

A novel psychophysiological Panchakarma protocol for functional dyspepsia integrating the gut–brain axis: a case report

¹Dr Krupa Parmar and ²Dr Sangeeta Toshikhane

¹PG Scholar, Department of Panchakarma, Parul Institute of Ayurved, Parul University, Vadodara, Gujarat, India

²Professor and Head, Department of Panchakarma, Parul Institute of Ayurved, Parul University, Vadodara, Gujarat, India.

Received: 14th June, 2026; Revised: 20th July 2026; Accepted: 20th August, 2026; Available Online: 05th September 2026

ABSTRACT

Background: Functional dyspepsia is a common upper gastrointestinal disorder in which gut–brain axis dysregulation, impaired gastric accommodation, visceral hypersensitivity and psychological co-morbidity coexist, and which responds only partially to pharmacological monotherapy. In Ayurveda the condition corresponds to *Agnimandya* and *Grahani dosha*, attributed to deranged *Samana Vata* and *Pachaka Pitta*.

Case presentation: A 38-year-old woman presented with a five-year history of Rome IV functional dyspepsia, postprandial distress syndrome subtype, refractory to proton pump inhibitors, prokinetics and low-dose tricyclic antidepressants.

Intervention: A 45-day, three-phase *Panchakarma* protocol, designated the Gut–Brain Axis *Panchakarma* Framework, comprising *Takradhara* with a medicated buttermilk containing *Bacopa monnieri*, *Convolvulus pluricaulis*, *Nardostachys jatamansi* and *Asparagus racemosus*; *Shirodhara* with *Sesamum indicum* oil; mild *Virechana*; *Matra basti* with a compound sesame oil formulation; and *Shashtika shali pinda svedana*, sequenced to address the central autonomic, enteric and spinal afferent levels of the axis. Five oral Ayurvedic medicines were given concurrently throughout the 90-day observation period.

Outcomes: Between baseline and day 90 the Leeds Dyspepsia Questionnaire score fell from 22 to 4, the Gastrointestinal Symptom Rating Scale composite from 3.8 to 1.3 and the Perceived Stress Scale-10 from 28 to 11. The root mean square of successive differences rose from 24 to 52 ms, indicating restored cardiac vagal tone. Repeat endoscopy and laboratory safety parameters were normal.

Conclusion: This *Panchakarma* protocol, given with concurrent oral Ayurvedic medicines, was associated with sustained symptomatic, psychometric and autonomic improvement in refractory functional dyspepsia and provides a reproducible framework for controlled trials. Causal inference from a single uncontrolled case is not possible.

Keywords: Functional dyspepsia; Agnimandya; Panchakarma; Takradhara; Gut–brain axis; Heart rate variability; Ayurveda

How to cite this article: Parmar K, Toshikhane S., A novel psychophysiological Panchakarma protocol for functional dyspepsia integrating the gut–brain axis: a case report Int J Drug Deliv Technol. 2026;16(83s): 279-286; DOI: 10.25258/ijddt.16.83s.31

Source of support: Nil.

Conflict of interest: None

1. INTRODUCTION

Functional dyspepsia (FD) is one of the most common functional gastrointestinal disorders encountered in primary and specialist care. Defined by the Rome IV consensus criteria as the presence of one or more of postprandial fullness, early satiation, epigastric pain or epigastric burning that is not explained by structural disease on upper endoscopy, FD affects an estimated 10–30% of the general population worldwide, with substantial heterogeneity across geographic regions and diagnostic criteria¹. In India, the prevalence of dyspepsia in community studies ranges from 7.6% to 30.4%, with

urban, working-age women constituting a substantially burdened subgroup².

The pathophysiology of FD is multidimensional. Peer-reviewed evidence implicates impaired gastric accommodation to meals, antral hypomotility, delayed gastric emptying, duodenal micro-inflammation with increased eosinophil and mast cell infiltration, increased mucosal permeability, visceral hypersensitivity mediated by sensitised mechanoreceptors and chemoreceptors, and bidirectional dysregulation of the gut–brain axis (GBA)³. The GBA, a neuro-hormonal network linking enteric, autonomic, hypothalamic-pituitary-adrenal (HPA) and central nervous systems through the vagus nerve, spinal

afferents, serotonergic signalling and the gut microbiome, is understood to be functionally aberrant in a substantial proportion of patients with FD⁴. Reduced vagal tone,

Current evidence-based pharmacotherapy for FD includes proton pump inhibitors, H₂-receptor antagonists, prokinetic agents (itopride, domperidone, acotiamide), low-dose tricyclic antidepressants (amitriptyline, nortriptyline) and selective serotonin reuptake inhibitors. While these agents provide symptomatic relief in subsets of patients, meta-analytic data report modest effect sizes, high relapse rates on discontinuation and persistent impairment of quality of life in a meaningful proportion of patients⁶. Psychological interventions including cognitive behavioural therapy and gut-directed hypnotherapy show efficacy, particularly where anxiety is prominent, but access barriers limit their adoption⁷.

Ayurvedic classical texts describe a syndrome closely corresponding to FD under the nomenclature Agnimandya (diminished or sluggish digestive fire), *Grahani roga* (disease of the intestinal absorption zone) and *Ajeerna* (indigestion). The Ayurvedic framework conceptualises digestion as dependent on the synergistic function of the thirteen Agni (metabolic fires) described classically, comprising one Jatharagni, five Bhutagni and seven Dhatvagni, together with harmonious *dosha* equilibrium, particularly *Samana Vata*, which governs gastric motility and peristalsis, and *Pachaka Pitta*, which mediates digestive secretions and biochemical transformation⁸. Derangement of *Prana Vata*, the sub-*dosha* governing neuro-cerebral activity, is described as an upstream trigger that precipitates *Samana Vata* dysregulation through a mind-gut continuum that parallels the modern GBA model⁸.

Several Ayurvedic case reports have described the management of Agnimandya or Grahani using internal medicines such as Chitrakadi vati, Agnitundi vati and Hingvashtaka churna. To the authors' knowledge, no published case report has employed a structured *Panchakarma* protocol explicitly conceptualised around gut–brain axis modulation and incorporating concurrent HRV monitoring, validated psychometric instruments and a sequenced psychophysiological framework. The present report addresses this gap by describing a reproducible, theoretically grounded herbal *Panchakarma* protocol for Rome IV FD, applied in a treatment-refractory patient over 45 days with 90-day outcome follow-up.

2. CASE PRESENTATION

2.1. Patient information and history

A 38-year-old married female schoolteacher presented to the *Panchakarma* out-patient department with a five-year history of recurrent upper abdominal distress. Her primary complaints were postprandial fullness persisting for two to three hours after meals (rated 7/10 on a numerical rating scale), early satiation limiting normal meal completion, intermittent epigastric bloating, belching and mild nausea without vomiting. She reported no dysphagia, haematemesis, melaena or unintentional weight loss.

Bowel habits were normal, with neither predominant diarrhoea nor constipation. There was no family history of gastrointestinal malignancy. Menstrual cycles were regular and she was not pregnant at the time of consultation.

Her past medical history included physician-diagnosed Rome IV FD (postprandial distress syndrome subtype), confirmed approximately three years earlier at a tertiary gastroenterology centre, with upper gastrointestinal endoscopy at that time reporting normal-appearing mucosa, no *Helicobacter pylori* on rapid urease test and no structural lesion. She had received pantoprazole 40 mg once daily for twelve months, domperidone 10 mg three times daily for eight months and low-dose amitriptyline 10 mg at night for four months, none of which produced sustained relief. Amitriptyline had been discontinued six months before presentation because of daytime sedation and worsening constipation. Pantoprazole and domperidone were continued unchanged throughout.

2.2. Clinical findings

On detailed Ayurvedic clinical evaluation the patient presented with *Vishama Agni* (irregular digestive fire characterised by variable appetite), *Anaha* (abdominal distension), *Udgarabahulya* (excessive belching) and *Aruchi* (reduced taste and appetite). Her *Prakriti* (constitutional type) was determined as *Vata-Pitta* dominant. Her *Vikriti* (current pathological state) showed aggravated *Samana Vata* and *Pachaka Pitta* with notably impaired *Prana Vata*. Assessment of *Manas Prakriti* (psychological constitution) revealed a *Rajasika-Tamasika* tendency, compatible with chronic stress reactivity and emotional suppression.

Physical examination revealed a thin-built woman (body mass index 19.8 kg/m²), mildly anxious affect and mild epigastric tenderness on deep palpation without guarding or rigidity. Bowel sounds were normal and the tongue was mildly coated. Pulse examination (*Nadi pariksha*) revealed a rapid, fine and irregular *nadi*, consistent with *Vata-Pitta vikriti*. All routine laboratory investigations (complete blood count, liver function tests, renal function tests, thyroid profile, blood glucose and serum electrolytes) were within normal limits.

2.3. Diagnostic assessment

The diagnosis of FD was re-confirmed on the basis of: (i) Rome IV diagnostic criteria, namely bothersome postprandial fullness or early satiation on at least three days per week (postprandial distress syndrome), or bothersome epigastric pain or burning on at least one day per week (epigastric pain syndrome), present over the previous three months with symptom onset at least six months earlier; (ii) exclusion of a structural cause by the previous endoscopy and normal biochemistry; and (iii) absence of alarm features⁹. The postprandial distress syndrome subtype was assigned on the basis of meal-provoked postprandial fullness and early satiation as the dominant symptom cluster.

The Ayurvedic differential diagnosis included *Grahani dosha*, *Visuchika* and *Shoola*. *Grahani dosha* was selected

as the primary Ayurvedic diagnosis on the basis of chronic *Agnimandhya*, altered intestinal function and the classical triad of *Anaha*, *Atopa* (gurgling sounds) and *Aruchi* described in *Charaka Samhita*, *Chikitsa Sthana* 15¹⁰.

Validated instruments administered at baseline comprised the Leeds Dyspepsia Questionnaire (LDQ; 0–40, higher scores indicating worse symptoms), the Gastrointestinal Symptom Rating Scale (GSRS; 1–7 Likert score per domain), the Perceived Stress Scale-10 (PSS-10; 0–40, higher scores indicating greater stress), the Generalised Anxiety Disorder-7 scale (GAD-7; 0–21) and the Pittsburgh Sleep Quality Index (PSQI; 0–21). Baseline scores were LDQ 22, GSRS composite 3.8, PSS-10 28, GAD-7 17 and PSQI 13. Objective autonomic assessment was performed by five-minute resting HRV measurement using a photoplethysmography-based device (Inner Balance; HeartMath, Boulder Creek, CA, USA), capturing the root mean square of successive differences (RMSSD) as the primary parasympathetic index. The baseline RMSSD of 24 ms was consistent with reduced vagal tone¹¹.

3. THERAPEUTIC INTERVENTION: THE GUT–BRAIN AXIS PANCHAKARMA FRAMEWORK

The treatment protocol developed for this case, designated the Gut–Brain Axis *Panchakarma* Framework (GBAPF), is a three-phase, 45-day structured *Panchakarma* intervention designed to modulate the bidirectional gut–brain axis. It is conceptually distinct from conventional *Panchakarma* sequences for digestive complaints in that: (a) each procedure is mapped to a specific neurophysiological or gastrointestinal target; (b) psychometric and HRV monitoring is integrated as a feedback loop within the protocol; (c) the sequencing follows a descending neurological hierarchy from central autonomic modulation, to enteric nervous system (ENS) regulation, to mucosal and luminal restoration; and (d) a medicated buttermilk-based cranial streaming therapy (*Takradhara*) is combined with standard *Panchakarma* procedures. The complete protocol is summarised in Table 1.

3.1. Phase 1: neuropsychological preparation and autonomic re-regulation (days 1–14)

The rationale for Phase 1 rests on the primacy of autonomic dysregulation in FD. Reduced parasympathetic tone, measurable as low RMSSD, is associated with impaired gastric accommodation, attenuated vagal anti-inflammatory signalling and altered brainstem nociceptive modulation¹². Phase 1 aimed to restore parasympathetic dominance before gastrointestinal function was addressed, on the principle that visceral healing is contingent on a conducive autonomic milieu.

Snehapana (internal oleation). The patient received *Shunthi ghrita* (ghee medicated with *Zingiber officinale* Roscoe) 30 ml on an empty stomach each morning for seven days. *Shunthi* is classically described as a *deepana-pachana* (digestive and metabolic stimulant) drug, and its pungent principles 6-gingerol and 6-shogaol have been

reviewed for gastroprotective and prokinetic activity¹³. Ghee served as the lipoidal vehicle, consistent with its Ayurvedic description as a *medhya* (neurotropic) carrier.

Takradhara (medicated buttermilk cranial stream). This procedure, the cornerstone of Phase 1, involved continuous pouring of warm medicated buttermilk (*takra*) over the forehead and scalp in a rhythmic, laminar stream for 45 min daily on days 1–10. The buttermilk base was prepared by churning freshly fermented curd from cow's milk and combining it with a decoction of *Brahmi* (*Bacopa monnieri* (L.) Pennell), *Shankhapushpi* (*Convolvulus pluricaulis* Choisy), *Jatamansi* (*Nardostachys jatamansi* DC.) and *Shatavari* (*Asparagus racemosus* Willd.) in a ratio of 3:2:1:2 respectively, at 5% w/v concentration. The buttermilk was maintained at 36–37 °C throughout the procedure¹⁴.

Proposed rationale for *Takradhara*. Rhythmic thermal and mechanical stimulation of the forehead engages cutaneous thermoreceptors and mechanoreceptors projecting to the trigeminal nucleus, which has anatomical connectivity with brainstem nuclei involved in autonomic integration. This trigemino-vagal route is offered here as a hypothesis to account for the observed autonomic change; it was not tested in this case. Among the botanical constituents, a standardised *B. monnieri* extract reduced anxiety and depression scores in a randomised, double-blind, placebo-controlled trial in older adults¹⁵, while *Jatamansi* is classically described as *medhya* and *nidrajanana* and is used in Ayurvedic practice for anxiety and disturbed sleep¹⁶. Prolonged scalp-level application may additionally engage the neuroendocrine machinery of the skin, since cutaneous keratinocytes, melanocytes and hair-follicle epithelium express a functional equivalent of the HPA axis, including corticotropin-releasing hormone, pro-opiomelanocortin, adrenocorticotrophic hormone and local cortisol synthesis¹⁷.

Sesame oil is rich in sesamin, sesamol and sesamolin. *Shirodhara* has been reported in human studies to reduce salivary cortisol and plasma catecholamines and to alter electroencephalographic activity, consistent with hypothalamic-limbic modulation¹⁸.

3.2. Phase 2: gastrointestinal bio-purification and enteric nervous system modulation (days 15–30)

Phase 2 targeted the gastrointestinal tract directly, once an improved autonomic baseline had been established. Procedures in this phase were chosen for their capacity to reduce duodenal inflammation, to restore normal gastric motility patterns through ENS modulation and to clear accumulated *ama* (metabolic residue) from the gastrointestinal mucosa, a concept that overlaps with contemporary proposals regarding mucosal barrier restoration in FD.

Deepana-Pachana (days 15–17, preparatory digestive kindling). Before therapeutic purgation, the patient received *Trikatu churna* (powders of *Z. officinale*, *Piper nigrum* L. and *Piper longum* L. in equal parts, 3 g twice daily with warm water) for three days, in accordance with

classical pre-purgation practice. Piperine, the principal alkaloid of *P. nigrum* and *P. longum*, is a documented bioavailability enhancer in healthy human volunteers¹⁹.

Mridu Virechana (mild therapeutic purgation, day 18). Controlled mild purgation was induced with *Eranda taila* (castor oil, *Ricinus communis* L.) 15 ml mixed with 100 ml warm cow's milk at bedtime. *Virechana* is the classical *Pitta*-pacifying elimination therapy and *Eranda taila* the classical *mridu virechana* drug²⁰. The intent was to clear the gastrointestinal lumen of accumulated antigenic and dysbiotic substrate, addressing the duodenal micro-inflammation and impaired mucosal integrity described in FD³.

Matra Basti with a compound oil (days 19–30). A daily *matra basti* (small-dose retention enema of 60 ml) was administered using a compound medicated oil prepared specifically for this protocol and designated *Grahani Neuro-Basti Taila*. The formulation was prepared by processing *Tila taila* (sesame oil base, 200 ml) with a decoction of seven botanicals — *Bilva* (*Aegle marmelos* (L.) Corrêa), *Shunthi*, *Musta* (*Cyperus rotundus* L.), *Kutaja* (*Holarrhena pubescens* Wall. ex G.Don), *Bala* (*Sida cordifolia* L.), *Brahmi* and *Ashwagandha* (*Withania somnifera* (L.) Dunal) — in a 4:1 decoction-to-oil ratio, with kalka at one-quarter the weight of the oil, according to classical *taila paka krama*. The oil was administered at 36 °C with retention encouraged for a minimum of 30 min²¹.

Proposed rationale for *Grahani Neuro-Basti Taila*. The colonic route of administration delivers constituents into contact with colonic enteroendocrine cells, submucosal

plexus neurons and the luminal microbial community, and avoids hepatic first-pass metabolism. The seven botanicals were selected from the classical *Grahani* and *Vata*-pacifying groups: *Kutaja* and *Bilva* for *Grahani* and *Atisara*, *Musta* as a *deepana* and *grahi* drug, and *Bala*, *Brahmi* and *Ashwagandha* as *Vata*-pacifying *rasayana* drugs, the last being one of the most widely characterised Ayurvedic adaptogens²². The specific receptor-level mechanisms suggested by these classes of constituents were not measured here and are presented as rationale rather than as demonstrated effects.

3.3. Phase 3: neuroprotective rebuilding and mucosal regeneration (days 31–45)

Phase 3 aimed to consolidate the gastrointestinal gains of Phase 2 while continuing central reconditioning. *Shashtika shali pinda svedana* was administered on days 31–45 as full-body bolus fomentation using warm boluses of cooked *shashtika shali* (60-day rice) prepared in *Bala* root decoction and cow's milk, for 45 min daily. The procedure induces sudation and sustained cutaneous afferent stimulation over the thoracic and lumbar dermatomes and is classically indicated as a *brimhana* (nourishing) and *Vata*-pacifying measure²³.

3.4. Concomitant oral Ayurvedic medicines

Alongside the *Panchakarma* procedures the patient received oral Ayurvedic medicines throughout the 90-day observation period, as detailed in Table 2. These comprised *Shankha vati*, *Sutashekhara rasa*, *Ashwagandha churna*, *Avipattikara churna* and *Yashtimadhu churna* (*Glycyrrhiza glabra* L.), the last selected for its demulcent and mucosal healing properties, for which a comparative clinical trial in peptic ulcer has been reported²⁴.

Table 1: The Gut-Brain Axis Panchakarma Framework, a three-phase Panchakarma intervention.

Phase	Duration	Procedure	Mechanism target	Gut–brain axis level
Phase 1	Days 1–14	<i>Snehapana</i> + <i>Takradhara</i> + <i>Shirodhara</i>	Autonomic and HPA re-regulation; cortisol reduction; vagal tone enhancement	Central → peripheral ANS
Phase 2	Days 15–30	<i>Deepana-Pachana</i> + <i>Virechana</i> + <i>Matra basti</i> (<i>Grahani Neuro-Basti Taila</i>)	Mucosal inflammation; ENS neurotransmitter modulation; microbiome substrate	ENS → portal axis
Phase 3	Days 31–45	<i>Shashtika shali pinda svedana</i> + oral medicines	Visceral pain threshold; neuropeptide modulation; mucosal regeneration	Peripheral → spinal afferents

ANS, autonomic nervous system; ENS, enteric nervous system; HPA, hypothalamic-pituitary-adrenal.

Table 2: Oral Ayurvedic medicines prescribed throughout the protocol.

Drug	Dose	Anupana	Rationale / indication	Duration
<i>Shankha vati</i>	500 mg twice daily	Warm water	Antispasmodic, antacid, carminative; pacifies <i>Samana Vata</i>	90 days
<i>Sutashekhara rasa</i>	250 mg twice daily	Honey	<i>Pachaka Pitta</i> pacifying; gastroprotective; anti-emetic	90 days
<i>Ashwagandha churna</i>	3 g twice daily	Warm milk	<i>Rasayana</i> and adaptogen; <i>Vata</i> -pacifying	90 days
<i>Avipattikara churna</i>	3 g twice daily before meals	Water	Prokinetic, mild laxative; <i>ama-pachana</i>	90 days
<i>Yashtimadhu churna</i>	2 g twice daily	Honey	Mucosal demulcent; anti-inflammatory	90 days

4. OUTCOMES AND FOLLOW-UP

Outcome assessments were performed at baseline (day 0), on completion of the protocol (day 45) and at follow-up (day 90), with an interim review on day 30. The clinical course is summarised in Table 3 and the full outcome data are presented in Table 4.

4.1. Symptomatic outcomes

The LDQ total score declined progressively across all assessment points, with an interim day 30 score of 14. The GSRS composite score fell in parallel, with the most pronounced improvements in the dyspepsia and indigestion sub-domains. Clinically, the patient reported the ability to complete full meals without premature satiation by day 20, with belching and bloating substantially resolved by day 35.

4.2. Psychometric outcomes

PSS-10 and GAD-7 scores declined progressively, with an interim day 30 PSS-10 score of 18. The patient reported improved sleep quality on the PSQI and described a subjective restoration of equanimity in her daily occupational stressors from week 3 onward, concurrent with the *Takradhara* sessions.

4.3. Autonomic outcomes

Resting RMSSD rose progressively from 24 ms at baseline to 35 ms at day 14 (end of Phase 1), 44 ms at day 45 and 52 ms at day 90, a 117% increase in the primary parasympathetic index over 90 days. This trajectory provides an objective, quantified record of autonomic change accompanying the protocol, consistent with increased cardiac vagal tone. The magnitude of change is comparable to that described in a systematic review of HRV outcomes following gut-directed hypnotherapy²⁵.

4.4. Endoscopic and laboratory follow-up

Repeat upper gastrointestinal endoscopy performed on day 90 confirmed the continued absence of structural pathology. A duodenal biopsy obtained on day 90, not performed at baseline, showed normal villous architecture and no eosinophilic infiltration, in contrast with published histopathological findings of elevated duodenal eosinophil counts in FD cohorts²⁶. Liver function tests, renal function tests and complete blood count on day 90 were within normal limits. Serum electrolytes and blood pressure were not re-assessed at day 90; in the absence of these, and of heavy-metal assay of the herbo-mineral preparation used, no positive safety claim is made for the formulations. No adverse events were observed or reported by the patient at any point during the 90-day observation period.

Table 3: Clinical timeline

Timepoint	Event, intervention or finding
2020	Onset of postprandial fullness, bloating and early satiation, attributed to occupational stress
2022	Gastroenterology out-patient department: Rome IV FD (PDS subtype) diagnosed; upper gastrointestinal endoscopy normal; <i>H. pylori</i> negative
2022–2024	Sequential trials of pantoprazole, domperidone and amitriptyline; no sustained relief
January 2025	Referral to <i>Panchakarma</i> out-patient department; baseline assessments: LDQ 22, GSRS 3.8, PSS-10 28, RMSSD 24 ms
Days 1–14	Phase 1: <i>Snehapana</i> (7 days), <i>Takradhara</i> (10 days), <i>Shirodhara</i> days 11–14 (4 sessions)
Days 15–30	Phase 2: <i>Deepana-Pachana</i> , <i>Virechana</i> (day 18), <i>Matra basti</i> (days 19–30)
Day 30	Interim review: LDQ 14, RMSSD 35 ms; belching reduced; improved sleep reported
Days 31–45	Phase 3: <i>Shashtika shali pinda svedana</i> and continued oral medicines
Day 45	Post-protocol: LDQ 7, GSRS 1.9, PSS-10 13, RMSSD 44 ms; full meal completion reported
Day 90	Follow-up: LDQ 4, GSRS 1.3, PSS-10 11, RMSSD 52 ms; endoscopy normal; duodenal biopsy normal

FD, functional dyspepsia; GSRS, Gastrointestinal Symptom Rating Scale; LDQ, Leeds Dyspepsia Questionnaire; PDS, postprandial distress syndrome; PSS-10, Perceived Stress Scale-10; RMSSD, root mean square of successive differences.

Table 4: Outcome measures at baseline, day 45 and day 90.

Outcome measure	Baseline (day 0)	Post-protocol (day 45)	Follow-up (day 90)
Leeds Dyspepsia Questionnaire (0–40)	22	7	4
GSRS composite score (1–7)	3.8	1.9	1.3
GSRS dyspepsia sub-domain (1–7)	4.1	2.1	1.4
Perceived Stress Scale-10 (0–40)	28	13	11
GAD-7 (0–21)	17	10	9
Pittsburgh Sleep Quality Index (0–21)	13	8	7
HRV RMSSD (ms)	24	44	52

GAD-7, Generalised Anxiety Disorder-7; GSRS, Gastrointestinal Symptom Rating Scale; HRV, heart rate variability; RMSSD, root mean square of successive differences.

5. DISCUSSION

This case report makes three contributions to the literature on integrative management of FD. First, it describes the GBAPF protocol, a three-phase *Panchakarma* sequence constructed around contemporary gut–brain axis physiology. Second, it integrates HRV measurement as an objective intra-treatment monitoring tool rather than as a terminal outcome only. Third, it reports a compound colonic oil, *Grahani Neuro-Basti Taila*, formulated for this indication.

5.1. The gut–brain axis in functional dyspepsia

The GBA comprises several principal communication pathways: the vagus nerve, mediating bidirectional neuroimmune signalling; the ENS, with semi-autonomous reflex arcs governing gastric motility; the HPA axis, providing stress-responsive neuroendocrine modulation of mucosal function and immunity; spinal visceral afferents, transmitting nociceptive information; and the gut microbiome, contributing to serotonin precursor availability, short-chain fatty acid signalling and immune regulation²⁷. FD is characterised by dysfunction across several of these pathways simultaneously, which may partly explain the limited efficacy of single-target pharmacotherapy. The GBAPF addresses these pathways in sequence: Phase 1 is directed at the vagus-HPA axis, Phase 2 at the ENS and luminal environment, and Phase 3 at spinal visceral afferents.

5.2. Autonomic outcome and the role of *Takradhara*

The 117% improvement in RMSSD over 90 days is the most notable objective finding. HRV is an established, non-invasive index of cardiac vagal tone reflecting the balance of sympathetic and parasympathetic output. Published data indicate that patients with FD have significantly lower HRV than healthy controls and that HRV correlates inversely with symptom severity scores⁵. The RMSSD trajectory in the present case, from a baseline markedly below the reported healthy adult range to 52 ms at follow-up, represents a clinically meaningful restoration of vagal tone. This is consistent with the clinical framework in which parasympathetic signalling exerts an anti-inflammatory influence on visceral tissues²⁸, although no mechanistic measurement was undertaken here.

The contribution of *Takradhara* warrants specific comment. *Takradhara* is a classical Ayurvedic procedure described principally in the context of *kushtha* (skin disease) and selected psychiatric conditions, and its use in psychosomatic gastrointestinal disease is not described in canonical texts nor, to the authors' knowledge, in the modern literature. Its inclusion in the GBAPF is therefore the principal novel element of the protocol. Because it was delivered together with several other procedures, its independent contribution cannot be isolated from this single case.

5.3. Phytochemical rationale of the formulations

The composition of the *Takradhara* buttermilk is of pharmacological interest. A standardised *B. monnieri* extract improved anxiety and depression scores in a

randomised, double-blind, placebo-controlled trial¹⁵. *Shatavari* is described in Ayurvedic pharmacology as a *madhura-sheeta rasayana* used in *Pitta*-predominant gastric complaints, and its steroidal saponins have been documented in standard pharmacognostic sources^{16,29}. Percutaneous permeation of topically applied compounds through human skin is established³⁰; however, transdermal delivery of the buttermilk-borne constituents was not measured in this case and remains an untested assumption of the protocol's rationale.

Grahani Neuro-Basti Taila was designed to act on the ascending arm of the GBA. Visceral hypersensitivity is a recognised component of FD and is linked to psychological stress³¹, which provided the rationale for including *Musta* and *Kutaja*, both classical *Grahani* drugs. *Bilva*, whose botany, phytochemistry and traditional use have been reviewed, was included on the same classical indication³². These selections are grounded in classical indications and reviewed phytochemistry; the receptor-level mechanisms are hypothesised and were not assessed.

5.4. Durability of benefit

The sustained improvement at 90-day follow-up, well after completion of the 45-day protocol, is clinically relevant, although it does not exclude a placebo or expectation effect. Durability of this kind is consistent with the concept of neuroplastic GBA reconditioning and parallels the maintained benefit reported months after cessation of gut-directed hypnotherapy in functional gastrointestinal disorders³³.

5.5. Limitations

This report describes a single patient without a control, so no causal inference is possible. The individual contributions of the protocol components cannot be disentangled, and the concurrent administration of oral Ayurvedic medicines throughout the observation period further limits attribution of the observed changes to the *Panchakarma* procedures alone. No baseline duodenal biopsy was available for histological comparison. Psychometric outcomes may have been influenced by the therapeutic relationship and by the intensity of the daily contact inherent in *Panchakarma*. The objective markers used (HRV, endoscopy and laboratory safety parameters) are less susceptible to these confounders but do not overcome the limitations of a single-case design. The patient was a woman, and the protocol has not been evaluated in men; sex and gender were not analysed as variables in this report, which limits the generalisability of the findings.

This report describes the application of a structured, multi-phase herbal *Panchakarma* protocol, the Gut–Brain Axis *Panchakarma* Framework, in a patient with Rome IV functional dyspepsia of the postprandial distress syndrome subtype refractory to conventional pharmacotherapy. The protocol was accompanied by clinically meaningful improvement in dyspepsia symptoms and psychometric stress and anxiety scores, and by a 117% increase in resting RMSSD consistent with restored vagal tone,

sustained at 90 days. The inclusion of *Takradhara* with a psychobutanical buttermilk formulation, the use of HRV as an intra-treatment monitoring tool and the compound *Grahani Neuro-Basti Taila* constitute the novel elements of the protocol. As a single uncontrolled observation these findings cannot establish efficacy; they provide a reproducible and theoretically grounded template for randomised controlled trials of *Panchakarma* as a gut–brain axis-modulating intervention in functional gastrointestinal disorders.

Ethical approval and consent

The case was managed and reported in accordance with the principles of the Declaration of Helsinki and the CARE reporting guideline for case reports. Written informed consent was obtained from the patient for publication of this case report and all accompanying clinical details and outcome data, before submission. The patient's identity has been anonymised and no identifying information is included. The signed consent form is retained by the authors and is available to the Editor on request.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

CRedit authorship contribution statement

Krupa Parmar: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Writing – original draft. Sangeeta Toshikhane: Conceptualization, Methodology, Supervision, Both authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The de-identified clinical data supporting the findings of this case report are available from the corresponding author on reasonable request. The data are not publicly available in order to protect patient privacy.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

No generative AI and AI-assisted technologies were used in the preparation of this manuscript.

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