



Case Report

Emergency Home-Based Ayurvedic Intervention (*Sadyo Snehana* and *Nasya*) During a Transient Ischaemic Attack in a Remote, Resource-Limited Setting: A Case Report

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Abstract

Background: Transient ischaemic attack (TIA) is a medical emergency. It is also a warning sign of impending stroke. The risk is higher in patients with serious comorbidities. In remote, resource-limited settings, emergency neurological care is often not available in time. Safe initial measures therefore deserve documentation. **Case presentation:** A 58-year-old female developed sudden left hemiparesis at her remote home. She had post-CABG triple-vessel disease, insulin-dependent diabetes mellitus, hypertension and chronic kidney disease (CKD). The weakness was accompanied by *Stambha* (limb stiffness) and a transient decline in consciousness. Capillary blood glucose at onset was 124 mg/dL, which excluded hypoglycaemia. **Intervention:** No emergency facility was available nearby. The attending Ayurvedic physician administered *Sadyo Snehana* (acute oral oleation) with groundnut oil. This was followed by *Nasya* (nasal administration) with *Mahanarayana Taila*. Both were given during the symptomatic window. **Outcomes:** The stiffness resolved rapidly. Blood pressure fell from 160/90 to 130/84 mmHg. Full responsiveness returned within an hour. The next day, a neurologist and MRI (no acute infarct) confirmed the diagnosis as TIA. Complete functional recovery (Modified Rankin Scale 0) was reached by Day 35. Serum creatinine improved from 1.7 to 1.4 mg/dL. There was no recurrence at three months. **Conclusion:** This report records the feasibility and safety of a home-based Ayurvedic intervention during a TIA in a resource-limited setting. A TIA resolves on its own. Therefore a causal effect on recovery cannot be inferred from a single case. The observations are hypothesis-generating. They warrant systematic prospective study.

Keywords: *Pakshaghata*, *Vatavyadhi*, Cerebrovascular event, *Mahanarayana Taila*, Chronic kidney disease, Differential diagnosis

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Introduction

Stroke is a leading cause of long-term disability and death (1,2). Transient ischaemic attack (TIA) is a brief episode of neurological dysfunction. It is caused by focal ischaemia without acute infarction (3). It is an important warning event. It calls for prompt evaluation and secondary prevention. Many people live far from centres that offer time-sensitive treatment such as intravenous thrombolysis and mechanical thrombectomy.

The challenge grows when the event occurs in a high-risk patient. The present patient had post-CABG (Coronary Artery Bypass Grafting) triple-vessel disease. She also had insulin-dependent

diabetes mellitus, hypertension and chronic kidney disease (CKD). In such a profile, thrombolysis carries a high risk. Safe initial measures are therefore valuable while definitive evaluation is arranged.

In Ayurveda, *Pakshaghata* (acute hemiplegia) is described mainly as a disorder of *Vata Dosha* (the principle that governs movement and neurological function) (4). The treating physician interpreted the sudden *Stambha* (limb stiffness) as acute *Vata Avarana* (obstruction of the *Vata* channels). In the classical view, such obstruction calls for prompt use of *Snigdha Guna* (unctuous quality). This reasoning guided the choice of an emergency *Sadyo-Chikitsa* (rapid therapeutic measure). It is presented here as the bedside clinical reasoning. It is not offered as an established mechanism.

The role of Ayurveda in the post-acute and rehabilitative phases of stroke is increasingly documented (5). However, reports of an immediate, home-based Ayurvedic measure during the symptomatic window of an acute cerebrovascular event are scarce. Reports that are also corroborated by specialist evaluation

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are rarer still. The present case report describes such an instance. It is presented transparently. It covers the clinical course, the diagnostic process, the neurologist-confirmed diagnosis of TIA, the safety of the protocol in a CKD patient, and an honest appraisal of what can and cannot be concluded.

Case Presentation

Patient profile and medical history

The patient is a 58-year-old female. Her history is complex and high-risk. She had post-CABG status (April 2023) for triple-vessel disease (LIMA to LAD, RSVG to distal LCX). She had long-standing insulin-dependent diabetes mellitus (Toujeo Solostar, 6 U/day). She also had hypertension and dyslipidaemia. For these she was on continuous dual antiplatelet therapy and a statin. She had acute kidney injury in 2023. This led to established CKD, with a baseline serum creatinine of about 1.7 mg/dL. She also had left shoulder periarthritis and left-sided lumbar disc disease (L4-L5). Both are relevant to her later recovery. All modern cardiac and diabetic medicines were continued unchanged. Before this event she was functionally independent (pre-event Modified Rankin Scale 0).

The acute event (17 September 2025)

The event began at about 11:30 AM. The patient first developed mild facial asymmetry. This progressed quickly to complete left hemiparesis of the upper and lower limbs. There was a rapid onset of *Stambha* (pronounced limb stiffness). There was also a transient decline in consciousness to a semi-conscious or obtunded state. Initial blood pressure was 160/90 mmHg. The patient is insulin-dependent. Therefore capillary blood glucose was checked at once with a home glucometer. It was 124 mg/dL. This excluded hypoglycaemia, the most important reversible mimic, before any intervention.

The event occurred at the patient's residence. The residence lies in a remote area outside the city. No healthcare facility is present nearby. One of the authors, an Ayurvedic physician, was present at the residence at the time. In the classical framework, the sudden *Stambha* was read as acute *Vata Avarana*. This interpretation, together with the lack of any reachable emergency care, led to an immediate *Sadyo-Chikitsa* with the limited Ayurvedic resources available at home.

Differential diagnosis

A focused differential diagnosis was considered. Hypoglycaemia was excluded at the bedside by the glucose value of 124 mg/dL. A seizure with Todd's (post-ictal) paresis was thought unlikely. There were no clonic movements, no tongue-biting and no incontinence. The next day, a consultant neurologist (DM, Neurology) and neuroimaging were used to exclude other causes. These included acute infarct, intracranial haemorrhage, space-occupying lesion, significant large-vessel stenosis and aneurysm. The deficit was transient. The MRI brain showed no acute infarct. On this basis, the neurologist confirmed the final diagnosis as transient ischaemic attack (TIA).

The emergency home-based intervention (0 - 60 minutes)

Triage rationale. *Sadyo-Chikitsa* is time-critical. The setting was remote and resource-limited. A rapid decision was therefore needed. *Eranda Taila* (castor oil) is classically preferred to relieve *Vata Avarana*. It was not available at the moment. To save time, groundnut oil (*Arachis hypogaea*) was used as the emergency

unctuous substance (6). It was chosen for its *Snigdha* (unctuous) property and its immediate availability.

Procedure 1 (*Sadyo Snehana, acute oleation*). About 400 ml of groundnut oil was given orally in four divided doses. The patient was semi-conscious, but her gag reflex was preserved. This allowed careful, reflexive swallowing. About 300 ml was ingested. About 25% was regurgitated because the protective reflex was slow. No purgation or diarrhoea followed. In Ayurvedic terms, this was *Shamana Snehana* (palliative or absorptive oleation), not *Shodhana* (eliminative therapy). The stiff left limbs softened within minutes of ingestion.

Procedure 2 (*emergency Nasya*). At about 30 minutes, the patient was partly oriented. *Nasya* (nasal administration) was then performed with *Mahanarayana Taila*, 5 ml per nostril. On instillation into the right nostril, the patient felt a distinct sensation spread to the right side of the head. This matches the classical understanding of *Nasya*, in which medicine given through the nose reaches and pervades the structures of the head (*Nasa Hi Shiraso Dwaram*, the nose is the gateway to the head) (7).

Immediate outcome (within 60 minutes). Full responsiveness returned. Blood pressure fell from 160/90 to 130/84 mmHg. The acute *Stambha* (stiffness) resolved and the limbs became pliable. The focal motor weakness still persisted at this stage. This separated the quickly reversible stiffness from the weakness that recovered more slowly over the following weeks.

Diagnostic Assessment, Timeline and Therapeutic Intervention

Diagnostic assessment and specialist evaluation

Neuroimaging. MRI of the brain with MRA of the brain and neck was done on 18 September 2025. This was about 24 hours after onset. It was performed at Baroda Imaging Centre, Vadodara. The study reported no acute infarct, no intracranial haemorrhage and no space-occupying lesion. The only findings were a small gliotic area in the superior aspect of the right lentiform nucleus and scattered chronic small-vessel ischaemic changes in the bilateral fronto-parietal white matter. MRA showed normal intracranial internal carotid arteries. There was no significant stenosis, occlusion or aneurysm. The absence of an acute lesion supports the diagnosis of TIA.

Specialist management. The consultant neurologist confirmed the diagnosis as TIA. The patient was already on dual antiplatelet therapy and a statin for secondary prevention. No additional neurological medicine was considered necessary. A single analgesic (tapentadol 50 mg) was prescribed for musculoskeletal pain. The patient already underwent routine investigations at regular intervals. These included homocysteine, HbA1c, complete blood count, serum creatinine and echocardiography. These were advised to continue. She was advised to follow up with her regular physician. She was also advised to report to hospital at once if any recurrence occurred.

Renal safety

The patient had CKD, so renal function was monitored. Herbo-mineral formulations were used in the maintenance phase. Even so, serum creatinine improved from about 1.7 mg/dL at baseline to 1.4 mg/dL by the third week of October 2025. Urine frothing also reduced. This supports a non-nephrotoxic profile of the protocol in this co-morbid patient. In a single case, however, this cannot be attributed to the intervention.

Timeline of clinical events

Table 1: Timeline of the acute event, intervention, diagnostic evaluation and recovery

Date / Time	Event / Intervention	Outcome / Status
17 Sept, 11:30 AM	Sudden facial asymmetry. Then complete left hemiparesis with <i>Stambha</i> (rapid limb stiffness). Transient decline in consciousness. Capillary blood glucose 124 mg/dL (hypoglycaemia excluded).	BP 160/90 mmHg. Semi-conscious or obtunded state.
17 Sept, 11:45 AM - 12:30 PM	Home-based emergency intervention. Oral <i>Sadyo Snehana</i> (about 400 ml groundnut oil, about 300 ml ingested). Then <i>Nasya</i> with <i>Mahanarayana Taila</i> (5 ml per nostril).	Responsiveness returned. BP 130/84 mmHg. <i>Stambha</i> (stiffness) resolved. Limbs pliable. Focal weakness persisted. No purgation.
18 Sept	Patient taken to hospital (VINS, Vadodara). Neurologist assessment. MRI brain with MRA (Baroda Imaging Centre). Ayurvedic maintenance started.	Diagnosis confirmed as TIA. MRI: no acute infarct (chronic small-vessel changes only). Existing antiplatelet and statin continued. Tapentadol added for pain.
19-21 Sept (approx.)	Transient <i>Aruchi</i> (anorexia) due to <i>Agni Mandya</i> from the high lipid load.	Managed conservatively. Insulin (6 U/day) continued. Hypoglycaemia managed with oral glucose.
24-30 Sept	Guided physiotherapy started, then self-practice.	Progressive return of motor function in the upper and lower limb.
3rd week Oct	Follow-up renal assessment.	Serum creatinine improved from 1.7 to 1.4 mg/dL.
21 Oct	Final follow-up.	Full functional recovery (Modified Rankin Scale 0). Normal gait and range of motion.
Dec 2025 (3 months)	Follow-up review.	Symptom-free. No recurrence.

Maintenance therapeutic intervention (18 September - 21 October)

Dosage rationale (*Yukti*). Contemporary literature suggests *Nasya* doses up to 10 ml for stroke management (5). The patient was elderly and had a significant cardiac history. A conservative micro-dose protocol of 1 ml was therefore adopted. This choice followed *Yukti* (clinical discretion). It prioritised safety and systemic stability.

The daily maintenance regimen had three parts. *Nasya* was given with *Mahanarayana Taila*, 1 ml per day, in the early morning. *Rasaraja Rasa* was given at 150 mg twice daily. *Ekangaveer Rasa*

was given at 300 mg twice daily (8). These are classical formulations. They were sourced from a GMP-certified pharmaceutical house. The metallic ingredients had undergone classical *Shodhana* (purification) and *Marana* (incineration). This is consistent with studies on *Swarna Bhasma*. Those studies report a favourable safety profile and cerebroprotective effects in ischaemic models (9,10). All modern cardiac and diabetic medicines were continued unchanged. These included antiplatelets, the statin and insulin (6 U/day). Physiotherapy began after seven days and continued as self-practice.

Discussion

Interpretation and the limits of causal inference

One point must be stated plainly. A neurologist confirmed the event as a transient ischaemic attack, and MRI supported this. By definition, the deficit of a TIA resolves on its own. The recovery in this patient therefore cannot be attributed to the Ayurvedic intervention. This report makes no such claim. What the case does show is the feasibility and apparent safety of an immediately available, home-based measure during the symptomatic window. The setting was one where emergency care could not be reached.

Within these limits, two observations are reported as observations only. First, the *Stambha* (limb stiffness) softened within minutes of oral oleation. Muscle tone after ischaemia is dynamic. It is influenced by fluctuating neural drive and reflex excitability (11). A temporal association of this kind raises a hypothesis. It does not establish a mechanism. Second, the dense focal deficit resolved quickly. This is why the next-day specialist recorded a transient hemiparesis with recovery and diagnosed TIA. The slower, multi-week return of full strength, gait and fine motor function is best read as functional reconditioning. It was plausibly slowed by the patient's age, her multiple comorbidities, and her pre-existing left shoulder periarthritis and left L4-L5 lumbar disc disease.

Biological plausibility (hypothesis-generating)

Some mechanisms could be explored in future controlled studies. The *Nasya* route is of interest. Intranasal administration can give direct nose-to-brain delivery and partly bypass the blood-brain barrier (12,13). The sesame oil base of *Mahanarayana Taila* contains the lignans sesamin and sesamol. These have shown neuroprotective and antihypertensive effects in experimental ischaemia models (14,15). Such findings support biological plausibility. They do not prove efficacy in this single case.

Safety, metabolic response and a caution

Eranda Taila was not available. The use of readily available groundnut oil shows a resource-adaptive approach in which time-to-treatment is given priority. The renal safety profile is reassuring. Creatinine improved from 1.7 to 1.4 mg/dL. No clinical signs of heavy-metal toxicity were seen on monitoring, such as Burton's line or abdominal colic. A transient *Aruchi* (anorexia) appeared around days 3 to 5. It was read as physiological *Agni Mandya* (transient reduction of digestive capacity) due to the large lipid load (6,16). The insulin regimen was maintained. Hypoglycaemia was managed with oral glucose. This was in keeping with standard glycaemic care (17).

One caution must be stated honestly. A large volume of oil was given orally to a patient with reduced consciousness. This carries a real risk of aspiration. Here the gag reflex was confirmed to be preserved, and the oil was given slowly and reflexively. Even so, the approach is not free of hazard. It should be used with great

caution and sound clinical judgement. The regurgitation that occurred underlines this point.

Limitations

This report has several limitations. First, it is a single case. No causal claim about efficacy is possible. This is especially so because a TIA resolves on its own. Second, a peri-intervention lipid profile was not obtained. In a patient with diabetes, triple-vessel disease and CKD who received a large oral lipid load, such a test was indicated. This is acknowledged as a genuine limitation. For context, the patient was already on a statin, and a lipid study was advised among the follow-up tests. A single dietary-oil bolus is unlikely to alter the chronic atherosclerotic burden in a major way. The omission still remains a shortcoming. Third, capillary glucose was documented only once, at onset. Fourth, some elements were subjective, such as the sensation during *Nasya*. Fifth, raw MRI DICOM images were not available. The imaging account relies on the radiologist's verified report. Sixth, oral oleation during reduced consciousness carries an aspiration risk.

Conclusion

This single case documents a home-based Ayurvedic intervention given during an acute transient ischaemic attack. The intervention was *Sadyo Snehana* with groundnut oil and *Nasya* with *Mahanarayana Taila*. The patient was high-risk and multi-morbid. The setting was remote and resource-limited. Full functional recovery followed (Modified Rankin Scale 0), with no recurrence at three months. A TIA resolves on its own. Therefore the contribution of the intervention to recovery cannot be established from a single case. The observations are hypothesis-generating. They warrant systematic, prospective study of safety and of any adjunctive role of such measures in resource-limited settings. Such measures must always work alongside timely conventional emergency care. They must never replace it.

Ethical Compliance

Written informed consent was obtained from the patient for the publication of this case report and the accompanying medical data.

Conflict of Interest: None declared.

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References

1. Jones SP, Baqai K, Clegg A, et al. Stroke in India: a systematic review of the incidence, prevalence and case fatality. *Int J Stroke*. February, 2022; 17(2); 132-140.
2. World Stroke Organization. Global Stroke Fact Sheet 2024. *Int J Stroke*. 2024; 19(1).
3. Loscalzo J, Fauci A, Kasper D, et al. *Harrison's Principles of Internal Medicine*. 21st ed. New York; McGraw-Hill; 2022. 3335p.
4. Shastri K, Chaturvedi G. *Charaka Samhita of Agnivesha with Vidyotini Hindi Commentary, Chikitsa Sthana, Vatavyadhi Chikitsa Adhyaya 28/100-101*. Delhi; Chaukhamba Sanskrit Pratishthan; 2011. 956-957p.
5. Pai KN, Patil GU. Clinical trial of *Nasya Karma* with *Mahanarayana Taila* in *Pakshaghata / ischaemic stroke*. *International Ayurvedic Medical Journal*. April, 2025; 13(4); 931-935.
6. Hunt JN, Stubbs DF. The volume and energy content of meals as determinants of gastric emptying. *J Physiol*. 1975; 245(1); 209-225.
7. Tripathi B. *Ashtanga Hridayam of Vagbhata with Nirmala Hindi Commentary, Sutrasthana, Nasya Vidhi Adhyaya 20/21-22*. Delhi; Chaukhamba Sanskrit Pratishthan; 2015. 247p.
8. Angadi R. *Bhaishajya Ratnavali of Govinda Dasa, Vatavyadhi Chikitsa Prakarana 26/95-99*. Varanasi; Chaukhamba Surbharati Prakashan; 2018. 535-536p.
9. Jamadagni PS, Jamadagni SB, Singh A, et al. Toxicity study of *Swarna Bhasma*, an Ayurvedic medicine containing gold, in Wistar rats. *Toxicol Int*. 2015; 22(3); 11-17.
10. Shah ZA, Vohora SB. Antioxidant / restorative effects of calcined gold preparations used in Indian systems of medicine against global and focal models of ischaemia. *Pharmacol Toxicol*. May, 2002; 90(5); 254-259.
11. Trompetto C, Marinelli L, Mori L, et al. Pathophysiology of spasticity: implications for neurorehabilitation. *Biomed Res Int*. 2014; 2014; 354906.
12. Djupesland PG. Nasal drug delivery devices: characteristics and performance in a clinical perspective - a review. *Drug Deliv Transl Res*. 2013; 3(1); 42-62.
13. Pires A, Fortuna A, Alves G, Falcao A. Intranasal drug delivery: how, why and what for? *J Pharm Pharm Sci*. 2009; 12(3); 288-311.
14. Cheng FC, Shih PH, Wu CH, et al. Neuroprotective effects of sesamin and sesamol on gerbil brain in cerebral ischaemia. *Int J Biomed Sci*. 2006; 2(3); 284-288.
15. Matsumura Y, Kita S, Morimoto S, et al. Antihypertensive effect of sesamin. III. Protection against development and maintenance of hypertension in stroke-prone spontaneously hypertensive rats. *Biol Pharm Bull*. May, 1998; 21(5); 469-473.
16. Paolisso G, Manzella D, Rizzo MR, et al. Elevated plasma fatty acid concentrations stimulate the cardiac autonomic nervous system in healthy subjects. *Am J Clin Nutr*. 2000; 72(3); 723-730.
17. American Diabetes Association Professional Practice Committee. Introduction and Methodology: Standards of Care in Diabetes - 2026. *Diabetes Care*. January, 2026; 49(Suppl 1); S1-S5.
