ISSN: 2322 - 0902 (P)
ISSN: 2322 - 0910 (O)

International Journal of Ayurveda and Pharma Research

Review Article

CONCEPTUALISING NEPHROTIC SYNDROME THROUGH AYURVEDIC PRINCIPLES: AN INTEGRATIVE REVIEW

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Article info

Article History:
Received: 25-06-2026
Accepted: 22-07-2026
Published: 15-08-2026

KEYWORDS:
Agnimandya, Kleda Vriddhi, Mutravaha Srotas, Nephrotic Syndrome, *Sopha, Vrikka Vikara*.

ABSTRACT

Nephrotic syndrome is a clinicopathological disorder characterized by massive proteinuria, hypoalbuminemia, generalised oedema, and hyperlipidaemia resulting from increased glomerular permeability. The condition may arise secondary to a wide range of primary glomerular diseases, systemic disorders, infections, drugs, and genetic abnormalities. Clinically, it presents with generalised oedema, urinary abnormalities, dyslipidaemia, and increased susceptibility to infections, significantly affecting the quality of life and long-term prognosis of affected individuals. From an Ayurvedic perspective, the condition can be understood through the concepts of *Agnimandya, Ama, Tridosha Dushti, Kleda Vriddhi, Mutravaha Srotodushti, Dhatu Dushti, Vrikka Karmahani,* and *Oja Kshaya*. The pathological process primarily involves the *Vrikka* and *Mutravaha Srotas* and progresses through different stages with varying *Dosha-Dushya* involvement. This review critically analyses the etiopathogenesis, clinical features, and diagnostic aspects of nephrotic syndrome from contemporary and Ayurvedic perspectives and proposes a stage-wise Ayurvedic understanding of *Samprapti*, thereby providing a holistic understanding of the disease.

INTRODUCTION

Nephrotic syndrome (NS) is a clinical disorder characterised by massive proteinuria that leads to hypoalbuminemia, subsequently resulting in hyperlipidaemia, oedema, and multiple systemic complications.[1] It arises from increased permeability of the glomerular filtration barrier, particularly due to damage to the glomerular basement membrane and podocytes, which may occur secondary to infections, thromboembolic events, or other pathological conditions. Nephrotic syndrome represents one of the major clinical manifestations of glomerulonephritis in both children and adults and is primarily associated with podocyte injury.[2] The condition develops as a consequence of altered glomerular permeability caused either by primary renal diseases confined to the kidney or secondary to systemic disorders such as congenital infections, diabetes mellitus, systemic lupus erythematosus, malignancies, or exposure to certain drugs. [3]

Access this article online

Quick Response Code

https://doi.org/10.47070/ijapr.v14i8.4283

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The glomerular filtration barrier consists of fenestrated endothelial cells, the glomerular basement membrane, and podocytes connected by slit diaphragms, which normally prevent the filtration of large plasma proteins such as albumin.[4] Structural or functional disruption of any of these components increases glomerular permeability, leading to excessive urinary protein excretion exceeding 3.5 g per day, which is the hallmark feature of nephrotic syndrome. [5]

Nephrotic syndrome is generally classified into primary forms, in which the pathology is restricted to the kidney, and secondary forms, in which the syndrome occurs as a complication of systemic diseases such as diabetes mellitus, systemic lupus erythematosus, infections, malignancies, or certain medications.[6] The most common primary causes include minimal change disease, focal segmental glomerulosclerosis, and membranous nephropathy. [7]

The loss of plasma proteins, particularly albumin, results in a reduction in plasma oncotic pressure, promoting the movement of fluid from the intravascular compartment into the interstitial space

and leading to the development of oedema.[8] Additionally, compensatory hepatic synthesis of lipoproteins increases in response to hypoproteinaemia, resulting in hyperlipidaemia and other lipid abnormalities.[9] Nephrotic syndrome is also associated with several complications, including increased susceptibility to infections, thromboembolic events due to a hypercoagulable state, and progressive decline in renal function, emphasising the importance of early diagnosis and timely management.[10]

Nephrotic syndrome can affect individuals of all ages; however, the underlying causes vary with age. Minimal change disease is more commonly observed in children, whereas membranous nephropathy and focal segmental glomerulosclerosis are more frequently reported in adults.[11] A thorough understanding of the underlying pathophysiological mechanisms is essential for accurate diagnosis and the development of effective therapeutic interventions.

Epidemiology

Nephrotic syndrome is a common glomerular disorder affecting both children and adults worldwide, with an annual incidence of 2–7 per 100,000 children and approximately 3 per 100,000 adults.[12,13] It occurs most frequently in children aged 2–6 years and shows a slight male predominance.[14] In India, it represents a major paediatric kidney disorder, with minimal change disease being the predominant cause in children, while focal segmental glomerulosclerosis and membranous nephropathy are increasingly observed in older age groups.[15,16] Regional epidemiological variations are influenced by socioeconomic, environmental, and systemic disease-related factors.[17]

Etiology

The aetiology of nephrotic syndrome is broadly classified into primary (idiopathic) and secondary causes.

Primary causes

Primary nephrotic syndrome arises from intrinsic glomerular diseases causing podocyte injury and increased glomerular permeability. Common causes include minimal change disease, focal segmental glomerulosclerosis, membranous nephropathy, and membranoproliferative glomerulo-nephritis, which disrupt the glomerular filtration barrier and result in proteinuria.[18] Minimal change disease is the predominant cause in children, whereas focal segmental glomerulosclerosis and membranous nephropathy are more common in adults. [19]

Secondary causes

Secondary nephrotic syndrome occurs due to systemic disorders that increase glomerular permeability, including metabolic diseases (diabetes mellitus, amyloidosis), autoimmune disorders (systemic lupus erythematosus, vasculitis), infections, drugs, malignancies, and genetic abnormalities affecting podocyte proteins such as nephrin and podocin. These conditions cause immune-mediated, metabolic, infectious, or toxic injury to the glomerular filtration barrier, resulting in massive proteinuria.[1,3]

Pathophysiology

Nephrotic syndrome develops due to structural and functional damage to the glomerular filtration barrier, composed of fenestrated endothelial cells, the glomerular basement membrane, and podocytes with slit diaphragms. These structures normally restrict the passage of large plasma proteins such as albumin. Injury to podocytes or disruption of the basement membrane increases glomerular permeability, leading to massive proteinuria.[1,20] Loss of albumin leads to hypoalbuminemia and decreased plasma oncotic pressure, resulting in oedema. Reduced protein levels also stimulate hepatic lipoprotein synthesis, leading to hyperlipidaemia.[21] Long-standing proteinuria is associated with significant complications such as a hypercoagulable state, recurrent infections, and progressive impairment of kidney function, potentially leading to chronic kidney disease and, in advanced cases, renal failure.[22] Nephrotic syndrome is often considered a podocytopathy, characterised by alterations in slit diaphragm proteins such as nephrin and podocin, cytoskeletal disruption, and foot process effacement, leading to impaired filtration barrier integrity and protein leakage into urine.[23]

Clinical features

Nephrotic syndrome commonly presents with oedema, initially observed as periorbital swelling and progressing to generalised oedema due to hypoalbuminemia.[1] Patients typically exhibit massive proteinuria, frothy urine, weight gain, fatigue, hyperlipidaemia, and lipiduria.[24] Loss of immunoglobulins increases susceptibility to infections, while loss of anticoagulant proteins predisposes to thromboembolic complications.[25] In prolonged cases, progressive renal dysfunction and chronic kidney disease may occur

Diagnosis

The diagnosis of nephrotic syndrome is based on characteristic clinical and laboratory findings. It is typically defined by heavy proteinuria (≥3.5gm per 24 hours), hypoalbuminemia, oedema, and hyperlipidaemia. Urinalysis and blood investigations confirm protein loss, reduced serum albumin, and elevated lipid levels, while additional tests help identify underlying causes.[26,18] Renal biopsy, particularly in adults, is often required to establish the underlying histopathological diagnosis and guide management.[27]

Complications

Nephrotic syndrome may result in complications such as severe oedema, infections, thromboembolic events, persistent hyperlipidaemia, and progressive kidney damage leading to chronic kidney disease. Hypovolemia and malnutrition may also occur, particularly in severe or untreated cases.[28,29]

Ayurvedic perspective of nephrotic syndrome

From an Ayurvedic perspective, nephrotic syndrome involves the interplay of *Dosha*, *Dhatu*, *Agni*, *Ama*, and *Srotas*. The condition closely resembles the *Sopha* spectrum of disorders, with generalised oedema arising from *Kleda Vriddhi* and disturbed fluid metabolism, while urinary abnormalities indicate *Mutravaha Srotodushti*. Persistent loss of essential body constituents may lead to *Dhatu Kshaya* and *Oja Kshaya*, resulting in reduced vitality and increased susceptibility to illness. According to the concept of *Shatkriyakala,* the disease progresses through the stages of *Sanchaya* (accumulation) and *Prakopa* (aggravation) of *Doshas*, followed by *Prasara* (spread) through the *Srotas, Sthanasamshraya* (localisation) in susceptible sites such as the *Vrikka* and *Mutravaha Srotas*, *Vyakti* (manifestation) of clinical features including oedema and urinary abnormalities, and finally *Bheda* (complications) characterised by chronicity and progressive tissue damage. Furthermore, *Nidana Panchaka* offers a comprehensive diagnostic framework in Ayurveda by systematically describing the causative factors, disease mechanisms, clinical manifestations, prognosis, and therapeutic principles. Applying this framework to nephrotic syndrome enables an in-depth understanding of its origin, progression, and management from an Ayurvedic perspective.

Nidana Panchaka

In the assessment of nephrotic syndrome through *Nidana Panchaka*, the evaluation primarily begins with *Rupa* (clinical manifestations), as *Poorvarupa* (premonitory symptoms) are often *Avyakta* (indistinct or unmanifest) and may not be clearly identifiable. Based on the presenting *Lakshanas*, the clinical features can be categorised into different stages of disease progression. Careful assessment of the *Rupa*, along with detailed history taking, helps identify the probable *Nidanas* (etiological factors) responsible for the condition. Analysis of these *Nidanas* and *Rupa* facilitates the understanding of the underlying *Samprapti* (pathogenesis), *Dosha-Dushya* involvement, and the course of disease progression, thereby providing a comprehensive Ayurvedic perspective on nephrotic syndrome.

Roopa

1st stage

Features related to *Ama* formation, *Agnimandya*, and initial *Dosha* vitiation

- *Aruchi* (loss of appetite)
- *Gaurava* (feeling of heaviness)
- *Angamarda* (body ache)
- *Alasya* (lethargy)
- *Daurbalya* (easy fatigability)
- Mild *Sopha* (mild swelling of feet, mild facial or periorbital puffiness)
- *Klama* (fatigue)
- Mild *Mutra Vaikrti* (subtle urinary abnormalities)
- *Avipaka* (indigestion)

2nd stage

Clinical features resulting from *Kleda Vriddhi, Mutravaha Srotodushti,* and *Sopha*.

- *Sarvanga Sopha* – Generalised oedema
- *Akshi Sopha /Mukha Sopha* – Periorbital and facial oedema
- *Pada Sopha* – Pedal oedema
- *Gaurava* – Heaviness of the body
- Increased *Daurbalya* and *Alasya* – Weakness and easy fatigability, lethargy
- *Klama* – Fatigue and exhaustion
- *Aruchi* – Loss of appetite
- *Mutra Vaikruti* – Abnormalities in urine
- *Phenila Mutra* – Frothy urine

3rd stage

Features indicating *Dhatu Kshaya* and *Oja Kshaya*

- Severe generalised oedema with fluid accumulation.
- Marked *Daurbalya* (weakness)
- *Krishata*
- *Oja Kshaya Lakshanas* such as loss of strength, reduced vitality, and poor disease resistance.
- Recurrent infections
- Progressive impairment of renal function.
- Complications due to chronicity and long-standing disease.

Categorising *Lakshanas* into early, intermediate, and advanced stages provides a structured understanding of disease pathogenesis, progression, and severity. These stages, based on patient findings, enable a practical and clinically relevant approach for stage-specific diagnosis and management.

Nidana

A thorough evaluation of *Rupa*, combined with careful case taking and patient interrogation, facilitates

the identification of probable *Nidanas* (etiological factors) responsible for the condition.

***Aharaja Nidana* (Dietary Factors)**

- Excessive intake of *Guru Ahara* (heavy foods)
- Excessive *Snigdha Ahara* (unctuous and fatty foods)
- Excessive *Madhura* & *Lavana Rasa Sevana*
- *Abhishyandi Ahara*
- *Viruddha Ahara* (incompatible food combinations)
- *Adhyashana* (eating before digestion of the previous meal)
- Excessive intake of curd, dairy products, fried foods, and processed foods

***Viharaja Nidana* (Lifestyle Factors)**

- *Divaswapna* (day sleep)
- *Avyayama* (lack of physical activity)
- Sedentary lifestyle
- Suppression of natural urges, especially *Mutravega Dharana*

***Beeja* and *Sahaja* Factors**

- *Beeja Dushti* (hereditary or congenital nephrotic syndromes)

***Agantuja* Factors (External Causes)**

- Infections
- Drug-induced renal injury
- Autoimmune disorders
- Environmental toxins

***Manasika* factors**

- *Chinta* (excessive worry)
- *Shoka* (grief)
- *Bhaya* (fear)
- *Krodha* (anger)

Samprapti

In Ayurveda, *Samprapti* describes the evolving sequence through which *Doshic* imbalance progresses to overt disease. Each stage shows different degrees of *Dosha, Dushya*, and *Srotas* involvement, producing varied clinical presentations. Therefore, patients with nephrotic syndrome may present with different *Lakshanas* depending on the stage of the underlying pathology. For effective *Samprapti Vighatana*, the physician must keep the possible stage-wise *Samprapti* in mind for each patient, so that treatment can be tailored appropriately and disease progression can be arrested at the earliest feasible stage.

1st stage

Agni is the basis of digestion, absorption, and tissue nourishment, and its impairment (*Agnimandya*) is the root cause of many diseases. In nephrotic syndrome, *Jatharagni Mandya* leads to improper digestion and formation of *Ama*, which obstructs the *Srotas* and disturbs fluid balance. With progression, this *Ama*- often in a *Pitta-pradhana Ama Avastha*- may acquire *Amavisha* properties, circulate, and localize at *Khavaigunya*, leading to *Srotodushti*, *Tridosha involvement*, and progressive *Dhatvagni Mandya* affecting *Dhatus* from *Rasa* onwards. At the initial stage of *Dosha* accumulation, there is a *Pitta-pradhana* state where minute inflammatory changes begin at the *Vrikka* due to continued *Nidana Sevana*, producing subtle pathological changes. This stage lacks well-established *Dosha–Dushya* interaction but presents with mild prodromal features. It remains highly reversible, and timely *Nidana Parivarjana* can effectively halt further progression.

2nd stage

The pathological process of nephrotic syndrome involves progressive *Agnimandya, Ama* formation, *Dhatvagni* impairment, and *Srotodushti*, leading to sequential involvement of multiple *Dhatus*. *Rasa Dhatu* is predominantly affected, resulting in impaired *Dhatu Poshana* and systemic nutritional deficiency. As the disease progresses, *Rakta Dhatu* becomes secondarily involved due to the susceptibility of the *Vrikka* (*Rakta–Meda Prasadaja*), leading to further *Srotodushti*. Subsequent involvement of *Meda Dhatu* is reflected by hyperlipidaemia and excessive *Kleda*, thereby contributing to disease progression. At this stage, the clinical picture is predominantly *Shotha-pradhana* (edematous), with increased inflammatory changes due to predominant *Pitta* involvement, along with aggravated *Kapha* contributing to fluid retention and obstruction. *Vata* vitiation occurs secondary to *Srotorodha* caused by *Ama* and *Kapha*. Among its subtypes, *Apana Vata* is mainly affected, leading to impaired urinary regulation and disturbed fluid balance. Overall, the condition is characterised by *Pitta–Kapha pradhana Tridosha* vitiation, where *Pitta* intensifies inflammatory processes, *Kapha* contributes to *Ama, Kleda*, and obstruction, and *Vata* becomes secondarily involved, influencing disease expression through *Srotas* dysfunction.

3rd stage

In the third stage, persistent *Ama formation* and *Agnimandya* cause aggravated *Doshas* to lose site specificity and circulate through the *Srotas*, leading to widespread *Srotodushti* and systemic involvement. This results in increased *Kleda* and inflammatory activity, producing progressive oedema and functional disturbances. Ongoing *Dosha Dushti* leads to *Dhatvagni Mandya*, impairing proper *Dhatu* transformation and progressing to *Dhatu Paka* and *Dhatu Kshaya*, manifested as weakness, fatigue, reduced tissue strength, and further decline in vitality, culminating in *Oja Kshaya* with reduced *Vyadhikshamatva*. *Apana Vayu Vaigunya* causes disturbed urinary regulation and fluid imbalance, contributing to advanced clinical features of nephrotic syndrome. Progressive *Vrikka*

involvement due to *Dosha Dushti, Ama, Kleda Vriddhi*, and *Mutravaha Srotodushti* results in *Vrikka Karmahani*. This stage is characterised by *Vata–Pitta–Rakta pradhana Tridosha* vitiation, reflecting systemic spread and deeper structural-functional deterioration.

Upashaya -Anupashaya

Upashaya and *Anupashaya* in nephrotic syndrome are inferred from clinical response. Reduction in oedema, improved urine output, appetite, and strength indicate *Upashaya*, while worsening oedema, fluid retention, reduced urine output, and weakness indicate *Anupashaya*. Therapeutic measures that reduce *Dosha Dushti* and *Kleda*, improve *Agni*, maintain *Mutravaha Srotas* patency, and support *Dhatu* nourishment act as *Upashaya*, whereas factors that aggravate *Dosha Dushti*, impair *Agni*, increase *Kleda*, and cause *Srotorodha* lead to *Anupashaya*.

Therapeutic Considerations

The therapeutic approach in nephrotic syndrome should be individualized based on the stage of disease, dominant *Dosha–Dushya* involvement, *Srotas* affected, and the *Bala* of the patient. In the early stage dominated by *Agnimandya* and *Pitta-pradhana Ama* formation, management focuses on *Nidana Parivarjana*, *Pachana- Deepana*, and mild *Shamana* or *Shodhana* to restore *Agni* and prevent further *Ama* formation, thereby arresting progression at a reversible phase.

As the disease advances, the emphasis shifts to *Ama Pachana*, *Srotoshodhana*, *Pitta–Kapha Shamana*, restoration of *Mutravaha Srotas* function, and control of *Kleda* and inflammatory activity. In the advanced stage characterised by *Vrikka Karmahani*, *Sopha, Dhatu Kshaya,* and *Oja Kshaya*, management becomes supportive and restorative, focusing on *Balya*, *Rasayana*, *Dhatu Poshana*, and regulation of *Mutravaha* function to preserve residual renal capacity and prevent further deterioration. Special attention is required to correct *Vata–Pitta–Rakta* imbalance and stabilise systemic function.

Accordingly, therapeutic principles described for conditions such as *Mutraghata, Mutrakrichra, Adhogata Raktapitta, Shotha, Kaphodara,* and *Prameha* may be appropriately adopted based on clinical presentation.

CONCLUSION

Nephrotic syndrome is a multifactorial renal disorder characterised by oedema, proteinuria, hypoalbuminemia, and hyperlipidaemia. Its pathogenesis can be understood through the Ayurvedic concepts of *Agnimandya, Ama, Tridosha Dushti, Kleda Vriddhi, Mutravaha Srotodushti, Dhatu Dushti, Vrikka Karmahani,* and *Oja Kshaya*. The disease primarily affects the *Vrikka* and *Mutravaha Srotas*, leading to disturbances in fluid balance, tissue nourishment, and urinary function.

A stage-wise understanding of *Samprapti* is essential, as patients may present with varying clinical features depending on the extent of *Dosha, Dushya*, and *Srotas* involvement. This facilitates accurate *Samprapti Vighatana* and individualized management. Accordingly, treatment should focus on restoring *Agni*, eliminating *Ama*, correcting *Tridosha Dushti*, reducing *Kleda*, maintaining patency of *Srotas*, supporting *Dhatu Poshana*, preserving *Ojas*, and protecting *Vrikka* function to prevent disease progression and improve patient outcomes. Such a *Samprapti*-based approach provides a rational Ayurvedic framework for the comprehensive management of nephrotic syndrome.

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Cite this article as:
Anju S Jerad, P M Madhu. Conceptualising Nephrotic Syndrome Through Ayurvedic Principles. International Journal of Ayurveda and Pharma Research. 2026;14(8):156-161.
https://doi.org/10.47070/ijapr.v14i8.4283
Source of support: Nil, Conflict of interest: None Declared

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