

KAMPAVATA: A CRITICAL REVIEW OF AYURVEDIC AND MODERN CONCEPTS

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ABSTRACT

Kampavata is a neurological disorder described in *Ayurvedic* literature under the category of *Vata Vyadhi*. The condition is mainly characterized by tremors, rigidity, slowness of movement, disturbed posture, and impairment of voluntary motor activities due to aggravated *Vata Dosha*. Classical *Ayurvedic* texts describe various manifestations of *Kampavata* and explain its pathological basis through aggravation, *Dhatukshaya*, and *Avarana*. The clinical presentation of *Kampavata* closely resembles Parkinson's Disease, a progressive neuro-degenerative disorder affecting the extrapyramidal motor system. Parkinson's Disease is associated with degeneration of dopaminergic neurons and deficiency of dopamine, resulting in tremors, rigidity, bradykinesia, and postural instability. Ayurveda offers a holistic therapeutic approach for *Kampavata* through *Shamana Chikitsa*, *Shodhana* procedures, *Panchakarma* therapies, and *Rasayana* management aimed at pacifying *Vata Dosha* and improving neurological functioning. Understanding the *Nidana*, *Lakshanas*, *Samprapti*, and *Chikitsa* of *Kampavata* is important for proper diagnosis and management. This review highlights the *Ayurvedic* and modern perspective of *Kampavata* and its correlation with Parkinson's Disease.

KEYWORDS: *Kampavata*, *Panchakarma*, *Dhatukshaya*, *Chikitsa*, *Vata Vyadhi*.**INTRODUCTION**

Ayurveda explains neurological disorders primarily under the broad category of *Vata Vyadhi*, where disturbance of *Vata Dosha* affects normal neuromuscular functioning. *Kampavata* is one such condition characterized mainly by involuntary tremors, stiffness, slowness of movement, and impairment of motor activities. The disease has clinical similarities with Parkinson's Disease described in contemporary medicine.

Among the three *Doshas*, *Vata* governs all voluntary and involuntary movements, coordination, sensory-motor activities, and higher neurological functions. Any disturbance in its normal functioning may lead to disorders affecting movement and posture. Classical *Ayurvedic* texts describe manifestations such as *Kampa* (tremors), *Stambha* (rigidity), and *Chestha Hani*

(difficulty in movements), which resemble the clinical presentation of Parkinsonism.

Different *Ayurvedic* scholars have discussed symptoms related to *Kampavata* under various contexts. *Acharya Charaka* described *Vepathu* among *Vata* disorders^[1], while later texts such as *Madhava Nidana* and *Basvarajiyam* elaborated the disease with characteristic symptoms including *Sarvanga Kampa*, *Karapada Tala Kampa*, *Nidrabhanga*, and *Deha Bhramana*.^[2-3] These descriptions provide an important foundation for understanding *Kampavata* from both classical and modern perspectives.

Parkinson's Disease is a chronic neurodegenerative disorder involving progressive impairment of dopaminergic neurons, resulting in tremors, rigidity, bradykinesia, and postural instability. Despite advances in modern symptomatic management, long term

treatment remains challenging. *Ayurvedic* management aims not only at symptomatic relief but also at correction of *Doshic* imbalance, nourishment of tissues, and improvement in functional capacity.

AIM AND OBJECTIVE

1. To understand the concept of *Kampavata* according to Ayurveda and modern medicine.
2. To understand aetiopathogenesis of *Kampavata* and Parkinson's Disease.

MATERIAL AND METHODS

A literary review was conducted using classical Ayurvedic texts such as *Charaka Samhita*, *Sushruta Samhita*, *Madhava Nidana*, *Basvarajiyam*. Modern medical literature regarding Parkinson's Disease is also reviewed.

NIDANA^[4]

According to the concept of *Vata Vyadhi Utpatti Hetu Nidana*, etiological factors are responsible for the manifestation of disease. Classical Ayurvedic texts do not describe any specific *Nidana* exclusively for *Kampavata*. However, Acharyas have considered the general causative factors of *Vata Vyadhi* as responsible for the development of *Kampavata* (*Vepathu*). These include excessive intake of light and cold food (*Ati Laghu and Sheeta Ahara*), inadequate food consumption (*Alpana*), excessive sexual activity (*Ativyavata*), irregular therapeutic measures (*Vishamopachara*), excessive blood loss (*Ati Asruka Sravana*), overexertion (*Aticheshta*), depletion of body tissue (*Dhatu Kshaya*), emaciation due to chronic disease (*Roga Atikarshana*), trauma to vital organs (*Marmaghata*), and other forms of injury (*Abhighata*). Although classical texts such as *Madhava Nidana* and *Basvarajiyam* provide additional description of *Kampavata*, They also emphasize that the general etiological factors of *Vata* disorders are the primary causative factors for *Kampavata*.

HETU^[5]

Sannikrishta Hetu

The immediate causative factors can be grouped into the following categories:

- Suppression of natural urges such as *Mutra* (urination), *Udgara* (belching), and *Jrumbha* (yawning).
- Trauma or injury to the *Krukatika Marma*.
- Complications due to excessive or improper therapeutic procedures.
- Intake of *Vata* aggravating diet and lifestyle factors (*Vata Prakopa Ahara-Vihara*).

Viprakrishta Hetu

- Excessive consumption of foods having *Kashaya* (astringent), *Katu* (pungent), and *Tikta* (bitter) tastes.
- Intake of substances possessing *Ruksha* (dry), *Sheeta* (cold), and *Laghu* (light) qualities.
- Improper dietary habits such as *Alpahara*, *Vishmashana*, and *Adhyashana*.

- Psychological factors such as *Chinta* (stress), *Shoka* (grief), *Krodha* (anger), and *Bhaya* (fear).

PURVAROOPA

Purvaroopa refers to the early warning signs that appear before the complete manifestation of a disease. These symptoms arise during the fourth stage of *Kriya Kala* known as *Sthana Samsraya*.^[6] Early recognition is important because timely intervention may prevent disease progression and structural damage.

According to *Charaka Samhita*, these symptoms are considered *Avyakta Lakshanas* of *Vata* disorders.^[7]

The Purvaroopa of Kampavata include

- *Angamarda*- generalized body ache
- *Udvega*- anxiety
- *Anavasthita Chittatva*- unstable mind
- *Moha*- confusion
- *Smriti Hani*- memory impairment
- *Aswastha Mana*- irritability
- *Gatraruka*- localized pain
- *Avasada*- nervousness or restlessness
- *Supti*- numbness or paresthesia
- *Ushna Pratiti*- sensation of warmth
- *Klama*- fatigue

These mild manifestations reflect the initial aggravation of *Vata* and, when detected at an early stage, may aid in the timely diagnosis and improved management of *Kampavata*.

ROOPA^[8]

करपादतले कम्पो देहभ्रमणदुःखिते ।

निद्राभङ्गो मतिः क्षीणा कम्पवातस्यलक्षणम्॥

(*Basvarajiyam*)

The clinical features of *Kampavata* include:

- *Karapada Tale Kampa*- tremors in hands and feet
- *Deha Bhramana*- body instability or whirling sensation
- *Nidrabhanga*- disturbed sleep
- *Kshina Mati*- dementia

Others associated symptoms include:

- *Stambha*- rigidity
- *Cheshta Hani*- slowness of movement
- *Vak Vikriti*- speech disturbances

These symptoms can also be observed in other *Vata Vyadhi* conditions.

SAMPRAPTI

According to *Charaka Samhita*, exposure to various etiological factors causes aggravation of *Vata Dosha*. The aggravated *Vata* occupies weakened or empty channels (*Rikta Srotas*) and produces different types of *Vata* disorders affecting either the entire body or specific body parts.^[9]

The aggravation of *Vata* mainly occurs through two mechanisms:

1. *Dhatukshaya* (tissue depletion)
2. *Avarana* (obstruction of bodily channels)^[10]

SAMPRAPTI GHATAKA

- **Dosha**- Predominantly *Vata* along with *Kapha*
- **Dushya**- *Rasa*, *Rakta*, *Mamsa*, *Meda*, *Majja*, and *Snayu*

CHIKITSA

Shamana Chikitsa^[11]

Following drugs are mentioned in different classics for treatment of *Kampavata*: -

Sr. No.	Drugs	Samhita
1.	<i>Ksheerbala Taila</i>	<i>Ashtanga Hridaya</i>
2.	<i>Nakuladhya Ghrita</i>	<i>Bhaishajya Ratnavali</i>
3.	<i>Brihat Chagaladi Ghrita</i>	<i>Bhaishajya Ratnavali</i>
4.	<i>Nakula Taila</i>	<i>Bhaishajya Ratnavali</i>
5.	<i>Mahanarayana Taila</i>	<i>Bhaishajya Ratnavali</i>
6.	<i>Rasna Taila</i>	<i>Bhela Samhita</i>
7.	<i>Mahamasha Taila</i>	<i>Chakradatta Samhita</i>
8.	<i>Nakula Taila</i>	<i>Harita Samhita</i>
9.	<i>Sarvang Kampa Rasa</i>	<i>Rasa Ratnakara</i>
10.	<i>Triguna Rasa</i>	<i>Sahasra Yoga</i>
11.	<i>Sahacharadi Taila</i>	<i>Sahasra Yoga</i>
12.	<i>Varuni Taila</i>	<i>Sharangadhara Samhita</i>
13.	<i>Dhatturadi Taila</i>	<i>Sharangadhara Samhita</i>
14.	<i>Mashadi Taila</i>	<i>Sharangadhara Samhita</i>
15.	<i>Maharasnadi Taila</i>	<i>Sharangadhara Samhita</i>
16.	<i>Devdarvadi Taila</i>	<i>Sharangadhara Samhita</i>
17.	<i>Masha Taila</i>	<i>Vangasena Samhita</i>

Shodhana Chikitsa^[12]

वायुं वेपथुनामानं स्वेदाभ्यङ्गानुवासनैः।

उपाचरेन्निरुहैश्च शिरोबस्ति विरेचनम्॥

(*Vangasena Samhita*)

According to *Acharya Vangasana*, therapies like *Swedana*, *Abhyanga*, *Anuvasana Basti*, *Niruha Basti*, *Shirobasti*, and *Virechana* are indicated in *Kampavata Roga*.

PATHYA^[13]

Cereals: *Yava* (barley), *Kulattha* (horse gram), *Kodrava*, *Rakta Shali*, and well-aged *Shashtika Shali* rice.

Vegetables: *Vastuka*, *Shigru* (drumstick), *Karella* (bitter gourd), *Patola* (pointed gourd), *Surana* (elephant foot yam), and *Kakamachi*.

Fruits: *Darsa*, *Kushmanda* (ash gourd), and *Amalaki* (Indian gooseberry).

Dairy Products: Cow's milk, Goat milk, Buffalo milk, Medicated buttermilk, and Garlic-processed buttermilk.

Meat of Birds: Quail, Partridge, Pigeon, Sparrow.

Beverage: Warm water.

APATHYA

Curd, *Mastu* (whey), Jaggery, Excessive milk, *Masha* (Black gram), *Viruddha Ahara* (incompatible food

• **Strotas**- *Rasavaha*, *Raktavaha*, *Mamsavaha*, *Medovaha*, and *Majjavaha*

• **Srotodushti**- *Sanga*, *Vimarga*, *Gamana*, and *Atipravritti*

• **Agni**-*Mandagni*

• **Udhbhava Sthana**- *Aamashaya* and *Pakvashaya*

combinations), *Asatmya* *Bhojana* (unsuitable/unwholesome foods), *Vishamasana* (irregular eating habits), *Abhishyandi*, *Guru*, and *Picchila dravyas* (Uncooked meat, and heavy, slimy, or channel obstructing foods).

Lifestyle Practices: *Viruddha Cheshta* (Incompatible physical activities), *Vegavarodha* (suppression of natural urges), and *Ratri Jagarana* (Night awakening).

PARKINSON'S DISEASE

Parkinson's Disease is a slowly progressive neurological disorder that primarily affects body movements and muscular coordination. The condition develops due to degeneration of neurons responsible for dopamine production, especially within the substantia nigra region of the brain. Reduction in dopamine levels interferes with smooth execution of motor activities and gradually produces characteristic movement-related abnormalities.

The disease commonly affects elderly individuals and progresses over time with varying severity among patients. Motor manifestations generally include resting tremors, muscular rigidity, slowness of voluntary movements, difficulty in maintaining posture, and gait disturbances, mood changes, autonomic dysfunction, and cognitive impairment.

From a clinical perspective, Parkinson's Disease significantly affects daily activities and quality of life. The disease progression is usually gradual, beginning with mild unilateral symptoms and later involving bilateral motor impairment. Although modern medicine offers symptomatic improvement through dopaminergic therapy, it does not completely halt neuronal degeneration.

AETIOPATHOGENESIS^[14]

The pathogenesis of Parkinson's Disease involves progressive degeneration of dopaminergic neurons located in the substantia nigra pars compacta of the mid brain. These neurons form part of the nigrostriatal pathway, which plays an essential role in regulating voluntary movements and muscular coordination.

Loss of dopamine results in altered neurotransmitter balance within the basal ganglia, leading to impaired motor control. Clinical manifestations generally become evident only after substantial neuronal loss has occurred. This explains the gradual onset and progressive nature of the disease.

Several factors have been implicated in the development of Parkinson's Disease, including aging, oxidative stress, mitochondrial dysfunction, environmental toxins, and genetic predisposition. Histopathological findings commonly include degeneration of pigmented neurons and formation of Lewy bodies within affected neural tissues.

The combined effect of neuronal degeneration and neurotransmitter imbalance ultimately produces tremors, rigidity, bradykinesia, and postural instability seen in affected individual.

CLINICAL FEATURES^[15]

General Features

- Expressionless face (Hypomimia)
- Stooped posture
- Soft and indistinct speech (Dysphonia)

Gait Disturbances

- Difficulty initiated walking
- Short and rapid steps (Festinating gait)
- Impaired balance while turning

Tremors

- **Resting Tremors**- Frequency of 3-4 Hz
Commonly begins in one hand ("Pill-rolling tremor")

May involve jaw and legs

Present at rest and reduced during movement

- **Postural Tremors**- Present while stretching the arms.
- **Re-emergent Tremors**- Tremors reappear after maintaining posture for few seconds

Rigidity

- Cogwheel rigidity
- Lead-pipe rigidity

Akinesia

- Slowness of movement
- Decreased amplitude of movement

DIAGNOSIS^[16]

The diagnosis of Parkinson's Disease remains primarily clinical, as there is currently no definitive test available to confirm the disease during life in most cases. PD is characterized clinically by parkinsonism and pathologically by the degeneration of dopaminergic neurons in the substantia nigra pars compacta, along with the presence of intraneuronal lewy bodies.

Clinical diagnosis is based on the identification of the cardinal motor features, including bradykinesia, rigidity, resting tremor, and postural instability. Although several diagnostic criteria have been proposed, no universally accepted standard exists. A positive response to levodopa or other dopaminergic therapies is considered an important supportive features for diagnosis.

By the time Parkinson's Disease is diagnosed using clinical criteria, substantial loss of dopaminergic neurons in the substantia nigra and a significant reduction in striatal dopamine levels have usually already occurred.

Neuroimaging techniques can assist in diagnosis and differentiation. Positron emission and differentiation. Positron emission tomography (PET) using 18F-dopa and other ligands can distinguish idiopathic Parkinson's Disease from other parkinsonian disorders in approximately 80% of cases, although its availability is limited. Magnetic resonance imaging (MRI) is particularly useful in identifying Parkinson-plus syndromes and has become an increasingly important tool in neurological diagnostics. Additional investigations, including genetic testing, olfactory assessment, dopamine transporter single-photon emission computed tomography (DAT-SPECT), computed tomography (CT), and MRI, may provide supportive evidence and aid clinical decision making.

TREATMENT^[17]

Early Treatment

Patients with early Parkinson's Disease may be considered for treatment with:

- Levodopa plus dopa decarboxylase inhibitor
- Oral or transdermal dopamine agonists
- Monoamine oxidase B inhibitors

Note-Ergot derived dopamine agonists should not be used as first line treatment.

Anticholinergic drugs should not be used as first line treatment.

Motor Fluctuations

- Motor fluctuations may be treated with either monoamine oxidase B inhibitors or dopamine agonists.
- Dopamine agonists may be used to manage motor complications in patients with advanced Parkinson's Disease. The non-ergot agonists are preferable to the ergot agonists.
- Intermittent or infusion of subcutaneous apomorphine may be used to manage advanced Parkinson's Disease.
- Catechol-O-methyl-transferase (COMT) inhibitors may be used to reduce off-time in patients with advanced Parkinson's Disease.

Surgery^[18]

The three different mechanisms of surgical response for Parkinson's Disease are:

Ablative surgery, stimulation surgery or deep brain stimulation (DBS), and transplantation or restorative surgery. Target areas for DBS or lesions include the thalamus, the globus pallidus, or the subthalamic nucleus.

- Deep brain stimulation: it is helpful in tremors.
- Advocated balance diet
- Rehabilitative approaches like physical exercise, gait exercise, auditory exercise and so on.
- Speech therapy
- Occupational therapy
- Musical therapy
- Palliative care.

DISCUSSION

Kampavata is described in *Ayurveda* under *Vata Vyadhi* and shows close resemblance with Parkinson's Disease in terms of tremors, rigidity, bradykinesia, postural instability, and impaired movements. Aggravated *Vata Dosha*, particularly due to *Dhatukshaya* and *Avarana*, plays a major role in its pathogenesis. Degeneration of dopaminergic neurons and dopamine deficiency in Parkinson's Disease can be correlated with *Vata* dysfunction affecting neuromuscular activities.

Ayurvedic management focuses on pacifying *Vata Dosha*, nourishing tissues, and improving motor functions through therapies like *Abhyanga*, *Swedana*, *Basti*, *Shirobasti*, and *Rasayana*. Along with *pathya ahara* and lifestyle modifications, *Ayurveda* offers a holistic approach for symptomatic relief and better quality of life.

CONCLUSION

Kampavata can be correlated with Parkinson's Disease based on similarities in clinical features and disease progression. *Ayurveda* explains the condition through *Vata* aggravation, *Dhatukshaya*, and *Avarana*, while modern medicine attributes it to degeneration of dopaminergic neurons. *Ayurvedic* therapies including *Panchakarma*, *Shamana*, and *Rasayana* may help in reducing symptoms, slowing disease progression, and improving functional capacity. Further clinical studies

are needed to validate the effectiveness of *Ayurvedic* management in *Kampavata*.

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