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Imaging of the Temporomandibular Joint

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Introduction

The temporomandibular joint (TMJ) is a unique structure as it is the only diarthroidal synovial joint in the body. It is continuously loaded, even when not functioning, and needs to be biomechanically sound in order to withstand the high mechanical loading that it is subject due during function. This article will discuss the anatomy and function of the TMJ and the methodology of investigating the breakdown of its biomechanical integrity radiographically and some of the less common but important pathology will be demonstrated. Trauma to the TMJ and developmental anomalies are not included in this article.

Anatomy and Function

The TMJ is a complex structure that affects the growth, development, and maintenance of many of the components of the craniofacial complex.[1,2] The TMJ is made up of extracapsular components, such as the capsule, ligaments, nerves and vessels, and these will not be covered in this article as they are not readily demonstrable on TMJ imaging. The intracapsular components (Fig. 1) can be divided into osseous components and soft tissue components.

The osseous components are (1) *the mandibular condyle* and (2) *the glenoid (mandibular) fossa* and *articular eminence*. These osseous components are best evaluated with cross-sectional hard-tissue imaging, such as cone beam CT (CBCT) or CT. CBCT is preferred as there is less radiation associated with this modality and the ability to generate submillimetric custom cross sections from the same volume facilitates the evaluation for early breakdown of the articular surface.

The soft tissue components are (1) the *TMJ disc* (sometimes erroneously called "the meniscus") which is made up of a thick *posterior band* and *anterior band* and a thin *intermediate zone* and, (2) the *disc attachments*, which are made up of (a) the posterior attachments: the *temporal posterior attachment* (TPA) or *superior lamina*, the *condylar posterior attachment* or *inferior lamina* and the *intermediate posterior attachment* (the area where the TPA and the condylar posterior attachment meet and attach to the posterior band of the disc), and (b) The *medial and lateral collateral ligaments*, which attach the medial and lateral aspects of the disc to the neck of the condyle. On an axially corrected sagittal view of a normal TMJ in the closed mouth position, the posterior band of the disc should sit at the 12 o'clock position on the condyle. The intermediate zone of the disc is interposed between the convex articular surfaces of the condyle and the posterior slope of the eminence, and the anterior band sits on top of the superior belly of the lateral pterygoid muscle. In this view, the disc is biconcave and resembles a bowtie (Fig. 1b). On an axially corrected coronal view, the disc posterior band should resemble a crescent sitting evenly on top of the condyle that tapers toward the condylar poles (Fig. 1c). In the open mouth position, a normal TMJ disc will translate anteriorly and inferiorly on the posterior slope of the eminence. The portion of the disc between the articular surfaces should be the junction between the anterior band and the intermediate zone, and the condyle should be positioned inferior to the level of the articular eminence[3] (Fig. 2).

There are two joint spaces. The space between the disc and the condyle is called the *inferior joint compartment* and the space between the disc and the fossa is the *superior joint compartment*. The joint compartments have anterior and posterior recesses lined by synovial lining that secretes synovial fluid into the joint spaces providing lubrication for the joint. The space posterior to the disc and condyle is called the "*retrodiscal tissue*" or the "*bilaminar zone*" which is very rich in vessels and nerve endings.

The articular surfaces are lined by *fibrocartilage*. The fibrocartilage has several functions. In a growing individual it is a primary growth site for the condyle and the mandible and the healthy proliferation of its undifferentiated mesenchymal cells is key for the growth and development of the mandible and, subsequently, the face. If this thin layer of cells is damaged prior to the cessation of growth (due to trauma,

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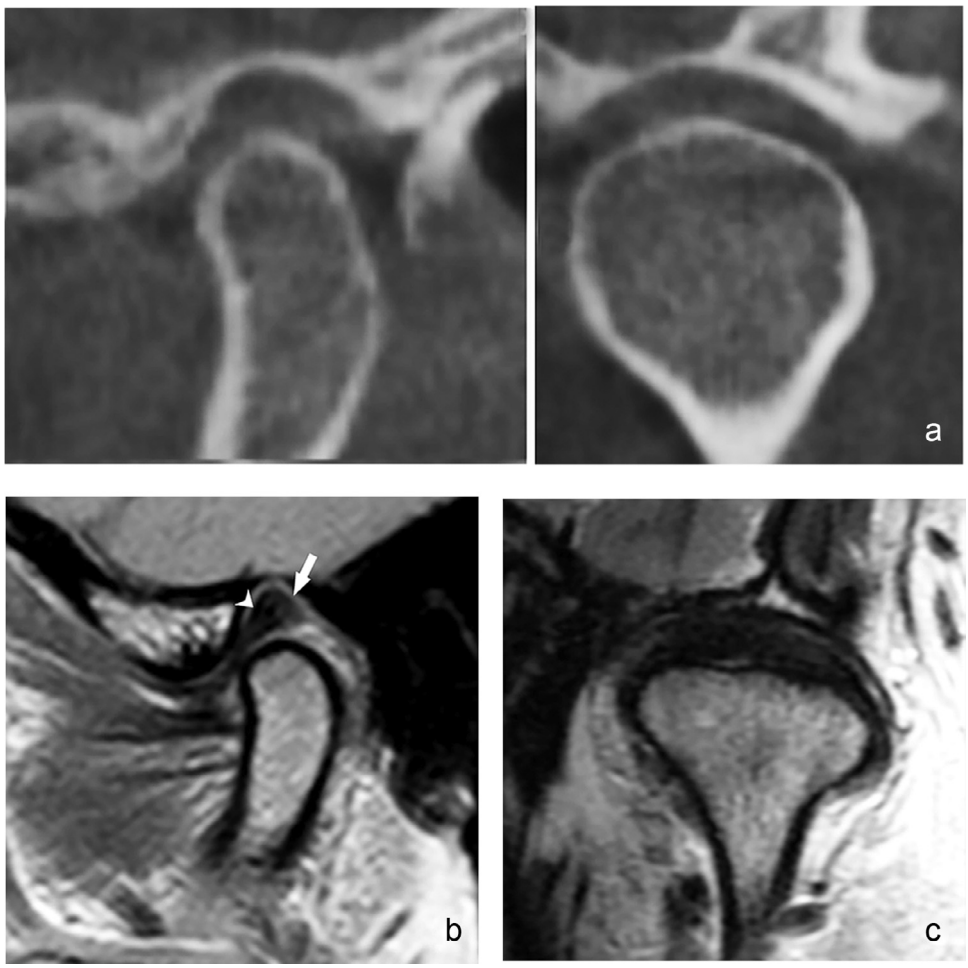

Figure 1 CBCT and MRI Axially corrected sagittal and coronal views show the normal anatomy of the TMJ. (a) On CBCT or CT: The osseous components are rounded and form an incongruent articulation. The condylar articular cortex is very thin in an adult condyle. The marrow spaces are uniform and not sclerotic. The normal disc space in the sagittal plane should reflect the bow-tie shaped disc that is interposed between the articular surfaces, and on the coronal should reflect the crescent shape of the posterior band of the disc. (b) Sagittal MRI, the posterior band of the disc sits at the 12 o'clock position on the condyle (arrowhead). The intermediate zone is between the incongruent articulations and the anterior band sits on the lateral pterygoid muscle fibers. The IPA (white arrow) is attached to the posterior band of the disc. The marrow spaces are homogenous and are bright on T1 and intermediate on T2. The cortical outlines are uniform and continuous. (c) On the coronal MRI, the posterior band of the disc resembles a crescent and sits evenly superior to the condyle, tapering toward the poles and attaching to the neck of the condyle with the lateral and medial collateral ligament.

inflammatory or a degenerative process) mandibular and facial growth will be compromised. If the damage to the cell layer occurs unilaterally on one condyle facial asymmetry will result (Fig. 3). If this happens bilaterally then not only is the facial profile more convex due to resulting posterior rotational growth of the mandible but also there is an increased risk for sleep-disordered breathing as a result of the posterior repositioning of the mandible and the attached soft tissues of the tongue into the oropharyngeal airway (Fig. 4).[4-6]

Thus lies the importance of reviewing the TMJ as a part of a whole complex and not just as a separate and individual structure. CBCT allows for a comprehensive review of the effect of the TMJ function and dysfunction on the facial skeleton, skull base, dental occlusion, and upper respiratory tract. Magnetic resonance imaging (MRI) will give adequate information on the soft tissue and osseous components of the TMJ, but to obtain the full diagnosis for the patient's TMJ condition one must observe the entire craniofacial complex as a whole if possible.

Imaging Modalities and Protocols

There are several options available for the imaging of the TMJ. Some plain film methods that were used in the past such as the transcranial, Towne, submentovertex, and lateral tomographic projections have been replaced with three-dimensional imaging that offers more accurate information

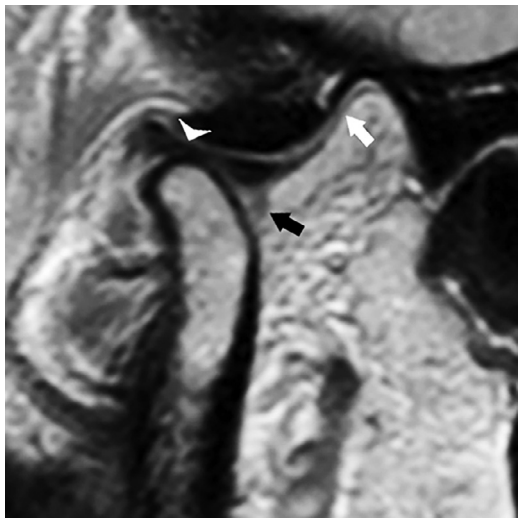

Figure 2 T1WI MR of the TMJ in the open mouth position shows the normal position of the disc in relationship to the osseous components. The junction between the intermediate zone and the anterior band (arrowhead) should lie between the articular surfaces. The TPA (white arrow) and the CPA (black arrow) are better visualized due to the more intense signal in the retrodiscal tissue with the dilatation of the blood vessels in this area upon opening.

CPA, condylar posterior attachment; TMJ, temporomandibular joint; TPA, temporal posterior attachment.

on the morphology of the tissues and as well as their spatial relationships. These plain films techniques have been described in the literature[3] and can be used if other more advanced imaging modalities are not available, but these are not discussed in this article. A panoramic projection can offer general information about the osseous components, but the TMJ spatial relationships cannot be accurately assessed as the teeth are in a protrusive position during acquisition to bring both jaws into the focal trough. Cone beam CT offers a comprehensive view of not only the osseous components of the TMJ but also the effect of the TMJ condition on the rest of the craniofacial complex. This includes analysis of asymmetry (enlargement or decrease in size of one side), growth changes, occlusal (bite) changes, neck posture, and risk factors for sleep-disordered breathing. To evaluate all of these, a 16 cm or more field of view is required to visualize all these structures and their interaction with one another in one volume. The teeth should be in maximum intercuspation and the head posture should be as close to natural head posture as possible. Custom cross sections of the TMJs can be generated from the CBCT volume. Some standard reformations that help visualization of the TMJs and the craniofacial complex are the TMJ sagittal and coronal oblique sections, TMJ axial view, panoramic reformation, 3D frontal and lateral reformations, and airway morphologic and volumetric

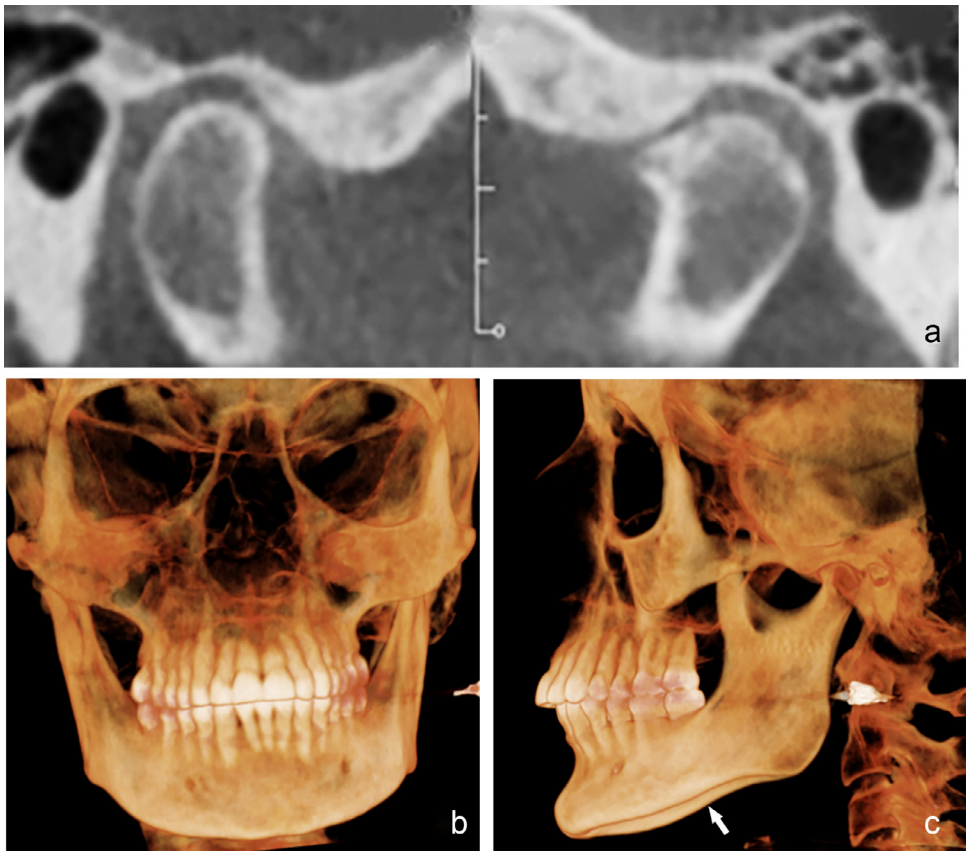

Figure 3 These 3D renderings show mandibular asymmetry, with the left side being smaller than the right due to the smaller condyle on the left side, as seen on the cross sections (a). When superimposing the two sides of the skull base and zygomas on the lateral view, the inferior border of the left side of the mandible appears higher than the right (c, arrow). Changes in the size and height of one condyle, regardless of the cause (developmental, degenerative or inflammatory) will result in mandibular asymmetry.

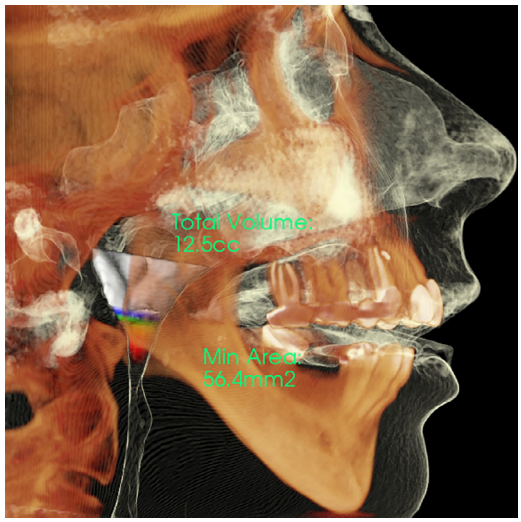

Figure 4 This 3D rendering of cone beam CT data shows some of the craniofacial features that occur when there is bilateral condylar height loss (regardless of the etiology): Posterior rotation of the mandible which leads to the posterior displacement of the soft tissue of the tongue, narrowing the oropharyngeal airway, anterior open bite, a long and convex facial profile and a steep mandibular plane (inferior border of the mandible) (Courtesy M. Noujeim DDS).

analysis. These reformation functions are readily available in most dental DICOM viewing software. CT-generated images can serve the same purpose, but as the radiation is higher with CT, CBCT should be used when available as it will provide the same information at less radiation exposure to the patient.[6]

MRI will offer insight to the position and condition of the soft tissue components of the TMJs. The disc and its attachments are made up largely of collagen which contrasts against the synovial fluid and vessel-rich retrodiscal tissue on proton density (PD) and T2-weighted imaging. The bone marrow integrity can be examined and the morphology of the osseous components assessed, but the fine changes that occur with early active degenerative joint disease are better visualized with CBCT. The presence of a joint effusion and the evaluation for the presence of soft tissue neoplasia or neoplastic-like lesions are best assessed with MRI. The protocols commonly used are T1 (or PD) and T2 (and/or STIR) in the closed mouth (maximal intercuspal) position and the open mouth position. The diagnostic cross sections to be obtained are the axially corrected sagittal oblique and coronal oblique cross sections along the long axes of the condyles and perpendicular to them. The closed mouth position will demonstrate the position and condition of the disc and other soft tissues with the teeth together in maximum intercuspation to determine the presence of a disc displacement. The open mouth position will determine if a displaced disc was recaptured by the condyle upon opening. The spectrum of internal derangement of the discs that can be evaluated on MRI will be discussed in the following section.[5,6]

Imaging of TMJ Pathology

Internal Derangement and Degenerative Joint Disease

Internal derangement (changes in disc morphology or position) can occur in the TMJ and may be caused by trauma, malocclusion, laxity of ligaments, or other conditions that may force the disc out of position. Once the disc is out of position the breakdown of the normal joint function begins.[5-7] The following will describe the sequence of the internal derangement into degenerative joint disease, as well as the variations of these derangements and stages of degenerative joint disease.

Disc Displacement Degree

A disc displacement may be partial or complete. In a partially displaced disc, the disc morphology changes to either a biplanar appearance (flattening of the posterior band) or biconvex (thickening of the posterior band, usually seen with posterior position of the condyle) (Fig. 5). A completely displaced disc that has been acutely displaced maintains its biconcave appearance (Fig. 6), but may gradually lose its shape and anteroposterior length and may eventually atrophy (Fig. 7).

Disc Displacement Directions

Depending on the altered biomechanics of the TMJs, the disc displacement can occur in various directions. The most commonly seen is the anterior disc displacement (Fig. 8). Other variations include anterior rotational (slippage of the disc off of only one pole while maintaining normal relationship with the other portions of the condyle) (Fig. 9), sideways (displacement in the purely lateral or medial direction) (Fig. 10), or posteriorly (which should be differentiated from the thickening of the intermediate posterior attachment, also called "pseudodisc formation" Fig. 11).

Disc Displacement With and Without Reduction

When the disc recaptures into its normal position upon opening, it is "reduced." In the closed mouth position, the disc will be displaced in a specific direction. Upon opening the mouth to maximal opening, the disc returns to its normal open mouth relationship to the condyle, only to become displaced again upon closing the mouth. When the disc is recaptured, there is usually an audible click or pop associated with the reduction (Fig. 8). In disc displacement without reduction, the disc remains displaced throughout the entire mouth opening cycle and, in the acute phase, is painful and associated with a closed lock, but range of motion is restored and symptoms subside as time progresses and the tissues adapt (Fig. 12).

Joint Effusion and Bone Marrow Edema

Synovitis and subsequent joint effusion can best be visualized on MRI T2 sequences and appears as areas of hyperintensity. The fluid accumulation can occur in only one compartment or both, creating an "arthrographic effect" where the fluid highlights the disc shape[8,9] (Fig. 13). The

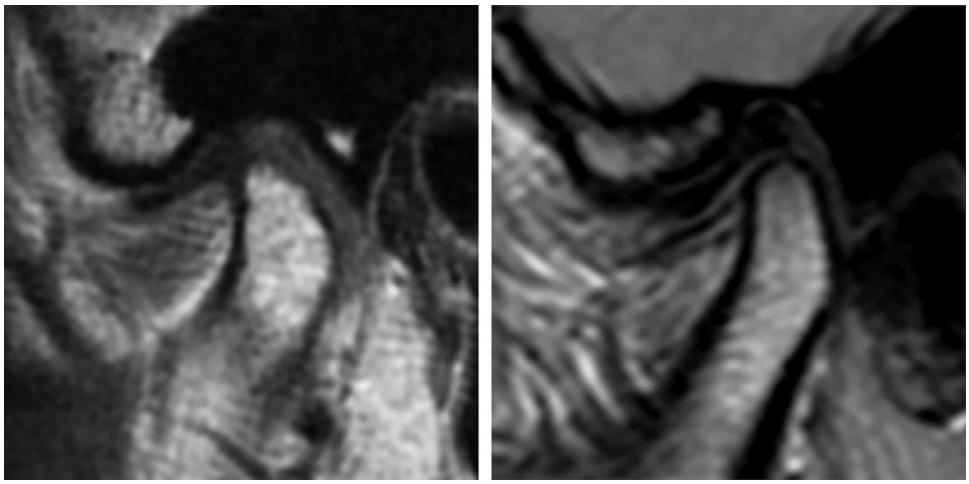

Figure 5 PD MR axially corrected sagittal cross section shows posterior position of the condyle in the fossa and the posterior band of the disc position at the 10 o'clock position to the condyle. The condyle position should be assessed in relation to the fossa and not just the disc relation to the condyle.

fluid can show a perforation of the disc or attachments if the effusion is only in one joint compartment, with the fluid seeping into the other compartment (Fig. 13). The fluid can also be seen shifting within a compartment upon opening, and can be used to visualize detachment of the TPA from the glenoid fossa (Fig. 13). In acute micro- or macro-trauma, the bone marrow may become edematous, which can present as high signal intensity subchondral bone on T2-weighted images that is intermediate to low signal on T1 or PD-weighted images.

Disc Adhesion and Subluxation

Disc adhesions can occur secondary to microtrauma or microtrauma to the TMJ. Fibrous attachment of the disc to either the superior compartment (more common and more limited range of motion) or in the inferior compartment can occur. In superior compartment adhesions, the condyle translates upon opening but the disc does not change position when compared to the closed position (Fig. 14a and b).

In subluxation, the disc and condyle translate more anteriorly than the normal position and the condyle is positioned anterior and superior to the crest of the eminence and the portion of the disc between the osseous components is the anterior band, which often sits between the posterior aspect of the condyle and the anterior slope of the eminence (Fig. 14c). This hypermobility can be seen in patients with joint laxity and Ehler's Danlos syndrome.

Degenerative Joint Disease

Degenerative joint disease (DJD), also known as osteoarthritis, is a non-inflammatory degenerative process that may occur in the TMJ. It may occur in a single joint or bilaterally.

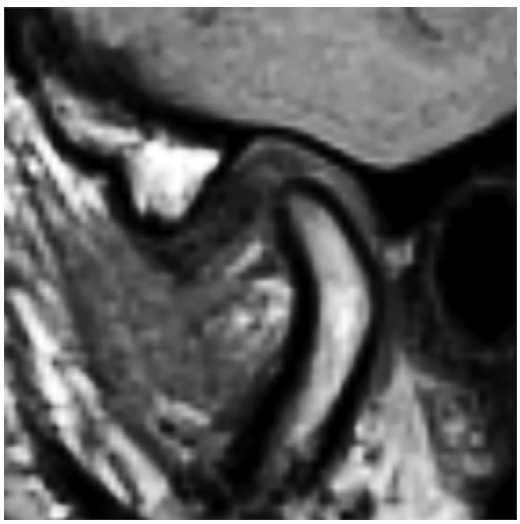

Figure 6 T1 MR axially corrected sagittal cross section shows that an acutely displaced disc maintains its anteroposterior dimension and its biconcave appearance. The condyle is posterior displaced.

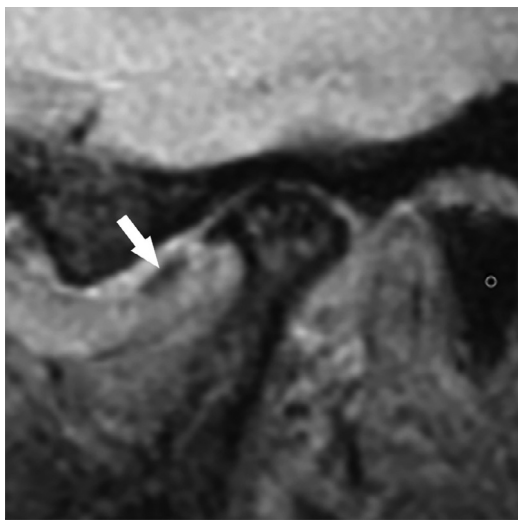

Figure 7 T1 MR axially corrected cross sections shows end-stage degenerative joint disease with sclerosis of the condyle and the eminence and a chronically displaced disc which has atrophied (arrow).

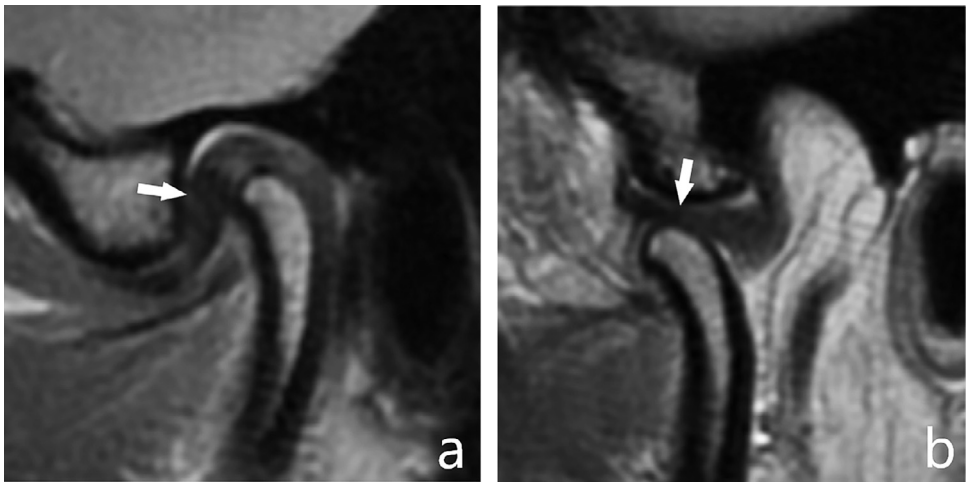

Figure 8 T1 WI MR axially corrected sagittal cross sections demonstrate anterior disc displacement with reduction: (a) The posterior band of the disc can be seen in the 9 o'clock position to the condyle. (b) The condyle translates to a point inferior to the crest of the eminence with the portion of the disc between the articular surfaces being the junction of the anterior band and the intermediate zone, which is the normal open mouth relationship of the components of the TMJ.

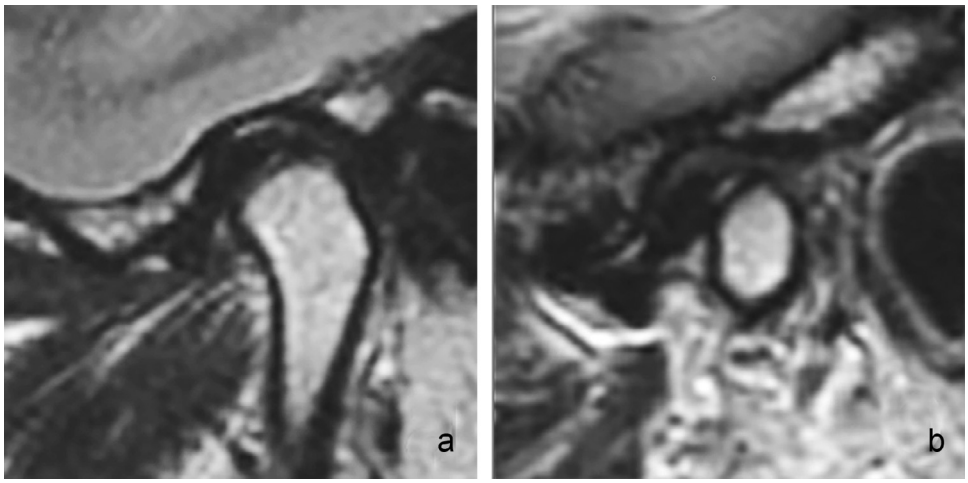

Figure 9 PD MR axially corrected sagittal cross sections demonstrate anterior rotational disc displacement: (a) The center portion of the TMJ shows a normal relationship between the disc and the condyle. (B) The lateral portion of the TMJ shows the posterior band of the disc being positioned anterior to the condyle.

The progression may also occur at different stages in the bilaterally joints, resulting in changes in the morphology and function of the mandible.[4] It is characterized by the breakdown of the articular surfaces of the joint and is precipitated by the derangement and displacement of the TMJ disc. Once the disc is displaced, the biomechanical forces in the joint will no longer be distributed properly and the shock absorption of the disc will no longer be effective.

In the initial stage of disc displacement, early changes to the articular surfaces occur such as flattening and subchondral sclerosis. These are called "functional remodeling" and are an effort to increase the articular surface area to withstand the excess force (Fig. 15). As the forces overcome the capability of the tissues to compensate, the bone begins to break down causing erosion to occur. This initially occurs on the superior surface of the mandibular condyle. It can be visualized on axially-corrected sagittal cross sections as breakdown of the cortical outline and loss of trabecular structure beneath the surface (Fig. 16). This stage is called "active DJD." As the erosions heal and become recorticated, the condyle appears smaller with loss of vertical height and volume (Fig. 3). Erosions that heal at the surface without filling the trabecular bone are called "subchondral bone cysts" (Fig. 17). These are not true cysts with epithelial lining, but rather cavity voids. An alternative explanation for this phenomenon is the seepage of synovial fluid into microcracks in the articular surface, giving these areas the characteristic high signal intensity on T2-weighted imaging and intermediate signal on T1 and PD-weighted imaging. Recortication or partial recortication of the articular surface is considered part of the healing stage.

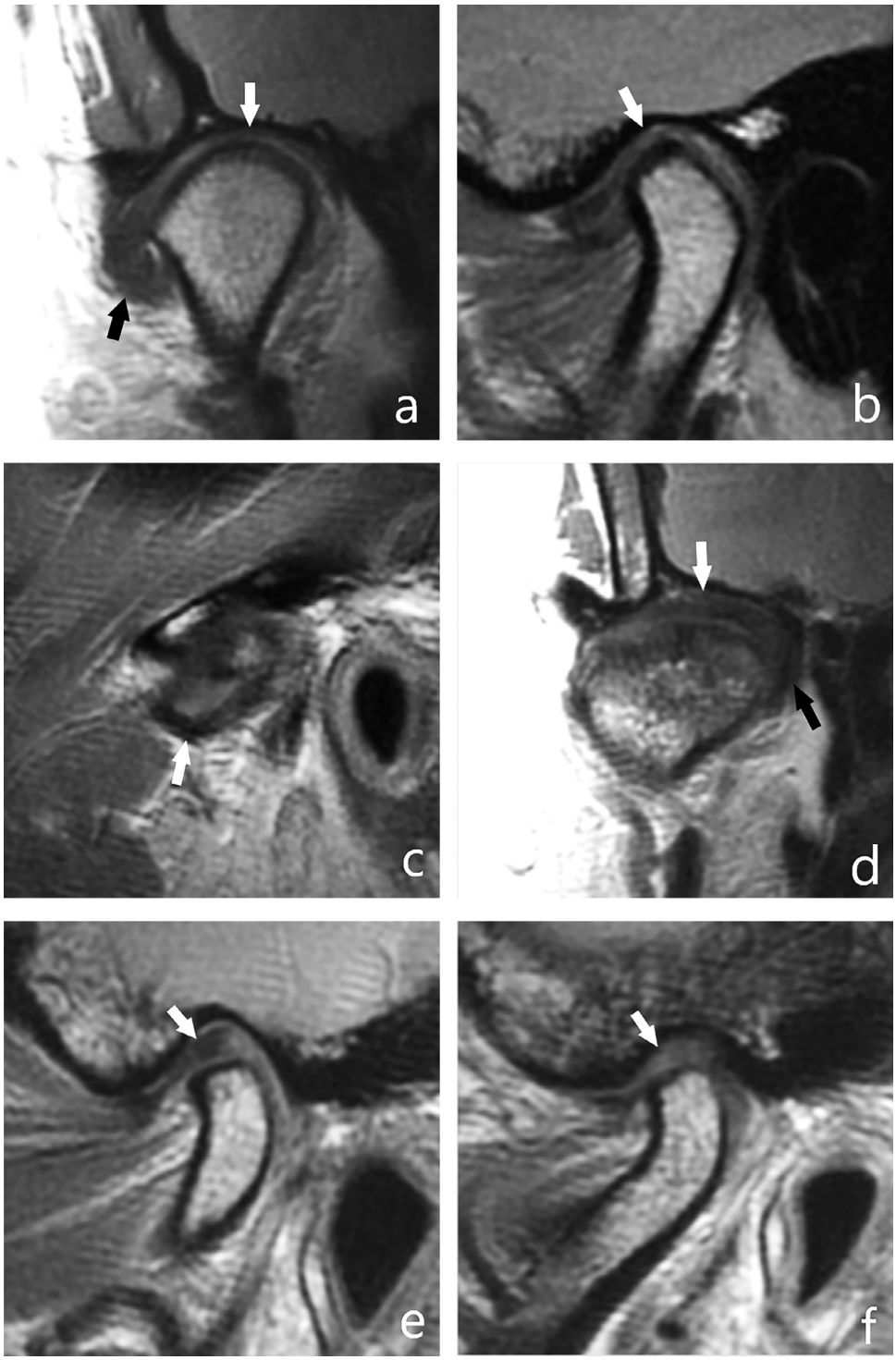

Figure 10 T1WI MR axially corrected sagittal and coronal cross sections demonstrate sideways disc displacement: (a) Coronal view shows the low signal lateral portion of the disc folded over the lateral pole (black arrow) and the medial superior portion is devoid of disc (white arrow). (b) Sagittal view shows that the center portion of the TMJ does not demonstrate a posterior band of the disc superior to the condyle, (c) Sagittal view in the lateral aspect of the TMJ shows the low signal of the disc folded over the lateral pole. (d) Coronal view shows the low signal medial portion of the disc folded over the medial pole (black arrow), with a portion of the posterior band of the disc still present in the center portion of the disc space (white arrow) (e) sagittal view on the center portion of the TMJ shows the posterior band is at the 11 o'clock position to the condyle. (f) The lateral portion of the TMJ shows no disc signal superior to the condyle (Courtesy M. Noujeim DDS).

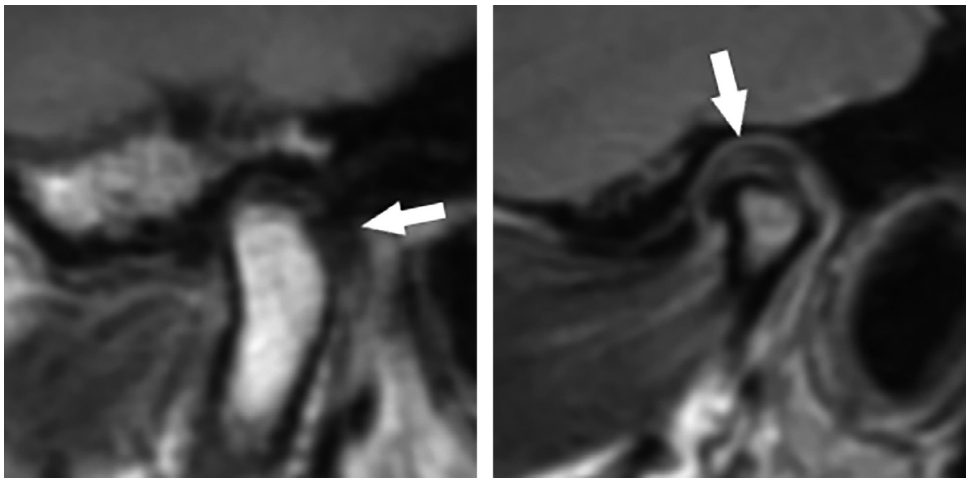

Figure 11 T1WI MR axially corrected sagittal cross section shows the formation of a pseudodisc superior to the condyle and posterior to the displaced disc. The fibrosis and thickening of the posterior attachment gives the appearance of a disc, and in some cases can be misinterpreted as a posteriorly displaced disc.

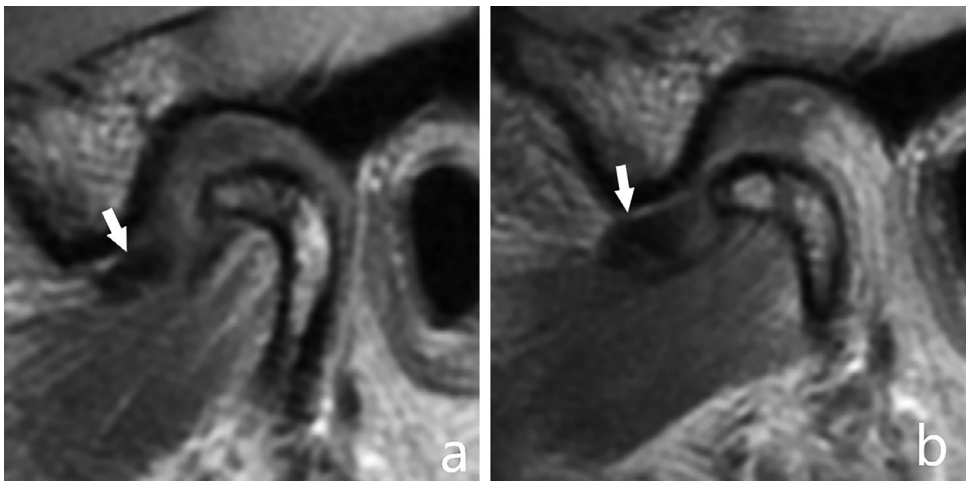

Figure 12 T1WI MR axially corrected sagittal cross sections demonstrate anterior disc displacement without reduction. (a) The disc has been chronically displaced, is reduced in anteroposterior dimension and has lost its biconcave appearance. (b) Upon opening, the disc remains anterior to the condyle and the condylar range of motion is reduced as the condyle is positioned posterior and superior to the crest of the eminence.

Once recortication is complete, the DJD is considered to be in the "Stable" (Fig. 18). Other radiographic signs of stable DJD are osteophyte formation (usually on the anterior aspect of the condyle) and loose articular bodies (joint mice) that can be considered a synovial chondromatosis secondary to DJD (Fig. 19). If excess forces are reapplied, the entire process may reactivate and further erosion and condylar volume loss may occur.[6,7]

Inflammatory Disorders

Chronic inflammatory conditions occur in the TMJ the most common of which are rheumatoid arthritis, juvenile idiopathic arthritis and pigmented villonodular synovitis. Other inflammatory conditions may affect the joint however they will not be discussed here.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is an autoimmune disorder which manifests itself as inflammation in the synovial joints throughout the body. The TM joint is no exception. This chronic inflammatory disease creates a synovitis which can lead to formation of synovial granulation tissue (pannus). This pannus then releases enzymes which destroy the surrounding osseous surfaces of the joint. Progressive bony erosions will be present with loss of condylar height and shape. Characteristic appearance of a joint affected by RA is that of a "sharpened pencil."[6] This differs from the appearance of the condyle with DJD as the erosions in DJD show more of a

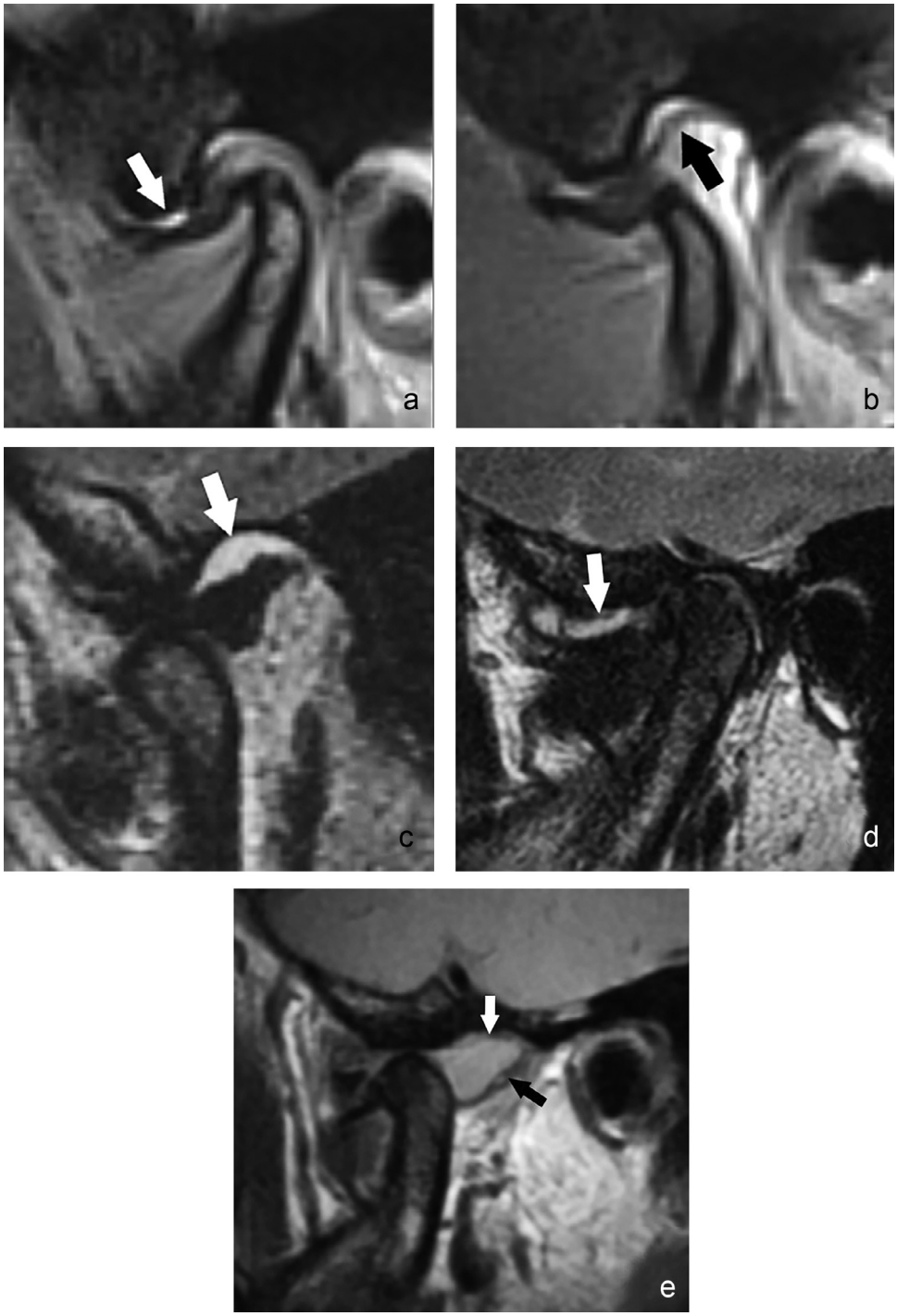

Figure 13 Axially corrected sagittal cross sections demonstrate joint effusion. (a) T2WI in the closed mouth position shows the synovial fluid in the anterior recess of the superior compartment outlining the shape of the disc and the posterior attachment. (b) Upon opening, the fluid travels to the posterior recess of the superior compartment and the TPA can be seen detaching slightly from the roof of the fossa (arrow). (c) T1WI FS shows moderate joint effusion in the posterior recess of the superior compartment upon opening detaching the TPA (arrow). (d) T1WI FS shows joint effusion in the anterior recess of the superior compartment. (e) The fluid traveled into the posterior recess of the inferior compartment, distending the capsule (black arrow). This is suggestive of the presence of a perforation connecting the superior and inferior joint compartments. The TPA can be seen abutting the roof of the fossa (white arrow) (Courtesy M. Noujeim DDS and T Deahl DDS).

TPA, temporal posterior attachment.

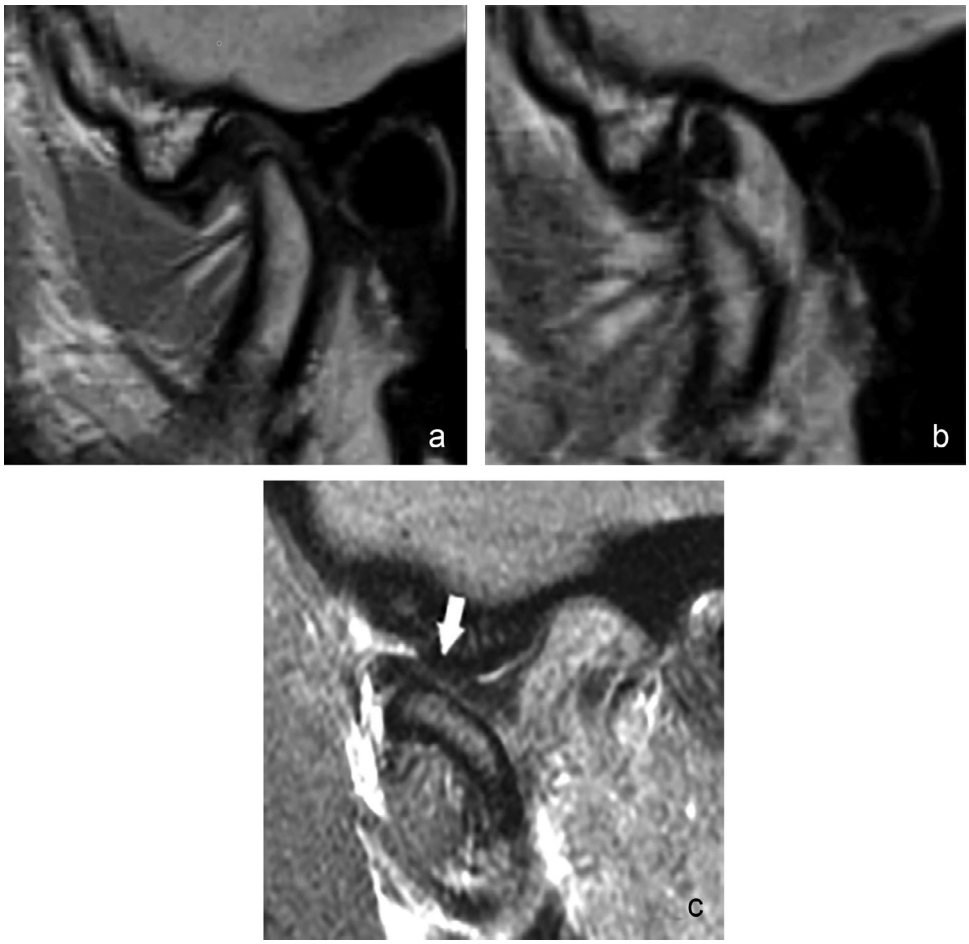

Figure 14 T1WI axially corrected cross sections show extremes of hypomobility and hypermobility. (a) In the closed mouth position, the condyle is posterior position in the fossa and the disc is in the normal relationship with the fossa. (b) In the open mouth position, the disc position does not change in relationship to the fossa but the condyle moves to the intermediate zone of the disc. These findings are suggestive of adhesion in the superior compartment of the TMJ. (c) This open mouth position for a different patient shows the condyle translating to a point anterior and superior to the crest of the eminence. The intermediate zone of the disc is positioned between the posterior aspect of the condyle and anterior slope of the eminence (arrow).

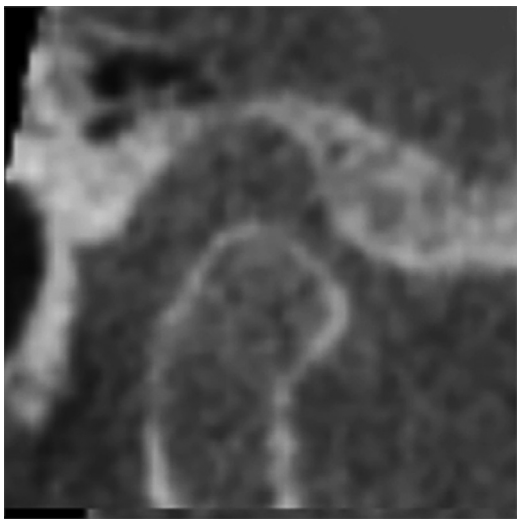

Figure 15 CBCT sagittal cross section shows flattening of the articular surface of the condyle, suggestive of functional remodeling. This is an attempt by the bone to increase the surface area to dissipate the increased loading on the articular surface.

pattern of focal erosions and subsequent volume loss. The 2 processes may be included in the same differential in milder cases. In addition RA commonly occurs bilaterally due to the systemic nature of the condition vs DJD which may occur in a single joint. In some cases the two conditions may coexist if the disc is displaced and DJD occurs. This is characterized by the presence of erosions, subchondral bone cysts, and osteophytes. The osseous changes in Rheumatoid Arthritis are well visualized using CBCT, however the pannus cannot be seen in this modality. In MR T1-weighted images, a decreased bone marrow density is seen. The pannus will have an intermediate signal intensity on both T1 and T2. T2-weighted images will show high signal intensity in the joint space due to the fluid (synovitis) and also an increased condylar marrow intensity due to edema (Fig. 20). The disc may or may not be displaced in this condition.

Juvenile Idiopathic Arthritis

Juvenile idiopathic arthritis (JIA) also known as juvenile rheumatoid arthritis or juvenile chronic arthritis is an

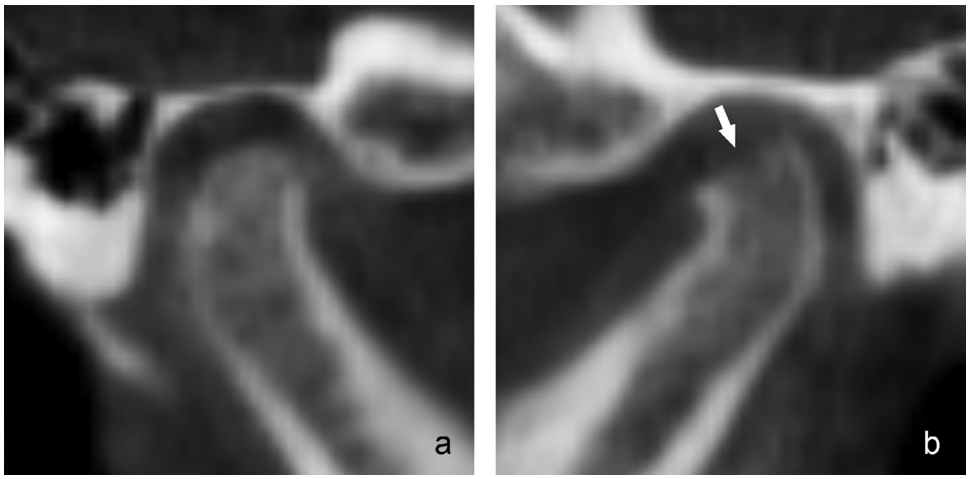

Figure 16 CBCT sagittal cross sections show the right and left TMJs of a patient undergoing unilateral active degenerative joint disease. (a) The right side is normal. (b) The left side shows decortication and erosion of the articular surface (arrow).

autoimmune inflammatory disease that affects the musculoskeletal system in children. It is the most common rheumatic disease in childhood.[10,11] The TMJ is commonly involved in this disease and is likely to be involved when the pattern is systemic. Seventy percent of patients are asymptomatic. Radiographically, the TMJ will show bilateral flat, grossly misshapen condyles with a wide glenoid fossa on CBCT (Fig. 21). It may be unilateral but more commonly bilateral. Secondary changes representing osteoarthritis may be seen such as osteophyte formation. The coronoid processes are often elongated and superiorly positioned. Clockwise rotation of the mandible may be seen due to the shortened condyles. In MR T1-weighted images a confirmation of the flat condyles will be present. T2-weighted images will show hyperintense joint effusion, bone marrow edema, and thin or perforated discs. The disc may also be absent. T1-weighted images with contrast reveal joint space enhancement (Fig. 22). Early inflammation may be detected before joint destruction. Using contrast in the suspected JIA cases may be useful for early detection.

Pigmented Villonodular Synovitis

Pigmented villonodular synovitis (PVNS) is a rare, benign, yet locally aggressive proliferation of mononuclear histiocytes and giant cells involving the synovium of the joint, tendon sheaths, and bursae with an incidence of 1.8 annual cases per million people.[12]

PVNS is classified as the localized form which involves only a part of tendon sheath or the joint lining and the diffuse form which is more common and involving the whole synovial joint membrane or bursa.[13] PVNS of the TMJ is a rare variant with <70 cases reported in the literature.[14]

Radiographic examination reveals remarkable bone erosion with involvement of skull base in approximately one-third of the cases.[15] The vast majority of the bone destruction involves the TMJ. Skull base, sphenoid bone, maxilla, coronoid process, auditory structures, and zygomatic arch can be affected as well.[16] The most common clinical symptoms of PVNS are preauricular swelling or mass with progressive pain and restricted mouth opening. Trismus, discomfort in mastication, impaired hearing, clicking, and tinnitus are less frequent findings.[15] Neurologic symptoms, such as hearing impairment, vomiting, and otalgia were rarely mentioned in the literature.[16]

PVNS is seen in all ages but more commonly in the 3rd to 5th decades of life (mean age, late 30s) with no gender predilection.[13] The etiology of PVNS remains unclear.

The imaging characteristics of PVNS represent its pathologic courses that are synovial proliferation and hemosiderin staining.[13] CT depicts bone erosion and sclerosis. In CT, PVNS is often seen as a soft tissue lesion with focal or all regions of high attenuation and further enhancement following contrast material administration[15] The thickened synovium and hyperdense regions produced by iron deposits are enhanced in CT. Thus, CT not only reveals the radiographic features above, but it can also show the tumor extension.[12]

The most specific and sensitive technique for the diagnosis of PVNS is MRI, which shows the preoperative tumor extension and recurrence. In MRI, depends on the amount of fluids, cellular elements, pannus, fibrous stroma, hemosiderin and lipids, PVNS can vary in appearance.[12] For instance, hemosiderin deposition leads to low signal intensity on proton density-, T1- and T2-weighted images (WI).[12] Additionally, hemosiderin will cause susceptibility or "blooming" artifacts on echo planar or gradient echo sequence (GRE) due to its paramagnetic properties, resulting in signal loss on the images.[13] Because of "blooming" the atypical tissue can be seen larger on T2-weighted spin echo and GRE sequences (Fig. 23). Lipid accumulation in foamy macrophages can cause high signal on T1-weighted spin echo sequence like in subcutaneous fat. In addition, loculated cyst of joint fluid regions gives high signal on T2WI. Hyperplastic synovium and bone erosion with inadequate intraarticular space due to outwards proliferation of the synovium are other well-known features of PVNS on MRI.[12] It is stated that GRE sequence

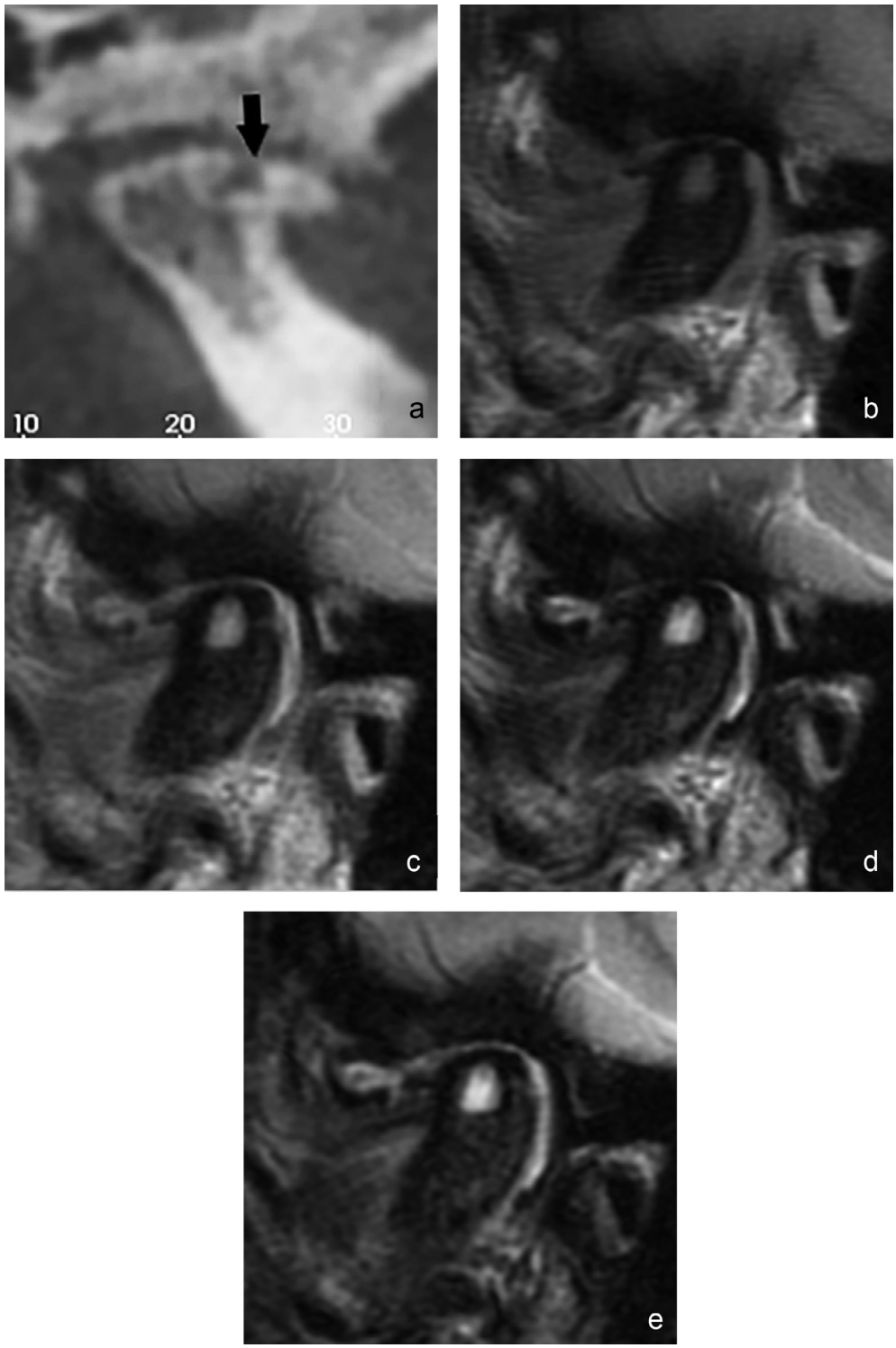

Figure 17 This series demonstrates the appearance of a subchondral bone cyst with multiple protocols. (a) CBCT shows a well-defined low density structure just deep to the articular surface connected to the surface with a small low density "tail". On a different patient, the MR images are of the same condyle and show the signal differences between protocols. (b) T1, (c) PD, (d) T2, (e) STIR (Courtesy, M. Noujeim DDS, MS).

can be useful to confirm the diagnosis when the diagnosis of PVNS is uncertain.[13]

The differential diagnosis of PVNS of TMJ contains parotid gland lesions (benign or malignant) along with tumors that affect the synovium and adjacent structures such as synovial chondromatosis, differentiated by the presence of small cartilaginous or ossific bodies, malignant synovioma identified by the presence of amorphous calcification and synovial sarcoma.[13] Gout, that is often polyarticular; rheumatoid arthritis, characterized by decrease in joint space and demineralization; and septic

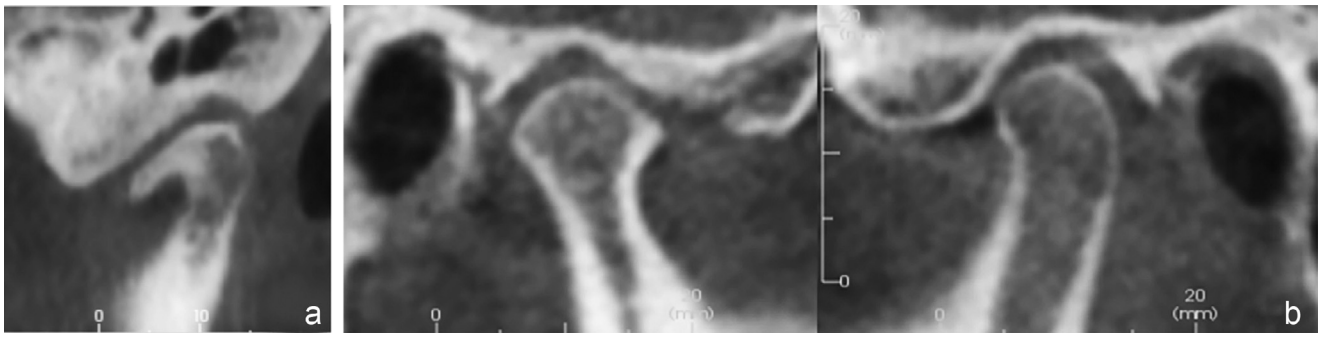

Figure 18 CBCT cross sections show the appearance of stable end-stage degenerative joint disease for (a) adult degenerative joint disease and (b) juvenile degenerative joint disease (idiopathic condylar resorption) on the right compared to the normal left side.

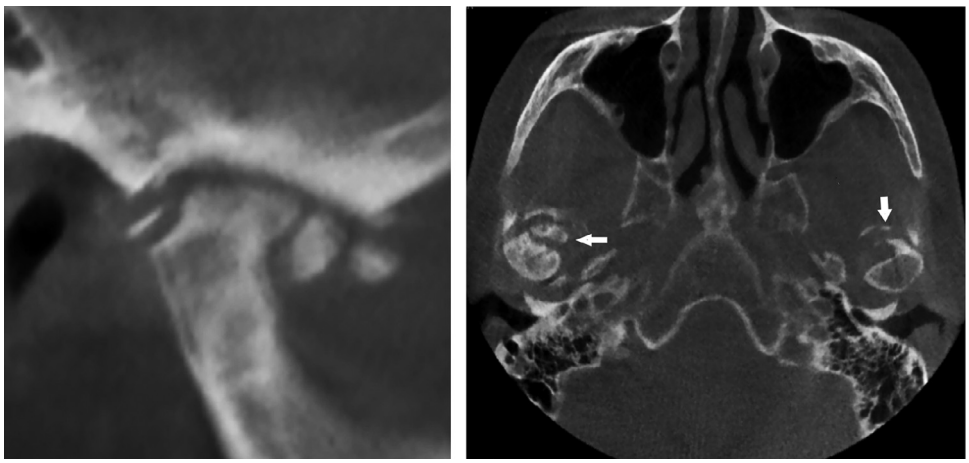

Figure 19 CBCT axial and sagittal cross section shows the appearance of the loose articular bodies seen secondary to degenerative joint disease (also known as "secondary synovial chondromatosis" or "joint mice"). The osseous components of the TMJ show degenerative joint disease changes but no mass effect that can be seen with primary synovial chondromatosis.

arthritis, presents with ill-defined borders and more cortical destruction should be also included in the differential diagnosis. Despite being rare, external auditory meatus or middle ear carcinoma is important differential diagnosis as well with presence of auditory canal and osseous destruction. Osseous destruction in PVNS is frequently limited to the joint space with no communication in the ear.[13] Presence of hemosiderin deposition, bone erosion, soft tissue mass and synovial proliferation are very diagnostic of PVNS and these features aid to differentiate PVNS from giant-cell tumors, hematomas, and pseudoaneurysm that occur following hemorrhage.[17] Also, in cases with signs and symptoms suggestive of TMJ dysfunction but with presence of accompanying symptoms (ie, altered sensation, trismus, or swelling), further evaluation is needed to rule out PVNS.

Mass-Like Lesions

Calcium Pyrophosphate Dihydrate (CPPD) Deposition Disease

Calcium pyrophosphate dihydrate (CPPD) deposition is a rare, metabolic disease characterized by CPPD crystal deposition in the synovial fluid leading to calcification of articular cartilage, and acute arthritis called pseudogout.[6] Calcium pyrophosphate dihydrate deposition disease (CPDD) has been defined as "pseudogout" due to gout-like symptoms and differs from gout which presents with precipitation of uric acid crystals instead of CPPD.[6,18]

CPDD has a tendency for joints with fibrocartilage and mostly affects large joints such as knee, ankle, hips, wrist, shoulder and rarely TMJ[6,19] with polyarticular involvement in almost two-thirds of the cases.[19] It is generally seen in the middle or older ages with male predominance although some studies reported female predilection.[6]

Despite that the etiology of CPDD has not yet been elucidated, it has been etiologically grouped into 4 classes: associated with metabolic disease, sporadic (idiopathic), hereditary, and associated with trauma or surgery.[6,18] The most common signs and symptoms of CPDD include preauricular swelling, pain, and trismus. Other findings are restricted condylar movement, otalgia, hearing loss, tinnitus, and sometimes asymptomatic.[6,20]

Radiographically, in CT/CBCT, early stage of CPDD shows thin, small, evenly distributed, cloud-like multiple synovial calcifications in the joint space, usually

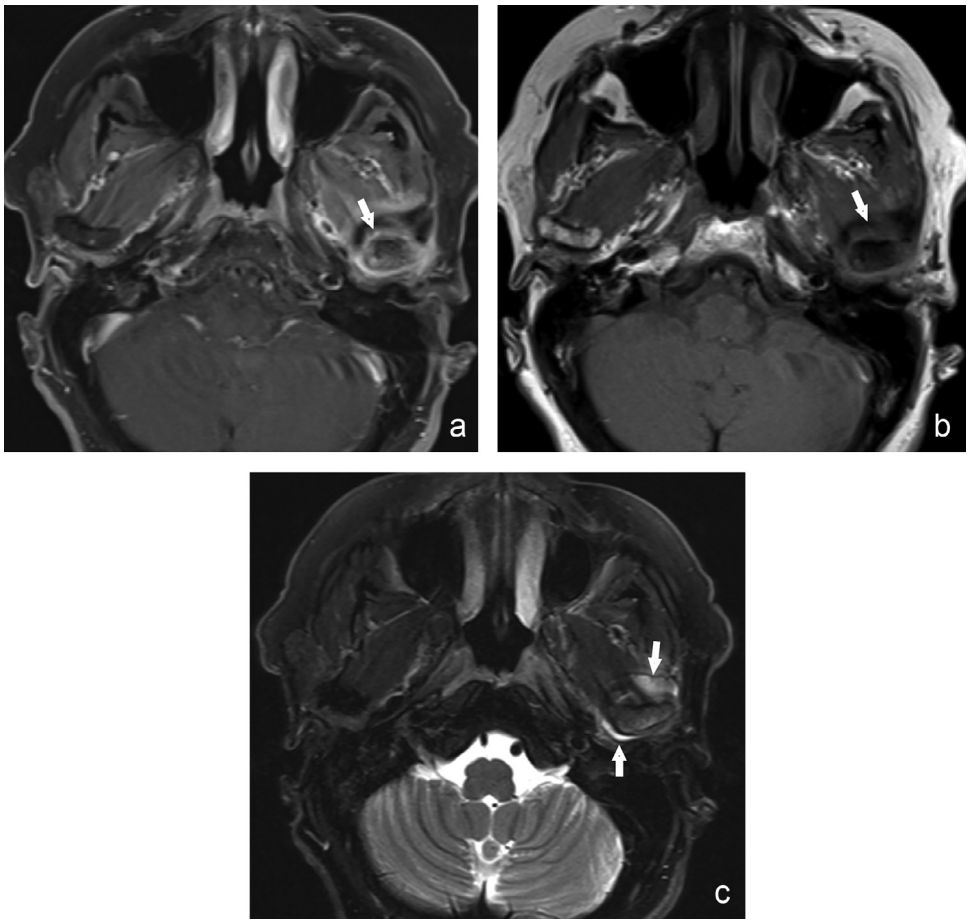

Figure 20 The appearance of rheumatoid arthritis on MR imaging: (a) T1 C+ FS shows enhancement of the vessel-rich inflamed synovial lining, (b) T1 shows no enhancement of the lining and, (c) T2 shows enhancement of the fluid within in the joint spaces. (Courtesy, R. Wiggins, MD).

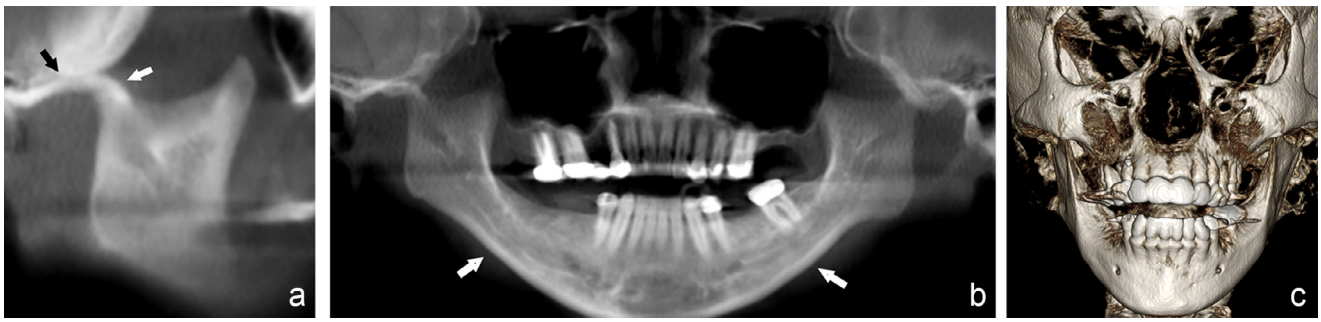

Figure 21 CBCT reformations show some of classic features of JIA: (a) Sagittal oblique view of the right side of the mandible shows a condylar stump (white arrow) that is positioned anterior to the eminence. The fossa and eminence are not developed (black arrow) as this condition most likely occurred earlier in life before their development. The coronoid process appears long in comparison to the condylar process, (b) Panoramic reformat shows steep mandibular planes (arrows) and very short mandibular rami, (c) 3D surface rendering shows an anterior open bite has occurred due to the posterior rotation of the mandible. The oropharyngeal airway is often compromised due to this posterior rotation due to posterior displacement of the tongue (Courtesy, M. Noujeim DDS, MS).

surrounding the condyle. Articular cartilage is linearly calcified. In the late stage, diffusely calcified mass with possible ground-glass appearance is prominent. Condyle and neighboring structures may demonstrate mass effect, erosion or remodeling (Fig. 24). In MRI, CPDD gives low to intermediate T1WI signal with heterogeneous contrast enhancement and low T2WI signal with edema of adjacent muscles. In plain radiography, linear or punctate

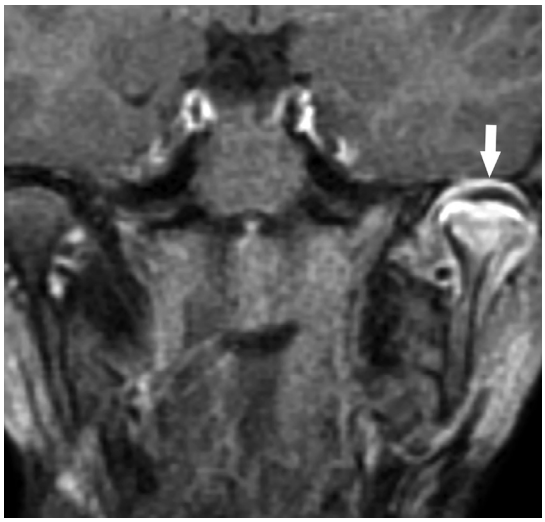

Figure 22 Coronal T1C+ FS shows enhancement of the synovium on the left TMJ in this patient with Juvenile idiopathic arthritis. This condition can occur unilaterally or bilaterally. (Courtesy, R. Wiggins, MD).

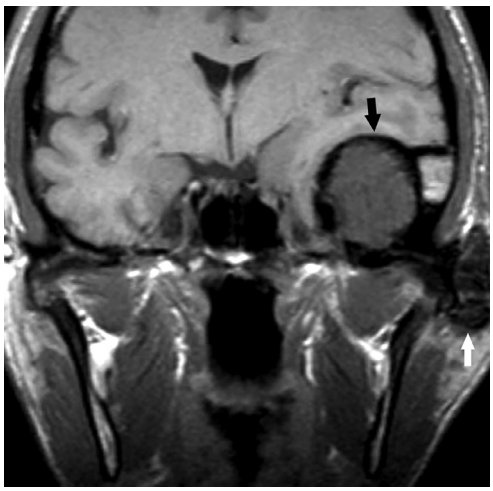

Figure 23 Coronal T1WI MR shows an example of PVNS hypointense right TMJ mass with a peripheral lower signal intensity (white arrow). A hypointense contiguous middle cranial fossa extra-axial mass is evident with similar markedly low signal peripheral rim (black arrow). (Courtesy R. Wiggins, MD).

PVNS, pigmented villonodular synovitis; TMJ, temporomandibular joint.

calcifications in fibro- or hyaline cartilage are the characteristic features of CPDD.[19]

Sometimes, the calcified lesion is spread into nearby regions (ie skull base, middle cranial fossa)[20] and can imitate malignancy.[6] The clinical and radiographic findings of CPDD of TMJ are unspecific, making the accurate diagnosis difficult and may mimic other lesions like synovial chondromatosis, degenerative disease, osteochondroma, and chondroblastoma or chondrosarcoma.[20] Therefore, the gold standard of CPDD diagnosis is the observation of CPPD crystals in synovial fluid and occurrence of metaplastic chondrocytes.[19] In terms of prognosis, the most significant misdiagnosis is chondrosarcoma.[18]

Synovial Chondromatosis

Synovial chondromatosis (SC) is a rare, benign, non-neoplastic lesion characterized by growth of cartilaginous nodules within subsynovial membranes that eventually ossify, detach, and seen as loose bodies floating within joint space.[6,21] It generally affects large joints (ie hip, knee or elbow) and hardly involves the TMJ.[22]

TMJ is the rarest region for SC and only about 200 cases reported in the literature.[22] Superior joint compartment of TMJ is more frequently affected with rare involvement of the inferior joint compartment.[6]

The average age at onset is 40 to 50 years, with mostly unilateral occurrence. Bilateral presentation of SC in the TMJ was rarely documented.[23] Females are more frequently affected than males with a ratio of 4:1.[6]

Despite the etiology of SC still remains unclear, the disease has been classified as primary and secondary synovial chondromatosis.[6] Primary synovial chondromatosis is more aggressive, associated with metaplasia, cartilaginous foci development and calcification of the cartilage bodies that widen and detach from synovial membrane.[6,21] Secondary synovial chondromatosis, as reflected by its name, occurs secondary to microtrauma, joint overuse, non-inflammatory arthropathy or inflammatory joint disease. Cartilage tissue fragments eventually dislodge in synovial fluid and ossify.[6] Primary synovial chondromatosis shows a diffuse and patchy calcification, while secondary synovial chondromatosis presents ring-like calcification.[21]

The common signs and symptoms are preauricular swelling and pain, compression of eustachian tube, crepitus, limited mouth opening and condylar movement, malocclusion and masticatory muscle myalgia,[6,21,23,24] which simulate temporomandibular joint disorder (TMD), causing difficulty and delay in diagnosis. Thus, radiologic examination (conventional radiography, computed tomography (CT), cone-beam CT, and MRI plays a key role in diagnosis of SC.[23] Multiple calcified / ossified loose bodies around the mandibular condyle, widening of the joint space, degenerative changes of the condyle, soft tissue swelling/mass and seldom intracranial extension are the most common radiographic features (Fig. 25)[21,23-25] In MRI, heterogeneous, hypointense loose bodies seen in the superior joint space on T1WI and hyperintense effusion ± expansion and "Ring-like" signal due to hyperintense fluid surrounding group of hypointense loose bodies seen on T2WI are characteristic findings. In T1WI with contrast administration, the synovium is enhanced.[6]

Differential diagnosis of SC includes DJD, rheumatoid arthritis calcium pyrophosphate dihydrate deposition disease, osteochondritis dissecans, avascular necrosis, and chondrosarcoma.[6,22] SC and DJD have several common clinical findings and the diagnosis of SC is usually delayed due to misdiagnosis. However, SC and DJD can be differentiated based on their radiographic findings. SC is frequently seen in

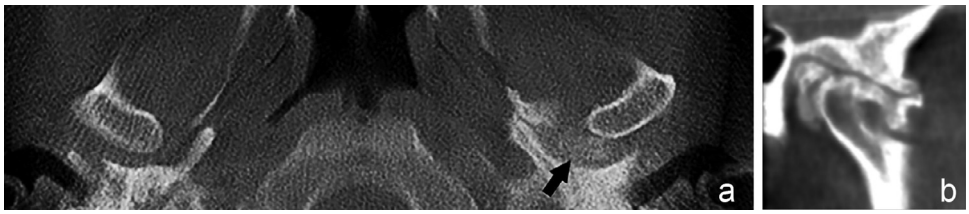

Figure 24 (a) Cropped Axial CBCT shows a diffuse cloud-like mass on the medial posterior aspect of the left condyle suggestive of CPPD deposition disease. (b) Sagittal cross section of a TMJ with CPPD deposition disease shows a granular high density mass posterior to the condyle as well as more solid (more mature) calcifications noted anterior to the condyle (Courtesy M Noujeim, DDS, MS).

CBCT, cone beam CT; CPPD, calcium pyrophosphate dihydrate.

the superior joint cavity,[6,21] generally presents round loose bodies with normal mandibular condylar shape along with effusion and expansion of the joint cavity.[6,21,25] On the other hand, DJD is usually found in the inferior joint cavity, presents angular loose bodies with apparent condylar degenerative changes (ie osteophytes in the anterior aspect).

Benign Tumors

The most common benign tumors of the TMJ are osteochondromas and osteomas.

Osteochondroma (OC) is a cartilage-capped bony exostosis developing from the external surface of the bone and including a marrow cavity which is continuous with the underlying bone.[26] Some authors categorize it as hamartoma and some as tumor.[6] It might arise from bones which are formed by endochondral ossification.[26] OC is frequently seen in the metaphysis of long bones such as proximal part of tibia and distal part of femur. Condyle and coronoid process are more often affected in jaws. In the mandibular condyle, OCs are 52% situated on the medial aspect, 20% on the anterior position, and 1% on the lateral location.[6,27]

Mandibular condylar OCs have a wide age range (13-76 years) with a mean age of 40 years.[6,26] Females are slightly more effected than males (F:M = 1.5:1).[6]

Clinical symptoms of osteochondromas involve hard, painless swelling, vertical face elongation on the affected side causing facial asymmetry, mandible deviation to unaffected side, limited condylar movement, cross-bite on unaffected side, open bite on affected side, disc displacement, and infrequently pain.[6,26]

Pathologic characteristics of osteochondroma usually mirror its radiographic presentation. The typical feature of osteochondroma is continuation of mass with medullary canal and bone cortex (Fig. 26).[27] Radiographically, it presents with nodular or mushroom-like appearance.[26,28] Due to its cartilage constituent, OC in TMJ is seen as a mixed density lesion on panoramic radiograph[26] CT shows calcification on the cartilage cap while MRI confirms the diagnosis by revealing the cartilaginous cap that is characteristic of

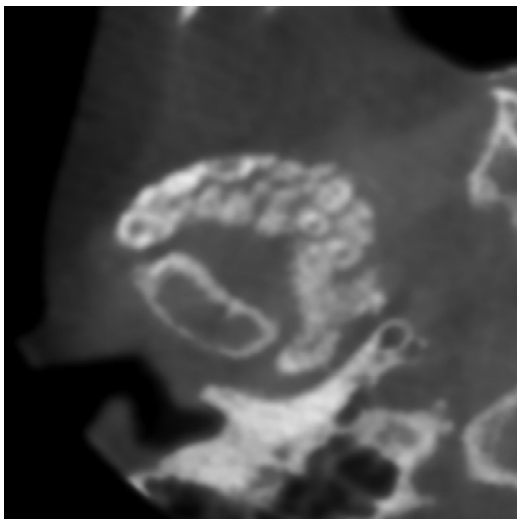

Figure 25 Cropped axial CBCT of the right TMJ with primary synovial chondromatosis shows multiple well-defined calcified structures with low density internal structures organized in the location of the anterior recess of the superior joint compartment (inferior to the crest of the eminence) (Courtesy, M. Noujeim DDS MS).

Figure 26 Sagittal CBCT of the left TMJ shows a hyperintense mass attached to the superior aspect of the condyle. The native condylar cortex can be visualized and the mass appears to be displacing the condyle inferiorly and remodeling the opposing fossa and eminence (Courtesy M. Noujeim, DDS, MS).

CBCT, cone beam CT; TMJ, temporomandibular joint.

osteochondroma.[27,28] Furthermore, abnormal metabolic bone activity may be detected by a bone scan due to increased 99mTc-HDP uptake in the affected area.[26]

Condylar hyperplasia, degenerative joint disease (DJD), coronoid hyperplasia, osteoma, chondroma, chondrosarcoma mainly develop in TMJ region and should be considered in the differential diagnosis of osteochondroma.[6,26]

Osteoma is a benign, slow growing, bone-forming tumor developing from cancellous or compact bone proliferation that originates from endosteum (central osteoma), periosteum (peripheral osteoma) or extra skeletal soft tissue.[6,29] It is usually limited to the craniofacial skeleton with rare involvement of TMJ.[30] Although the mandibular condyle is more commonly affected, the glenoid cavity, coronoid process and mandibular notch can be involved as well.[6]

Osteomas develop at any age, but are usually seen in people older than 40 years with no gender predilection.[6,29] They arise from membranous bones of the face and skull, from the embryonal endosteum, periosteum or cartilage. Osteomas can be classified as cortical (ivory) type, cancellous type or a combination of both.[29] Compact osteoma demonstrates well-differentiated mature dense bone with scarce marrow. Cancellous osteoma demonstrates lamellar trabeculae with rich marrow.[6] They may present as a pedunculated lesion on the condylar neck or head or as tissue proliferation that replaces the condyle (Fig. 27).[29]

Osteomas generally do not cause symptoms and can be seen as an incidental finding during a routine examination. If symptomatic, they mimic TMD with limited jaw opening and sporadic pain. Ipsilateral open-bite in condylar involvement, swelling, facial asymmetry, contralateral mandibular deviation are other associated findings.[6,30]

Radiographically, compact (ivory) osteoma is seen as a well-defined, uniform, radiopaque lesion. Cancellous osteoma manifests as a well-defined radiopaque lesion with presence of internal trabeculae. Depends on the marrow cavity, mixed radiolucent-radiopaque region might be present in the centers of osteomas.[29] On CT, it demonstrates homogeneous, bony, hyperdense lesion with normal internal trabecular structure (Fig. 26). On MRI, it is hypointense on T1WI with or without hyperintense central yellow marrow. On T2WI, it gives low signal or similar to marrow signal with or without hypointense cortical rim.[6]

As a differential diagnosis of osteomas, osteochondroma and condylar hyperplasia should be considered. Osteochondroma is a cartilage capped tumor which develops with endochondral ossification and continuous with trabecular bone. However, osteoma arises from periosteal regions. Condylar hyperplasia keeps the original shape of the condyle but osteoma has a lobulated or pedunculated appearance.[6]

In presence of multiple osteomas Gardner syndrome (familial adenomatous polyposis) should be considered. Gardner syndrome is associated with multiple osteomas of skull, jaws, and long bones accompanied by premalignant colorectal polyposis, multiple sebaceous or epidermoid cysts and multiple impacted supernumerary or permanent teeth.[6]

Malignancy

Malignancies of the TMJ are very rare. The more common TMJ malignancies are chondrosarcomas, osteosarcomas, and metastatic disease.

Chondrosarcoma (CS) is a malignant tumor that arises from embryogenic cartilaginous cells (pure hyaline cartilage differentiation). It is characterized by the production of cartilage without bone.[31,32] Chondrosarcoma may be divided as primary or secondary depending on its origin. Secondary chondrosarcoma develops from a pre-existent benign tumor, either an enchondroma or an osteochondroma and can be associated with Maffucci syndrome and Ollier disease.[32,33]

Chondrosarcomas account for 10%-20% of all malignant bone tumors and are usually seen in ribs, pelvis, and long bones with only 1%-2% originating in the head and neck region. In TMJ, CS is extremely rare, with only 30 cases mentioned in the literature until 2016.[31] When present, coronoid process is more commonly involved compared to condyle.[6]

Chondrosarcoma is a tumor of adulthood and old age with a mean age of 50 years (ranges from 11 to 75 years) with female predominance (F:M = 2:1) for TMJ region.[6] Secondary chondrosarcomas are found in younger ages, with a mean age at the time of diagnosis about 35 years.[32] These features differ from head and neck chondrosarcomas.[31] The etiology of CSs is still unknown.[33]

The major symptom of TMJ chondrosarcomas is preauricular swelling, followed by preauricular pain and trismus.[31] Deviation of the mandible, pain on mastication, obstruction of external auditory canal, limited jaw opening, and hearing loss may be seen as well. These findings imitate TMD which delay the diagnosis.[33]

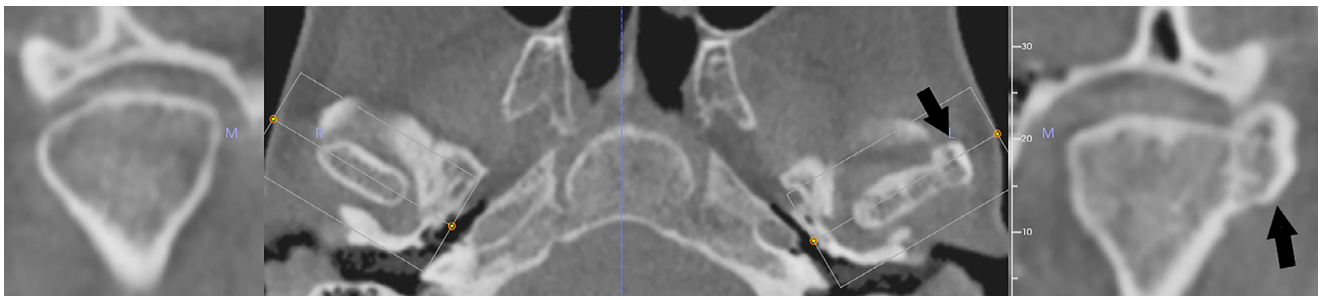

Figure 27 Coronal and axial CBCT of a cancellous osteoma shows a well-defined pedunculated mass attached to the lateral pole of the condyle. It has a thick cortical outline and the internal surface shows trabecular patterns similar to the native condyle. The differential diagnosis includes an osteochondroma.

There is no pathognomonic finding of chondrosarcoma. On panoramic or plain radiographs, it is seen as a lesion including single or multiple radiolucent areas.[33] On CT, CS appears as a soft tissue mass with flocculent calcifications in the joint space and surrounding the condyle with or without bone destruction (Fig. 28).[6] In many cases, widening of the joint space, attributed to condylar resorption, and elongation of the condylar neck with high attenuation of the condyle are noted.[31,33] Erosion of the glenoid cavity and invasion to the cranial fossa have been reported as frequent findings.[6,31] On MRI, CS gives low to intermediate T1WI signal comparing gray matter with possible hypointense foci suggestive of calcification of the tumor and high T2WI signal. It shows heterogeneous enhancement on T1WI with contrast administration with frequently present whorls of enhancing lines in the tumor matrix.[6]

Chondrosarcoma of the condyle can be confused with osteochondroma. In this case, patient age and growth rate are the key factors to aid differentiating them with osteochondroma seen in relatively younger age groups and having slower growth pattern than chondrosarcoma.[32] In addition, osteochondroma appears as a well-defined bony lesion with an organized low-density center and continuous with medullary cavity and cortex and has a thin cartilage cap. Chondrosarcoma demonstrates a soft tissue mass with a heterogeneously mineralized center. It can also cause bone destruction and neighboring soft tissue abnormality. Thickness of the hyaline cartilage cap is a very significant measure in deciding malignant transformation of osteochondroma. The thickness of the cartilage cap ranges between 0.1 and 3.0 cm; average, 0.6 and 0.8 cm in benign osteochondromas and between 1.5 and 12 cm; average, 5.5 and 6.0 cm in secondary chondrosarcomas.[32] No uptake in the whole-body bone scan eliminates malignant transformation of osteochondroma. Osteosarcoma, metastatic disease, calcium pyrophosphate dihydrate deposition disease and synovial chondromatosis are the other diseases that can be confused with chondrosarcoma.[6]

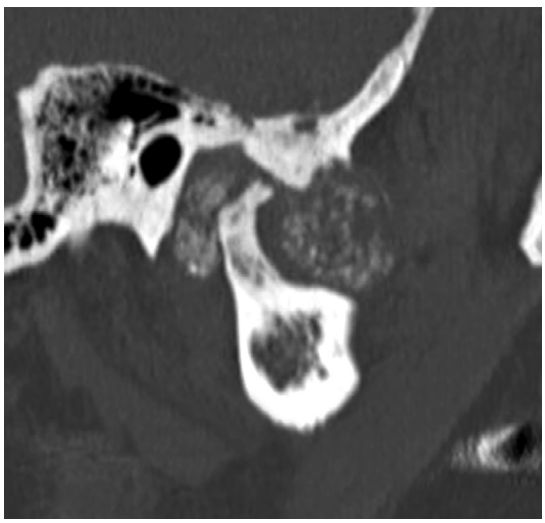

Figure 28 Sagittal reformat of noncontract-enhanced CT shows flocculent whorl-like calcifications anterior and posterior to the condyle in the soft tissues surrounding the joint. The condyle is anteriorly displaced by the posterior mass and resorbed posteriorly by this same mass. The eminence is resorbed by the anterior mass. (Courtesy, R. Wiggins).

Osteosarcoma (OS) is a highly malignant bone tumor arising from the osteogenic mesenchymal matrix, in which osteoid, fibrous, cartilaginous and osseous tissues are formed by malignant stroma.[6]

Osteosarcoma constitutes about 20% of primary bone tumors and generally affects the most rapidly growing sites of the skeleton like long bones (ie proximal tibia, humeral and distal femoral metaphysis).[34,35] It rarely occurs in the jaws, approximately 6% of all OSs with uncommon mandibular condylar involvement.[34] Osteosarcomas of the jaws are not as aggressive as OSs of long bones and do late metastasis comparing OSs of long bones.[6]

Jaw OSs are usually seen in the third decade with a mean age of 35 years, contrary to long bone OSs that peak in the second decade, approximately 10 years earlier than jaw OSs.[6,34] They occur in males more than females (M:F = 2:1) although female predilection was reported by some studies. (Dania)

The most common presentation of TMJ osteosarcomas are limited mouth opening, limited lateral movements, obstruction of external auditory canal, unilateral preauricular swelling, headache, TMJ dysfunction or pain which can be easily misdiagnosed as one of the more common TMJ diseases.[6,34]

Radiographically, TMJ osteosarcomas may have radiolucent (lytic), mixed, or sclerotic appearance with rare periosteal reaction such as sunray spicules or hair on end appearance in right angles to cortex. They may be laminated as well. The solid component of OS is moderately enhanced in CT with contrast administration. On MRI, it gives heterogeneous signal both on T1WI and T2WI. While non-mineralized solid tumors give intermediate T1 and high T2 signal; low T1 and T2 signal can be seen in mineralized tumors with heterogeneous contrast enhancement when applied.[6] Periosteal reaction manifests as a hypointense region on different MRI sequences.[36] Fat suppression sequences with contrast administration show fine details of tumor borders.[36,37]

In the TMJ, osteosarcoma radiographically presenting as a zone of bone permeation lacking tumor and new bone production may not be distinguished from metastatic diseases.[35] OS also resembles osteochondroma (OS), chondrosarcoma (CS) or aneurysmal bone cyst, however, soft tissue constituent in OS might be a hint to differentiate it from osteochondroma.[34,37] If the tumor has some radiopacities chondrosarcoma should be considered and presence of typical arc or whorl shape calcification suggests CS. Aneurysmal bone cyst causes pain and expands fast.[6]

Metastatic Malignancies

Among the malignancies potentially affecting the TMJ, non-primary tumors were defined with the primary tumor

originating from the breast, lung, prostate, liver, skin, or rectum.[38] The literature mentions few cases of condylar metastasis (24 metastatic cases to the condyle between 2000 and 2016 and 48 metastatic cases based on a review published in 2010).[38,39]

Cancers in the TMJ region are hard to diagnose due to their profound location and symptoms imitating TMD.[40] TMD like symptoms may be a hint for metastasis and/or relapse.[39] A tumor appearing in the TMJ area might be a metastatic malignancy. Metastatic malignancies of oro-facial region are relatively infrequent (about 1%-1.5% of all oral malignancies) and most commonly seen in patients between 40 and 70 years.[41] Most of the metastases affect the body of the mandible and metastasis to the condyle is rare.[39] This rarity may be due to paucity of hematopoietic (red) marrow and poor local blood supply in the condyle or to the existence of an osseous plate which limits the metastasis to the marrow of the condyle. In addition, spread from primary lesion to the TMJ region is generally hematogenous and metastasis to a minor joint like the condyle frequently occurs in the terminal stage of cancer when other metastases are already clinically obvious.[41] However, there are reports claiming mandibular condyle may be the first metastatic site recognized. When the metastasis is present it is generally unilateral, but bilateral TMJ metastasis have been reported as well. TMJ metastasis is frequently associated with mandibular or generalized skeletal metastases and solitary TMJ involvement is sporadic.[6]

Most common primary malignant tumors metastasizing to TMJ are breast, lung, prostate, kidney, and thyroid. Signs and symptoms of metastatic malignancies mimic TMD such as pain, restricted lateral movement, clicking, crepitation, mandibular deviation, trismus, and malocclusion. Preauricular swelling, paresthesia, pathologic fracture, hearing loss, diminished visual acuity, facial asymmetry, and tinnitus may be observed as well.[6]

Metastatic tumors in the TMJ generally manifest as poorly defined destructive lesion with invasive margins. Most metastatic malignancies do bone destruction; however, breast, lung and prostate metastases can form bone and have sclerotic or mixed radiographic presentation. Spiculated periosteal reaction found in other bones may not be present in condyles. In CT, metastatic tumors with a soft tissue mass and/or destruction of adjacent structures (ie skull base) can be seen. Increased signal or enhancing condylar mass may be observed on MRI.[6]

The differential diagnosis of several diseases affecting TMJ region usually is complicated by the fact that most common symptoms (limited movement and pain) are common to most of them. Thus, a special attention must be given to prevent a misdiagnosis of TMD and not to overlook the metastatic malignancies.[40] Osteosarcoma, chondrosarcoma, multiple myeloma, and osteomyelitis should be considered in the differential diagnosis of metastatic malignancies. Osteosarcoma usually has spiculated periosteal reaction, multiple myeloma has additional well-defined lesions either in mandible or other bones and osteomyelitis has mostly parallel periosteal reaction around the cortex.[6]

References

1. Lei J, Liu MQ, Yap AU, et al: Condylar subchondral formation of cortical bone in adolescents and young adults. Br J Oral Maxillofac Surg 51:63-68, 2013
2. Enlow DH, Hans MG: Essentials of facial growth. (ed 2). Ann Arbor MI: Distributed by Needham Press, 2008
3. Katzberg RW, Tallents RH: Normal and abnormal temporomandibular joint disc and posterior attachment as depicted by magnetic resonance imaging in symptomatic and asymptomatic subjects. J Oral Maxillofac Surg 63:1155-1161, 2005
4. Schellhas KP, Piper MA, Omlie MR: Facial skeleton remodeling due to temporomandibular joint degeneration: An imaging study of 100 patients. AJR Am J Roentgenol 155:373-383, 1990
5. Goncalves JR, Gomes LC, Vianna AP, et al: Airway space changes after maxillomandibular counterclockwise rotation and mandibular advancement with TMJ Concepts(R) total joint prostheses: Three-dimensional assessment. Int J Oral Maxillofac Surg 42:1014-1022, 2013
6. Tamimi D, Hatcher DC: Specialty Imaging: Temporomandibular Joint. (ed 1). Salt Lake City, Utah: Elsevier, 2016
7. Ahmad M, Hollender L, Anderson Q, et al: Research diagnostic criteria for temporomandibular disorders (RDC/TMD): Development of image analysis criteria and examiner reliability for image analysis. Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol 107:844-860, 2009
8. Larheim TA, Katzberg RW, Westesson PL, et al: MR evidence of temporomandibular joint fluid and condyle marrow alterations: Occurrence in asymptomatic volunteers and symptomatic patients. Int J Oral Maxillofac Surg 30:113-117, 2001
9. Tomas X, Pomes J, Berenguer J, et al: MR imaging of temporomandibular joint dysfunction: A pictorial review. Radiographics 26:765-781, 2006
10. Gare BA: Epidemiology of rheumatic disease in children. Curr Opin Rheumatol 8:449-454, 1996
11. Muller L, Kellenberger CJ, Cannizzaro E, et al: Early diagnosis of temporomandibular joint involvement in juvenile idiopathic arthritis: A pilot study comparing clinical examination and ultrasound to magnetic resonance imaging. Rheumatology (Oxford) 48:680-685, 2009
12. Kim IK, Cho HY, Cho HW, et al: Pigmented villonodular synovitis of the temporomandibular joint - computed tomography and magnetic resonance findings: A case report. J. Korean Assoc Oral Maxillofac Surg 40:140-146, 2014
13. Wong JJ, Phal PM, Wiesenfeld D: Pigmented villonodular synovitis of the temporomandibular joint: A radiologic diagnosis and case report. J Oral Maxillofac Surg 70:126-134, 2012
14. Safaee M, Oh T, Sun MZ, et al: Pigmented villonodular synovitis of the temporomandibular joint with intracranial extension: A case series and systematic review. Head Neck 37:1213-1224, 2015
15. Romanach MJ, Brasileiro BF, Leon JE, et al: Pigmented villonodular synovitis of the temporomandibular joint: Case report and review of the literature. Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol 111: e17-e28, 2011
16. Damodar D, Chan N, Kokot N: Pigmented villonodular synovitis of the temporomandibular joint: Case report and review of the literature. Head Neck 37:E194-E199, 2015
17. Cheng XG, You YH, Liu W, et al: MRI features of pigmented villonodular synovitis (PVNS). Clin Rheumatol 23:31-34, 2004
18. Vellone V, Bracciolini V, Ramieri V, et al: Synovial chondromatosis and calcium pyrophosphate deposition of the temporomandibular joint: Challenging diagnosis. J Craniofac Surg 29:e792-e794, 2018
19. Wickrematilake GW: Calcium pyrophosphate dihydrate deposition disease in young patients: Two case reports. Arch Rheumatol 32:80-83, 2017
20. Kwon K.J., Seok H. Calcium pyrophosphate dihydrate deposition disease in the temporomandibular joint: Diagnosis and treatment. 2018;40:19.
21. Kim HS, Lee W, Choi JW, et al: Temporomandibular joint synovial chondromatosis accompanying temporal bone proliferation: A case report. Imaging Sci Dent 48:147-152, 2018
22. Holtmann H, Bottinger T, Kubler NR, et al: Intra- and extracapsular synovial chondromatosis of the temporomandibular joint: Rare case and

review of the literature. SAGE Open Med Case Rep 6, 2018. 2050313x18775307
23. Liu X, Huang Z, Zhu W, et al: Clinical and imaging findings of temporomandibular joint synovial chondromatosis: An analysis of 10 cases and literature review. J Oral Maxillofac Surg 74:2159-2168, 2016
24. Testaverde L, Perrone A, Caporali L, et al: CT and MR findings in synovial chondromatosis of the temporo-mandibular joint: Our experience and review of literature. Eur J Radiol 78:414-418, 2011
25. Meng J, Guo C, Yi B, et al: Clinical and radiologic findings of synovial chondromatosis affecting the temporomandibular joint. Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol 109:441-448, 2010
26. Kwon YE, Choi KS, An CH, et al: Recurrent osteochondroma of the mandibular condyle: A case report. Imaging Science Dent 47:57-62, 2017
27. Kamble V, Rawat J, Kulkarni A, et al: Osteochondroma of bilateral mandibular condyle with review of literature. J Clin Diagn Res: JCDR 10: Td01-Td02, 2016
28. Zhang J, Wang H, Li X, et al: Osteochondromas of the mandibular condyle: Variance in radiographic appearance on panoramic radiographs. Dentomaxillofac Radiol 37:154-160, 2008
29. Misra N, Srivastava S, Bodade PR, et al: Osteoma of temporomandibular joint: A rarity. BMJ Case Rep 2013, 2013
30. de Souza NT, Cavalcante RCL, de Albuquerque Cavalcante MA, et al: An unusual osteoma in the mandibular condyle and the successful replacement of the temporomandibular joint with a custom-made prosthesis: A case report. BMC Res Notes 10:727, 2017
31. Lee K., Kim S.H. Temporomandibular joint chondrosarcoma: A case report and literature review. 2016;42:288-294.
32. Xu B, Shi H, Wang S, et al: Secondary chondrosarcoma in the mandibular condyle. Dentomaxillofac Radiol 40:320-323, 2011
33. Giorgione C, Passali FM, Varakliotis T, et al: Temporo-mandibular joint chondrosarcoma: Case report and review of the literature. Acta Otorhinolaryngol Ital 35:208-211, 2015
34. Uchiyama Y, Matsumoto K, Murakami S, et al: MRI in a case of osteosarcoma in the temporomandibular joint. Dentomaxillofac Radiol 43:20130280, 2014
35. Wang S, Shi H, Yu Q: Osteosarcoma of the jaws: Demographic and CT imaging features. Dentomaxillofac Radiol 41:37-42, 2012
36. dos Santos DT, Cavalcanti MG: Osteosarcoma of the temporomandibular joint: Report of 2 cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol 94:641-647, 2002
37. Lee YY, Van Tassel P, Nauert C, et al: Craniofacial osteosarcomas: Plain film, CT, and MR findings in 46 cases. AJR Am J Roentgenol. 150:1397-1402, 1988
38. Guarda-Nardini L, Stellini E, Di Fiore A, et al: A rare case of misdiagnosed silent lung cancer with solitary metastasis to the temporomandibular joint condyle. J Oral Facial Pain Headache 31:180-185, 2017
39. Dodo M, Kumagai M, Kato Y, et al: Metastasis in the mandibular condyle: A case report. J Med Case Rep 11:323, 2017
40. Singh S, Desai B, Laskin D: Metastatic melanoma misdiagnosed as a temporomandibular disorder: A case report and review of the literature. J Am Dent Assoc 145:1052-1057, 2014
41. Klasser GD, Echandi L, Shannon M: Hepatocellular carcinoma metastasis to the condyle: A case report and review of the literature. J Am Dent Assoc 145:1063-1067, 2014