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Ayurvedic Perspective on Hyperuricemia: Linking Classical Concepts with Contemporary Evidence - A Review Article

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ABSTRACT

Hyperuricemia is an increasingly prevalent metabolic disorder characterized by elevated serum uric acid levels exceeding 6.8 mg/dl and is a major risk factor for gout, renal disorders, and cardiovascular diseases. Although hyperuricemia is well defined in modern medicine as a disorder of purine metabolism or impaired renal excretion of uric acid, there is no direct disease entity described in Ayurveda corresponding to hyperuricemia. Hence, understanding this condition through Ayurvedic principles requires an analysis of *Dosha, Dhatu, Srotas*, and *Agni* involved in its pathogenesis.

This conceptual review attempts to elucidate the applied physiology and etiopathogenesis of hyperuricemia in the light of classical Ayurvedic principles. A detailed review of Ayurvedic literature was undertaken to interpret hyperuricemia from the perspective of *Panchamahabhuta* predominance, *Agni Vaigunya*, and *Dosha–Dushya Sammurchana*. Parallels have been drawn between modern concepts of uric acid metabolism and Ayurvedic descriptions of *Vidagdha Pitta, Rakta Dushti*, and *Vata Prakopa*, particularly in relation to *Purvarupa* of *Pittadhika Vatarakta*.

Hyperuricemia, especially in its asymptomatic stage, is proposed to represent an early stage of *Samprapti* rather than a distinct *Vyadhi*, functioning as a precursor for metabolic disorders such as gouty arthritis. The review aims to throw a light on ayurvedic understanding of hyperuricemia. Understanding hyperuricemia through this integrative approach may help in early diagnosis and formulation of preventive and therapeutic strategies based on Ayurvedic principles.

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INTRODUCTION

Hyperuricemia, defined as serum uric acid levels exceeding 6.8 mg/dl,[1] is a metabolic disorder that predisposes individuals to gout, renal dysfunction, cardiovascular diseases, and other non-communicable disorders[2]. In modern medicine, hyperuricemia is primarily attributed to altered purine metabolism, resulting either from increased uric acid production or decreased renal excretion[3]. Despite its clinical importance, hyperuricemia often remains asymptomatic in its early stages and is detected incidentally during laboratory investigations

Recent epidemiological evidence highlights the growing global burden of hyperuricemia. A systematic review published in February 2025 estimated the worldwide prevalence of hyperuricemia to be 13.85%, indicating a substantial and rising public health concern[4]. The increasing prevalence over the past two decades has been largely attributed to lifestyle modifications, including excessive intake of purine-rich foods, red meat, alcohol, fructose-containing products, sedentary habits, and the rising incidence of obesity, insulin resistance, and metabolic syndrome[2]. Genetic predisposition and long-term use of medications such as diuretics further contribute to the development of hyperuricemia.

The pathophysiology of hyperuricemia extends beyond crystal deposition, involving oxidative stress, endothelial dysfunction, and progressive end-organ

damage[5]. Emerging evidence suggests that molecular and vascular alterations begin even before serum uric acid reaches the conventional diagnostic cutoff, challenging the earlier notion that asymptomatic hyperuricemia is a benign condition. Consequently, hyperuricemia is now increasingly recognized as a metabolic and vascular disorder rather than merely a precursor to crystal deposition, emphasizing the need for early identification and intervention.[2]

Conventional management primarily relies on xanthine oxidase inhibitors and uricosuric agents. However, long-term pharmacotherapy is often associated with inadequate response, risk of severe allergic hypersensitivity reactions, and the necessity for prolonged drug use, thereby increasing the likelihood of adverse effects[6]. These limitations underscore the importance of preventive strategies and safe therapeutic alternatives, particularly during the asymptomatic stage of the disease.

Ayurveda does not describe hyperuricemia as a distinct Vyadhi; however, it offers a comprehensive framework to interpret such metabolic disturbances through the principles of *Agni, Dosha, Dhatu, Mala,* and *Srotas*. Conditions representing early metabolic derangements without overt clinical manifestations are explained as stages of Samprapti, especially Purvarupa[7]. From this perspective, hyperuricemia can be conceptualized as an early pathological stage predisposing to diseases like *Vatarakta* specifically *Pittadhika Vatarakta.*

This review attempts to systematically interpret hyperuricemia by correlating modern biochemical understanding of uric acid metabolism with Ayurvedic concepts such as *Panchamahabhuta* predominance, *Agni Vaigunya, Dosha dushti* and *Rakta Dushti*. Such an integrative approach may facilitate early diagnosis, preventive intervention, and the development of safer therapeutic strategies rooted in Ayurvedic principles.

AIM AND OBJECTIVES

To critically analyse hyperuricemia from an Ayurvedic perspective and establish a probable conceptual understanding and correlation with contemporary biomedical insights**.**

MATERIALS AND METHODS

This is a conceptual study where literature search was performed using various classical Ayurveda text books, Modern Medical text books and Published articles. Logical explanations were given by critically evaluating the *Dosha, Dhathu* and *Srotas* involved in understanding the *Samprapti* of the condition and hence could arrive at a probable correlation of hyperuricemia in Ayurveda.

DISCUSSION

Hyperuricemia, defined as elevated serum uric acid levels, can be meaningfully interpreted through Ayurveda only after establishing the Ayurvedic perspective of uric acid.

Panchamahabhoutika analysis of Uric acid

From a modern perspective, uric acid is an acidic, relatively insoluble metabolic end product with a tendency to crystallize when present in excess[8,9]. When analyzed through *Panchamahabhuta Siddhanta*, its acidic and reactive nature indicates predominance of *Agni Mahabhuta*, reflected by *Ushna* and *Teekshna* properties[10]. Its dryness corresponds to *Vayu Mahabhuta*, characterized by *Ruksha Guna*[10]. Furthermore, the crystallization tendency of uric acid suggests *Prithvi Mahabhuta* predominance in its solidified state[10].

This *Panchabhautika* composition explains why dietary and lifestyle factors possessing *Agni-Vayu* predominance (*Amla, Lavana, Vidahi Ahara*, alcohol, excessive exertion) promote increased uric acid production through *Sajathiya Guna Vrddhi*[11]. Thus, modern biochemical characteristics of uric acid align closely with Ayurvedic *Mahabhuta* theory.

Physiological aspects of Uric acid – *Prakrita Pitta* analogy

In normal physiology, uric acid functions as a potent antioxidant, scavenging free radicals and protecting tissues from oxidative damage[12]. Modern science recognizes this protective role at physiological levels.

In Ayurveda, similar protective and metabolic regulatory functions are attributed to *Prakruta Pitta* and *Agni*, which maintain normal *Paka, Varna, Prabha*, and cellular metabolism. Since uric acid exhibits *Agneya Bhuta* predominance and antioxidant activity, its physiological state can be conceptually correlated with one aspect of *Prakruta Pitta Bhava* in the body[13].

Pathological aspects of Uric acid – *Vikruta Pitta* and *Vidagdha Avastha*

When uric acid levels increase beyond physiological limits, it shifts from a protective antioxidant to a pathological pro-oxidant, leading to oxidative stress, endothelial dysfunction, inflammation, and crystal deposition[12]. Excessive oxidative activity mirrors the pathological state caused by aggravated *Pitta*, where increased *Daha* (burning sensation), *Raga* and *Paka* (inflammation) are observed[13]. *Agneya bhuta* dominant substances in an abnormal state is responsible for *Daha* and *Paka* which resembles well with oxidative stress and inflammatory changes caused by excess uric acid. This can be correlated well with the *Vikrta* or *Vidagdha Pitta bhava* in

Sarira. Thus, pathological hyperuricemia can be correlated with *Vidagdha Amla Pitta Pradhana Mala*, particularly when associated with *Rakta Dushti*.

To understand the Pathological role of uric acid in detail, the role *of Agni, Dosha* and *Dushya* should be understood which will be discussed below:

Agni and Uric acid production – Modern and Ayurvedic parallels

Although uric acid is not explicitly mentioned in Ayurvedic literature, still the synthesis can be conceptually understood as the byproduct of *Agnipaka,* which parallels the modern biochemical view of uric acid as the metabolic waste product of purine degradation.

While modern medicine attributes the production of uric acid, the end product of purine metabolism, to both exogenously from dietary sources and endogenously within the body, a parallel conceptual framework can be constructed using the principles of Ayurveda. This model integrates the functions of different forms of *Agni* to explain the multifaceted origins of uric acid.

Exogenous Uric acid production

Modern medicine explains exogenous uric acid formation through intestinal digestion of dietary purines, followed by enzymatic degradation via xanthine oxidase. Initially, nucleic acids are broken down into purine bases by proteolytic enzymes, followed by their conversion through the action of xanthine oxidase, leading to the formation of uric acid[1,12]. Ayurveda explains a similar process through Jataragni and Bhutagni, where continuous intake of *Pitta*-aggravating *Nidana* produces *Vidagdha Pitta Pradhana Ahara Rasa* during second stage of *Avasthapaka (Amla Avasthapaka)* in *Pachyamana Asaya*[13]. This metabolized product *(Vidagdha Ahara Rasa)* subsequently enters *Rasa* and *Rakta Dhatu*, resulting in formation of *Vidagdha Pitta Pradhana Mala* in *Rakta*. This end product may be considered as *Agni* and *Vayu Bhuta* predominant due to its *Ushna Teekshna Ruksha* qualities[10]. The uric acid, possessing properties analogous to *Agni- Vayu Bhuta* predominance, can thus be correlated in Ayurveda to *Pitta Pradhana Mala Sanchaya in Rakta*, produced by the combined action of *Jataragni, Bhutagni* and *Dhatwagni* as part of exogenous metabolism.

Endogenous Uric acid production

Endogenous uric acid production parallels *Rakta Dhatwagni Vaigunya*. When *Rakta* is directly vitiated by *Nidana*, impaired *Dhatwagni* leads to accumulation of *Vidagdha Amla Pitta Pradhana Mala* within *Rakta*. This explains hyperuricemia independent of dietary purine intake, similar to endogenous overproduction described in modern medicine.

Uric acid as *Mala* – Ayurvedic interpretation

The word *Mala* in Ayurveda refers to either the by product or end product of metabolism that maintains normal physiology when properly eliminated but causes pathology when accumulated[14]. Uric acid, being a terminal metabolic product of purine metabolism[3], fits this definition. Under normal conditions, it is excreted efficiently. Excess production or impaired elimination results in *Vidagdha Amla Pitta Pradhana Mala Sanchaya* in *Rakta*, corresponding to hyperuricemia.

Hyperuricemia as *Purvarupa* of *Pittadhika Vatarakta*

Hyperuricemia lacks overt clinical manifestations in its early stages, similar to *Purvarupa Avastha* in Ayurveda. The *Nidana* of *Pittadhika Vatarakta* such as *Amla, Lavana, Vidahi Ahara*, alcohol, sedentary lifestyle, and obesity closely resemble etiological factors of hyperuricemia. This establishes hyperuricemia as an initial stage of *Pittadhika Vatarakta* rather than a separate disease entity.

Correlation of *Nidana* of *Pittadhika Vatarakta* and Etiology of Hyperuricemia

***PITTADHIKA VATARAKTA NIDANA*[15]**	**CAUSES OF HYPERURICEMIA**
Amla	Excess intake of certain sour foods particularly those high in fructose such as beer, sugary sodas[16]
Shaka	Excessive intake of purine rich vegetarian food items like spinach, cauliflower, mushroom etc[17]
Kulatha, Masha, Nishpava	Legumes like dried beans, peas and other pulses can also increase the uric acid level by excessive consumption[17]
Anoopa Mamsa, Ambuja Mamsa	Nonvegetarian food especially seafood like Salmon, Mackerel, Sardine, Tuna and red meat etc are purine rich[17]
Aranala, Sauvira, Sura, Sukta, Asava, Madya	fermented drinks, excessive alcohol consumption[16]
Anashna, Langhana	The starvation like condition can result in oxidative stress.

Pramada	Intoxication, i.e., any chronic poisoning like lead poisoning. The continuous intake of certain drugs like aspirin, diuretics also predisposes hyperuricemia[16].
Ikshu, Mishtanabhojana	Fructose rich diet such as fructose rich sweetened soda[16]
Achamkramana seeli	Sedentary lifestyle[19]
Ativyayama, Ativyavaya	Excessive muscular exertion, by doing heavy work.[19]
Staulya	Obesity[16]
Rogaischa Atipeedita	Hyperuricemia is mainly associated with many other comorbidities like hypertension, metabolic syndrome, psoriasis, cancer.[20]

Dosha, Dhatu, and _Srotas_ involved in hyperuricemia

In order to understand Hyperuricemia in Ayurvedic view, the *Dosha, Dhathu and Srotas* involved in the Pathogenesis should be understood. By analysing the above etilogy of hyperuricemia we can infer that it can directly vitiate *Pitta, Rakta* and can lead to *Vataprakopa.*

Pitta – In *Pittadhika Vatarakta* the *Nidana* mentioned are mainly *Pitta Prakopa* due to the *Ushna Teekshna* and Amla Swabhava. This *Nidana* correlate well with some of the causative factors of Hyperuricemia, suggesting that the etiological factors of hyperuricemia lead to *Pitta* aggravation. In *Pittadhika Vatarakta, Vidagdha* Amla *Pitta Pradhana Mala Sanchaya* occurs at the *Rakta Dhathu* level. Since similar *Nidanas* are observed in the pathogenesis of hyperuricemia, the end product formed as a result of impaired metabolism i.e., uric acid can be correlated with *Vidagdha Pitta Pradhana Mala Sanchaya.* So, *Pitta* can be considered as main dosha involved in hyperuricemia. The production of Uric acid can be interpreted as outcome of enzymatic activities and these activities may be considered to be under the domain of pitta as one of its primary functions is *Pakthi* or *Pachana.*

Rakta- *Rakta Dhatu* can be considered as the main pathological entity in hyperuricemia. The very factors that cause an increase in uric acid, such as excessive intake of meat, alcohol, and salty or sour foods, are also known to vitiate *Rakta.* Among these, *Lavana* is predominantly composed of *Agni* and *Jala Bhuta*, while *Amla* is *Agni* and *Prthvi Bhuta* predominant. Consequently, such *Nidana* can directly render the *Rakta, Amla Vidagdha Visra* and *Kleda Bahula*. Given that *Rakta, Pitta* and uric acid share a similar *Agni Bhuta* predominance, they possess a strong affinity, making *Rakta* the primary site of pathology and accumulation.

Vata- *Vata Dosha* can be considered as an associated or *Anubandha Dosha*, playing a significant role in the progression of the condition. The proper functioning of *Vata* is essential for digestion and metabolism in the body and a similar role can be inferred in uric acid metabolism. When *Samana Vata becomes vitiated due to Vata Prakopa Nidana,* the proper *Sara Kitta Vibhajana* does not takes place, hence accumulation of *Kitta* occurs. At *Rakta Dhatu* level, due to *Pitta Prakopa* and *Rakta Prakopa Nidana Sevana*, the *Kitta Bhaga of Rakta Dhatu* increases, resulting in production of *Vidagdha Pitta Pradhana Mala*. *Vyana Vata* is responsible for the circulation of all metabolites including uric acid. Here *Vidagdha Amla Kleda Bahula Rakta* causes obstruction to the normal pathway of *Vata* and hence *Rukshatha, Chala* and *Sukshma Guna* of *Vata* increase and does the *Soshana* of *Rakta*. The vitiated *Rakta* is then carried throughout the body by *Asukari* action of *Vata*. During circulation, *Vata* does the *Soshana* of *Rakta* which can be considered as crystallisation of uric acid which becomes deposited in the joints. This parallels the pathological process of symptomatic hyperuricemia, where uric acid crystals get accumulate in the periarticular soft tissues and joints and cause pain. Thus, a disturbance in function of *Vata* contributes to the improper handling and accumulation of uric acid.

Upadhatu of Rakta- When *Vidagdha Amla Pitta Pradhana Mala Sanchaya* manifests in Rakta, it can lead to the involvement of *Kandara* and *Sira*, the *Upadhatu* of *Rakta*[21], thereby rendering them susceptible to pathological changes. This involvement manifests clinically in symptoms such as *Stambha* (stiffness),*Nistoda, Sopha*[21]. From a modern perspective, a comparable mechanism is observed when blood becomes supersaturated with uric acid beyond 6.8 mg/dl, resulting in crystallization and deposition not only within the joints but also in periarticular structures such as tendons, ligaments, and bursae[22]. Thus, the Ayurvedic understanding of *Kandara* and *Sira* involvement in this pathology correlates well with the modern pathophysiological explanation of hyperuricemia and its periarticular manifestations.

Srotas- The *Nidana* described in *Pittadhika Vata Rakta* are directly responsible for *Raktavaha Sroto Dushti*, indicating that the primary *Srotas* involved is *Raktavaha*. Considering the modern view, uric acid is produced in

liver due to activity of xanthine oxidase enzyme. In Ayurveda, the *Yakrt* (liver) is described as one of the *Moola* of *Rakta vaha Srotas*. Due to the influence of *Nidana,* the metabolic by product formed becomes lodged in *Raktavaha Srotas*. This concept closely aligns with pathophysiology of hyperuricemia wherein the byproduct of purine metabolism when produced in excess, accumulates in blood. Therefore, *Raktha Vaha Srotas* can be considered the prime site for both production and accumulation of uric acid. When the condition progresses and involves *Utharothara Dhatu*, other *Srotas* will also get involved in the disease progression and manifestation.

Correlating the *Purvarupa* of *Pittadhika Vatarakta* and Hyperuricemia

Hyperuricemia is usually asymptomatic in its early stages and is often detected incidentally through laboratory investigations. Despite the absence of evident clinical manifestations, disturbances in digestion and metabolism can be observed, reflecting altered purine metabolism and the subsequent accumulation of urate in the blood.

From an Ayurvedic perspective, the physiological and pathological aspects of uric acid metabolism and the *Samprapti Ghataka* involved in hyperuricemia suggest its correlation with *Pittadhika Vatarakta*, particularly in the stage of *Purvarupa*, as the complete manifestation of the disease is not yet evident. Several *Purvarupa* described in *Vatarakta* are observed in hyperuricemic individuals, such as Sopha, Nistoda in *Hasta-Pada-Anga Sandhi, Swedadhikya, Alasya*, and *Gaurava*[23]. These symptoms indicate impaired metabolism consistent with early pathological changes.

In this condition, *Sthanasamshraya* first occurs in the *Rakta Dhatu*, and subsequently, its *Upadhatu* become involved in the pathogenesis. This leads to clinical features such as pain and swelling in the joints, which are the sites of *Kandara* and *Sira.*

Therefore, considering the pathological entities observed in hyperuricemia, it may be broadly understood in Ayurveda as the *Purvarupa (*initial stage) of *Pittadhika Vatarakta*, since the complete clinical picture of the disease has not yet manifested.

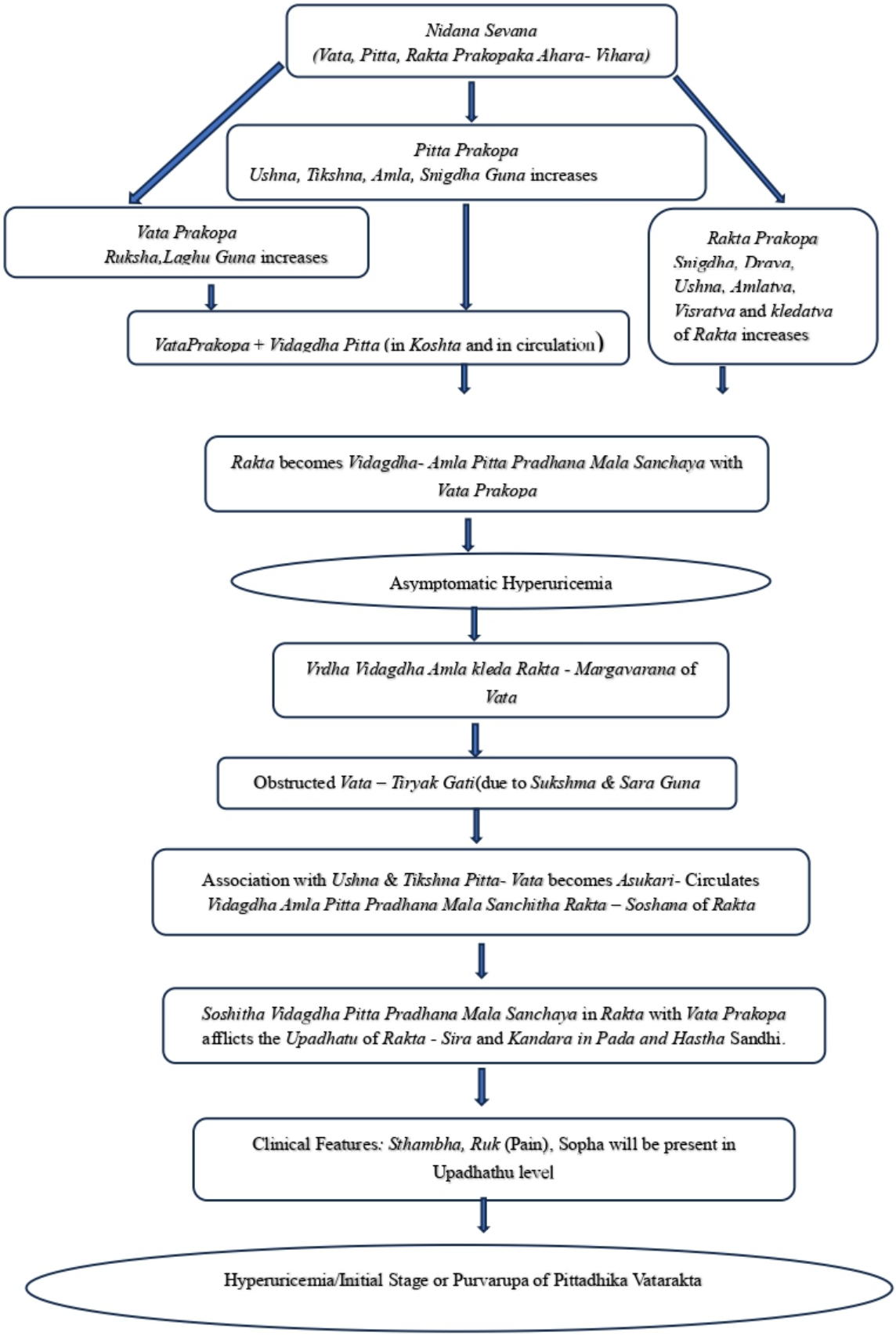
Progression to *Rupa* and *Vyakta Avastha*

As hyperuricemia progresses, uric acid crystals deposit in joints and periarticular tissues, producing classical symptoms of gout. This corresponds to *Vyakta Avastha* of *Pittadhika Vatarakta*[24] with appearance of characteristic clinical features such as pain, swelling, stiffness and tenderness exactly in *Asthi Sandhi*, corresponds with documented findings in literature, thereby confirming the correlation of hyperuricemia in its progressive stage with *Pittadhika Vatarakta.* Uric acid deposition is not limited to the joints but may also occur in sites such as the kidneys and vascular endothelium, thereby impairing renal function, cardiac health, and normal circulation. From an Ayurvedic perspective, this progression can be correlated with the involvement of *Uttarottara Dhatus* such as *Meda* (renal calculi) and *Asthi-Sandhi* (gouty arthritis) in the advanced stages of the disease process.

***Samprapti* of Hyperuricemia**

By analysing the *Doṣa, Dhatu, Srotas, Nidana*, site of *Sthana Saṃsraya*, and *Vyaktavastha*, the probable *Samprapti* of hyperuricemia is depicted in Chart No.1.

Chart No.1: *Samprapti* of Hyperuricemia



CONCLUSION

Hyperuricemia is not described as a distinct disease entity in Ayurveda; however, its physiological and pathological aspects can be comprehensively understood through Ayurvedic principles. Uric acid can be interpreted as an *Agni-Vayu Bhuta* predominant *Mala*, which in its normal state exhibits characteristics comparable to *Prakruta Pitta*, while in excess manifests as *Vidagdha Amla Pitta Pradhana Mala Sanchaya* in *Rakta*.

Hyperuricemia represents an early stage of *Samprapti*, corresponding to the *Purvarupa* of *Pittadhika Vatarakta. Agni Vaigunya* at *Jataragni, Bhutagni,* and *Dhatwagni* levels, along with *Rakta Dushti, Pitta Prakopa*, and *Vata Anubandha,* plays a central role in its pathogenesis. Progressive involvement of *Rakta Upadhatu* and *Uttara-Uttara Dhatus* explains the transition from asymptomatic hyperuricemia to symptomatic gout and systemic complications.

Therefore, the Ayurvedic approach to hyperuricemia should focus on *Agni Deepana, Rakta Prasadana, Pitta-Vata Shamana*, and prevention of *Mala Sanchaya*, emphasizing early intervention to prevent disease progression.

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