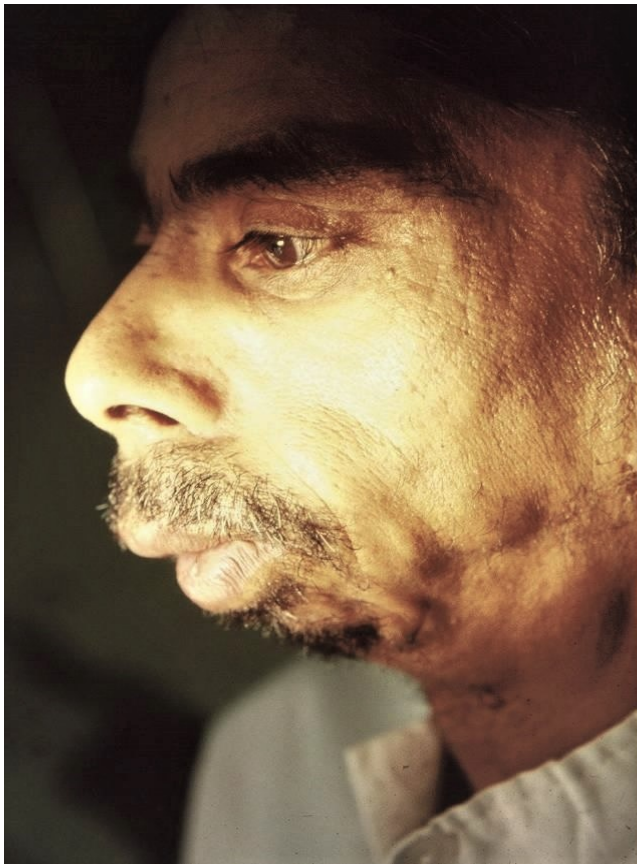

Fig. (4): Facial disfigurement, micrognathia (bird face).

Diagnosis of TMJ ankylosis

The diagnosis of TMJ ankylosis is based on clinical and radiographic features. Conventional radiographs; mainly panoramic and posterior-anterior views of the mandible, are very helpful in the diagnosis and provide valuable information with regard to the extent and type of ankylosis. However, clear information on the extent of the ankylosis are taken mainly with CT scan and three-dimensional CT scan. The radiographic picture (Figs. 5-8), in most cases, was reduced or complete obliteration of the joint space with fusion of the condyle to the glenoid fossa and elongation of the coronoid process. In addition, marked antegonial notching, flattening of the condylar head, and reduced vertical height of the ascending ramus of the mandible

are common features (11). In those with fibrous ankylosis, the joint space was diminished in size but one could still delineate the bony margins.

Differential diagnosis should be made by other pathological conditions that cause a restriction of the mobility of the mandible (trismus). Specifically, limited mobility of lower jaw may be due to an infection of the surrounding tissue (due to infection of an anatomical space, pericoronitis etc.), lesions, swelling of the muscles, after local anesthesia of the Inferior alveolar nerve (formation and organization hematoma), syndrome of masticator myalgia - dysfunction, tetanus or hysteric trismus (12).

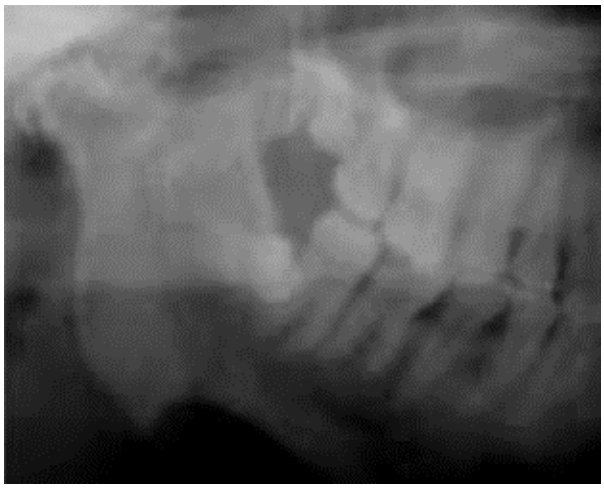

Fig. (5): Radiographically, ankylosed joint is characterized with obliterated joint cavity, short ramus, and accentuated antigonial notch.

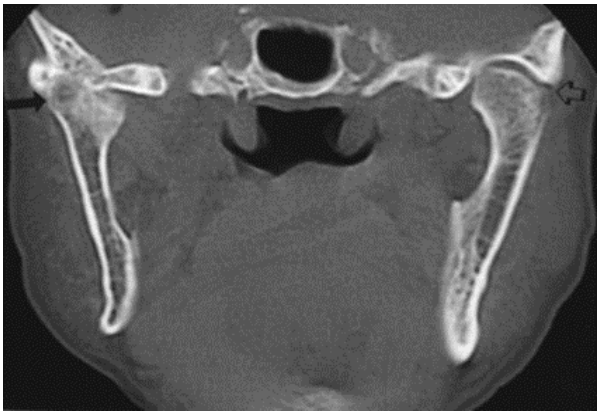

Fig. (6): Coronal CT scan shows bony ankylosis of the right joint.

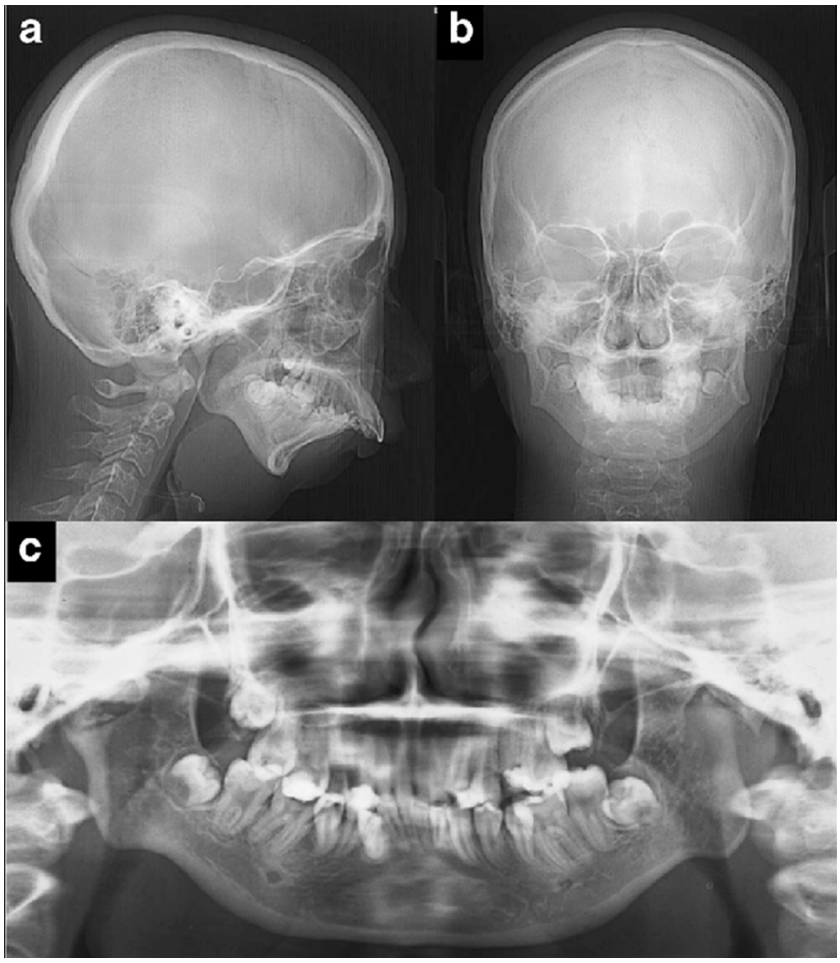

Fig. (7): Radiographic picture of bilateral ankylosis; (a) lateral cephalogram, (b) skull P-A, (c) OPG.

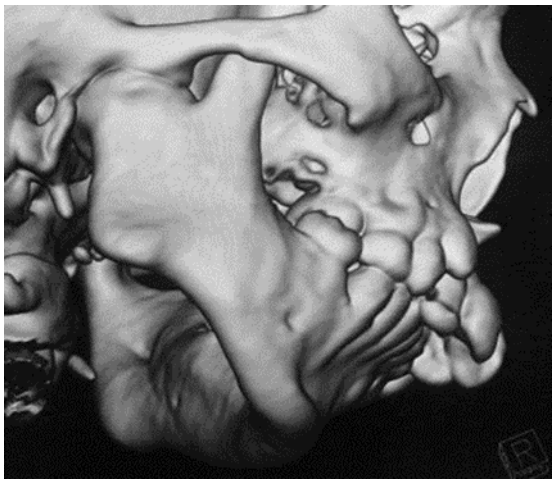

Fig. (8): 3-D CT scan showing ankylotic mass.

Management of TMJ ankylosis

A variety of techniques have been described for the management of TMJ ankylosis, yet no single technique has proved to be universally successful. The management of TMJ ankylosis has always been difficult and frustrating, particularly when dealing with long-standing or recurrent cases, or if the case is complicated by deformities of the jaw bones. The main objectives of treatment for TMJ ankylosis are to restore joint function, to improve the patient's aesthetic appearance and quality of life (13), to avoid re-ankylosis (14), to facilitate the maintenance of good oral hygiene (15), and to relieve airway problems if these occur (10). Carlson (16), classified the surgical procedures for release of the ankylosed condyle into 3 groups: condylectomy, gap arthroplasty and interpositional arthroplasty.

Condylectomy and gap arthroplasty

Although condylectomy and gap arthroplasty (Fig.9) can result in pseudo-articulation, these procedures are associated with a high recurrence rate. For prevention of re-ankylosis, a gap of minimum 1- 2 cm is recommended (17). Increased bony resection theoretically decreases the likelihood of re-ankylosis, but at the expense of ramus height. Moreover, the creation of a big gap and associated short mandibular ramus, probably leads to mandibular deviation in unilateral cases, posterior premature contact and open bite (10). Complications of gap arthroplasty (GA) reported in unilateral cases are posterior premature occlusion, open bite on the affected side and contralateral side, while in bilateral cases, severe anterior open bite may develop preventing mastication and causing aesthetic and respiratory issues (10). Some authors have reported improvements in appearance in patients treated with GA at an early age (18). Others however, prefer using GA as a primary procedure followed by reconstruction with costochondral graft (CCG) as a secondary procedure (19).

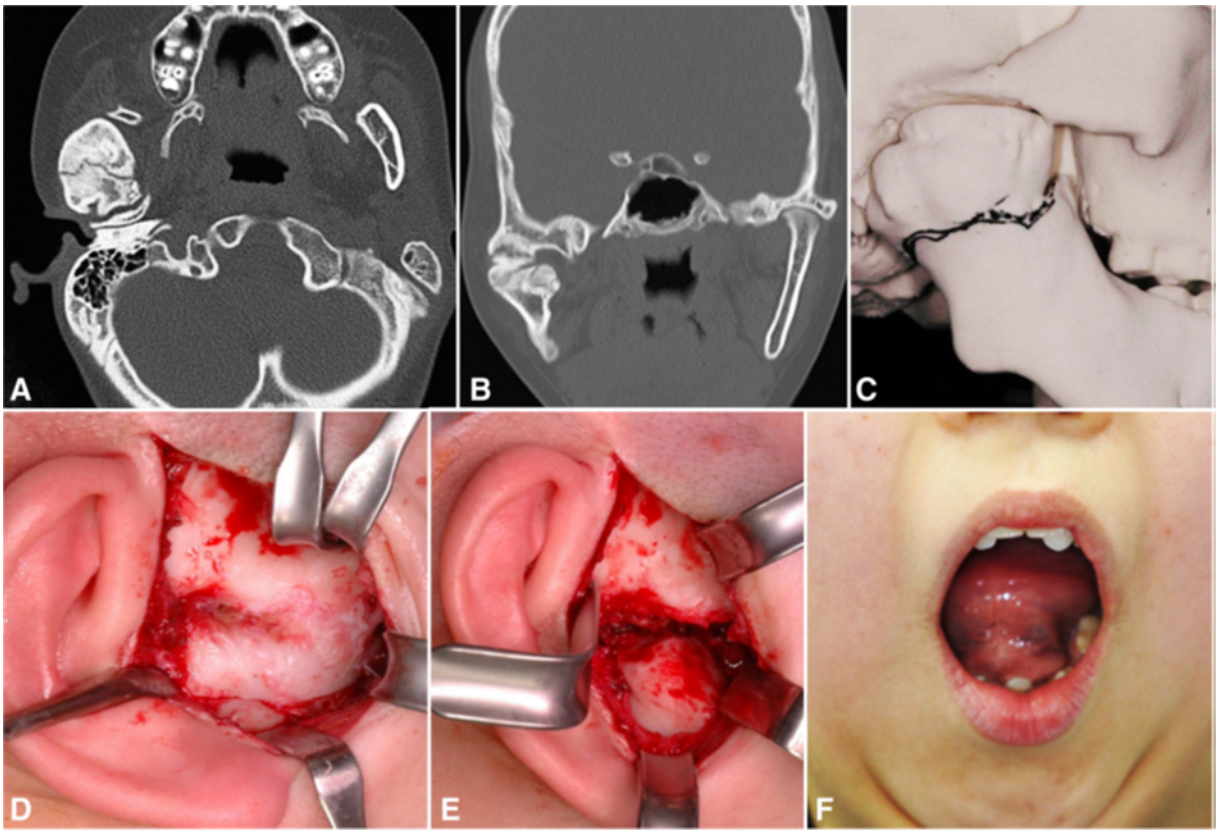

Fig. (9): Right side TMJ bony ankylosis; axial (a) and coronal (b) CT scan, model used for surgical simulation in gap arthroplasty and coronoidectomy (c), endaural approach (d), complete resection (e), and mouth opening appearance after active mouth opening exercise (f).

Interpositional arthroplasty

Interpositional arthroplasty (IA) refers to minimal resection of bone between the ramus of the mandible and mandibular fossa, and the placing of an interpositional material between the two bone segments (Figs.10,11). In IA autogenous materials are placed in the area between two bone segments to prevent recurrence of ankylosis, by preventing adhesion between the two bony cut surfaces, and maintain the length of the condylar and ramus area of the mandible. Huang et al (20), suggested that making a gap of correct size, choosing the proper interpositional material and diligent postoperative physical therapy are all important factors in preventing re-

ankylosis and in contributing to the success of the treatment. Various interpositional materials have been used, such as temporal fascia, auricular cartilage and dermis (21). Alloplastic materials have also been described (22). Gap arthroplasty with interpositional grafting remains the mainstay of join reconstruction following ankylosis. Temporalis fascia is commonly used tissue because of it is close proximity to the surgical area, which can be used from the same incision, good blood supply, easy preparation and harvesting, minimal cosmetic and functional morbidity. Topazian (17), compared 40 patients treated with gap arthroplasty, with and without the use of interpositional grafting. He found that half of the patients without grafting had re-ankylosis, and those who were grafted had no recurrence. It was hypothesized that interpositional grafting shields the exposed bony surface, preventing re-ankylosis. Güven (23), reported that the treatment using temporalis fascia as autogenous material and GA with active mouth opening exercises, provides an effective functional outcome and also assists in the prevention of re-ankylosis. However, gradual collapse of the gap by contraction forces from the masticatory muscles can also lead to recurrent TMJ ankylosis (17).

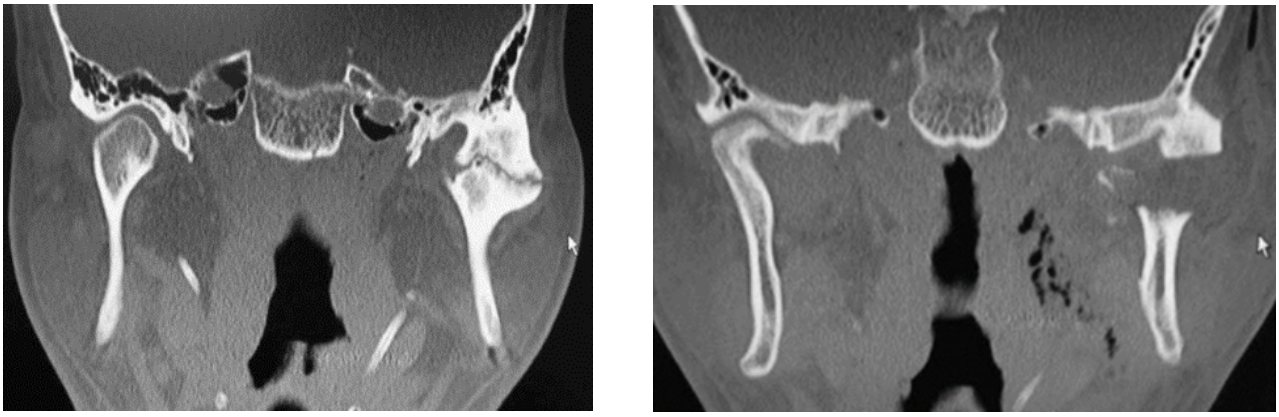

Fig. (10): Coronal computed tomographic scan of ankylosed left before and after interpositional gap arthroplasty.

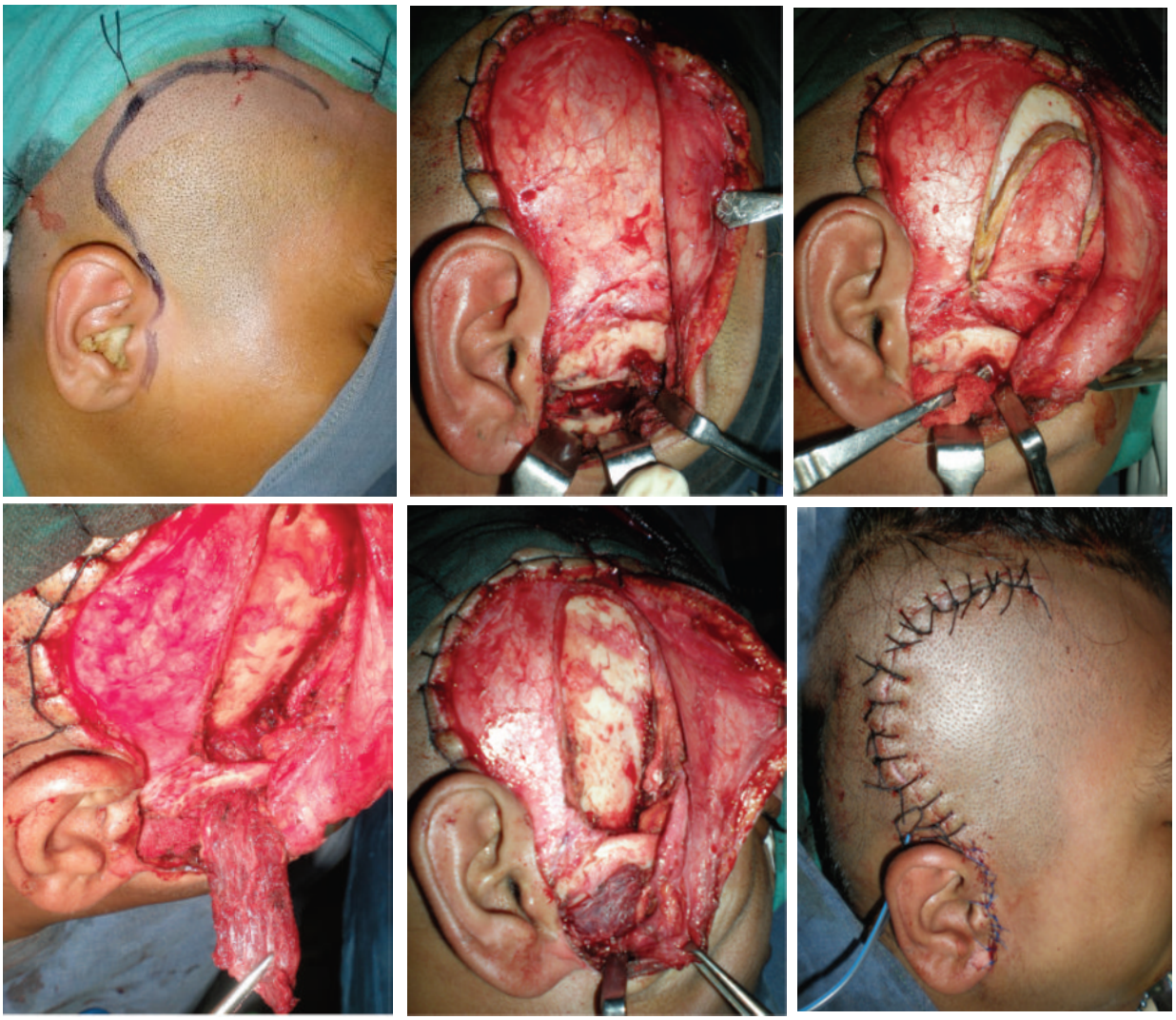

Fig. (11): Gap arthroplasty and temporalis graft interpositioning through preauricular approach.

Reconstruction of TMJ

Autogenous bone and alloplastic materials have been used for reconstruction of the TMJ. Reconstruction of the joint is done in children to transplant the condylar growth center, and in children and adults to restore the vertical height of the ramus and thereby restore mandibular symmetry. Autogenous bones that have been used as grafts in the management of TMJ ankylosis are metatarsal head, costo-chondral, sternoclavicular joint, fibular head, iliac crest and coronoid processes (24). Costo-

chondral grafts have proved best among the autogenous tissues used for reconstruction of the mandibular condyle. The main reason for its success is the similarity of the costochondral graft to the condyle and its capacity to regenerate and grow. Costo-chondral graft decreases the incidence of re-ankylosis after gap arthroplasty and keep the proper ramus height, while the advantages are biological compatibility and functional adaptability (19). Moreover, the graft prevents jaw deviation or production of an open bite and in children is an active growth center (25). Potential problems with the use of costo-chondral graft include fracture, donor site morbidity and the variable growth behavior of the graft. The growth of the transplanted rib is unpredictable as its growth may be proportional to the rest of the mandible or may overgrow or may undergo resorption (26). Saeed and Kent (27), used costo-chondral grafts for management of TMJ ankylosis. They reported a high incidence of re-ankylosis, and a limited improvement in mouth opening. The most prevalent hypothesis for re-ankylosis is that the overgrowth of the mandible after costochondral grafting occurs because of the transplantation of excessive amounts of cartilage (28). In accordance with that theory, Perrott et al. (29), reported that when only 2 to 4 mm of cartilage were transplanted with the rib, no overgrowth of the grafts occurred. In the study by Qudah et al. (30), although only 2 to 3 mm of cartilage was transplanted, 2 of 16 patients in the costochondral graft group showed overgrowth of the mandible on the operated side during the second year of follow-up.

Alloplastic joint prostheses have been developed for use in the reconstruction of TMJ ankylosis to avoid the complications associated with costo-chondral graft treatment. Alloplastic materials are used to maintain the height of the condyle-ramus region and to reduce recurrence rate of the ankylosis (Fig. 12). A comprehensive and well documented systematic review of the history of alloplastic TMJ prosthesis has recently been published (31). Henry and Wolford (32), evaluated patients who under-

went multiple TMJ surgeries and reported a higher success rate with custom-made total joint prostheses than with various autogenous grafts (88% vs. 13–31%). Wolford et al. (33), demonstrated adequate function of artificial TMJ prostheses during a follow-up period of 5 to 8 years. However, such prostheses are also associated with problems such as wear, mobility or fracture of the implant, hypersensitivity, and a foreign body reaction.

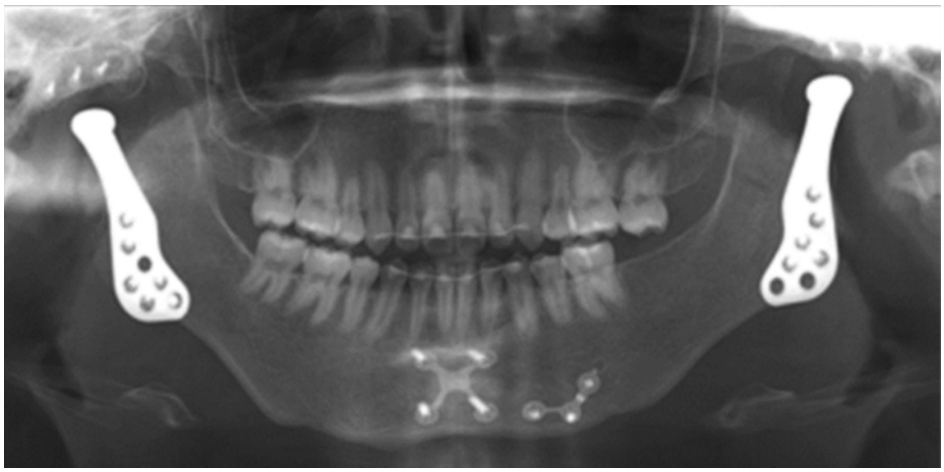

Fig. (12): Panoramic radiographs after temporomandibular joint reconstruction with alloplastic implants.

Surgical procedures

Patients with TMJ ankylosis always present with challenges of management of airway. The intubation process in TMJ ankylosis is difficult because of the possibility of failure, trauma and bleeding from the upper airway due to the distorted soft tissue anatomy. Furthermore, the intubation challenge in children results from unequal growth of the two halves of the mandible and reduced mandibular space with overcrowding of soft tissue. Three techniques of intubation that have been reported include blind nasal intubation, tracheostomy and fiber-optic intubation (14). Tracheostomy should be avoided if possible as complications with this technique

may be severe. Lately blind nasal intubation has been replaced with glide scope assisted intubation (34).

The surgical technique for release of TMJ ankylosis could be summarized in the following steps: -

Incision through the skin is limited to the preauricular and submandibular approaches that provide adequate exposure without injury to the facial nerve. These incisions (Fig. 13) include the hockey stick (35), T-shaped (18), L-shaped, horizontal or / and vertical incision (36), and Al kayat and Bramley (37). The submandibular approach is used for access to the lateral aspect of the ramus for fixation of the costo-chondral graft or alloplastic joint. Stripping of any attachment such as muscles and ligaments that attach the mandible to the cranium (masseter, medial pterygoid, stylomandibular and pterygomandibular ligaments) is then performed. The next step is aggressive excision of the fibrous and/or bony ankylotic mass, coronoidectomy on the affected and possibly unaffected side if coronoid processes are elongated. The unaffected side is treated if a maximal incisal mouth opening of 35 mm is not achieved during ankylosis release. Temporalis myofascial flap may be used as an interpositional material. In cases indicated for joint reconstruction, costo-chondral graft is harvested from 5th, 6th, or 7th rib. Rigid fixation of the graft is then performed with minimal inter-maxillary fixation for less than 10 days, and early mobilization of the jaw.

Prefabricated occlusal splint is used to improve the malocclusion after release of maxillo-mandibular wire fixation (MMF), it remains in place for approximately three months, without adjustment. The splint creates an open bite on the affected side to permit setting of the bone graft, and to allow over-eruption of the maxillary segment to improve the occlusal cant.

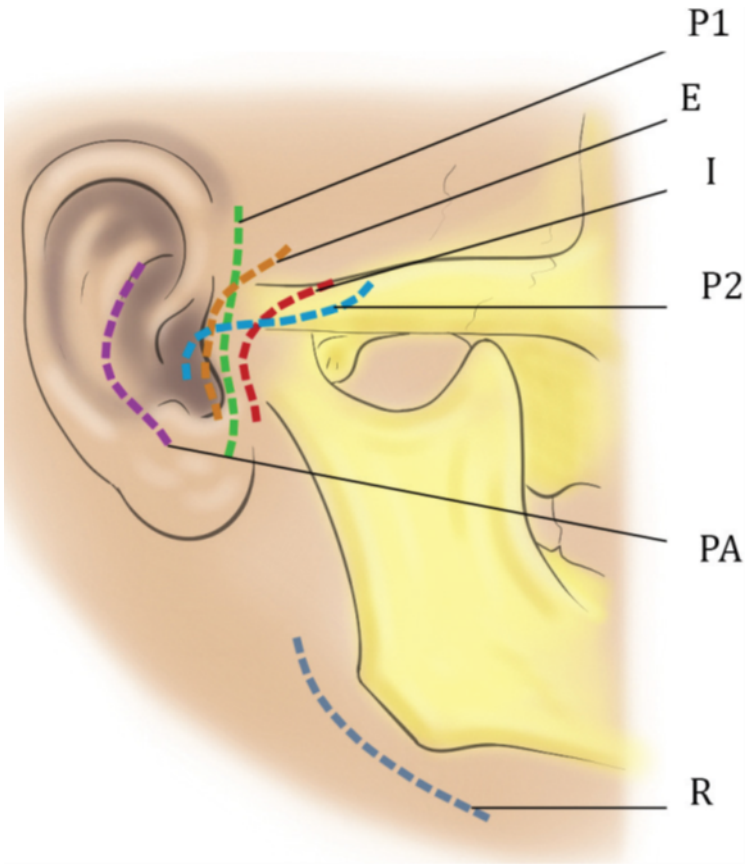

Fig. (12): Surgical approaches to the TMJ. P1 and P2- preauricular approaches; PA- postauricular approach; I- inverted hockey stick approach; E-endaural approach; R-retromandibular approach.

The major problem of surgical correction of the TMJ ankylosis is the high recurrence rate and the failure to maintain the result. Chossegros et al (38), reported that early physiotherapy and choice of interpositional material are important in preventing recurrence. The immediate postoperative period is the most critical time for successful treatment of TMJ ankylosis. Postoperative vigorous physiotherapy is required from the 2nd day, which includes passive and energetic exercises of the mouth opening at least once a day, and lasts several months until no further improvement is expected. Tongue blades (Fig. 14) that can be increased in number

each day according to the patient's tolerance are simple and reliable method for jaw exercise.

Fig. (14): Tongue blades used for jaw exercise regimen.

Distraction osteogenesis

Several reports (39,40), have demonstrated the feasibility of using distraction osteogenesis to correct mandibular deficiencies secondary to TMJ ankylosis (Fig. 15). Management of TMJ ankylosis could be single stage or two stage treatment. Single stage procedure involves performing distraction osteogenesis and ankylotic mass removal simultaneously. This could improve the restriction of mouth opening and maxillofacial deformity in one go, avoid second surgery, and reduce the economic burden of patients. However, the effect of distraction osteogenesis may be unsatisfactory because of the unstable condyle, there may exist interference between physical exercises and distraction and there still needs the second surgery to remove the distraction devices. Recently, transport distraction technique has been utilized to reconstruct a neocondyle without the need of bone graft (40). In two stage procedure, distraction osteogenesis of the mandible is performed as the initial surgery, followed

by a second stage surgery for arthroplasty or TMJ reconstruction. Since TMJ ankylosis frequently leads to more complex facial deformity which are not amenable by simple distraction procedure, some patients require orthognathic surgery to improve occlusion and facial profile along with or following arthroplasty or TMJ reconstruction. Few other surgeons persist that arthroplasty should be performed at the first stage to solve the problem of limited mouth opening, and distraction osteogenesis at the second stage (41).

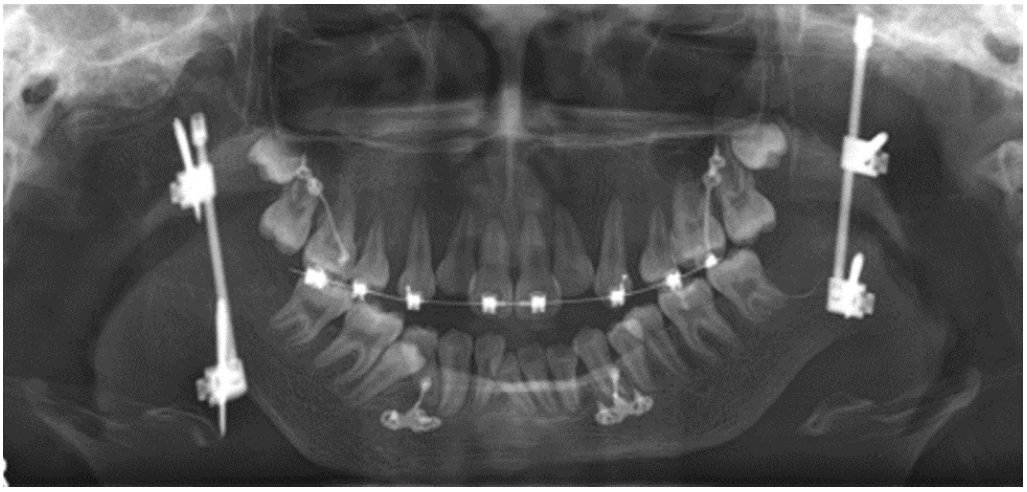

Fig. (15): Post-operative OPG at the end of distraction phase.

Current algorithm of treatment

Kaban et al. (4,42) proposed a management protocol for TMJ ankylosis consisting of aggressive resection, ipsilateral coronoidectomy, contralateral coronoidectomy when necessary, lining of the TMJ with temporalis fascia or cartilage, reconstruction of the ramus with a costochondral graft, rigid fixation, and early mobilization and aggressive physiotherapy. Since then, most authors have reported cases that followed the basic tenets of the principles for management suggested in that protocol. Disagreement still persists concerning the optimal reconstructive method utilized.

El-Sheikh (10), presented retrospective review of 204 patients with temporomandibular joint (TMJ) ankylosis treated according to a definitive protocol in the Cranio-Maxillo-Facial Department of the Alexandria University Hospital during the period 1990-1996 with a follow-up varying from 1.5 to 7 years. A history of trauma was confirmed in 98% of cases. Patients were grouped into: (1) Those with ankylosis not associated with facial deformities. The management involves release of the ankylosed joint(s) and reconstruction of the condyle ramus unit(s) (CRUs) using costochondral graft(s) (CCGs). (2) Those with mandibular ankylosis complicated by facial bone deformities, either asymmetric or bird face. The treatment consists of release of the ankylosis, reconstruction of the CRUs, and correction of jaw deformities-all performed simultaneously. Respiratory embarrassment was an important presenting symptom in the second group, all of whom complained of night snoring, eight of whom had obstructive sleep apnea. In this latter group, respiratory obstruction improved dramatically after surgical intervention. The degree of mouth opening, monitored as the interincisal distance improved from a range of 0-12 mm to over 30 mm in 62% of patients and to 20-30 mm in 29% of patients. However, re-ankylosis was still around 8% and was attributed to lack of patient compliance in 75% and to iatrogenic factors in 25% of patients. CCGs resorption, whether partial or complete, occurred in 27% of patients, resulting in retarded growth, relapse of deformities and night snoring.

In the study by Sadakah et al (43), there were 9 patients (5 males and 4 females), their ages ranging from 14 to 35 years (mean 19 years), presenting with TMJ ankylosis. Seven of them were unilateral and 2 bilateral. Six patients had recurrent ankylosis following previous failed surgery, while 3 were de novo cases. Pre and postoperative assessment of all patients included facial and occlusal evaluation, panoramic radiographs, frontal and lateral cephalograms, photographs and study models. Micrognathia, a deviated central line, canting of the occlusion and sleep

apnea were the main disorders in this study. In that study, patients were treated by a 2-stage surgical protocol: (1) bimaxillary distraction osteogenesis to correct the maxillary and mandibular deformities and (2) releasing of the TMJ ankylosis for mandibular motion at a later stage. Postoperative physiotherapy included gradual intraoral bilateral insertion of wooden spatulas (2-mm thick); 10 spatulas (20 mm) were used immediately after surgery, followed by daily number increase until an optimal mouth opening was attained.

The study by Elgazzar et al (44), reports the experience in managing TMJ ankylosis in Delta Nile, Egypt (1995 - 2006) and compares the surgical modalities used. 101 patients (109 joints) were reviewed in this retrospective study. Pre- and post-operative assessment included history, radiological and physical examination, and mouth opening. Age, sex, etiology, joint(s) affected, surgical modality, complications and follow up periods were evaluated. Various types (fibrous, fibro-osseous and bony) of TMJ ankylosis were diagnosed; trauma was the commonest etiology. The patients' age range was 2 - 41 years, 62% were female, and the follow up period ranged from 14 to 96 months. Average mouth opening was significantly increased from 5.3 mm pre-operatively to 32.9 mm 12 months postoperatively (P = 0.0001). Marked improvement in mouth opening was documented when the ramus-joint complex was reconstructed using distraction osteogenesis (34.7 mm), costochondral graft (34.4 mm) and Surgibone (34.6 mm). Gap arthroplasty showed least satisfactory mouth opening compared with other techniques (P = 0.001). Minor and major complications were encountered in 33% of cases, including 5% recurrence rate. Early release of TMJ ankylosis; reconstruction of the ramus height with distraction osteogenesis or bone grafting combined with interpositional arthroplasty, followed by vigorous physiotherapy is successful for managing TMJ ankylosis.

Hegab (45), proposed surgical protocol for treatment of temporomandibular joint ankylosis based on the pathogenesis of ankylosis and re-ankylosis. The protocol

consisted of the following 9 steps: 1) perioperative indomethacin for 2 weeks; 2) the creation of a minimal gap of 5 to 10 mm; 3) ipsilateral coronoidectomy and (if required) contralateral coronoidectomy; 4) pterygomasseteric sling and temporalis muscle release; 5) interpositional dermis fat graft fixed to the condylar stump; 6) insertion of a suction drain; 7) immediate aggressive physiotherapy for at least 6 months; 8) regular long-term follow-up; and 9) delayed reconstruction using distraction osteogenesis. He discussed that medium to high doses of perioperative nonsteroidal anti-inflammatory drugs (indomethacin) clearly produce substantial decreases in the incidences of the recurrent formation of heterotopic bone and recurrent ankylosis. The study comprised 14 patients (3 male and 11 female) with TMJ ankylosis. Of these patients, 9 and 5 exhibited unilateral and bilateral ankylosis, respectively, and their ages ranged from 12 to 38 years (median, 18.5 yrs.). The follow-up period ranged from 24 to 48 months (mean, 32.5 months). The final outcome results showed a positive coloration with the magnitude of ankylosis; cases with a greater degree of ankylosis and cases with bilateral ankylosis were associated with a decrease mouth opening in final outcome and an increase of the degree of relapse. Moreover, these cases were associated with a longer operating time, more postoperative edema, and temporary mild facial nerve paresis (grade II). He concluded that the surgical protocol appears to be effective in the treatment of TMJ ankylosis and the prevention of re-ankylosis.

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Chapter 8: Condylar hyperplasia and hypoplasia

Condylar hyperplasia

Condylar hyperplasia (CH) is a rare disorder characterized by excessive bone growth that usually presents unilaterally, resulting in facial asymmetry (1). In many cases, occlusal discrepancies and temporomandibular joint disorder are concurrent symptoms with facial asymmetry. Current research has yet to define an exact etiology for CH. However, several causal factors have been reported, such as post-trauma, mandibular condyle hyper-remodeling, joint infection, hormonal disorders, mandibular condyle hypervascularization, intrauterine changes and genetic factors (2).

Diagnosis

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. 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 (3). The patient's main complaint is paramount, and their concerns and expectations should be central to the plan. Aesthetic and functional problems should be recorded. Progressive asymmetry of the lower face that presents during the pubertal growth spurt supports a diagnosis of condylar hyperplasia. Asymmetry that has been present since birth probably suggests another disease, and photographs may help. A full medical and social history should be taken, as with any surgical patient.

Plain radiographs, such as panoramic (Fig.1), frontal (Fig.2) and profile teleradiography, are useful to evidence skeletal changes typical of mandibular condylar hyperplasia. However, decisions about management require more detailed anatomical and functional imaging. Lateral cephalometry is of limited value, as

bilateral structures are superimposed. Computed tomography (CT) provides detailed anatomical information (Fig.3) and can be reformatted into 3-dimensional images (Fig.4) for use in surgical planning (4). Nuclear imaging is capable of providing details of bone metabolism and activity. SPECT is an essential diagnostic tool to assess active growth (Fig.5). In this quantitative method, 99mTc-MDP is injected and absorbed into hydroxyapatite crystals and calcium in the bone (5). The bone is then scanned using the SPECT technique, and the hyperplastic condyle is quantitatively compared to the contralateral side. Differences greater than 10% between two condyles are considered to indicate active growth due to CH.

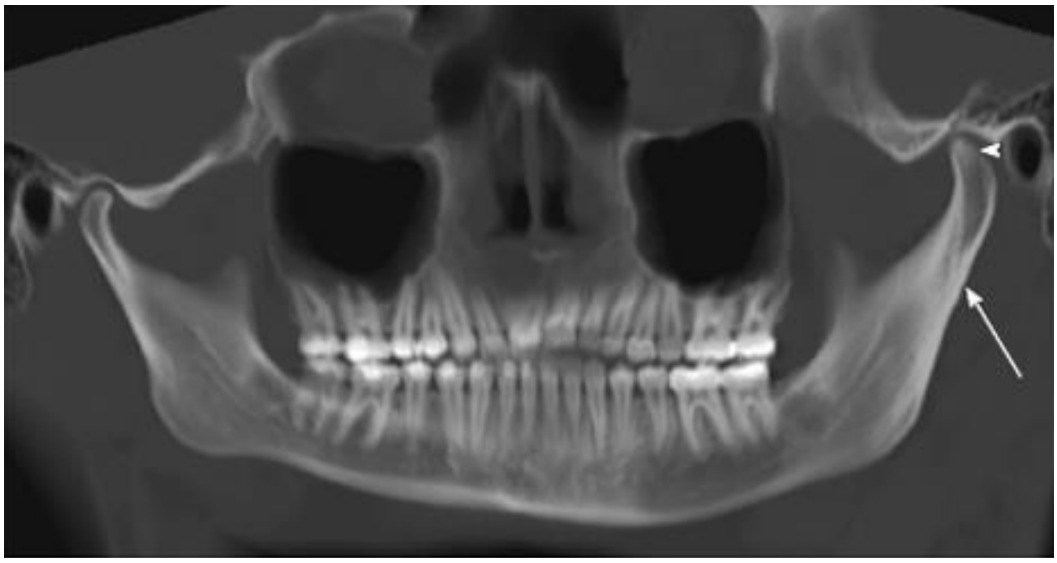

Fig. (1): Panoramic reformation of computed tomography demonstrates hyperplasia of the left condyle (arrowhead) in comparison to the right side. Associated hypertrophy of the ramus and the neck (arrow) of the left hemi-mandible is also noted.

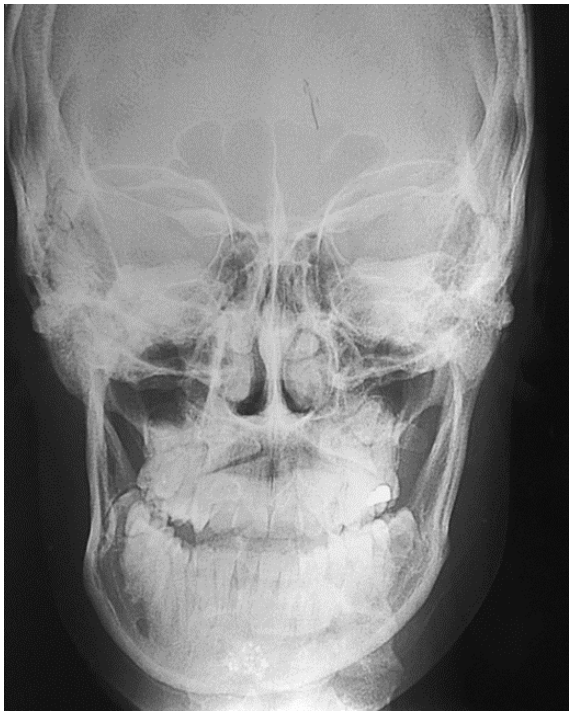

Fig. (2): Skull P-A showing elongated condyle and thickened mandibular rami on the left side.

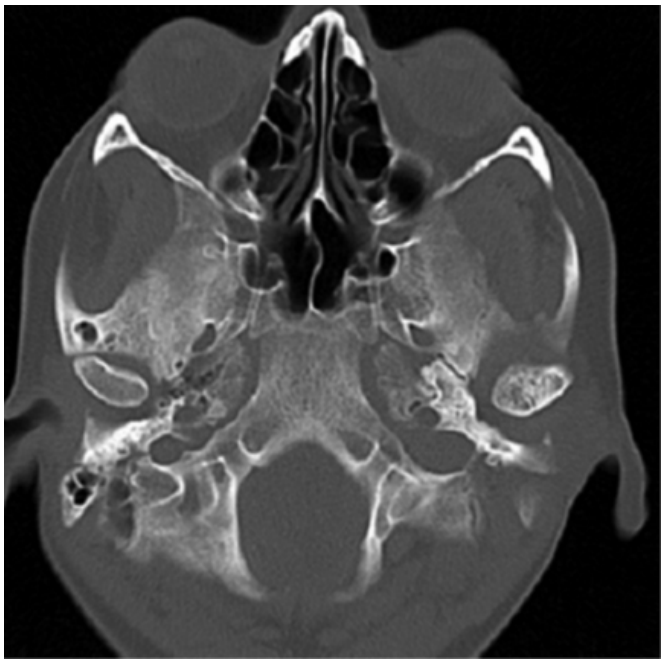

Fig. (3): Axial CT exhibiting hyperplastic left condyle.

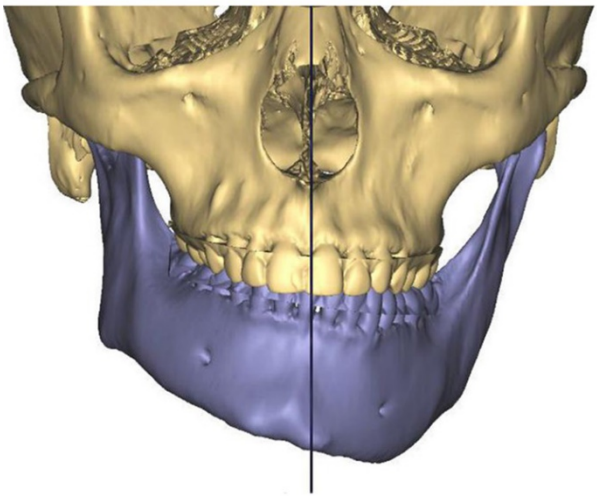

Fig. (4): 3-dimensional virtual reconstructions of patient with left-sided condylar hyperplasia.

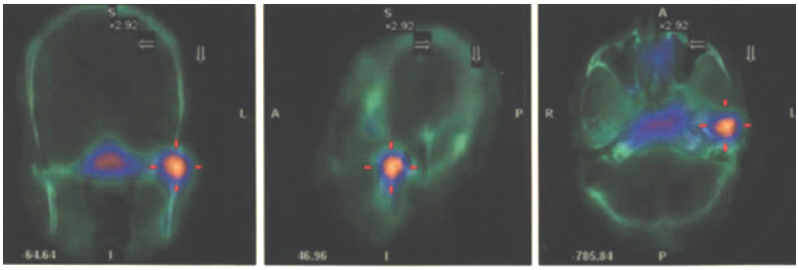

Fig. (5): Bone scan: the isotope uptake demonstrates the active nature of the condylar hyperplasia.

Classification and clinical characteristics

Several classifications have been proposed for CH. Obwegeser and Makek (6), developed a classification system based on the asymmetry and predominant growth vector. In their article, they classified CH into 3 different categories. They defined Type 1 as hemimandibular elongation, with excessive growth displayed in the horizontal vector. Type 1 CH is associated with chin deviation toward the unaffected side, with no corresponding vertical asymmetry. Due to the overgrowth, the mandibular midline is also shifted to the contralateral side. As a result, the contralateral mandibular molars often deviate lingually in order to remain in proper occlusion with the maxillary molars. If the contralateral molars are unable to adapt

to the growth, a crossbite may develop. Normally, the condyle is unaffected, but the neck is often misshapen and slender. The ramus is elongated, which is the basis for referring to Type 1 as hemimandibular elongation. Type 2 CH was defined as hemimandibular hyperplasia, which is associated with excessive growth in the vertical vector. It is often characterized by bowing of the inferior border of the mandible, obliteration of the antigonial notch, and minimal chin deviation. Due to the excessive downward growth of the mandible, the maxillary molars on the affected side compensate by following the mandible's downward growth. The maxillary alveolar bone on the ipsilateral side grows excessively to maintain occlusion. If the maxillary molars are not able to follow the excessive downward growth, an open bite on the affected side results (Fig.6) In Type 2 CH, the condyle often appears enlarged, and the head is usually irregular or deformed. The neck of the condyle has also been reported as thickened and/or elongated. Type 3 CH is a combination of Types 1 and 2. Nitzan and colleagues (7), 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.

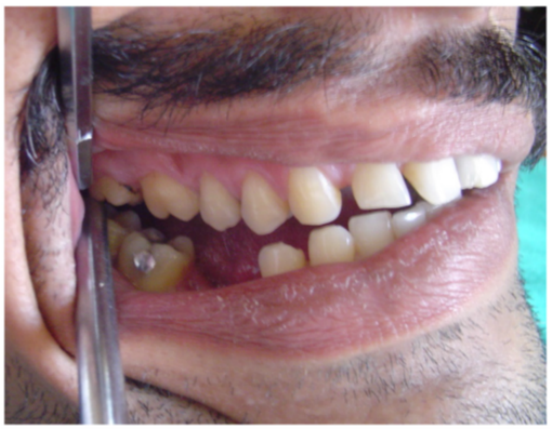

Fig. (6): Intraoral photograph showing posterior open bite.

Wolford et al. (8), developed an updated classification system that they considered more inclusive of pathologies causing CH. 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. Type 1 is characterized by an accelerated and prolonged growth that causes condylar and mandibular elongation and split into 1A and 1B. CH Type 1A is defined as mandibular elongation that occurs bilaterally, while CH Type 1B occurs unilaterally. It generally appears in subjects in the growth phase, mainly in adolescence or young adults (7). CH Type 2 consists of unilateral overgrowth of the condyle caused by an osteochondroma and results in vertical overgrowth of the mandible, and accompanying compensatory downward growth of the maxilla. It has been estimated that females can present with the active pathology more frequently than males (9). Wolford et al. (10), further classified CH Type 2A and B. Type 2A results from vertical elongation of the condylar head and neck. Type 2B involves horizontal exophytic tumor growth of the condyle in addition to vertical elongation of the head and neck. CH Type 3 consists of other benign tumors that cause CH, including but not limited to osteomas, neurofibromas, and fibrous dysplasia, and results in unilateral facial enlargement. Type 4 CH is caused by malignant tumors that originate in the condyle and cause enlargement and facial asymmetry. Some malignant tumors attributed to Type 4 CH include chondrosarcoma, multiple myeloma, osteosarcoma, and Ewing sarcoma. Generally, tumors are rare, but osteosarcoma and chondrosarcoma do present. Metastatic carcinoma is the most frequently occurring malignancy; however, the jaws are an uncommon site for metastasis, especially the mandibular condyle. When considering benign tumour, osteochondroma is the most common, but this is still very rare. Features that may suggest the possibility of a tumour in the TMJ include pain, swelling, paresthesias,

trismus and occlusal changes. There may rarely be auditory changes secondary to eighth nerve involvement.

Treatment

Treatment is essentially surgical. The treatment options supported by the literature are: 1) high condylectomy of the condyle with CH, 2) high condylectomy of the condyle with CH and bimaxillary orthognathic surgery, 3) orthognathic surgery of the residual facial deformity or 4) cosmetic procedures associated with orthodontic compensation.

Treatment considerations of CH type 1

CH type 1 is self-limiting relative to growth; patients in their mid-20s or older will not have further jaw growth related to CH type 1, so routine orthognathic surgical procedures can usually be performed to correct the dentofacial deformity and malocclusion. If active growth is confirmed, then there are 2 options for treatment.

Option 1

The surgical protocol for active CH type 1 consists of (Fig.7): 1- Bilateral or unilateral (depending if type 1A or 1B) high condylectomy (4–5 mm of the top of the condylar head), including the medial and lateral pole areas. 2- Disc repositioning, using a bone anchor. 3- Orthognathic surgical procedures, often requiring double jaw surgery to optimize the functional and esthetic outcomes. The series of patients in the study by Wolford et al. (10), showed that the high condylectomy in conjunction with orthognathic surgery was acceptable to treat the functional and esthetic problems. In addition, it indicated that performing orthognathic surgery without condylar treatment would not limit subsequent condylar growth, and that recurrence and new surgical procedures were possible. This protocol predictably stops mandibular growth and provides highly predictable and stable outcomes.

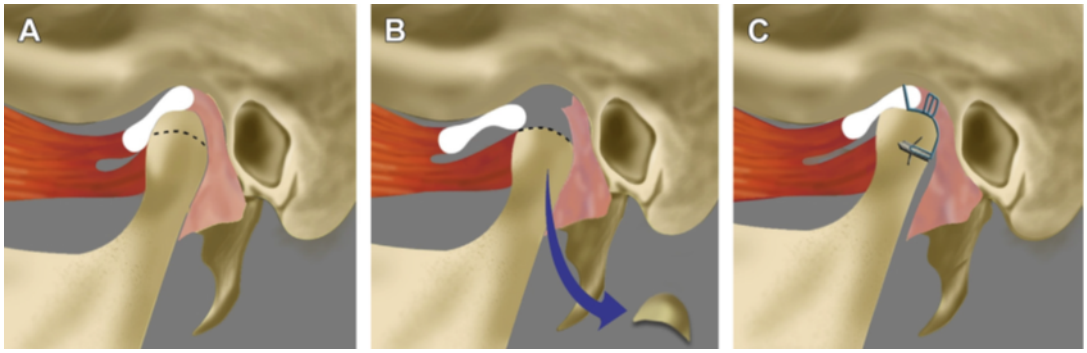

Fig. (7): For CH type1 in active growth, a high condylectomy (A) will arrest any further mandibular growth. The articular disc is repositioned and stabilized on the condyle (B,C) with a bone anchor.

Option 2

Surgery is delayed until growth is complete, which could be in the early to mid-20s, and then only orthognathic surgery is performed. However, the longer the abnormal growth is allowed to precede, the worse the facial deformity, asymmetry, occlusion, and dental compensations will become, in addition to warping of the mandible and ipsilateral excessive soft tissue development. This will increase the difficulties in obtaining optimal functional and esthetic results, in addition to the adverse effects on occlusion, dental compensations, mastication, speech, and psychosocial development (8). It is recommended to delay surgery until the age of 15 for girls and 17 for boys, when most of the normal facial growth is complete. A unilateral high condylectomy will arrest growth on the operated side, but normal growth can continue on the contralateral side and could cause development of facial and occlusal asymmetry later if the surgery is performed at a younger age (10).

Treatment considerations of CH type 2

In the presence of joint tumors, condylectomy will be essential. Low condylectomy to remove the tumor in its entirety allows completely removing hyperplastic tissues and preventing the recurrence of the deformity. Shankar et al. (11), stated that with low condylectomy the results are predictable and stable because it allows to remove

all remnants of fibrocartilage from the condyle, especially in those cases that present condylar hyperplasia due to a tumor (osteoma, osteochondroma). Surgery (Fig.8) would include the following steps (12): 1- Low condylectomy to remove the tumor in its entirety, 2- Reshape the condylar neck, 3- Reposition the articular disc over the remaining condylar neck, 4- An ipsilateral sagittal split osteotomy is then performed, and the disc/condylar stump complex is seated into the fossa, 5- If indicated, orthognathic surgery is performed to correct the maxillary and mandibular asymmetries, 6- If needed, inferior border ostectomy can be performed on the involved side to reestablish vertical balance of the mandible. The risk of recurrence of this benign lesion is low after surgical removal (13).

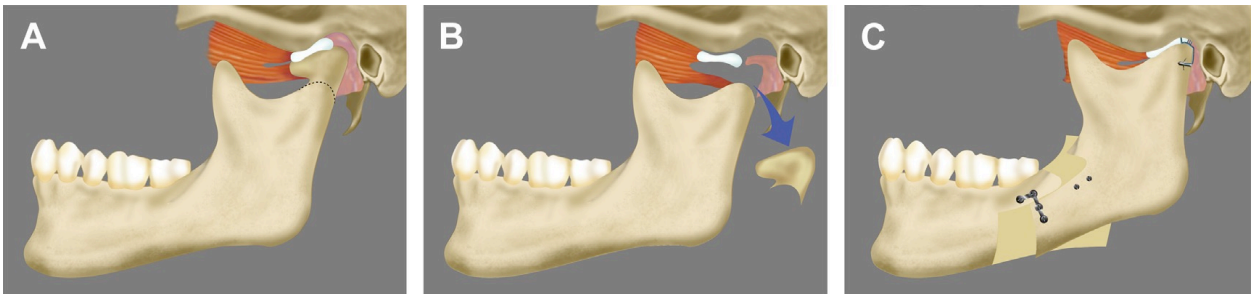

Fig. (8): Treatment of CH type 2 includes a low condylectomy (A), the condylar neck is recontoured (B), the articular disc is repositioned with a bone anchor; and sagittal split osteotomies are completed (C).

Condylar hypoplasia

Condylar hypoplasia is a bone disease characterized by the underdevelopment of one or both the mandibular condyles (14). It presents as progressive facial asymmetry which is usually asymptomatic. Condylar hypoplasia may be congenital or acquired and each of these may present as unilateral or bilateral entity (1). Congenital condylar hypoplasia is idiopathic in origin, presents at birth and is often associated with various craniofacial abnormalities. The most commonly associated syndromes are Hemifacial microsomia, Treacher Collins syndrome and Hallermann-Steiff

syndrome (15). Acquired condylar hypoplasia may be caused by local or systemic factors. The most common causes are mechanical injury, such as trauma, infection of the joint itself or the middle ear, childhood rheumatoid arthritis, radiotherapy, and parathyroid hormone-related protein deficiency which affect bone formation and chondrocyte differentiation (15,16). Acquired condylar hypoplasia may develop after the loss of one or both condylar growth centers in very early stages of life and is sometimes accompanied by ankylosis. Several authors confirmed that mandibular deficiency can occur without any defined etiology (17). When an affected side fails to grow downward and forward, a three-dimensional asymmetry is produced. The mandibular skeletal midline deviates to the affected side, a lack of vertical growth on the same side produces a cant of the occlusal plane and mandibular retrognathia is seen as a result of the hypoplasia. The severity of the deformity depends on the degree of hypoplasia or agenesis of the tissue involved, and the more severe the deformity the greater is the probability that it will worsen with growth (18).

Diagnosis

The diagnosis is arrived at by a correlation of the clinical findings with the radiological findings. Conventional radiographs, and computed tomographic images (Figs.9-12) usually reveal a poorly developed condylar head associated with a short condylar neck, an overall decrease in the height of the ramus of the mandible and prominent antegonial notch. Plain film radiography is generally inadequate for assessing disorders of the TMJ. Three-dimensional imaging in form of conventional CT or cone beam computed tomography (CBCT) must be considered in the investigation of the osseous morphology of the temporomandibular joint.

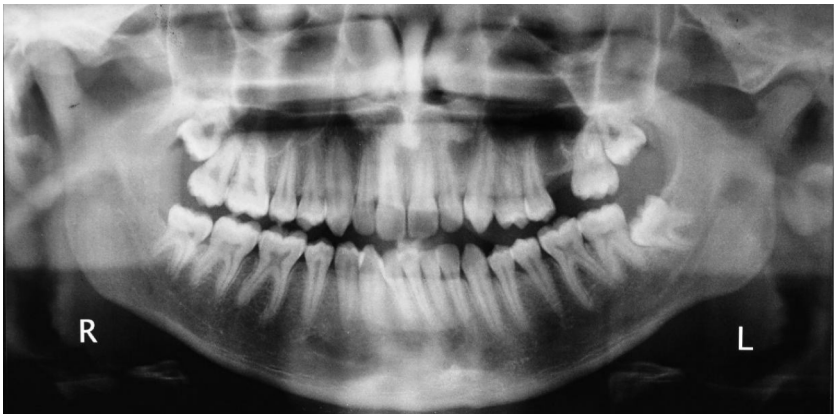

Fig. (9): Panoramic radiograph showing a poorly developed left condylar head associated with a short condylar neck, an overall decrease in the height of the ramus, and prominent antigonial notch.

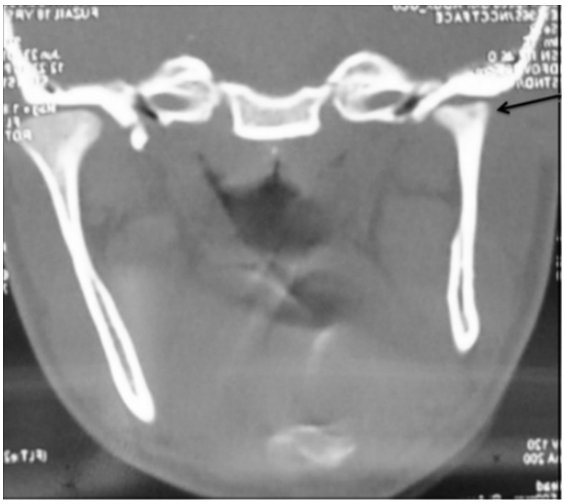

Fig. (10): Coronal CT showing a significant reduction in the mediolateral width of the left condyle as compared to the right one.

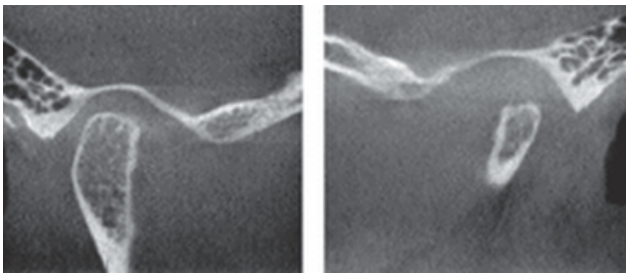

Fig. (11): Corrected sagittal CBCT sections of left condylar hypoplasia compared to normal right condyle.

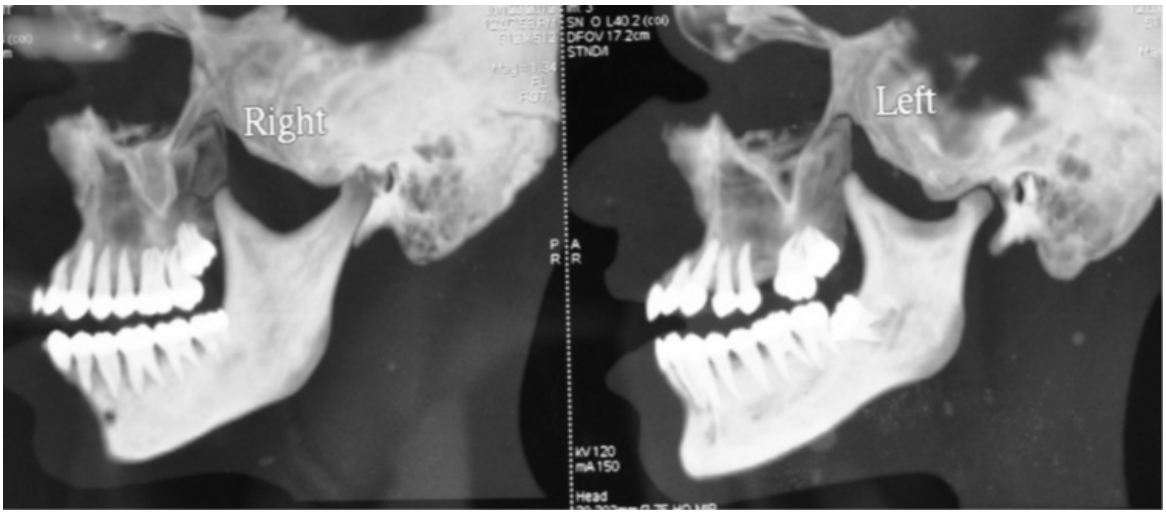

Fig. (12): Three-dimensional reconstruction CT in the sagittal plane comparing right (normal) and left (hypoplastic) side of the mandible.

Treatment

The treatment modalities for mandibular condylar hypoplasia vary dependent on the age of the patient. In growing patients, orthopedic treatment with functional appliances is often helpful in correcting deformities or in reducing the worsening of deformities with growth. If the facial asymmetry develops progressively during orthopedic treatment, mandibular distraction osteogenesis or surgical reconstruction of the TMJ with a costochondral graft of the remaining ramus tissue may be considered. After the patient growth spurt is completed, skeletal deformities can be corrected only by double jaw surgery and/or genioplasty or unilateral mandibular augmentation (18).

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