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Review

Imaging of the temporomandibular joint

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Temporomandibular disorders are common, especially in young to middle-aged women, and most settle with supportive treatment. Imaging is indicated for the small percentage of cases that do not respond to conservative management and when the diagnosis is no doubt. The temporomandibular joint (TMJ) is a bilateral synovial articulation between the mandible and skull base. It has an intra-articular disc dividing the joint into superior and inferior compartments and the articular surfaces are lined with fibrocartilage. The normal imaging anatomy of the TMJ is described and illustrated. Different movements occur in each joint compartments: a hinge movement in the inferior joint space and translation or gliding in the superior joint space. Internal derangement is the commonest disorder affecting the TMJ and is most commonly due to disc displacement, followed by osteoarthritis and inflammatory arthritides. The imaging findings, primarily on magnetic resonance imaging (MRI) and computed tomography (CT), of internal derangement and less common disorders of the joint, are reviewed and illustrated. Optimal imaging protocols are discussed with detailed reporting guidelines.

Introduction

The temporomandibular joint (TMJ) is a complex synovial articulation between the mandibular condyle and the glenoid fossa and articular eminence of the temporal bone. An intra-articular disc (meniscus) divides the joint into superior and inferior compartments. The TMJ is normally subject to high biomechanical load from mastication and is unique in that movement of one joint always results in movement of the contralateral side as they are connected by the horseshoe-shaped mandible.

Temporomandibular disorder (TMD) is a collective term involving several disorders of the muscles of mastication, the TMJ, or both. Patients with TMD most frequently present with pain, limited or asymmetric mandibular motion, and joint noises. TMD is common, affecting up to 28% of the population and it predominates in young women; however, only 5–10% of those with symptoms require treatment, and in up to 40% of patients, the symptoms resolve spontaneously.[1]

Pain in the muscles of mastication (masticatory myalgia) is the commonest TMD. Articular causes of TMD include internal derangement of the joint, usually due to displacement of the disc and inflammatory and degenerative

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arthritides. Less frequently, the TMJ may be involved by a range of congenital, developmental, and acquired conditions including trauma and neoplasia.[2]

Indications for imaging of TMD

The referral will usually describe the clinical presentation and indicate whether the aetiology is likely to be muscular or joint in origin. Imaging is rarely indicated for masticatory myalgia unless there is a history of a precipitating event, such as dental treatment or trauma, or if there are other symptoms and signs, such as trismus, swelling or fever (Fig 1).

Internal derangement is defined as interference with a joint's smooth action and in the TMJ is most commonly due to disc displacement (DD); however, magnetic resonance imaging (MRI) studies have shown that mild DD is present in one-third of asymptomatic volunteers. DD is highly prevalent and of greater severity in symptomatic patients, but 20% will have a normally positioned disc.[3,4] Osteoarthritis (OA) is the second most common cause of internal derangement.

The principal indications for imaging include suspected advanced internal derangement; arthritis; failure of medical treatment; and in cases when the diagnosis of TMD is in doubt: atypical pain, sensory or motor dysfunction, or a palpable mass.

Table 1
Imaging protocols for the temporomandibular joint (TMJ).

CBCT of the TMJs (exact parameters vary with the vendor)
• Closed-mouth acquisition with a field of view (FOV) of 150×50 mm. The height of 50 mm allows coverage from above the TMJ to the mid-ramus level with a 15 s imaging time
• Voxel size of between 0.1–0.3 mm. Resolution is proportional to the radiation dose
• Open-mouth scans can also be performed to assess condylar mobility
• Large FOV (160×160 up to 240×190 mm), lower resolution (and dose) scans are used for assessment of dentofacial deformity, including the TMJs, for treatment planning
MDCT of the TMJs
• Multidetector (64–256 sections) CT acquisitions in the closed- and open-mouth positions from above the TMJ to the mid-ramus level, 140kV
• Thin overlapping images reconstructed into coronal oblique (i.e., parallel to the long axis of the mandibular condyle) and sagittal oblique (perpendicular to the long axis of the condyle) reconstructions, using bone and soft-tissue algorithms
MRI of the TMJs
• Ideally, at 3 T, using dual phased-array surface coils
• Proton-density-weighted coronal oblique and sagittal oblique sequences of each TMJ
• T2-weighted fat-saturated sagittal oblique sequences of each TMJ
• With maximum mouth opening (the mouth can be held open, for example with a syringe), fast T2-weighted sagittal oblique of each TMJ

CBCT, cone-beam computed tomography; MDCT, multidetector CT; MRI, magnetic resonance imaging.

Imaging techniques

The main imaging methods used in the evaluation of TMD are dental panoramic tomography (DPT), cone-beam computed tomography (CBCT), multidetector CT (MDCT), MRI, ultrasound, and nuclear medicine techniques. Table 1 presents the imaging protocols for CBCT, MDCT and MRI of the TMJ.

DPT

The DPT is commonly called an orthopantomogram (OPG). It is a curved tomogram that provides an overview of the dentition, jaws, and TMJs. The main value of the DPT is to demonstrate dental diseases, such as excess wear of the occlusal surfaces (attrition), caries, periapical pathology, and periodontitis, which may be relevant to the patient's symptoms and proposed treatment. Asymmetry of the mandible and condyles on an optimally performed DPT reflects asymmetric growth or resorption of the condyles.

Because of the superimposition of overlying bony structures and variable obliquity of the condyle, DPT is of limited value in the assessment of osseous abnormalities of the TMJ. It provides a lateral view of the joint and can usually demonstrate moderate to severe arthritis involving the condyle, but is insensitive for mild disease.[2,5]

Figure 1 Muscles of mastication. (a) There is diffuse hypertrophy of the muscles of mastication (M, masseter; T, temporalis) in a patient with bruxism and myalgia. The bulky, fat-replaced parotid glands (P) are laterally displaced by the enlarged masseter muscles. (b) This patient developed trismus following a left inferior alveolar nerve block for a large restoration in the lower left second molar. There is marked swelling of the left medial pterygoid muscle (white open arrows) secondary to a haematoma. The right mandibular foramen (asterisk), medial pterygoid (MP) and lateral pterygoid (LP) muscles are indicated.

CBCT

CBCT provides excellent bone detail and multiplanar two-dimensional (2D) and three-dimensional (3D) reconstructions of the TMJ, maxillofacial region, and adjacent skull base, but no soft-tissue information. It has similar, high accuracy to MDCT in detecting cortical abnormalities and sub-articular lucencies and sclerosis.[6]

A major review article concluded that there was a poor correlation between osseous changes on CBCT, pain, and other clinical signs and symptoms of OA.[7] Overall, assessment of CBCT led to changes in the primary diagnosis and management in more than half the patients with TMD who were initially assessed by clinical examination and DPT at a tertiary referral centre.[8]

MDCT

Low-dose MDCT is a highly effective technique when MRI is contraindicated, not tolerated, or unavailable (Table 1).[2,9] Bone detail of the TMJ is similar or better than CBCT mainly because, although MDCT is of lower spatial resolution it is less affected by image noise. The radiation dose of MDCT is usually higher than that of CBCT.

The major advantage of MDCT over CBCT is far superior soft-tissue contrast, which allows detection of significant DD and extra-articular pathology (including neoplastic causes of pain) that would not be detected by CBCT.

MRI

MRI is the optimal imaging technique for evaluating patients with TMD. The excellent inherent soft-tissue contrast combined with a high signal-to-noise ratio provided by dual-phase array surface coils and the high field strength of 3 T results in excellent soft-tissue, cortical, and bone marrow evaluation.

MRI of the TMJ allows assessment of DD, effusions, synovitis, the articular cortices, sub-articular marrow, the muscles of mastication, adjacent parotid tissue, and the external auditory canal.[3,4,10,11] Fat saturation T2 and post-gadolinium enhanced sequences provide an optimal assessment of intra-articular inflammation and mass lesions.

CBCT and MDCT have been reported to superior to MRI in the evaluation of the articular cortices, but it is also recognised that the accuracy of MRI is highly dependent on both the standard of equipment and the experience of the observer.[2,5] In middle-aged to elderly patients, MDCT may be preferred for evaluation of TMD when the underlying aetiology is more likely to be due to arthritis rather than DD.[9]

Research using functional MRI supports the concept that TMD is very similar to other chronic pain conditions in that there is abnormal pain processing in the trigeminal system including central sensitisation.[12]

Ultrasound

A recent systematic review article concluded that ultrasound could be used to screen for the presence of anterior DD and effusions in patients with TMD. Laterally situated cortical erosions and capsular thickening can also be visualised. A high-frequency probe is utilised in the axial plane with initial scanning with the teeth in occlusion. If a DD is visualised, the scan should be repeated with the mouth open to determine if the disc is reduced.[13,14]

Ultrasound-guided injection of corticosteroids into the inferior joint space can be used for the treatment of pain due to TMD if preliminary diagnostic imaging has demonstrated intra-articular soft-tissue or osseous pathology.[15]

Nuclear medicine

The combination of single-photon-emission CT (SPECT) and low-dose CT: SPECT/CT, is primarily used to quantify the differential growth of the condyles when unilateral condylar hyperplasia is suspected in adolescents with mandibular asymmetry. Uptake differences of 10% or more between the left and right condyles, with increased uptake on the side of condylar hyperplasia, are considered to be evidence of active growth.[16] This dictates the optimal time for corrective maxillofacial surgery to be performed.

Arthrography

Imaging-guided injection of contrast medium into the TMJ and subsequent fluoroscopy/radiography, CT, or MRI is no longer advocated for the evaluation of TMD because it is invasive, uncomfortable, technically demanding, and may involve a significant radiation dose.

Anatomy and function

The TMJ is a ginglymoarthrodial (i.e., both a hinge and gliding) synovial joint between the condyle of the mandible and the glenoid fossa and articular eminence of the squamous temporal bone. It has a fibrous capsule, external ligaments, lining synovial membrane, and is divided into two compartments by a fibrocartilaginous disc (Fig 2).

Uniquely, the articular surfaces are covered by fibrocartilage rather than hyaline cartilage. Normal fibrocartilage and articular cortex cannot be separated on MRI sequences; they are seen as a hypointense band of uniform thickness. In contrast, MDCT, CBCT, and radiographs depict only the cortex so the “white” outline of the articular surfaces is thinner than the “black” outline shown by MRI. On cross-sectional imaging, the mandibular condyle is oval in the axial plane, round in the sagittal plane, and it has a convex to mildly flattened contour in the coronal plane.[17] In contrast, the temporal articulation is S-shaped with the articular eminence being rounded in the sagittal plane (Fig 3).

The disc is biconcave in the sagittal dimension with an anterior band, a slightly thicker posterior band, and a thin intermediate zone; a normal disc is of uniform low signal on

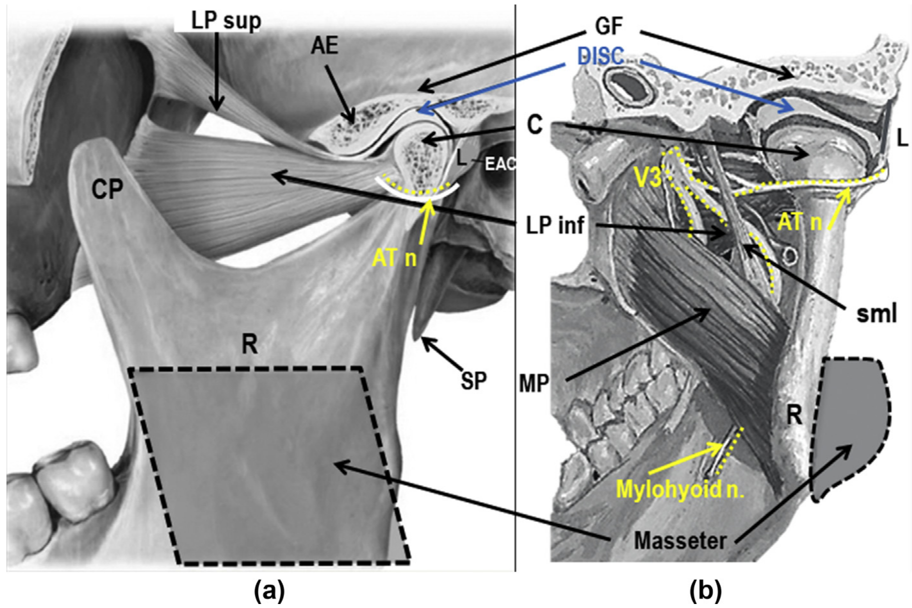


Figure 2 Anatomy of the temporomandibular joint region. (a) Sagittal (viewed from the lateral aspect) and (b) coronal (viewed from the posterior aspect) diagrams of the key osseous, muscular, ligamentous, and neural anatomy of the TMJ and surrounding structures. The left mandibular ramus (R), condyle (C) and coronoid process (CP) are indicated as is the glenoid fossa (GF) and articular eminence (AE) of the squamous temporal bone, and the styloid process (SP). Only the inferior portion of the masseter muscle is shown (grey shapes with a black dashed outline) and the medial pterygoid (MP) is visualised on the coronal diagram. The superior portion of lateral pterygoid (LP sup) inserts into the disc (D) and the inferior portion (LP inf) into the condylar neck. The lateral, intrinsic ligament (L) and one of the two extrinsic ligaments: the sphenomandibular ligament (sml), are shown. Innervation (yellow dotted outline) of the TMJ, the anterior wall of the external auditory canal (EAC) and pre-auricular skin is from the auriculotemporal nerve (AT n) which is a branch of the mandibular division of the trigeminal nerve (V3). V3 exits the skull base via the foramen ovale and passes inferolaterally to enter the mandibular foramen on the medial (lingual) surface of the mandible.

proton density and T2-weighted MRI sequences. The disc is firmly attached to the medial and lateral poles of the condyle and anteriorly to the joint capsule and the superior tendon of lateral pterygoid (Fig 3a,d).

The bilaminar zone (BZ) forms the posterior disc attachment; it extends posteriorly from the posterior band and divides into superior and inferior laminae, which attach to the squamous temporal bone and condylar neck, respectively. Between these fibroelastic laminae is richly innervated and vascularised connective tissue; together, they are known as the retrodiscal soft tissue. The BZ elongates with mouth opening as the condyle translates anteriorly; upon closing the mouth, the BZ aids in retraction of the disc as the condyle returns to the glenoid fossa (Fig 3b). Opening the mouth leads to distension of the venous plexus within the retrodiscal soft tissue accounting for T2 hyperintensity and enhancement with gadolinium as a normal finding.

The normal position of the disc is usually defined such that the junction of the posterior band and the BZ is at the 12 o'clock position on an oblique sagittal MRI scan (Fig 3a). Using this criterion, MRI studies of asymptomatic patients demonstrate mild DD in 34%. Therefore, it has been suggested that minor degrees of DD are probably a normal variant and the normal position should be defined as 11 to 12 o'clock.[2,3]

To open the jaw, the condyle rotates on the inferior surface of the disc in the inferior joint compartment (hinge) followed by a translation of the condyle and attached disc over the articular eminence in the superior joint compartment (gliding). The narrowest point of the disc: the intermediate zone corresponds to the narrowest inter-bony distance between the maximum convexity of the condyle and articular eminence, throughout the opening and closing cycle. The superior convexity of the condyle should be situated directly inferior or just anterior (1–2 mm) to the maximum convexity of the articular eminence during maximum opening (Fig 3b).

Lateral pterygoid (LP) is the principal muscle involved in opening the mouth, supported by several suprahyoid muscles. The tendon of the superior head of LP primarily inserts into the disc and the inferior tendon inserts into the pterygoid fovea situated on the anterior surface of the condyle (Figs 2 and 3a,b). The temporalis, medial pterygoid, and masseter muscles close the jaw. Innervation of the TMJ is from the auriculotemporal branch of V3 (Fig 2) supplemented by its temporal and deep masseteric branches.[1,10]

Aetiology of TMD

The aetiology of TMD is multifactorial and complex. Biological, psychological, and social factors, usually in combination, contribute to the development of the signs and symptoms of TMD.[1,18] Trauma, either as a single initiating episode such as prolonged mouth opening during a

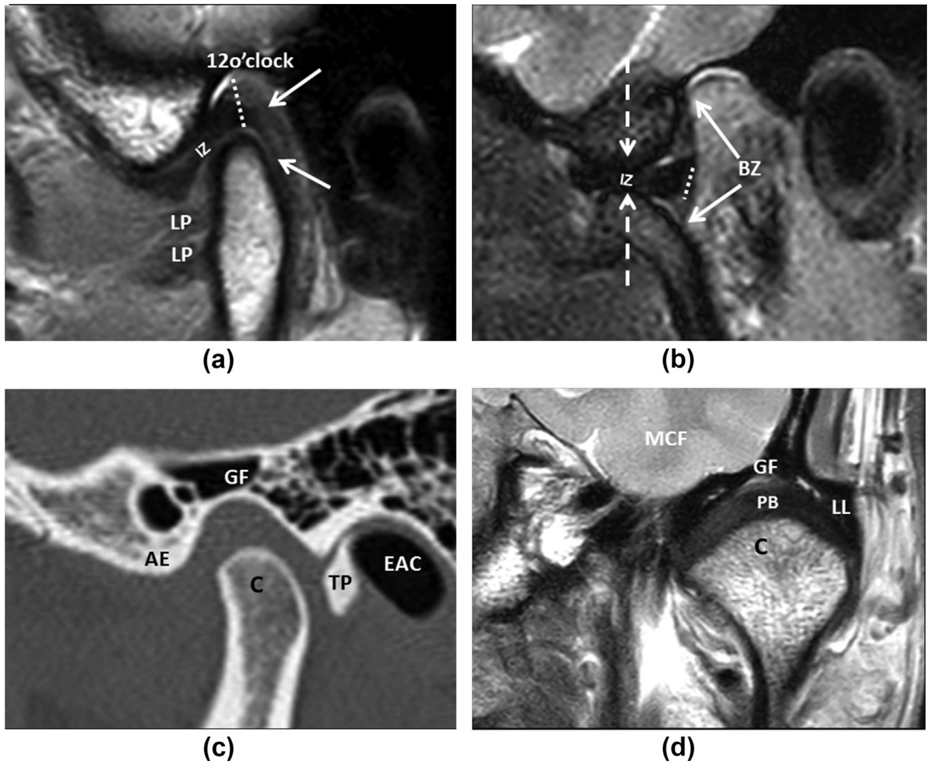


Figure 3 Radiological anatomy. (a) A proton-density weighted oblique sagittal image through the mid-portion of the TMJ demonstrating the normal soft-tissue and osseous anatomy. The disc is of low signal and shaped like a bow-tie with a thin intermediate zone (IZ). The junction (white dotted line) of the posterior band of the disc and the low signal laminae (white arrows) of the bilaminar zone (BZ) is at 12 o'clock. The lateral pterygoid tendons are indicated (LP). The low-signal outline of the condyle represents the combined layers of cortical bone and the articular fibrocartilage. Normal fatty marrow in the condyle and temporal sides of the joint is of high signal on PD sequences unless the temporal surface is pneumatised. (b) A fast T2 scan in the open-mouth position. The disc is positioned between the condyle and the articular eminence with a normal range of translation (mobility) of the condyle. The superior and inferior laminae of the BZ are widely separate with intervening, relatively high signal soft tissue (white arrows). (c) The normal osseous anatomy of the TMJ depicted by an oblique sagittal reconstruction from MDCT. The articular eminence (AE) and glenoid fossa (GF) form the temporal side of the articulation. The condyle (C), the external auditory canal (EAC) and tympanic plate (TP) of the temporal bone are also indicated. (d) A slightly oblique coronal PD scan through the mid-portion of the left TMJ. The posterior band (PB) of the disc, thickened lateral capsule (LL, lateral ligament), glenoid fossa, middle cranial fossa (MCF) and condyle (C) are shown.

dental procedure, or repetitive low-grade trauma is a common trigger for TMD.

Muscles of mastication and myalgia

A recent review article evaluated research using ultrasound and MRI of patients with masticatory myalgia/TMD.[19] The masseter is usually the only muscle that is evaluated, being a superficial structure that is easily evaluated by ultrasound. In patients with TMD, the masseter enlarges with a reduction in the number and increased thickness of echogenic intra-muscular septae. Sonoelastography demonstrates a raised elastography index in the masseter muscles of TMD patients as compared with controls and MRI using magnetisation transfer contrast and diffusion-tensor imaging, has shown an increase in water content of painful master muscles consistent with oedema.

Excessive clenching or grinding of teeth (bruxism), especially during sleep, results in hyperactivity of the muscles of mastication and attrition of the dentition. Up to 48% of patients with sleep bruxism have a sleep disorder, including obstructive sleep apnoea.[20] Chronic bruxism is associated with hypertrophy of the muscles of mastication as demonstrated by ultrasound, MDCT, and MRI (Fig 1a). Hypertrophy is usually symmetric; asymmetric hypertrophy of the masseter may be confused with enlargement of the anterior aspect of the parotid gland on clinical examination.

A haematoma may follow inadvertent injection of local anaesthetic into a muscle of mastication for regional anaesthesia before a dental procedure. This includes medial pterygoid (complicating an inferior alveolar block) or temporalis (complicating a posterior superior alveolar block) for anaesthesia in the mandible and maxilla, respectively (Fig 1b).

Internal derangement

Internal derangement is defined as interference with a joint's smooth action and is most commonly due to DD. Moderate to severe DD, rather than mild displacement, is associated with articular surface remodelling, intra-articular inflammation and degeneration, deformity and perforation of the disc. OA, other arthritides and miscellaneous causes, such as intra-articular bodies and adhesions, are additional causes of internal derangement.

DD

The TMJ can withstand a high biomechanical load during normal function. Excessive load causes a deterioration of the structural integrity of the posterior band of the disc and the BZ leading to laxity and DD.[17] In early internal derangement, the DD is recaptured on mouth opening and this is usually associated with a "click," which is frequently audible. A normal relationship of the disc to the condyle is present in the fully open mouth position (Fig 4a and b.) Symptoms are mild and patients infrequently seek medical attention.

If internal derangement is more marked, the DD is not recaptured on mouth opening and it acts as a mechanical block to anterior condylar translation limiting mouth opening, which is described by clinicians as a "closed lock" (Fig 4c and d). In addition to limited mouth opening, pain is generally more severe and patients more commonly seek medical attention.

MRI is the reference standard imaging technique for the assessment of DD. MDCT and ultrasound can be used for the same purpose but are less accurate. DD in the closed mouth position is most commonly anterior (44%) or anterolateral (29%) with anteromedial, medial, and lateral displacement being uncommon.[11] A comparison of the sagittal and coronal sequences is essential to determine the degree of rotational or sideways displacement. If the posterior band is anterior to the anterior margin of the condyle on all sagittal images from lateral to medial, DD is designated as complete. Partial DD describes the displacement of the disc in a portion of the TMJ, usually the lateral aspect. An open mouth sequence is performed to assess disc recapture and the degree of condylar translation.

Remodelling

If functional loading of the TMJ chronically matches the biological threshold of the fibrocartilage and the articular surfaces, morphological changes occur to adapt and distribute these forces over a large surface area. On imaging, this is demonstrated by thickening, sclerosis, and flattening of the articular surfaces, especially the condyle (Fig 5a).[2,17] Remodelling of the articular surfaces does not progress if the functional load and adaptive capacity of the TMJ remain in balance. Articular surface remodelling is commonly seen in older patients with few or no symptoms.[2]

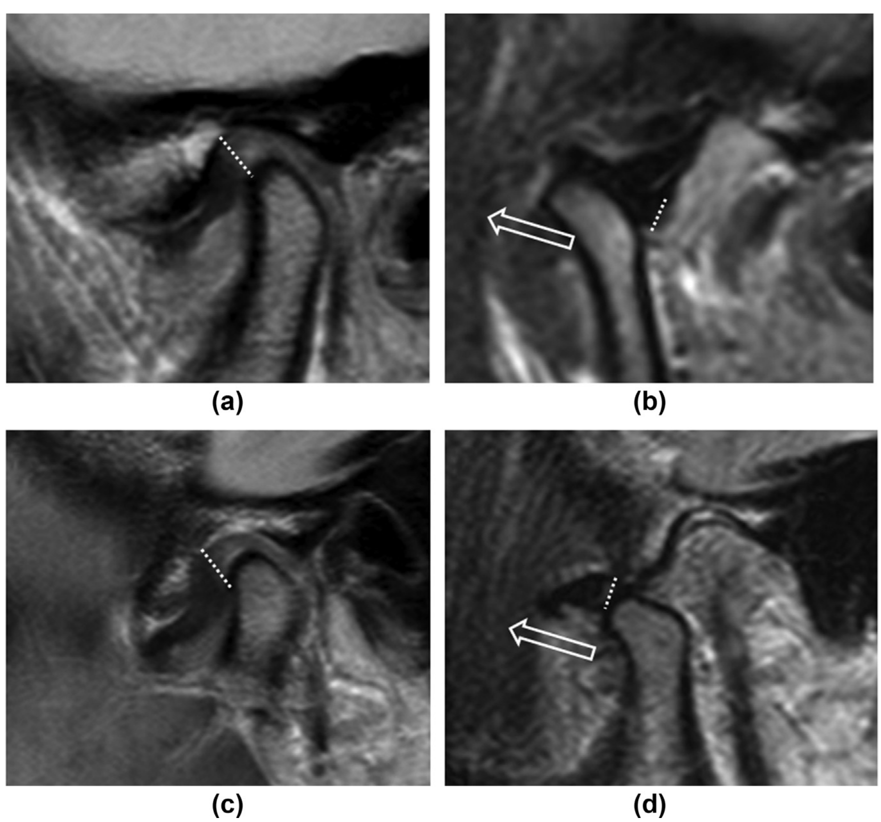


Figure 4 Common patterns of disc displacement. (a) There is complete anterior disc displacement (PD sagittal oblique) with (b) recapture on mouth opening (T2 sagittal oblique in open mouth position); the condyle is slightly hypermobile (white open arrow). (c) Complete anterior disc displacement (DD) is more marked within this joint (PD sagittal oblique). (d) With mouth opening, the disc is not recaptured (T2 sagittal oblique, and becomes progressively further anteriorly displaced). There is a normal range of condylar translation.

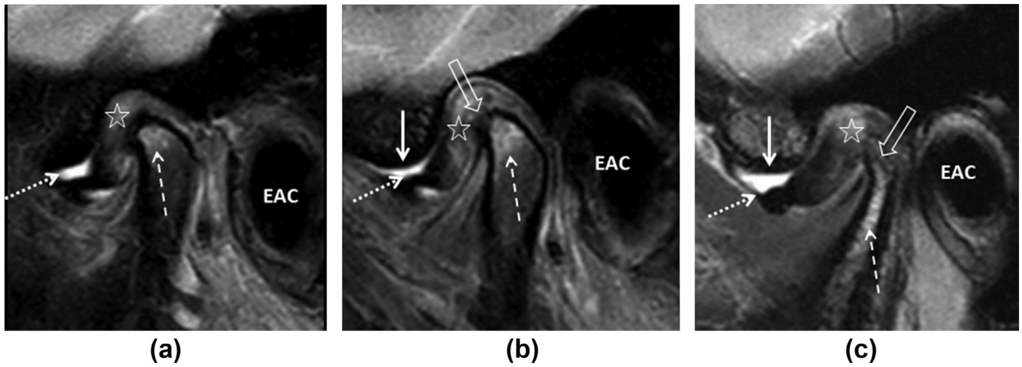


Figure 5 Complete disc displacement, intra-articular inflammation and condylar erosions. Three different examples of complete anterior DD, swelling of the posterior band of the disc and intra-articular inflammation are shown on fat-saturation T2 sagittal images. (a) Small effusions are present in the anterior recesses of the superior (white dotted arrows) and inferior joint spaces. There is mildly hyperintense tissue/synovitis superior to the condyle involving the BZ (white star). Flattening and thickening of the condylar articular surface are consistent with remodelling. There is focal sub-articular marrow oedema (white dashed arrow). (b) There is a moderate-sized effusion in the anterior recess of the superior joint space (white dotted arrow), synovitis in the inferior joint space extending inferior to the BZ (white star), subtle erosion of the condyle (white open arrow) and flattening of the inferior surface (white arrow) of the low-signal articular eminence (suppressed signal from fatty marrow and/or sclerosis). There is focal sub-articular hyperintensity of marrow in the condyle (white dashed arrow). (c) Extensive condylar resorption is present in an adolescent female consistent with idiopathic (progressive) condylar resorption (white open arrow). A moderate-sized effusion is shown in the superior joint space (white dotted arrow) with extensive oedema/synovitis of the retro-discal soft-tissues (white star). There is remodelling of the articular eminence (white arrow) and diffuse marrow oedema in the condyle (white dashed arrow).

Intra-articular inflammation

Further increase in functional loading causes intra-articular biochemical alterations leading to oxidative stress and the generation of free radicals, synovitis, and an effusion; the joint fluid has altered viscosity and contains inflammatory cytokines. MRI, ultrasound, and to a lesser extent MDCT, can demonstrate effusions but only MRI can depict synovitis and oedema of the retrodiscal soft-tissues including the BZ (Fig 5). Excess joint fluid and synovitis are strongly associated with TMJ pain.[21–26]

Intra-articular adhesions are a complication of intra-articular inflammation. They are present in 5–20% of cases of internal derangement and are implied on MRI when disc movement is absent between the open and closed mouth sagittal sequences.[4]

Morphology of the disc, BZ, and lateral pterygoid tendon

Complete DD without reduction is commonly associated with anterior or anterolateral crumpling of the disc, a swollen posterior band of increased signal (Fig 5), an attenuated anterior band and occasionally, perforation of the disc or the junction of the disc and BZ (Fig 6).[3,4,10,11,26] In joints involved by OA, the disc may be small, biconvex in shape, fragmented, and detached from a retracted BZ.

Oedema of the retrodiscal soft tissue on closed mouth fat-saturation T2 sequences occurs in painful DD without

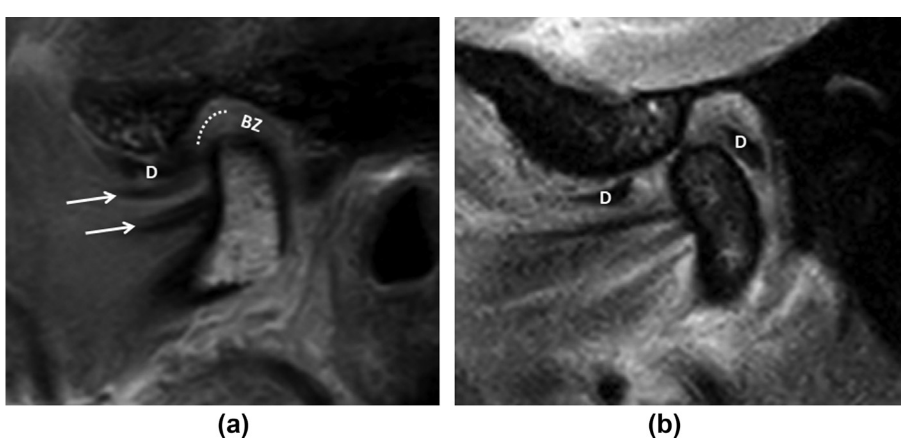


Figure 6 Abnormal disc. (a) Oblique sagittal fat-saturation T2 through the medial aspect of the condyle. There is complete anteromedial disc displacement (D); the disc is crumpled and separate from the thin BZ (white dotted curved line). There is hypertrophy of the superior and inferior tendons of the lateral pterygoid muscle (arrows) giving a “triple disc” appearance. (b) An elderly female patient presented with an acute “closed lock” whilst chewing. The intermediate zone of the disc is torn and the fragments are displaced anteriorly and posteriorly (D) secondary to the pull of LP and the BZ respectively.

reduction, usually with other imaging evidence of intra-articular inflammation (Fig 5). Low-signal thickening of the superior (temporal) lamina of the BZ is a described feature of chronic and severe DD giving a "pseudo-disc" appearance, which is probably due to fibrosis.[11,24] In our experience, plication or cauterisation of the lamina at arthroscopy can give an identical appearance.

Hypertrophy of the tendons of the lateral pterygoid may be seen with significant and chronic DD especially when there is a component of medial displacement; this gives a "double" or "triple disc" appearance (Fig 6a).[11,24]

OA/degenerative joint disease (DJD)

Chronic and excessive functional loading of the TMJ that exceeds the adaptive capacity of the TMJ leads to accelerated loss of articular cartilage. It is currently accepted that OA is an inflammatory, as well as a degenerative process[2,6,27]; both factors lead to cortical defects (erosions) and subarticular marrow oedema (Figs 5b and 7b). Marrow oedema is strongly associated with joint pain.[21,23–26]

Synovitis, cortical erosions, and marrow oedema predominate in the inferior joint space and condyle (Fig 5) for reasons that are not entirely understood. This inferior compartment predominance is probably related to different biomechanical forces in the inferior and superior joint spaces as they allow different movements: rotation and translation of the condyle, respectively. Degeneration and deformity of a displaced disc primarily decrease the cushioning effect on the articular surface of the condyle to which the disc is firmly attached; this is analogous to meniscal injury in the knee.[27]

The imaging features of the proliferative phase of OA in the TMJ are articular surface flattening, sclerosis, cortical thickening and irregularity, osteophytes, subcortical lucencies, and ossified intra-articular bodies (Fig 7c). The severity of imaging findings is more marked within the condyle than the temporal articular surface.[6,9] OA involving the TMJ is often asymptomatic apart from joint noises and low-grade pain7. There is a recognised association between high-grade DD and OA; both are significantly more common in females with OA predominating in an older age-group than DD.[28]

Osteonecrosis

Osteonecrosis of the condyle results from impaired blood flow secondary to DD leading to marrow oedema and increased intra-osseous pressure. If extensive, condylar collapse results and OA develops. Condylar osteonecrosis should be suspected when there is marked subarticular sclerosis on CBCT/MDCT (reduced marrow signal on T1) and hyperintensity or heterogeneous marrow signal on a fat-saturation T2 sequence.[2,11]

Idiopathic condylar resorption

This condition, also known as progressive condylar resorption, represents an accelerated process of condylar erosion and resorption that occurs in adolescent females with DD who usually have a small or retrognathic mandible (Fig 5c). Hormonal influences are thought to make the condyle vulnerable to excessive biomechanical load.[29] Following active resorption, there is remodelling leading to small condyle with a flattened articular surface. The principal differential diagnosis is juvenile idiopathic OA, which occurs in a similar age group.

Juvenile idiopathic arthritis (JIA)

JIA is the most common rheumatological condition in childhood and adolescence and is commoner in girls. It should begin before 16 years of age and last for at least 6 weeks. Involvement of the TMJ occurs in up to 87% of patients with JIA and is most common with long disease duration, young age at disease onset, and in polyarticular or systemic sub-types. TMJ involvement is highly significant because the mandibular growth plate is located inferior to the articular fibrocartilage and is susceptible to damage

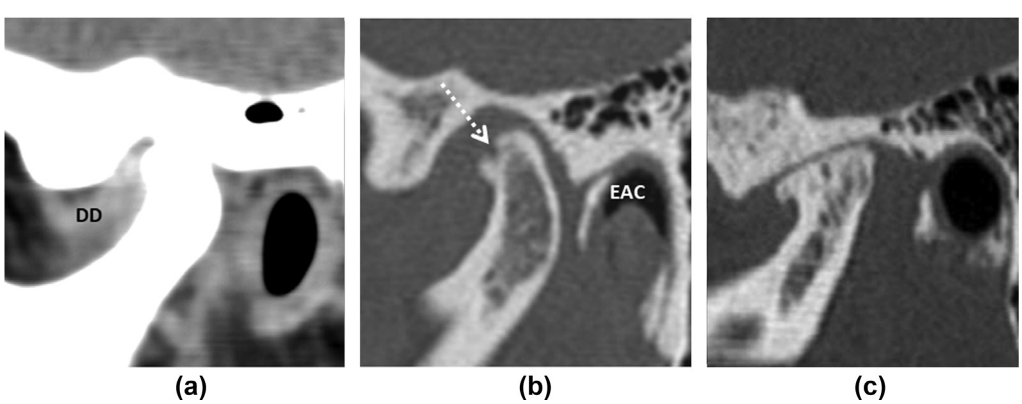


Figure 7 OA. (a) An oblique sagittal soft-tissue reconstruction demonstrating complete anterior DD. (b) A bone window reconstruction of the same joint as in (a) demonstrates early OA with a small erosion (white dotted arrow) within the thickened articular surface of the condyle. The condyle is posteriorly situated within the glenoid due to the pronounced anterior DD. The opacity within the EAC represented a plug of cerumen. (c) Marked OA in a different patient shown on an oblique sagittal bone-window MDCT reconstruction. There is diffuse joint space loss, articular surface flattening, sclerosis, and an anterior condylar osteophyte and small subcortical lucency.

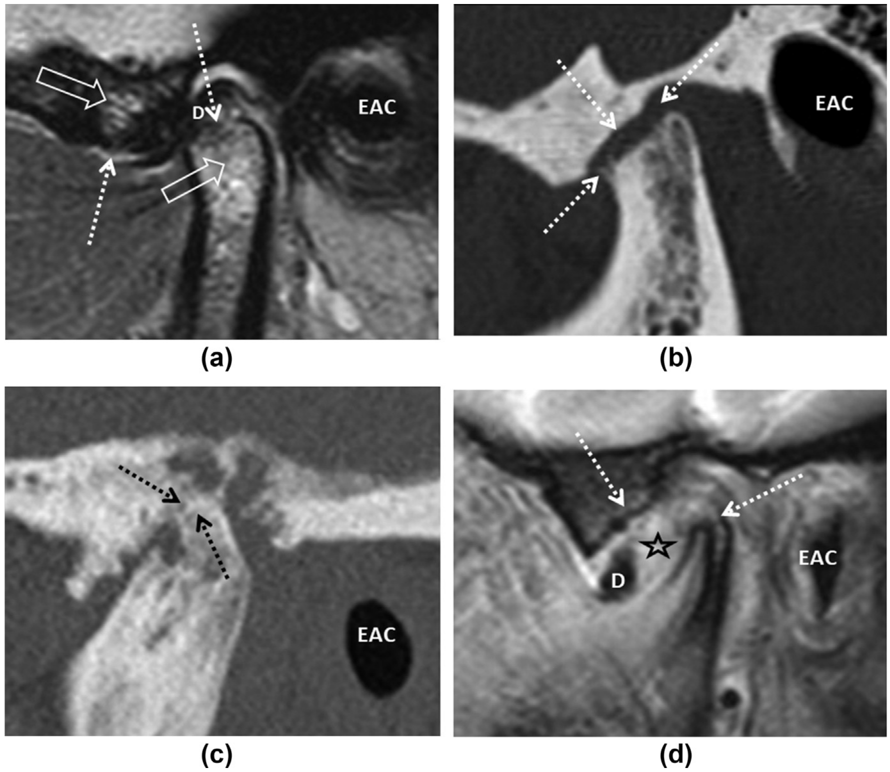


Figure 8 Erosive arthritides. (a) A fat-saturation T2 oblique sagittal image of a 16-year-old female patient with JIA who presented with TMJ pain. There are small effusions in both joint spaces outlining a normally positioned disc. Erosion of the condyle and to a lesser extent the articular eminence (white dotted arrows) is associated with subarticular marrow oedema (white open arrows). (b) Diffuse erosion of both articular surfaces of the TMJ is demonstrated on an oblique sagittal reconstruction from an MDCT scan with long-standing rheumatoid arthritis (RA) who re-presented with TMD. There is marked sclerosis of the temporal articular surface and cortical thickening of the condyle. (c) Fibrous and osseous ankylosis (black dotted arrows) of the TMJ in a patient with longstanding, polyarticular RA is demonstrated on an oblique sagittal MDCT reconstruction. There had been a progressive restriction of mouth opening for at least 12 months with no anterior condylar translation when the scan was repeated with attempted mouth opening. (d) A fat-saturation T2 oblique sagittal T2 image shows "arthritis mutilans" with gross erosion and resorption of both articular surfaces (white dotted arrows) of the TMJ in a patient with active RA and TMD. There is marked anterior displacement of a biconvex disc (D) with hyperintense synovitis distending the expanded joint (black star).

from inflammation. Untreated bilateral JIA leads to a small mandible (retrognathism) and, if unilateral, to mandibular asymmetry.[11,30]

Seventy percent of patients with TMJ involvement are asymptomatic and imaging plays a key role in the diagnosis and monitoring of treatment. MRI, with gadolinium, is the reference standard in the assessment of the TMJ. Imaging features can be divided into acute and chronic changes. Acute findings include an effusion, synovial enhancement and thickening, enhancing erosions, and bone marrow oedema (Fig 8a). Chronic changes include pannus, flattening, loss of volume and irregularity of the articular surfaces, disk deformity and displacement, and hypertrophic bone formation.

Diffusion-weighted imaging (DWI) can distinguish between active and inactive JIA in the knee based on ADC values and correlates closely with gadolinium-enhanced studies[31]; this may not have the same accuracy in relatively small joints such as the TMJ.

Ultrasound of paediatric joints is operator dependent, and the developing skeleton poses unique challenges in interpretation with sonographic findings that can mimic pathology and vice versa.[32] Only the superficial aspect of the TMJ can be evaluated by ultrasound and the results from available studies are inconclusive about the value of this technique in JIA.[33]

Rheumatoid arthritis (RA)

RA is a chronic inflammatory polyarticular disease most common among women in the fifth decade. It is the most common inflammatory arthritis in adults to affect the TMJ, and 50–75% of patients with RA have TMJ involvement.[33] Active RA leads to synovial proliferation, excess joint fluid, articular surface erosion on both sides of the joint and marrow oedema (Fig 8a and b). Intra-articular inflammation tends to be more pronounced in RA as compared with the arthropathy associated with severe DD. DD is not

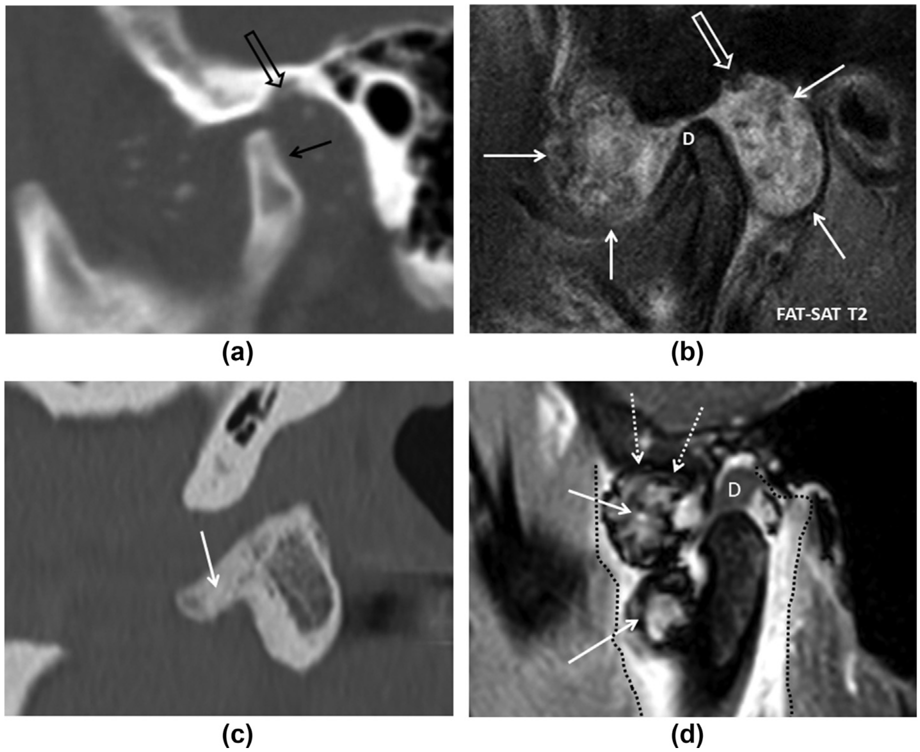


Figure 9 Proliferative disorders and benign neoplasms. (a) An oblique sagittal MDCT reconstruction and (b) fat-saturation T2 sagittal sequence demonstrate synovial chondromatosis. There is marked distension of the superior joint space (white arrows in b) by synovial proliferation containing chondral bodies, some of which are calcified. There is focal erosion of the glenoid fossa (open arrows) and smooth scalloping of the posterior aspect of the C head (black arrow in a). (c) A lateral positioned, oblique sagittal MDCT reconstruction demonstrates an osteochondroma (white arrow) arising from the anterior aspect of the condyle. (d) Pigmented villonodular synovitis of the TMJ demonstrated on a post-gadolinium T1 sagittal sequence. Two large round foci anterior to the condyle (white arrows) demonstrate heterogeneous enhancement with the superior focus having a virtually complete low-signal haemosiderin rim. There is florid intra-articular, capsular and peri-capsular enhancement (black dotted outline). The inferior aspect of the articular eminence is eroded (white dotted arrows) and the disc (D) is posteriorly displaced.

infrequent in RA, secondary to weakening of the disc attachments and is not a reliable distinguishing feature.

Imaging signs of chronic disease depicted by all techniques include gross loss of volume and deformity of the articular surfaces, marked joint space narrowing, and secondary OA.[11,33] The most severe outcome is joint destruction (arthritis mutilans) and ankylosis (Fig 8c and d).

Other arthritides

The deposition of calcium pyrophosphate dihydrate (CPPD) in articular cartilage, synovium and the capsule of a joint constitute chondrocalcinosis and are associated with degenerative changes and prominent subchondral “cysts”.[11] As with CPPD arthritis affecting other joints, the patients may present with acute onset of pain, limited function, and swelling secondary to the shedding of crystals into the joint. A rare presentation is of a painless tumour-like mass secondary to extensive chondrocalcinosis within hypertrophied synovium associated with diffuse and irregular erosion of both articular surfaces.[34]

Seronegative arthritides may involve the TMJ. In addition to erosions, psoriatic arthritis is characterised by periosteal new bone formation.[11]

Septic arthritis of the TMJ can arise from a penetrating wound, as a complication of a therapeutic joint injection/procedure or as an uncommon complication of acute otitis media in children[35] or otitis externa in immunocompromised adults (necrotising external otitis).

Condylar hypermobility and dislocation

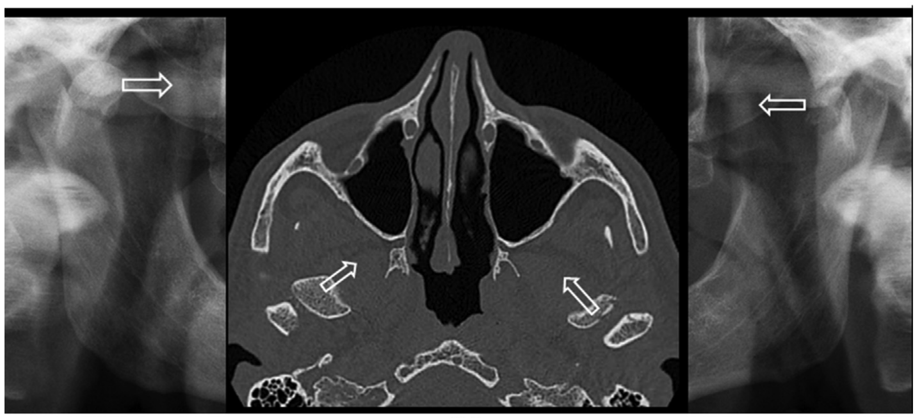
Hypermobility

Hypermobility is defined on imaging as a condyle that moves >2 mm anterior and superior to the crest of the eminence. It probably reflects developmental laxity of the ligaments and capsule of the TMJ (Fig 4b). If the condyle cannot be relocated posteriorly into the glenoid fossa without manipulation, it is described by clinicians as an “open-lock”.

Dislocation

Dislocation (luxation) is defined as the excessive and fixed displacement of the condyle anterior to the articular eminence secondary to an episode of trauma or wide mouth opening. Imaging is rarely required in the acute situation, but if performed demonstrates that the condyle is situated

(a)



(b)

Figure 10 Condylar fractures. (a) There is an undisplaced, low left condylar neck fracture (black arrows), which is shown on cropped images of the condyles and rami from DPT and a central posteroanterior radiograph. (b) Cropped images of the rami and condyles from DPT and central axial bone-window axial MDCT demonstrate bilateral high condylar neck/head fractures. The fragments attached to the lateral pterygoid muscles are displaced anteromedially by the action of these muscles (white open arrows). MDCT is essential for surgical planning; in older patients, these fractures are often treated conservatively.

anterior to the articular eminence in both the open and attempted closed-mouth radiographs.

Ankylosis

Ankylosis of the TMJ can be fibrous or bony and is usually secondary to an insult to the joint. Trauma (haemarthrosis) is the most common cause, followed by inflammatory arthritides (Fig 7d) and previous joint surgery and infection.

Cysts, proliferative disorders and neoplasia

Synovial cysts (ganglion)

These rare cysts have a synovial lining and are usually asymptomatic findings on imaging; occasionally, they present with pain or a pre-auricular mass.[36]

Synovial chondromatosis

Synovial chondromatosis is a metaplastic condition of the synovium. In the TMJ, it is commoner in females and involves the superior joint space. The characteristic imaging appearance is distension of the superior joint space by fluid containing numerous small chondral bodies of similar size, which progressively calcify (Fig 9a and b). There are usually erosions and sclerosis of the glenoid fossa and articular eminence.[11] Recurrence after excision is not uncommon; malignant transformation and intracranial extension are rare.

Pigmented villonodular synovitis

This is a neoplastic synovial process; the synovium thickens and contains haemosiderin causing the characteristic reduced T2 signal on MRI (Fig 9c). Extensive erosion of the articular surfaces and cyst formation are also characteristic. Intracranial extension occurs in 33% of cases.[37]

Benign tumours

Osteochondromas are the commonest tumour, and tend to arise from the anterior aspect of the condyle and may be confused with a bifid condyle or large osteophyte (Fig 9d). Osteomas arise from the non-articular surface of the condyle.[9,17]

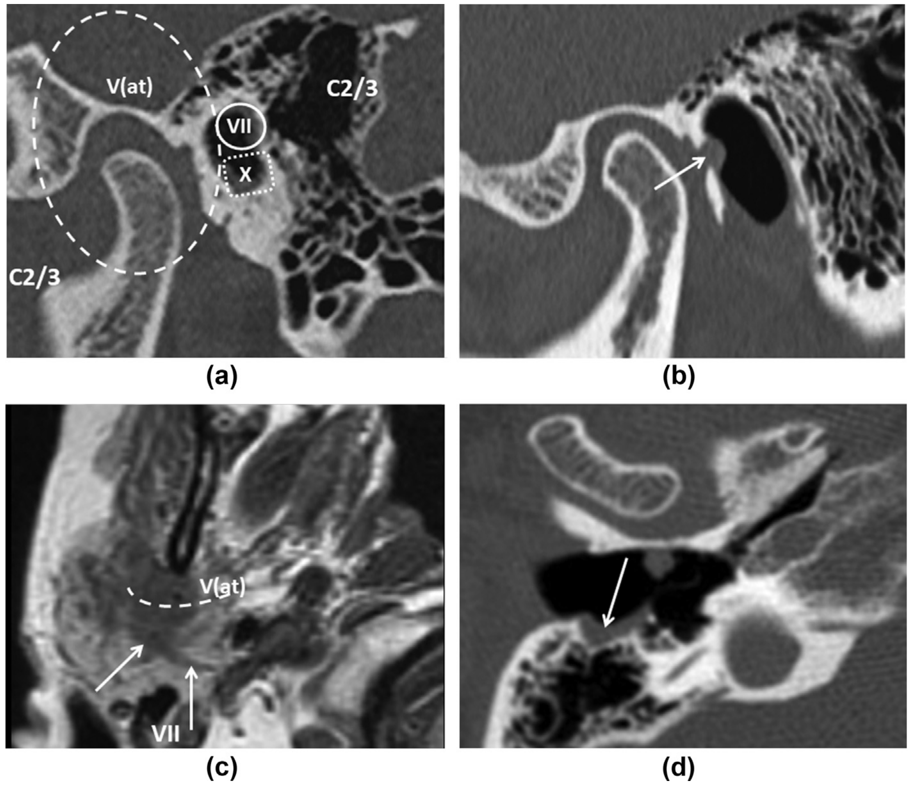


Figure 11 The TMJ and the ear. (a) Several cranial nerves (trigeminal: V, facial: VII, and vagus: X) innervate the external auditory canal (EAC) and the cervical plexus (C2/3) supplies the skin situated anteroinferior and posterior to the pinna. The auriculotemporal branch of the V nerve (V(at)) is the principal sensory supply to the TMJ, the anterior wall (tympanic plate) of the EAC, and skin situated anterosuperior to the pinna. The complex innervation of this region explains why it may be impossible, on clinical grounds, to distinguish between otalgia and pain originating in the TMJ or referred pain from other structures in the neck, pharynx or oral cavity innervated by the same cranial nerves or cervical plexus. (b) The patient presented with a sensation of irritation and noise in the ear, which was worse on chewing. Otoscopy demonstrated a small cyst along the anterior wall of the EAC, which changed in size with opening and closing the mouth. An oblique sagittal reconstruction from MDCT can showed focal protrusion of a "hernia" of the TMJ capsule via a developmental defect in the anterior wall of the tympanic plate (white arrow). (c) A middle-aged patient was investigated for pain, thought to be of TMJ origin, with CBCT (not shown); no abnormality was demonstrated. MRI was requested when pain progressed, there was paraesthesiae of skin situated anterior to the EAC and subtle facial (VII) nerve weakness. A T1-weighted axial MRI scan of the right parotid gland shows an irregular mass involving the auriculo-temporal nerve (V(at): white curved, dashed line) with perineural tumour spread along the right VII nerve (white arrows). Ultrasound-guided biopsy demonstrated undifferentiated carcinoma. (d) MDCT was requested by an oral medicine specialist for investigation of pain in the TMJ region without other symptoms of TMD. There is focal erosion of the posterior wall of the EAC on an axial, bone-window reconstruction of the right temporal bone from an MDCT scan. The defect is filled with soft-tissue, which represented cholesteatoma (white arrow). Focal debris was also present along the anterior wall of the EAC.

Osteoid osteoma and osteoblastoma rarely involve the TMJ and predominantly involve the temporal joint surface. Symptoms, especially pain, may be confused with TMD.

Malignant tumours

The most common malignant tumours involving the TMJ are sarcoma and metastatic disease.[11]

Developmental disorders

Bifid condyle

A bifid condyle is a rare entity characterised by partial division of the mandibular condyle. It can be developmental or acquired; the latter is usually post-traumatic.

Condylar hypoplasia and aplasia

Hypoplasia is a developmental abnormality in which the condyle is smaller than the contralateral side because of a thinner layer of, or insult to, the condylar growth centre. The condyle is of normal morphology but small, and the ramus is usually shorter than the normal side resulting in mandibular asymmetry and deviation of the mandibular midline to the affected side. Aplasia of the condyle is seen in several craniofacial syndromes, notably hemifacial microsomia.[11,17]

Condylar hyperplasia

Hyperplasia results from overactivity of the condylar growth centre and is most commonly idiopathic. The condyle and ramus are elongated and the affected hemimandible may be enlarged. The occlusal plane is tilted inferiorly on the affected side and the mandibular midline deviates to the contralateral side. Lateral bowing of the affected ramus and an inferior convex border of the mandibular body on the affected side are important diagnostic features.[17]

Trauma

Fractures of the condyle may be subtle on plain radiographs and are easily missed. The DPT provides a lateral view of the condyle and should be supplemented with posteroanterior and open-mouth reverse Towne's views (Fig 10 superior). MDCT or CBCT should be performed if the radiographs are equivocal and there is a strong clinical suspicion of a fracture.[9]

Condylar fractures account for 25–50% of mandibular fractures and are classified as condylar neck (low, medium, or high) and condylar head (extra- or intracapsular). If the fracture is displaced, there is anteromedial displacement of the main condylar fragment secondary to the pull of the lateral pterygoid muscle (Fig 10 inferior). The disc is displaced with the condyle to which it is firmly attached and soft-tissue trauma (disc, retrodiscal soft tissue and capsule) is more pronounced than with undisplaced fractures.[38]

Malunited condylar fractures may cause a bifid appearance of the condyle when they occur in the growing skeleton; the condylar growth centre forms a neo-condyle, which is lateral to the displaced condylar fragment.

The TMJ and the ear

The squamous temporal bone forms the superior articular surface of the TMJ and branches of V3 innervate the muscles of mastication, the joint, the anterior wall of the external auditory canal (tympanic plate) and a portion of the middle ear, including the tensor tympani muscle (Figs 2 and 11a). Small ligaments connect the disc and the malleus within the middle ear.[39] A developmental defect in the medial aspect of the anterior wall of the tympanic plate (foramen of Huschke) can allow posterior bulging of the TMJ capsule into the external canal, which is visible on otoscopy (Fig 11b).

The prevalence of otological signs and symptoms in adult patients with TMD is high.[40] The most prevalent symptom is ear fullness (75%), followed by otalgia (55%) and tinnitus (52%). The TMJ is an important review area for patients being imaged with otological signs and symptoms and vice versa (Fig 11c and d).

Table 2
Imaging of the temporomandibular joints (TMJs): a guide to reporting.

1. Disc (MRI >> MDCT)
   - Position: normal or displaced
   - Severity of DD: partial or complete
   - Direction of DD: anterior > anterolateral > anteromedial > medial/lateral >>posterior
   - Recapture: not recaptured OR partial or complete recapture on mouth opening
   - Morphology, normal: black "bow-tie" shape, no internal signal, smooth contour
   - Morphology, abnormal: increased signal, abnormal shape, small or large, fragmentation
2. Condylar translation (MRI, MDCT + CBCT)
   - Normal, reduced (hypomobile) or excessive (hypermobile)
   - Grade abnormal mobility: mild – moderate – marked
3. Intra-articular inflammation (MRI >>MDCT: large effusions, calcification)
   - Joint fluid: normal trace of fluid increased mild – moderate – marked effusion
   - Synovitis: less hyperintense than fluid on T2; early post-Gd sequences are optimal
   - Oedema of the retrodiscal soft tissue: assess on closed mouth fat-saturated T2
4. Articular surfaces (MDCT + CBCT > MRI)
   - Normal: continuous "black/white line", preserved convexity of condyle and articular eminence
   - Abnormal: involvement of inferior (condyle), superior (temporal) or both joint spaces
   - Remodelling: flattening and cortical sclerosis + thickening
   - OA: erosions (condyle), cortical thickening + sclerosis, osteophytes, narrow joint space
   - Primary erosive arthropathies: erosions on both sides of the joint
5. Marrow (MRI >> MDCT + CBCT: sclerosis)
   - Normal: high signal on T1 and PD sequences; uniform reduction in signal on fat-saturated T2
     the articular eminence may be of low signal on all sequences if pneumatised
   - Abnormal: increase in signal on fat- saturated T2 due to oedema
     decrease in signal on T1 due to sclerosis
     heterogeneous signal due to osteonecrosis
6. Muscles of mastication (MRI> MDCT)
   - Usually normal
   - Oedema, hypertrophy or atrophy should be excluded
7. Review areas (MRI + MDCT >> CBCT: temporal bone)
   - Temporal bone: otitis externa, otomastoid opacification, mass lesions should be excluded
   - Parotid gland: mass lesions, evidence of parotitis, duct dilatation and calculi
   - Nasopharynx and skull base: mass ± invasion
8. Clinical relevance (Refs.: 20–24). Pain is strongly associated with:
   - Complete DD without reduction
   - Joint effusions, synovitis, erosions, marrow oedema
   - Marked OA

A sequential, eight-step approach to reporting of MRI is advised. MDCT and especially CBCT, allow a less comprehensive evaluation.
DPT, dental panoramic tomography; CBCT, cone-beam computed tomography; MDCT, multidetector CT; MRI, magnetic resonance imaging; DD, disc displacement, Gd, gadolinium, PD, proton density, OA, osteoarthritis.

Conclusion

TMD is common and predominates in adults aged 20–50 years of age. Females have more severe symptoms and are much more likely to attend for treatment; they outnumber males by at least 4:1.[41] The reasons for this are multifactorial.[1,18,42]

MRI is the preferred technique for the evaluation of adolescents or young adults with significant internal derangement of the TMJ. It has an accuracy of 94% as compared with arthroscopy, and demonstrates the type and severity of DD, intra-articular inflammation as well as disease of the articular surfaces and sub-articular marrow.[43] MDCT is preferred for patients >45 years in whom OA is the commonest cause of internal derangement. The absence of soft-tissue contrast is a major disadvantage of CBCT, but it is the preferred technique for the evaluation of TMD amongst a large proportion of the dental profession. A guide for reporting of imaging of the TMJ is shown in Table 2.

The terminology used for TMD has changed and become more standardised in the last decade.[44] The terms TMD and DJD have replaced TMJ dysfunction and osteoarthrosis respectively. DJD and OA are used interchangeably by radiologists; the identification of inflammatory markers in joint fluid and research using MRI has confirmed the role of inflammation and specifically synovitis, as aetiological factors in this disease in addition to degeneration of articular cartilage.[27,45]

The first widely accepted staging system TMJ disease was the Wilkes's classification from 1989.[46] Specialists in oral medicine and orofacial pain have adopted the *Diagnostic Criteria for TMDs* formulated by a consortium of TMJ specialists and published in 2014.[44]

Treatment of TMD is beyond the scope of this article. Non-surgical management is the most effective way of managing >90% of patients and aims to reduce joint and muscle pain, increase joint function, and prevent further TMJ damage.[1,41,47] This conservative approach is supported by the results of a 15-year MRI review of patients with DD and articular surface remodelling, which demonstrated the stability of these findings when the TMD is mild.[48]

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

1. Scrivani SJ, Keith DA, Kaban LB. Temporomandibular disorders. *N Engl J Med* 2008;**359**:2693–705.
2. Larheim TA, Hol C, Ottersen MK, *et al*. The role of imaging in the diagnosis of temporomandibular joint pathology. *Oral Maxillofac Surg Clin N Am* 2018;**30**:239–49.
3. Tomas X, Pomes J, Berenguer J, *et al*. MR imaging of temporomandibular joint dysfunction: a pictorial review. *RadioGraphics* 2006;**26**:765–81.
4. Whyte AM, McNamara D, Rosenberg I, *et al*. Magnetic resonance imaging in the evaluation of temporomandibular joint disc displacement a — review of 144 cases. *Int J Oral Maxillofac Surg* 2006;**35**:696–703.
5. 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 Endod* 2009;**107**:844–60.
6. Larheim TA, Abrahamsson A-K, Kristensen M, *et al*. Temporomandibular joint diagnostics using CBCT. *Dentomaxillofac Radiol* 2015;**44**:20140235. https://doi.org/10.1259/dmfr.20140235.
7. Palconet G, Ludlow JB, Tyndall PA, *et al*. Correlating cone-beam CT results with temporomandibular joint pain of osteoarthritic origin. *Dentomaxillofac Radiol* 2012;**41**:126–30.
8. de Boer EWJ, Dijkstra PU, Stegenga B, *et al*. Value of cone-beam computed tomography in the process of diagnosis and management of disorders of the temporomandibular joint. *Br J Oral Maxillofac Surg* 2014;**5**:241–6.
9. Boeddinghaus R, Whyte A. Computed tomography of the temporomandibular joint. *J Med Imaging Radiat Oncol* 2013;**57**:448–54.
10. Styles C, Whyte A. MRI in the assessment of internal derangement and pain within the temporomandibular joint: a pictorial essay. *Br J Oral Maxillofac Surg* 2002;**40**:220–8.
11. Petscavage-Thomas JM, Walker EA. Unlocking the jaw: advanced imaging of the temporomandibular joint. *AJR Am J Roentgenol* 2014;**203**:1047–58.
12. Barkhordarian A, Demerjian G, Chiappelli F. Translational research of temporomandibular joint pathology: a preliminary biomarker and fMRI study. *J Transl Med* 2020;**18**:22. https://doi.org/10.1186/s12967-019-02202-0.
13. Dong XY, He S, Zhu L, *et al*. The diagnostic value of high-resolution ultrasonography for the detection of anterior disc displacement of the temporomandibular joint: a meta-analysis employing the HSROC statistical model. *Int J Oral Maxillofac Surg* 2015;**44**:852–8.
14. Almeida FT, Pacheco-Pereira C, Flores-Mir C, *et al*. Diagnostic ultrasound assessment of temporomandibular joints: a systematic review and meta-analysis. *Dentomaxillofac Radiol* 2019;**48**:20180144. https://doi.org/10.1259/dmfr.20180144.
15. Levorova J, Machon V, Hirjak D, *et al*. Ultrasound-guided injection into the lower joint space of the temporomandibular joint. *Int J Oral Maxillofac Surg* 2015;**44**:491–2.
16. Yang Z, Reed T, Longino BH. Bone scintigraphy SPECT/CT evaluation of mandibular condylar hyperplasia. *J Nucl Med Technol* 2016;**44**:49–51.
17. Tamimi D, Jalali E, Hatcher D. Temporomandibular joint imaging. *Radiol Clin N Am* 2018;**56**:157–75.
18. Durham J, Wassell RW. Recent advancements in temporomandibular disorders (TMDs). *Rev Pain* 2011;**5**:18–25.
19. Ariji Y, Ariji E. Magnetic resonance and sonographic imagings of masticatory muscle myalgia in temporomandibular disorder patients. *Jpn Dent Sci Rev* 2017;**53**:11–7.
20. Whyte A, Gibson D. Imaging of adult obstructive sleep apnoea. *Eur J Radiol* 2018;**102**:176–87.
21. Takahara N, Nakagawa S, Sumikura K, *et al*. Association of temporomandibular joint pain according to magnetic resonance imaging findings in temporomandibular disorder patients. *J Oral Maxillofac Surg* 2017;**75**:1848–55.
22. Roh HS, Kim W, Kim YK, *et al*. Relationships between disk displacement, joint effusion, and degenerative changes of the TMJ in TMD patients based on MRI findings. *J Craniomaxillofac Surg* 2012;**40**:283–6.
23. Farina D, Bodin C, Gandolfi S, *et al*. TMJ disorders and pain: assessment by contrast-enhanced MRI. *Eur J Radiol* 2009;**70**:25–30.
24. Wahaj A, Hafeez K, Zafar MS. Association of bone marrow oedema with temporomandibular joint (TMJ) osteoarthritis and internal derangements. *CRANIO* 2017;**35**:4–9.
25. Hasan NMA, Abdelrahman TEF. MRI evaluation of TMJ internal derangement: degree of anterior disc displacement correlated with other TMJ soft tissue and osseous abnormalities. *Egypt J Radiol Nucl Med* 2014;**45**:733–45.
26. Vogl T, Lauer H-C, Lehnert T, *et al*. The value of MRI in patients with temporomandibular joint dysfunction: correlation of MRI and clinical findings. *Eur J Radiol* 2016;**85**:714–9.
27. Braun HJ, Gold GE. Diagnosis of osteoarthritis: imaging. *Bone* 2012;**51**:278–88.
28. Cortés D, Exss E, Marholz C, *et al*. Association between disk position and degenerative bone changes of the temporomandibular joints: an imaging study in subjects with TMD. *Cranio* 2011;**29**:117–26.
29. Hatcher DC. Progressive condylar resorption: pathologic processes and imaging considerations. *Semin Orthod* 2013;**19**:97–105.

30. Navallas M, Inarejos EJ, Iglesias E, *et al*. MR imaging of the temporomandibular joint in juvenile idiopathic arthritis: technique and findings. *RadioGraphics* 2017;**37**:595–612.
31. Barendregt AM, van Gulik EC, Lavini C, *et al*. Diffusion-weighted imaging for assessment of synovial inflammation in juvenile idiopathic arthritis: a promising imaging biomarker as an alternative to gadolinium-based contrast agents. *Eur Radiol* 2017;**27**:4889–99.
32. Basra HAS, Humphries PD. Juvenile idiopathic arthritis: what is the utility of ultrasound? *Br J Radiol* 2017;**90**:20160920.
33. Kretapirom K, Okochi K, Nakamura S, *et al*. MRI characteristics of rheumatoid arthritis in the temporomandibular joint. *Dentomaxillofac Radiol* 2013;**42**:31627230.
34. Marsot-Dupuch K, Smoker WRK, Gentry L, *et al*. Massive calcium pyrophosphate dihydrate crystal deposition disease: a cause of pain of the temporomandibular joint. *AJNR Am J Neuroradiol* 2004;**25**:876–9.
35. Ishikawa S, Watanabe T, Iino M. Acute septic arthritis of the temporomandibular joint derived from otitis media: a report and review of the English and Japanese literature. *Oral Maxillofac Surg* 2017;**21**:83–5.
36. Maribo Y, Stokbro K, Aagaard E, *et al*. Synovial cysts in the temporomandibular joint: a case report and critical review of the literature. *J Oral Maxillofac Res* 2019;**10**:e4. https://doi.org/10.5037/jomr.2019.10104.
37. Damodar D, Chan N, Kokot N. Pigmented villonodular synovitis of the temporomandibular joint: case report and review of the literature. *Head Neck* 2015;**37**:E194–9.
38. Wang P, Yang J, Yu Q. MR Imaging assessment of temporomandibular joint soft tissue injuries in dislocated and nondislocated mandibular condylar fractures. *AJNR Am J Neuroradiol* 2009;**30**:59–63.
39. Lee SY, Park JW, Park SE, *et al*. Clinical implications of magnetic resonance imaging in temporomandibular disorders patients presenting ear fullness. *Laryngoscope* 2018;**128**:1692–8.
40. Porto De Toledo I, Stefani FM, Porporatti AL, *et al*. Prevalence of otologic signs and symptoms in adult patients with temporomandibular disorders: a systematic review and meta-analysis. *Clin Oral Investig* 2017;**21**:597–605.
41. Dimitroulis G. Management of temporomandibular joint disorders: a surgeon's perspective. *Aust Dent J* 2018;**63**(Suppl. 1):S79–90.
42. Yasa Y, Akgül HM. Comparative cone-beam computed tomography evaluation of the osseous morphology of the temporomandibular joint in temporomandibular dysfunction patients and asymptomatic individuals. *Oral Radiol* 2018;**34**:31–9.
43. Alarabawy RA, El Ahwal HM, El Sattar El Sergany MA, *et al*. Magnetic resonance imaging evaluation of temporomandibular joint disorders, criterial analysis and significance in comparison with arthroscopy. *Egypt J Radiol Nucl Med* 2016;**47**:467–75.
44. Schiffman E, Ohrbach R, Truelove E, *et al*. Diagnostic criteria for temporomandibular disorders (DC/TMD) for clinical and research applications: recommendations of the international RDC/TMD consortium network and orofacial pain special interest group. *J Oral Facial Pain Headache* 2014;**28**:6–27.
45. Sokolove J, Lepus CM. Role of inflammation in the pathogenesis of osteoarthritis: latest findings and interpretations. *Ther Adv Musculoskelet Dis* 2013;**5**:77–94.
46. Wilkes CH. Internal derangements of the temporomandibular joint. Pathological variations. *Arch Otolaryngol Head Neck Surg* 1989;**115**:469–77.
47. Gauer RL, Semidey MJ. Diagnosis and treatment of temporomandibular disorders. *Am Fam Physician* 2015;**91**:378–86.
48. Salé H, Bryndahl F, Isberg A. Temporomandibular joints: 15-year follow-up clinical and MR study. *Radiology* 2013;**267**:183–94.