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ROLE OF AYURVEDIC DIETARY REGIMENS IN THE NON-PHARMACOLOGICAL MANAGEMENT OF ASYMPTOMATIC HYPERURICEMIA: A NARRATIVE REVIEW

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Abstract

Background: Asymptomatic hyperuricemia is characterized by persistently elevated serum uric acid concentrations in the absence of overt gouty arthritis or uric acid nephrolithiasis. Although long regarded as a benign biochemical curiosity, accumulating evidence links it to gout, chronic kidney disease, cardiovascular disease, and metabolic syndrome. Current management emphasizes lifestyle and dietary correction during this silent phase, before irreversible joint or renal damage occurs.

Objective: To critically examine the role of Ayurvedic dietary regimens (*Pathya Ahara*, wholesome diet) in the non-pharmacological management of asymptomatic hyperuricemia by integrating classical Ayurvedic reasoning with contemporary nutritional science.

Methods: A narrative review was undertaken using classical Ayurvedic compendia and indexed biomedical literature from PubMed, Scopus, and Google Scholar (2000–2026). Material relating to Vatarakta, its causative factors (Nidana), and constitutional (Prakriti) and seasonal (Ritucharya) dietary planning was extracted and thematically synthesized.

Results: Ayurveda treats dietary correction as the primary therapeutic lever through Nidana Parivarjana. The causative profile of Vatarakta — excess sour, salty, pungent, alkaline, hot, and unctuous foods, red meat, fermented beverages, overeating, and sedentary habits — mirrors recognized dietary risk factors for hyperuricemia. Individualizing this framework by Prakriti, Agni, and Ritucharya sharpens its preventive value, and several classical Pathya

items show documented uricosuric or anti-inflammatory activity in modern pharmacological studies.

Conclusion: Ayurvedic dietary regimens offer a holistic, individualized, and low-cost strategy for arresting asymptomatic hyperuricemia before progression to symptomatic gout. The classical framework is biologically plausible and partly corroborated by modern nutritional science, but robust randomized controlled trials are still needed to establish standardized protocols and confirm long-term clinical benefit.

Keywords: Hyperuricemia; Vatarakta; Pathya Ahara; Nidana Parivarjana; Prakriti; Ritucharya.

1. Introduction

Asymptomatic hyperuricemia — persistently elevated serum uric acid in the absence of gouty arthritis or renal stone disease — is an increasingly common and frequently overlooked finding in routine biochemistry.[1] Because it produces no immediate symptoms, it is often dismissed as clinically inconsequential. Yet a growing body of evidence identifies it as an early warning sign of future disease, with links to cardiovascular disorders, metabolic syndrome, and chronic kidney disease that extend well beyond joint health.[2,4] Global dietary transitions toward purine-dense foods, sugar-sweetened beverages, and sedentary living have driven a steady rise in population uric acid levels over the past two decades.[3]

Pharmacological therapy remains the standard of care once gout becomes symptomatic, but there is growing interest in non-pharmacological strategies for the preceding asymptomatic phase, motivated by the desire to avoid long-term drug exposure, reduce healthcare expenditure, and equip patients with sustainable self-management tools.[5] Ayurveda offers a distinct vantage point on this problem: rather than targeting serum urate directly, it seeks to correct the underlying causative factors (*Nidana*) that disturb metabolic equilibrium.[6] Diet (*Ahara*, food or diet) is one of the three pillars of life (*Trayopastambha*, the three pillars of life) in classical Ayurvedic physiology, and dietary regulation (*Pathya Ahara*) is considered both preventive and curative.[7,8]

Although asymptomatic hyperuricemia has no direct nosological equivalent in classical texts, its natural history overlaps substantially with the early (*Purvarupa*, premonitory stage) of *Vatarakta*, a disorder arising from combined vitiation of *Vata* (the bio-humor governing

movement) *Dosha* (bio-functional humor governing physiology) and *Rakta Dhatu* (blood tissue).[6,9] The dietary and behavioral triggers described for *Vatarakta* — excess sour, salty, pungent, alkaline, hot, and unctuous foods, heavy meat consumption, fermented preparations, alcohol, overeating, irregular meal timing, daytime sleep, and physical inactivity — bear a striking resemblance to the modern risk-factor profile for hyperuricemia and gout.[9,10,11]

This review synthesizes classical Ayurvedic dietary reasoning — *Pathya–Apathya Ahara*, *Prakriti*-based planning, *Agni*-centered digestion, and *Ritucharya* — with contemporary evidence on diet and serum urate, in order to evaluate the case for Ayurvedic diet therapy as a first-line, non-pharmacological approach to asymptomatic hyperuricemia.

2. Methodology

This is a narrative, rather than systematic, review, reflecting the heterogeneous and largely non-indexed nature of classical Ayurvedic source material. Primary textual material was drawn from *Charaka Samhita*, *Sushruta Samhita*, *Ashtanga Hridaya*, *Bhaishajya Ratnavali*, *Yogaratnakara*, and *Bhava Prakash*, focusing on the *Vatarakta* and Amavata chapters and their *Nidana*, *Samprapti* (pathogenesis), and *Pathya–Apathya* sections.[7,9,10,12,23,27] Supplementary biomedical literature was retrieved from PubMed, Scopus, and Google Scholar using combinations of the terms "hyperuricemia," "gout," "purine diet," "uric acid," "Ayurveda," and "*Vatarakta*," restricted to English-language publications from 2000 to 2026, with earlier foundational studies included where directly relevant. Editorial priority was given to systematic reviews, meta-analyses, and controlled trials for the biomedical component, and to primary classical commentary for the Ayurvedic component. Given the narrative design, no formal risk-of-bias or PRISMA-based screening was applied; this limitation is discussed further in Section 12.

3. Understanding Asymptomatic Hyperuricemia

3.1 Pathophysiology and Causes

Uric acid is the terminal product of purine catabolism, generated from dietary nucleic acids and endogenous cell turnover. Serum concentration reflects the balance between production and excretion: roughly two-thirds is cleared renally, with the remainder eliminated through the gastrointestinal tract. Increased purine intake, impaired renal excretion, and genetic predisposition can each disturb this equilibrium.[15] High-purine foods — red meat, organ

meat, shellfish, and certain fish — raise serum urate when consumed in excess, while sugar-sweetened beverages and alcohol, particularly beer, increase production and simultaneously blunt renal clearance.[3] Obesity and metabolic syndrome compound the problem through increased tissue turnover and reduced renal function.[16]

3.2 Prevalence and Risk Factors

Asymptomatic hyperuricemia is common worldwide and more prevalent in men, reflecting hormonal differences in urate handling; global prevalence continues to rise alongside dietary change, physical inactivity, and obesity.[17] Recognized risk factors include age, sex, body mass index, dietary pattern, hypertension, diabetes, and chronic kidney disease, with a discernible familial and genetic component, including variants in urate transporter genes such as SLC2A9.[18,19]

3.3 Health Consequences

Some consequences deserve particular attention. First, roughly one in five individuals with sustained asymptomatic hyperuricemia will develop gout within 10–20 years, with attendant joint damage if untreated.[20] Second, elevated urate is associated with hypertension, endothelial dysfunction, and a higher incidence of heart failure and ischemic heart disease.[21] Third, hyperuricemia and metabolic syndrome interact bidirectionally, each amplifying the other through shared mechanisms of insulin resistance and dyslipidemia.[18] Fourth, chronic urate crystal precipitation in renal tubules contributes to uric acid nephropathy and accelerates progression of chronic kidney disease.[22] Fifth, the anticipatory anxiety of eventually developing gout, though rarely discussed, can itself erode quality of life and warrants acknowledgement in comprehensive care.[16]

4. Ayurveda: An Overview and the Concept of *Vatarakta*

Ayurveda defines health as a dynamic equilibrium of the three *Doshas* (Vata, Pitta, Kapha), *Agni*, the seven *Dhatus* (bodily tissues), and *Mala* (waste products), together with clarity of mind, senses, and spirit*[9]*

Diet and lifestyle correction (*Pathya*) is treated as inseparable from treatment itself. *Vaidya* (physician) Lolimbaraja's oft-quoted verse from *Vaidyajivanam* captures this succinctly, that medicine becomes superfluous when *Pathya* is observed, and futile when it is not.

Classical texts attribute this vitiation to excess *Amla* (sour), *Lavana* (salty),*Katu* (pungent),*Kshara* (alkaline),*Ushna* (hot), and *Snigdha* (unctuous) foods; *Ajirnabhojana*

(eating before the previous meal is digested); heavy reliance on horse gram, black gram, radish, leafy vegetables, marshy-animal meat, dried meat, curd, sugarcane products, and fermented preparations (Sura, Asava, Arishta, Souviraka, Shukta); and lifestyle factors such as physical inactivity, *Divaswapna* (daytime sleep), *Prajagarana* (night vigil), *Krodha* (anger), and chronic stress.[7,10,24]

5. *Prakriti*-Based Dietary Individualization

Ayurveda's central methodological contrast with conventional dietetics is its insistence on constitutional individualization. *Prakriti* — the fixed psychophysical constitution established at conception — determines an individual's baseline susceptibility to *Dosha* vitiation and therefore modulates dietary recommendations even within a single disease category.

Vata-predominant individuals, who are inherently prone to dryness, irregularity, and *Vata* aggravation, require particular vigilance against cold, dry, and light foods and benefit from warm, lightly unctuous, *Vata*-pacifying preparations, regular meal timing, and *Abhyanga* (therapeutic oil massage), since further *Vata* provocation directly worsens the *Vatarakta* process. *Pitta* (the bio-humor governing metabolism and heat)-predominant individuals, already biologically closer to *Rakta* vitiation through inherent heat and sharpness, must be especially strict in avoiding *Ushna*, *Amla*, and *Katu* foods, alcohol, and fermented preparations, as these compound *Pitta*-driven *Rakta Dushti* (vitiation of blood tissue). *Kapha* (the bio-humor governing structure and lubrication)-predominant individuals, who are more prone to obesity, sluggish *Agni*, and *Ama* (undigested metabolic toxin) accumulation, benefit most from avoidance of Guru and Abhishyandi foods such as curd and excess sesame, together with increased physical activity to prevent metabolic stagnation.[7,13]

In practice, most patients present with a dual *Prakriti* (e.g., Vata-Pitta or Pitta-Kapha), and dietary counselling is calibrated to the dominant and provoked *Dosha* at the time of assessment rather than to a single fixed template. This constitutional lens parallels, in principle, the contemporary recognition that dietary responses to purine load and fructose intake are not uniform across individuals and are shaped by genetic and metabolic phenotype.[1,19]

6. *Agni*: Digestive Fire and Metabolic Regulation

Agni governs the transformation of ingested food into usable tissue and the elimination of metabolic byproducts; its balance is considered a precondition for *Dosha* equilibrium. Four

functional states are described: *Vishama Agni* (irregular digestive fire), *Tikshna Agni* (excessively sharp digestive fire), *Manda Agni* (weak digestive fire), and *Sama Agni* (balanced digestive fire). Weak or irregular *Agni* predisposes to the formation of *Ama* — a poorly digested, toxic metabolic residue that classical texts implicate directly in *Dosha* vitiation and channel obstruction (*Srotorodha*), including obstruction of the *Rakta* and urinary (*Mutravaha*) channels through which uric acid is conceptually eliminated.[7,9]

From this standpoint, Ayurvedic dietary advice for hyperuricemia is not restricted to avoiding high-purine foods; it equally targets restoration of *Sama Agni* through appropriately spiced, warm, freshly prepared meals, avoidance of *Viruddha Bhojana* (incompatible food combination), and regular meal timing. This *Agni*-centered framing offers a plausible mechanistic bridge to modern observations that impaired insulin sensitivity and sluggish hepatic and renal metabolic handling — themselves downstream of poor dietary quality — are closely tied to elevated serum urate.[16,18]

7. *Ritucharya*: Seasonal Dietary Regimen

Ayurveda divides the year into six seasons (*Ritu*) — Shishira (late winter), Vasanta (spring), Grishma (summer), Varsha (monsoon), Sharad (autumn), and Hemant (early winter) — each associated with characteristic accumulation, aggravation, or pacification of specific *Doshas*, and each carrying its own dietary counsel (*Ritucharya*).[10,13]

Two seasons are of particular relevance to *Vatarakta* and, by extension, to hyperuricemia management. Varsha *Ritu* is classically associated with *Vata* accumulation due to variable digestive strength and damp, cool weather; light, warm, freshly cooked, minimally fermented food is advised, with strict avoidance of stagnant water sources and raw leafy vegetables that are more liable to microbial contamination during this period. Sharad *Ritu*, immediately following, is the season in which accumulated *Pitta* becomes provoked and *Rakta Dhatu* is considered most vulnerable to vitiation — precisely the pathological axis of *Vatarakta*. Classical physicians therefore recommend seasonal moderation of sour, salty, and pungent tastes, avoidance of direct sun exposure and *Ushna Ahara* (excessively hot food), and preferential use of sweet, bitter, and astringent, cooling foods such as barley, rice, ghee, Amalaki, and bitter gourd during Sharad.[10]

Applied to hyperuricemia, *Ritucharya* suggests that dietary vigilance need not be uniform year-round: patients with borderline or fluctuating serum urate may benefit from tightened

dietary discipline specifically during the Varsha-to-Sharad transition, when *Rakta Dushti* is constitutionally favored, complementing rather than replacing the constant, *Prakriti*-based baseline diet.

8. Rationale for Exclusive Dietary Management

Dietary correction occupies the foundation of Ayurvedic therapeutics because food is regarded as the primary determinant of both health and disease. Because asymptomatic hyperuricemia represents an early, functionally reversible metabolic disturbance without structural joint damage, this stage offers a genuine window in which dietary correction alone may restore equilibrium before symptomatic gout develops.[6,17]

Modern evidence corroborates this window: excess purine intake, fructose-sweetened beverages, alcohol, obesity, and insulin resistance all independently raise serum urate by increasing production or reducing renal clearance,[1,25] and interventional studies show that weight reduction, low-purine diets, restriction of fructose and alcohol, and regular physical activity measurably lower serum urate and improve broader metabolic markers.[17,26] This is functionally identical to the Ayurvedic principle of *Nidana Parivarjana* — removing the causative factor as the first act of treatment.[7] By replacing *Apathya Ahara* with *Pathya Ahara*, calibrated to *Prakriti*, *Agni*, and *Ritucharya*, Ayurveda aims to restore *Dosha* equilibrium, strengthen digestion, protect *Rakta Dhatu*, and normalize waste elimination — collectively addressing the causal chain of hyperuricemia rather than only its biochemical marker.[7,11] It is this root-cause orientation, rather than any claim of superiority over pharmacotherapy in overt disease, that underlies the review's emphasis on the "exclusive" role of diet during the asymptomatic phase specifically.

9. Dietary Regimens in Ayurveda for Hyperuricemia

Ayurvedic dietary management follows a dual strategy: systematically incorporating *Pathya Ahara* that support *Agni*, *Dosha* balance, and *Rakta Shuddhi* (blood purification), while eliminating *Apathya Ahara* that drive purine load, *Ama* formation, or *Rakta Dushti*. Table 1 consolidates the recommendations cross-referenced across *Charaka Samhita*, *Sushruta Samhita*, *Bhaishajya Ratnavali*, and *Yogaratnakara*.[7,9,10,12,27]

Table 1. Classical Pathya–Apathya Ahara for Vatarakta, applied to dietary management of asymptomatic hyperuricemia

***Pathya* (Wholesome)**	***Apathya* (Unwholesome)**
Purana Yava (aged barley)	*Masha / urad* (black gram)
Godhuma (wheat)	*Kulattha* (horse gram)
Nivara and Shashtika Shali (rice varieties)	*Nishpava* (flat beans)
Yusha of Shimbi Dhanya with Ghrita (pulse soup with ghee)	*Kalaya* (garden pea)
Adhaki (pigeon pea)	*Kshara* (alkaline salts)
Chanaka (chickpea)	*Anupa Mamsa* (meat of marshy/aquatic animals)
Masura (lentil)	*Viruddha Bhojana* (incompatible combinations)
Makushthaka (moth bean)	*Dadhi* (curd)
Mulaka (radish, cooked)	*Ikshu Vikara* (sugarcane products)
Sunishnaka (Marsilea minuta)	*Madira* (alcohol)
Kakamachi (black nightshade)	*Kanji* (fermented sour gruel)
Tila (sesame, moderate)	*Excess Katu, Lavana, Amla Rasa* (pungent, salty, sour tastes)
Shatavari (Asparagus racemosus)	*Abhishyandi Ahara* (obstructive, sticky foods)
Vastuka (Chenopodium)	*Ushna Ahara* (excessively hot foods)
Upodika (tender greens)	*Guru Ahara* (heavy-to-digest foods)
Patola (pointed gourd)	*Divaswapna* (day sleep)
Karavellaka (bitter gourd)	*Prajagarana* (night vigil)
Kushmanda (ash gourd)	
Amalaki (Indian gooseberry)	
Draksha (grapes/raisins)	
Adraka (fresh ginger)	
Naveena Ghrita (fresh ghee)	
Go Dugdha (cow's milk)	
Meat of gallinaceous/small/pecker birds, in light soup	
Abhyanga, Parisheka, Pradeha (external oleation and fomentation therapies)	

9.1 Correspondence with Modern High-Purine and Metabolic Risk Foods

Several *Apathya* categories map directly onto recognized modern risk factors. Marsh/aquatic-animal meat (Anupa Mamsa), dried meat (Shushka Mamsa), and pulses such as black gram (Masha) and horse gram (Kulattha) correspond to high-purine protein sources; alcohol (Madira) and fermented sour gruel (Kanji) correspond to alcohol and fermented beverages known to raise urate and impair clearance; curd (Dadhi) and excess sesame (Tila) correspond to heavy, obstructive foods that slow digestion and promote *Ama*; and sugarcane products (Ikshu Vikara) parallels the well-documented fructose–uric acid relationship.[3,25] *Viruddha Ahara* (such as milk with sour fruit or fish with milk) has no precise biomedical equivalent but is traditionally associated with impaired digestion and toxin accumulation, broadly consonant with the modern emphasis on food-pattern quality over single-nutrient analysis.

9.2 Notable *Pathya* Items with Emerging Pharmacological Support

Amalaki (Emblica officinalis) is rich in vitamin C and polyphenols and has shown uricosuric and antioxidant activity in preliminary pharmacological studies; related Terminalia species have demonstrated measurable reductions in serum urate in a controlled pilot trial.[14] Barley (Purana Yava) and ash gourd (Kushmanda) are low-purine, alkalizing foods traditionally favored for their diuretic and *Vata-Pitta* pacifying properties. Ghrita, used in moderation with pulse preparations, is proposed in classical texts to aid *Agni* and reduce the Abhishyandi quality of legumes when cooked together — an example of Ayurvedic food-combination logic that merits further biochemical characterization.

10. Hydration and Waste Elimination

From a modern medical perspective, adequate hydration is important for maintaining normal kidney function and supporting the body's removal of waste products. Water helps the kidneys excrete uric acid more efficiently, which can reduce the chance of uric acid crystals forming[15].

11. Discussion

This section considers the significance of the evidence presented above, situates Ayurvedic dietary management alongside conventional pharmacological approaches, and identifies the limitations of the current evidence base and priorities for future research.

11.1 Comparison with Pharmacological Management

Pharmacological management remains central once hyperuricemia becomes symptomatic, but is not without drawbacks that strengthen the case for early dietary intervention. Xanthine oxidase inhibitors such as allopurinol and febuxostat reduce urate synthesis but carry risks: allopurinol can precipitate hypersensitivity reactions, including Stevens–Johnson syndrome, and gastrointestinal disturbance,[28] while febuxostat has been linked to increased cardiovascular risk in comparative trials.[29] Uricosuric agents such as probenecid enhance renal urate excretion but can predispose to uric acid nephrolithiasis through the resulting excretory load.[30]

Beyond direct adverse effects, sustained pharmacotherapy imposes a cumulative economic burden on patients and health systems, an issue that is especially acute where insurance coverage or drug access is limited. Dietary intervention, by contrast, carries no comparable pharmacological risk profile, is comparatively low-cost, and confers ancillary benefits to overall metabolic and cardiovascular health rather than targeting a single biochemical marker in isolation — though its success depends heavily on sustained patient adherence, which itself requires structured counselling and follow-up.

11.2 Strengths and Limitations of the Review

Several limitations qualify the conclusions above. First, this is a narrative rather than systematic review; source selection, while broad, was not governed by a pre-registered protocol or formal risk-of-bias assessment, introducing potential selection bias. Second, classical Ayurvedic texts were composed over many centuries by different authors, and terminology and specific food identifications (particularly botanical equivalents of Sanskrit plant names) vary across commentaries and translations, which can affect the precision of *Pathya–Apathya* correspondence. Third, direct clinical trial evidence testing whole Ayurvedic dietary protocols against serum urate endpoints in humans remains sparse; much of the supporting biomedical evidence addresses individual foods, nutrients, or lifestyle factors rather than the integrated dietary system described here. Fourth, asymptomatic hyperuricemia itself lacks a single internationally agreed management threshold, which complicates direct comparison across the classical and biomedical literatures cited. These gaps do not undermine the biological plausibility of the framework but do constrain any claim of proven clinical efficacy at this stage.

11.3 Future Directions

Priority should be given to well-designed randomized controlled trials comparing standardized Ayurvedic *Pathya*-based dietary protocols against conventional low-purine diets and/or usual care, with serum urate, inflammatory markers, and renal function as outcomes. *Prakriti*-stratified trial designs would allow direct testing of whether constitutional individualization improves outcomes relative to a uniform diet. Mechanistic studies isolating the uricosuric, anti-inflammatory, and antioxidant activity of specific *Pathya* items (Amalaki, barley, ash gourd, and ghee-based pulse preparations) would help translate classical reasoning into pharmacologically grounded recommendations. Finally, integrative clinical pathways that combine dietary counselling, *Ritucharya*-based seasonal adjustment, and conventional biochemical monitoring merit pilot evaluation in primary-care and rheumatology settings.

12. Conclusion

Asymptomatic hyperuricemia represents a reversible, early metabolic disturbance and a genuine opportunity for preventive intervention before the onset of gout and its systemic complications. Ayurveda offers a comprehensive, individualized dietary framework grounded in *Pathya Ahara*, *Nidana Parivarjana*, *Prakriti*-based planning, and *Ritucharya*, whose classical description of *Vatarakta* closely parallels the recognized dietary and lifestyle determinants of hyperuricemia.

This framework extends beyond simple purine restriction to address digestive efficiency (*Agni*), *Dosha* equilibrium, tissue protection (*Dhatu Samyata*), and metabolic waste elimination (*Mala Nirharana*), and several of its specific dietary recommendations find partial support in contemporary pharmacological and nutritional research. As a low-cost, low-risk, and patient-centered non-pharmacological strategy, Ayurvedic dietary management holds meaningful potential for the prevention and long-term control of asymptomatic hyperuricemia. Realizing this potential will require rigorously designed clinical trials to standardize dietary protocols, confirm efficacy against serum urate and downstream clinical outcomes, and support integration into evidence-based preventive care.

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Conflict of interest

The authors declare no conflict of interest.

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