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

International Journal of Ayurveda and Pharma Research

Review Article

PAKSHAGHATA IN AYURVEDA: A CRITICAL NARRATIVE REVIEW OF CLASSICAL DESCRIPTIONS, CLINICAL CORRELATES, AND CONTEMPORARY EVIDENCE FOR STROKE REHABILITATION

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Article info

Article History:
Received: 19-09-2026
Accepted: 25-10-2026
Published: 17-11-2026

KEYWORDS:
Pakshaghata, Hemiplegia, Ayurveda, Stroke Rehabilitation, *Panchakarma*, Narrative Review.

ABSTRACT

Pakshaghata is a classical Ayurvedic description of predominantly unilateral functional impairment that overlaps clinically with hemiplegia but cannot independently identify stroke subtype. **Objective:** To examine the classical descriptions, critically synthesize contemporary clinical research on Ayurveda-based stroke rehabilitation, and identify safety and research priorities. **Methods:** This critical narrative review drew on specified Ayurvedic textual loci, targeted searches of published clinical studies, accessible journal records and contemporary stroke guidelines through November 2026. An author-supplied teaching compendium informed verse identification but was not treated as primary clinical evidence. **Results:** A 2025 systematic review identified 30 heterogeneous clinical studies, with widespread risk-of-bias and reporting limitations. The multicenter RESTORE randomized trial enrolled 140 ischemic-stroke survivors and did not establish superiority of Ayurvedic rehabilitative treatment over conventional physiotherapy at three months (adjusted p=0.057). A 52-participant non-randomized comparison suggested short-term mobility differences with massage added to physiotherapy; selection bias prevents causal attribution. An 18-participant blinded pilot demonstrated feasibility of procedure-based comparative research, not definitive efficacy. **Conclusion:** Classical descriptions permit cautious clinical-phenotype comparisons but not claims of equivalent disease mechanisms. Current evidence is insufficient to establish an additional functional benefit or long-term safety of Ayurvedic interventions beyond guideline-based rehabilitation. Adequately powered adjunctive trials with standardized interventions, validated outcomes, independent assessment and rigorous adverse-event monitoring are needed.

INTRODUCTION

Stroke is a major cause of death, persistent disability, and dependence worldwide. The World Stroke Organization estimated nearly 12 million incident strokes and approximately 93.8 million prevalent cases in 2021; contemporary burden estimates also document substantial geographical inequalities and a large contribution from modifiable vascular risk factors[1,2]. Hemiparesis or hemiplegia is a frequently encountered neurological consequence, affecting mobility, self-care, occupational participation, and psychosocial functioning. Restoration of meaningful activities requires organized rehabilitation in addition to optimal acute treatment and secondary prevention. The present synthesis uses the SANRA methodological domains as a writing and appraisal framework [3].

Ayurveda describes *Pakshaghata* (also written *Pakshavadha* or *Pakshavata* in some sources) under *Vatavyadhi*. Literal and textual interpretations emphasize compromise of one side of the body, reduced voluntary movement (*Cheshta-nivritti* or *Akarmanyata*), and occasionally sensory loss, speech disturbance, pain, or contracture[4,5,6,7,8,9,10]. Its resemblance to post-stroke hemiplegia has generated considerable interest in integrative neurorehabilitation. However, the traditional syndrome belongs to a descriptive medical framework

Access this article online
Quick Response Code
https://doi.org/10.47070/ijapr.v14i10.4486

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distinct from modern lesion localization, vascular pathophysiology, and neuroimaging. An equivalence based only on laterality risks conflating different diagnostic categories.

This review therefore addresses three questions: (i) which features of *Pakshaghata* are consistently described in primary Ayurvedic literature, and where do the classical authorities disagree; (ii) how should these features be clinically interpreted alongside contemporary stroke diagnostics and rehabilitation; and (iii) what is the strength and applicability of evidence for Ayurvedic treatment modalities, particularly as adjuncts to standard care?

Literature search and review approach

This article is a critical narrative review, not a systematic review or meta-analysis. Classical concepts were located through *Charaka Samhita* (*Chikitsa Sthana* 28), *Sushruta Samhita* (*Nidana Sthana* 1; *Chikitsa Sthana* 5), *Ashtanga Hridaya* (*Nidana Sthana* 15; *Chikitsa Sthana* 21), *Madhava Nidana* (chapter 22) and selected later treatises. A 120-page teaching compilation supplied for this review helped identify candidate chapter/verse locations; the compilation was not included as a primary study. Because independent critical collation of Sanskrit editions was not undertaken, disputed textual terms and edition-specific verse locators remain explicitly qualified[4,5,6,7,8,9,10].

Targeted, non-exhaustive searches of PubMed/MEDLINE, publisher pages and accessible original reports were used to identify published evidence through 10 November 2026. Identified systematic reviews, comparative trials, observational studies, process evaluations and stroke care guidelines were prioritized according to clinical relevance and interpretive rigor. Reported data were compared by study design, participant characteristics, intervention/comparator, sample size, functional outcomes, clinical effect estimates, adverse-event reporting and risk of bias. No preregistered protocol, complete multi-database search log, double independent screening, PRISMA flow, new RoB 2 assessment, GRADE grading or meta-analysis was undertaken. The selection is therefore illustrative and cannot support exhaustive negative claims[3,11,12,13,14].

The classical textual comparison distinguishes directly cited passages from interpretation; biological or anatomical equivalence to modern vascular disease is not assumed. Published clinical observations were considered separately from Sanskrit therapeutic recommendations. Some edition, commentary and verse interpretations were drawn from the supplied teaching compilation and require independent verification from the cited print editions before submission [4,5,6,7,8,9,10].

Classical conceptualization and diagnostic identity

Definitions and clinical descriptors

In *Charaka Samhita*, *Chikitsa Sthana* 28/53–55, aggravated *Vata* is described as affecting the right or left side and producing loss of voluntary activity, pain, and speech disturbance. The adjoining verses also describe wasting of *Sira* and *Snayu*, contraction of a hand or foot, and distributional differences between *Ekangaroga* and *Sarvangaroga*[4]. *Sushruta Samhita*, *Nidana Sthana* 1/60–62, explicitly names *Pakshaghata* in association with disruption of joint-related bodily function and incapacitation of a body half, including altered consciousness or sensation in severe descriptions[5]. Vagbhata and Madhavakara similarly emphasize involvement of half the body, diminished function, and sensory disturbance [6,7].

Three qualifications follow. First, "*Pakshaghata*" is a syndrome-level description and does not itself distinguish cerebral infarction, intracranial hemorrhage, tumor, abscess, trauma, or metabolic mimics. Second, classical words such as *Sira*, *Snayu*, *Dhamani*, and *Sandhibandhana* should be interpreted in their own textual context; equating them directly with modern arteries, tendons, corticospinal axons, and synapses would be an untested retrospective anatomical mapping. Third, the severity of dysfunction varies: partial weakness (hemiparesis) and complete paralysis (hemiplegia) should not be merged when designing present-day studies.

Nomenclature and intertextual differences

The compendium reveals a meaningful philological distinction. Charaka discusses *Pakshaghata*, *Ekangaroga*, and *Sarvangaroga* in contiguous but separately characterized passages [4]. Sushruta also lists *Pakshaghata* and *Ekangaroga* separately in the context of *Basti* indications (*Chikitsa Sthana* 35/5)[5]. By contrast, the wording "*Ekangarogam tam kechid anye pakshavadham viduh*" in *Ashtanga Hridaya* (*Nidana Sthana* 15/38–40) and *Madhava Nidana* (22/39–41) permits variant naming for a closely related condition[6,7]. Therefore, it would be inaccurate to assert that all these terms are unconditionally synonymous across authors. *Ardita*-sometimes described with hemifacial or broader unilateral manifestations- also needs separate diagnostic consideration rather than automatic assimilation to *Pakshaghata* [4].

Table 1: Classical comparison with cautious contemporary interpretation

Source and locus	Textually supported phenotype	Interpretive qualification
Charaka Samhita, Chi. 28/53–55 [4]	One-sided loss of movement, pain, speech disturbance; adjoining *Ekangaroga*/ *Sarvangaroga* descriptions	Phenotypic overlap with hemiplegia; named entities not uniformly identical
Sushruta Samhita, Ni. 1/60–63 [5]	One side functionally incapacitated, severe sensory/motor compromise; prognosis qualified	Clinical severity description, not vascular-subtype diagnosis
Ashtanga Hridaya, Ni. 15/38–40 [6]	Half-body dysfunction, wasting and disturbed joint function; alternate naming	Terminological equivalence is author-specific
Madhava Nidana, 22/39–43 [7]	Motor/sensory deficit; *Vata* with *Pitta*/*Kapha* -associated features; prognostic categories	*Dosha*-associated features are not validated stroke mechanism markers
Sharangadhara, *Purva Khanda* 7/105–108 [8]	*Pakshaghata* enumerated among *Vata* disorders	Taxonomic citation, not a mechanism or treatment trial

Critical correspondence between classical terms and neurological findings

A clinically useful comparison must operate at the level of observable signs, not inferred biological identity. The terms *Cheshta-nivritti* and *akarmanyata* describe restricted voluntary activity; the same finding may appear after stroke but also after spinal, traumatic, neoplastic, infectious, or functional disorders. *Vakstambha* indicates a speech disturbance without distinguishing dysarthria, aphasia, apraxia or impaired consciousness. *Vicetana*/*Achetana* expressions in the classical text should not be mapped automatically to a specific modern sensory tract lesion. Similarly, *Sira* and *Snayu* reflect historically situated anatomical concepts whose extension is broader than modern arteries, veins, peripheral nerves or tendons [4,5,6,7]. The contrasts are summarized below.

Table 2: A phenotype-level classical–modern crosswalk; proposed parallels are interpretive rather than validated biological equivalences

Classical descriptor / source	Observable point of overlap	Why direct equivalence is unwarranted
Cheshta-nivritti/ *Akarmanyata* [4,5]	Marked restriction of one-sided voluntary movement	Does not determine ischemic versus hemorrhagic stroke, brain versus spinal lesion, or stroke mimic.
Vakstambha [4]	Speech disturbance may coexist with limb weakness	Cannot specify dysarthria, aphasia, language dominance or lesion location.
Vicetana/ *Achetana* [5,6,7]	Possible sensory impairment or reduced responsiveness	Translations vary; does not provide a modern sensory examination or Glasgow Coma Scale.
Sira-snayu Shoshana [4,6,7]	Descriptions of wasting or altered limb function	Historical tissue terms cannot be assumed to represent corticospinal axonal injury.
Sandhibandha-Vimoksha [5,6,7]	Functional instability or loss of limb control	Does not identify a specific neurological or biomechanical lesion.

***Nidana* and *Samprapti*: A historically situated reading**

Classical *Vata*-provoking factors include depletion, overexertion, inadequate nourishment, irregular routines, injury, and the broader concept of *Dhatukshaya* (Charaka *Chikitsa Sthana* 28/15–19)[4]. Sushruta describes severe injury involving selected *Marma* regions and excessive blood loss in association with paralysis-like outcomes[5]. These should be read as etiological observations and internal explanatory categories of the classical system; they must not be presented as demonstrated causes of ischemic or hemorrhagic stroke. Established biomedical risk factors include hypertension, diabetes, atrial fibrillation, dyslipidemia, smoking, and other vascular/environmental exposures [1,2,15].

The *Samprapti* discussed by Vagbhata and Madhavakara invokes aggravated *Vata*, wasting or impairment of *Sira* and *Snayu*, and loss of activity on one side[6,7]. In present-day research, such a framework may help define patient-reported symptom clusters, functional dependency, pain, perceived stiffness, or nutritional vulnerability. It does not establish that *Vata* corresponds biologically to a particular neurotransmitter, perfusion abnormality, or vascular lesion. The proposed relationship is best understood as a cross-disciplinary clinical analogy, not a mechanistic equivalence.

The source compendium also lists a composite *Samprapti-ghataka* schema (*Vata* predominance, *Sira*/*Snayu* involvement, *Shiro-pradesha* as principal

locus, and unilateral manifestation). Because that schema is a later didactic synthesis rather than an identically stated formula in all surveyed classical works, it is not treated as a universal ancient definition. Such attribution discipline is essential when translating educational compilations into scholarship.

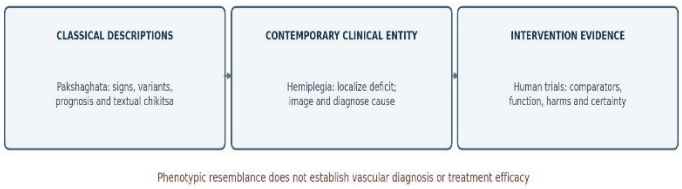


Fig. 1. Conceptual separation of classical descriptions, modern etiological diagnosis and contemporary intervention evidence. Figure supplied as a separate JPEG file

Differential diagnosis and the medical urgency of acute hemiplegia

Modern hemiplegia is a sign of dysfunction affecting descending motor pathways rather than an independent etiological diagnosis. A cortical or subcortical cerebral lesion usually causes contralateral weakness, whereas brainstem pathology may produce crossed neurological findings. Sudden focal weakness must be treated as a possible stroke until urgent evaluation identifies the cause. Mimics include hypoglycemia, post-ictal Todd paresis, functional neurological disorder, migraine, intracranial mass lesions, and selected inflammatory or infectious conditions. Emergent assessment requires time of last known well, examination of airway and swallowing risk, blood glucose testing, standardized neurological evaluation (often NIHSS), and brain imaging; vascular imaging is used when indicated for thrombectomy selection [16,17].

For acute ischemic stroke, the 2026 American Heart Association/American Stroke Association guideline supports rapid reperfusion assessment and, in selected eligible patients, intravenous thrombolysis with alteplase or tenecteplase and endovascular thrombectomy [16]. Intracerebral hemorrhage requires a fundamentally different pathway involving careful stabilization, appropriate blood-pressure management, reversal of anticoagulation when relevant, and specialist neurosurgical assessment[17]. Clinical appearance or Ayurvedic *Dosha* categorization cannot distinguish hemorrhage from infarction reliably. Any intervention that delays hospital transfer, imaging, or evidence-based reperfusion care can materially worsen prognosis.

The phase distinction is decisive: this review considers Ayurvedic interventions only as possible components of medically supervised rehabilitation after diagnostic clarification and clinical stabilization, not as a substitute for acute stroke treatment or evidence-based secondary prevention [15,16,17,18,19,20].

Classical principles of *Chikitsa* and their present-day appraisal

Snehana, *Abhyanga*, and *Swedana*

Snehana (oleation), *Abhyanga* (external oil massage), and *Snigdha Swedana* (selected heating/ fomentation measures) are prominent in general *Vatavyadhi* management, including Charaka *Chikitsa Sthana* 28/75–83 and 28/104–106[4]. Sushruta *Chikitsa Sthana* 5/19 describes a sequence of oleation, sweating, gentle purification, *Basti*, and specialized head-related procedures for selected *Pakshaghata* patients [5]. Their traditional aims include promoting comfort, reducing perceived stiffness, and supporting functional care. Contemporary interpretations may reasonably pose hypotheses about comfort, pain experience, therapeutic touch, engagement, or ease of movement; however, these are hypotheses until tested against suitable controls and clinically meaningful endpoints.

Human evidence concerning *Abhyanga* is limited but directly relevant. A 2019 prospective case-control report of 52 post-stroke inpatients compared self-selected Ayurvedic massage plus standard physiotherapy (25 patients) with standard physiotherapy alone (27 patients). Patients choosing massage had lower discharge spasticity scores and better standing and walking outcomes. Because participants selected their intervention, rather than being randomized, baseline confounding and differences in motivation or prognosis cannot be excluded; this report provides a signal for prospective adjunctive testing, not proof of an efficacy mechanism [13].

In a survivor with sensory impairment, cognitive deficits, impaired skin integrity, venous disease, or autonomic instability, intense heat, prolonged pressure, or massage may cause harm. Interventions should be modified by a qualified clinician, not delivered according to an undifferentiated protocol. Such supportive procedures also cannot replace task-specific upper-limb or gait rehabilitation, swallowing management, or stroke-prevention drugs [18,19,20].

Basti, *Virechana*, *Nasya*, and other procedures

Charaka *Chikitsa Sthana* 28/100 and *Ashtanga Hridaya Chikitsa Sthana* 21/44 refer to *Sneha*-associated therapeutic principles in *Pakshaghata*; Sushruta *Chikitsa Sthana* 5/19 provides a detailed treatment sequence including *Mridu shodhana* and *Basti* [4,5,6]. Later authors describe differing purgation intensity, exemplifying an important variation in source traditions[10]. *Nasya* and *Shirobasti* appear in related protocols, including later pharmaceutical compendia[5,8,9,10]. The presence of a recommendation

in a classical treatise is evidence of historical practice, not clinical proof of efficacy for neural recovery after stroke.

In medically fragile or anticoagulated stroke survivors, purgation and invasive or nasal procedures raise concerns relating to dehydration, electrolyte disturbance, aspiration, mucosal injury, altered drug absorption, and interactions with ongoing rehabilitation. No standardized stroke-specific benefit-risk profile is established for these interventions. Consequently, they should not be administered routinely as a substitute for neurological care, and should be evaluated only under explicit clinical eligibility, safety monitoring, and institutional oversight.

Internal medicines and formulation-specific uncertainty

The compendium identifies formulations across *Sharangadhara Samhita*, *Chakradatta*, and *Bhaishajya Ratnavali*, including *Maharasanadi Kwatha*, selected *Masha*-based preparations, *Narayana Taila*, and various *Rasaushadhi* formulations [8,9,10]. Their textual indications differ, and some formulations appear primarily in broad *Vatavyadhi* contexts rather than being exclusive to *Pakshaghata*. Historical listing does not establish equivalence among products differing in ingredients, processing, manufacturer, route, dose, or batch quality.

Potential pharmacological mechanisms occasionally proposed for botanicals- such as anti-inflammatory or antioxidant activity- are largely mechanistic or preclinical hypotheses and cannot be extrapolated directly to clinically significant recovery after stroke[21]. For mineral- or metal-containing formulations, standardized identity testing, contaminant assessment, toxicological evaluation, and interaction screening are particularly important. A published product survey identified potentially hazardous concentrations of lead, mercury, or arsenic in some Ayurvedic medicines sold online; its findings should not be generalized to every formulation but justify robust quality control [22].

Product quality, concomitant medicines, and treatment-specific safety

Stroke survivors frequently receive antiplatelets, anticoagulants, statins, antihypertensives and glucose-lowering agents, while some are affected by dysphagia, cognitive impairment or renal/hepatic comorbidity. Before any investigational oral formulation, researchers should reconcile medications and evaluate potential pharmacodynamic and pharmacokinetic interactions; theoretical concerns are not substitutes for formulation-specific interaction data. Herbal-mineral preparations also require validated identity, manufacturing provenance and batch-wise contaminant testing. A published analysis of commercially available Ayurvedic products documented contamination with lead, mercury or arsenic in some samples[22], a finding that demonstrates a quality-control concern but should not be generalized to all Ayurvedic products or individual classical formulations.

Physical and procedural safety require separate protocols: *Abhyanga* and heating interventions should account for hemianesthesia, skin integrity, pressure injury and hemodynamic tolerance; emetic/purgative and *Basti*-related procedures require attention to hydration, electrolytes, bowel pathology and procedural risk; *Nasya* in individuals with dysphagia or impaired airway protection needs a particularly cautious risk assessment. Any invasive, sedating or aspiration-associated practice should be deferred in medically unstable patients. These safeguards are precautionary clinical interpretations, not evidence that every named procedure carries the same quantified risk. Neither acute reperfusion treatment nor guideline-directed secondary prevention should be interrupted to implement an unproven adjunct [16,17,18,20].

Evidence from contemporary clinical studies

Systematic evidence synthesis

A 2025 systematic review by Akashlal and colleagues specifically evaluated Ayurvedic interventions for hemiplegia/*Pakshaghata* and searched literature through July 2023. Thirty reports met eligibility criteria: nine randomized controlled trials, eight non-randomized comparative trials and 13 pre-post studies. The review reported 1,202 participants in its randomized studies, 417 participants in the non-randomized comparative studies, and 412 participants in the pre-post studies; these figures describe heterogeneous study groups and must not be treated as a pooled effect estimate. Reviewers used RoB 2 and ROBINS-I tools. All but one study had substantial concerns for bias. Common problems were inadequate masking and sampling, poor allocation reporting, non-validated outcome scales, inconsistent diagnostic confirmation and stroke chronicity, limited follow-up, and absent or incomplete adverse-event reporting. Owing to incompatible designs, interventions and outcomes, meta-analysis was not feasible. The authors could not definitively establish efficacy or safety[11]. These findings weigh more heavily than the favorable within-group improvements reported in individual small trials.

The synthesis provides unusually concrete evidence-quality indicators. Of the 30 included studies, 21 (70%) did not adequately report adverse-event information, 20 (67%) omitted dropout or withdrawal information, 23 (77%) employed non-validated outcome tools, and 24 (80%) omitted follow-up data; 23 (77%) failed to specify stroke type and 19 (63%)

did not adequately specify duration of stroke in their eligibility descriptions [11]. These percentages refer to reporting in historical studies and do not establish that an intervention either caused or did not cause harm. Similarly, apparent within-group motor gains may reflect spontaneous post-stroke recovery, concurrent physiotherapy, regression to the mean or practice effects rather than a specific Ayurveda-related treatment effect.

RESTORE randomized trial and process evaluation

The investigator-initiated RESTORE trial was conducted at four comprehensive stroke centers in India, with prospective parallel-group randomization and blinded outcome assessment. Of 403 screened patients, 140 ischemic-stroke survivors were enrolled (70 Ayurvedic rehabilitation therapy [ART], 70 conventional physiotherapy [PT]). At three months the investigators reported upper-extremity sensorimotor assessment values of 71.97±23.88 for ART and 81.97±24.57 for PT; the unadjusted between-group difference favored PT (p=0.023). Following adjustment for age, baseline stroke severity, baseline Fugl-Meyer score and risk factors, the comparison did not meet conventional statistical significance (p=0.057). No secondary outcome was significantly better in ART, and no major serious adverse events were reported during the study period [12]. Because the abstract calls the outcome FMA-UE while reporting values above the 66-point motor subscale maximum, its precise sensorimotor score composition and normalization must be checked in the full trial report before secondary effect-size calculations. The justified conclusion is the absence of demonstrated ART superiority, not proven equivalence; these replacement-arm results do not directly estimate the effect of adding ART to standard PT.

A subsequent mixed-methods process evaluation of RESTORE used case-report data, therapy logs and 38 interviews involving patient-caregiver dyads and health professionals. Improvements in mobility were reported in both groups; participants allocated to ART also described perceived relief of pain and emotional distress, and acceptability varied geographically. Investigators proposed three implementation-related hypotheses for why ART had not outperformed PT: fixed treatment standardization may have compromised individualization, *Nasya* was excluded for stroke-related safety concerns, and a one-month intervention period might have been insufficient [23]. These are explanatory hypotheses and qualitative observations, not confirmatory tests of clinical efficacy or grounds to relax safety restrictions. Such work identifies feasibility questions and patient-relevant symptoms worth testing as predefined outcomes in an add-on trial.

Table 3: Published indexed studies: Quantitative findings and study-specific methodological appraisal (no pooled effects calculated)

Publication / design	Sample, intervention and reported endpoints	Strength of inference / main limitation
Akashlal et al., 2025; systematic review [11]	30 studies: 9 randomized (n = 1,202), 8 nonrandomized comparative (n = 417), 13 pre-post (n = 412). Adverse-event information omitted in 21; follow-up omitted in 24; non-validated outcomes in 23.	Formal risk-of-bias appraisal; heterogeneity precluded pooling. Does not establish effectiveness or safety.
Sylaja et al., 2025; RESTORE, assessor-blinded multicentre RCT [12]	140 participants (70 ART, 70 PT), 1-month assigned intervention. At 3 months, reported upper-extremity sensorimotor scores 71.97 ± 23.88 vs 81.97 ± 24.57; unadjusted p = 0.023 (PT favored), adjusted p = 0.057.	ART superiority over PT not established. Not an adjunct trial. Exact reported FMA-UE score convention requires verification.
Aggithaya et al., 2014; blinded pilot randomized comparison [24]	18 chronic hemiplegia patients (9/arm): 7 days *Navarakizhi* versus *Pinda Sweda*; common oral *Ayurveda* medicines for 14 days; blinded patient and assessor.	Shows methodological feasibility; tiny sample, short-term active-comparator study without PT-only comparator.
Sankaran et al., 2019; self-selected prospective comparative cohort [13]	52 patients (25 massage + PT, 27 PT). Standing with minimal assistance: 6.0 ± 2.16 versus 9.42 ± 4.55 weeks (p = 0.001); FIM walking at discharge: 5.1 ± 1.1 versus 4.2 ± 1.2 (p = 0.007).	Potential adjunctive signal; self-selection and group differences prevent causal attribution.

Ediriweera & Perera, 2011; unbalanced single-blind placebo comparison [14]	30 patients (25 formulation, five placebo); 14-day course. Reported complete relief among treated: slurred speech 64%, mouth deviation 68%, swallowing difficulty 44%.	Very small placebo arm, short follow-up, symptom-based outcomes; no established functional or safety benefit.
Raju et al., 2026; RESTORE mixed-methods process evaluation [23]	38 interviews alongside trial logs and process records; perceptions of acceptability, burden and symptom experience.	Acceptability and implementation evidence; not a new efficacy comparison.

Procedure-comparator evidence:

Double-blind *Navarakizhi* pilot

Aggithaya and colleagues (2014) conducted a double-blind, randomized, active-comparator pilot trial in 18 participants with hemiplegia of 6 months to 2 years duration. Nine participants per group were assigned to *Navarakizhi* or *Pinda Sweda* for seven days; both groups also received the same Ayurvedic oral medicines over 14 days. Patients and the physiotherapist assessing muscle tone, strength, reflexes, range of movement and balance were blinded. Reported interquartile-range patterns were more favorable with *Navarakizhi* for some assessments, but the small sample, short follow-up and lack of a standard-physiotherapy-only comparator preclude a reliable claim of efficacy or superiority over evidence-based rehabilitation[24]. Its main contribution is methodological: patient and outcome-assessor masking of procedure-based Ayurveda interventions is possible, a point recognized in the 2025 systematic review [11,24].

Earlier indexed clinical studies: Quantitative outcomes and bias

Sankaran and colleagues (2019) studied 52 stroke survivors who received either Ayurvedic massage plus a common physiotherapy program (n=25) or physiotherapy alone (n=27). All were evaluated in an inpatient rehabilitation setting; massage began approximately one month after stroke onset and comprised five or ten treatment sessions. Reported time to standing with minimal assistance was 6.0±2.16 weeks with massage versus 9.42±4.55 weeks in controls (p=0.001). Discharge Functional Independence Measure walking scores were 5.1±1.1 versus 4.2±1.2 (p=0.007), and Modified Ashworth Scale results favored massage (reported comparison p = 0.001). These between-group findings are clinically interesting but vulnerable to self-selection, small sample size, heterogeneous clinical severity, potential co-interventions and incomplete protection against outcome-assessment bias [13]. They should not be read as evidence that massage regenerates corticospinal pathways or that a similar effect would persist in a randomized trial.

An earlier small study in AYU (Ediriweera and Perera, 2011) enrolled 30 patients with *Pakshaghata* of less than one-month duration. Participants were assigned unequally to *Chandra Kalka* 250mg with *Mahadalu Anupanaya* 40ml three times daily for 14 days (n=25) or an internal placebo (n=5), reportedly with single blinding. In the treated group the authors reported complete relief of speech slurring in 64%, swallowing difficulty in 44%, tongue fasciculation in 52%, and mouth deviation in 68%, with no statistically significant symptom reduction in the placebo group[14]. These are symptom-level results; the very small control arm, unequal allocation, short follow-up, limited standardized functional measurement and incomplete stroke-subtype ascertainment prevent dependable estimates of treatment-specific motor recovery or safety. The authors' favorable conclusion should therefore be treated as preliminary rather than definitive [11,14].

Outcome measures, effect sizes, and interpretive cautions

Cross-study interpretation is complicated by incompatible endpoints. RESTORE assessed three-month upper-extremity sensorimotor recovery using an FMA-based measure, the 2019 massage study evaluated spasticity (Modified Ashworth Scale), standing, and FIM walking, while the 2011 AYU study primarily evaluated graded *Pakshaghata* symptoms [12,13,14]. The Fugl-Meyer upper-extremity MOTOR subscale has a maximum of 66 points, whereas broader sensorimotor implementations are scored differently; investigators must identify the full scoring convention before comparing studies or deriving effect sizes [25]. A robust efficacy study should report the baseline and follow-up values by arm, adjusted between-group effect estimate, 95% confidence interval, intention-to-treat population, adverse events, and one prospectively specified clinically important primary outcome. Validated secondary scales may include modified Rankin Scale, Barthel Index, FIM, Berg Balance Scale, gait speed, quality of life, and pain/sleep instruments when applicable. The AVERT trial demonstrates why rehabilitation timing and intensity require separate scrutiny [26]. Importantly, a low p value from a self-selected cohort or symptom-scale pre-post comparison does not compensate for biased treatment assignment.

The RESTORE abstract labels its primary endpoint Fugl-Meyer Assessment–upper extremity yet reports mean scores exceeding the conventional upper-extremity motor subscale maximum of 66 [12,25]. These values have therefore been retained only as originally reported in the study description and Table 3; the precise sensorimotor scale construction requires confirmation against the full paper or author clarification before reanalysis. No unverified transformation, effect-size estimate or claim of equivalence has been made. The adjusted p-value and unadjusted p-value should not be treated as interchangeable. This scoring caveat is a report-interpretation limitation, not grounds to disregard the entire randomized trial.

Limitations of the existing clinical evidence

Comparative validity and confounding. Many early studies compared treatment packages without robust randomization, adequate allocation concealment or blinded outcome assessment. Sankaran et al. used patient-selected groups; preference, motivation, unequal age distributions and differences in care intensity could influence walking and standing outcomes despite similar baseline stroke severity [13]. The 2011 placebo study allocated only five patients to the control arm, creating highly imprecise estimates and limiting detection of both treatment benefit and harm [14]. The 2014 pilot was randomized and blinded but too small and short to provide stable estimates [24].

Clinical heterogeneity. Ischemic stroke, intracerebral hemorrhage and other causes of hemiplegia differ in pathophysiology, acute treatment and prognosis. Many historical reports did not document CT/MRI confirmation, lesion characteristics, onset-to-enrollment interval, stroke severity or comorbidity. Mixing acute, subacute and chronic recovery without prespecified subgroup analysis obscures spontaneous-recovery gradients and makes it difficult to determine which population, if any, might benefit [11,15,16,17,18].

Intervention heterogeneity and attribution. Named treatments such as *Abhyanga*, *Swedana*, *Basti*, *Nasya* and oral formulations vary in material composition, dose, frequency, sequencing, practitioner technique and treatment duration. Trials frequently administer several components together, sometimes alongside physiotherapy or conventional medicines. Improvement following a bundle does not isolate a causal contribution from any constituent. The RESTORE trial tested an Ayurveda rehabilitation program against conventional physiotherapy, not as a rigorously controlled additive intervention; its primary result therefore cannot establish the efficacy or inefficacy of an Ayurveda-plus-physiotherapy combination [12,23].

Outcome and reporting limitations. Short treatment windows and limited longitudinal assessment are inappropriate for a disorder with variable neuroplasticity and functional recovery over months. Non-validated symptom scores, selective emphasis on within-group p-values, lack of confidence intervals, poorly described missing data and insufficient minimal clinically important difference benchmarks reduce comparability and interpretability. The disagreement between the RESTORE abstract outcome label and its reported score range further argues against uncritical numerical pooling [11,12,25].

Safety and external validity. Failure to document adverse events is not evidence of safety. Historical studies rarely provided systematic monitoring for hypotension, dehydration, aspiration, skin injury, bleeding, drug interactions or formulation contamination; rare harms could not be excluded in small, short studies. Most available studies arise from a narrow geographical and service context, with little information about training, provider fidelity, cost, patient preferences or sustained adherence; consequently generalizability to diverse stroke populations and routine practice remains uncertain [11,22].

Table 4: Evidence gaps and corresponding design remedies for future *Pakshaghata* trials

Deficiency in published evidence	Consequence for interpretation	Recommended correction
Uncertain stroke subtype and chronicity [11]	Biologically disparate participants; heterogeneous spontaneous recovery	CT/MRI-defined diagnosis, onset window, NIHSS or equivalent baseline severity
Inadequate allocation, blinding and controls [11,13,14]	Selection, performance and measurement bias	Concealed randomization, credible control, blinded assessor, equal core rehabilitation
Multicomponent intervention packages [11,12,23]	Impossible to attribute effects to a single treatment	Manualized intervention, dose/fidelity logs, preplanned component analyses

Nonvalidated endpoints, short follow-up [11]	Poor relevance, reproducibility and durability assessment	FMA-UE motor score with stated range; mRS, Barthel, gait and quality of life; 3-12-month follow-up
Incomplete harms and withdrawals [11]	Potential adverse effects remain unobservable	Prospective adverse-event definitions, laboratory/clinical monitoring and complete attrition reporting

Evidence-based clinical integration: A phase-specific framework

A defensible integrative model separates three consecutive but overlapping domains. The hyperacute domain is entirely governed by emergency stroke protocols, neurological stabilization, appropriate reperfusion or hemorrhage treatment, and complication prevention[16,17]. The subacute domain emphasizes specialist multidisciplinary rehabilitation, including physiotherapy, occupational therapy, speech-language and swallow assessment, cognitive/psychological care, caregiver education, prevention of immobility-related complications, and adapted exercise[18,19,20]. The chronic domain focuses on sustained function, social participation, cardiovascular prevention, adherence, and symptom management [15,18,19,20]. Any candidate Ayurvedic intervention belongs to the latter two domains only after a documented individualized risk-benefit assessment, explicit consent, and continuity of standard rehabilitation.

Such a framework respects patient preferences without creating false equivalence between evidence levels. It also creates a testable distinction between substitution research (*Ayurveda* instead of conventional physiotherapy) and add-on research (standard care plus a specified intervention versus standard care plus a matched comparator). Until credible adjunctive efficacy data exist, routine recommendation for improved neurological outcomes cannot be justified [11,12].

Table 5: Proposed safeguards for clinical assessment and future research pathways

Phase / decision	Non-negotiable clinical requirement	Potential research question
Suspected new stroke	Emergency transfer, glucose check, imaging and specialist pathway [16,17]	None: do not delay acute treatment
Medically stabilized rehabilitation	Standard physiotherapy/OT/SLT; secondary prevention [15,18,19,20]	Can a standardized adjunct improve comfort, pain or adherence?
Persistent unilateral impairment	Validated impairment, activity and participation scales [25]	Does the adjunct produce clinically important between-group functional gains?
Any oral/herbal/ mineral intervention	Medication reconciliation, quality testing and adverse-event reporting [11,22]	What are interaction, toxicological and tolerability profiles?
Follow-up at 3–12 months	Repeat functional, quality-of-life and vascular-risk evaluations	Are benefits durable and cost-effective?

Classical prognosis and modern outcome prediction

The classical literature is explicit that *Pakshaghata* may be difficult to treat. Charaka *Chikitsa Sthana* 28/72–74 describes the complexity of severe *Vatavyadhi* and emphasizes relative advantages of a recent presentation, stronger patient condition, and fewer complications [4]. Sushruta *Nidana Sthana* 1/63 and *Madhava Nidana* 22/43 distinguish prognostic categories related to unassociated *Vata*, *dosha* concurrence, and depletion-related states [5,7]. These should not be reinterpreted as validated prognostic categories for specific ischemic stroke subtypes; the apparent correspondence between "recent disease" and time-sensitive treatment is a hypothesis of limited conceptual similarity, not a demonstrated shared biological theory.

Contemporary prognosis incorporates baseline neurological severity, infarct/hematoma characteristics, reperfusion, age, comorbidities, complications, cognition, social support, and access to structured rehabilitation [15,16,17,18,19,20]. Standardized risk stratification provides a transparent basis for trial randomization, outcome adjustment, and individualized goal-setting. Classical categories might be recorded as additional exploratory variables once their reliability, construct validity, and inter-rater consistency have been established independently.

Research gaps and a priority agenda for *Kayachikitsa*

The main research gap is not the absence of historically described therapies but absence of reproducible, sufficiently unbiased estimates of treatment-specific benefit and harm. RESTORE tested ART against PT and did not establish ART superiority; Sankaran et al. provided a nonrandomized adjunctive signal; Ediriweera and Perera provided a very small, short-duration placebo comparison; and the 2025

systematic review found pervasive limitations in older studies [11,12,13,14,23]. A next-generation multicenter trial should specifically test a predefined adjunct against standard physiotherapy plus an appropriate control rather than withdrawing established rehabilitation. It should identify one protocol or adequately documented individualized treatment framework; specify medicinal product identity, preparation, dose, route, batch quality, provider qualifications, session intensity, adherence, co-interventions and prespecified safety rules. Methods should include concealed random allocation, blinded assessors, sample-size justification based on a defensible minimal clinically important difference, intention-to-treat analysis, prespecified missing-data management, trial registration, harms surveillance, and transparent CONSORT 2025 reporting [27].

Population heterogeneity requires explicit stratification by ischemic versus hemorrhagic stroke, time since onset, lesion localization, severity, cognitive ability, dysphagia and anticoagulant exposure. Equipoise and appropriate controls should determine whether a pragmatic additive trial or a mechanistic pilot is most appropriate. Patient-valued outcomes such as pain, fatigue, sleep, caregiver burden, mood, participation, and accessibility may be important secondary questions; however, multiple comparisons should not become substitutes for a clearly justified primary endpoint. Mixed-methods implementation research, undertaken alongside trials, can address acceptability and generalizability without being mistaken for causal efficacy evidence [23].

An additional scholarly priority is philological: systematic collation of Sanskrit editions, commentarial differences, and modern translations may reduce the conflation of *Pakshaghata*, *Ekangaroga*, *Sarvangaroga*, and *Ardita*. Prospective studies could examine whether robustly defined Ayurvedic phenotypes carry independent information about rehabilitation needs after accounting for conventional clinical variables. A priori hypotheses should avoid retrospective claims that ancient nosology predicted modern cerebrovascular mechanisms.

A pragmatic research pathway would begin with an externally monitored feasibility pilot in medically stable adults with a specified stroke subtype and onset interval; assess recruitment, intervention fidelity and the feasibility of blinded endpoint assessment; then progress to a multicenter, adequately powered superiority trial of one defined adjunct plus conventional rehabilitation versus conventional rehabilitation plus a credible comparator. Functional outcomes should be assessed by blinded rehabilitation professionals, with rehabilitation dose in both groups documented. A six- to twelve-month follow-up, health-related quality of life, caregiver outcomes, cost/resource use and prospective adverse-event surveillance would determine whether any short-term signal translates into sustained patient-important benefit. These are proposed study-design recommendations rather than outcomes established by current evidence [11,18,19,20,27].

Position relative to previous IJAPR publications

A literature-based article focused specifically on *Nasya* in *Pakshaghata* and individual reports of multimodal management in post-stroke hemiplegia [28,29]. Those publications offer useful classical interpretations and clinical descriptions but cannot, by themselves, establish comparative therapeutic efficacy. The present review differs in systematically distinguishing classical phenotype from etiological stroke diagnosis, scrutinizing randomized and nonrandomized comparative evidence, and separating clinical effectiveness from procedural feasibility, acceptability and safety. This difference in purpose is the principal contribution of the present review rather than a claim to describe *Pakshaghata* for the first time.

Limitations of the present narrative review

The findings of this review must be interpreted within several methodological boundaries. First, literature identification was purposive rather than exhaustive. Although major pertinent PubMed-indexed publications and clinical guidelines were inspected, a preregistered search protocol, full reproducible multi-database search histories, duplicate independent title/abstract screening, grey-literature retrieval, citation-chaining audit and prospective eligibility adjudication were not conducted. Consequently, additional eligible studies may have been missed, especially unpublished trials, regional-language material and journals not indexed in MEDLINE. The reviewed examples were chosen for interpretive value and evidence level, not to represent a complete census of all *Pakshaghata* studies; publication and selection biases cannot be excluded. SANRA informed reporting quality, but its use does not confer systematic-review status [3].

Second, the synthesis of historical Ayurvedic sources was initiated from an educational compendium supplied by the author. The compendium is rich in verse locators but is a secondary educational aid, not an independent critical edition or standardized translation. A full philological comparison of original manuscripts, Sanskrit editions, commentaries and contested readings was beyond this review. Terms such as *Sira*, *Snayu*, *Achetana* and *Ekangaroga* may vary by textual context and commentator, and translation choices can influence apparent similarity to present-day neurological language. This review therefore describes plausible phenotypic overlap, not verified anatomical or causal identity, and cannot establish equivalence between classical *Dosha*/

Samprapti models and ischemic or hemorrhagic pathobiology [4,5,6,7,8,9,10].

Third, this review relied on available peer-reviewed reports, original abstracts and selected accessible full texts rather than individual participant data, original case-report forms or independent statistical reanalysis. No meta-analysis, de novo Cochrane risk-of-bias assessment, GRADE rating or pooled adverse-event estimate was attempted. The underlying studies differ substantially in age, stroke diagnosis, disease duration, intervention components, physiotherapy exposure, outcome measures and follow-up. Accordingly, numerical results are presented as source-specific observations and must not be combined into an implied pooled estimate. Some reports provide unadjusted p-values but not adjusted between-group estimates or precision intervals, which limits their clinical interpretation [11,12,13,14,24].

Fourth, outcome-score and harms reporting constrain the certainty of selected comparisons. The RESTORE publication abstract labels its primary measurement FMA-UE while reporting scores that exceed the 66-point upper-extremity motor subscale maximum; absent full scoring documentation, the precise scale construction cannot be verified in this manuscript and no secondary effect calculation was made [12,25]. "No major serious adverse events reported" in a time-limited trial is narrower than demonstrated long-term safety of every Ayurvedic procedure or compound. Similarly, omission of harm reporting in most historical studies prevents an inference of absence of risk. No formulation-specific pharmacovigilance meta-analysis or laboratory quality audit was performed [11,12,22].

Finally, external validity is limited by the evidence base itself. Most selected empirical studies involved South Asian service settings, modest samples and distinct therapist expertise. Patient selection, affordability, geography, treatment acceptability, dose fidelity, co-interventions and rehabilitation access may differ markedly in other healthcare systems. This narrative review did not include a formal patient/public priority-setting exercise or health-economic evaluation. Its principal contribution is therefore a transparent conceptual and methodological appraisal that identifies testable questions; it must not be read as a practice guideline, definitive efficacy estimate or endorsement of replacing acute stroke care or established rehabilitation [11,12,18,19,20,23].

CONCLUSION

Classical *Pakshaghata* descriptions provide a historically structured account of predominantly unilateral dysfunction, including impaired movement and, in certain accounts, speech or sensory disturbance. These features can be critically compared with the clinical phenotype of hemiplegia, but cannot specify modern stroke subtype or establish shared pathophysiology. The contemporary evidence remains insufficient to conclude that Ayurveda-based procedures or formulations replace or add clinically meaningful benefit to established multidisciplinary post-stroke rehabilitation. A 2025 review identified pervasive reporting and bias limitations, RESTORE did not demonstrate superiority over conventional physiotherapy, the small procedure-comparison pilot supports research feasibility rather than efficacy, and encouraging observations from nonrandomized or short clinical studies remain hypothesis generating. Medically supervised, adequately powered additive trials using reproducible interventions, blinded validated outcomes, robust adverse-event monitoring and patient-valued long-term endpoints offer the most defensible research direction for *Kayachikitsa*.

Author contributions

Dr. Ayush Gupta- Conceptualization, literature search, data collection, critical analysis and interpretation of the literature, and preparation of the original manuscript.

Dr. Aman Raj- Literature review, data extraction, synthesis of scientific evidence, and critical review and revision of the manuscript.

Dr. Sujata Yadav- Conceptual guidance, academic supervision, critical evaluation of the manuscript, and revision for important intellectual content.

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Cite this article as:
Ayush Gupta, Aman Raj, Sujata Yadav. Pakshaghata in Ayurveda: A Critical Narrative Review of Classical Descriptions, Clinical Correlates, and Contemporary Evidence for Stroke Rehabilitation. International Journal of Ayurveda and Pharma Research. 2026;14(10):276-288.
https://doi.org/10.47070/ijapr.v14i10.4486
Source of support: Nil, Conflict of interest: None Declared

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