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REVIEW ARTICLE

Gut–Brain Axis and Amlapitta (Hyperacidity): A Review of *Jatharagni* Modulation and Microbial Homeostasis via *Ayurvedic* Detoxification

Randhir Kaushal[1]*, Sudha Sharma[2]

[1]Associate Professor, Department of Panchkarma, Government Ayurveda College, Sikar, Rajasthan, India.
[2]Principal, Shri Vinod Durgadutt Tibrewala Institute of Ayurveda Medical College and Hospital, Jhunjhunu, Rajasthan, India.

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ABSTRACT

Functional dyspepsia is a highly prevalent disorder of gut–brain interaction under the Rome IV classification, characterized by post-prandial fullness, early satiety, epigastric pain, and burning. Traditional treatments often rely on acid-suppressive therapies that can induce long-term dysbiosis. In contrast, Ayurveda describes this clinical spectrum as *Amlapitta*, a pathology of the *Annavaha Srotas* (gastrointestinal tract) driven by *Jatharagnimandya* (depressed central metabolic fire) and the qualitative vitiation of *Pachaka Pitta* (specifically, increases in *Dravatva* and *Amlatva*). This review explores the structural and functional convergence between the classical Ayurvedic principle of *Pittadhara Kala Sahayo Majjadhara Kala* and the modern gut–brain–microbiome (GBM) axis. We evaluate how traditional Ayurvedic biopurification (*Shodhana*), specifically therapeutic emesis (*Vamana*) and purgation (*Virechana*), acts as a biological "reset" of the GBM axis. By analyzing classical Samhita literature, clinical symptomatology, and herbo-mineral pharmacology (*Sutshekhar Rasa*, *Kamadudha Rasa*, *Avipattikar Churna*, and *Amlapitta Mishran Suspension*), we present an evidence-based, integrative therapeutic paradigm. Furthermore, we outline how the graduated post-purification dietary protocol (*Samsarjana Krama*) functions as a structured prebiotic reconstruction algorithm that stabilizes short-chain fatty acid-producing bacterial cohorts, repairs mucosal barrier tight junctions, and restores autonomic gut–brain communication.

1. INTRODUCTION

Functional dyspepsia (FD) represents a major clinical challenge in modern gastroenterology, affecting approximately 10–30% of the global adult population. According to the Rome IV diagnostic consensus, FD is clinically characterized by the persistence of bothersome post-prandial fullness, early satiety, epigastric pain, or epigastric burning for at least 3 months, with symptom onset occurring at least 6 months before diagnosis, in the absence of any structural or organic disease.[1] Standard conventional treatments, primarily consisting of acid-suppressive therapies such as proton pump inhibitors (PPIs) and H2-receptor antagonists, fail to achieve satisfactory symptom resolution in approximately 86% of patients, demonstrating a low overall efficacy rate of around 14%. Furthermore, long-term administration of PPIs disrupts the gastric acid barrier, predisposing patients to systemic complications such as hypochlorhydria, osteoporosis, and profound intestinal dysbiosis, including small intestinal bacterial overgrowth.[2]

In classical Ayurvedic medicine, this cluster of upper gastrointestinal symptoms is classified as *Amlapitta*, a pathological condition affecting the *Annavaha Srotas* (the channels carrying food). *Amlapitta* is characterized by the qualitative and quantitative vitiation of *Pachaka Pitta* (the digestive subtype of *Pitta*), wherein its natural alkaline and sharp properties (*Katu Rasa*) undergo abnormal acidification, transforming into a highly sour and liquid state (*Amla* and *Drava Guna* increase). This pathological shift is driven by *Jatharagnimandya* (depressed central metabolic fire), which leads to incomplete digestion of food and the subsequent generation of *Ama,* a highly reactive, toxic, and undigested metabolic intermediate.

Despite the therapeutic success of Ayurvedic interventions, a significant translation gap exists between classical literature and modern clinical science. Modern gastroenterology now conceptualizes FD not as an

Corresponding Author:
Randhir Kaushal, Associate Professor,
Department of Panchkarma, Government Ayurveda College, Sikar, Rajasthan, India.
Email: santoshrandhir14@gmail.com

isolated disorder of gastric secretion, but as a complex disorder of gut–brain interaction governed by the gut–brain–microbiome (GBM) axis. This bidirectional communication is mediated by neural, endocrine, metabolic, and immunological pathways, which closely mirror the traditional Ayurvedic principle of *Pittadhara Kala Sahayo Majjadhara Kala* (PDK-MDK), the mutualistic and physiological dependency between the digestive membrane (*Pittadhara*) and the nervous system/brain tissue (*Majjadhara*).[3,4]

2. METHODS

To compile a comprehensive repository of classical and contemporary evidence, a systematic search of both classical Ayurvedic texts and modern peer-reviewed databases was conducted. The foundational treatises of Ayurveda, including the *Brihatrayi* (*Charaka Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya*) and the *Laghutrayi* (*Madhava Nidana*, *Kashyapa Samhita*, and *Bhavaprakasha*), were critically reviewed to map the historical evolution, etiological factors (*Nidana*), pathogenesis (*Samprapti*), clinical features (*Rupa*), and therapeutic formulations of *Amlapitta*.[5,6]

3. RESULTS

3.1. Pathophysiology of *Amlapitta* and FD: A Dual-perspective Analysis

3.1.1. Ayurvedic pathophysiology (Samprapti)

The *Samprapti* (pathogenesis) of *Amlapitta* is an intricate process of *Dosha-Dushya Sammurchana* (interaction of humors and tissues) initiated by chronic exposure to *Aharaja* (dietary) and *Viharaja* (lifestyle) *Nidanas* (etiologies). The consumption of incompatible food combinations (*Viruddha Ahara*), excessively sour (*Amla*), pungent (*Katu*), salty (*Lavana*), heavy (*Guru*), and deep-fried foods, along with psychological triggers such as *Chinta* (anxiety), *Shoka* (grief), and *Krodha* (anger), severely impairs the *Jatharagni*. This state of diminished digestive fire, known as *Jatharagnimandya*, compromises the breakdown of ingested materials, leading to the formation of *Ama* [Figure 1].[7]

As *Ama* accumulates, it causes obstruction in the functional channels of the body (*Srotorodha*), specifically within the *Annavaha* and *Rasavaha Srotas*. Concurrently, the *Pachaka Pitta* (located in the *Amashaya* or stomach and the *Grahani* or duodenum) becomes hyperactive. Its natural alkaline, sharp, and pungent qualities (*Katu Rasa*) are corrupted into an excessively acidic (*Amla*) and fluid (*Drava*) state, a phenomenon termed *Vidagdhatva*. This fluid accumulation of *Vidagdha Pitta* overwhelms the local buffering capacities of the gastric mucosa, irritating the tissue and resulting in the clinical manifestation of *Amlapitta*.[8,9]

3.1.2. Modern pathophysiology of FD

Modern biomedical research defines FD as a multifactorial disorder characterized by dysregulation of the GBM axis. The core pathophysiological nodes include:

- Vagal autonomic dysfunction: Efferent parasympathetic vagal fibers, which innervate the myenteric and submucosal plexuses of the stomach, are critical for coordinating the gastric accommodative reflex (the relaxation of the proximal stomach to store food) and antral motility. In patients with FD, autonomic neuropathy or impaired vagal tone disrupts these reflexes, leading to early satiety and delayed gastric emptying of solids and liquids.
- Visceral hypersensitivity: This involves an abnormal sensory response of the enteric nervous system (ENS) and vagal afferents to mechanical stretching, chemical irritation, and luminal acid. Central sensitization of sensory processing areas in the brain, such as the anterior cingulate cortex, amplifies these normal digestive signals into clinical pain or burning sensations.[8]
- Mucosal low-grade inflammation and barrier defect: Tight junction dysfunction in the duodenal mucosa allows the paracellular translocation of luminal antigens and pathobionts. This triggers localized mast cell and eosinophil activation, releasing inflammatory mediators that alter ENS signaling and impair local muscle contractility.
- Microbial dysbiosis: Imbalances in the gastrointestinal microecology – specifically a reduction in beneficial, short-chain fatty acid (SCFA)-producing taxa and an overgrowth of facultative anaerobes – deplete protective mucosal barriers. The subsequent loss of luminal SCFAs (such as butyrate and acetate) weakens mucosal repair and fuels local immune reactivity [Figure 2].

4. AYURVEDIC DETOXIFICATION (SHODHANA) METHODS IN AMLAPITTA[10]

Ayurvedic therapeutics prioritize the physical extraction of vitiated humors (*Shodhana*) before initiating palliative herbomineral therapies (*Shamana*). This is critical because administering heavy palliative formulations in a state of chronic channel obstruction (*Srotorodha*) and low digestive capacity (*Mandagni*) results in poor drug absorption and metabolic toxicity.[11]

4.1. *Purvakarma* (Preparatory Protocol)

Before physical biopurification is performed, the patient undergoes a structured preparatory regimen designed to mobilize deep-seated toxins (*Ama*) from peripheral tissues (*Shakha*) into the gastrointestinal tract (*Koshtha*).

- *Deepana-Pachana*: Administration of carminative and digestive herbs such as *Shunthi* (*Zingiber officinale*), *Maricha* (*Piper nigrum*), and *Chitrakadi Vati* to stimulate *Jatharagni* and metabolize systemic *Ama*.
- *Snehapana* (Internal oleation): Graduated oral administration of medicated lipid vehicles, typically *Mahatiktaka Ghrita* or *Sukumara Ghrita*, in increasing daily doses. The lipid vehicle penetrates cellular barriers, dissolving lipophilic toxins and packaging them for excretion.
- *Snehana-Swedana* (External Oleation and Sudoration): Passive whole-body massage with warm oils followed by steam chamber therapy to dilate microvascular channels (*Srotas*), liquefying mobilized toxins and channeling them toward the stomach and intestines.

4.2. *Vamana Karma* (Therapeutic Emesis)

Acharya Kashyapa establishes *Vamana* as the primary therapeutic choice for *Amlapitta*. *Vamana* targets the upper gastrointestinal tract (*Amashaya*), the physiological home of *Kapha* and *Pachaka Pitta*.

- Mechanism: The physical act of controlled emesis, induced through formulations containing *Madanaphala* (*Randia dumetorum*) and *Yashtimadhu* (*Glycyrrhiza glabra*) decoctions, evacuates the accumulated, highly acidic, and fermented mucus barrier of the stomach.
- Indication: Primarily indicated in *Urdhwaga Amlapitta* (upward-moving acid reflux) dominated by *Kapha–Pitta* symptoms such as nausea, sweet-sour regurgitation, and throat burning. Kashyapa states that performing *Vamana* is like destroying a poisonous tree by cutting its roots.

4.3. *Virechana Karma* (Therapeutic Purgation)[12]

Virechana is the premier purification therapy for *Pitta* disorders. It targets the small intestine (*Grahani*) and liver-bile secretory apparatus, physically purging the corrupted *Vidagdha Pitta* through the lower gastrointestinal tract.

- Mechanism: It is executed using specialized purgative compounds such as *Trivrit Churna* (*Operculina turpethum*), *Eranda Taila* (castor oil), or *Avipattikar Churna*. This induced osmotic purgation drains the portal circulation, clearing accumulated bile acids and systemic inflammatory mediators.
- Indication: Highly effective in both *Urdhwaga* and *Adhoga Amlapitta*, particularly when characterized by severe epigastric burning, abdominal discomfort, and bitter belching.

4.4. *Sadyovirechana* (Acute Purgation)

In cases of acute metabolic distress or when the patient lacks the structural strength (*Bala*) to undergo the full, multi-week Panchakarma protocol, *Sadyovirechana* is employed.

- Mechanism: It bypasses the long internal oleation (*Snehapana*) phase, administering a single, therapeutic dose of *Avipattikar Churna* (5–10 g) at bedtime with warm water. This provides rapid downward mobilization of the *Doshas* (*Pitta Anulomana*), bringing immediate relief from acute heartburn and duodenal inflammation.

4.5. *Basti Karma* (Medicated Enemas)

Although *Basti* primarily targets the colon (*Pakvashaya*), its systemic impact on the autonomic nervous system makes it vital for modulating the gut–brain axis.

- Mechanism: Administering oil enemas (*Anuvasana Basti*) or herbal decoction enemas (*Niruha Basti*) stimulates the rich enteric nervous network of the rectal mucosa. This downregulates hypothalamic–pituitary–adrenal axis hyperreactivity, reduces visceral hypersensitivity, and modulates the central pain matrix, resolving the neurosensory symptoms of FD.

5. CLINICAL PRESENTATION AND DIAGNOSTIC CRITERIA CORRELATION

To ensure clinical precision, the symptoms of *Amlapitta* must be mapped onto modern diagnostic categories. The classical texts divide *Amlapitta* based on the direction of the vitiated *Pitta*'s movement: *Urdhwaga* (upward) and *Adhoga* (downward). These presentation pathways closely correlate with the Rome IV subgroups of FD: Post-prandial distress syndrome and epigastric pain syndrome. Table 1 details the clinical alignment between classical Ayurvedic signs and symptoms (*Rupa*) and modern diagnostic criteria.

6. *SAMHITA*-BASED COMPARATIVE ANALYSIS OF *AMLAPITTA* [MENTIONED IN TABLE 2]

6.1. Theoretical Trajectory of *Samhita* Literature

The conceptual understanding of *Amlapitta* has undergone a clear evolutionary trajectory across classical Ayurvedic history. In the early classical period (*Brihatrayi*), *Amlapitta* was not classified as an independent disease entity. In the *Charaka Samhita*, it is conceptualized primarily as a metabolic symptom or clinical complication (*Upadrava*) arising from underlying systemic pathologies, such as *Vidagdhajirna* (acidic indigestion) or *Grahani Roga* (intestinal malabsorption). Charaka attributes its pathogenesis to the ingestion of incompatible food (*Viruddha Ahara*), which corrupts *Pachaka Pitta* in the stomach, transforming the nutrient fluid (*Rasa Dhatu*) into an acidic toxin.[13]

Similarly, the *Sushruta Samhita* focuses on the physiological biochemistry of the humor, stating that *Pitta* is naturally of *Katu Rasa* (pungent taste) but undergoes a molecular transition to *Amla Rasa* (sour taste) when it becomes *Vidagdha* (improperly digested or fermented). Sushruta's commentator, Dalhana, provided the critical physiological link by detailing PDK-MDK, establishing that the membranes of digestion and neural processing share a deep, mutualistic, and bidirectional dependency.[14]

In the later medieval period, the clinical prevalence of the condition led to its systematization as an independent, standalone pathology (*Vyadhi* or *Mahagada*). The *Kashyapa Samhita* (specifically the *Khila Sthana*) was the first to dedicate an independent chapter to *Amlapitta*, describing its etiology, pathogenesis, and pediatric/obstetric presentation. This was followed by the *Madhava Nidana*, which provided the definitive nosological classification, dividing the disease into *Urdhwaga* (upward) and *Adhoga* (downward) pathways based on the physical movement of the vitiated fluid. Finally, the *Bhavaprakasha* consolidated these classifications, expanding the clinical picture to include secondary systemic and psychosomatic symptoms like giddiness (*Bhrama*) and skin eruptions (*Kotha*). This evolutionary perspective is shown in the table below.[15]

7. CLINICAL DIFFERENTIAL DIAGNOSIS

Mentioned in table 3.

8. PHARMACOLOGICAL COMPARISON OF CLASSICAL FORMULATIONS[16]

Ayurvedic therapeutics for *Amlapitta* combine targeted herbo-mineral formulations (*Shamana*) with physical purification (*Shodhana*). Table 4 details the quantitative composition, pharmacodynamics, and modern mechanisms of action of four major classical formulations.

9. DISCUSSION

The clinical efficacy of classical bio-purification (*Shodhana*), specifically *Virechana Karma* (therapeutic purgation), in managing *Amlapitta* lies in its capacity to physically evacuate deeply seated, aggravated *Pitta* from its primary physiological sites in the *Amashaya* (stomach) and *Grahani* (duodenum). Modern microbiological and metabolic evaluations validate this approach, demonstrating that *Virechana* significantly corrects gut flora dysbiosis by reducing pathogenic, facultatively aerobic bacteria such as *Escherichia coli*. This clearance curtails the luminal pool of circulating lipopolysaccharides, thereby arresting a major systemic trigger for chronic, low-grade mucosal inflammation. Furthermore, *Virechana* acts as a systemic metabolic reset by lowering fecal fat content, fasting blood glucose, and serum triglycerides, while actively reversing steatotic changes in visceral tissues by enhancing insulin sensitivity at the skeletal muscle level. At the crossroads of the GBM axis, this biopurification optimizes hepatic clearance and bile acid kinetics, stimulating the release of glucagon-like peptide 1 and Peptide YY from enteroendocrine cells through TGR5 receptor binding to normalize gastric accommodation, antral motility, and central satiety networks. This neuroendocrine cascade directly addresses the core pathophysiology of FD, illustrating the functional alignment between the central digestive fire (*Jatharagni*) and cellular metabolism. In this framework, *Sama Agni* reflects intestinal eubiosis where a diverse

microbiome generates SCFAs to maintain mucosal immune tolerance, whereas *Mandagni* (depressed digestive fire) drives dysbiosis and the generation of toxic *Ama* (endotoxins and pro-inflammatory intermediates). This biological interdependence is structurally codified in Ayurveda via the PDK-MDK axis, establishing a mutualistic link between the digestive mucosa (*Pittadhara*) and the nervous system (*Majjadhara*). This axis regulates neurotransmitter homeostasis – such as the microbial processing of dietary tryptophan into serotonin (5-HT) to modulate local peristalsis and vagal signaling – validating why brain-targeted *Medhya Rasayanas* require optimal processing by *Jatharagni* to nourish the *Majja Dhatu* effectively. This neural loop matches the functional equilibrium between *Prana Vata* and *Saman Vayu*, explaining how psychological distress (*Chinta, Shoka,* and *Krodha*) destabilizes *Agni*, and how chronic *Pitta* vitiation or *Viruddha Ahara* (incompatible diet) can precipitate psychological pathologies (*Maanas Vyaadhi*) such as *Unmaad* and *Apasmaar*. To safely rebuild this delicate equilibrium post-purification without inducing osmotic shock, Ayurveda mandates *Samsarjana Krama*, a highly structured, graduated dietary algorithm. Transitioning from *Manda* and *Peya* (supernatant rice waters) to *Vilepi* and *Yusha* (thick gruels and lentil soups), and finally *Maamsa Rasa* (meat broths), this protocol functions as a targeted prebiotic reconstruction strategy. It first provides easily assimilable carbohydrates to protect the hypersensitive epithelium and prime basal enzyme secretions, then delivers complex plant fibers to selectively cultivate protective bacterial cohorts such as *Bifidobacteria* and *Lactobacilli* for tight-junction repair and SCFA synthesis, and ultimately supplies bioavailable amino acids such as glutamine and glycine for structural tissue repair (*Dhatu Upachaya*). By systematically scaling nutritional complexity in strict alignment with the step-by-step recovery of *Agni*, *Samsarjana Krama* successfully rebuilds a stable gut microbiome, repairs mucosal barriers, and permanently resets the metabolic intelligence of the gut–brain axis.

10. CONCLUSION

This comprehensive, evidence-based review demonstrates that the classical Ayurvedic model of *Amlapitta* and *Jatharagni* aligns with contemporary gut–brain–microbiome science. The *Samprapti* of *Amlapitta*, characterized by *Jatharagnimandya* and *Ama* formation, corresponds to the pathophysiological processes of FD under the Rome IV criteria, including vagal autonomic dysfunction, visceral hypersensitivity, mucosal barrier defects, and gut microbiota dysbiosis.

By evaluating these processes through the lens of the PDK-MDK axis, we gain an integrated understanding of the therapeutic mechanisms of Ayurvedic detoxification. Clinical and experimental data demonstrate that *Virechana Karma* is an effective biopurification process that corrects gut dysbiosis, significantly reduces *Escherichia coli* pathobionts, and improves liver-gut–brain signaling. Furthermore, post-cleansing dietary protocols such as *Samsarjana Krama* serve as structured prebiotic regimens, systematically rebuilding a diverse and stable gut microbiome while restoring metabolic fire (*Agni*).

Integrating targeted herbo-mineral formulations such as *Sutshekhar Rasa*, *Kamadudha Rasa*, *Avipattikar Churna*, and *Amlapitta Mishran Suspension* provides a multi-targeted approach to GI care. These remedies work by neutralizing excess gastric acid, stabilizing the mucosal barrier, promoting healthy gut motility, and reducing neuroinflammation. Utilizing these evidence-based Ayurvedic protocols alongside contemporary diagnostics provides a safe, effective, and personalized model of integrative gastroenterology, helping address the complex challenges of chronic functional gastrointestinal disorders.

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15. CONFLICT OF INTERESTS

The authors declare no conflicts of interest regarding the publication of this paper.

16. DATA AVAILABILITY STATEMENT

The data analyzed in this review were obtained from publicly available sources, including peer-reviewed articles, observational studies, and surveys accessible through databases.

17. DISCLAIMER/VIEW AND OPINIONS

The opinions expressed in this article are solely those of the authors and do not reflect the views of the International Research Journal of Ayurveda and Yoga or its editorial board.

18. AI-USE DECLARATION

The authors declare that no generative AI tools were used to create scientific content, interpret data, or draft any sections of this manuscript. AI-based tools were used solely for minor language and grammar refinements to improve clarity and readability. All scientific content, analysis, and conclusions remain the sole responsibility of the author.

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Table 1: Correlation of Ayurvedic Rupa with Rome IV criteria and underlying mechanisms

Classical ayurvedic feature	Modern medical symptom	Rome IV classification	Pathophysiological basis and axis modulation
Avipaka	Indigestion and delayed digestion	PDS	*Jatharagnimandya*; delayed gastric emptying and impaired gastric accommodation.
Gaurava/ Gurukoshthatva	Bothersome post-prandial fullness and abdominal heaviness	PDS	Accumulation of Ama in the Annavaha Srotas; poor motility of the proximal stomach.
Hrut-Kantha Daha	Retrosternal and epigastric burning (heartburn)	EPS	Vitiation of Ushna and Tikshna Guna of Pitta; mucosal exposure to excess gastric acid and visceral hypersensitivity.
Tikta-Amla Udgar	Bitter-sour eructations and acid regurgitation	EPS/GERD overlap	Downward flow impairment (Vata Vimargagamita); transient lower esophageal sphincter relaxation.
Aruchi	Anorexia or early satiation	PDS	Impairment of Bodhaka Kapha and Saman Vayu due to Ama; mechanical gastric distension hypersensitivity.
Utklesha	Nausea and occasional vomiting	PDS	Upward push of Vidagdha Kledaka Kapha and Pitta; antral-duodenal dysmotility and vagal afferent activation.
Amashayadaha	Epigastric pain or localized burning	EPS	Direct mucosal erosion by hyper-acidic, fluid Pitta; localized mucosal mast cell/eosinophil activation.
Shiroruja	Dyspeptic headache	Systemic gut–brain manifestation	Bidirectional transmission along the *Pittadhara Kala Sahayo Majjadhara Kala* axis; systemic absorption of inflammatory cytokines/lipopolysaccharides through leaky gut.

EPS: Epigastric pain syndrome, GERD: Gastroesophageal reflux disease, PDS: Post-prandial distress syndrome

Table 2: Comparative analysis of *Amlapitta* across classical ayurvedic texts

Classical text	Nosological status	Primary pathogenetic theory	Classifications described	Key recommended therapeutic interventions
Charaka Samhita	Symptom/*Upadrava* (secondary to *Vidagdhajirna*/*Grahani*)	*Viruddha Ahara* corrupts *Pachaka* Pitta and Rasa *Dhatu*, turning digestion acidic.	None are described as an independent disease.	*Deepana-Pachana*, use of *Mahatiktaka Ghrita*, and restoring gut integrity.
Sushruta Samhita	Physiological Variation of Pitta Guna	Pitta transitions from natural Katu Rasa to abnormal Amla Rasa when Vidagdha.	None described; details the *Pittadhara Kala Sahayo Majjadhara* axis dependency.	Bile acid management, alkaline therapies, and correcting local tissue heat.
Kashyapa Samhita	Independent Disease (*Mahagada*)	*Vidagdha* food remains in the stomach, impairing *Jatharagni* and provoking Pitta.	*Vatika, Paittika*, and *Shleshmika* subtypes.	*Vamana Karma* as the primary curative line; place change for mental peace.
Madhava Nidana	Independent Disease (Standalone Chapter)	*Vidagdha* and liquid Pitta (*Dravatva* increase) accumulate in the gastric lumen.	*Urdhwaga* (Upward) and *Adhoga* (Downward) Gati.	*Pitta-Shamana* formulations, *Mridu Virechana*, and strict dietary codes.
Bhavaprakasha	Independent Disease (Systemic Phase)	Vitiated Pitta spreads systemically, impacting skin, blood, and cognitive tissues.	*Urdhwaga*, *Adhoga*, and chronic multi-*dosha* variants.	Herbo-mineral formulations (*Sutshekhar Rasa*) and cooling rejuvenators.

Table 3: Clinical differential diagnosis of *Amlapitta* and allied pathologies

Clinical entity	Primary site	Cardinal symptoms	Pain-food relationship	Pathological biomarkers
Amlapitta	*Amashaya* (Stomach) and Annavaha Srotas	*Hrut-Kantha Daha (heartburn), Tikta-Amla Udgar, Utklesha* (nausea).	Generalized burning, aggravated by fasting or immediately after meals.	Qualitative vitiation of *Pachaka Pitta* (*Dravatva* and *Amlatva* increase); no mucosal erosion.
Pitta Grahani	Grahani (Small Intestine)	Loose stools with undigested food, severe thirst, and localized burning.	Typically post-prandial, during the active phase of small intestinal digestion.	Intestinal malabsorption, tissue wasting, is highly linked to gut dysbiosis (depleted Bacteroidetes).
Vidagdhajirna	*Amashaya* (Stomach)	Acute sour belching, excessive thirst, warm perspiration, and chest burn.	Occurs immediately after a meal, resolving once the food is digested or expelled.	Acute temporary metabolic overload; temporary inactivation of *Jatharagni* without channel block.
Parinama Shula	*Amashaya-Pakvashaya Sandhi* (Duodenum)	Severe, localized abdominal colic, bloating, and flatulence.	Occurs specifically during late stages of digestion; relieved temporarily by eating.	Corresponds to duodenal ulcers; *Vata-Pitta* dominance with mucosal tissue erosion.
Annadrava Shula	*Amashaya* (Stomach)	Constant, intense, burning epigastric pain unresponsive to dietary changes.	Persistent pain occurring regardless of food status; temporarily relieved by vomiting.	Corresponds to gastric ulcers; severe tissue destruction (Dhatu Kshaya) and mucosal damage.
GERD	LES and esophagus	Substernal burning, regurgitation of gastric contents, and chronic dry cough.	Aggravated by lying down, bending, or physical straining post-meals.	Reflux of gastric acid due to reduced LES tone and defective esophageal peristalsis.

GERD: Gastroesophageal reflux disease, LES: Lower esophageal sphincter

Table 4: Pharmacological mechanisms of classical formulations in *Amlapitta*[17,18]

Formulation and composition	Key active ingredients	Ayurvedic pharmacodynamics	Modern pharmacological and clinical mechanisms
Sutshekhar Rasa[19] *Swarna Bhasma, Shuddha Parada, Shuddha Gandhaka, Shankha Bhasma, Dhattura, Tankan, Tamra Bhasma, Bhringaraja, Pippali, etc.*	Dhatura stramonium alkaloids (atropine, scopolamine, hyoscyamine), Tankan (Borax), Shankha Bhasma (Calcium carbonate).	• *Rasa: Tikta, Katu, Kashaya* • *Virya: Ushna/Sheeta Vipaka: Katu* • *Action: Deepana, Pachana, Pittashamana, Amapachaka.*	• Antimuscarinic anticholinergic action: Dhattura alkaloids competitively block muscarinic acetylcholine receptors, inhibiting vagally-mediated acid secretion from gastric parietal cells. • Direct acid neutralization: Tankan and Shankha Bhasma act as local antacids, raising gastric pH.
Kamadudha Rasa Shankha Bhasma, Kapardika Bhasma, Muktashukti Bhasma, Pravala Bhasma, Pravala Pishti, and Guduchi Satva.	Calcium carbonate (mineral-calcite complexes), Guduchi Satva (*Tinospora cordifolia*).	• *Rasa: Madhura, Tikta* • *Virya: Sheeta* • *Vipaka: Madhura* • *Action: Atyamla-hara, Dahaprashamana, Balya.*	• Mucosal barrier support: Released calcium ions act as tight junction stabilizers in the mucosal intercellular matrices, strengthening the protective mucosal barrier against acid-induced erosion. Guduchi Satva provides antioxidant, anti-inflammatory, and adaptogenic properties.
Avipattikar Churna[20] *Triphala, Trikatu, Musta, Vidanga, Ela, Patra, Lavanga, Trivrit, and Sharkara.*	*Operculina turpethum (Trivrit), Syzygium aromaticum (Lavanga), Phyllanthus emblica (Amalaki), Cyperus rotundus (Musta).*	• *Rasa: Katu, Tikta, Madhura* • *Virya: Ushna* • *Vipaka: Katu* • *Action: Pitta-Virechana, Anulomana, Srotoshodhana.*	• Anti-secretory activity: Significantly reduces gastric volume, free acidity, and total acidity. • Prokinetic and anulomana action: Trivrit acts as an osmotic laxative, facilitating gastric emptying and reducing duodenogastric reflux. Prebiotic modulation: *Triphala* supplies rich polyphenols.
Amlapitta Mishran Suspension Bhunimbadi Kwath ingredients (Kalmegha, Ativisha, Lodhra, Musta, Indrayava, Amruta, Balaka, Dhanyaka, Bilva) along with Yashti and Shouktik Bhasma.	*Andrographis paniculata (Kalmegha), Glycyrrhiza glabra (Yashti),* processed seashell calcium carbonate *(Shouktik Bhasma).*	• *Rasa: Tikta, Kashaya, Madhura* • *Virya: Sheeta* • *Vipaka: Madhura* • *Action: Sama Pitta Pachana, Vrana Ropana, Dahashamaka.*	• Cytoprotective and ulcer healing: Yashti and Amruta promote mucus secretion, strengthening the gastric mucosal barrier and accelerating the healing of micro-erosions. • Hepatoprotective: Kalmegha stimulates bile flow and hepatic clearance.

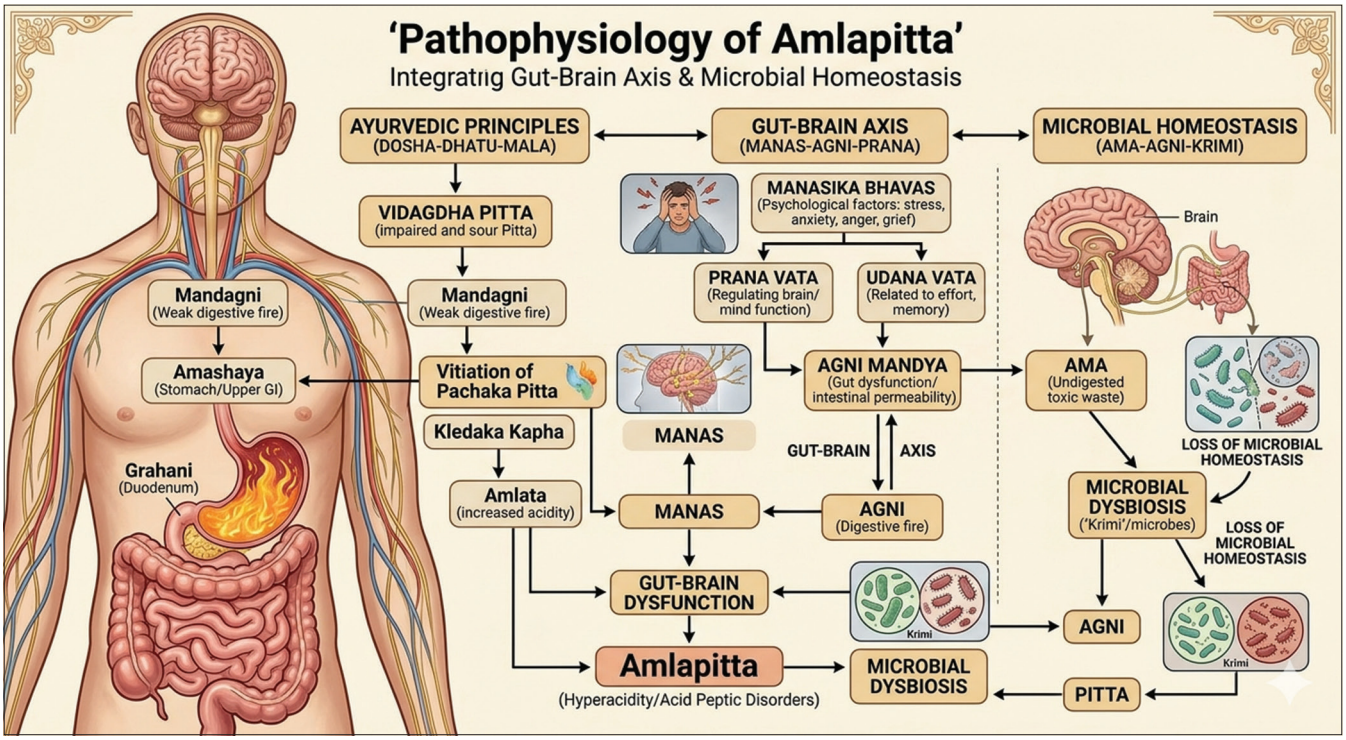

Figure 1: Pathophysiology (*Samprapti)* of *Amlapitta* and functional dyspepsia

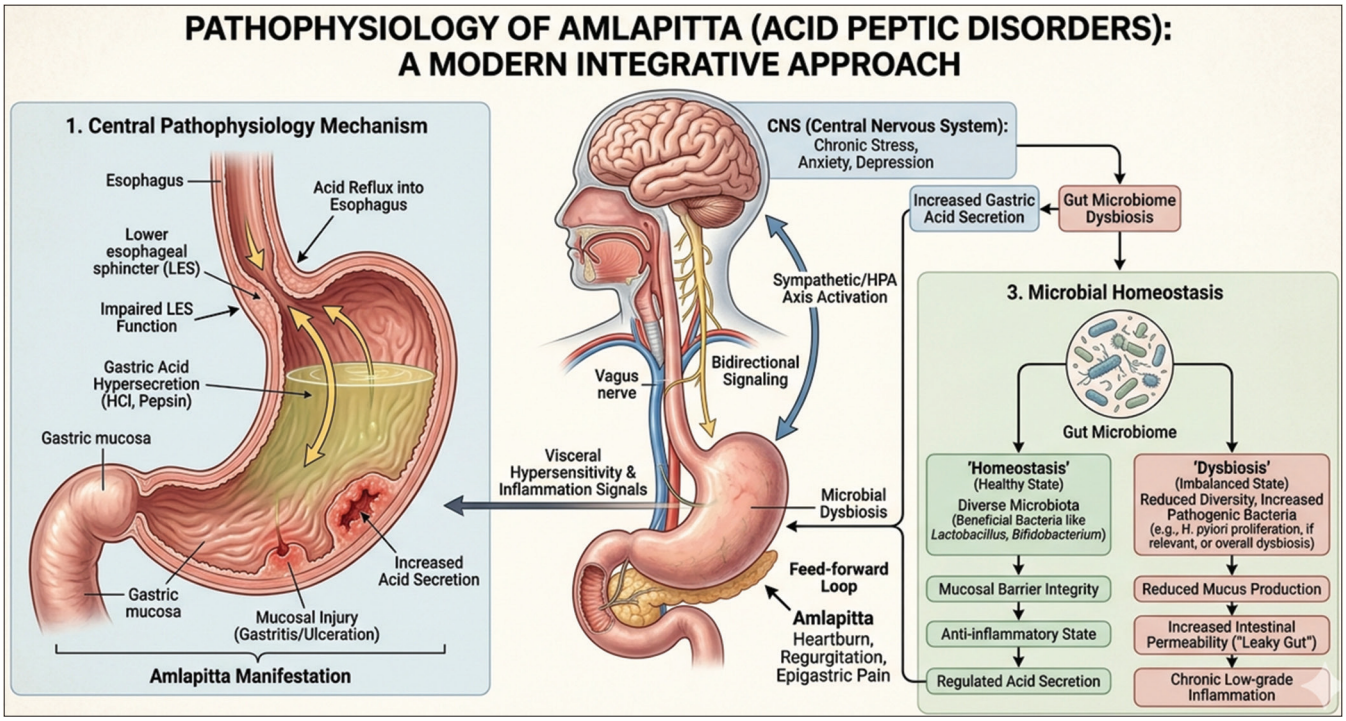

Figure 2: Modern pathophysiology of functional dyspepsia