

Amavata (Rheumatoid Arthritis) in Contemporary Modern Medical Science

KEYWORDS

Amavata, Ama, Agni, Ayurveda, Rheumatoid arthritis.

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Amavata is a particular type of disease that is mentioned in Ayurveda since the period of Madhavkar, under the category of Vata – Kaphaja disorder, which have positive correlation with Rheumatoid arthritis (RA) of conventional modern medicine. Rheumatoid arthritis is most important among those disorders, which can cripple an individual's life. Amavata is a severe form of chronic inflammatory autoimmune systemic disorders, which mainly affects the synovial joints. The synovial inflammation sometimes leads to destruction and ankylosis of affected joints along with substantial loss of functioning and mobility. The available treatment modalities in contemporary science is NSAIDS, DMRDS, steroids etc. but these drugs are related with hazardous side effects and remission is very big problem. The ancient Ayurvedic treatment not only devoid such type of ill effect, but also provides a better way by treating Agni and Ama at its roots.

Introduction:

The signs and symptoms of Amavata are very much similer to Rheumatoid arthritic(RA). RA is a systemic inflammatory disease characterized by serositis (inflammation of the lining surfaces of the joints, pericardium and pleura), rheumatoid nodules and vasocentric inflammation (vasculitides) [1]. The hallmark of the disease is chronic, symmetric, polyarthritis (synovitis) that affects the hands and feet, although any joint lined by a synovial membrane may be involved. Systemic involvement may lead to weight loss, low-grade fever, and malaise, in additional to the vascular destruction. The cause of RA is not completely understood. The process involves an inflammatory response of the capsule around the joints (synovium) secondary to swelling (turgescence) of synovial cells, excess synovial fluid, and the development of fibrous tissue (pannus) in the synovium. It also affects the underlying bone(focal erosions) and cartilage(thinning and destruction).[1] RA can also produce diffuse inflammation in the lungs, the membrane around the heart, the membranes of the lung (pleura), and whites of the eye, and also nodular lesions, most common in subcutaneous tissue. It is a clinical diagnosis made primarily on the basis of symptoms and physical examination. X-rays, laboratory testing, and synovial fluid analysis might help support a diagnosis or exclude other diseases with similar symptoms.

Treatments include both medication and nonpharmacological measures - the goal being to control joint inflammation and prevent joint damage and disability. Non-pharmacological treatment includes physical therapy, splints and braces, occupational therapy and dietary changes but these do not stop the progression of joint destruction. painkillers and anti-inflammatory drugs, including steroids, suppress symptoms, but do not stop the progression either. Disease-modifying antirheumatic drugs (DMARDs) may slow or halt the progress of the disease. [2] Biological DMARDS like anti-TNF agents are effective but usually avoided in persons with active disease or hypersensitivity to these agent.[3] They have been shown to decrease the number of tender or swollen joints and the pain and disability due to the disease but there is little data about side

effects.[4]

RA affects between 0.5 and 1% of adults in the developed world with between 5 and 50 per 100,000 people newly developing the condition each year.[5] Onset is most frequent during middle age, but people of any age can be affected.[6]

Signs and symptoms:

Morning stiffness: Stiffness in and around the joints lasting 1 hour before maximal improvement.

Arthritis of three or more joint areas: At least three joint areas, observed by a physician simultaneously, have soft tissue swelling or joint effusions, not just bony overgrowth. The 14 possible joint areas involved are right and left Proximal interphalangeal, metacarpophalangeal, wrist, elbow, knee, ankle and metatarsophalangeal joints[7][8].

Arthritis of hand joints: Arthritis of wrist, metacarpophalgeal joint or proximal interphalangeal joint[9].

Symmetric arthritis: Simultaneous involvement of same joint areas on both sides of the body.

Rheumatoid nodules: Subcutaneous bony prominences over extensor surfaces or juxtaarticular regions observed by a physician[7].

Serum Rheumatoid Factor: Demonstration of abnormal amounts of serum rheumatoid factor (auto antibodies reactive with Fc portion of IgG) by any method for which the result has been positive in less than 5 percent of normal control subjects.

Radiographic changes: Typical changes of RA on posteroanterior hand and wrist radiographs, which must include erosions or unequivocal bony decalcification localized in or most marked adjacent to the involved joints.

As the pathology progresses the inflammatory activity leads to tendon tethering and erosion and destruction of the joint surface, which impairs range of movement and

leads to deformity. The fingers may suffer from almost any deformity depending on which joints are most involved. Specific deformities, which also occur in osteoarthritis[10], include ulnar deviation, boutonniere deformity, swan neck deformity and "Z-thumb." "Z-thumb" or "Z-deformity" consists of hyperextension of the interphalangeal joint. fixed flexion and subluxation of the metacarpophalangeal joint and gives a "Z" appearance to the thumb.[9] The hammer toe deformity may be seen. In the worst case, joints are known as arthritis mutilans due to the mutilating nature of the deformities[11].

Diagnostic Criteria For Rheumatoid Arthritis:

In 2010 the 2010 ACR / EULAR Rheumatoid Arthritis Classification Criteria were introduced.[12] [13] The new criteria is not a diagnostic criteria but a classification criteria to identify disease with a high likelihood of developing a chronic form. [1] However a score of 6 or greater unequivocally classifies a person with a diagnosis of Rheumatoid arthritis. These new classification criteria overruled the "old" ACR criteria of 1987 and are adapted for early RA diagnosis. The "new" classification criteria, jointly published by the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) establish a point value between 0 and 10. Four areas are covered in the diagnosis: [12]

Joint involvement, designating the metacarpophalangeal joints, proximal interphalangeal joints, the interphalangeal joint of the thumb, second through fifth metatarsophalangeal joint and wrist as small joints, and shoulders, elbows, hip joints, knees, and ankles as large joints:

Involvement of 1 large joint gives 0 points

Involvement of 2-10 large joints gives 1 point

Involvement of 1-3 small joints (with or without involvement of large joints) gives 2 points

Involvement of 4-10 small joints (with or without involvement of large joints) gives 3 points

Involvement of more than 10 joints (with involvement of at least 1 small joint) gives 5 points

Serological Parameters - including the rheumatoid factor as well as ACPA - "ACPA" stands for "anticitrullinated protein antibody":

Negative RF and negative ACPA gives 0 points

Low-positive RF or low-positive ACPA gives 2 points

High-positive RF or high-positive ACPA gives 3 points

Acute Phase Reactants:

1 point for elevated erythrocyte sedimentation rate, ESR, or elevated CRP value (c-reactive protein)

Duration Of Arthritis: 1 point for symptoms lasting six weeks or longer.

Lab Diagnosis:

The RA (Amavata) can be diagnosed by assessing X-ray, magnetic resonance imaging (MRI), ultrasound, color Doppler and power Doppler ultrasound, RA Fector, Anti CCP (Anticitrullinated protein antibodies), C-Reactive Protein, Anti-MCV assay (antibodies against mutated citrullinated Vimentin) ESR, Interlukin- 1 & 6, Immunological markers and Others(CBC, LFT, RFT etc.).

Management:

According to Modern medical sciences, there is no cure for RA, but treatments can improve symptoms and slow the progress of the disease. Disease modifying treatment has the best results when it is started early and aggressively.[14] The goals of treatment are to minimize symptoms such as pain and swelling, to prevent bone deformity (for example, bone erosions visible in X-rays), and to maintain day-to-day functioning.[15] This can often be achieved using two main classes of medications: analgesics such as NSAIDs, and disease-modifying antirheumatic drugs (DMARDs). [16] RA should generally be treated with at least one specific anti-rheumatic medication. [15] The use of benzodiazepines (such as diazepam) to treat the pain is not recommended as it does not appear to help and is associated with risks. [17] Analgesics, other than NSAIDs, offer lesser, but some benefit with respect to pain.[8] whilst not causing the same level of gastrointestinal irritation.

Conclusion:

According to Ayurveda Amavata is a Disease Caused by Ama along with vitiated vata. Ama is a Sanskrit Word that means Apakva Annarasa or undigested or unriped food elements. It is formed when the Jatharagni is weak, the result is the accumulation of apakva Annarasa or Ama in the stomach. Vata dosha takes the ama in to systemic circulation and in to the body tissue, where Ama combines with the doshas and spreads all over the body and produces the symptoms like Sandhi shotha (Swelling), Sandhi shula (Pain), Stabdhata (stiffness) and other systemic sign and symptoms. The symptoms of Aamavata are mainly resembles the disease Rheumatoid Arthritis(RA) in modern medical science. RA is a chronic, systemic inflammatory disease involving the joints. Inflammation and damage to joints cause marked disability. This varies with time and is unique to an individual, depending on the exact ways their joints are involved. Some people with rheumatoid arthritis are simply unable to do normal things. The disability is also psychological and social. There is no any effective treatment for RA still today, but Ayurveda can give the appropriate solution of this problem.

References:

- Shah, Ankur. Harrison's Principle of Internal Medicine (18th ed.). United States: McGraw Hill. p. 2738. ISBN :978-0-07174889-6.
- Majithia V, Geraci SA (2007). "Rheumatoid arthritis: diagnosis and management". Am. J. Med. 120 (11): 936-9
- 3. Shah, Ankur. Harrison's Principle of Internal Medicine (18th ed.). United States: McGraw Hill. p. 2749. ISBN:978-0-07174889-6.
- Singh, Jaswinder (2009). "Biologics for rheumatoid arthritis: an overview 4. of Cochrane reviews". Cochrane Database of Systematic Reviews.
- Scott DL, Wolfe F, Huizinga TW (Sep 25, 2010)."Rheumatoid arthritis". 5. Lancet 376 (9746): 1094-108.
- "Handout on Health: Rheumatoid Arthritis". National Institute of Arthritis and Musculoskeletal and Skin Diseases. April 2009. Retrieved 2013-
- Landré-Beauvais AJ (1800). La goutte asthénique primitive (doctoral thesis). Paris. reproduced in LandréBeauvais AJ (2001). "The first description of rheumatoid arthritis. Unabridged text of the doctoral dissertation presented in 1800". Joint Bone spine 68 (2): 130-43.
- Turesson C. O'Fallon WM, Crowson CS, Gabriel SE, Matteson EL (2003). "Extra-articular disease manifestations in rheumatoid arthritis: incidence trends and risk factors over 46 years". Ann. Rheum. Dis. 62 (8): 722-7.
- Davidson's principles and practice of medicine. (21st ed.). Edinburgh: Churchill Livingstone/Elsevier. 2010. ISBN:978-0-7020-3084-0. |first1=

RESEARCH PAPER

- missing |last1= in Authors list (help)
- "An approach to Early Arthritis". Pn.lifehugger.com. 12 January 2009. Archived from the original on 2010-05-27.
- Gaffo A, Saag KG, Curtis JR (2006). "Treatment of rheumatoid arthritis".
 Am J Health Syst Pharm 63 (24):2451–2465.
- Aletaha D, Neogi T, Silman AJ et al. (2010). "2010 rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative". Ann. Rheum. Dis. 69 (9): 1580–8.
- Aletaha, Daniel; Neogi et. al. "2010 Rheumatoid arthritis classification criteria: An American College of Rheumatology/European League Against Rheumatism collaborative initiative". Arthritis & Rheumatism 62 (9): 2569–2581.
- Saag KG, Teng GG, Patkar NM, et al. (2008). "American College of Rheumatology 2008 recommendations for the use of nonbiologic and biologic disease-modifying antirheumatic drugs in rheumatoid arthritis". Arthritis Rheum
- Amy M. Wasserman (2011). "Diagnosis and Management of Rheumatoid Arthritis". American Family Physician
- Chris Deighton, Rachel O'Mahony, Jonathan Tosh, Claire Turner, Michael Rudolf, and Guideline Development Group (2009). "Management of rheumatoid arthritis: summary of NICE guidance". British Medical Journal
- Richards BL, Whittle SL, Buchbinder R (Jan 18, 2012). Richards, Bethan L, ed. "Muscle relaxants for pain management in rheumatoid arthritis". Cochrane database of systematic reviews