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Conceptual Paper

The Annealed Juncture- Osteoarthritis

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Preface

Osteoarthritis is a degenerative, non-neoplastic joint disorder comprised of progressive erosion of articular cartilage. The condition appears as a consequence of aging, trauma or occupational injury. Osteoarthritis is a frequently discerned arthritis engendering moderate to severe joint disability. Generally, a singular joint or a corresponding joint can be implicated bilaterally in antecedent instances. Individuals with osteoarthritis may be asymptomatic or experience severe disability and joint dysfunction.

Disease characteristics

Commonly, osteoarthritis arises secondary to obesity, enhancing age, diverse anatomical factors, muscular weakness and joint injury appearing due to specific occupation or sports.

Typically, osteoarthritis implicates proximal and distal interphalangeal joints, first carpometacarpal (CMC) joint, hips, knees, first metatarsophalangeal joint and joints of lower cervical or lumbar vertebral column [1,2].

Osteoarthritis depicts a female preponderance wherein male subjects display incrimination of hips whereas female subjects demonstrate implication of knees, hands, first metatarsophalangeal joint or lumbar vertebral column. Commonly, small joints of hands or wrist such as the first carpometacarpal joint may be affected [1,2].

Osteoarthritis is classified contingent to incriminated joints, age of disease onset, radiographic appearance, possible aetiology and disease progression [1,2].

Osteoarthritis may be categorized as primary osteoarthritis or secondary osteoarthritis denominated as:

- Primary or idiopathic osteoarthritis is unrelated to preceding arthritis, appears frequently and demonstrates an absence of predisposing factors such as trauma or associated diseases although aforesaid contributory features may eventually arise [1,2].

- Secondary osteoarthritis is associated with severe arthritis and is contingent to pre-existing joint anomalies. Trauma, joint injury, congenital joint disorder, inflammatory arthritis, avascular necrosis, infectious arthritis, obesity, ochronosis, Paget's disease, osteopetrosis, osteochondritis dissecans, metabolic disorders as hemochromatosis or Wilson's disease, haemoglobinopathy, Ehlers-Danlos syndrome or Marfan's syndrome may predispose to occurrence of secondary osteoarthritis [1,2].

Secondary degenerative joint disease or secondary osteoarthritis occurs in younger individuals. Generally, knee joint of basketball players is implicated with the disorder [2,3].

An estimated 80% of individuals beyond > 65 years are incriminated with osteoarthritis although disease representation occurs beyond 50 years. Around 80% individuals above > 65 years depict radiographic modifications associated with osteoarthritis with a disease incidence of ~ 3.5% of the population [1,3].

Osteoarthritis incriminates the joint in it's entirety and no tissue is exempt from the disease process. Apart from aforesaid contributory factors, mechanical stress and abnormal joint mechanics may engender the condition. Pro-inflammatory markers and proteases eventually mediate joint destruction although the comprehensive pathway which initiates destruction of the entire joint remains obscure [2,3].

Osteoarthritis exhibits preliminary modifications within the articular cartilage with the occurrence of surface fibrillation, irregularity and focal erosion. Cartilaginous erosion extends contiguously to the bone and incriminates entire joint surface [2,3].

Clinical elucidation

Characteristically, osteoarthritis represents with joint pain and decimated joint function although clinical representation may be variable. The condition may be asymptomatic or appear as an incidentally discovered, permanently disabling joint disorder [3,4].

Clinically, osteoarthritis can be adequately discerned with symptoms such as

- Pain which worsens upon activity and ameliorates with rest
- Incriminated subjects beyond > 45 years
- Morning stiffness below < 30 minutes
- Enlargement of joint enclosed bones
- Limited range of motion (ROM) [3,4].

Osteoarthritis can implicate a singular joint (mono-articular) or multiple joints (poly-articular) wherein disease progression is in different stages in diverse joints [4,5].

Common clinical symptoms accompanying osteoarthritis are pain which worsens upon joint motion, crepitus, limited range of motion (ROM) and nerve root compression. Heberden's nodes are discovered in fingers of incriminated female subjects which appear on account of osteophytes arising at distal interphalangeal joints [4,5].

Clinical representation and progression of osteoarthritis is variable and subject-specific. Classical triad of clinical symptoms associated with osteoarthritis are joint pain, stiffness and restricted locomotion. Muscular weakness and problems of balance may occur [4,5].

Pain appears with activity and disappears with rest. Disease progression is associated with continuous pain which affects activities of daily living and consequent severe, functional limitations [4,5].

Bone swelling, joint deformity and joint instability with a feeling of joint "giving way" or "buckling" due to muscle weakness may occur [5].

Histological elucidation

Upon gross examination, preliminary instances display uniform degeneration of articular surface of hyaline cartilage along with fibrillation of cartilaginous matrix and possible cartilage fragmentation. Gradually, cartilaginous thinning and overgrowth of opposing joint surfaces ensue [5,6].

During sectioning, articular surface appears soft and granular with altered outline. Additionally, sloughing of cartilage, bone eburnation where constant friction smoothens and polishes the exposed bone, akin to an ivory surface, joint mice composed of displaced pieces of cartilage and subchondral bone, cysts engendered by effusion of synovial fluid into fractures, osteophytes or bony outgrowths appearing at the margins of articular surface and pannus or fibrotic synovium which superimposes upon peripheral articular surface are exhibited. Secondary infarcts or foci of osteonecrosis are exceptional [5,6].

On microscopy, cartilage injury induces deterioration of collagen matrix with consequent proliferation and aggregation of chondrocytes. Phenotypic alteration into hypertrophic chondrocytes may ensue with cartilaginous outgrowths which ossify to configure osteophytes [5,6].

With continual injury to collagen matrix chondrocytes may undergo apoptosis. Inadequate mineralization of collagen engenders thickening of subchondral bone. Advanced disease may occasionally demonstrate bone cysts. Erosive osteoarthritis infrequently depicts bone erosion [5,6].

On histology, ghost chondrocytes or necrotic chondrocytes demonstrate a lack of nuclei. Significant irregularity of tidemark emerging between calcified and non calcified cartilaginous interface is delineated. Irregular thinning, fragmentation and fibrillation of attenuated cartilage is observed. Subchondral cysts imbued with mucoid fluid are circumscribed by sclerotic bone [6,7].

Generally, a significant inflammatory component is absent although advanced disease demonstrates synovial hyperplasia with emergence of lymphoid follicles. Sterile, acute subchondral inflammation may be exemplified [6,7].

Synovial inflammation and hypertrophy is moderate. Soft-tissue articulations such as ligaments, joint capsule or menisci are incriminated by the disease. End stage disease demonstrates depo-

sition of calcium phosphate and calcium pyrophosphate dihydrate crystals which possibly contribute to synovial inflammation [6,7].

Loose bodies may be configured if a segment of articular cartilage is disrupted. Generally, loose bodies are supported by the synovium, evolve continuously and demonstrate a "tree ring" appearance. Clumped, atypical chondrocytes or unevenly distributed chondrocytes are usually absent [6,7].

Upon cytogenetic analysis, clone specific aberrations of HMGIC gene situated upon chromosome 12q13-15 may be discerned within the synovium in around 5% subjects [6,7].

Differential diagnosis

Osteoarthritis requires a demarcation from conditions such as rheumatoid arthritis, psoriatic arthritis, crystalline arthritis, hemochromatosis, bursitis, avascular necrosis, tendinitis, radiculopathy and various soft tissue abnormalities [8,9].

Investigative assay

A comprehensive history and physical examination with focused assessment of the musculoskeletal system is necessitated. Upon physical examination, incriminated joints depict bony enlargement, crepitus, non-inflammatory joint effusion and limited range of motion (ROM). Tenderness upon joint lines and pain upon passive motion may be discerned [8,9].

Classically, osteoarthritis of the hand demonstrates posterolateral swelling of distal interphalangeal joint or Heberden's nodes, posterolateral swelling of proximal interphalangeal joint or Bouchard's nodes and "squaring" at the base of thumb or first carpo-metacarpal joint [8,9].

Haematological parameters such as complete blood count (CBC), erythrocyte sedimentation rate (ESR), rheumatoid factor (RF) and antinuclear antibodies (ANA) are usually within normal range [8,9].

White blood cell count within the synovial fluid is usually below < 2000 cell per microliter (uL). Cellular infiltrate is predominantly non-inflammatory and composed of mononuclear cells [8,9].

Plain radiographs of the incriminated joint depicts marginal osteophytes, narrowing of joint space along with subchondral sclerosis and cysts. Radiographic features are not concurrent to disease severity and preliminary disease may be devoid of pertinent findings [8,9].

On plain radiography, joint deformity, decimated bone substance or cartilage, reduced joint space, migration of joint, articulation of osteophytes, sclerosis of subchondral bone and subchondral bone cysts are observed [8,9].

Ultrasonography is optimally utilized to discern synovial inflammation, effusion and osteophytes arising due to osteoarthritis [8,9].

Magnetic resonance imaging (MRI) is usually not recommended although preliminary disease can be detected [8,9].

Therapeutic options

Treatment in osteoarthritis is directed towards reduction of pain and maintaining integrity of joint function. Comprehensive disease management is composed of non-pharmacological and pharmacological therapies [10,11].

Mild clinical symptoms can be managed by non-pharmacological therapy whereas advanced disease necessitates a combination of strategies. Non pharmacological therapy is constituted of

- Circumventing activities which overload or aggravate joint pain
- Exercises which ameliorate muscular strength
- Weight loss
- Occupational therapy for decreasing load upon joints with the adoption of brace, splint, cane or crutch [10,11].

Malalignment of joints requires mechanical correction with realignment of knee brace or orthotics [11].

Oral, topical or intra-articular pharmacological agents are efficacious in treating osteoarthritis. Acetaminophen and oral non-steroidal anti-inflammatory drugs (NSAIDs) are the initial and optimal pharmacological agents utilized to manage osteoarthritis [10,11].

Intra-articular, injectable drugs are effective in treating osteoarthritis with acute pain. Glucocorticoid or hyaluronic acid injections may be employed. Duloxetine is moderately efficacious and opioids may be employed in individuals with inadequate response to pharmacotherapy or candidates unfit for surgical intervention [10,11].

Surgical procedures are required for pain relief and enhancing joint function. Proportionate failure of knee or hip joint replacement surgery is minimal [10,11].

Prognostic outcome of osteoarthritis is contingent to incriminated joint, concomitant clinical symptoms and functional impairment. Joint replacement surgery may be associated with superior long-term outcomes in certain instances [10,11].

Osteoarthritis may be accompanied by complications such as pain, difficult ambulation with a tendency to fall, malalignment of joints, decreased range of motion (ROM) of incriminated joint or radiculopathy [10,11].

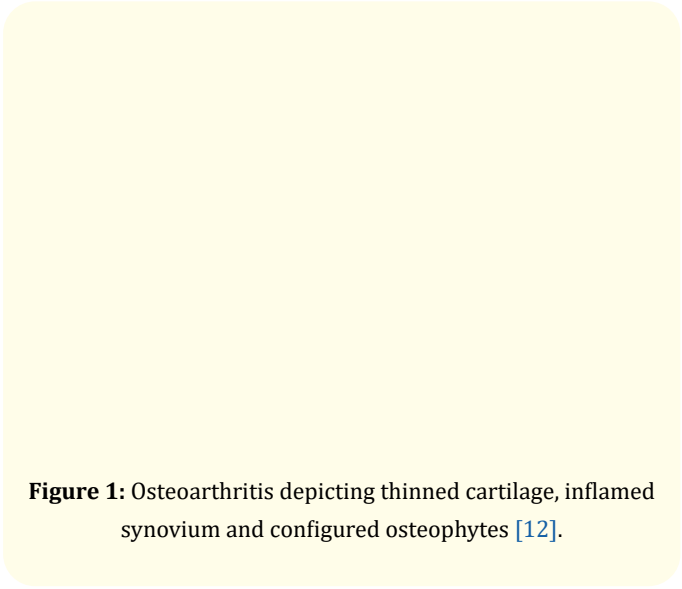

Figure 1: Osteoarthritis depicting thinned cartilage, inflamed synovium and configured osteophytes [12].

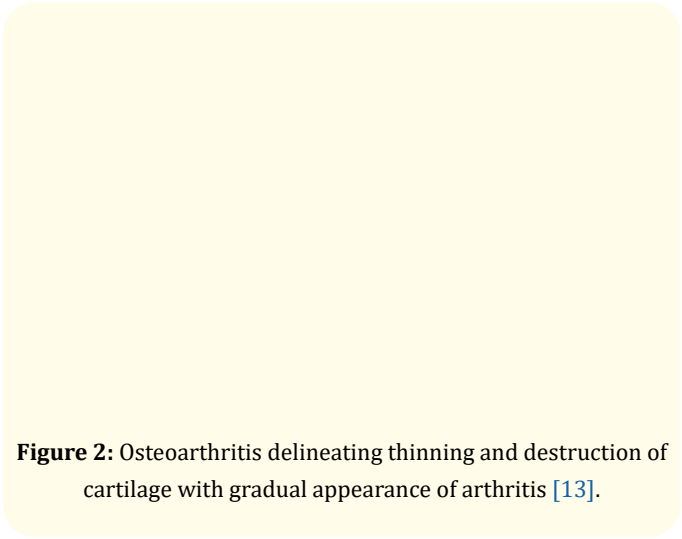

Figure 2: Osteoarthritis delineating thinning and destruction of cartilage with gradual appearance of arthritis [13].

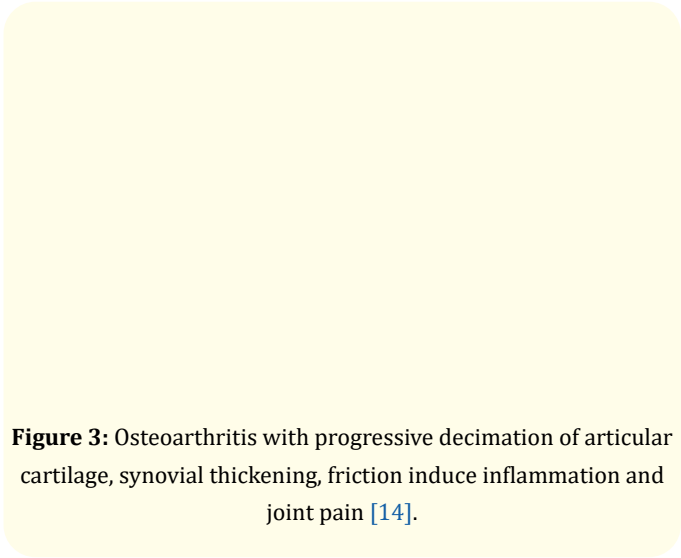

Figure 3: Osteoarthritis with progressive decimation of articular cartilage, synovial thickening, friction induce inflammation and joint pain [14].

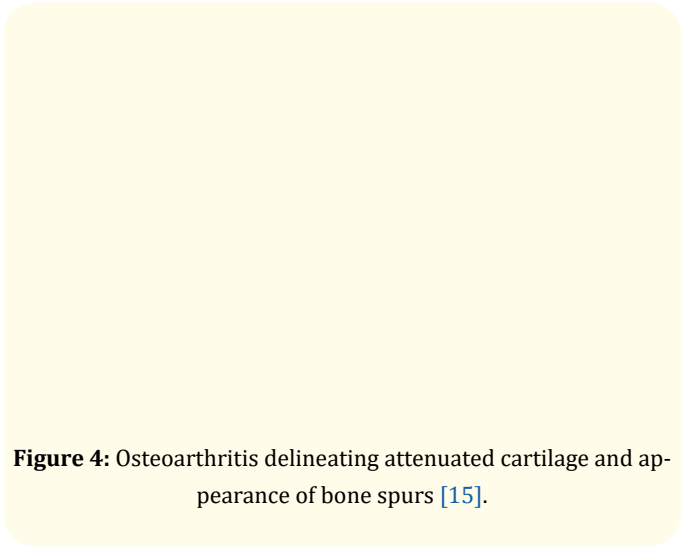

Figure 4: Osteoarthritis delineating attenuated cartilage and appearance of bone spurs [15].

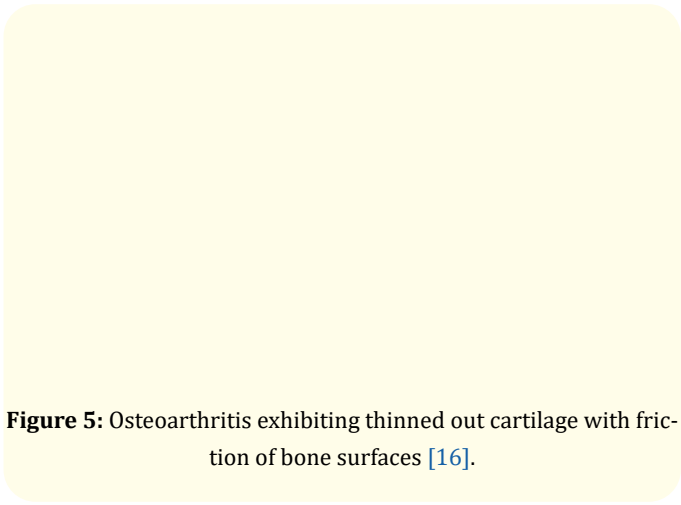

Figure 5: Osteoarthritis exhibiting thinned out cartilage with friction of bone surfaces [16].

Figure 6: Osteoarthritis exemplifying necrotic chondrocytes and irregularity of cartilaginous tidemark [17].

Figure 7: Osteoarthritis enunciating ghost chondrocytes, irregularity and thinning of superimposed articular cartilage [18].

Figure 8: Osteoarthritis demonstrating cartilaginous injury, risk factors, implicated joints and appearance of specific nodes [19].

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12. Image 1 Courtesy: Versus Arthritis.
13. Image 2 Courtesy: Histology A560.
14. Image 3 Courtesy: Osmosis.com.
15. Image 4 Courtesy: The Islander Magazine.

16. Image 5 Courtesy: Clinical Advisor.

17. Image 6 Courtesy: Plastic surgery key.

18. Image 7 Courtesy: Springer link.

19. Image 8 Courtesy: You tube.

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