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Integrative Management of Acute Diarrhoea: A Narrative Review of Modern Pharmacotherapy and *Arogyavardhini Vati* in Restoring Digestive Physiology

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ABSTRACT

Acute diarrhoea is a common illness that is usually infective but may also follow dietary, toxic or drug-related insults. Modern management centres on rehydration, with antimotility, antisecretory or antisecretory drugs chosen according to the clinical picture, and these control symptoms quickly. Many patients nevertheless report poor appetite, weak digestion, bloating and fatigue after the loose stools stop. Ayurveda describes the illness as *Atisara* and attributes delayed recovery to weakened Agni, accumulation of Ama and disturbed *Grahani* function. This narrative review sets the biomedical and Ayurvedic accounts of acute diarrhoea side by side, summarizes the mechanisms and positions of loperamide, racecadotril and the combination of ofloxacin and ornidazole, and examines the rationale for using *Arogyavardhini Vati* in the recovery phase. Sources were international guidelines, standard textbooks, classical Ayurvedic texts and indexed literature. The drugs act on motility, secretion and infection respectively and are not a fixed regimen. *Arogyavardhini Vati* is classically described as *Deepana, Pachana, Yakritottejaka,* supportive of *Grahani and Rasayana*, actions that correspond conceptually to appetite, digestion, bile flow, bowel rhythm and strength. It is not a classical indication for *Atisara,* controlled clinical evidence in this setting is lacking, and its metal-containing composition calls for quality assurance and qualified supervision. A sequential model in which modern medicine controls the disease and Ayurvedic measures support restoration of physiology is plausible, but requires well-designed trials before it can be recommended in routine care.

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1. Introduction

Acute diarrhoea is the passage of three or more loose or liquid stools in 24 hours, lasting less than 14 days [1-3]. Infection is the commonest cause, but dietary indiscretion, food intolerance, toxins and drugs such as antibiotics and laxatives also contribute [2,4]. Diarrhoeal disease caused roughly 1.6 million deaths worldwide in 2016 [5]. Oral rehydration, continued feeding and, in children, zinc supplementation remains the foundation of management [1,6,7].

Beyond rehydration, clinicians often add antimotility agents, antisecretory agents or antimicrobials depending on the clinical picture [2,8]. These measures relieve symptoms quickly but do not directly address what follows. A proportion of patients develop post-infectious irritable bowel syndrome [9,10], and the gut microbial community may recover only incompletely after antibiotic exposure [11]. In practice, many patients describe lingering anorexia, heaviness, weak digestion and fatigue after the stools have normalized.

Ayurveda describes the illness as *Atisara*, literally excessive flow, in the Charaka Samhita [12,13], Sushruta Samhita [14], Ashtanga Hridaya [15] and Madhava Nidana [16]. These texts place weakness of Agni (digestive and metabolic capacity), formation of *Ama* (products of incomplete digestion) and disturbance of *Grahani* (the seat of *Agni*) at the centre of both disease and recovery [12,15]. The concept of Agni has also been discussed as a bridge between traditional ideas of digestion and contemporary metabolic health [17]. *Arogyavardhini Vati*, a classical herbo-mineral formulation [18-20], is widely used for impaired digestion and hepatobiliary function, and its stated actions map onto problems seen in the recovery phase.

The objectives of this review are to (i) set the biomedical and Ayurvedic accounts of acute diarrhoea side by side, (ii) summarize the mechanisms and appropriate positions

of loperamide, racecadotril and ofloxacin with ornidazole, and (iii) examine the rationale, evidence and safety considerations for *Arogyavardhini Vati* in the recovery phase within an integrative continuum of care.

2. Materials and Methods

A narrative review design was used. Sources were international clinical guidelines on acute diarrhoea, standard textbooks of medicine and pharmacology, and peer-reviewed articles on the epidemiology, pathophysiology and treatment of acute diarrhoea, the pharmacology of the drugs discussed, post-infectious gut dysfunction, and the safety of metal-containing Ayurvedic products. Classical Ayurvedic sources were the Charaka Samhita (Chikitsa Sthana, chapters 15 and 19), Sushruta Samhita (Uttara Tantra, chapter 40), Ashtanga Hridaya (Nidana and Chikitsa Sthana chapters on Atisara and Grahani), Madhava Nidana (chapter 3), Bhaishajya Ratnavali, Rasa Ratna Samuccaya and the Ayurvedic Formulary of India. Findings were organized under six themes: aetiology and pathophysiology, Ayurvedic interpretation, modern management, residual digestive dysfunction, Arogyavardhini Vati, and the integrative continuum. Because the biomedical and Ayurvedic frameworks use different conceptual categories, correspondences drawn between them are interpretive and are identified as such. No patient data were collected. One clearly labelled hypothetical scenario is included in section 3.7 to illustrate decision points; it is not a case report and reports no outcomes.

3. Results and Discussion of Findings

3.1 Aetiology and pathophysiology

Infective causes include viruses (the commonest cause in children), bacteria and protozoa. Non-infective causes include dietary excess or intolerance, ingested toxins, and drugs such as antibiotics, laxatives and magnesium-containing antacids [2-4]. Mechanistically, acute diarrhoea may be osmotic, secretory, inflammatory or related to altered motility, and these often coexist [4,21].

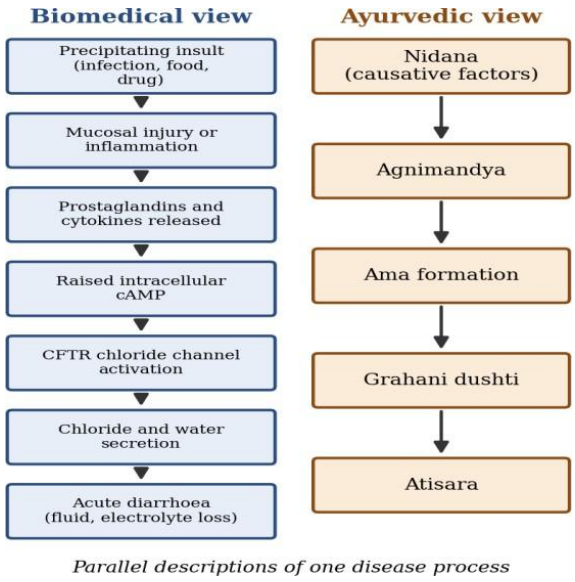


Fig. 1. Parallel biomedical and Ayurvedic descriptions of acute diarrhoea.

In secretory diarrhoea, toxins and mediators raise intracellular cyclic adenosine monophosphate, which activates the cystic fibrosis transmembrane conductance regulator and drives chloride, and with it water, into the lumen [21]. Mucosal injury releases prostaglandins and cytokines that add to secretion, while loss of absorptive enterocytes reduces fluid uptake [4,21]. The net result is loose stool with fluid and electrolyte loss (Fig. 1).

3.2 Ayurvedic interpretation

The classical texts attribute Atisara to dietary and behavioural causes (Nidana), including heavy, excessively unctuous, dry, incompatible or contaminated food and water, indigestion, and emotional disturbances such as fear and grief [12,15,16]. These factors weaken *Agni (Agnimandya),* incompletely digested material accumulates as *Ama*, and the aggravated doshas, mixed with the body's fluid, are expelled with the stool [12,16]. *Grahani*, regarded as the seat of *Agni*, is disturbed (*Grahani dushti),* so digestion and the formation and retention of stool are no longer regulated [12,15].

Classification is by dosha and by cause (Table 1), and a further distinction is made between the state with *Ama (Sama)* and the state without *Ama (Nirama)* [12,14,15]. Management follows this distinction: where *Ama* is present, measures that digest *Ama* and kindle *Agni (Pachana, Deepana, Langhana)* take priority and astringent or binding measures *(Stambhana)* are deferred; once *Ama* has been digested, binding and *Grahani*-supportive measures are used [12,15]. Rehydration and the correction of depletion are recognized concerns in the classical approach, which warns against neglecting a severely depleted patient [12]. These constructs are not equivalent to a single biomedical entity, but they describe a familiar clinical pattern in which impaired digestion precedes and persists beyond the loose stools.

Table 1. Classical types of *Atisara* (after Charaka) [12]

Type	Principal basis and features
Vataja	Vata predominance; scanty, frothy or dry stool with pain and flatus
Pittaja	Pitta predominance; yellowish or greenish stool with burning and thirst
Kaphaja	Kapha predominance; whitish, slimy, heavy stool with coldness
Sannipataja	Involvement of all three doshas with mixed features
Shokaja	Emotional origin (grief); managed with attention to the mental state
Bhayaja	Emotional origin (fear); managed with attention to the mental state

3.3 Modern management

Rehydration with oral rehydration solution is the first priority, with early resumption of feeding, and zinc in children [1,6,7]. Probiotics may modestly shorten the duration of acute infectious diarrhoea [22]. Drug therapy is adjunctive and individualized.

Loperamide is a peripherally acting μ-opioid receptor agonist. By acting on enteric neurons it reduces acetylcholine release and propulsive peristalsis, prolongs transit and allows greater water absorption, thereby reducing stool frequency [23]. It is appropriate for non-invasive watery diarrhoea in adults, but is avoided in bloody diarrhoea, high fever and suspected invasive

infection, and is not recommended in young children because of the risk of serious adverse effects [2,8,24].

Racecadotril is a prodrug whose active metabolite, thiorphan, inhibits enkephalinase. The resulting rise in endogenous enkephalins at intestinal opioid receptors lowers cyclic adenosine monophosphate and reduces chloride and water secretion without slowing motility [25]. A randomized trial in children with acute watery diarrhoea showed reduced stool output [26], and an individual patient data meta-analysis found shorter duration and lower stool volume when it was added to rehydration [27]. Evidence in adults is more limited.

Ofloxacin is a fluoroquinolone that inhibits bacterial DNA gyrase and topoisomerase IV, preventing DNA replication [28]. *Ornidazole* is a nitroimidazole that is reduced within anaerobic bacteria and protozoa to reactive intermediates that damage DNA; the class is active against anaerobes, *Entamoeba histolytica* and *Giardia* [23,29]. A fixed combination therefore covers bacterial and protozoal causes. Guidelines, however, do not support routine antimicrobial use in all acute diarrhoea, because most watery cases are viral or self-limiting, and they reserve antimicrobials for dysentery, febrile invasive disease, moderate to severe travellers' diarrhoea, immunocompromised hosts and confirmed treatable pathogens [2,8].

The three agents therefore act at different points (motility, secretion and infection) and are selected according to the clinical picture; they are not a fixed triple regimen. Their mechanisms and cautions are summarized in Table 2.

Table 2. Drugs used for rapid control of acute diarrhoea

Agent	Mechanism of action	Position and cautions
Loperamide	Peripheral μ-opioid agonist; reduces acetylcholine release and propulsive motility; increases transit time and absorption [23]	Non-invasive watery diarrhoea in adults. Avoid in bloody stool, high fever and young children [2,8,24]
Racecadotril	Enkephalinase inhibitor (active metabolite thiorphan); lowers cyclic adenosine monophosphate and secretion; motility preserved [25]	Adjunct to rehydration in watery diarrhoea; evidence strongest in children [26,27]
Ofloxacin + ornidazole	Inhibition of DNA gyrase and topoisomerase IV; nitroimidazole-induced DNA damage in anaerobes and protozoa [28,29]	Selected invasive bacterial or protozoal disease only. Resistance, adverse effects and microbiota disruption [2,8,11]

3.4 Residual digestive dysfunction

After the acute phase, some patients continue to have reduced appetite, weak digestive capacity, digestive fatigue and altered stool habit. Biomedical correlates include persistent disturbance of the gut microbial community, particularly after antibiotic exposure [11], and the risk of post-infectious irritable bowel syndrome [9,10].

In Ayurvedic terms this state corresponds to lingering *Agnimandya* with residual *Ama* and incomplete restoration of *Grahani* function [12,15]. Reduced hepatobiliary stimulation is also described in the Ayurvedic account, but a distinct biomedical counterpart has not been established and this remains a hypothesis.

3.5 Arogyavardhini Vati

Arogyavardhini Vati is a herbo-mineral *(Rasaushadhi)* preparation described in the Rasa Ratna Samuccaya and Bhaishajya Ratnavali and listed in the Ayurvedic Formulary of India [18-20]. Its ingredients are processed mercury *(Shuddha Parada),* processed sulphur *(Shuddha Gandhaka)*, and the incinerated preparations *(bhasmas)* of iron, mica and copper, combined with Triphala (*Terminalia chebula*, *Terminalia bellerica*, *Emblica officinalis*), purified Shilajatu, purified Guggulu, Chitrakamula (*Plumbago zeylanica*) and Katuki (*Picrorhiza kurroa*), with neem (*Azadirachta indica*) leaf juice as the levigating medium [18,19]. Dose and adjuvant *(anupana)* should follow the formulary and the treating physician.

Classically, the formulation is credited with *Deepana* (kindling Agni), *Pachana* (digestion of Ama), *Yakritottejaka* (stimulation of liver function and bile secretion), support of *Grahani*, and a *Rasayana* (restorative) effect [18,19]. The texts list it chiefly for skin disease and for digestive and hepatobiliary disorders [18,19], so its use after acute diarrhoea is an extension by rational application *(Yukti)*, not a classical indication for *Atisara*. Table 3 sets its classical actions beside the recovery-phase problems they are intended to address. The correspondences are proposed, not demonstrated.

Table 3. Classical actions of *Arogyavardhini Vati* and proposed recovery-phase correlates

Classical action	Proposed correlate in recovery
Deepana	Improved appetite and digestive enzyme activity
Pachana	Less heaviness and bloating; improved metabolism of ingested food
Yakritottejaka	Hepatobiliary stimulation; improved bile secretion and digestion of fat
Grahani support	Improved intestinal digestive capacity, nutrient absorption and bowel rhythm
Rasayana	Support for tissue recovery, strength and general homeostasis

Several constituents, notably *Katuki* and *Guggulu,* are classically regarded as mildly purgative or cholagogue [30]. This is a further reason to position the formulation after the acute loose stools have settled rather than during the acute phase.

3.6 The integrative continuum

Combining the above gives a sequential model (Fig. 2). In the acute phase, rehydration and the selected drug (antimotility, antisecretory or antimicrobial) control symptoms and, where relevant, infection. When acute symptoms have improved but residual digestive dysfunction persists, *Arogyavardhini Vati* is introduced to restore Agni and digestive physiology, and hence intestinal homeostasis and metabolic balance. In this view, modern medicine controls the disease and Ayurveda supports restoration of physiology.

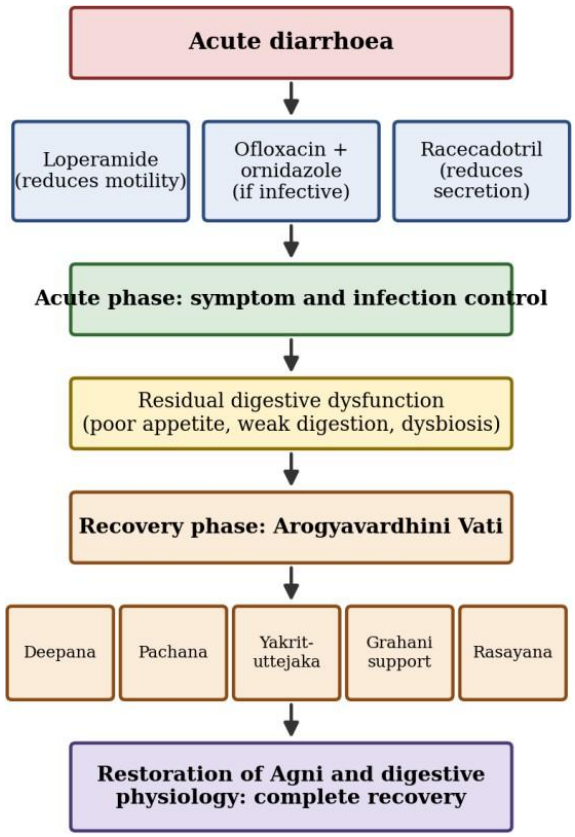


Fig. 2. Proposed integrative continuum from acute control to recovery.

3.7 Illustrative scenario (hypothetical)

This scenario is constructed for teaching purposes. It does not describe a real patient, and no clinical outcomes are claimed.

Consider a healthy adult who develops watery stools several times a day for two days after a meal outside the home, with mild abdominal cramps, no fever, no blood or mucus, and mild thirst but normal urine output.

Acute phase. Hydration status is assessed first [1]. With no warning features, oral rehydration and continued feeding are the mainstay, and stool investigation is not routinely needed. A non-invasive picture such as this would allow an antimotility agent like loperamide, or an antisecretory agent like racecadotril, if symptomatic relief is wanted [2,8]. Antimicrobials such as ofloxacin with ornidazole would not be indicated, because there is no dysentery, fever or other sign of invasive or protozoal disease [2,8]. If blood, high fever, persistent vomiting or dehydration appeared, the plan would change to urgent assessment.

Ayurvedic assessment. The clinician would record the probable causative factors, the dosha pattern, whether Ama is present, and the state of *Agni* [12,15]. If *Ama* predominates, digestion-promoting measures and a light diet would precede any binding measure.

Recovery phase. Suppose that, after the loose stools had settled, the person still felt poor appetite, heaviness and tiredness for several days. This is the situation in which the proposed model would consider *Arogyavardhini Vati*, after baseline liver and kidney tests, with a licensed product, a defined short course, and review of safety [18,31,32]. Appetite, bloating and stool form could be tracked with simple scales [33]. Many such patients recover spontaneously, so any improvement in a single patient could not be attributed to the formulation, which is why controlled studies are needed.

4. Discussion

The integrative model proposed here does not replace standard care. Oral rehydration remains the primary intervention in every patient and reduces diarrhoea mortality [6]. The Ayurvedic recovery-phase component is positioned on top of this foundation, and warning features such as blood in stool, high fever, persistent vomiting, severe dehydration or illness in infants and the elderly call for urgent medical assessment [1,2].

Two points of convergence between the frameworks are worth noting. First, current guidelines restrict antimicrobials and antimotility drugs to defined situations [2,8], and the classical principle of digesting Ama before applying binding measures [12,15] reflects a similar caution against premature suppression. Second, both frameworks recognize that recovery of function lags behind control of symptoms: the biomedical literature documents persistent microbial disturbance and post-infectious bowel symptoms [9-11], and Ayurveda names the corresponding state as impaired *Agni* and *Grahani*.

The principal limitation is evidence. To the authors' knowledge, controlled clinical trials of *Arogyavardhini Vati* in recovery from acute diarrhoea are not available, so its benefit in this setting is hypothesized from classical descriptions and from the plausibility of the correlates in Table 3. The model in Fig. 2 should therefore be read as a framework for generating hypotheses and not as a treatment recommendation. A further limitation is that the correspondences between Ayurvedic and biomedical concepts are interpretive.

Safety deserves particular emphasis. *Rasaushadhi* preparations contain processed mercury and other metals, and analyses of commercially available Ayurvedic products have found detectable lead, mercury or arsenic in a proportion of samples [31,32]. Use should be restricted to products from licensed manufacturers with documented quality control, prescribed and supervised by qualified practitioners, with attention to hepatic and renal function if use is prolonged, and avoided in pregnancy and where the physician judges it unsuitable.

A pragmatic randomized trial is the logical next step. Adults whose acute diarrhoea has been managed according to guidelines and whose acute symptoms have improved could be randomized to standard supportive care alone or with *Arogyavardhini Vati* for a defined short period. Primary outcomes could include time to restoration of normal appetite and stool pattern, measured with a validated stool-form scale and a structured Agni assessment. Secondary outcomes could include symptom scores at follow-up for post-infectious bowel symptoms, hepatic and renal safety markers, and, if resources permit, microbiome profiling. Registration in a clinical trials registry and ethics approval would be required.

5. Conclusion

Modern pharmacotherapy offers rapid, mechanism-based control of acute diarrhoea through reduction of motility,

reduction of secretion or eradication of the pathogen, and these agents are used selectively alongside rehydration. Ayurveda frames the illness as *Atisara* and the recovery period in terms of *Agni, Ama* and *Grahani,* and offers *Arogyavardhini Vati,* whose classical actions correspond conceptually to residual problems of appetite, digestion, bile flow and strength. A sequential integrative approach is therefore biologically plausible, but its clinical value remains unproven. Rigorous trials with careful safety monitoring are needed before it can be recommended in routine practice.

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