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Research Paper

Tmj Arthritis -A Review

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ABSTRACT:
The Temporomandibular Joint (TMJ) is one of the most important and complex joints in the body which provides the articulation between the movable mandible and the fixed temporal bone of the cranium. The TMJs are bilateral, diarthrodial , ginglymoidal , synovial, and freely movable. Temporomandibular joint arthritis is inflammation of TM joint could be rheumatoid arthritis, degenerative joint disorder (Osteoarthritis), septic arthritis, psoriatic arthritis, Ankylosing spondylitis. Severity of the disease differs from each other ranging from mild to severe. In this paper, etiopathogenesis, clinical features, investigations and the management of temporomandibular joint Arthritis are discussed.

KEYWORDS: *Temporomandibular Joint Arthritis, Rheumatoid Arthritis, Psoriatic Arthritis, Osteoarthritis, Ankylosing spondylitis*

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I. INTRODUCTION

Temporomandibular Joint arthritis affects the joint and the surrounding musculature. Like any other joint, causes of temporomandibular joint arthritis could be rheumatoid arthritis, osteoarthritis or psoriatic arthritis. The aim of this review is to discuss about the various causes of TMJ arthritis, etiopathogenesis, clinical features, investigations and the management of temporomandibular joint arthritis.

1. DEGENERATIVE JOINT DISEASES

Degenerative joint disease (OSTEOARTHRITIS) is a non inflammatory disorder of joints characterized by joint deterioration and proliferation characterized by loss of articular cartilage and bone erosion then bone formation at the articular surface and in the subchondral region.[1]

When bony changes are active the condition is called osteoarthritis. as remodelling occurs, the condition can become stable yet the bony morphology remains altered. this condition is referred to as osteoarthrosis.[2]

TYPES:

It may be categorized as primary or secondary although both are similar on histopathologic examination. Primary degenerative joint disease is of unknown origin, but genetic factors play an important role. It is often asymptomatic and is most commonly seen in patients above the age of 50 years, although early arthritic changes can be observed in younger individuals. Mainly due to overloading of the joint. This may occur when joint surfaces are compromised by disc dislocation and retrodiscitis. Secondary degenerative joint disease results from a known underlying cause,such as trauma, congenital dysplasia, or metabolic disease.[1]

ETIOPATHOGENESIS

The process begins in loaded articular cartilage, which thins and clefts (fibrillation) and then breaks away during joint activity. This leads to sclerosis of underlying bone, subcondylar cysts, and osteophyte formation. The fibrous tissue covering in patients with degenerative disease is preserved. This may be a factor in remodeling and the recovery that is usuallyexpected in osteoarthrosis and osteoarthritis.[1]

CLINICAL FEATURES:

It occurs in patients older than 40 years of age and 85% of them are older than 70, with a mean age of 53 years. Females are affected 6 times as frequently as males. The incidence of degenerative changes increases with age, and such changes are found in over 40% of patients over 40 years of age. Richards and Brown observed a direct relationship, irrespective of age, between the rate and extent of dental attrition and degenerative disease of the TMJs.[1,3]

Symptoms – There is unilateral pain over the joint, which may be sensitive to palpation. Patient also experience pain on movements or biting, which may limit mandibular function. Pain usually worse in the evening.

Signs – There is a deviation of the jaw towards the affected side. Affected joint is swollen and warm to touch. Stiffness of the joint.

It can occur any time the joint is overloaded. but it is most commonlyassociated with disc dislocation without reduction.[4]

Crepitation – there is presence of crepitation of the joint, the sound indicatesdegeneration within the articulating surfaces of the joint or disc.

Jaw movement – There is limitation of jaw movements, which becomes increasingly apparent with function. Pain is usually located to the immediate pre auricular region.[3]

INVESTIGATIONS:

Blood investigations to rule out Rheumatoid arthritis, OPG, CT, CBCT.

RADIOGRAPHIC FEATURES:

Degenerative changes seen on the lateral and anterolateral wall of the fossa. First evidence of erosion of condyle on the radiograph occurs on an average, 6 months after the onset of TMJ pain. This will result in enlargement and shallowing of mandibular fossa. Osteophytes may break off and lie free within the joint space (these fragments are known as joint mice), and these must be differentiated from other conditions that cause joint space radiopacities.[2,3]

MANAGEMENT:

Definitive treatment

Relieving the pressure on joint – a stabilizing appliance is indicated to decrease the loading force. **Elimination of the cause** – It includes occlusal adjustment or replacement of the missing teeth and ill fitting prosthesis, grinding, treatment of caries and periodontal disease. Pharmacotheraphy: Analgesic and anti-inflammatory drugs – For the relief of pain, The treatment includes the use of acetaminophen, NSAIDS, COX-2 inhibitors, topical analgesics. **Physiotherapy** – Heat therapy, diathermy and ultrasonic. **Myotherapy** – Muscle exercises, injection of local anesthetic in TMJ. **Arthoscopic lavage** – Arthroscopic lavage may give relief in some patients. **Doxycycline** – Nowadays, low dose doxycycline (collaganase inhibitor, anti matrix metalloproteinase) is giving relief in many patients.[4]

RHEUMATOID ARTHRITIS

Rheumatoid aribritis (RA) is an inflammatory disease characterized by pain, swelling, stiffness, and loss of function in the joints. It is a debilitating systematic disease of unknown origin, characterized by progressive involvement of the joint, particularly bilateral involvement of large joints. Bony components of the TMJ are affected secondary to the granulomatous involvement of the synovial membrane thatsubsequently spreads

to the articular surface of the condyle. It is non –suppurative inflammatory destruction of joint.[5] The percentage of rheumatoid arthritis (RA)patients with TMJ involvement ranges from 40 to 80%.[62]

ETIOPATHOGENESIS:

Phase one – It results from some systemic infection, which evokes an inflammatory response within the joint.

Phase two – As an autoimmune reaction to the antigen generated by the initial inflammation itself or it may be associated with derangement of the immune response to the exogenous antigen.

Active phase – In the active phase, TMJ may get involved bilaterally. Bony components of the TMJ are affected secondary to the granulomatous involvement of its synovial membrane that subsequently spreads to the articular surface of the condyle. The joint space enlarges with synovial effusion which attacks the fibrocartilage and ultimately produces erosion of the underlying bone. This causes pain, stiffness and limitation of movement.

Chronic phase – Chronic phase may follow after active phase. Here, there is a proliferation of the synovial membrane due to inflammation. This is called as*pannus* formation. This pannus then encroaches the joint space and causes destruction of the articular cartilage. In this, lipping of the condyle and marginal proliferation is seen, this result in narrowing of joint space. Here, predominant clinical findings are crepitus, pain on bitting and tenderness. The granulomatous tissue replaces the articular surface and small adhesions develop between the articular surface and disc.

Healing phase – The process then enters the healing phase, where the symptoms subside and remodelling of the articular surface occurs.

Psychosomatic – Emotional trauma, anxiety and environmental strain lead tothe onset of rheumatoid arthritis.

Immunological – The presence of rheumatoid factor in the serum and synovial fluid of affected patients suggest immunological etiology. Plasma cells and lymphocytes are also present on the histological examination.[4,6]

CLINICAL FEATURES:

It more commonly seen in women from 20 to 50 years of age. In typical cases, small joints of fingers and toes are the first to be affected. Swelling of the proximal but not the distal, interphalangeal joints give the finger spindle like appearance and swelling of the metatarsophalangeal joints results in broadening of feet.[7]

Symptoms include bilateral stiffness, crepitus tenderness and swelling over the joint. Fever, malaise, fatigue, weight loss, pain and stiffness in the limb are also evident. Polyarthritis develops subsequently, large and weight bearing joints are frequently affected.[1,9]

There is formation of subcutaneous nodules on the pressure points, sites of friction and various vascular lesions, both necrotizing and obliterative types. Severe deformities of extremity can occur as a result of joint collapse, tendon rupture and muscle involvement.[1]

The TMJs are usually bilaterally involved in RA. The most common symptoms include limitation of mandibular openingand joint pain. Pain is usually associated with the early acutephases of the disease but is not a common complaint in later stages. Other symptoms often noted include morning stiffness,joint sounds, and tenderness and swelling over the joint area.[7]

The symptoms are usually transient in nature, and only a small percentage of patients with RA of the TMJs will experience permanent clinically significant disability. The most consistent clinical findings include pain on palpation of the joints and limitation of opening.[7]

Crepitus alsomay be evident. Micrognathia and an anterior open bite are commonly seen in patients with juvenile RA. micrognathia to a combination of direct injury to thecondylar head and altered orofacial muscular activity. Ankylosis of the TMJ related to RA is rare. Anterior open bite is present due to bilateral destruction and anterosuperior positioning of the condyle.[7]

RADIOLOGICAL FEATURES:

The initial changes may be generalized osteopenia (decreased dens.ity) of the condyle and temporal component. The pannus may destroy the disk, resulting in diminished width of the joint space.[3]

Figure 1: Rheumatoid Arthritis Of TMJ

Bone erosions by the pannus most often involve the articular eminence and the anterior aspect of the condylar head, which permits anterosuperior positioning of the condyle when the teeth are in maximal intercuspation and results in an anterior open bite .

Erosion of the anterior and posterior condylar surfaces at the attachment of the synovial lining may result in a "sharpened pencil" appearance of the condyle. Erosive changes may be so severe that the entire condylar head is destroyed, withonly the neck remaining as the articulating surface.[8,9]

Subchondral sclerosis and flattening of articulating surfaces may occur, as well as subchondral "cyst" and osteophyte formation. Fibrous ankylosis or, in rare cases,o sseousa nkylosis,m ay occur; reduced mobility is related to the duration and severity of the disease.[8,9]

Radiological staging of rheumatoid arthritis:

Stage I – Periarticular osteoporosis

Stage II – Loss of articular cartilage

Stage III – Erosion

Stage IV – Subluxation and ankylosis[4]

INVESTIGATIONS :

Complete blood count (CBC), comprehensive metabolic panel (CMP) with lipids, and thyroid panel: Obtained to rule-out infection, anemia, and thyroid disease, and to check kidney and liver function.[10]

Rheumatoid factor: RF is an autoantibody, most relevant to rheumatoid Rose Waller test is positive in 70% of the patients withrheumatoid arthritis.

Antinuclear antibodies: ANAs are frequently elevated in inflammatory arthritis and indicate level of disease activity detected by indirect immunofluorescence. they are useful for tracking treatment efficacy.[10]

Other tests like High-sensitivity C-reactive protein (hs-CRP), Deoxy pyridinoline (DPD), Vitamin D , Food-specific IgG4 antibodies , Multi profile panel (amino acids) and essential elements ,essential fatty acids ,organic acids, and oxidative stress markers can be used to monitor condition.[10]

MANAGEMENT:

Supportive treatment – Adequate rest to the joint, soft diet is advocated. The patient should be placed on a soft diet during acute exacerbation of the disease process.

Use of a flat plane occlusal appliance may be helpful, particularly if parafunctional habits are exacerbating the symptoms. An exercise program to increase mandibular movement should be instituted as soon as possible after the acute symptoms subside.[11]

Intra – articular corticosteroid injections – Local injections of long acting steroids such as methyl prednisone acetate (20-80 mg for large joint and 4–10 mg for small joint) or triamcinolone hexa-acetomide (10-40 mg for large joint and 2-6 mgfor small joint) are given when patients have severe symptoms.[11]

Non- steroidal anti-inflammatory drugs –These drugs are inhibitory to prostaglandins. These are used for symptomatic relief. Salicylates (for pain) and anti- inflammatory agents like phenylbutazone, indomethacin, ibuprofen, diclofenac and piroxicam can be used.

Immunomodulator – azathioprine is found to be effective in both, high and low doses.Slow acting

anti-rheumatic drugs – These are the antimalarials like hydroxyl chloroquine suphate, sulphasalazine (500 mg/day) and methotrexate (D-Penicillamine and parental gold).

Physical treatment – It is done with heat, diathermy, jaw exercise or a mouth stretcher. Muscle strengthening exercise and hydrotherapy.[4]

Surgical treatment of the joints including placement of prosthetic joints, is indicated in patients who have severe functional impairment or intractable pain not successfully managed by other means.[4]

JUVENILE CHRONIC ARTHRITIS

Juvenile chronic arthritis (JCA), formerly called juvenile rheumatoid arthritis, is a chronic inflammatory disease that appears before the age of 16 years (themean age is 5 years). It is also called as 'Still's disease'

It is characterized by chronic, intermittent synovial inflammation that results in synovial hypertrophy, joint effusion, and swollen, painful joints. As the disease progresses, cartilage and bone are destroyed.[3]

Rheumatoid factor may be absent, hence the preferred use of the term JCA rather than juvenile rheumatoid arthritis. JCA differs from adult RA in that it has an earlier onset, and systemic involvement usually is more severe. TMJ involvement occurs in approximately 40% of patients and may be unilateral or bilateral.[3]

CLINICAL FEATURES

The patient usually has pain and tenderness in the affected joint or joints, although the disease can be asymptomatic. Unilateral onset is common, but contra lateral involvement may occur as the disease progresses.

Severe TMJ involvement results in inhibition of mandibular growth. Affected patients may have micrognathia and posteroinferior chin rotation, resulting in a facial appearance known as bird face, which may also be accompanied by an anterior open bite.

The degree of micrognathia is proportional to the severity of joint involvement and the early onset of disease. When only one TMJ is involved or if one side is more severely affected, the patient may have a mandibular asymmetry with the chin deviated to the affected side.[3]

RADIOGRAPHIC FEATURES

Osteopenia (decreased density) of the affected TMJ components may be the only initial radiographic finding. Radiographic findings are similar to those for the adult form except for the addition of impaired mandibular growth.Erosions may extend to the mandibular fossa, and the articular eminence maybe destroyed. Similarly, erosion of the anterior or superior aspect of the condyle may occur, and in more severe cases only a pencil-shaped small condyle remains; the condyle may be destroyed.

Manifestations of inhibited mandibular growth, such as deepening of the antegonial notch, diminished height of the ramus, and dorsal bending of the ramus and condylar neck, also may occur unilaterally or bilaterally, resulting in an obtuse angle between the mandibular body and ascending ramus.[3]

PSORIATIC ARTHRITIS

Psoriatic arthritis (PA) is an erosive polyarthritis occurring in patients with a negative rheumatoid factor who have psoriatic skin lesions. The skin lesions precede the joint involvement by several years.[12]

PA affects 5 to 7% of patients with psoriasis. Investigators suspect that the cutaneous and joint manifestations of the disease may be traced to the same immunologic abnormality. PA commonly involves the fingers and spine. Pitting of the nails is observed in 85% of patients.

TMJ involvement was once considered rare in PA, with only 28 cases having been reported in the world literature, but recent studies by **Könönen and Kilpinen** suggest that TMJ involvement is more common than previously believed.[12]

ETIOPATHOGENESIS:

Hereditary – The exact cause is unknown. In some cases, heredity is the factor, transmitted as simple dominant trait.

Precipitating factors – In some cases, the precipitating factors include infection by various microorganisms, metabolic disturbances, endocrine dysfunction, neurogenic factors and trauma.

HLA factors – There is evidence that disease is associated with human major histocompatibility (HLA) antigen complex, implying that either the HLA antigen itself or a linked gene is directly involved in the disease process.[12]

CLINICAL FEATURES:

Skin lesions are found on the trunk, arms, face and scalp. Skin lesions exhibit broad irregular papules or plaques, which are dull red to brownish in colour and are usually covered with a layer of fine silvery scales.

Auspitz's sign – When scrapped, they leave behind small bleeding points; this is called as 'Auspitz's sign.Exacerbation occurs after exposure to ultra violet light.

TMJ Involvement:

Limitation of mandibular movement, deviation to the side of the pain, andtenderness directly over the joint may be observed on examination. Preauricular pain, which is usually unilateral. There isdifficulty in opening the mouth.TMJ is tender. Crepitus, deviation towards the affected side and insmall proportion of cases, deformities are seen.[4,12]

RADIOLOGICAL FEATURES:

Radiographic findings show erosion of the condyle and glenoid fossae rather than proliferation Generalized appearance of irregularity of condylar articular surface. Generalized osteoporosis can occur. Proliferative changes with diminution of joint space.[3]

MANAGEMENT:

The management of PA is similar to the treatment of RA, with an emphasis on physical therapy and NSAIDS that control both pain and inflammation in many cases. Screw wedge appliance is made and is given to the patient.[4]

Antimalarial drugs should not be used because they may cause severe skin reactions in patients with psoriasis. Immunosuppressive drugs, particularly methotrexate, are used for atients with severe disease that does not respond toconservative treatment.[4]

Only when there is intractable TMJ pain or disabling limitation of mandibular movement is surgery indicated. Arthroplasty or condylectomy with placement of costochondral grafts has been performed successfully.[4]

INFECTIVE ARTHRITIS

It is also called as 'Septic arthritis'. It is the infection and inflammation of a joint that can result in joint destruction. It is rare in comparisonwith the incidence of degenerative joint disease and RA in the TMJ. It may be acute or chronic.[3]

ETIOLOGY:

Microorganisms – It is caused by direct spread of organisms like staphylococci, streptococci, pneumococci and gonococci, from an infected mastoid process, tympanic cavity or via blood. **Trauma** – It may also be caused by trauma directly to the joint or infection from maxillary molar parotid gland. **Osteomyelitis and middle ear infection** – It is acute in nature. It can be caused by osteomyelitis and suppurative middle ear infection. **Brucellosis** - Brucellosis can also cause infective arthritis usually of chronic type but acute infection can occur.[4]

PATHOGENESIS:

Dilation of blood vessels of synovial membrane with serous exudate accompanied by the usual stages of inflammation. This is followed by suppuration, with formation of pus andulceration of the synovial membrane. The articular disc is eroded and partly destroyed.The cartilage of the head of the condyle and the fossa are similarly affected.[13]

CLINICAL FEATURES:

It usually occurs in young children with no sex predilection It is always unilateral, in some cases of chronic variety bilateral involvement can occur.Symptoms of septic arthritis of the TMJ include trismus, deviation of the mandible to the affected side, severe pain on movement, and an inability to occlude the teeth, owing to the presence of inflammation in the joint space.

Examination reveals redness and swelling in the region of the involved joint. In some cases, the swelling may be fluctuant and extend beyond the region of the joint.

Large *tender cervical lymph nodes* are frequently observed on the side of theinfection, this helps to distinguish septic arthritis from more common types of TMJ disorders. Serious sequelae include osteomyelitis of the temporal bone, brain abscess, and ankylosis. Facial asymmetry may accompany septic arthritis of the TMJ, especially in children.

Evaluation of patients with suspected septic arthritis must include a review ofsigns and symptoms of

gonorrhea, such as purulent urethral discharge or dysuria.

Diagnosis is made by detection of bacteria on Grams stain and culture ofaspirated joint fluid. The affected TMJ should be aspirated and the fluid obtainedtested by Grams stain and specially cultured for *Neisseria gonorrhoeae*.[13]

RADIOGRAPHIC FEATURES:

No radiographic signs may be present in early stages of the disease, although the space between the condyle and the roof of the mandibular fossa may be widened because of inflammatory exudate in the joint spaces.

Osteopenic (radiolucent) changes of the joint components and mandibular ramus may be evident. More obvious bony changes are seen approximately 7 to 10 days after the onset of clinical symptoms. As a result of the osteolytic effects of inflammation, the condylar articular cortex may become slightly radiolucent, and discontinuity or subtle irregularity of the anterior cortical surface may be evident.

As the disease progresses, the condyle and articular eminence, including the disc, may be destroyed. Osseous ankylosis may occur after the infection subsides.

If the disease occurs during the period of mandibular growth, radiographic manifestations of inhibited mandibular growth may be evident.[3]

MANAGEMENT:

Rest to joint – Adequate rest to the joint should be given and patient is askedto limit movement of joint. Patient should be kept on liquid diet Appropriate antibiotics and analgesics should be given to patient.

Surgical – If there is suppuration, incision and drainage should be carried out.[14]

ANKYLOSING SPONDYLITIS

It is also called as 'Marie – Strumpell disease' and 'rheumatoid spondylitis'. It is a chronic inflammatory connective tissue disease that affects the axial skeleton and central joints including the TMJ.[62]

CLINICAL FEATURES:

It is more commonly seen in young adults and is more common in men than in women.It primarily involves spine joint. This will result in forward and stopped posture. Joint stiffness results from immobility (during sleep) and is typically relievedby heat and exercise.[3]

RADIGRAPHIC FEATURES:

There is flattening of articular surface of the TMJ. Osteophytic formation is common.In some cases, erosion of condylar head is seen.[3]

MANAGEMENT:

Analgesics – NSAID should be given to the patient to relieve pain and inflammation.Gentle physical therapy to improve joint joint mobility is indicated, on occasion moist heat and ultrasound therapy may also be helpful.[3]

II. CONCLUSION

More research is needed to create a better understanding of the etiology of temporomandibular disorder. More randomized, controlled, clinical trials are required to get a better understanding of the treatment approach that yield more successful results than those gained by the regression to the mean phenomenon and placebo effect. Early diagnosis and management is necessary to slow down the progression of disease which leads to a good quality of life.

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