Chapter 8
Gut and Joint Interconnections: A Reappraisal to Ayurvedic Understanding of Joint Diseases

Sanjeev Rastogi and Ranjana Rastogi

8.1 Introduction

Ayurveda, in true representation to disease prevalence pattern among human population, is marked by vivid elaboration to various common clinical entities. Infective conditions predominantly marked by fever and noninfective degenerative and metabolic conditions like diabetes, anemia, jaundice, and joint diseases are among few conditions described in Ayurveda in great details. Among them all, joint diseases make a proportionate sum to the net patient input to any Ayurvedic clinic seen in India [1, 2]. Among joint conditions commonly seen in Ayurvedic clinics, predominant ones are the diseases marked by joint pain, inflammation, and limitation to movements. Patients having joint pathologies visiting Ayurvedic outdoors are invariably found to have few common features. They are chronically disabled, are prediagnosed, and are marked with deformities, depression, and dependence [3, 4]. It is less common to find a newly diagnosed patient with joint disease in an Ayurvedic clinic. Ayurveda works admirably in joint afflictions; this layman's belief becomes an observable fact when we see a large number of these patients waiting for their turn of consultation or panch-karma therapy in an Ayurvedic setting [5]. Although an unavailability of dependable and safe remedy to many of these conditions through conventional medicine and a countrywide deficit of trained rheumatologists for their early admiration substantiates to escalating number of joint patients in Ayurvedic outdoors, it does not reduce the prima facie impression of effectiveness of Ayurvedic therapy in such conditions.

S. Rastogi (✉)
PG Department of Kaya Chikitsa, State Ayurvedic College, Lucknow University, Lucknow, Uttar Pradesh, India
e-mail: rastogisanjeev@rediffmail.com

R. Rastogi
Department of Obstetrics and Gynaecology, Vatsala Hospital, Lucknow, Uttar Pradesh, India
e-mail: rastogiranjana2010@rediffmail.com

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What should we expect from Ayurveda in a joint condition where the contemporary system of medicine is away from reaching to a dependable and reasonable management plan and where a patient is marked with lost hopes and fortunes of being recovered [3]. For obvious reasons of complexities associated with joint diseases, the expectations from Ayurveda for intervention in such conditions should be made balanced plausibly and pragmatically. Any plausibility of Ayurvedic interventions to act upon joint diseases lies onto its unique propatient understanding of a disease and its original gut–joint pathology theory, based on which Ayurveda holds promises of further exploration of its principles especially in the segment of joint pathologies to complement the existing management protocols within various joint diseases.

Food, its type, digestion process, and postdigestion physiology have remained the key aspects to an Ayurvedic understanding of health and subsequent pathogenesis. Besides their role to many other diseases, they are found to have a special bearing in joint diseases. Ayurveda groups inflammatory diseases of joint under the umbrella term of Amavata and proposes diet, digestion, and postdigestive mechanisms for being major incriminators to its pathogenesis. An association of diet with arthritis has remained a subject of debate since long. We have come across various studies where exacerbation of arthritis symptoms was observed by certain inclusion diets and similarly a remission was observed through exclusion diets [6–9]. Due to a huge variability in food consumption pattern across the globe, we however could not yet reach to a consensus for what are the foods that may lead to arthritis. This is, however, more or less agreed that a small component of all arthritis may get worsened and relieved by some diets [10]. There have also been the studies identifying an increased intestinal permeability in reference to excessive use of NSAIDs or otherwise as a cause to increased immunological gut responses in reference to various dietary antigens [11].

Ayurvedic understanding of diet in relation to joint pathology goes deeper than identifying type of food for arthritis symptom exacerbations. It actually points out to certain mechanism through which a normal food can also be converted as a precursor to various pathologies. Incidentally, a good account of lifestyle diseases including those afflicting the joints are found to have evidence-based links to trivial dietary faults existing for a considerable period of time.

Unfortunately, mere proposal of a hypothesis to understand a disease process may not really help to the actual disease management. In light of theoretical proposals and observations referring to clinical practice of Ayurvedic rheumatology, Ayurveda is required to realize more honestly what is still undone on their part of joint disease understanding and management. To be more honest, it requires paving ways to translate its founding principles of joint disease in galore to treat them more effectively. Ayurveda requires realizing the expectations of the patients who are making most of its clientele and requires finding ways to come true to those expectations. Needless to say, a thorough research with a clear vision of translating its conceptual excellence into utilizable repertory is the only way forward.

Contemporary researches to explore the rheumatologic conditions in Ayurveda have so far been diversified. By and large these researches are found limited to the exploration of possible clinical utility of herbal or herbomineral compounds alone or in addition to panch-karma procedures in various joint conditions [12]. Failing to find any admirable result through these clinical researches, of late, Ayurvedic rheumatology research has shifted from individual treatments to the development of a complete management plan, often a cocktail regimen, desirous to give some impact upon patients by adopting variable combination of a drug in oral or local forms, a diet, and a physico-biological procedure aptly called as panch-karma. Whatever idea of research is adopted so far to explore the subject of Ayurvedic rheumatology, it failed to contribute, and therefore despite of a huge patient input, it could not pave ways to the development of a dependable understanding and subsequent treatment protocols in Ayurvedic rheumatology. Forthcoming sections of this chapter are in realization to this important missing aspect of Ayurvedic rheumatology.

Considering the fundamentals of Ayurvedic pathogenesis, a disease may find its origin through multilevel imbalances to human physiology. These imbalances could be diversified, affecting various physiological functions like metabolism, digestion, absorption, circulation, synthesis, and elimination. *Agni* (fire of transformation) irregularity coupled with irregular functions of three founding physiological principles, namely, *vata*, *pitta*, and *kapha*, mark the basis of any pathogenesis in Ayurveda. Further to it, Ayurveda also identifies a few exclusive *hetu* or *nidana* (etiological factors) for most diseases. Diet forms an exclusive component to these etiological elaborations in Ayurveda.

Planning a dependable management in a disease condition thereby largely depends upon the understanding of its etiopathogenesis. It is therefore mandatory to reappraise Ayurvedic pathophysiological understanding to various rheumatologic conditions to design a definitive treatment protocol malleable as per the specific causes. *Amavata*, a syndromic entity in Ayurveda, is found to have reasonable resemblances with various inflammatory arthritic conditions [13]. Being one important representative to the subject of rheumatology, in terms of its prognosis and also in terms of its prevalence [5], *Amavata* is largely taken up as a clinical prototype in Ayurvedic rheumatology. A thorough reappraisal to its etiopathological understanding, therefore, is presumed essential to tailor an effective plan of its management and also to various other joint conditions where a similar etiopathogenesis is presumably involved.

8.2 *Amavata*: An Interplay of *Ama* and *Vata*

Amavata, an Ayurvedic parallel to rheumatologic syndromes, does not have a mention in the triad of ancient Ayurvedic classics (*Charaka Samhita*, *Sushruta Samhita*, *and Ashtanga Samgraha*). It finds its first ever mention as a syndrome in *Madhava Nidana* (sixth century AD), a text dedicated to Ayurvedic clinical diagnostics [14]. Thereafter, however, *Amavata* could find a regular mention to Ayurvedic texts and has become an indispensible part of Ayurvedic clinical medicine study and practice. This is interesting to note here about the apparent absence of this disease in particular from classical triads and its relative late entry into the Ayurvedic curriculum. Does it point out to some specific emergence–prevalence–incidence pattern to the disease which may have a relation to changes in local dietary, social, or environmental customs? Defining *Amavata*, *Madhava* explained it as a disease characterized by simultaneous vitiation of *ama* and *vata*, a duo of pathological entities, vitiating independently but in unison leading to a pathology complex enough to be explained through either of them alone.

8.2.1 Ayurvedic Understanding of a Disease Pathogenesis

Ayurveda proposes a novel mechanism to the beginning of a disease process in any individual. It identifies the same factors responsible for a disease process whom otherwise are responsible for the preservation of health during their normal occurrence and functioning. This proposal has a beautiful resemblance to the concept of eco-balancing where a definitive sum of one entity is found responsible for a sustainable eco-health and failing to which a population is presumed to suffer from a cascade of anomalies related directly or indirectly to the primary imbalance [15].

8.2.2 Etiology of Amavata

Etiological description of *Amavata* in Ayurveda renders a divergent spectrum of factors presumably leading to its origin. Dietary factors, metabolic status, physical activities done prior or immediately after the food intake, and average physical activities in general are principal components of this spectrum. An etiology to Amavata as depicted by *Madhava* clearly identifies two distinct roots to it. These are:

1. *Amavata* in metabolically and physically less active people who are also engaged in incompatible dietary practices. *Madhava* describes this as "*Viruddhaaharacheshtasya mandagnernischalasya cha*," a condition marked by a preexisting *mandagni* and hypoactive physical state clubbed with an eagerness to go for *viruddh-ahara*. This description of *Madhava* tries to differentiate one set of etiology from the other where a different set of etiology is found responsible to *Amavata*.
2. *Amavata* in physically and metabolically active people who are also having a practice of indulging into physical activities just after having heavy fatty meals. *Madhava* defines this state as "*Snigdhama bhuktavato hyannam vyayamam kurvatastatha*."

This set of etiology proposes the involvement of people with a good metabolic status and appetite and also with much agility. An exercise session following a heavy fatty meal which is presumed to be the reason of *Amavata* in these cases, incidentally, reminds us to the stressful urban working environments where one is constantly under substantial work stress and, as a consequence, is bound to rush to work even immediately after meals.

We can find that Ayurveda, through its etiopathogenesis, explores the possibilities of heterogeneous pathologies to the syndrome of *Amavata*. Any such possibility further gives us a clue as to why the presentation, responses to the treatment, and ultimate prognosis in *Amavata* are not identical in any two apparently similar cases. This divergent etiology of *Amavata* also explains about the heterogeneous spectrum of rheumatologic disease profile and their afflicting population in general.

8.2.3 *Ama*: *The Primary Root to Rheumatologic Diseases*

Ama is a novel concept of Ayurveda which adequately elaborates various rheumatologic pathogenesis marked by features of stiffness, heaviness, fever, swelling, and pain. *Ama* literally stands for something which is unripe. From physiological perspectives, production of *ama* refers to the under-operative state of *agni* leading to liberation of inadequately processed end products. These end products, being inadequately processed, have a macromolecular structure, which is bigger and grosser than those end products which might have produced if a complete processing would have taken place. *Ama*, thereby, owing to its macromolecular structure, in general, is told to have a clogging property with a predilection to block the *srotas* (fine conduits of the body), an important step through which most *ama*-related symptoms begin manifesting.

As *ama* is the produce of hypometabolic state, adequately referred as *mandagni* in Ayurveda, it can have multiple forms, depending upon the level of its origin. It can be produced in gastrointestinal tract where an inadequate digestion may give rise to production of macromolecular substances largely antigenic in nature for their under-processing into simpler forms. It can also be generated at tissue level where substrates are exposed to enzymatic actions for their conversion into ultimately utilizable forms in tissues. An under-processing of substrates at tissue level is proposed to give rise to *ama* at tissue level which is also referred to as *dhatu gata ama*.

8.2.3.1 Causes of *Ama* Production

Ayurveda identifies various dietary irregularities as the primary reason to *ama* production and subsequently to many *ama*-related diseases. Among all, an excessive intake of food is found to be the principal cause to such pathology (*Atimatram punah sarvadoshaprakopanama* – *Charaka Vimana* 2/7). An occasional excessive intake, however, produces transient symptoms only which are local to gastrointestinal tract and may not have a sustainable systemic effect required for the production of joint diseases.

Table 8.1 Food and its intake-related factors which may cause *Ama* production

Food quality–related factors	*Guru* (difficult to digest)
	Ruksha (dry mainly due to lack of fat)
	Sheet (cold)
	Shushka (dry mainly due to lack of hydration)
Food appearance–related factors	*Dwishta* (which is not liked in appearance)
Food effect–related factors	*Vishtambhi* (causing gastric discomfort)
	Vidahi (causing burning in abdomen)
Food preparation–related factors	*Ashuchi* (unclean)
Food composition–related factors	*Viruddha ahara* (incompatible food)
Food intake–related factors	*Akal* (food intake at improper time)
Psychological status at the time of food intake	*Kama* (extremely desirous)
	Krodha (angry), *Lobha* (lust), *Moha* (attachment), *Irshya* (jealous)
	Lajja (shameful), *Shoka* (grief)
	Abhimana (proud)
	Udvega (anxious)
	Bhaya (fearful)
	Up-*tapta* (sad)

Explaining further about the causes of *ama* production, *Charaka* identifies various other diet and its intake-related factors ultimate to *ama* production. Principal ones among them are shown in Table 8.1.

By observing the causes of *ama* production, it is apparent that besides quality of the food, an intake method and mood status at the time of food intake can also play a great role in determination of ultimate outcome to the consumed food. Effects of various psychological factors, especially the stress upon intestinal functioning, have increasingly been recognized [16]. A sustained stress is found to give rise to observable microscopic changes into gut epithelium and to its subsequent physiology. In many experimental studies, various types of physical and psychological stress are found to induce dysfunction of intestinal barrier, resulting in enhanced intake of potentially noxious material (e.g., antigens, toxins, and other proinflammatory molecule) from the gut lumen into the bloodstream [17]. This gives a very interesting proposition to see if these noxious materials play the role of ama as a progenitor to joint diseases as perceived in Ayurveda.

8.2.3.2 *Mandagni*: The Hypodigestive State

Mandagni is another Ayurvedic conceptualization for the possible reasoning to *ama* production. *Agni* is proposed to be the principal factor responsible for transformations in the body. As per its site of action, it is variously named as *Jatharagni* (transforming factor in gut), *Bhutagni* (transforming factor in substrates while they are in transition), and *Dhatvagni* (transforming factor in tissues responsible for active utilization of substrates to build up tissue). This is interesting to elaborate upon what *mandagni* is and how this may lead to production of *ama*. As we are aware, to

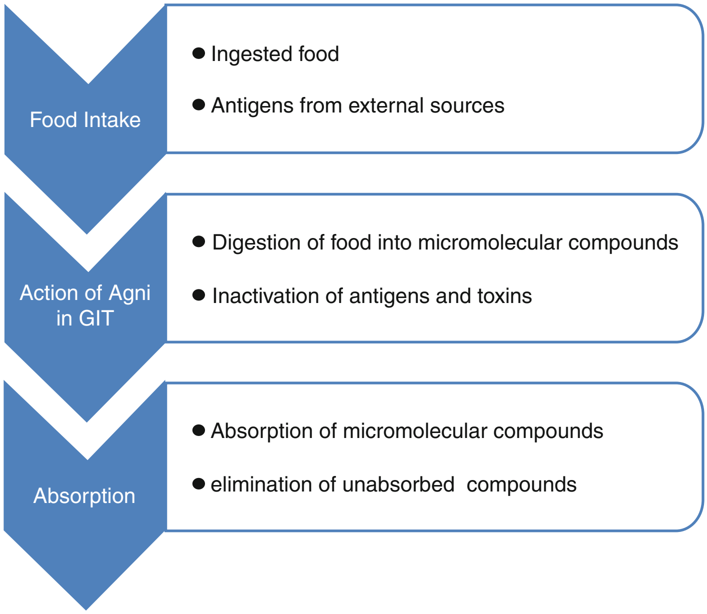


Fig. 8.1 Normal digestive physiology

complete the process of any transformation, an adequacy of *agni* is required; it is clear that if *agni* is inadequate, the produce of it would not be of the desired consistency. These macromolecular substances which are produced by incomplete processing of substrates are nothing but *ama* (unripe) for their potential to be broken further into smaller components (Figs. 8.1 and 8.2).

Mandagni or a hypodigestive state thereby is found to be operational at two levels which are mutually interdependent. At one end, it acts through a deficient enzyme state, leading to inadequate processing of the substrates and thereby producing inadequately treated end products of enzymatic reactions (Fig. 8.3). On the other hand, it also points out to an integral pathology to gut mucosa which is supposedly responsible for the poor enzymatic secretions. An inadequate secretion may also be consequential to food intake, food quality, or food composition–related factors, as is discussed earlier (Table 8.1). Through both of its operational mechanisms, mandagni gives rise to the following pathology:

1. Inadequate handling with the ingested substances
2. Enhanced uptake of improperly treated macromolecular substances into the systemic circulation

In whatever way the *ama* is produced, due to its increased influx in the gut mucosa in reference to *mandagni*-related epithelial changes, it makes the surrounding immune system exposed to various new antigens, a pathology which marks the beginning to various autoimmune disorders.

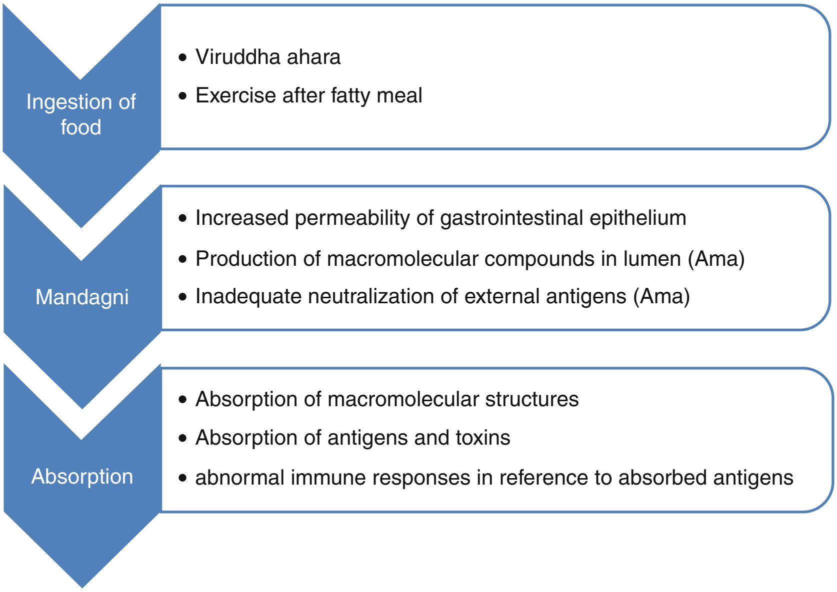


Fig. 8.2 Digestive physiology referring to production of *Ama* and subsequent pathology

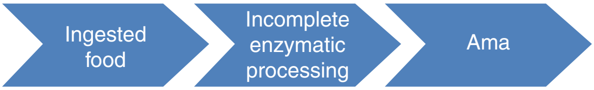


Fig. 8.3 Production of *Ama* through incomplete enzymatic processing of ingested food

An inadequate processing of food leading to production of *ama* can also be a result of reduced contact between food and enzymes, a factor which is aptly described in subsequent etiology to *Amavata*. An exercise following a fatty meal may lead to a rapid transit of food from the gut, making it less exposed to enzymatic activity. The result would again be the production of substrates none other than *ama*.

8.3 Etiopathogenesis of *Amavata*

Describing further to etiology of *Amavata*, *Madhava* stressed upon two principal etiologies predominant either to sedentary people marked with *mandagni* and habitual to *viruddh-ahara* or to active but stressed people who are compelled to make physical activities immediately after meals, especially the ones which contain fat. Interestingly, we can clearly say that these two etiologies are nothing but the two distinct mechanisms of producing *ama*, which once produced, chooses a common pathway to develop *Amavata*-related manifestations. This would also be interesting to elaborate these two distinct etiopathogenesis of *Amavata* in light of recent advances made in the field of digestive physiology. But before elaborating it further, it is important to have a look at the common pathway through which *Amavata* finally manifests.

8.3.1 Pathogenesis in Amavata

Once the *ama* acquires a sustainable formation and subsequent accumulation in the gastrointestinal tract due to either of its two principal etiologies, it goes further to subsequent morbid changes. *Ama* produced and accumulated in such ways is subsequently subjected to internalization and migration towards the places which are rich in substances having a property similar to that of *ama*. For general considerations, *kapha* has a reasonable resemblance to that of *ama*, and therefore, this movement of *ama* is largely directed towards *kapha* predominant places in the body. *Vata* plays an important role in *ama* internalization and also in its further movements within the body.

Accumulating further and reaching to a critical level, *ama* then flows through arteries where it gets an opportunity to react with various preexisting substrates of *vata–pitta–kapha* distinctions. This interaction gives rise to diverse products having profound adherence as a common feature. An *ama* produce of this variety quickly develops weakness and heaviness in the body as a forerunner to *Amavata*. A *vata* and *ama* accumulation following to this in joints mainly to the lumbo-saccral region gradually cause a stiffness to the whole body. This is how *Amavata* begins.

A few points conceptualized to *Amavata* pathogenesis in *Ayurveda* are truly phenomenal. *Ama*, the principal causative factor to *Amavata*, is said to be produced through variable ways. Once *ama* acquires a sustainable production owing to regularity of its etiology, it does not remain in gastrointestinal tract alone but, under the influence of *vata*, starts getting internalized to body tissues. *Vata* here depicts the factors which promote internalization of *ama* through the intestinal mucosa. A small amount of *ama* internalized in such a way remains limited to gut surrounding tissue and causes immune changes at local level. Under normal circumstances, the gut epithelium allows only minute quantity of intact antigens to cross into the mucosa, where they interact with the mucosal immune system to downregulate inflammation [17]. It is, however, important to observe that if *ama* is produced in higher quantities, it may absorb to reach to intestinal portal system. Once internalized, *ama* is subjected to interactions with various preexisting substrates having *vata–pitta–kapha* properties. As a consequence, *ama* is further converted into its highly adherent form. Interestingly, by considering *ama* as macromolecular antigenic substances, we can also presume exposure of these antigens to preexisting T lymphocytes in the peripheral circulation [18]. An antigen–T lymphocyte complex is then further exposed to immune reactions, leading to the ultimate production of antibodies against such antigens which are having a gross cross-reactivity, especially to *ama*-resembling substances of which *kapha* is the most important to be considered [19].

Table 8.2 General features of *Amavata*

Feature	Interpretation
Angamarda	Body ache
Aruchi	Loss of appetite
Trishna	Thirst
Aalasya	Malaise
Gaurava	Heaviness
Jvara	Fever
Apaka	Indigestion
Shunata-anganaam	Swelling in various body parts

8.3.2 Symptomatology in Amavata

Amavata in general is manifested through a variety of symptoms which are produced through a clogging of microconduits of body through *ama*. Besides this, *ama*, the principal morbid product in *Amavata* itself, has properties which promote inflammation in connection to its adherence with various preexisting tissues in the body. General features of *Amavata* as given in Ayurvedic texts are shown in Table 8.2.

It is important to understand that in *Amavata*, because of intangible nature of inherent pathogenesis and also because of need of accumulation of etiological factors to a critical level before *Amavata* is actually manifested, a time gap between etiological exposure and a symptom manifestation is obvious and can never be clearly predicted.

The symptom profile and intensity in *Amavata* and their time concordance in reference to etiological exposures are therefore variable in reference to various confounding factors like individual capacity to handle various noxious substances and individual response in reference to that exposure.

Once the pathogenesis of *Amavata* gets matured, it is manifested through various musculoskeletal symptoms which become the hallmark to this disease (Table 8.3). An association to *vata*, *pitta*, or *kapha* to disease-causing factors in *Amavata* further gives rise to a few identical features typical to these factors. Thereby an association of pitta is marked with burning and tenderness, *vata* with pain, and *kapha* with heaviness and itching (Table 8.4).

8.4 Reappraising *Amavata* Etiology to Contemporary Understanding

As we have understood so far, *Amavata* is found to have two distinct etiologies. It either initiates among people who are less active, have poor digestive capacities, and are habituated to *viruddha ahara* or among people who are active but stressed. The typical etiology narrated for latter subgroup is an exercise schedule just after heavy meals containing fat.

Table 8.3 Specific symptoms of *Amavata*

Features	Contemporary meaning
Saruja sandhi shotha	Painful swelling at joints
Hasta, *Pada*, *Shiro Gulpha*, *Trika Jaanu*, *Uru Sandhi*	Hand, foot, cervical, ankle, sacral, knee, and hip joints
Rujyatetyarthah vyaviddha iva vrishchike	Intense pain like a scorpion bite
Agni-daurbalya	Indigestion
Praseka	Excessive salivation
Aruchi	Loss of appetite
Gaurava	Heaviness
Utsaha hani	Loss of interest
Vairasya	Loss of taste
Daah	Burning
Bahu-mutrata	Polyurea
Kukshi kathinata	Hardness in abdomen
Shula	Pain in abdomen
Nidra viparyaya	Sleep diversion
Trit	Thirst
Chardi	Vomiting
Bhrama	Giddiness
Murcha	Syncope
Hrid griha	Heaviness in heart
Vida Vibaddhata	Constipation
Jadya	Stiffness
Aantra kujana	Increased sound in abdomen
Aanaha	Abdominal distention

Table 8.4 *Dosha* specific features in *Amavata*

Dosha	Features
Pitta	*Daha*, *Raga*
Vata	*Shula*
Kapha	*Stimit*, *Guru*, *Kandu*

This is interesting to elaborate these two distinct etiologies of *Amavata* in reference to their contemporary significance as per recent understanding of human physiology.

8.4.1 Amavata Among People with Mandagni–Nischalata–Viruddha ahara

A distinct subgroup of people characterized by less active routines, poor digestive capacities, added with habitual *viruddha ahara* intake is identified in Ayurveda as susceptible to *Amavata*. As the group is characterized by three independent pathological factors which are able to cause independent pathologies at their own, this is interesting to see as to how they interact together to give rise to a more complicated pathology like *Amavata*.

Mandagni can arise independently as a feature in response to various dietary or routine responses. It can also arise as a response to *Nishchalata*, an independent factor identified as a cause to *Amavata*. As we have understood so far, *mandagni* or the hypoactive metabolic state can manifest at gastrointestinal tract level through a reduction in independent or cumulative enzyme quantity secreted in response to the dietary intakes. Any such deficiency will lead to an inadequate transformation of dietary intake into the subsequent end products; the result will be the production of *ama*. A further quest into *mandagni* gives us a clue that *mandagni* itself is not limited to transiently reduced enzymatic secretions but simultaneously also involves the factors which cause a permanent deficit into the enzyme secretion mechanism. An important mechanism which may be involved in mandagni-related features could be a gradual destruction of intestinal mucosal cells responsible for enzyme secretion and also a poor intramural plexus response to dietary stimuli, leading to a neurohormonal mechanism for enzymatic secretions. Interestingly, dietary lectins from common dietary staples like cereal grains and legumes are found to have influence upon structure and function of enterocytes and lymphocytes [20, 21]. Wheat germ agglutinin and lectin in general are found bound to gut brush border epithelial cells, causing damage to the base of villi through disarrangement of cytoskeleton, increased endocytosis, and shortening of the microvilli [20–22]. The structural changes induced by this agglutinin on intestinal epithelial cells elicit functional changes including increased permeability [23] which may facilitate the passage of undegraded dietary antigens into systemic circulation [21]. This is important to reappraise if this could be the possible mechanism of working of *mandagni* and *ama* in reference to *Amavata* pathogenesis.

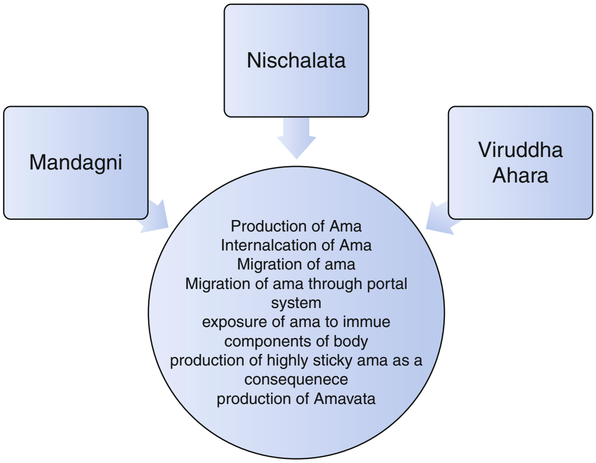
Nischalata or poor physical activity can reciprocally be associated to *mandagni*. A sedentary lifestyle eventually reduces the energy expenditure and subsequent energy requirements of human body. If it is sustained longer, it may lead to a sustained low energy transaction or *mandagni*. A mandagni of independent origin, in turn, can also cause *nischalata* as a consequence to poor digestion and subsequent production of *ama*.

Viruddha ahara is another important factor which contributes to the pathogenesis of *Amavata. Viruddha ahara* classically represents the combination of food which is incompatible if taken together. This dietary incompatibility as per Ayurveda is operational not only at level of food composition but also at levels like *Matra* (quantity), *Guna* (property), *Karma* (action), *Kala* (time duration), or *Sanskara* (qualitative transformations) of food. An incompatibility, therefore, may substantiate through any such factor making a food unsuitable to human consumption.

Viruddha ahara is proposed to give rise to many transient and stable clinical features. *Charaka* defines *viruddha ahara* for three of its distinct properties [24]:

1. Characters opposite to *dhatu*
2. *Dosha* vitiating property
3. Remains in the body even after the *dosha* vitiation

Fig. 8.4 An interplay of *Mandagni–Nischalata–Viruddha ahara* leading to *Amavata*



This clearly marks out the property of *viruddha ahara* for their stable antigenic properties leading to chronic and lasting illnesses.

In reference to *viruddha ahara*, it is also important to understand that a method of intake also can turn a wholesome food into the incompatible ones. Mental state of the person who is consuming the food plays a very important role in determining the net outcome to a dietary intake. Although acting primarily through *agni*, it can make the consumed food incompatible due to the poor enzymatic action and subsequent production of end products which are not compatible with body tissue. A net interplay of *mandagni–nischalata–viruddha ahara* can be speculated, as shown in Fig. 8.4. Once the *ama* is produced subsequent to etiological exposures, it acquires a common pathway to cause the disease. *Ama* produced gradually gets internalized under the influence of *vata*. This internalization of *ama* is variously facilitated by many factors coacting at the gut mucosa–lumen interface. Under normal circumstances, gut epithelium controls internalization of large antigenic molecules through an intestinal barrier function. This barrier includes physical diffusion barrier, regulated physiological and enzymatic barrier, and immunological barrier which are under neurohormonal control and can be affected by various mechanisms like stress and dietary intakes. A continuous epithelial cell layer interconnected with tight junctions restricts both transcellular and paracellular permeation of molecules, thus constituting the principal component of intestinal barrier. In addition, the epithelium exerts an important physiological defense by secretion of fluid and mucus, together with secretory IgA, into the lumen to dilute, wash away, and bind noxious substances [17]. A mechanism which operates to cause intestinal barrier dysfunction may be complex and may involve either of the barriers we discussed. A loosening at the mucosal tight junctions, besides direct cellular damages in response to stress or incompatible intakes, becomes one plausible mechanism through which ama can find a way to get internalized to the gut peripheral tissue. This tissue is invariably rich in lymphatics and gives a reason to Ayurvedic perception of *ama* migration to the places which are similar to *kapha*. Any *ama* which gets internalized in such a way to gut periphery gets exposed to various immune factors operating locally. As a consequence, it generates local immune reactions causing local inflammation, a phenomena required to produce gut immunity physiologically but can facilitate architectural disorganization of gut mucosa and thereby can augment *mandagni*. A large quantity of *ama* added with a substantial barrier dysfunction may give *ama* a way to get absorbed through portal system. Once internalized through portal system, *ama* is exposed to various food-related antigens and also to various indigenous proteins. Due to their complex structure, *ama* is presumed to have a cross-reactivity to various macromolecular proteins intrinsically available in the body, a phenomena recently studied in reference to various dietary proteins and their antigenicity in autoimmune conditions [19]. As a primary immune reaction, *ama* is exposed and trapped by T lymphocytes through their target receptors. Once trapped, *ama* is presented further to B lymphocytes to produce antibodies against such *ama* antigens. Ayurveda beautifully elaborated the whole phenomena as an *ama–vata–pitta–kapha* interaction leading to huge production of highly adherent and disease-causing type of *ama*.

8.4.2 Amavata Among Snigdha–Bhuktavato–Vyayama People

Another etiology speculated for *Amavata* is for people where *mandagni* is not a primary pathology and where *ama* is produced through a mechanism of escaping enzymatic action upon dietary components because of their rapid transit. *Snigdha ahara* itself has an affinity for being rapidly transmitted. An exercise following a fatty meal elaborates about the mental and social state of the person who is consuming food. A compulsion to go for work immediately after meals is a good example which elaborates the Ayurvedic *snigdha–bhuktavato–vyayama* proposition of *Amavata*. Interestingly, a rapid transit not only gives a poor exposure of enzymes upon their respective substrates, it also reduces the neutralization of various dietary toxins through respective action of many neutralizing factors in the gut lumen. The result is the production of *ama* which could be a net sum of inadequately digested end products and also inadequately neutralized toxins.

A habit or a compulsion of intruding into exercise after meals more adequately is expressed through the stress which might be related to one's profession. A continuous stress gives rise to certain phenomenal changes to gut mucosa in reference to its permeability and absorptive capacity. Under stress, macromolecular molecules from the gut are found easily internalized into the gut mucosa, a phenomena which is less commonly observed in a normal unstressed mucosa.

It is therefore easy to speculate that under stress, a person is more liable to have increased production of *ama* as a combination to inadequately processed end products of digestion and also to inadequately neutralized dietary toxins. At the same time, it is also noteworthy that under stress, this *ama* gets a better opportunity to get internalized into the gut mucosa, a phenomena which is crucial for the genesis of *Amavata* subsequently (Fig. 8.5).

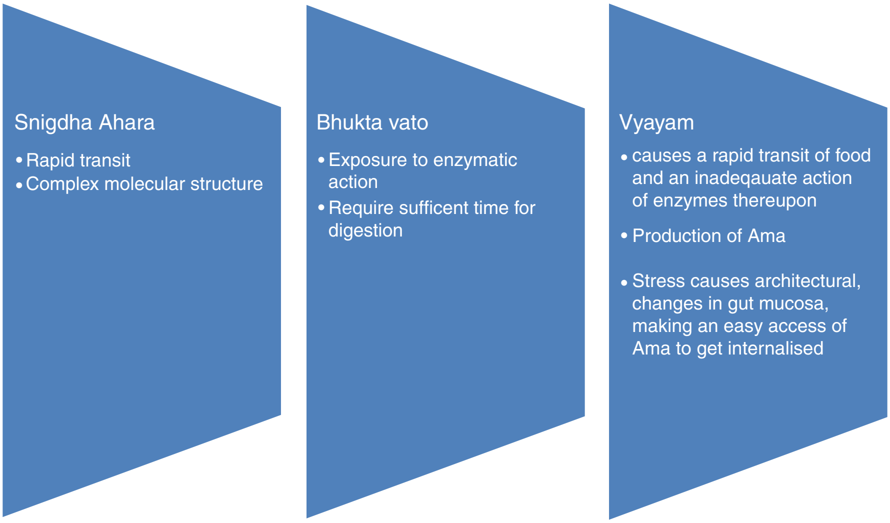


Fig. 8.5 Pathogenesis of *Amavata* among *Snigdha–Bhuktavato–Vyayama* people

8.4.3 Final Pathway to Amavata Production

We can find that production of *ama* may have different pathways, but once it is produced and internalized, it adopts a common pathway of reacting with body tissue, ultimately leading to production of *Amavata*.

Ama, once internalized, is usually migrated to the gut periphery, a tissue which is usually rich in lymphatics. Ayurveda perceives it as the primary migration of *ama* to *kapha* predominant tissue. This migration is supposed to give rise to local reactions which can also augment the architectural changes in gut mucosa. In case of sustained and copious production of *ama*, it can get absorbed through the portal system of intestine, a mechanism which makes *ama* exposed to more diversified and systemic actions. During its migration through portal system, *ama* gets opportunities to react with various body tissues, of which more important are the T lymphocytes. *Ama* trapped through T lymphocytes is further presented to B lymphocytes for a subsequent production of antibodies against trapped *ama*. Interestingly, *ama* or the macromolecular antigens internalized in such a way are found to have a cross-reactivity with various tissue proteins due to molecular mimicry [25]. Antibodies, therefore, produced against such *ama* particles are presumed to act against many self-proteins too, a mechanism through which a cascade of autoimmune disease begins.

Ama, after its reaction to *vata–pitta–kapha*, is proposed to become highly adherent (*picchila*) and noxious, an elaboration which truly fits to the subsequent antibody production in reference to *ama* presentation to B lymphocytes. This *ama* has a high affinity to body tissue which is adequately elaborated through its clogging *picchila* property.

Ayurveda portrays all the subsequent events of *Amavata* pathogenesis referring to the production of *picchila ama*. A clogging of microconduits of the body has been proposed as the possible mechanism to subsequent manifestations.

8.5 Portraying the Plan of Management in *Amavata*

Once the pathogenesis of *Amavata* is understood, it becomes easy to understand how Ayurveda approaches towards its management. So far we have understood the mechanism of *ama* production as the primary step from where a footing to *Amavata* begins.

An approach to manage *Amavata*, therefore, fundamentally includes:

1. Reduction in *ama* production
2. Reduction in *ama* internalization
3. Dissociation of internalized *ama*

8.5.1 Reduction in Ama Production

A reduction in *ama* production could be the primary step to handle *Amavata*. As *ama* is the outcome of *mandagni*, a recorrection of *agni* could be considered as the primary step to deal with *Amavata*. This is important to understand that a recorrection of *agni* here essentially involves an architectural correction of intestinal mucosa which is ultimately responsible for the enzyme secretions. We are aware that intestinal mucosa observes a regular shedding off phenomena with its renewal at regular intervals. By avoiding the mechanisms which may be involved in gut mucosal destruction and by adopting a supportive mechanism to protect gut mucosal lining, a restoration of gut mucosa and subsequently *agni* may be approached.

At the same time, we also need to understand that a supplementation in the form of additional enzymes from external sources or the drugs which may stimulate the enzymatic secretions may not be an efficient remedy to *mandagni* as it would either transiently patch up the problem instead of making a correction, or it would stimulate the compromised mucosa for more enzyme production, a measure which will augment the cell destruction in long run.

A *mandagni* correction, therefore, in *Amavata* should only be approached, initially by avoidance of etiological factors which are causing a *mandagni* and subsequently by exposing the intestinal mucosa to dietary substrates which may promote tissue restoration. The best approach is to begin with food which requires simple digestion. This food is called *laghu* (the food which requires less time to get digested) in Ayurveda.

For restoration of *agni*, it is also important that *agni* be given a chance to recover on its own by providing it a period of rest. This can be approached through a reduction in daily calorie intake in comparison to the presumptive expenditure. A relative negative energy balance for few days is proposed to help elimination of stored junks within the tissue, thereby making the system more clean. Ayurveda approaches to relative negative energy balance through *laghvasana* (less food intake) and *langhana* (fasting). Interestingly, both of these approaches are considered as cornerstones of *Amavata* management in Ayurveda. Interestingly, beneficial effects of fasting and vegetarian diet upon markers of rheumatoid arthritis are now been identified through many researches including the systematic reviews [26–30].

A reduction in *ama* production should also be approached through reduction of intakes which are *ama* promotive. *Viruddha ahara* and *snigdha ahara* are such intakes which themselves are *ama* promotive. They thereby are required to be reduced. An observation of reduced symptom intensity among rheumatoid arthritis patients consuming a vegetarian diet substantiates this management approach [26–29]. Activities which are *ama* promotive should also be reduced at the same time when we are trying to reduce *ama* through dietary corrections. A sedentary lifestyle with minimum of physical activities or a stressful lifestyle where one is compelled to be involved in extraneous physical activities just after food intake should, therefore, also be avoided.

8.5.2 Reduction in Ama Internalization

Through the discussions of preceding parts of this chapter, we are aware that internalization of *ama* is primarily dependent upon architectural changes to the gut mucosa in reference to *mandagni* or stress. A reduction of *ama* internalization, thereby, can only be achieved if an architectural correction to gut mucosa is approached. To initiate with, this is important to avoid the factors like stress and *viruddha ahara* which may promote the mucosal destruction.

Besides this, the gut mucosa is also required to be provided with enough rest to get an opportunity to restore its deficits, if there are any. Conventionally, this is being done with the observance of *langhana* (fasting) or *lagvasana* (relative fasting).

8.5.3 Dissociation of Internalized Ama

Simultaneous to reduction in *ama* production and its internalization, among established cases of *Amavata*, we also required to deal with *ama* which is already internalized and has interacted with body tissue to generate noxious and adherent form of *ama*.

A dissociation of the macromolecular structures of *ama* into smaller fractions which independently may not have the adherence property of *ama* is commonly employed approach of Ayurveda to dismantle internalized macromolecular *ama*.

The herbs rich in *katu* (bitter), *tikta* (pungent taste), and *lavana* (salt) *rasa* (taste) are found good to offer dissociative effects to *ama*. Ayurvedic formulations offering *dipana* (ignition) and *pachana* (dissociation) effects are mainly composed of components which are predominant in these *rasa*.

A comprehensive management plan of *Amavata*, therefore, can be tailored around the idea of *ama* formation, its internalization, and its reaction to body tissue. Essentially, a reversal to *Amavata* pathology should also be sought to the same order.

An important thing which needs to be stressed at this juncture is the presumption of time which may be required to give an observable effect among *Amavata* patients through an Ayurvedic *Amavata* management protocol. We need to reiterate here about prospective etiopathogenesis and level of ama which might be present in individual cases. If there are no good reasons to produce explicit amount of *ama* and if the *agni* is not compromised, possibly the recovery may be faster in comparison to a person where there are ample opportunities for *ama* to surface because of preexisting *mandagni* added with dietary inputs promotive to *ama* production.

If *ama* is available in ample amount and has interacted well to the body tissue, it is obvious to think that this would take longer to be recovered in comparison to other conditions where *ama* remained less offensive. Furthermore, due to huge variability to the *ama*–tissue interaction, an *ama* produce may also be variable in terms of its dissociability. This gives us a reason to understand huge individual variation to Ayurvedic treatment response among *Amavata* patients.

To sum up, we need to reiterate that a management plan to *Amavata* should be confronted at various fronts simultaneously. Obviously and for this reason too, a management to *Amavata* is time-consuming and cumbersome, a phenomena which is routinely observed in the clinical practice of Ayurvedic rheumatology.

8.6 Conclusion

Rheumatoid arthritis in particular and various autoimmune arthritic conditions in general are still the big challenges to the human health-care system. *Amavata* in Ayurveda is conceptualized as a group of conditions marked predominantly by joint afflictions besides many other systemic manifestations. It is for this reason that *Amavata* is often placed parallel to what is understood about rheumatoid arthritis in modern medicine. Interestingly, it is not only their symptom resemblance but rather a pathogenesis too which gives us a clue to think for a common wisdom behind the understanding of autoimmune joint conditions within these two streams of health care. Ayurveda, since antiquity, argued for a gut initiative to *Amavata*, a phenomena explored only lately through the contemporary science. These explorations have dissected into the gut–joint interactions to find a feasibility of generating a joint disease in the future. It is found that architectural changes at gut epithelium are primarily responsible to many immunological changes in the body which may further initiate the process of more complex autoimmune reactions due to their properties of molecular mimicry and cross-reactivity. *Mandagni*, a beautiful elaboration of hypodigestive state from Ayurveda, further explains about possible architectural changes in gut lumen which subsequently may lead to a hypoenzymatic condition, a prerequisite to *ama* production. Ayurvedic management approach to *Amavata* comprehensively handles the issues individually involved in its pathogenesis. At this juncture, we can conclude that an evidence base to Ayurvedic management of *Amavata* is almost ripe on the conceptual grounds. It only requires to be tested clinically and experimentally to give it a more sharpened effect so as to offer a more pragmatic, dependable, reproducible, and biological approach to deal with various autoimmune joint conditions.

Deciphering the Ayurvedic attributes of a pathogenesis in tune to the contemporary knowledge is the foremost prerequisite of articulating a trustworthy Ayurvedic intervention protocol in any condition [31]. Making a hypothesis based upon classical thoughts and associating it judicially with contemporary knowledge serves as a foundation to an evidence-based synthesis. If sustained and subjected to rigorous methods of scientific enquiry, it may ultimately help in modulating evidence-based Ayurveda [32, 33]. *Amavata* exposition in this purview may prove to be a milestone in unearthing the ways of synthesizing evidence base to Ayurveda.

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