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REVIEW ARTICLE

The Interplay between Gut Microbiota and *Amavata*: An Ayurvedic and Modern Perspective

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ABSTRACT

Amavata is a disorder characterized by the simultaneous vitiation of *Ama* (undigested toxic metabolic by-products) and *Vata*, which closely resembles rheumatoid arthritis (RA) in modern medicine. The disease manifests with pain, stiffness, swelling of joints, impaired mobility, and systemic features of inflammation. Contemporary research has increasingly highlighted the pivotal role of gut microbiota (GM) in immune modulation and the pathogenesis of autoimmune conditions, such as RA. This convergence opens a promising interface between classical Ayurvedic concepts and modern biomedical insights. From an Ayurvedic standpoint, deranged *Agni* (digestive fire) leads to the formation of *Ama*, which circulates through *Srothas* and accumulates in joints, triggering inflammation under the influence of vitiated *Vata*. Modern studies suggest that dysbiosis of GM contributes to increased intestinal permeability, altered immune tolerance, and systemic inflammation, which parallels the Ayurvedic description of *Ama* entering systemic circulation. Moreover, specific bacterial strains have been linked with RA pathogenesis, further strengthening the connection between gut health and joint disorders. Thus, the interplay between GM and Amavata illustrates a profound convergence of traditional Ayurvedic wisdom and modern biomedical science. Understanding this relationship offers novel integrative strategies for prevention and management, emphasizing the centrality of gut health in systemic well-being.

1. INTRODUCTION

Amavata is a chronic immune-inflammatory systemic disorder caused by the formation of *Ama* and its association with *Vata* at *Kaphasthana* (joints). Ama is a maldigested product, which is not homogeneous for the body. Vitiated *Vayu* circulates the *Ama* all over the body through *Dhamanis,* takes shelter in the *Shleshma Sthana (Amashaya, Sandhi*, etc.) Whenever that Ama gets localized in the body tissue or joints, it can lead to the production of pain, stiffness, swelling, tenderness, etc., in the related joints. The features of *Amavata* are much identical to rheumatoid arthritis (RA), an autoimmune disorder that causes chronic inflammatory and symmetrical polyarthritis.[1] The equilibrium of gut microbiota (GM) plays a role in the homeostasis of host immune mechanisms. Hence, when there is any alteration in GM, the immune mechanism is vulnerable to an inflammatory condition like *Amavata.*[2] It is one of the crippling diseases claiming the maximum loss of human power. It is not only a disorder of the locomotor system, but is also a systemic disease and is named after its chief pathogenic constituents, which are *Ama* and *Vata*.[3] Derangement of the *Kapha dosha*, especially *Shleshaka* kapha in the *Amavata*, which produces joint pain and swelling with tenderness, can be correlated with RA and derangement of the *Pitta dosha* along with *Ama* taking shelter in the *Avalambaka Kapha sthana*, which can be correlated with rheumatic fever because of the cardiac involvement, due to repeated fever, resulting in rheumatic heart diseases.[4] Humans are one of the most complex microbial ecosystems on the planet, hosting over 100 trillion bacteria, mainly in the distal gut. Some gut bacteria species can induce autoimmunity in genetically predisposed animal models. Dysbiosis of specific bacterial lineages and alterations in GM metabolism led to changes in the host immune profile that contribute to RA.[5] Notably, different strains of gut bacteria can have profoundly different regulatory effects on immune system function. Some strains can stimulate an immune response, benefiting immunocompromised patients, while others can suppress the immune response, affecting immune regulation in RA patients. It is widely recognized that the GM can affect almost all aspects of the host, and its dysregulation is

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associated with dysregulated immune tolerance and RA development. Indeed, changes in the GM can precede the onset of RA and are closely related to disease activity afterward. Analysis of GM composition can also predict susceptibility to RA, and has become a useful method to predict and control RA incidence.[6]

1.1. Aims and Objectives

a. To explore the interplay between GM and *Amavata* (RA) through Ayurvedic and modern scientific perspectives
b. To correlate the Ayurvedic concepts of *Agni*, *Ama*, and *Srothssodushti* with the modern understanding of gut dysbiosis and immune dysfunction
c. To highlight integrative approaches for the prevention and management of *Amavata* by linking Ayurvedic interventions with emerging microbiome-based therapies.

2. MATERIALS AND METHODS

A comprehensive literary search was done to collect the information related to *Agni*, *Amavata nidana, samprapthi, Pathya,* and *apathya* related to it from Classical Ayurvedic texts along with their commentaries. Furthermore, electronic databases were also searched using keywords, such as *Amavata,* RA, and GM.

2.1. *Amavata*

Improper diet plans and a flawed way of lifestyle bring a lot of disturbances in human life, both physically and psychologically. This leads to impairment of *Jatharagni,* which results in the genesis of *ama*. *Amavata* is a condition that occurs when the *Ama* and *Vatadosha* become vitiated at the same time and enter the *Trika Pradesh*a and *Sandhi* (joints), resulting in *stabdhata* (stiffness) of the body. Acharaya Madhavkara has clearly quoted the *roopas* (signs and symptoms) of *Amavata* in Madhava Nidana. The *pratyathma linga* (Cardinal symptoms) are: *Gatrastabdhatha*, *sandhishoola*, *sandhishotha, sparshasahatva,* and *samanya linga* (generalized symptoms) include: *Angmarda, aruchi, trishna, alashya, gaurava, jvara, apaka.*[6]

The root cause behind the development of *Amavata* is *Mandagni* (~reduced digestive power), which further leads to the formation of *Ama* (~indigested products) and it goes through several steps finally resulting in Amavata (Table 1). When this *Ama* gets lodged in *Sandhi* (~joints), it produces localized symptoms, such as pain, stiffness, and swelling. In classical texts, specific sequential management is described.[7] As per avastha of amavata its represents various sign and symptoms like in "Tivraavasta janya amavata" (acute condition) there are pain and inflammation in joints of hands, legs, ankles, sacrum, knees, thigh region; poor appetite, excessive salivation, anorexia, burning sensation, polyuria, pain and heaviness in body, changing in sleep pattern, vomiting, vertigo, stiffness in cardiac region and all over the body, constipation whereas in "Jirnaavasta janya amavata" (chronic condition) there are deformity in bony joints, dryness and stiffness in muscle and tendons, deformities in fingers etc.[8]

In the stage of *Ama*, any medicine should not be given as *Agni* is already in *Durbalaavastha* (~weak stage). While treating *Amavata*, the very first step is *Amapachana* (~digestion of undigested metabolic waste) and *Agnideepana*. *Langhana* (~depletion treatment) is the best modality for achieving *Amapachana* and it increases *Agni*. The purpose of *Langhana* is to make the body light and to clear the obstruction of channels due to increased *Kapha* and *Ama*.[7]

2.2. *Amavata* and RA

RA is a systemic autoimmune disease that predominantly affects the joints. The prevalence of RA varies globally, with generally a higher prevalence in industrialized countries, which may be explained by exposures to environmental risk factors, but also by genetic factors, differing demographics, and under-reporting in other parts of the world. Over the past three decades, strong trends of the declining severity of RA probably reflect changes in treatment paradigms and overall better management of the disease.[9] The worldwide prevalence of RA in the Global Burden of Disease 2010 Study is about 0.24%.[10] According to epidemiologic data, RA is more prevalent in women compared to men, with a lifetime risk of RA of 3.6% in women compared to 1.7% in men.[11] RA risk also increases with age, with a peak incidence between the age 65 and 80 years of age.[12] A systematic review of population-based studies (including 60 studies) showed a worldwide period prevalence of RA of 0.51% (1955–2015).[13]

Amavata has similarities to many arthritic disease conditions, but it most closely resembles RA, a long-term autoimmune inflammatory systemic condition that primarily affects synovial joints but can also cause extra-articular symptoms.[14] RA is a systemic disease that primarily causes joint inflammation, pain, loss of function, and eventual joint destruction and deformity. The disease is of variable severity, ranging from mild inflammation in a few joints to symmetric involvement in multiple joints, mainly in the hands and feet. One of the oldest records of the disease is a brief description in the *Rigveda*, which roughly dates back to 1500 B.C. In the 9th century A.D., Indian physician, Madhava wrote a full description of *Aamavata,*[15] but it was not until 1800 that the disease, described by French physician Augustin Jacob Landré-Beauvais, was recognized in the Western world. In 1859, British rheumatologist Alfred Baring Garrod, named the disease RA.[16]

The well-known pathogenesis of RA includes the production of autoantibodies, the mediation of immune cells, activation of inflammatory pathways, and proliferation of synovium. However, these have not offered enough support for us to seek a cure for RA. Whether these immunologic, epigenetic, and metabolic targets could be transformed into predictive factors or clinical interventions need more innovative study designs. Numerous therapeutic options make RA, a highly disabling disease; become controllable, especially biologic DMARDs and JAK inhibitors. Although inflammation was extinguished and tissue damage decelerated, drug-free remission is still far from reach in RA.[17]

2.3. Clinical manifestation: (*Amavata,* RA)

Generally, the disease begins with gradual pain, swelling, and stiffness of multiple joints in the hands and feet at the metacarpophalangeal, proximal interphalangeal, metatarsophalangeal, wrist, and ankle. Elbows, shoulders, knees, and temporomandibular joints are also commonly affected. Hip and lower back pain are less common. The neck joints are frequently affected in children. Morning stiffness lasting for 1 h or more is very common. There are also complaints of muscle pain, fatigue, low-grade fever, depression, weight loss, lack of appetite, and thirst in about one-third of patients. Less frequently, patients may experience monoarticular arthritis at the onset of the disease, with more joints affected later. In palindromic rheumatism, there is episodic pain and swelling of the joints lasting anywhere from a few hours to a few days, and may reoccur days, weeks, or months later. Aside from manifestations in the joints, muscles, and tendons, there are also extra-articular manifestations in the form of uveitis, scleritis, episcleritis,

rheumatoid nodules, anemia, pleural and pericardial effusions, sicca syndrome, splenomegaly, vasculitis, neuropathy, and renal disease. Moreover, there is an increased incidence of coronary artery disease in patients suffering from RA.[16]

2.4. GM and *Amavata*

In *Amavata*, the manifestation of systemic inflammation, joint stiffness, and immune dysregulation aligns with the pathological consequences of gut dysbiosis. Pro-inflammatory cytokines, such as tumor necrosis factor-α and interleukin-6, are elevated in both conditions, highlighting the common underlying mechanisms. Furthermore, the Ayurvedic emphasis on Agni correction and Ama digestion is supported by modern interventions targeting gut health, such as probiotics, prebiotics, and dietary modifications.[18]

It has been proposed that the mechanism by which GM imbalance leads to RA may be related to the regulation of immune function by metabolites produced by gut microbes. Immune T and B cells have position-specific phenotypes and functions in the mucosa, influenced by the microbiota.[5] In turn, bacterial peptidoglycan components are found in the synovial tissue of RA patients, which may contribute to inflammation within the microenvironment of the joint.[19,20] Notably, different strains of gut bacteria can have profoundly different regulatory effects on immune system function. Some strains can stimulate an immune response, benefiting immunocompromised patients, while others can suppress the immune response, affecting immune regulation in RA patients.[5] For example, segmented filamentous bacteria (SFB) have a unique ability to drive T helper 17 cell accumulation in the small intestine's lamina propria through SFB-derived antigens presented by dendritic cells.[21-23] In contrast, the colonization of *Bacteroides fragilis* is associated with enhanced activity of regulatory T cells, which may alleviate autoimmune disease.[24,25] Therefore, the relative abundance of different bacterial lineages may lead to changes in the host immune profile and drive inflammatory responses contributing to RA.

2.4.1. GM and Ama

Disruptions in gut flora (dysbiosis) lead to increased intestinal permeability, triggering the formation of inflammatory metabolites akin to Ama described in Ayurveda. Research suggests that microbial dysbiosis contributes to the development of autoimmune conditions by promoting systemic inflammation and immune dysregulation.

Linking Gut Dysbiosis and *Amavata* Pathophysiology:

a. Ama and leaky gut syndrome: Ama, formed due to impaired Agni (digestive fire), parallels the concept of endotoxins generated in leaky gut syndrome. These toxins enter the bloodstream, eliciting an inflammatory response akin to *Amavata* symptoms
b. Immune dysregulation: Dysbiosis disrupts the gut immune axis, leading to the activation of pro-inflammatory cytokines, similar to the pathogenesis of *Amavata* as described in Ayurveda
c. Vata aggravation: Modern research correlates gut dysbiosis with neural inflammation, resonating with the Vata vitiation described in *Amavata*'s progression.[18]

2.4.2. Pathya, Amavata, and GM

The equilibrium of GM plays a role in the homeostasis of host immune mechanisms. Hence, when there is any alteration in GM, the immune mechanism is vulnerable to inflammatory conditions, such as RA. Diet can thus play a major role in the modulation of GM in RA. The presence of Bowman–Birk inhibitor in *Kulattha* (horse gram); bioactive components, such as polyphenols and flavonoids in *Lashuna* (garlic); butyrate, and SCFA in *Yava* (barley) controls the etiopathogenesis of RA by restoring GM. *Shigru* (moringa), *Takra* (buttermilk), *Gomutra* (cow urine), and *Lashuna* (garlic) can inhibit inflammatory markers, such as IL-6 and IL-17. Thus, dietary regimen has a direct link with GM of RA, and especially the dietary regimen mentioned as *pathya* and *apathya* in the treatment of *Amavata* can be effective in the management of RA.[2]

3. DISCUSSION

The human microbiome is considered here as the collection of microbes, their genes, and their products that colonize our body since birth and are transferred vertically. While all body sites are colonized, the highest microbial numbers are found in the gut, which has been studied extensively.[26] Multiple factors contribute to the establishment of the human GM during infancy. Diet is considered as one of the main drivers in shaping the GM across the life time. Intestinal bacteria play a crucial role in maintaining immune and metabolic homeostasis and protecting against pathogens. Altered gut bacterial composition (dysbiosis) has been associated with the pathogenesis of many inflammatory diseases and infections.[27]

Amavata is a disease in which vitiation of *Vata Dosha* and accumulation of *Ama* take place in joint(s), and it simulates RA at modern parlance.[1] RA is a multifactorial autoimmune disease with a high disability rate. It is characterized by destructive and symmetrical joint diseases and synovitis, which seriously threaten human health. Recently, growing evidence demonstrates that GM plays an important role in RA. At present, the mechanisms of GM participating in RA pathogenesis mainly include regulating the differentiation of immune cells, inducing the production of inflammatory mediators, and molecular simulation. Disturbed GM can trigger the innate and adaptive immunity abnormally, which may lead to aberrant systemic immunity.[28]

The alternation of GM can be applied in the prediction and treatment of RA. Before the onset of RA, there are some presages about the high risk of the disease, such as rheumatoid factor (RF) and anti-citrullinated protein (anti-CCP).[29,30] The gut microbiome of Anti-CCP/RF-positive high-risk individuals without clinical synovitis differs significantly from that of healthy individuals. The study of the SCREEN-RA cohort also uncovered the gut microbiome as a risk factor for RA development.[31] This can help discover new biological markers of progression toward RA, which is beneficial to identify high-risk populations precisely.

Obviously, evidence is sufficient to prove that there is a correlation between GM and RA, but many details are unclear at present. Some bacteria have been reported to have significant differences between healthy and RA individuals, or affect immune function, or be related to other autoimmune diseases, but there is no specific report on RA, which is not much and deep-going.[32,33]

4. CONCLUSION

The Ayurvedic concept of *Ama* and the modern understanding of gut dysbiosis reflect two perspectives of the same pathophysiology in *Amavata*. Both emphasize the centrality of gut health in systemic inflammation and autoimmunity. Strengthening *Agni* and restoring microbial balance emerge as common therapeutic goals. In a word, this field has already established a solid foundation, and more efforts are needed to build it higher so as to seek new strategies for the treatment of *Amavata* (RA) patients.

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6. AUTHORS' CONTRIBUTIONS

All authors have contributed equally to conception, design, data collection, analysis, drafting, and final approval of the manuscript.

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8. ETHICAL APPROVALS

This study does not require ethical clearance as it is a review article.

9. CONFLICTS OF INTEREST

Nil.

10. DATA AVAILABILITY

This is an original manuscript and all data are available for only review purposes from the principal investigators.

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Table 1: *Samprapti chakra* of *Amavata*[8]

Stage	Event	Explanation
1.	*Agnimandya*	Weak digestive fire due to improper diet/lifestyle
2.	Formation of *Ama*	Formation of toxic material (Ama) due to indigestion
3.	*Srothorodha*	Blockage of body channels by *Ama*
4.	*Vata prakopa*	Aggravation of Vata due to obstruction
5.	*Samyoga of Vata* and *ama*	Vata carries Ama to joints
6.	*Sandhi Sthapana*	Localization of Ama and Vata in joints
7.	*Sheeta, Shoola, Sthambha*	Symptoms, such as stiffness, pain, and coldness appear
8.	*Upadrava*	Involvement of other doshas and tissues leading to complications.