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DEGENERATIVE ARTHRITIS (*THAHAJJAR-E-MAFASIL*): A CLASSICAL UNANI PERSPECTIVE

***A.M. Muthalib[1*], M.M.M Nifras[2], S. M. Mujahid[3]**

[1]Department of Unani Clinical Medicine, Faculty of Indigenous Medicine, University of Colombo, Sri Lanka.

[2]Department of Unani Pharmacology, Faculty of Indigenous Medicine, University of Colombo, Sri Lanka.

[3]Bandaranayaka Memorial Ayurveda Research Institute, Navinna, Maharagama, Sri Lanka

*Corresponding Author's Email ID: mujasha@yahoo.com

Abstract

In classical Unani literature, Osteoarthritis or Degenerative Arthritis is described as *Tahajjar-ul-Mafasil* under the broader category of *Waja-ul-Maf*asil. According to Unani scholars, all arthritic pains arise due to humoural imbalance. When morbid matter (*mawad-e-fasida*) is diverted from vital organs toward the joints, it tends to accumulate there because the joints are relatively empty spaces that are constantly in motion. Additionally, joints possess a cold temperament and are located in the extremities, far from the heart—the primary source of internal heat. As a result, the accumulated matter becomes thick and hardened, particularly when the *Akhlat* (humours) remain immature or retained for long periods.

From a modern biomedical perspective, Osteoarthritis is recognized as a chronic, progressive, degenerative musculoskeletal disorder with a multifactorial etiology. It is

primarily a non-inflammatory condition affecting movable joints and is characterized by cartilage degradation, joint space narrowing, and structural deterioration that may lead to significant functional impairment.

The objective of this study was to consolidate and analyze Unani concepts related to *Thahajjar-e-Mafasil*. This review is based on classical Unani texts, contemporary journal articles, and online scholarly resources. According to Unani physicians, the main predisposing factor for Osteoarthritis is the accumulation of *ghair tabayi akhlat* (abnormal humours), such as dominance of *Damavi* (sanguine), *Safravi* (choleric), *Balghami* (phlegmatic), *Saudavi* (melancholic) humour, a combination of two abnormal humours, or *reeh* (abnormal gases). Another important factor is *Su'-e-Mizaj* (abnormal temperament), which may manifest as either *Su'-e-Mizaj Sa'ada* (without associated morbid matter) or *Su'-e-Mizaj Maddi* (with associated morbid matter).

Keywords: Degenerative Arthritis, Osteoarthritis, *Tahajjar-ul-Mafasil*, Unani Medicine, *Waja-ul-Mafasil.*

Introduction

In authoritative Unani literature, Osteoarthritis (OA) is described as *Tahajjar-ul-Mafasil* under the category of *Waja-ul-Mafasil*. Unani physicians attribute arthritic conditions to humoural imbalance, wherein morbid matter is diverted from vital organs and accumulates within the joints. Because joints are hollow structures, constantly in motion, possessing a cold temperament, and situated in the extremities—far from the heart, the principal source of heat—these accumulated humors tend to thicken and harden, especially when the *Akhlat* (humours) remain immature or retained.

In contrast, modern medicine defines OA as a chronic, progressive, degenerative musculoskeletal disorder with a multifactorial etiology. It is primarily a non-inflammatory disease of movable joints, characterized by cartilage degradation, joint space narrowing, *and structural deterioration.*

The objective of this review is to compile and analyze the Unani concepts related to Thahajjar-e-Mafasil. This study draws upon classical Unani texts, peer-reviewed journal articles, and

relevant online sources. According to Unani scholars, the major predisposing factor for OA is the accumulation of *ghair tabayi akhlat* (abnormal humours)—including dominance of *Damavi* (sanguine), *Safravi* (choleric), *Balghami* (phlegmatic), *Saudavi* (melancholic) humours, mixtures of abnormal humours, or excess *reeh* (gases). Another important cause is Su'-e-Mizaj (abnormal temperament), which may occur either as *Su'-e-Mizaj Sa'ada* (without morbid matter) or *Su'-e-Mizaj Maddi* (with morbid matter).[1, 2, 3&4.]

Aim and Objectives

The objective of the study was to compile the Unani concepts on *Thahajjar-e-Mafasil* (OA).

Materials and Methods

A systematic search was conducted in both classical Unani literature sources and modern electronic databases. Such as Regional Research Institute of Unani Medicine, Srinaga, Jamia Hamdard Library, New Delhi, CCRUM Library, New Delhi, Electronic Databases such as ,Google Scholar, PubMed, and MEDLINE. Search Terms Used:

Unani Medicine,*Tahajjar-ul-Mafasil*, *Waja-ul-Mafasil*, Osteoarthritis Degenerative Arthritis

Results and discussion

According to Unani System of the Medicine

In most of the authentic *Unani* literature, OA is described as "*Tahajjar-ul-Mafasil*" under the heading of "*Waja-ul-Mafasil*." The cause of all arthritic pains is described to be humoural imbalance. When the morbid matter is diverted from the vital organs toward the joints, it accumulates in the joints, as they are empty and subjected to movement and activity. Moreover, they have cold temperament and lie in the extremities far away from the heart (the center of heat production). Hence, this matter becomes thick and hard, especially if the *Akhlat* (humours) are immature and retained.

According to the *Unani* scholars, the predisposing factors of OA is accumulation of *ghair tabayi akhlat* (abnormal humour), such as dominance of *Damavi* (sanguine/plethoric), or *Safravi* (choleric/bilious), or *Balghami* (phlegmatic), or *Saudavi* (melancholic) khilt (humour), or a mixture of any two *ghair tabayi akhlat* or *reeh* (gases)[1, 2, 3&4].

Wajaul Mafasil is caused by the abnormal change in the one of the four humours and has been categorized into; *Wajaul Mafasil Balghami*, *Wajaul Mafasil Damvi*, *Wajaul Mafasil Safravi* and *Wajaul Mafasil Saudavi*.

Wajaul Mafasil Murakkab When the *change is in more than one humour and at least two humours are involved i.e. Safra (Yellow bile) with Sauda (Black Bile), Dam (Blood) with Balgham (Phlagma), Dam and Safra etc . Wajaul Mafasil Reehi,* this type of *Wajaul Mafasil* is caused by the *Reeh Ghaleez* literally meaning (Bad Gases) [1, 2, 3&4].

The other predisposing factor is *Su e mizaj* (abnormal temperament/derangement in temperament): that can be either *Sue mizaj saada* (without any morbid matter) When the derangement is based on the temperament without the involvement of the humours or *su e mizaj maddi* (with morbid matter) When the derangement involves humours or gasses.

According to the Unani Medicine the term *mizaj* (temperament) is used to describe the normal biochemical equilibrium of the cells, tissues, organs and body as a whole. Any change in this equilibrium is termed as *su-e-mizaj* or derangement of temperament, which may affect either a whole body or may be confined to the vital organs (*A'azae-Raeesa*). This abnormal change in the temperament causing arthritis is of 3 types: 1) *Su-e-Mizaj Har Multahib* 2) *Su-e-Mizaj Barid Munjamid* 3) *Su-e-Mizaj Yabis Munqabiz*.

Asbabe Arzi or Secondary/Precipitating Factors

There are total of 7 aggravating factors mentioned in the Unani classics: 1) Giving up the exercise (Tarke Riyazat) 2) Weakness of stomach (Zaufe M'ada) leading to the absorption of impaired matter 3) Derangement (Su-e-Tarteeb) 4) Sedentary life style 5) Regular and excessive use of alcohol 6) Excessive coitus and exercise after meals 7) Cold and catarrh[4].

Other causes of accumulation of bad humours in the joint are as follows: 1) Giving up the voluntary habitual excretion (Tarke Istefragh-e-Aadati) e.g. vomiting, purgation, venesection etc. 2) Cessation of normal involuntary excretion e.g. menstruation, piles etc. 3) Intestinal colic 4) Drinking of water on empty stomach 5) Anxiety, depression, insomnia etc[4].

According to Allopathy view

Osteoarthritis (OA), the most common form of joint disease, affects mainly the hips, knees, hands and feet, leading to severe disability and loss of quality of life, particularly in the

elderly population. Its importance grows every year with the aging of the population, with a large increase in the elderly population compared to younger patients. The progressive understanding of the pathophysiology of OA, the perception that the process is not purely mechanical and / or aging, and clarification of the inflammatory pathways involved led recently to the clinical application of various drugs and other measures.

In the OA are most significant there is enzymatic degradation of the major structural components - Aggrecan and Collagen principally by aggrecanase, collagenase & stromelysin. There is eventual fissuring of the cartilage surface (fibrillation), development of deep vertical clefts, localized chondrocyte death and decrease in cartilage thickness. The bone immediately below compromised cartilage increases its trabecular thickness. At margins of joint there is production of new fibrocartilage that undergoes ossification to form an Osteophyte (abnormal cell in the bone tissue instead of normal osteocyte). Bone re modelling and cartilage thinning slowly alter the joint, increasing its surface. The synovium undergoes variable degrees of hyperplasia. The outer capsule also thickens and contracts. The muscles that act over the joint commonly show fibre atrophy[5].

This disease is an enigmatic, homogeneous, ubiquitous condition related to age[6]. OA is the most frequent form of arthritis and joint disorder worldwide. In India, it is the second most common joint disease with a prevalence rate of 22%–39%[7]. It is estimated that 11% individuals of age 65 years or more have symptoms of OA, while <1% of individuals of age of 25–34 years experience knee OA[8]. It strikes mainly weight-bearing joints, such as knee and hip joints. In that, Knee arthritis alone accounts for 30%–40%[9].

Epidemiologic principles can be used to describe the distribution of OA in the population and to examine risk factors for its occurrence and progression. For the purpose of epidemiologic investigation, OA can be defined pathologically, radiographically, or clinically. Radiographic OA has long been considered the reference standard, and multiple ways to define radiographic disease have been devised. The most common method for radiographic definition is the Kellgren-Lawrence (K/L) radiographic grading scheme and atlas which has been in use for over four decades. This overall joint scoring system grades OA in five levels from 0 to 4, defining OA by the presence of a definite osteophyte (Grade≥2), and more severe grades by the presumed successive appearance of joint space narrowing, sclerosis, cysts, and

deformity. Other radiographic metrics including semi-quantitative examination of individual radiographic features, such as osteophytes and joint space narrowing, or the direct measurement of the interbone distance as an indicator of the joint space width in the knees and hips are used to investigate progression in epidemiologic studies and clinical trials of disease modifying therapies. More sensitive imaging methods using magnetic resonance imaging (MRI) can visualize multiple structures in a joint and are undergoing evaluation for their role in defining OA and for their usefulness in detecting the effects of potential disease-modifying interventions more quickly than possible with conventional radiographs[10,11&12].

Studies of OA in people who have joint symptoms may be more clinically relevant, because not all persons who have radiographic OA have clinical disease, and not all persons who have joint symptoms demonstrate radiographic OA. Each set of clinical and radiographic criteria may yield slightly different groups of subjects defined as having OA[14].

Clinical features:

Stiffness, pain especially during movement, inflammation of joints, loss of movement are the main symptoms. Main focus of treatment is reduction of pain and inflammation. The ultimate treatment for a disabling joint is replacement[5&6].

Diagnosis:

It is chiefly based on history and clinical examination. X-rays may be used as confirmative tool for diagnosis. Typical changes seen on x- rays include: 1. Joint space narrowing 2. Sub Chondral sclerosis (increased bone formation around the joint) 3. Subchondral cyst formation 4. Osteophytes[10.&11].

Risk Factors for OA

OA has a multifactorial etiology, and can be considered the product of an interplay between systemic and local factors. For example, a person may have an inherited predisposition to develop OA but may only develop it if an insult to the joint has occurred. The relative importance of risk factors may vary for different joints, for different stages of the disease, for the development as opposed to the progression of disease, and for radiographic versus symptomatic disease. There is even some evidence suggesting that risk factors may act differently according to individual radiographic features, such as osteophytes and joint space

narrowing. Whether some of these differences are genuine or are spurious results of different study populations, definition of risk factors and OA, or statistical power, or analytic methods is open to debate.

Aggravating factors in

1. Excessive consumption of dry, cold or stale food. 2. Exposure to severe cold and dry weather. 3. Excessive use of joint. 4. Obesity and overweight. 5. Joint injury. 6. Hereditary. 7. Old age. 8. Biochemical imbalances that put stress on joints. 9. Cellular disorders that lead to abnormal breakdown of cartilage, further chronic disease/s, congenital weakness of the organ, emotional upsets, alcoholism, insomnia, sedentary life, menstrual disturbances in women, heredity, and so on[1,2,5].

Gender and hormones

Women not only are more likely to have OA than men, they also have more severe OA. The definite increase in OA in women around the time of menopause has led investigations hypothesize that hormonal factors may play a role in the development of OA. However, results on effect of oestrogen, either endogenous or exogenous, on OA from observational studies have been conflicting. In a randomized clinical trial (the Heart and Estrogen/Progestin Replacement Study) in a group of older postmenopausal women with heart disease, no significant difference was found in the prevalence of knee pain or its associated disability between those taking estrogen plus progestin therapy or those taking placebo. Data from the Women's Health Initiative showed that, women on estrogen replacement therapy were 15% less likely to require total knee or hip arthroplasty than those not taking such therapy (hazard ratio 0.86; 95% Confidence Interval, 0.70-1.00), but that estrogen combined with progestin therapy was not associated with the risk of joint replacement[15,16,17&18]. the knee is the most studied joint, the highest OA prevalence estimates were found in hand joints. OA of the knee tends to be more prevalent in women than in men independently of the OA definition used, but no gender differences were found in hip and hand OA[19].

Age

Age is a one of the strongest risk factors for OA of all joints. The increase in the prevalence and incidence of OA with age probably is a consequence of cumulative exposure to various

risk factors and biologic changes that occur with aging that may make a joint less able to cope with adversity, such as cartilage thinning, weak muscle strength, poor proprioception, and oxidative damage[14]. The incidence of hand, hip, and knee OA increased with age, and women had higher rates than men, especially after age 50. A leveling off or decline occurred for both groups around the age of 80[19]. In a large study of symptomatic OA we observed incidence rates that increased with age. In women ages 70-89, the incidence of knee OA approached 1% per year[20]. The effect of age is greatest in the elderly for knee and hip OA, but around the menopause for hand OA[21].

Obesity and overweight

Increase in 2 major risk factors for knee OA, aging, and obesity. This underscores the immediate need for greater use of clinical and public health interventions, especially those that address weight loss and self-management, to reduce the impact of having knee OA[22]. Kin. J *et al* (2017) reported that the lifetime risk of symptomatic hand OA among individuals with obesity (47.1%, 95% CI 37.8-56.7%) was 11 percentage points higher than that in individuals without obesity (36.1%, 95% CI 29.7-42.9%)[23].

Race/ethnicity

The prevalence of OA and patterns of joints affected by OA vary among racial and ethnic groups. Kin. J *et al* (2017) concluded that the nearly 1 in 2 women (47.2%, 95% CI 40.6-53.9%) had an estimated lifetime risk of developing symptomatic hand OA by age 85 years, compared with 1 in 4 men (24.6%, 95% CI 19.5-30.5%). Race-specific symptomatic hand OA risk estimates were 41.4% (95% CI 35.5-47.6%) among whites and 29.2% (95% CI 20.5-39.7%) among African Americans [23]. Jordan. JM et al (2003) revealed that serum levels of cartilage oligomeric matrix protein (COMP) levels vary by ethnicity and sex[24].

Diet

Dietary factors are the subject of considerable interest in OA, results of studies, however, are conflicting. One of the most promising nutritional factors for OA is vitamin D. Without sufficient vitamin D, bones can become thin, brittle, or misshapen.

Low vitamin C dietary intake was associated with an increased risk of progression, but not incidence, of both radiographic and symptomatic knee OA among the participants in the

Framingham Study. In the Johnston County Osteoarthritis Project subjects with a high ratio of alpha:gamma tocopherol had 50% lower risk in development of radiographic knee OA. However, results from a controlled clinical trial of vitamin E failed to ameliorate symptoms in patients who had symptomatic knee OA or to prevent knee OA progression, as measured by cartilage volume by MRI.

Animal studies have shown that selenium deficiency is associated with irregular bone formation, decreased bone strength, and abnormalities in type I and II collage in cartilage. In areas of China and eastern Asian where selenium levels in the soil is extreme low, the prevalence of Kashin-Beck Disease, an early onset of osteoarthropathy, was also high and food supplement of selenium decreased the incidence of this disease. Preliminary results from the Johnston County Osteoarthritis Project have shown that sub-optimal selenium levels, measured in toenails, were associated with worse knee OA. However, others have reported that high selenium intake was significantly associated with increased risk of both hip and knee OA.

In one study, high levels of serum vitamin K were associated with a low prevalence of radiographic hand OA in one study, particularly for the presence of large osteophytes.

Sufficient vitamin K status combined with sufficient vitamin D status was associated with better lower-extremity function in 2 knee OA cohorts. These findings merit confirmation in vitamin K and D co-supplementation trials [25]. Zittermann, A reported that very high pharmacological doses of the vitamin K2 menatetrenone has impressively been used to prevent further bone mineral loss and fracture risk in osteoporotic patients[26].

Genetics

Studies have implicated linkages to OA on chromosomes 2q, 9q, 11q, and 16p, among others. Genes implicated in association studies include VDR, AGC1, IGF-1, ER alpha, TGF beta, CRTM (cartilage matrix protein), CRTL (cartilage link protein), and collagen II, IX, and XI. Genes may operate differently in the two sexes, at different body sites, and on different disease features within body sites. OA is a complex disease, and understanding its complexity should help us find the genes and new pathways and drug targets. Results from several studies have shown that OA is inherited and may vary by joint site. Twin and family studies have

estimated the heritable component of OA to be between 50 and 65% with larger genetic influences for hand and hip OA than for knee OA[27]

Conclusion

Osteoarthritis is a common condition with significant impact on quality of life of affected individuals. The Correction of imbalanced humors, relieving the symptoms and restoration of movements. Information about Unani pathology, aeteopathogenesis, Associated factors and types of arthritis were mentioned in Unani literature Thus if all the above information is taken into consideration there is more comprehensive mention of and in those days of life.

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