WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.453
Volume 15, Issue 2, 460-466. Review Article ISSN 2277–7105

AMLAPITTA AS A PRECURSOR OF METABOLIC SYNDROME: AN INTEGRATIVE REVIEW FROM AYURVEDIC AND BIOMEDICAL PERSPECTIVES

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Article Received on 15 Dec. 2025,
Article Revised on 05 Jan. 2025,
Article Published on 16 Jan. 2026,

https://doi.org/10.5281/zenodo.18264548

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How to cite this Article: Dr. Ratnesh Kumar Shukla*[1], Dr. Shraddha Sharma[2] (2026). Amlapitta As A Precursor Of Metabolic Syndrome: An Integrative Review From Ayurvedic And Biomedical Perspectives. World Journal of Pharmaceutical Research, 15(2), 460–466.

ABSTRACT

Background: Metabolic Syndrome (MetS) is a rapidly escalating global health crisis involving insulin resistance, central obesity, and dyslipidemia. While modern medicine focuses on managing established metabolic risks, emerging evidence suggests that functional digestive disturbances and low-grade inflammation precede these overt metabolic changes. **Objective:** This integrative review aims to conceptualize the *Ayurvedic* diagnosis of *Amlapitta* not merely as a gastric disorder, but as a prodromal (early warning) stage of Metabolic Syndrome. **Discussion:** In *Ayurveda*, *Agni* (digestive fire) is central to health, and *Amlapitta* arises from *Agni* impairment and *Pitta* aggravation. Classical texts describe a progression from *Amlapitta* to *Medovaha Srotodushti* (fat metabolism disorder) and *Prameha* (diabetes). This parallels biomedical findings where Gastroesophageal Reflux Disease (GERD) is strongly linked to visceral adiposity, cytokine-induced inflammation, and insulin resistance. Both systems indicate that metabolic dysregulation begins in the gut. **Conclusion:** Identifying *Amlapitta* as an early metabolic marker offers a novel window for prevention. Integrating *Ayurvedic* correction of *Agni* with modern lifestyle interventions at this stage could prevent the progression to full-blown Metabolic Syndrome.

KEYWORDS: *Amlapitta*, Metabolic syndrome, GERD, *Agni*; *Ama*.

INTRODUCTION

Metabolic syndrome (MetS) has emerged as one of the most significant non-communicable disease clusters of the modern era. It markedly increases the risk of type 2 diabetes mellitus, cardiovascular disease, and premature mortality.[1,2] Despite extensive research, modern medical management largely focuses on treating established metabolic abnormalities rather than identifying early reversible stages.[3]

Recent evidence indicates that metabolic syndrome evolves gradually, beginning with subtle functional disturbances in digestion, metabolism, and inflammatory regulation.[4–6] Gastrointestinal disorders such as gastroesophageal reflux disease (GERD) and functional dyspepsia are increasingly recognized as being closely associated with obesity, insulin resistance, and systemic inflammation.[7–10]

Ayurveda places digestion and metabolism at the center of health maintenance. Classical texts state that *Agni* is the foundation of life and that its derangement initiates disease processes.[11–13] *Amlapitta*, a condition arising from impaired *Agni* and aggravated *Pitta*, presents with symptoms strikingly similar to GERD. Importantly, *Ayurveda* describes progression from *Amlapitta* to *Medovaha Srotodushti*, *Sthaulya*, and *Prameha*, suggesting a metabolic continuum. This review explores whether *Amlapitta* can be conceptualized as a precursor stage of metabolic syndrome.

Ayurvedic Concept of *Amlapitta*

Amlapitta is described primarily in *Madhava Nidana* and elaborated in *Charaka Samhita* as a disorder resulting from vitiation of *Pitta* along with deranged *Agni*.[14,15] Etiological factors include excessive intake of sour, spicy, oily, and incompatible foods, irregular dietary habits, suppression of natural urges, sedentary lifestyle, and psychological stress.[16]

The central pathological mechanisms involve:

* *Agnimandya* (diminished digestive fire) or *Tikshnagni* (sharp digestive fire)
* Formation of *Ama* (metabolic toxins)
* *Pitta Prakopa* (aggravation of *Pitta*)
* Abnormal movement of vitiated *Pitta* (*Vimarga Gamana*)

Clinical features include *Amla Udgara* (sour belching), *Hrit-Kantha Daha* (heartburn), *Avipaka* (indigestion), *Utklesha* (nausea), *Chhardi* (vomiting), *Aruchi* (anorexia), and *Klama*

(fatigue). Classical texts clearly state that untreated or chronic *Amlapitta* leads to *Medovaha Srotodushti*, *Sthaulya*, and *Prameha*.[17,18], indicating its role in metabolic disease progression rather than being a mere gastric disorder.

Modern Biomedical Perspective: GERD and Metabolic Dysfunction

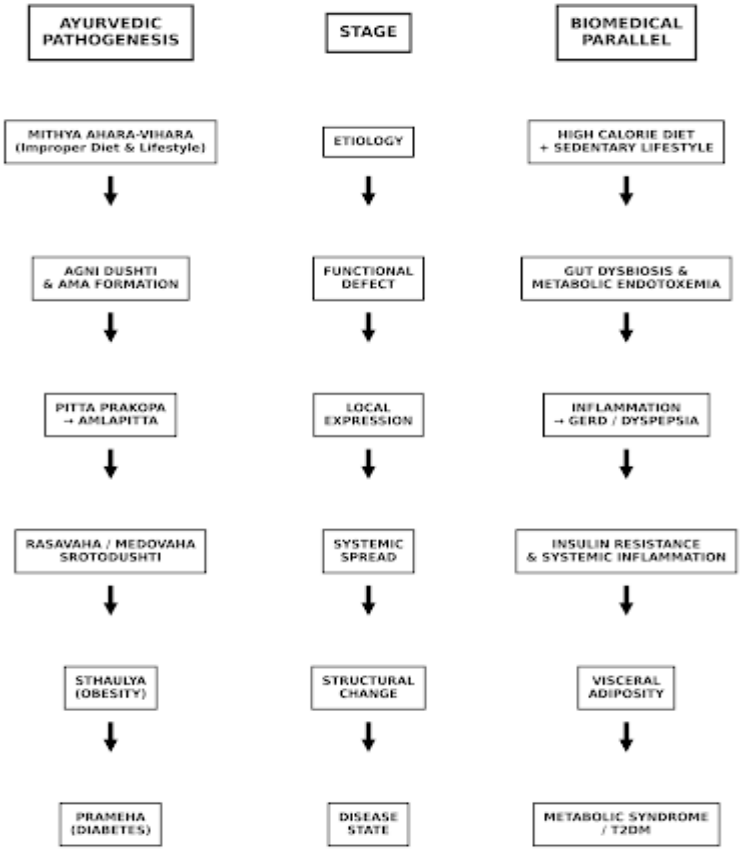
GERD is defined as a condition resulting from reflux of gastric contents causing troublesome symptoms or complications. Traditionally viewed as a mechanical acid-related disorder, GERD is now increasingly recognized as a systemic condition influenced by metabolic factors.[19,20]

Numerous epidemiological studies demonstrate a significantly higher prevalence of GERD among individuals with metabolic syndrome.[21–23] Visceral obesity increases intra-abdominal pressure and alters gastroesophageal junction physiology.[24] Insulin resistance impairs autonomic regulation and gastric motility, worsening reflux symptoms.[25-26]

GERD patients exhibit elevated inflammatory markers such as C-reactive protein, TNF-α, and IL-6, reflecting chronic low-grade inflammation — a hallmark of metabolic syndrome.[27–29] Thus, GERD may represent an early clinical manifestation of metabolic dysregulation rather than an isolated gastrointestinal condition.

Integrative Pathogenesis: A Comparative View

Both systems suggest that digestive dysfunction is the starting point of metabolic disease. The table below illustrates the alignment between the *Ayurvedic Samprapti* (pathogenesis) and the modern biomedical cascade.



Both systems converge on the concept that digestive and metabolic dysfunction precede overt metabolic disease.

DISCUSSION

The concept that metabolic syndrome develops from early functional disturbances rather than abrupt biochemical derangements is increasingly supported by modern research.[30–32] *Ayurveda* anticipated this concept through its emphasis on Agni as the central regulator of health.[11,33]

GERD and functional dyspepsia are now recognized as manifestations of the altered gut–brain–metabolic axis.[34,35] Gut microbiota dysbiosis, increased intestinal permeability, and metabolic endotoxemia play a crucial role in the development of insulin resistance and obesity.[36–38] These mechanisms closely parallel *Ayurvedic* descriptions of *Ama* spreading from the gastrointestinal tract and obstructing *Srotas*, leading to systemic metabolic dysfunction.[39,40]

Persistent *Pitta Prakopa* in *Amlapitta* corresponds to oxidative stress and inflammatory cytokine activation described in metabolic syndrome. Visceral adipose tissue acts as an endocrine organ, secreting pro-inflammatory mediators that further impair insulin signalling.[41–43] *Ayurveda* conceptualizes this state as *Medovaha Srotodushti*.

Importantly, both systems emphasize reversibility at early stages. *Amlapitta* is described as a *Saadhya* condition when managed early through dietary regulation, lifestyle modification, and correction of *Agni*.[17,18] Modern preventive medicine also highlights the effectiveness of early lifestyle intervention, though GERD is rarely viewed as a metabolic warning sign.[44–46]

Amlapitta, classically described as a disorder of *Agni* and aggravated *Pitta*, represents more than a localized acid-peptic condition. Improperly digested food undergoes fermentative transformation, producing systemic effects that indicate early metabolic imbalance. Clinical features such as fatigue (*Klama*), heaviness (*Gaurava*), and lethargy (*Alasya*) reflect impaired energy utilization and decreased metabolic efficiency, resembling early preclinical manifestations of metabolic syndrome. Persistent *Amlapitta* can influence *Meda Dhatu* metabolism due to prolonged *Agni* dysfunction and *Pitta* aggravation. As *Meda* metabolism is dependent on *Agni*, chronic *Amlapitta* may result in deranged adipose tissue accumulation (*Medodushti*), analogous to visceral obesity and dyslipidemia observed in metabolic syndrome. Formation of *Ama* due to *Agni* impairment further obstructs metabolic channels (*Srotas*), creating systemic low-grade inflammation—a key mechanism in the development of insulin resistance. From an integrative perspective, *Amlapitta* functions as a clinical marker of early metabolic dysfunction, where digestive impairment, inflammatory processes, and altered tissue metabolism coexist. Recognition and management of *Amlapitta* at this stage may prevent progression to *Medodushti* and *Prameha*, conditions that closely resemble the modern concept of metabolic syndrome.[12]

Clinical and Preventive Implications

Reframing *Amlapitta* as a precursor of metabolic syndrome suggests several shifts in clinical practice.

Screening: Patients presenting with GERD or *Amlapitta* should be screened for metabolic risk factors.

Focus on Digestion: Emphasis on digestive health is crucial in metabolic disease prevention.

Dietary Integration: Integration of *Ayurvedic* dietary and lifestyle principles with modern care offers cost-effective preventive strategies for non-communicable diseases.

CONCLUSION

This integrative review demonstrates strong conceptual and scientific parallels between *Amlapitta* and metabolic syndrome. Evidence from both *Ayurveda* and modern biomedical research suggests that digestive dysfunction and acid-peptic disorders may represent early metabolic warning stages. Conceptualizing *Amlapitta* as a precursor of metabolic syndrome aligns ancient *Ayurvedic* wisdom with contemporary scientific understanding and highlights the importance of early intervention in preventing metabolic disease.

REFERENCES

1. Alberti KGMM, Zimmet P, Shaw J. Lancet, 2005; 366: 1059-62.
2. Grundy SM. Circulation, 2008; 118: 594-7.
3. Reaven GM. Diabetes, 1988; 37: 1595-607.
4. Ford ES. Diabetes Care, 2005; 28: 2745-9.
5. Kassi E. Metabolism, 2011; 60: 113-20.
6. Misra A. Natl Med J India, 2007; 20: 193-9.
7. Moshkowitz M. World J Gastroenterol, 2011; 17: 4238-41.
8. Kim BJ. J Gastroenterol Hepatol, 2008; 23: 1360-5.
9. Chang P. Gastroenterol Clin North Am, 2014; 43: 161-73.
10. Park JH. J Neurogastroenterol Motil, 2017; 23: 421-9.
11. Sharma R, Dash B. Charaka Samhita.
12. Tripathi B. Madhava Nidana.
13. Shastri A. Sushruta Samhita.
14. Lad V. Textbook of Ayurveda.
15. Sharma PV. Ayurveda Ka Vaigyanik Itihas.
16. Tripathi I. Grahani Chikitsa.
17. Yadav R. J Ayurveda Integr Med, 2015; 6: 1-7.
18. Rastogi S. J Ayurveda Integr Med, 2012; 3: 67-72.
19. Fass R. Gut, 2007; 56: 1313-9.
20. Pandolfino JE. Clin Gastroenterol Hepatol, 2008; 6: 970-6.
21. Sonnenberg A. Gut, 2016; 65: 882-8.
22. Zhang X. Obes Rev, 2013; 14: 951-62.
23. Vaezi MF. N Engl J Med, 2018; 379: 2222-31.
24. Camilleri M. Gastroenterology, 2018; 154: 1571-81.
25. Moayyedi P. Am J Gastroenterol, 2017; 112: 988-1013.

26. Hotamisligil GS. Nature, 2006; 444: 860-7.
27. Tilg H. Nat Rev Immunol, 2006; 6: 772-83.
28. Aggarwal BB. Antioxid Redox Signal, 2010; 13: 731-43.
29. Turnbaugh PJ. Cell, 2009; 136: 44-56.
30. Cani PD. Gut, 2007; 56: 1391-8.
31. Kolterman O. Metabolism, 2019; 92: 36-44.
32. Hankey A. J Altern Complement Med, 2014; 20: 1-6.
33. Patwardhan B. J Ayurveda Integr Med, 2010; 1: 1-5.
34. Gupta R. Indian Heart J, 2016; 68: 646-54.
35. Tripathi YB. Indian J Exp Biol, 2013; 51: 90-6.
36. Tilg H. Gastroenterology, 2020; 159: 1099-112.
37. Holtmann G. Gut, 2014; 63: 706-16.
38. De Groot NL. Aliment Pharmacol Ther, 2009; 30: 110-21.
39. Konturek PC. J Physiol Pharmacol, 2011; 62: 591-9.
40. Camilleri M. Gastroenterology, 2018; 154: 1571-81.
41. Hotamisligil GS. Nature, 2006; 444: 860-7.
42. Tilg H. Nat Rev Immunol, 2006; 6: 772-83.
43. Aggarwal BB. Antioxid Redox Signal, 2010; 13: 731-43.
44. Moayyedi P. Am J Gastroenterol, 2017; 112: 988-1013.
45. Sonnenberg A. Gut, 2016; 65: 882-8.
46. Vaezi MF. N Engl J Med, 2018; 379: 2222-31.