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A CONCEPTUAL REVIEW ARTICLE ON *TAMAKA SHWASA* (BRONCHIAL ASTHMA)

Dr. Asmita Rani*[1], Prof. (Dr.) Ravi Sharma[2]

[1]MD Scholar, PG Department of Kayachikitsa, Madan Mohan Malviye Government Ayurved College and Hospital, Udaipur (Raj).

[2]Professor and Head of Department of Kayachikitsa, Madan Mohan Malviye Government Ayurved College and Hospital, Udaipur (Raj).

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***Corresponding Author**

Dr. Asmita Rani

MD Scholar, PG Department of Kayachikitsa, Madan Mohan Malviye Government Ayurved College and Hospital, Udaipur (Raj).

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ABSTRACT

Bronchial asthma remains one of the most prevalent chronic non-communicable respiratory conditions worldwide, and its long-term management with bronchodilators and corticosteroids continues to raise concerns around dose dependency and adverse effects. Ayurveda's classical description of *Tamaka Shwasa* offers a disease model built on *Vata-Kapha* vitiation within *Pranavaha Srotas* that maps in striking, though not exact, correspondence onto the modern understanding of reversible airway obstruction. This review revisits the classical construction of *Tamaka Shwasa* — its *Nidana*, *Samprapti*, *Purvarupa* and *Rupa* — and reads it against the extrinsic/intrinsic classification of bronchial asthma described in contemporary pulmonary medicine, with particular attention to where the two frameworks converge (a two-humor, biphasic model of airway narrowing) and where they diverge (the classical inclusion of *Pitta* at the site of origin versus the immunological IgE-mediated basis identified in modern pathology). The review argues that the value of this comparison lies less in establishing a one-to-one equivalence between the two systems and more in showing how each supplies an explanatory layer the other does not: Ayurveda's staged, causal *Kriyakala* model for tracing when in the disease course intervention is most effective, and modern immunology's account of the specific inflammatory mediators responsible for bronchoconstriction and remodeling.

KEYWORDS: *Tamaka Shwasa*, Bronchial Asthma, *Pranavaha Srotas*, Ayurveda integrative respiratory medicine.

INTRODUCTION

Respiratory disorders account for a disproportionate share of global morbidity, and among these, bronchial asthma is singled out by its episodic but recurring course, its tendency to begin early in life, and its capacity to erode a patient's quality of sleep and daily function over years rather than resolve outright. Contemporary estimates place the global burden in the hundreds of millions, with meaningfully higher prevalence in urban, pollutant-heavy environments than in rural ones[10,13] — a pattern that itself echoes an older observation in Ayurvedic nosology that environmental exposure (*Rajas* or dust, *Dhuma* or smoke, and adverse seasonal *Vata*) is not incidental to this disease but central to its onset.[1,2,3]

Among the five varieties of *Shwasa Roga* recognised in the classical texts — *Kshudra*, *Tamaka*, *Chinna*, *Maha* and *Urdhva Shwasa* — only *Tamaka Shwasa* is treated as a fully independent (*Swatantra*) disease entity with its own etiology, staged pathogenesis, and dedicated management protocol; the remaining varieties are either symptomatic of other conditions or represent terminal states.[1,2,3,4] It is this independent standing, together with its episodic, nocturnally-aggravated presentation, that has made *Tamaka Shwasa* the variety most consistently correlated with bronchial asthma in Ayurvedic literature.[5,6] This review re-examines that correlation, drawing on the classical treatises directly and situating the comparison against current pulmonary pathophysiology, rather than repeating the correlation as settled.

Bronchial asthma is a major global public health concern affecting individuals across all age groups. According to the Global Burden of Disease Study 2021, approximately 260.48 million people worldwide were living with asthma in 2021. India accounts for a substantial proportion of this disease burden, with an estimated 32.05 million prevalent cases in the same year. The prevalence of asthma varies considerably according to age, geographical location, environmental exposures, and the diagnostic criteria employed.[10,12] A systematic review and meta-analysis of 33 studies involving 167,626 children in India reported a pooled asthma prevalence of 7.9% (95% confidence interval: 6.3–9.6%). The prevalence reported in individual studies ranged from 2% to 18.2%, highlighting considerable variability across populations and study settings. These findings emphasize the importance of standardized epidemiological assessments, early diagnosis, and appropriate management strategies to

reduce the burden of bronchial asthma in India.[12,13]

Nomenclature and Disease Framing

Tamaka derives from a root indicating darkness or suffocation — the classical gloss *"Tamyati Iti Tamaka"* captures a patient's own description of an asthmatic attack: the sensation of the world narrowing into darkness as air is withheld.[1,4] *Shwasa* itself denotes the act of breathing, so the compound name is, in effect, a clinical description rather than an arbitrary label — a naming convention that is unusual among classical disease categories and suggests the condition was recognised and named from direct bedside observation of paroxysmal breathlessness, well before its internal mechanism was theorised.

The texts differ slightly on how severe a case must be before it is named as such. *Charaka* distinguishes two allied intensifications of the base condition — *Pratamaka Shwasa*, where fever and fainting accompany the breathlessness and where *Pitta* involvement is inferred from the fact that the patient obtains rapid relief from cooling measures, and *Santamaka Shwasa*, the more severe state in which the patient reports the sensation of drowning in darkness.[1] *Sushruta* and *Vagbhata* do not preserve this two-tier distinction, folding both presentations under the single term *Pratamaka*; and even within the Charaka tradition, the commentator Chakrapani reads the two as synonyms differing only in the intensity of a single attack rather than as separate clinical entities. This is a genuine point of internal disagreement in the classical corpus, not a settled taxonomy, and it is worth stating plainly rather than smoothing it over.[1,2,3]

Nidana — Causative and Precipitating Factors

The classical texts separate the causative factors of *Tamaka Shwasa* along dosha lines, and the resulting list is unusually granular for a single disease entity. Dietary causes are grouped by which dosha they provoke: dry, irregular and excessively gapped meals (*Rukshana*, *Vishamashana*, *Anasana*) aggravate *Vata*; sour, salty, hot and burning foods aggravate *Pitta*; and heavy-to-digest items — pulses such as *Masha* and *Nishpava*, curd, unboiled milk, and meat of aquatic or marshy animals — aggravate *Kapha*.[1,3,4] Behavioural and environmental causes overlap more heavily with what modern medicine would call triggers: exposure to dust and pollen (*Rajas*), smoke (*Dhuma*), cold wind and cold water, excessive exertion, and suppression of natural urges are named consistently across Charaka, Sushruta and Ashtanga Hridaya alike.[1,2,3]

A structurally important but easily overlooked category is disease-derived onset. *Tamaka Shwasa* is described not only as arising on its own from the exposures above (*Nidanottha*) but also as a sequel of other conditions — *Kshaya* (a wasting condition consistent with tuberculosis), *Panduroga* (anaemia), *Amatisara* and chronic constipation (*Vibandha*) are each named as antecedents (*Rogottha*).[1,3,4] This secondary pathway has a reasonably direct modern parallel: comorbid states such as gastro-oesophageal reflux, chronic sinusitis and nutritional deficiency are recognised triggers or aggravators of asthma control today, even though the underlying mechanism proposed for the link differs entirely between the two systems.[7,9]

Samprapti — Staged Pathogenesis

The classical description of pathogenesis is organised through the six-stage *Kriyakala* model, and applying it to *Tamaka Shwasa* gives the disease a temporal structure that a simple symptom list does not convey.[2,5] In the accumulation stage (*Sanchaya*), *Vata* gathers in *Pranavaha Srotas* while *Kapha* gathers separately in the chest (*Uras*). Seasonal and environmental triggers — cold weather, overcast conditions — provoke this accumulated dosha into the aggravation stage (*Prakopa*), at which point the vitiated humors begin to spread (*Prasara*), impairing digestive fire (*Agnimandhya*) and generating the toxic metabolic residue *Ama*. It is at the localisation stage (*Sthanasanshraya*) that the spreading dosha lodges specifically in a weakened respiratory channel (*Khavaigunya*), producing the first prodromal signs, before manifesting fully (*Vyakta*) as obstructed, reversed-direction *Vata* movement (*Pratiloma Gati*) — the mechanistic core of the disease, in which air that should move outward on expiration is instead held back by *Kapha*-driven obstruction.[1,3,6] Left untreated, the condition can progress to the final stage (*Bheda*), marked by lasting structural change in the channels — a concept that anticipates, in a very different vocabulary, the modern recognition of irreversible airway remodeling in chronic asthma.[15]

Nidana Sevana: *Raja* (Dust particles), *Dhuma* (Smoke), *Prag-Vatasevana, Marmaghata, Vata-Kapha Prakopaka Ahara Vihara* etc.[1,3]

Sanchayavastha: *Sanchaya* of *Vata* occurs in *Pranavaha Srotas* and that of *Kapha* in *Uras.*[1,3]

***Prakopavastha*:** Sheeta Kaala and Durdina etc. will provoke the *Sanchita Doshas*. As a result the *Vata Dosha* in *Pranavaha Srotas* and Kapha in Uras tries to move to other places.[1,3]

Purvarupa and Rupa — Prodrome and Clinical Presentation

Before the attack fully manifests, patients are described as experiencing abdominal distension and flatulence (*Anaha*), pain along both sides of the chest (*Parshwa Shoola*), a sensation of tightness over the cardiac region (*Peedanam Hridayasya*), and an early reversal of normal respiratory rhythm.[1,3,4] Once the attack is established, the clinical picture becomes considerably more specific and, notably, closely resembles a bedside description of an acute asthmatic episode as a modern clinician would chart it: rapid, laboured breathing with audible wheeze (*Ghurghurukama*), a cough that offers only partial and temporary relief once sputum is expectorated, pronounced discomfort on lying flat with corresponding ease when sitting upright (orthopnea, in modern terminology), sweating localised to the forehead, and, in severe paroxysms, a deterioration of consciousness.[1,2,3] The texts also note that cloudy, damp weather and cold water reliably worsen the attack — an observation about environmental triggering that survives essentially unchanged in modern clinical teaching on asthma exacerbation.[7,9]

Principles of Management

Classical management follows a sequence rather than a fixed prescription, and the sequencing itself carries clinical logic. *Nidana Parivarjana* — systematic avoidance of the identified causative factors — is treated as the non-negotiable first step, on the reasoning that continued exposure will simply re-aggravate the dosha regardless of what treatment follows.[1,2,3] For a robust patient with dominant *Kapha* and *Vata*, purificatory therapy (*Samsodhana*) is indicated, proceeding through oleation with salt-medicated oil (*Snehana*), sudation to loosen the channels (*Swedana*), therapeutic emesis to expel accumulated *Kapha* (*Vamana*), medicated smoke inhalation to reach residual dosha (*Dhumapana*), and, where indicated, purgation (*Virechana*). Notably, oil-based enema (*Sneha Basti*) is the one purificatory measure explicitly excluded — the *Yogaratnakara* names it as the sole exception among *Shodhana* procedures unsuitable for this condition.[1,3]

Patients too weak for purificatory therapy — including children and the elderly — are managed instead with palliative measures (*Samsamana*): digestive and carminative therapy (*Deepana*, *Pachana*) alongside drugs that pacify both *Kapha* and *Vata* together with agents that specifically clear *Pranavaha Srotas*.[1,3,5,6] Because recurrent attacks progressively deplete the patient's tissue strength (*Dhatukshaya*), the classical authors place unusual emphasis — more than for most other conditions — on restorative (*Brimhana*) and

rejuvenative (*Rasayana*) therapy administered between attacks, precisely because this chronic, relapsing course is what limits how aggressively a patient can be treated during any single acute episode.[1,3]

Recent Advances

The development and progression of bronchial asthma are influenced by multiple factors, including advancing age, exposure to biomass smoke, respiratory infections, female sex, atopic predisposition, tobacco smoke, occupational exposures, and dietary factors.[7,12,14] Although asthma has traditionally been regarded as a reversible airway disease, recent evidence suggests that persistent structural alterations may develop in the airways. These changes, collectively known as airway remodeling, include subepithelial fibrosis, submucosal gland hyperplasia, airway smooth muscle hypertrophy and hyperplasia, and increased vascularization. Airway remodeling contributes to disease progression, increased severity, persistent airflow limitation, and, in some individuals, partial irreversibility of airway obstruction.[15] Furthermore, chronic asthma has been associated with an accelerated decline in lung function over time.[7,9]

Asthma has also been reported frequently among young adults, particularly those aged 16–25 years, although its prevalence varies across populations and age groups.[12,18] Emerging evidence suggests a possible association between maternal thyroid dysfunction during pregnancy and an increased risk of asthma in offspring, with some studies indicating a greater risk when maternal thyroid dysfunction remains untreated.[16] Additionally, recent research has demonstrated an association between psychological conditions, particularly depression and anxiety, and poor asthma control.[17] These findings highlight the multifactorial nature of asthma and emphasize the importance of considering environmental, physiological, and psychological factors in understanding its pathogenesis and clinical outcomes.[16,17]

The Modern Disease Picture: Extrinsic, Intrinsic and Mixed Asthma

Contemporary pulmonary medicine defines bronchial asthma as a condition of heightened tracheobronchial reactivity producing reversible, widespread narrowing of the airways, and separates it clinically into three patterns.[7,9,10] Extrinsic (atopic) asthma, the more common presentation, typically begins in childhood, correlates with a personal or family history of allergic conditions such as rhinitis or eczema, and is driven by an IgE-mediated Type I hypersensitivity reaction with a recognisable two-phase course: an immediate response, in which mast-cell degranulation releases histamine and leukotrienes to cause

bronchoconstriction and mucus hypersecretion, followed by a delayed, more sustained late-phase reaction driven by recruited eosinophils and neutrophils that is largely responsible for the prolonged symptoms of an attack.[8,9] Intrinsic (non-atopic) asthma instead appears later in life, without a personal or family allergic history and with normal IgE levels, often following a viral upper respiratory infection, and carries its own recognised trigger — hypersensitivity to aspirin in a meaningful minority of patients.[7,9] A mixed pattern, showing features of both, is also common in practice.

Points of Convergence and Divergence

Where the two frameworks align

The strongest correspondence between the two systems is structural rather than mechanistic: both describe a disease driven by two interacting causal factors rather than one, and both describe it as unfolding in phases rather than appearing all at once. The classical two-dosha model (*Vata* obstructed by *Kapha*) and the modern two-phase immunological model (immediate mast-cell response followed by a delayed leukocyte-driven phase) are different explanatory systems, but both insist on the same clinical fact: that an asthma attack is not a single event but a build-up followed by a more prolonged secondary process, which is why relief on expectoration is only partial and temporary in both descriptions.[1,7,9] Environmental triggering also aligns closely — cold, damp, and particulate exposure feature in both accounts as reliable precipitants rather than incidental associations.[3,10] And the classical warning that unmanaged, recurrent disease produces permanent channel changes (*Bheda*) anticipates, without the underlying cell biology, the modern finding that chronic asthma produces genuine structural remodeling — subepithelial fibrosis, smooth-muscle hyperplasia, and neovascularisation — that is not fully reversible.[3,15]

Where they diverge

The most important divergence is at the level of origin. The classical model treats the disturbance as arising within the digestive tract (*Amashaya* or *Pittasthana*) and travelling to the chest, and correspondingly gives dietary regulation and digestive strength a causal role that has no direct equivalent in the immunological account, which instead locates the disease's origin in airway-resident immune cells and circulating antibody.[1,3,9] This is not a minor terminological difference — it changes where each system directs primary prevention: the classical tradition toward diet, digestion and constitutional strength, modern medicine toward allergen avoidance and immune modulation.[3,10] Equally, the classical corpus does

not distinguish an allergic from a non-allergic subtype in the way modern medicine does; the extrinsic/intrinsic split has no clean classical parallel, and forcing one onto the *Pratamaka*/*Santamaka* distinction (which tracks severity, not immunological trigger) would overstate the correspondence.[1,7] These are not failures of either system so much as evidence that they were built to answer different questions — one organised around observable pattern and causal sequence for the purpose of guiding treatment, the other around cellular and molecular mechanism for the purpose of targeted pharmacology.[8,9,11]

DISCUSSION

Read together, the classical and modern descriptions of this disease are best understood as complementary rather than translatable one-for-one. The *Kriyakala* framework's real contribution is its insistence that intervention should be matched to disease stage — that avoidance and constitutional strengthening belong at the earliest stages, purification at the stage of active aggravation, and restorative therapy in the interval between attacks specifically because repeated exacerbation is understood to deplete the patient rather than leave them unchanged.[1,2,3] This staged logic is arguably under-emphasised in standard step-wise pharmacological asthma management, which tends to escalate or de-escalate medication by symptom severity at a point in time rather than by where the patient sits in a longer arc of disease progression and tissue depletion.[10,11]

At the same time, the classical model's explanatory limits should not be understated for the sake of a tidy comparison. It has no mechanism for explaining why one patient develops an allergic pattern from childhood while another develops the disease later without any allergic history — a distinction that materially changes modern treatment choice and one that *Pitta*-based reasoning about *Pratamaka Shwasa* does not capture.[1,7,9] An integrative approach, rather than asserting equivalence, should treat the classical framework as a guide to timing, constitutional management, and relapse prevention, and the modern framework as the more reliable guide to acute pharmacological intervention and to distinguishing clinical subtypes that carry different long-term risk.[9,10]

CONCLUSION

Tamaka Shwasa and bronchial asthma describe recognisably the same clinical phenomenon — episodic, nocturnally-worsened, wheeze-associated breathlessness with a chronic, relapsing course — through two independently developed explanatory systems.[1,3,7,9] The correspondence is real at the level of clinical description, staged progression, and

environmental triggering, but it is partial at the level of mechanism, and treating the two as fully interchangeable would understate what each has to offer on its own terms. The more useful reading is one that draws on the classical model's staged, preventive logic for long-term disease management and on modern immunology's mechanistic precision for acute treatment and subtype-specific care — an integration that respects the boundaries of what each system was actually built to explain.

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