



Amavata: Ayurvedic Insights and Its Correlation with Rheumatoid Arthritis

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Abstract

Introduction:

Amavata is a classical Ayurvedic disease characterized by the simultaneous aggravation of *Ama* (toxic, incompletely digested metabolic by-products) and *Vata*, primarily localizing in the joints and periarticular structures. Historically, its earliest systematic description is credited to Madhavakara in Madhava Nidana (9th century AD), although the conceptual foundations of *Ama* and joint disorders trace back to the Vedic and Samhita periods. Modern studies correlate *Amavata* with Rheumatoid Arthritis (RA) due to similar clinical features, pathogenesis, and chronicity.

Materials and Methods:

This review is based on a comprehensive analysis of classical Ayurvedic texts, including Madhava Nidana, Charaka Samhita, Sushruta Samhita, Harita Samhita, Bhela Samhita, Bhavaprakasha, and later medieval texts (Vagbhata, Chakradatta, Yogaratnakara), alongside modern clinical literature on RA. The historical evolution, etiopathogenesis, clinical features, classifications, and therapeutic approaches of *Amavata* were systematically reviewed. The pathophysiological correlation between *Ama* accumulation, *Vata* aggravation, and joint involvement was analyzed, along with modern correlations to autoimmune polyarthritis.

Results and Conclusion:

Amavata results from impaired digestive and metabolic functions (*Agnimandya*), leading to *Ama* formation, which, in association with aggravated *Vata*, localizes in *Kapha*-prone joints, producing pain, stiffness, swelling, and functional limitation. Classical treatment emphasizes *Ama pachana* (digestion of *Ama*), *Vata* pacification, *svedana* (sudation), *langhana* (fasting), *virechana* (therapeutic purgation), *sneha*

(oleation), and *basti* (medicated enema). *Pathya* (dietary and lifestyle modifications) complements pharmacological therapy. The disease closely resembles RA in clinical presentation, progression, and systemic manifestations. Understanding the classical Ayurvedic pathogenesis and correlating it with modern RA facilitates integrative management and guides individualized treatment strategies.

Keywords: *Amavata, Ama, Vata, Rheumatoid Arthritis, Samprapti, Sandhi* (joints), *Agnimandya*

Introduction

Historical Overview of *Amavata*

The earliest comprehensive and systematic description of *Amavata* as an independent disease is credited to Madhavakara in *Madhava Nidana* (900 AD), where it appears in the chapter *Amavata Nidana* (Chapter 25, Verses 1–12) (Madhavkara, 2016). Prior to this, the conceptual foundations of *Amavata* developed gradually across different historical periods—*Vedic* (Ancient era), *Samhita* (Classical Period), *Sangraha* (Medieval Period), and *Adhunika Kala* (Modern eras). During the Vedic period, although the term *Amavata* is not explicitly mentioned, the concept of *Ama* is evident in the *Rigveda* through terms like *Amayath* (*Rigveda*) and *Amayatha* (*Rigveda*), and in the *Atharvaveda* through *Amaya* and *Amayama* (*Atharva Veda*), which denote toxic or morbid states resembling the pathological nature of *Ama*. The *Atharvaveda* also describes the five fold classification of *Vata* (*Prana, Samana, Udana, Vyana, and Apana*)(*Atharvaveda*) and refers to joint disorders (*Sandhi Vikriti*) arising from vitiation of *Sleshma*, indicating an early understanding of the pathological interaction between *Doshas* and joints.

In the *Samhita* period, which represents the classical golden age of Ayurveda, the concept of *Ama* is extensively described, though *Amavata* is rarely recognized as a distinct clinical entity. Among the classical texts, the *Harita Samhita* uniquely provides an independent chapter on *Amavata*, detailing its etiology, symptomatology, pathogenesis, and treatment, although the authenticity of the current version of this text remains debated. (*Harita Samhita*) Charaka *Samhita* offers a highly detailed account of *Ama*, emphasizing its role as a major pathogenic factor, its similarity to poison (*Visha*) (*Charaka Samhita*, 2022), its clinical features, and its management. References to the term *Amavata* appear indirectly in therapeutic contexts such as the use of *Kamsa Haritaki* (*Charaka Samhita*, 2022) and *Vishaladi Phanta* (*Charaka Samhita*, 2022), and in discussions of *Vata* occlusion (*Avarana*) by *Ama* (*Charaka Samhita*, 2022). Furthermore, Charaka's description of *Amapradoshaja Vikaras* (*Charaka Samhita*, 2022) and the management principles of *Shariragata Ama* in *Grahani Chikitsa* (*Charaka Samhita*, 2022) closely parallel the later therapeutic approach to *Amavata*. Similarly, *Bhela Samhita* does not directly name *Amavata* but presents a full chapter on *Amavata* titled *Atha Ama Pradoshiya Adhyaya*, whose symptom complex and treatment strategies closely resemble the clinical picture of *Amavata* as understood later (*Bhela Samhita*, 2022).

During the Sangraha period, Vagbhata introduced the concept of *Samavata* (Ashtang Hridaya) in Ashtanga Hridaya, describing a pathological condition produced by the association of *Ama* and *Vata*, which bears a striking similarity to *Amavata*. Although he did not establish *Amavata* as a separate disease entity, the term appears in therapeutic contexts, particularly with reference to the use of formulations such as *Vyoshadi Yoga* (Ashtang Hridaya). The most significant milestone in the historical evolution of *Amavata* occurred with *Madhavakara*, who, in *Madhava Nidana*, Chapter-25, was the first to clearly and systematically establish *Amavata* as an independent disease. He provided a structured account of its etiological factors, detailed pathogenesis, clinical manifestations, subtypes, and prognosis, thereby laying the foundation for the classical understanding of *Amavata* that continues to guide Ayurvedic clinical practice and research today.

Table no.1 : Books of Sangraha Kala which include Amavata as a separate entity

Text	Chapter / Section
<i>Bhavaprakasha</i>	<i>Amavata Adhikara</i>
<i>Yogaratanakara</i>	<i>Amavata Adhikara</i>
<i>Rasaratna Samuchchaya</i>	<i>Vatavyadhi Chikitsanam, 21/47-50</i>
<i>Vrindamadhava</i>	<i>25th Chapter Amavata Adhikara</i>
<i>Gadanigraha</i>	<i>Part-II/22nd Chapter, Amavata Nidanam</i>
<i>Vrihadyogatarangani</i>	<i>Amavata 93</i>
<i>Vasvarajiyam</i>	<i>Ashiti Vatanidana Chikitsa (Sashath Prakarana)</i>
<i>Bhaishajya Ratnavali</i>	<i>29th Chapter, Amavata Chikitsa Prakaran</i>
<i>Chakradatta</i>	<i>25th Chapter, Amavata Chikitsa</i>
<i>Rasendra Chintamani</i>	<i>9th Chapter, Amavata Adhikara</i>
<i>Vrindhavaidyaka</i>	<i>Amavata Adhikara 29</i>
<i>Yogatarangani</i>	<i>Amavata Chikitsa 42</i>
<i>Vangasena Samhita</i>	<i>Amavata Adhikara</i>
<i>Rasendra Sara Samgrah</i>	<i>Amavata Chikitsa, Dwitiya Adhyaya</i>

In the modern period (*Adhunka Kala*), *Amavata* has been reinterpreted by several scholars in the context of evolving clinical understanding and contemporary disease classification. Kaviraj Gananath Sen (1943) made a notable contribution by classifying joint disorders into five distinct types and introducing a condition termed *Rasavata*, which he considered synonymous with *Amavata*. Through this concept, he emphasized the central role of improperly processed *Rasa Dhatu* in initiating the pathological process of the disease. He further described four varieties of *Manyastambha* in *Ayurveda Rahasya Deepika*, identifying one variant as a clinical manifestation of *Amavata*. Subsequently, scholars such as Prof. Y. N. Upadhyaya (1953) and others correlated *Amavata* with Rheumatoid Arthritis based on the close resemblance in symptomatology, disease course, and pathological characteristics. In summary,

while the concept of *Ama* gained medical importance during the Samhita period, it was *Madhavakara* who first established *Amavata* as an independent disease entity by recognizing its distinctive etiopathogenesis. In later periods, Chakradatta elaborated its therapeutic approaches, Bhavaprakasha enriched the clinical and pathological descriptions, and the concept attained comprehensive clarity in *Bhaishajya Ratnavali*. On the basis of clinical presentation and underlying pathological features, *Amavata* is now widely correlated with Rheumatoid Arthritis in modern medical science.

***Amavata*: Etymology and Definition**

In Ayurveda, diseases are named on the basis of the involvement of vitiated *Doshas*, affected *Dushyas*, nature of pain, site of manifestation, involved organs, Gati/Marga (pathway of disease spread), and distinctive clinical features. The term *Amavata* is derived from the union of two words: *Ama* and *Vata*, which together reflect the central pathogenic factors responsible for the disease (Madhavkara,2016). According to Shabdakalpa Druma, *Ama* is formed due to impaired digestive and metabolic processes, and when this toxic, improperly formed *Anna-Rasa* associates with vitiated *Vata*, the clinical entity of *Amavata* is produced (Shabdkalpadruma,2022). Siddhanta Nidana also support this view by explaining that incompletely processed nutritive fluid (*Anna-Rasa*) is termed *Ama*, which subsequently vitiates *Vata* and gives rise to the disease (Siddhanta Nidana).

Classically, *Amavata* is defined in *Madhava Nidana* as a condition in which there is stiffness of the body (*Gatra-stabdhata*) due to the simultaneous aggravation of pathogenic factors that localize in the *Trika* (lumbosacral region) and *Sandhi* (joints) (Madhavkara, 2016). The term *Yugapata* (simultaneously) in the definition has been interpreted differently by commentators. The *Madhukosha* commentary explains it as the simultaneous vitiation of *Vata* and *Kapha*, whereas the *Atankadarpana* commentary interprets it as the concurrent vitiation of *Ama* and *Vata*. These interpretations are not contradictory, as *Ama* is structurally and functionally similar to *Kapha*. However, an important distinction lies in the fact that *Kapha* becomes pathological only after vitiation, while *Ama* is inherently pathological from the moment of its formation.

Interpretation of *Trika* in Ayurvedic Literature

The concept of *Trika* has been variably interpreted by classical Ayurvedic scholars, reflecting anatomical and functional perspectives. Vachaspati Vaidya described *Trika* as the *Kati–Manya–Ansa Sandhi*, (Madhavkara,2016) emphasizing the shoulder girdle region. Arundatta interpreted *Trika* as *Prishthadhara*, relating it to the vertebral column(Ashtang Hridaya). Hemadri considered it the ilio-sacral and lumbo-sacral junction,(Ashtang Hridaya) highlighting the region of pelvic–spinal articulation. Dalhana, in his commentary on the Sushruta Samhita, described *Trika* as involving both hip joints and scapular articulations (Sushruta Samhita,2022). From a practical anatomical viewpoint, *Trika* may be understood as complex joints where more than two bones articulate, whereas the term *Sandhi* broadly denotes any structural junction between anatomical components.

Concept of *Ama* in Ayurveda

Ama is a pathological, toxic by-product of defective digestion and metabolism and is regarded as a fundamental factor in the initiation of many diseases. The significance of *Ama* is reflected in the term *Amaya*, which is often used synonymously with *Vyadhi* (disease). Assessment of the presence or absence of *Ama* is essential before initiating treatment, as the line of management differs markedly between *Samavastha* (presence of *Ama*) and *Niramavastha* (absence of *Ama*). In the context of disorders such as *Amavata*, *Ama* forms the basic pathological substrate responsible for symptom manifestation and disease progression.

Classical authors have defined *Ama* in multiple ways. Madhavakara described *Ama* as improperly formed *Adya Ahara Dhatu* (*Rasa Dhatu*) resulting from diminished *Kayagni* (Madhavkara, 2016). It is also explained as the digestive residue produced due to hypofunction of *Agni* and is regarded as the root cause of many diseases. Some Acharyas consider *Ama* to be *Apakva Annarasa*, while others describe it as *Mala* or the early vitiated state of *Doshas*, *Ama* is characterized as a foul-smelling, heavy, sticky, and partially digested substance that circulates throughout the body (Madhavkara, 2016). Vagbhata further described *Ama* as the undigested and vitiated *Adya Dhatu* produced due to hypoactivity of *Jatharagni* (Ashtang Hridaya, 2022).

Ayurveda recognizes thirteen types of *Agni*: one *Jatharagni* (central digestive fire), five *Bhutagni* (elemental metabolic fires), and seven *Dhatvagni* (tissue-specific metabolic fires). The process of digestion and metabolism occurs sequentially through these three levels, wherein *Jatharagni* initiates digestion in the gastrointestinal tract, *Bhutagni* carries out elemental transformations, and *Dhatvagni* governs tissue-level metabolism. When any of these metabolic fires become impaired, incomplete digestion and metabolism occur, leading to the formation and accumulation of *Ama*. Among these, dysfunction of *Jatharagni* is considered the primary and most significant factor.

Etiological Factors (*Nidana*) Responsible for *Ama* Formation

The primary cause of *Ama* is *Agnimandya* (weak digestive fire), and the factors that diminish *Agni* are considered etiological contributors. Dietary causes include excessive intake of food, consumption of heavy, cold, dry, and incompatible foods, fasting, and eating during indigestion (Charaka Samhita, 2022). Lifestyle factors such as suppression of natural urges, daytime sleep, nocturnal vigil, improper posture, excessive water intake, and incorrectly administered Panchakarma procedures further weaken *Agni*. Psychological factors like anger, grief, fear, jealousy, and anxiety contribute to *Agnidourbalya* through neuro-psychological pathways. Miscellaneous factors such as chronic illnesses, adverse climatic conditions, and improper living environments further predispose to *Ama* formation.

Samprapti (Pathogenesis) of Ama

Ama is primarily produced due to derangement in *Agni* at various levels. At the level of *Jatharagnimandya*, improper digestion results in the formation of *Ama* instead of normal *Rasa Dhatu*, (Ashtang Hridaya,2022) leading to manifestations such as *Chardi*(vomiting), *Atisara* (diarrhea), *Visuchika*, (Charaka Samhita,2022) *Grahani* (Charaka Samhita,2022), and even formation of *Amavisha* (Charaka Samhita,2022). At the level of *Bhutagni* impairment, the elemental components of food fail to transform appropriately, producing *Bhutagni-janya Ama* (Charaka Samhita,2022) *.Dhatvagnimandya* may arise either due to impairment of *Jatharagni* (Ashtang Hridaya,2022) or due to localized *Dhatu*-level dysfunction, as seen in disorders like *Medoroga* (Sushruta Samhita,2022). Additionally, accumulation of gross and subtle metabolic wastes (*Mala-sanchaya*) obstructs the channels (*Srotorodha*), further promoting *Ama* production.

In the early stage of disease development (*Sanchaya Avastha*), the *Doshas* begin to accumulate and remain in an immature, toxic state, which is equated with *Ama*. This stage, termed *Prathama Doshadushti*, signifies the initial pathological phase where *Doshas* are not yet fully aggravated but are qualitatively altered (Ashtang Hridaya,2022), (Sushruta Samhita,2022) ,This conceptual understanding explains why early intervention aimed at *Ama pachana* can prevent progression of disease.

When *Ama* undergoes fermentation or further pathological transformation, it becomes *Amavisha*, a highly toxic variant possessing poison-like properties. Management of *Amavisha* is particularly challenging because hot therapies tend to aggravate the *Visha* component, while cold therapies increase the *Ama* component (Charaka Samhita,2022). This dual nature makes *Amavisha* therapeutically complex and clinically severe. In the specific context of *Amavata*, *Ama* predominantly originates from *Jatharagnimandya* and later combines with vitiated *Vata* to localize in the joints.

Clinical Features of *Ama*

Ama manifests through a variety of systemic and local symptoms. These include obstruction of channels (*Srotorodha*), loss of strength (*Balabhramsha*), heaviness (*Gaurava*), impaired movement of *Vata* (*Anilamudhta*), lethargy (*Alasya*), indigestion (*Apakti*), excessive salivation (*Nishthiva*), constipation (*Malasanga*), anorexia (*Aruchi*), and fatigue (*Klama*). These symptoms serve as clinical indicators for the presence of *Ama* and guide the physician in selecting appropriate therapeutic measures.

Clinical Features of *Samavata (Amavata)*

In *Amavata*, the combination of *Ama* and aggravated *Vata* produces characteristic manifestations. These include abdominal pain, distension, borborygmi, impaired appetite, constipation, joint pain, pricking pain, stiffness, body ache, swelling, coldness, and heaviness. Additional symptoms include *Anaha* (obstruction), *Angamarda* (generalized pain), body ache, *Bhrama* (giddiness), *Agnimandya* (reduced digestive capacity), *Parshva-Prishtha-Kati-graha* (pain and stiffness in the flanks,

back and waist), *Sira-akunchnana* (constriction of vessels), and *Stambha* (marked rigidity) (Charaka Samhita,2022). Symptoms are typically aggravated by oleation therapy, during early morning hours, at night, and during cloudy weather, reflecting the influence of *Kapha* and *Ama* predominance (Ashtang Hridaya,2022).

Nidana of *Amavata*

Nidana serves as the essential initiating factor in the pathogenesis of *Amavata*. Acharya Madhavakara provided the first comprehensive description of the etiological factors of this disorder, and these causative factors have been consistently reiterated in classical texts such as Yogaratnakara and Bhaishajya Ratnavali. The *Nidana* primarily involve factors that induce *Agnimandya* and promote the simultaneous vitiation of *Vata* and accumulation of *Ama*, ultimately leading to localization of the pathological complex in the joints and periarticular tissues.

Table no. 2: *Nidana* of *Amavata* according to different classics

S.No.	<i>Nidana</i>	M.N.	H.S.	B.P.	V.S.	Y.R.
1	<i>Virudha Ahara</i>	+	-	+	+	+
2	<i>Virudha Chesta</i>	+	-	+	+	+
3	<i>Mandagni</i>	+	+	+	+	+
4	<i>Snigdha bhuktavato vyayama</i>	+	-	+	+	+
5	<i>Avyayama</i>	+	-	+	+	+
6	<i>Gurva ahara</i>	-	+	-	-	-
7	<i>Kandaska Sevana</i>	-	+	-	-	-
8	<i>Vyavaya</i>	-	+	-	-	-

*M.N: *Madhava Nidana*, H.S: *Harita Samhita*, B.P: *Bhavprakash*, Y.R: *Yogratnakar*

Acharya Charaka defines *Viruddha Ahara* as dietary substances and combinations, including certain medicines, that may temporarily pacify the *Doshas* but ultimately disturb their physiological balance. Eighteen varieties of such incompatible dietary practices are described in the Charaka Samhita (Charaka Samhita , 2022). Regular consumption of *Viruddha Ahara* results in the formation of *Ama* due to impaired digestion and metabolism. This *Ama* acts as the initial pathological factor and triggers the *Samprapti* (pathogenesis) of *Amavata* by obstructing channels and facilitating *Dosha–Dushya* interaction.

Viruddha Chesta refers to incompatible lifestyle practices that disturb *Dosha* balance. Activities like strenuous exercise immediately after consuming *Snigdha* (heavy, unctuous) food impair *Agni*, leading to *Agnimandya*, *Vata* vitiation, and *Ama* formation. This combination initiates the pathogenesis of *Amavata*.

Mandagni is considered the central pathological factor in the development of *Amavata*. As described by Vagbhata, impaired function of *Agni* is the root cause of a wide spectrum of diseases (Ashtang Hridaya, 2022).

Nischalata is habitual physical inactivity that weakens *Agni*, increases *Kapha*, and promotes *Ama* formation. When combined with *Vata* aggravation, it predisposes to the development of *Amavata*.

Excess *Snigdha* food weakens *Agni* and forms *Ama*, while immediate *Vyayama* causes joint vulnerability (*Khavaigunya*), together acting as an important *Nidana* for *Amavata*.

***Purvarupa* (Premonitory Symptoms of *Amavata*)**

Although *Purvarupa* of *Amavata* are not clearly described in classics, early signs like weakness (*Daurbalya*), chest heaviness (*Hrid-gaurava*), and body stiffness (*Gatra-stabdhata*) indicate developing *Agni* impairment and *Ama* accumulation.

***Rupa* (Fully Manifested Clinical Features)**

Rupa (*Vyaktavastha*) is the fully manifested stage of *Amavata* marked by clear symptoms, including pain, joint involvement, systemic features, and *Ama*-related signs, aiding clinical assessment and diagnosis.

Table no.3: *Rupa* of *Amavata* according to different classics

S.No.	<i>Rupa</i>	M.N. (Madhavkara,2016)	B.P. (Bhavmisra,2022)	Y. R. (Yogratna kara,2018)	H. S. (Harita Samhita,2022)
1.	<i>Angamarda</i>	+	+	+	-
2.	<i>Aruchi</i>	+	+	+	-
3.	<i>Alasya</i>	+	+	+	-
4.	<i>Apaka</i>	+	+	+	-
5.	<i>Angasunyata</i>	+	+	+	-
6.	<i>Agnisada</i>	+	+	+	-
7.	<i>Antrakunjana</i>	+	+	+	-
8.	<i>Anaha</i>	+	+	+	-
9.	<i>Amatisara</i>	-	-	-	+

10.	<i>Angvaikalya</i>	-	-	-	+
11.	<i>Bahumutrata</i>	+	+	+	-
12.	<i>Bhrama</i>	+	+	+	-
13.	<i>Chhardi</i>	+	+	+	-
14.	<i>Daurbalaya</i>	+	+	+	-
15.	<i>Daha</i>	+	+	+	-
16.	<i>Gatrastabdha ta</i>	+	+	+	-
17.	<i>Grahanidosh a</i>	-	-	+	-
18.	<i>Gaurava</i>	+	+	+	-
19.	<i>Hridgaurava</i>	+	+	+	-
20.	<i>Hridgraha</i>	+	+	+	-
21.	<i>Jadyata</i>	+	+	-	-
22.	<i>Jwara</i>	+	+	+	+
23.	<i>Kukshishula</i>	+	+	+	-
24.	<i>Kandu</i>	+	+	+	-
25.	<i>Kukshikathin ya</i>	+	+	+	-
26.	<i>Murchha</i>	+	+	-	-
27.	<i>Nidranasha</i>	-	-	-	-
28.	<i>Nidravipraya ya</i>	+	+	+	-
29.	<i>Praseka</i>	+	+	+	+
30.	<i>Sandhiruja</i>	+	+	+	+
31.	<i>Sandhoshoth a</i>	+	+	+	+
32.	<i>Sandhigraha</i>	+	+	+	-
33.	<i>Shirashula</i>	+	+	+	-
34.	<i>Staimitya</i>	+	+	+	-
35.	<i>Trishna</i>	+	+	+	+
36.	<i>Trikshula</i>	+	+	+	+
37.	<i>Utsahahani</i>	+	+	+	-
38.	<i>Vibandha</i>	+	+	+	-
39.	<i>Vairasya</i>	+	+	+	-

Samprapti (Pathogenesis) of Amavata:

Nidanas impair *Agni*, leading to *Ama* formation and *Dosha* vitiation, especially *Vata*. Aggravated *Vata* circulates *Ama*, which lodges in structurally weak joints (*Khavaigunya*), particularly *Kapha*-dominant *Sandhis*. This causes *Srotorodha* and progressive joint pathology, resulting in the manifestation of *Amavata*.

Samprapti Ghataka (Sharma AK, 2022)

Dosha	<i>Tridosha</i> involvement, with predominance of <i>Vata</i> and <i>Kapha</i>
Dushya	<i>Rasa, Rakta, Mamsa, Asthi, Snayu, Sandhi, Kandara</i>
Srotas	<i>Rasavaha</i>
Adhithana	<i>Sleshma-sthana (Sandhi)</i>
Udbhava-sthana	<i>Amasaya</i>
Roga-marga	<i>Madhyama</i>
Vyadhi-swabhava	<i>Asukari, Kastaprada</i>

Bheda (Classification of Amavata)

Amavata is classified in classical Ayurvedic texts based on *Dosha* predominance, severity, and specific clinical patterns. Based on the involvement of *Doshas*, *Madhava Nidana* describes seven types: *Vata-pradhana*, *Pitta-pradhana*, *Kapha-pradhana*, *Vata-Pitta-pradhana*, *Vata-Kapha-pradhana*, *Pitta-Kapha-pradhana*, and *Sannipatika* varieties (Madhavkara, 2016). According to severity, *Amavata* is broadly divided into two types, namely *Samanya Amavata* (mild or uncomplicated form) and *Pravridha Amavata* (advanced or severe form). Furthermore, *Harita Samhita* classifies *Amavata* into five distinct types based on clinical presentation: *Vistambhi Amavata*, *Gulmi Amavata*, *Snehi Amavata*, *Sarvangi Amavata*, and *Pakva Amavata* (*Harita Samhita*, 2022). This classification helps in understanding the clinical diversity of the disease and guides individualized therapeutic planning.

Although classical texts do not list separate complications for *Amavata*, the advanced (*Pravridha*) features are considered *Upadravas*. These include deformity (*Khanja*), contractures (*Sankocha*), and functional disability (*Angavaikalya*), reflecting the crippling nature of the disease.

Measures that pacify *Ama* and *Vata* such as *Ushna* therapies, *Tikta-Katu rasa*, *Deepana* drugs, light diet, warm lifestyle, and *Pathya Ahara* act as *Upashaya*. Cold, heavy, and unctuous foods, cold exposure, and rainy season aggravate symptoms and are considered *Anupashaya*.

Sadhyata-Asadhyata

Amavata is generally *Kricchra-Sadhyata* (difficult to cure). Single *Dosha* involvement with recent onset is *Sadhyata*, whereas multi-*Dosha* involvement, long duration, and multiple symptoms make it

difficult to manage. *Sannipatika Amavata* with generalized edema is considered particularly difficult to treat. (Madhavkara, 2016).

Treatment

The treatment principles for *Amavata* were first systematically described by *Chakrapani*. He recommends *Langhana*, *Svedana*, *Tikta*, *Deepana* and *Katu* drugs, followed by *Virechana*, *Snehapana* and *Anuvasana* with *Saindhavadi Taila*, as well as *Kshara Basti* for *Amavata* patients (Chakradatta,2022). *Bhavaprakasha* mentions *Ruksha Sveda* using *Valuka Pottali* and *Upanaha* without *Sneha* for managing *Amavata* (Bhavmisra,2022). *Yogaratriakara* gives a similar therapeutic outline as *Bhavaprakasha* (Yogaratriakara,2022). In *Amavata*, *Ama* and *Vata* are the main pathogenic factors with opposing qualities, so treatment requires caution. According to *Chakrapani*, therapy should prioritize *Ama Pachana*, then restoration of *Agni*, and finally regulation of *Vata*. *Langhana* is the first-line treatment in *Amavata*, especially *Upavasa*, to reduce and digest *Ama* and restore *Agni*. It should be stopped once *Nirama Vata* is achieved to avoid aggravating *Vata*.

Svedana induces sweating, relieves stiffness, heaviness and coldness (Charaka Samhita,2022). *Svedana Dravyas* possess *Ushna*, *Tikshna*, *Sara* or *Sthira*, *Snigdha* or *Ruksha*, *Drava*, *Sukshma* and *Guru* qualities (Charaka Samhita,2022). *Snigdha Sveda* increases symptoms in *Amavata* because *Snigdha* enhances *Ama*. Therefore, *Ruksha Sveda* is preferred, as its *Ushna Guna* digests *Ama* in affected joints and removes *Srotorodha*. Using warm water internally is also beneficial. It supports *Deepana*, *Pachana*, *Srotoshodhana*, *Jvaraghna*, *Balya*, *Ruchikara* and *Svedakara* actions (Charaka Samhita,2022). In chronic *Amavata* where *Rukshata* is predominant, *Snigdha Sveda* may be employed. *Charaka* advises that if vitiated *Vata* is located in a *Sleshma Sthana*, *Ruksha Sveda* should be followed by *Snigdha Sveda* (Charaka Samhita,2022).

Svedana is especially beneficial in *Stambha*, *Gaurava*, *Jadya*, *Sheeta* and *Shula*—key features of *Amavata*. *Atapa Sevana* and *Snana* with *Ushna Jala* processed with *Vataghna Dravyas* are also useful forms of *Svedana*. *Tikta* and *Katu rasa* possess *Laghu*, *Ushna* and *Tikshna* properties, making them highly effective for *Ama Pachana*. They also perform *Deepana* and *Pachana* functions. These qualities help in digesting *Ama*, restoring *Agni*, reducing excess *Kledaka Kapha*, and mobilizing *Doshas* from the *Shakhā* to the *Koshta*. Since *Tikta–Katu Dravyas* aggravate *Vata*, caution is needed. Therefore, selected drugs should be both *Tikta–Katu* in *rasa* and also *Vatahara*.

Virechana is a *Shodhana* therapy that expels *Doshas* through the *Adhomarga* (Charaka samhita,2022). *Doshas* often remain lodged in *Srotasas*, and without *Shodhana* they may spread again. Therefore, complete elimination is essential.

After *Langhana*, *Svedana*, *Deepana* and *Pachana*, *Doshas* reach *Nirama* state, and *Virechana* can be administered with appropriate drugs. Symptoms like *Anaha*, *Vibandha*, *Antrakunjana* and *Ushna Gati* of *Vata* improve significantly with *Virechana*.

Snehapana imparts *Snigdha*, *Vishyandana*, *Mriduta* and *Kledana* to the body. *Sneha* has *Drava*, *Sukshma*, *Sara*, *Snigdha*, *Pichhila*, *Guru*, *Sheeta*, *Manda* and *Mridu* qualities (Charaka Samhita,2022). Previous treatments may cause *Rukshata* and provoke *Vata*, potentially worsening the condition. *Snehapana* counteracts these effects. *Sneha* processed with *Ushna*, *Katu* and *Tikta Dravyas* is particularly effective for both *Ama* and *Vata*. Due to the chronicity of *Amavata*, severe *Dhatu-Kshaya* occurs, making *Snehapana* beneficial. *Samana Snehapana* supports Agni by softening food and aiding digestion (Charaka Samhita,2022). *Snehapana* is also indicated in *Asthi-Majjagata Vata* (Charaka Samhita,2022). Since *Asthi* and *Majja Dhatus* are significantly involved in *Amavata*, *Snehapana* proves especially helpful.

Among all Ayurveda therapies, *Basti* is considered the best treatment for vitiated *Vata*. Since *Vata* is a major pathogenic factor in *Amavata*, *Basti* is highly beneficial. As the disease becomes chronic, *Vata* increases further. Both *Anuvasana* and *Niruha (Asthapana) Basti* are indicated. *Anuvasana Basti* counteracts the *Rukshata* caused by *Deepana* and *Pachana*, helps control *Vata*, maintains Agni and nourishes tissues. *Asthapana Basti* eliminates *Doshas* mobilized into the *Koshta* and relieves *Anaha*, *Vibandha* and related symptoms.

Pathyapathya

Pathya:

Yava, *Kulattha*, *Shyamaka*, *Kodrava*, *Raktashali*, *Purana Shashti* and *Shali*, *Vastuka*, *Shigru* (Bhavmisra,2016).

Apathya:

Kshira, *Dadhi*, *Matsya*, *Guda*, *Upodika*, *Mashapishtaka*, *Dushtanira*, *Viruddhanna*, *Asatmya ahara*, *Anupa mamsa*, *Guru-abishyandi anna*, *Pichchhila anna*. (Yogratnakara,2022),(Bhavmisra,2022).

Clinical Features & Onset:

RA is a chronic, systemic autoimmune disease that primarily presents as symmetric polyarticular arthritis, often accompanied by constitutional symptoms like fatigue, malaise, muscle wasting, and low-grade fever. Onset can be acute, gradual, or insidious: over half of patients experience a slow, subtle onset, while about 10% present with rapidly progressive, severe symptoms. Early features include joint pain, swelling, and morning stiffness, initially affecting the hands, wrists, and feet, though any joint may be involved. While RA is typically symmetric, early asymmetry may occur.

Articular Involvement & Deformities:

Small Joints: MCP, PIP, MTP, and IP joints are affected first.

Large Joints: Wrists, elbows, knees, ankles, and later hips may be involved. Cervical spine involvement can cause instability and neurological compromise.

Hands & Wrists: Tenosynovitis, ulnar deviation, swan neck, boutonniere, mallet finger, Z-thumb deformities, and piano-key sign.

Elbows & Shoulders: Loss of extension and rotator cuff weakness in advanced disease.

Feet & Ankles: Synovitis causes forefoot widening, metatarsalgia, hallux valgus, hammer toes, flatfoot, and tarsal tunnel syndrome.

Knees: Synovial effusion, quadriceps wasting, Baker's cysts, instability, and valgus deformity.

Hips: Pain in groin, buttocks, or knee; flexion deformity; trochanteric bursitis; femoral head collapse in late stages.

Cervical Spine: Atlanto-axial synovitis may lead to subluxation, quadriparesis, or medullary compression.

Extra-Articular Manifestations:

RA is systemic, often associated with high rheumatoid factor levels, and can affect multiple organs:

Hematological: Anemia, lymphadenopathy

Pulmonary: Airway obstruction, pleural effusion, interstitial lung disease

Connective Tissue: Rheumatoid nodules, tenosynovitis

Cutaneous: Livedo reticularis, pyoderma gangrenosum, palmar erythema

Cardiac: Mitral regurgitation, pericarditis, reduced cardiac output

Vascular: Raynaud's, digital vasculitis, systemic vasculitis

Neurological: Entrapment neuropathies, cervical myelopathy, peripheral neuropathy

Ocular: Keratoconjunctivitis sicca, episcleritis, scleritis

Cricothyroid Joint: Dysphagia, hoarseness, stridor

Musculoskeletal: Myositis, osteoporosis, spinal fusion, subcutaneous nodules

Renal & Reticuloendothelial: Proteinuria, Felty's syndrome, Sjögren's syndrome, splenomegaly, amyloidosis

Patients may also experience fatigue, pain, depression, weight loss, constipation, and increased susceptibility to infections.

Diagnosis & Investigations:

RA diagnosis relies on clinical criteria (ACR 1997), requiring **≥4 of 7 features** (morning stiffness,

arthritis in ≥ 3 joint areas, hand joint involvement, symmetric arthritis, rheumatoid nodules, positive rheumatoid factor, radiographic changes).

Laboratory: Elevated ESR and CRP; RF positive in ~75%; ANA may be positive.

Radiology: Early – soft tissue swelling, joint space narrowing, juxta-articular osteoporosis; Late – cysts, erosions, bone hypertrophy, subluxation.

Special Tests: Synovial fluid analysis, biopsy, arthroscopy, ultrasound, CT, MRI, bone scans.

Management:

Goals: **pain relief, inflammation reduction, joint protection, functional preservation, and systemic control.**

Non-pharmacological: Patient education, physiotherapy, occupational therapy, lifestyle modifications.

Pharmacological:

NSAIDs for symptom relief.

Glucocorticoids for acute flares.

DMARDs (methotrexate, sulfasalazine, gold) to slow progression

Biologics (TNF- α inhibitors) for refractory disease

Immunosuppressants (azathioprine, cyclophosphamide) for severe cases

Surgery: Synovectomy, arthroplasty, arthrodesis, osteotomy for pain relief, deformity correction, and functional improvement.

Conclusion

Amavata represents a unique disease entity in Ayurveda where the pathological interaction between *Ama* and *Vata* leads to chronic joint and systemic involvement. Historically, its recognition evolved from conceptual discussions on *Ama* and *Vata* disorders to a clearly defined nosological entity by Madhavakara in Madhava Nidana. Classical descriptions provide detailed insight into etiological factors, pathogenesis, clinical features, classifications, and therapeutic principles. Modern correlations align *Amavata* with Rheumatoid Arthritis, emphasizing autoimmune and systemic aspects alongside articular manifestations. Integrating classical Ayurvedic management with modern understanding can optimize treatment outcomes, highlighting the importance of early intervention to digest *Ama*, pacify *Vata*, and restore metabolic balance, thereby preventing chronicity, joint deformities, and functional disability.

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