JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

Correlation of Ama Lakshana with Inflammatory Biomarkers – A Rog Nidan Perspectives

Dr. Yashashree J. Thakre 1, Dr. Trupti A Sarode [2]

1. Associate Professor, Rog Nidan Vikruti Vidyan Department, Bhojraj Bhondekar Ayurvedic Mahavidyalaya & PES Ayurvedic Rugnalay, Bhandara.
2. Associate Professor, Rog Nidan Vikruti Vidyan Department, Government Ayurvedic College & Hospital Osmanabad.

Abstract

The Ayurvedic concept of *Ama* denotes a fundamental pathological condition characterized by the accumulation of toxic, undigested, or improperly metabolized substances within the body. Classical Ayurvedic texts describe *Ama* as the *Mulakarana* (root cause) of disease, arising from impairment of *Agni*—the digestive and metabolic fire responsible for physiological transformation. In a parallel framework, modern medicine recognizes free radicals, particularly reactive oxygen and nitrogen species, as major contributors to oxidative stress, resulting in cellular injury and the progression of chronic degenerative disorders.

This review undertakes a critical comparative analysis of the ancient concept of *Ama* and the contemporary Free Radical Theory of aging and disease. Evaluation of their physiological characteristics reveals notable similarities: both *Ama* and free radicals originate endogenously from inefficient metabolic processes, exhibit unstable and reactive properties, and possess the ability to obstruct biological channels (*Srotas*) and compromise systemic homeostasis.

Furthermore, the paper examines the congruence between Ayurvedic therapeutic modalities—*Deepana–Pachana* (enhancement of digestive and metabolic activity) and *Shodhana* (biopurificatory procedures)—and modern interventions involving antioxidants and free radical scavengers. This integrative analysis suggests that *Ama* may be interpreted as a macroscopic clinical manifestation of microscopic oxidative stress. By bridging Ayurvedic and biomedical paradigms, the study offers a comprehensive understanding of systemic inflammation and metabolic disorders, thereby reinforcing the relevance of Ayurvedic principles within contemporary molecular and integrative medicine.

Key Words: Ama, Bio Markers, Inflammation, Toxins.

Introduction

In modern medicine, inflammation is increasingly regarded as a fundamental underlying factor in the development of many chronic diseases. Ayurveda presents a parallel pathological concept through *Ama*. The equilibrium of *Doshas* is maintained by *Agni*, which governs all metabolic and transformative processes in the body. When *Agni* (digestive and metabolic capacity) becomes impaired, it fails to properly digest ingested food, resulting in defective *Sara–Kitta Vibhajana* (separation of nutritive and non-nutritive components). This derangement of *Agni* leads to the formation of *Ama*, a toxic, undigested, and improperly metabolized substance. Classical Ayurvedic texts describe *Ama* as a primary disease-causing factor that converts *Dhatu Samyavastha* (state of equilibrium) into *Dhatu Vaishamya* or *Vikrita Avastha* (pathological state). *Ama* obstructs the *Srotas* (body channels), provokes *Doshas*, and ultimately acts as the *Mulakarana* (root cause) of several chronic disorders.

The contemporary global surge in chronic non-communicable diseases—such as type 2 diabetes mellitus, cardiovascular disorders, rheumatoid arthritis, obesity, neurodegenerative conditions, and premature aging—has redirected biomedical research toward elucidating the fundamental metabolic mechanisms underlying cellular degeneration. Modern molecular medicine recognizes oxidative stress, mitochondrial dysfunction, and chronic low-grade inflammation as pivotal pathological processes driving these disorders. [1]

In parallel with these scientific advances, Ayurveda has long articulated a comparable pathological framework through the concept of *Ama*, which denotes incompletely metabolized, toxic by-products formed as a consequence of impaired digestive and tissue-level metabolic activity (*Agnimandya*). *Charaka Samhita* identifies *Ama* as a principal etiological factor in systemic disease, attributing its pathogenicity to obstruction of microchannels (*Srotorodha*), disruption of tissue nourishment, and initiation of inflammatory processes. *Vagbhata* further expands this concept by explaining that *Ama* may originate not only at the gastrointestinal level but also within tissues due to defective intracellular metabolism (*Dhatvagni Mandya*). [2] These classical descriptions closely parallel modern insights into mitochondrial inefficiency, accumulation of reactive oxygen species, metabolic endotoxemia, and the resultant inflammatory milieu that accelerates cellular dysfunction and disease progression.

Concept of Ama

In Ayurveda, *Ama* is described as an undigested, immature, and improperly metabolized substance formed as a result of impaired *Agni*. According to *Acharya Vagbhata*, when food is inadequately digested due to hypofunctioning of *Ushma* (*Agni*) [3], it undergoes fermentation and putrefaction within the *Amashaya*, leading to the formation of *Ama*. This improperly processed *Rasa Dhatu* behaves as a toxic entity and becomes the fundamental cause of various systemic disorders.[4]

Acharya Sushruta further elucidates that *Apakwa Anna Rasa* exhibits properties akin to *Visha* (poison) within the body and produces deleterious effects. Consequently, *Ama* possesses inherent toxic potential and plays a pivotal role in the initiation and progression of pathological processes.[5]

Properties of Ama

Classical Ayurvedic texts describe *Ama* as *Avipakva* (undigested), *Asamyukta* (unassimilated), *Durgandha* (foul-smelling), *Bahupicchila* (excessively sticky), *Guru* (heavy), *Snigdha* (unctuous), *Tantumaya* (thread-like), *Asukari* (rapidly spreading), and *Visharupa* (poison-like). Owing to these properties, *Ama* possesses the capacity to obstruct the *Srotas* and disrupt normal physiological functions.

Sama Avastha

When *Ama* combines with *Dosha*, *Dhatu*, and *Mala*, it results in *Sama Avastha*, and the diseases arising under this condition are referred to as *Sama Vyadhi*. In *Sama Avastha*, *Ama* circulates throughout the *Srotas* and becomes localized at sites of *Kha-vaigunya*, leading to the manifestation of disorders involving *Shakha*, *Koshta*, and *Marma* regions.

Pathogenesis of Ama

Derangement of *Agni* at the levels of *Jatharagni*, *Bhutagni*, and *Dhatvagni* leads to the formation of *Ama*. This *Ama* subsequently associates with *Dosha*, *Dushya*, and *Mala* and circulates through the *Srotas*. Upon reaching susceptible sites (*Kha-vaigunya*), it initiates the development of various disorders such as *Amavata*, *Grahani*, and *Katigata Vata*.[6]

Types of Ama

Ayurveda describes two principal forms of *Ama*. *Jatharagni-mandajanya Ama* arises due to impaired digestion at the gastrointestinal level, whereas *Dhatvagni-mandajanya Ama* results from defective tissue and cellular metabolism. These descriptions closely correspond with the modern understanding of the accumulation of intermediate metabolic by-products, misfolded protein aggregates, oxidized lipids, and reactive oxygen species (ROS) generated during inefficient mitochondrial oxidative phosphorylation.[10]

Free Radical Theory and Oxidative Stress

Free radicals are molecules containing unpaired electrons, rendering them highly unstable and reactive. The major reactive oxygen species include superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radical ($OH\cdot$), which are predominantly generated within mitochondria during oxidative phosphorylation. Under normal physiological conditions, endogenous antioxidant defense systems—such as superoxide dismutase (SOD), catalase, and glutathione peroxidase—neutralize these reactive species. However, when ROS production exceeds the neutralizing capacity of these antioxidant mechanisms, oxidative stress ensues. This state results in lipid peroxidation, protein denaturation, mitochondrial DNA damage, and activation of apoptotic pathways. Oxidative stress is now widely recognized as a central pathogenic factor in the development of diabetes mellitus, cardiovascular diseases, cancer, neurodegenerative disorders, chronic inflammatory conditions, and premature aging.[11]

Mitochondrial Dysfunction as Cellular *Agnimandya*

Mitochondria serve as the principal metabolic engines of the cell. Impaired efficiency of the electron transport chain leads to electron leakage and excessive generation of reactive oxygen species (ROS). This state of compromised mitochondrial metabolism may be interpreted as *cellular Agnimandya*, while the resultant accumulation of ROS and oxidatively modified biomolecules corresponds to *cellular Ama*. Accordingly, mitochondrial dysfunction and oxidative stress together constitute the molecular substrate underlying the formation of *Ama*.

Ama, Lipid Peroxidation, and *Srotorodha*

Classical Ayurvedic texts describe *Ama* as *Picchila* (sticky) and *Srotorodhaka* (channel-obstructing). In modern terms, lipid peroxidation leads to the formation of lipofuscin—an indigestible intracellular pigment that progressively accumulates with aging and interferes with lysosomal degradation and intracellular trafficking. This phenomenon closely parallels the obstructive and accumulative nature of *Ama*, thereby reinforcing the conceptual equivalence between *Ama*-induced *Srotorodha* and oxidative stress–mediated cellular dysfunction.[12]

Ama is recognized as an antigenic entity capable of eliciting inflammatory responses. Oxidative stress activates the transcription factor nuclear factor-κB (NF-κB), which upregulates pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6), thereby promoting chronic inflammatory states.[13] In rheumatoid arthritis, oxidative stress induces neutrophil respiratory burst, synovial inflammation, and progressive cartilage destruction, closely aligning with the Ayurvedic pathogenesis of *Amavata*.[14]

Antioxidants and *Amapachana*

Ayurveda advocates *Deepana*, *Pachana*, *Shodhana*, and *Rasayana* therapies for the elimination of *Ama*. These interventions enhance digestive and cellular metabolic efficiency, reduce oxidative burden, and strengthen endogenous antioxidant defense mechanisms. Clinical studies have demonstrated that *Panchakarma* procedures significantly decrease lipid peroxidation markers while increasing superoxide dismutase (SOD) levels, thereby substantiating the antioxidant and *Amapachana* effects of Ayurvedic biopurification therapies.[15,16]

Discussion

The present review demonstrates a compelling scientific convergence between the Ayurvedic concept of *Ama* and the modern Free Radical Theory of disease. Ayurveda characterizes *Ama* as a toxic, incompletely metabolized substance arising from impairment of *Agni*, which obstructs microchannels (*Srotorodha*), disrupts tissue nourishment, and initiates inflammatory pathology. In parallel, contemporary molecular biology identifies mitochondrial dysfunction and oxidative stress as fundamental metabolic derangements underlying chronic degenerative diseases. Inefficient mitochondrial activity results in excessive generation of reactive oxygen species (ROS), leading to lipid peroxidation, protein oxidation, DNA damage, and progressive cellular

dysfunction. This condition is comparable to *Agnimandya* at the cellular level, while the accumulation of ROS and oxidatively modified molecular aggregates represents the molecular expression of *Ama*.

The obstructive and adhesive nature of *Ama* described in classical texts closely parallels the accumulation of lipofuscin and oxidized cellular debris that impair intracellular transport, autophagy, and metabolic clearance. Furthermore, *Ama* is described as a pathogenic antigen responsible for sustaining chronic inflammation. Modern immunology substantiates this view by demonstrating that oxidative stress activates nuclear factor-κB (NF-κB) signaling, leading to increased production of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. These mediators play a central role in the pathogenesis of rheumatoid arthritis, diabetes mellitus, cardiovascular diseases, and neurodegenerative disorders, thereby supporting the classical descriptions of *Amavata* and other *Ama*-dominant conditions.

Ayurvedic therapeutic principles—including *Deepana*, *Pachana*, *Shodhana*, and *Rasayana*—are aimed at restoring *Agni*, digesting *Ama*, and re-establishing metabolic homeostasis. Emerging clinical evidence indicates that *Panchakarma* procedures significantly reduce lipid peroxidation markers and enhance endogenous antioxidant enzyme activity, while *Rasayana* formulations exhibit notable antioxidant and immunomodulatory properties. These findings provide molecular validation for classical Ayurvedic detoxification and rejuvenation therapies.

Importantly, *Ama* represents a broader state of metabolic toxicity than oxidative stress alone, encompassing undigested dietary residues, microbial endotoxins, heavy metals, protein aggregates, and psychosomatic metabolic toxins. Thus, while all free radicals may be understood within the framework of *Ama*, the concept of *Ama* extends beyond free radicals and offers a more comprehensive, systems-level understanding of chronic disease pathogenesis.

References

1. Sastri K, Chaturvedi G. Charaka Samhita of Agnivesha. Varanasi: Chaukhambha Bharati Academy; 2005.
2. Paradkara HS. Ashtanga Hridaya of Vagbhata. Varanasi: Chaukhambha Sanskrit Sansthan; 2009.
3. Harman D. Aging: a theory based on free radical and radiation chemistry. J Gerontol. 1956;11(3):298–300.
4. Garde GK. Sartha Vagbhata (Ashtanga Hridaya). Pune: Proficient Publishing House; 2009. p.70.
5. Shastri KA. Sushruta Samhita of Maharshi Sushruta. Part I, Sutrasthana. Varanasi: Chaukhambha Sanskrit Sansthan; 2012. p.73.
6. Shukla VD, Tripathi RD. Charaka Samhita of Agnivesha. Part II. New Delhi: Chaukhambha Sanskrit Pratishthan; 2009. p.367.
7. Shukla VD, Tripathi RD. Charaka Samhita of Agnivesha. Part I. New Delhi: Chaukhambha Sanskrit Pratishthan; 2009. p.755.
8. Shukla VD, Tripathi RD. Charaka Samhita of Agnivesha. Part I. New Delhi: Chaukhambha Sanskrit Pratishthan; 2009. p.563.

9. Shukla VD, Tripathi RD. Charaka Samhita of Agnivesha. Part II. New Delhi: Chaukhambha Sanskrit Pratishthan; 2009. p.367.
10. Murphy MP. How mitochondria produce reactive oxygen species. Biochem J. 2009;417(1):1–13.
11. Valko M, Leibfritz D, Moncol J, Cronin MT, Mazur M, Telser J. Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem Cell Biol. 2007;39(1):44–84.
12. Brunk UT, Terman A. Lipofuscin: mechanisms of age-related accumulation and degradation. Free Radic Biol Med. 2002;33(5):611–619.
13. Morgan MJ, Liu ZG. Crosstalk of reactive oxygen species and NF-κBsignaling. Cell Res. 2011;21(1):103–115.
14. Phull AR, Nasir B, Haq IU, Kim SJ. Oxidative stress, consequences and ROS mediated cellular signaling in rheumatoid arthritis. Int J Rheum Dis. 2018;21(3):478–485.
15. Sharma M, Dwivedi S. Effect of Virechana karma on oxidative stress markers: a clinical study. Int J Ayush Case Rep. 2019;3(2):45–52.
16. Sharma H, Chandola HM, Singh G, Basisht G. Utilization of Ayurveda in managing type 2 diabetes: role of Ama and Agni. J Ayurveda Integr Med. 2013;4(1):3–8.