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OVERVIEW OF AMAVATA

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ABSTRACT

Rheumatoid arthritis is a disease of Rasavaha strotasa, is frequently contrasted. Amavata is the result Of Agnidushti, Amotpatti, and Sandhivikruti. The most effective remedy for this disease will be one that restores Agni to normal, breaks down Ama, regulates Vata, and keeps Sandhi and Sandhistha Shleshma in good condition. The disease and its aftereffects, which cause joint deformities that last a lifetime, are not well known to many members of society. The classical texts mention a variety of herbal and Ayurvedic remedies that are highly successful in treating amavata.

KEYWORDS: Amavata, Ama, Rheumatoid Arthritis, Agni-dushti, Amotpatti, Sandhivikruti.

INTRODUCTION

Amavata is a disease in which vitiation of Vata Dosha and accumulation of Ama take place in joints, which simulate rheumatoid arthritis (RA) in modern parlance.[1] Ama is a maldigested product, which is not homogeneous for the body. Whenever that Ama gets localized in the body tissue or joints, it can lead to production of pain, stiffness, swelling, tenderness, etc., in the related joints.[2] The features of Amavata are much identical to RA, an autoimmune disorder which causes chronic inflammatory and symmetrical polyarthritis.[3] In Ayurveda, Nidana Parivarjana (avoidance of causative factors) is considered as the first and foremost line of management for any disease. Virechanakarma is a Shodhana process (biological purification of the body) to balance the vitiated Dosha in general and Pitta Dosha in particular.[4] Hence, this study included both the treatment modalities, i.e. Nidana Parivarjana and Virechanakarma to manage Amavata effectively.

Women are affected approximately 3 times more often than men. Studies suggest that genetics & environmental influences are important in the susceptibility to R.A. There is no doubt modern system of medicine play an important role in overcoming agony of pain, restricted movement & disability caused by the disease. Simultaneously prolonged use of allopathic medicines are not only giving rise to many side effects, toxic symptoms & adverse reactions even including many organic impairments.

Etymology of Amavata

1. "Amena sahita vata Amavata". The virulent Ama circulates in the whole body propelled by the vitiated vata doshas producing blockage in the body channels that stations itself in the sandhi giving rise
2. to Amavata.
3. The combination of Ama & Vata form Amavata, it shows the predominance of Ama & vata in the samprapti of Amavata.
4. Amavata is produced by Jeerna when combined with vata. clarification The Jatharagni and Dhatwagnis produce Ama through Agnimandya. As the primary causative factor in Amavata, ama is responsible for many ailments, notwithstanding this fact. Joints of the hasta, pada, sira, trika, gulpha, janu, and uru are the primary sites of disease manifestation as Ama and Vata vitiated simultaneously. Alasya, Gouravam, Shotha, Aruchi, Angamarda, and Trishna are the primary symptoms that are generated. In Amavata, Ama's role Ama is the one who causes the Amavata manifestation. Rheumatoid arthritis and amavata, a disease of Rasavaha strotasa, are frequently contrasted. Of Agnidushti, Amotpatti, and Sandhivikruti, Amavata is the result. The treatment plan that would restore Agni to normal, Metabolize Ama, Regulate Vata, and Maintain good Sandhi and Sandhistha Shleshma.

In many cases, rheumatoid arthritis results in permanent incapacity since it affects not just the joints but also the internal organs. This autoimmune condition currently has no known cure; instead, each patient's symptoms are treated individually. Here, we provide a brief overview of the traditional and modern therapies that can be used to manage this complicated illness in patients.This will be the most effective in treating this illness and regulate Agni, Metabolize Ama, Regulate Vata, and Maintain Healthy Sandhi and Sandhistha Shleshma.

The inflammatory, symmetrical, and chronic rheumatoid arthritis (RA) is an autoimmune disease that starts off small and gradually spreads to larger joints, the skin, eyes, heart, kidneys, and lungs. Joint bone and cartilage are frequently damaged, and ligaments and tendons deteriorate.[5] Patients typically experience excruciating pain as a result of all this joint deterioration, which also results in bone erosion. Some common signs and symptoms of RA are sore, swollen, and heated joints; weariness; fever; weight loss; and subcutaneous rheumatoid nodules. In the morning, the affected joints may remain stiff for more than thirty minutes. With remissions and exacerbations, this disease often manifests itself between the ages of 35 and 60. In addition, juvenile RA (JRA), which is comparable to RA but does not have the rheumatoid factor, can affect young children even before the age of sixteen.[6,7,8]

Clinically, RA can be distinguished from OA because RA affects the proximal interphalangeal (PIP) and metacarpophalangeal (MP) joints, whereas OA usually affects the distal interphalangeal (DIP) joint. The most prevalent kind of arthritis, OA is brought on by deterioration rather than an autoimmune disease. The immune system, heart, or lungs are unaffected by it. Furthermore, unlike RA, which is symmetrical, OA usually only affects one side of the body. The prolonged morning stiffness that RA patients experience for at least ≥1 hour is another distinguishing feature. Patients with OA may experience stiffness in the morning, although this usually goes away or becomes better in 20 to 30 minutes.[8,9]

The goals of treatment for RA are to reduce joint inflammation and pain, maximize joint function, and prevent joint destruction and deformity. Treatment regimens consist of combinations of pharmaceuticals, weight-bearing exercise, educating patients about the disease, and rest. Treatments are generally customized to a patient's needs and depend on their overall health. This includes factors such as disease progression, the joints involved, age, overall health, occupation, compliance, and education about the disease.[10]

Ayurvedic management

The following treatments are used to address amavata according to Ayurveda

•Langhana, or therapeutic fasting,

•Swedana, or dry fermentation

•Appetizer usage (Deepana)

•Use of bitter and strong-tasting foods and medication

•medicinal urging (virechana)

•Basti, a therapeutic enema.[11]

First-Line Management

NSAIDS and Corticosteroids

The overall goal of first-line treatment is to relieve pain and decrease inflammation. Medications, considered to be fast-acting, are nonsteroidal anti-inflammatory drugs (NSAIDs) including acetylsalicylate (Aspirin), naproxen (Naprosyn), ibuprofen (Advil and Motrin), and etodolac (Lodine). Aspirin is an effective anti-inflammatory for RA when used at high doses, due to the inhibition of prostaglandins. It is one of the oldest NSAIDs used for joint pain. Side effects of aspirin at high doses include tinnitus, hearing loss, and gastric intolerance. There are other NSAIDs that are newer on the market than aspirin and just as effective. In addition, these drugs require fewer doses per day. NSAIDs work by inhibiting cyclo-oxygenase to prevent the synthesis of prostaglandins, prostacyclin, and thromboxanes. Common side effects are nausea, abdominal pain, ulcers, and gastrointestinal (GI) bleeding. These symptoms can be reduced if taken with food, antacids, proton pump inhibitors, or misoprostol (Cytotec), An even newer NSAID called celecoxib (Celebrex) is a selective Cox-2 inhibitor that has less risk of GI side effects.[12]

Corticosteroids are a more potent anti-inflammatory medication than NSAIDs, but they come with greater side effects. For this reason, they are only indicated for a short period of time at low doses, during exacerbations or flares of RA. Intra-articular injections of corticosteroids can be used for the local symptoms of inflammation.[13] They work by preventing the release of phospholipids and decreasing the actions of eosinophils, thereby decreasing inflammation. Their side effects include bone-thinning, weight gain, diabetes, and immunosuppression. Advising the patient to take calcium and vitamin D supplementation can prevent thinning of the bone. Side effects can be reduced by gradually tapering doses as a patient's condition improves. It is important to not abruptly discontinue injected or oral corticosteroids as this can lead to suppression of the hypothalamic-pituitary-adrenal axis (HPA) or flares of RA.[14]

Opioid Analgesics

Whittle et al.[15] addressed the question of the use of opioid analgesics for patients with pain due to RA. From their conclusions, weak opioids such as codeine, dextropropoxyphene, and tramadol may play an effective role in the short-term management of pain caused by RA, but the adverse effects outweigh the benefits. They recommend that other analgesics be considered first.[16]

Second-Line Management

Disease-Modifying Antirheumatic Drugs

The overall goal of second-line treatment is to promote remission by slowing or stopping the progression of joint destruction and deformity. Medications are considered to be slow-acting because they take from weeks to months to be effective. Diseasemodifying antirheumatic drugs (DMARDs) can also reduce the risk of developing lymphoma that can be associated with RA.[17]

Methotrexate (MTX) is the initial second-line drug (also considered an anchor drug). It is an analog to folic acid that competitively inhibits the binding of dihydrofolic acid (FH2) to the enzyme that is responsible for converting FH2 to folinic acid (FH4).

Without FH4, the metabolism of purine and pyrimidine is impaired, and the synthesis of amino acids and polyamine is inhibited. MTX is an immunosuppressive drug that requires regular blood tests due to its side effects, i.e., liver problems, cirrhosis, and bone marrow deterioration. Folic acid supplementation can reduce the risk of side effects. It is an effective DMARD, has a lower incidence of side effects than other DMARDs, and has dosage flexibility, meaning that doses can be adjusted as needed.[18] Until now, there is convincing data showing the benefits of combinations of conventional synthetic DMARDs over MTX monotherapy. However, biological and synthetic DMARDs in combination are reported to be better than MTX but with more side effects and greater costs.[14,19]

Hydroxychloroqyine (Plaquenil) is an antimalarial drug and can be used for longterm treatment of RA. This drug decreases the secretion of monocyte-derived proinflammatory cytokines. Common side effects include problems in the GI tract, skin, and central nervous system. The eyes, in particular, can be affected when this drug is taken at high doses. Patients on this medication require routine consultation with an ophthalmologist.[20]

Sulfasalazine (Azulfidine) is a DMARD typically used in the treatment of irritable bowel disease. Combined with anti-inflammatory medications, this DMARD can be used to treat RA. The mechanism of action of this drug in the treatment of RA has not been identified. It is thought that sulfapyridine, a reduced form of the medication after administration, may reduce secretions of interleukin (IL)-8 and monocyte chemoattractant protein (MCP). This drug has side effects of GI and central nervous system symptoms as well as rash. It is usually well-

tolerated among patients, but should be avoided in patients with sulfa allergies since it contains sulfa and salicylate compounds.[21]

Gold salts, such as aurothioglucose (Solganal), auranofin (Ridaura), gold sodium thiomalate (Myochrysine), and D-penicillamine (Depen and Cuprimine) have been used frequently in the treatment of RA. These DMARDs require frequent blood and urine tests due to damage to the bone marrow and kidneys. They have not been used recently due to the more effective treatments, particularly MTX. Other immunosuppressive medications like azathioprine (Imuran), cyclophsphamide (Cytoxan), chlorambucil (Leukeran), and cyclosporine (Sandimmune) can be employed but are typically reserved for patients with very aggressive RA or complications of the disease.[22,23]

Newer Medications

Leflunomide is an oral medication that is converted to malononitrilamide, which inhibits the synthesis of ribonucleotide uridine monophosphate pyrimidine. It relieves symptoms and retards the progression of RA. It is recommended to be used in combination with MTX but can constitute a monotherapy if patients do not respond to MTX. Side effects include hypertension, GI upset, liver damage, leukopenia, interstitial lung disease, neuropathy, rash, and bone marrow damage.[24,25]

Biologics, also known as biological DMARDs, are rapidly effective in retarding the progression of the joint damage caused by RA. They are considered to be a more "direct, defined and targeted" method of treatment.[26] Nonetheless, biologics pose the problem of serious side effects, such as increased risk of infections. Other common side effects include neurologic diseases like multiple sclerosis and lymphoma.[27,28,29]

Tumor necrosis factor (TNF) is a messenger protein that promotes inflammation in joints. Biologic medications such as etanercept (Enbrel), infliximab (Remicade), adalimumab (Humira), golimumab (Simponi), and certolizumab pegol (Cimzia) are all TNF inhibitors that prevent the recruitment of the cells that cause inflammation, bringing rapid symptom relief. They are recommended if other second-line medications are not effective. Unfortunately, these medications tend to be very expensive and their role in treating patients at various stages of RA and with various mechanisms of action is a matter of continuous investigation. They are often used in combination with other DMARDs, especially MTX. TNF inhibitors

are contraindicated in patients with congestive heart failure of demyelinating diseases. Each biologic medication has a different mode of administration.[30,31,32]

Anakinra (Kineret) is a drug that is injected subcutaneously daily. It works by binding to IL-1, a chemical messenger of inflammation. It can be used in combination with other DMARDs or as a monotherapy, but due its low response rate compared to other biologics, it is not used as frequently.[33,34] Rituximab (Rituxan) is useful in RA because it depletes the B cells responsible for inflammation and the production of abnormal antibodies. Typically used in the treatment of lymphoma, this drug can be used in cases of RA where TNF inhibitors have failed. In addition, rituximab has shown benefits in treating the complications of RA, such as vasculitis and cryoglobulinemia. It is administered as an intravenous infusion in 2 doses, 2 weeks apart, every 6 months.[35, 36] Abatacept (Orencia) is a biologic medication that works by blocking T cell activation. This is given as an intravenous infusion once a month or subcutaneously once a week. It is used in patients who have not been effectively treated with traditional DMARDs.[37]

Tocilizumab (Actemra) is a biologic that works by blocking IL-6, a chemical messenger of inflammation. It is administered via intravenous infusion given monthly or via weekly subcutaneous injections. It is also used for patients who have not been effectively treated with traditional DMARDs.[38] Lastly, tofacitinib (Xeljanz) has a different mechanism of action and works by blocking Janus kinases within cells, which are enzymes of inflammation. For this reason, it is known as a JAK inhibitor. This medication is used for patients who have not been effectively treated with MTX. Tofacitinib is taken orally twice daily, alone or in combination with MTX. It should not be used in combination with traditional biologic medications or other potent immunosuppressants.[39,40]

Surgery

Joint surgery in patients with RA reached a peak in the 1990s. However, a 2010 study showed decreased rates of joint surgery in RA patients 40–59 years of age. In contrast, patients older than 60 years had increased rates of surgery.[41] Surgery is a last resort for the treatment of RA. Indications include intractable joint pain or functional decline due to joint destruction after all nonsurgical approaches have failed. At this point, the disease is considered “end-stage.” The goal of surgical management is to relieve pain for the patient and restore the function of the joints. A patient needing surgical treatment should be evaluated based on their customized needs because there are many different types of surgery.

A tenosynovectomy involves the excision of inflamed tendon sheaths or repairing a recent tendon rupture, most commonly in the hand.[42] Radiosynovectomy is an alternative to surgical synovectomy; it involves intra-articular injection of small radioactive particles, is cost-effective, and can treat multiple joints simultaneously.[43] Repair of ruptured tendons can also be done through arthroscopy, most commonly in the rotator cuff of the shoulder. Excision of an inflamed synovium via arthroscopy or open synovectomy is no longer commonly used due to the availability of more effective options. Another surgical option is osteotomy. In this procedure, weight-bearing bones are realigned to correct valgus or varus deformities, most commonly in the knee.[44] Joint fusion can be done to stabilize joints that are not easily replaceable such as the ankle, wrist, thumb, and cervical spine. A procedure for soft-tissue release can be done to correct severe contractures around joints causing decreased range of motion; this is an older procedure that is not commonly utilized.[45] Small-joint implant arthroplasty can be done to reduce pain and improve hand function, most commonly in the metacarpophalangeal joints. Metatarsal-head excision arthroplasty is done to alleviate severe forefoot pain. Lastly, a total joint replacement involves removing the damaged joint and replacing it with a metallic, plastic, or ceramic prosthesis. This is most commonly done in the shoulder, elbow, wrist, hip, knee, and ankle.[46,47] The major contraindication for surgical joint replacements is the presence of active systemic articular infection.

Other Therapies

It has been found that, in contrast to suggestions in the past, there are no specific foods that patients with RA should avoid. The idea that diet can "aggravate" symptoms is no longer accepted as true.[48] Home remedies have been proven to be helpful for patients suffering from RA, although they are not as effective as DMARDs. Fish oils and omega-3 fatty acid supplements are beneficial for the short-term symptoms of RA. Cumin has been shown to have anti-inflammatory effects in patients with this disease. Calcium and vitamin D supplementation can be helpful in preventing osteoporosis. Lastly, folic acid can help to prevent the side effects of MTX.[49]

Patients with RA also benefit from physical and occupational therapy. It is recommended that they perform exercise regularly to maintain joint mobility and strengthen the muscles around the joints. Movement exercises that are less traumatic for joints but good for muscle strength include swimming, yoga, and tai chi. Applying heat- and cold-packs before and after exercise minimizes painful symptoms. Studies are being done on different types of connective tissue

collagen, to better understand and reduce RA disease activity. Lastly, with the scientific advancements and enhanced understanding of the molecular mechanisms, newer and better treatment options should become available in the near future.[50,51,52,53,54,55]

CONCLUSION

RA is a debilitating, chronic, inflammatory disease, capable of causing joint damage as well as long-term disability. Early diagnosis and intervention are essential for the prevention of serious damage and loss of essential bodily functions. The treating physician should consider adhering to treat-to-target (T2T) recommendations[56], by first outlining the aims and then implementing the protocols to achieve and assess them. Furthermore, early referral to a specialist can help to ensure better treatment outcomes.

With advances in the field of molecular medicine, we have a better understanding of disease mechanisms which can aid in the designing of more effective treatments. Old treatment modalities have been optimized and new ones have been produced. Gene array analysis is proving beneficial in finding out which patients will be more responsive to specific medications. This customization will allow for more rapid treatment as well as decrease the likelihood of disease progression during the experimental phase to seek an appropriate treatment for a particular patient. Gene array analysis is also being used to determine which patients are at greater risk for more aggressive forms of RA. It is foreseen that treatment methods will face tremendous improvements in the management of RA.

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