



Avascular Necrosis of Temporomandibular Joint: A Discrete Condition or a Chaos?

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Abstract

Purpose: To emphasize the importance of unifying temporomandibular joint (TMJ) avascular necrosis (AVN) and similar diagnoses from expanded taxonomy from diagnostic criteria for temporomandibular disorders (DC/TMD) and to emphasize the need for developing standardized criterion for diagnosing the condition.

Materials and Methods: The author conducted a literature search on PubMed and Ovid Medline databases. Key terms used were "osteonecrosis", "avascular necrosis", "jaw", and "jaw diseases". Excluded term was "bisphosphonates". The search was filtered to the articles published in last 10 years [2007 - 2017 (August)] in English only. Studies on magnitude, causation, and diagnostic foundation of osteonecrosis of jaw were selected. The information has been analyzed from already published data, IRB is not required.

Results: Out of 583 relevant articles (344 on PubMed and 239 on Ovid Medline), most literature pertained to AVN of femoral head and knee, and evidence relating this review was historic and contemplating medication related osteonecrosis of jaw. Studies that comprehend our area of interest are discussed and serve the basis for the recommendations made herein.

Conclusion: AVN of jaw constitutes a series of events leading to life disturbing pain and functional limitation. Evidence for diagnostic categorization of TMJ AVN is limited and there is a need for establishing diagnostic criterion to facilitate communication and formulate plan of care. The criteria can be based on the same as for femoral bone necrosis and portend easy clinical application. The term "necrosis" can only be used after histopathological confirmation and so making this diagnosis based on radiological studies is questionable, suggesting revision of the terminology.

Keywords: *Avascular Necrosis; Osteoarthritis; Subchondral Cyst; Idiopathic Condylar Lysis*

Introduction

In 1979, Reiskin was the first to report osteonecrosis of mandibular condyle as shown by microscopic evidence of bony changes in association with symptomatic TMJ internal derangements [1]. The condition has been described in the literature using interchangeable terms such as avascular necrosis (AVN), aseptic necrosis, ischemic necrosis, or a more neutral term osteonecrosis [2,3].

Review of AVN of TMJ

Incidence

Prevalence of temporomandibular disorders (TMD) and related pain is approximated up to 12% of the U.S. population [4,5]. However, exact incidence and prevalence of avascular necrosis (AVN) of jaw is still to be found [6]. TMJ osteoarthritis (OA) which is a related sub-classification of TMD has radiographic and clinical features

resembling those of avascular necrosis. Radiology is considered a standard clinical coadjutant for diagnosing TMJ OA [7] and AVN and estimates the incidence of AVN in less than 10% of joints with TMD [8,9] as detected by abnormal bone marrow signal. AVN most commonly affects bones such as femoral head, knee and shoulder joints. Other bones reported to be affected include ankle, elbow, wrist, calcaneus, tarsal navicular, cuneiform, cuboid, and metacarpal head [10].

Impact on quality of Life: (Why is AVN important?)

The available evidence makes it clear that AVN is becoming more prevalent in younger age and its impact on the patient's daily quality of life is proportionally increasing [11]. Patients with TMJ OA which has similar features frequently report painful and defective jaw mechanics with reduced quality of life [7]. A crucial finding that makes radiological features of OA and AVN compatible per DC/TMD imaging criteria is the presence of subchondral cyst on computed tomography (CT). Most of the discussion following is based on the studies for TMJ OA which is a closely related radiological entity. However, of note is that RDC/TMD criteria which has been used most often in TMD studies does not consider subchondral cyst as the sole standard diagnostic criterion for TMJ osteoarthritis; it may or may not exist with other findings [12]. OHRQoL measured by Oral Health Impact Profile (OHIP) is a multifaceted composition for valuation of the adverse influences of orofacial pain disorders on average daily oral functions. A sample of adult Chinese population, 18 - 70 years of age, diagnosed with symptomatic TMJ OA per Research diagnostic criteria for temporomandibular disorders (RDC/TMD) Axis I demonstrated a converse relation of Oral health-related quality of life (OHRQoL) with pain and functional domains [13]. The results of another Oral health-related quality of life (OHRQoL) study in Slovenia where 81 TMD patients per aforementioned diagnostic criteria were compared with 400 controls, reinforced the reverse association of OHRQoL with pain and showed even higher OHIP of psychological discomfort from TMDs especially OA [14].

Signs and symptoms

Patients with advanced degenerative disease (aka OA) of TMJ may complain of pain, joint stiffness, limited range of motion, grad-

ually progressive skeletal deformities of face. Clinical signs may be comparable to other TMDs but radiographic signs may include collapse of the articular surface [15]. Symptomatic osteonecrosis is often unilateral; bilateral presentation is commonly asymptomatic. In a retrospective study [16] of a cohort of a hundred patients who presented with the chief complaints of recently acquired facial deformities-deviation of chin to a side at rest, deviation of mandible to one side on opening and occlusal changes-ranging from anterior open bite, posterior open bite or cross bite, or unstable shifting posterior bite, showed signs of affected TMJ mechanics. Mechanical abnormalities included clicking noises, locking, asymmetric jaw motion and pain in the form of headache, facial pain, neck pain and earache. The study population denied a history of and exhibited no radiographic signs of mandibular or facial fractures or corrective jaw surgery. Radiographic findings based on submentovertex, anteroposterior with protruded mandible and lateral face views, and open and closed lateral TMJ tomograms, followed by MRI, revealed asymmetric or deformed mandibular condylar morphology and height, and altered condylar position in the glenoid fossa. These findings explain reduction in posterior mandibular and facial height, articular space narrowing, articular space widening, AVN, retrognathia, or deviation of chin to the side of deformed mandibular condyle respectively. Three patients (five joints) with a history of pain, mechanical TMJ symptoms, progressive retrognathia, open bite, and loss of posterior mandibular height exhibited radiographic signs of complete absence of condyle and most or whole of condylar neck suggesting progression of AVN to osseous collapse and resorption. Radiography also manifested articular surface abnormalities varying from erosions of condylar cartilage to articular surface depression (with or without remodeling) justifying AVN. Furthermore, histologic evaluation of removed osseous fragments by core decompression of diseased condyles from the study sample revealed areas of normal marrow between areas of softened or sclerosed bone, and complete lack of bleeding in focal areas, indicating AVN. All cases of AVN had advanced stages of disc derangements usually with perforation. Loss of vertical dimension lead to occlusal instabilities causing contralateral anterior open bite and ipsilateral premature posterior teeth contacts and/or cross bite, creating facial deformities e.g. chin deviation to the side of the collapsed joint.

Vascular basis of mandibular condyle

The major blood supply to the mandible and condylar head comes from the inferior alveolar artery and arteries in the TMJ capsule which can branch either directly from the maxillary artery, or from any of its following branches; masseteric artery, lateral pterygoid artery or superficial temporal artery. The main arteries pierce the TMJ capsule branching further and penetrating the periosteum of the condylar head. The additional arteries splitting from maxillary artery directly or as a continuation of the lateral pterygoid artery approach the condylar neck from the medial side, at the border of TMJ capsule [17].

Risk factors and etiology for AVN

A risk factor is a variable associated with the increased probability of a disease. Alcohol, systemic medications (e.g. bisphosphonates, corticosteroids, immunosuppressive agents), infections e.g. Human immunodeficiency virus (HIV), direct exposure to chemicals (e.g. aluminum) [18], trauma (e.g. intracapsular mandibular condylar fracture) [19], radiations and low levels of protein C and S, high levels of plasminogen activator inhibitor (causing hypofibrinolysis), liver disease, Factor V Leiden mutation, high levels of lipoprotein A and Von Willebrand factor, at least one abnormal coagulation factor, and Gaucher's disease (deficiency of beta-glucocerebrosidase), all have been found in patients with AVN when compared to healthy controls [15,20,21]. Patients with sickle cell disease, pregnancy, gout, systemic lupus erythematosus (SLE), and Caisson's disease have also exhibited AVN in different joints [22-24]. A dormant osteoarthritic joint if stressed by trauma (including barotrauma), iatrogenic manipulation, and/or systemic inflammation (e.g. pancreatitis) or connective tissue disorders can potentiate development of AVN [16]. The lateral part of the condylar head generally has poor blood supply and so a fracture detaching the condylar head from neck can disconnect it from main arterial supply of the mandible especially in the absence of any additional arteries supplying the capsule and can increase the likelihood for developing AVN [17]. A fourth of the cases, however, can be idiopathic with no known etiology. The role of genetic predisposition in conjunction with systemic medications mentioned above has also been reported with AVN of femoral head [11].

Histological classification of mandibular condyle

In an *in vitro* study [25] of human mandibular condyles re-

moved from 63 TMD patients (by high condylectomy), the articular surface of the mandibular condyle was subdivided into articular zone, proliferative zone, intermediate zone, cartilage zone, and cortical bone zone. Microscopic observation of the condyles refractory to conservative management of TMJ related problems showed local areas of abnormal tissue in approximately 50% whereas complete absence of articular zone was noted in more than 40% of the specimens. The histological picture of each of these zones was differentiated into five classes by the presence or absence of any changes and were described as normal, reduced, thickened, absent, or abnormal. The presence or absence of inflammation was also assessed in each zone. The cortical zone was frequently normal; however, local areas of reduced cortical bone thickness and resorption was relatively common finding. In cortical bone zone, remodeling was assessed based on ongoing activity (active or passive) and nature of tissue being replaced-no replacement, bone, cartilage, fibrous tissue, bone and cartilage, fibrous tissue and cartilage. The proliferative zone was totally absent in 45% of cases, and the cartilage zone was found to be generally or locally absent in more than 80% of specimens. The study had some limitations including gender bias in sample (more females), absence of a control group, variable amount of trauma induced during surgical removal of specimens, also affecting the orientation of the specimen.

Physiopathology of AVN and associated pain

AVN is characterized as a primary subchondral osseous breakdown of the mandibular condyle with secondary articular surface collapse [26]. Microvascular studies of femoral head bone have demonstrated higher susceptibility of the blood supply of subchondral bone to vascular interruption (from fractures or dislocation), intravascular occlusion (from thrombi or embolic fat), extravascular compression, and/or venous obstruction (from lipocyte hypertrophy i.e. Gaucher cells) causing necrosis of hematopoietic cells and adipocytes, culminating in interstitial bone marrow edema. A decrease in blood flow is known to diminish intraosseous partial pressure of oxygen dysregulating the balance between need and supply of oxygen. This can elicit marked ischemia leading to necrosis as illustrated in figure 1. However, it is not the necrosis itself but the resorption from the repair process that causes loss of structural integrity and subchondral fracture of the bone. The loss of conformity of subchondral trabeculae is responsible for the subchondral fracture [15]. Compartment syndrome concept of the pathogenesis

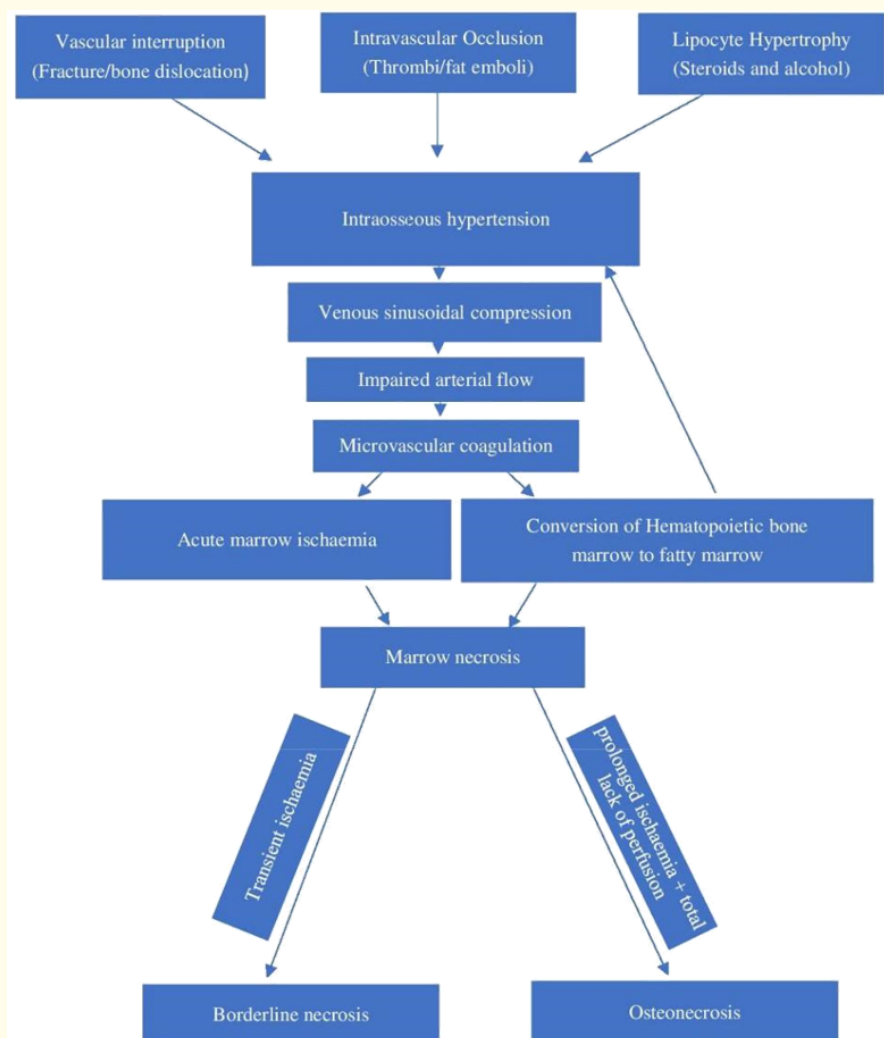


Figure 1: Flow chart of events involved in pathogenesis of osteonecrosis of bone. Adapted from pathogenesis of AVN in Femoral Head [6].

of AVN gives deeper insight into the pathogenesis. Viewing osseous medulla as a starling resistor (a rigid compartment through which one or more flexible tubes pass), the flow through the tubes is determined by the pressure outside the compartment relative to that inside. An increase in the intra compartmental or intramedullary pressure will decrease blood flow inside the space; whereas increased pressure in the extramedullary space will decrease venous outflow. Decreased flow of blood from increased extravascular pressure could result from multiple reasons aforesaid under the

heading of risk factors. Presumably, ischemia of the joint should be throughout the condylar head; howbeit, mechanical loading clearly seems to play a role to augment this effect. Condylar collapse from AVN typically precedes similar changes in the glenoid fossa. Fossa changes are the consequence of mechanical trauma caused by incongruity of the articulating surfaces. Elevated intraosseous pressure, however, may not be the only cause for the pathogenesis of the clinical occurrence [15] soft tissue derangements in the joint and disc displacements are associated with higher predisposition

of TMJ to AVN particularly anterior disc displacement without reduction. It is also conceivable that mechanical impingement of the disc against the lateral pterygoid muscle could potentially interrupt the arterial inflow and venous outflow causing a rise in intramedullary pressure which is the basis of AVN [15,19,20,27]. Pain of AVN is believed to be due, principally, to increased intraosseous venous pressure and secondary synovitis with or without effusion or due to periosteal irritation by surface irregularities or osteophytes. Prognosis is better with optimal general health and good repair capacity of the condyle especially in the absence of surface collapse [26].

Histopathology of AVN

Histological studies reveal that osteocyte necrosis is reflected by pyknosis of nuclei in the beginning and by empty osteocyte lacunae later (approximately two to three hours post anoxic injury). However, pyknosis alone is not considered a reliable indicator of cell death in osteonecrosis because there is a normal attrition of osteocytes with age. Complete absence of osteocytes within localized areas of trabecular bone is, however, a reliable sign of osteonecrosis. The signs of osteocyte death to appear histologically take approximately 24 to 72 hours after hypoxia. Osteocyte lysis can take 48 hours up to 4 weeks or even longer to complete. Subsequently, reactive hyperemia and capillary revascularization in the periphery of the necrotic zone and a repair process begins. Repair process consists of both processes-bone resorption and deposition and can partially replace dead with living bone. New viable bone is coated over dead trabeculae with incomplete resorption of dead bone. In the subchondral trabeculae, bone resorption overtakes bone formation resulting in net loss of bone and compromised structural integrity of these trabeculae, generating subchondral fracture and joint incongruity [15]. Interestingly, foci of bone necrosis can be visualized on biopsy specimen, even when magnetic resonance imaging (MRI) suggests marrow edema only. The histologic criteria for AVN encloses an acellular marrow, large cysts, occasional calcifications in the marrow, large zones of empty lacunae, granulation tissue marking the demarcation between necrotic and viable bone (reflecting healing attempts), and thickening of the trabeculae as a result of new bone deposition on the necrotic or viable bone [20].

Imaging-MRI based classification of mandibular condylar marrow

MRI has been established as a technique for the diagnosis of early AVN [28]. Diagnosis of AVN is based on alterations of the MR signal from the mandibular condyle. Internal derangements of TMJ are categorized into five categories as described by Wilkes [29]. This categorization is founded on changes in osseous morphology, disc morphology, disc position, presence or absence of intrinsic MR signal changes in disc and presence or absence of disc and/or attachment perforation. MRI signal changes of the mandibular condyle with or without articular collapse have been predominantly found in stage V internal derangements beneath cartilaginous erosions and perforations. The condylar marrow abnormality has traditionally been classified into two patterns; an edema pattern and a sclerosis pattern (Table 1). The edema pattern gives a hypo intense signal on T1 but a hyperintense signal on T2. In contrast, the sclerosis pattern returns hypo intense signals on both T1 and T2 weighted images. This difference in intensity is consistent with the stages of AVN; acute AVN is hypointense on T1-weighted images and hyperintense on T2-weighted images versus chronic stage which is characterized by hypointense marrow signal on both T1- and T2-weighted images and is analogous to the sclerosis pattern [30]. Surgical findings have been in sync where the diagnosis for condylar AVN was suggested preoperatively with tomograms and/or MRI [19].

MRI staging and pattern of TMJ AVN			
Stages of mandibular condylar	MRI pattern	MRI Signal	
		T1	T2
AVN			
Acute	Edema pattern	Hypointense	Hyperintense
Chronic	Sclerosis pattern	Hypointense	Hypointense

Table 1: Adapted from the work of Lieberman, et al. 1996 [30].
 *Hyperintense (bright/white), hypointense (dark).

Correlation of histological and radiological findings

Radiographically, lucent areas reflect bone resorption while areas of sclerosis can comprise of both-repaired living bone and/or dead trabeculae. Histopathologic changes of the femoral head AVN are known to reflect in the radiographic appearance of scler-

rosis and lucency [15]. A study [2] correlated MR images and histological pictures of bone marrow of mandibular condyle of TMD patients with painful internal derangements. It compared MRI of 50 symptomatic TMJs with 124 asymptomatic joints and examined core biopsies of mandibular condyles from symptomatic TMJs. When analyzed for fluid in joint compartments and edema and sclerosis in marrow, total 18 joints displayed abnormal findings-nine joints revealed edema and joint effusion with cortical erosions suggesting inflammatory arthritis and nine joints suggested osteonecrosis on histology as shown by edema, marrow fibrosis, fat cell necrosis, bone resorption, reactive bone formation, and inflammation-mimicking histological picture of hip osteonecrosis. Cone beam computed tomography (CBCT) images of necrosed TMJs in patients with pain, demonstrated cyst-like radiolucent le-

sions underneath the superior cortical margin with discontinuity of the condylar head [28]. Whether advanced degenerative changes (aka OA) precede or follow necrosis is yet to be found. In hip joints, however, osteoarthritis is known to follow osteonecrosis. In TMJ, bone marrow alterations (a sign of AVN) may represent early osteoarthritic changes [8,9,31].

Histological and radiological stages of progression of AVN

The histopathological progression of AVN has been staged. Evidence of marrow necrosis adjacent to viable bone and marrow with an intervening zone of repair on the same section of bone reinforces the histological diagnosis of osteonecrosis. During the initial stage, stage 1 of AVN, routine plain radiographs do not reveal changes in tissue density; more advanced techniques like MRI or bone scintigraphy are needed. A reparative reaction on the margins appears as low signal intensity band on T1-weighted images and high signal intensity band on T2-weighted images. Histologically, the band represents the reactive interface, and completely empty lacunae can be seen in the necrotic bone within the band. In stage 2, radiologic appearance of lucency and sclerosis can be observed produced by blood vessels entering the necrotic zone attempting repair by bone resorption and formation. Toward the outer margin of the reactive interface, previously dead cancellous bone is fractionally invested by ber or lamellar bone. This area is enveloped by a reinforcing zone of viable trabecular bone, which appears as demarcating sclerosis on x-ray images. The classical terms for this type of bone formation are “creeping substitution” or “creeping apposition.” By stage 2, no collapse is detected in femoral head AVN. However, reinforcing bony architecture is continuously being weakened by ongoing resorption of trabecular bone. It is during stage 2 that stress from mechanical forces can result in subchondral fracture and eventual collapse which will mark stage 3. Fragmentation and compaction of debris from subchondral bony fracture develops a subchondral lucent zone along the fracture line, which is denoted as crescent sign. Due to the predominant anterior location, crescent sign is usually seen on lateral radiographic views of the femoral head, even though staging is based on both anteroposterior and lateral x-ray images of the femoral head. Stage 3 is further subdivided into three types depending upon the size of surface collapse, as shown in table 2. Articular collapse creates an incongruent articular surface propagating to stage 4 which is characterized by degenerative joint disease (DJD) [32].

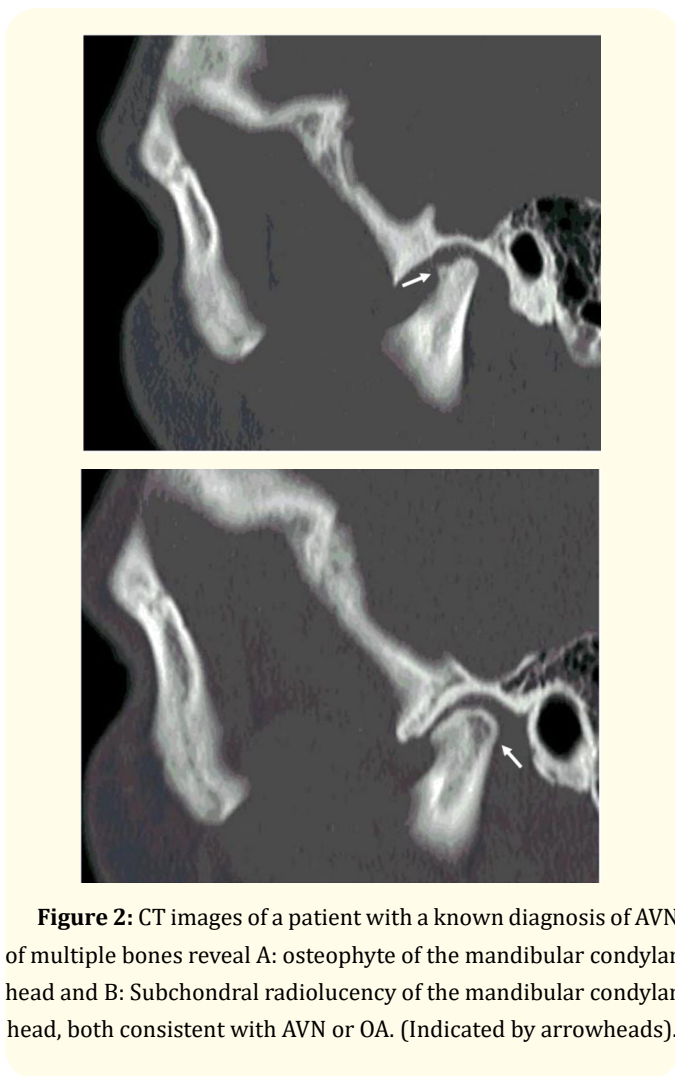


Figure 2: CT images of a patient with a known diagnosis of AVN of multiple bones reveal A: osteophyte of the mandibular condylar head and B: Subchondral radiolucency of the mandibular condylar head, both consistent with AVN or OA. (Indicated by arrowheads).

Stages	Radiologic findings			Histological findings
Stage 1 location: Medial Central Lateral Radiographic quantitation: Based on area of femoral head involved Mild 15% Moderate 15 - 30% Extensive > 30%	Plain Radiographs No changes	CT No need	MRI T1: Hypointense band along the margin of avascular region T2: Hyperintense band along the margin of avascular region	Reactive interface, completely empty lacunae in the necrotic bone
Stage 2 location: Medial Central Lateral Radiographic quantitation: Based on area of femoral head involved Mild 15% Moderate 15 - 30% Extensive > 30%	Appearance of radiolucency and sclerosis or mottled appearance, osteolysis, or focal osteoporosis No collapse of bone detected		Not needed	Necrotic zone attempting repair by bone resorption and formation. Investment of fiber or lamellar bone. Envelop of viable trabecular bone
Stage 3 location: Medial Central Lateral Radiographic quantitation: Based on area of femoral head involved Mild 15% Moderate 15 - 30% Extensive > 30% Or Based on amount of flattening Depression of bone: < 2 mm 2 - 4 mm > 4 mm	Flattening of articular surface. Subchondral lucent zone crescent Sign. Collapse of bony surface		Not needed	Subchondral fracture. Fragmentation and compaction of debris
Stage 4 Sub classification of location or radiographic quantitation not needed anymore	Incongruent articular surface, joint degeneration and joint space narrowing	No need	Not needed	Degenerative joint disease

Table 2: Radiologic and histologic staging of femoral head AVN.

Adapted from Association Research Circulation Osseous (ARCO) International Classification of osteonecrosis of femoral head [33].

Discussion

In many cases, AVN presages the development of occlusal disharmonies. Condylar alterations are noted in association with acute or chronic pain and are important to diagnose to ensure timely management of pain, restoration of function, and prevention of progression to facial skeletal deformities or skeletal growth

disturbances. We know from the available evidence that pain, limited range of motion and functional disability can all affect multiple domains of a healthy lifestyle [19]. Acute and subacute AVN can precipitate severe pain, joint dysfunction, and rapid structural disintegration of the condyle leading to unstable occlusal harmony. The cause of advanced condylar osseous degeneration, as seen in OA may not be distinguishable from AVN. Chronic stage V internal

derangements of TMJ may be painful and disabling depending on the extent of joint inflammation and/or coexisting marrow pathology-which may be a representation of AVN. This stresses the need for timely diagnosis to reduce associated morbidity.

Importance of staging

The important information to note is that epidemiological rates do not directly correspond to the need for management of AVN [10] and staging can generally help separate the cases that warrant management from the ones that do not. Much of our current understanding of the disease process in TMJ is in consort with that of other articular joints, especially femoral head bone AVN. Although biological basis of pathogenesis is related to that of other joints, additional studies on TMJ are required owing to its unique structural and functional anatomy. TMJ AVN has been under reported in the literature and more studies may unveil if flattening or resorption of the condylar head and presence of altered bone pattern is only a radiologic sign of osseous changes or an actual presentation of an ongoing pathological phenomenon. To my knowledge, this is the first review providing a concerted understanding of the magnitude, impact, risk factors, signs and symptoms and physiopathology of AVN of TMJ.

Need for Unification of related TMD taxonomy

The expanded taxonomy by Peck, *et al.* [33] suggests separate categories for idiopathic condylar resorption (ICR), and avascular necrosis of TMJ. The diagnosis of ICR needs subjective and objective findings of progressive dental occlusal changes and resorption of part or all condylar head. Analyzing closely, the same applies for the diagnosis of progressive avascular necrosis and OA. Broadly speaking, DJD, AVN and CLR represent common clinical and radiographic findings. The imaging guidelines from DC/TMD based on computed tomography (CT) recommend that subcortical pseudocyst, one of the three cardinal features, be used as reference standard for the diagnosis of DJD of TMJ; the other two being erosion and osteophyte formation. The pseudocyst has been defined as a cavity below the articular surface that shows deviation from normal bone marrow pattern and is reflected by loss of trabeculations [12,34]. This leads to uncertainty because a subcortical cyst may also represent AVN of bone and could be a possible manifestation of systemic condition or psychosocial risk factors like excessive alcohol intake. This similarity of the tomographic signs calls for the need for unifying the terms and buttresses the inquisition

of whether DJD, AVN, or ICR are different entities. If yes, what distinguishes the three? Osteonecrosis may also occur within an old quiescent osteoarthritic joint and it is not uncommon to find both osteonecrosis and osteoarthritis together in a joint. Presence of focal areas of surface necrosis in OA, makes histopathological diagnoses of OA and AVN indistinguishable in advanced stages [19,30]. On the other hand, idiopathic condylar lysis is an unclear clinical diagnosis implying condylar resorption due to unknown cause. The question is, is the cause truly unknown or a missed systemic illness or risk factor?

Emphasizing radiologic monitoring of bony alterations

The developers of DC/TMD do not recommend routine image acquisition and endorse that it be guided by the "clinical needs" of the patients. The need also arises for defining "clinical needs". If only the symptomatic joints at the time of evaluation necessitate radiological assessment, overlooking a history of or existence of known systemic risk factors, can serve to miss an important disabling bone condition. In such an occurrence, the importance of radiological screening to look for early bone changes, in the absence of clinical symptoms, cannot be disregarded. Every joint showing similar radiographic signs (e.g. OA) can be closely monitored to detect disease progression. In the absence of well-defined single clinical entity and applicable consistent diagnostic criteria for it, this can be puzzling and can compromise patient prognosis.

Proposing use of an umbrella term-Focal osseous defect of TMJ

The name avascular necrosis implies histopathological diagnosis, which brings forward the question-is there a need to biopsy every predisposed or affected mandibular condyle to confirm the diagnosis of AVN? It is hereby proposed that we change the terminology to focal osseous defect based on radiological assessment of TMJ and reserve histopathological evaluation only for cases that are refractory to conservative management options and warrant surgical treatment. This will help facilitate communication, remove any ambiguities regarding diagnosis until future research allows us true distinction between the related clinical conditions. It will also reduce the risks associated with biopsy in all cases of altered radiologic bone pattern.

Recommended approach for staging

There is a need for classifying the stages of osseous defect using a system as ARCO staging system for femoral head described

above. Such a criterion is worth the time of clinicians to help measure and compare degrees of severity across populations and standardize diagnostic protocol for optimum management especially in advanced cases.

Careful measurements of the size of the bone marrow abnormality can help compare the progression of the process over time and correlate it with the clinical severity of the disease. A more detailed sub classification for TMJ may not be possible beyond categorizing the size and location of the lesion as mandibular condylar head is a smaller bone when compared to femoral head. However, as per ARCO staging, radiologic quantitation, clinical quantitation of symptomatic and asymptomatic joints in patients with known systemic risk factors (based on different questionnaires assessing pain and degree of limitation of joints) can be done and any specific findings outside the recommended guidelines may be noted separately. Advanced imaging techniques like scintigraphy may be recommended for symptomatic cases [35,36].

Conclusion/Clinical Relevance

The invaluable evolution of imaging guidelines for RDC/TMD and recommendations from Axis I of DC/TMD criteria provide a scaffolding for efficient diagnoses of TMD. It is hereby stressed to assess the importance of coalescence of less defined and less common diagnoses with similar features to one making the assessment methodology adept yet thorough. The review not only highlights the diagnostic comparison with other similar conditions like OA and ICR of TMJ and requests a more insightful and apt terminology to describe them all, but also stresses the need for establishing diagnostic criteria for the condition. The diagnostic criteria will prove promising for understanding the clinical staging, predicting need for treatment, and improving patient's quality of life.

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