

THERAPEUTIC ROLE OF *LEKHANIYA GANA BASTI* IN *STHAULYA* WITH SPECIAL REFERENCE TO DYSLIPIDEMIA: A COMPREHENSIVE REVIEW

Dr. Shriram S. Ragad¹, Dr. Santosh E. Chavan²

¹Ph.D (Scholar), Department of Panchakarma, Bharati Vidyapeeth (Deemed University) College of Ayurved, Pune, Maharashtra, India- 4110043

¹Assistant Professor- Department of Panchakarma, K.V.T.R Ayurveda College, Boradi, Ta-Shirpur, Dist-Dhule, Maharashtra, India- 425428 (0009-0000-0919-8242) shriramragad1992@gmail.com

²Professor & Head of Department of Panchakarma, Bharati Vidyapeeth (Deemed University) College of Ayurved, Pune, Maharashtra, India 4110043 (0009-0001-0195-9577) santosh.chavan@bharativedyapeeth.edu

*Corresponding Author: Dr. Santosh E. Chavan: santosh.chavan@bharativedyapeeth.edu

ABSTRACT

Sthaulya is described in Ayurveda as a *Santarpanajanya* disorder characterized by excessive and functionally disturbed *Meda*, whereas contemporary obesity is a chronic relapsing disease associated with cardiometabolic morbidity. Dyslipidemia commonly accompanying obesity is characterized by abnormalities in triglyceride-rich lipoproteins, HDL cholesterol, non-HDL cholesterol, apolipoprotein B and LDL particle characteristics rather than by a uniform rise in LDL cholesterol alone. The Ayurvedic literature places *Sthaulya* among the clinically undesirable constitutional states and describes dietary excess, reduced physical activity, day sleep and other Kapha-*Meda*-promoting factors in its pathogenesis. *Lekhaniya Mahakashaya* is a ten-drug group described in Charaka Samhita for Lekhana-oriented therapeutic action and is therefore conceptually relevant to Kapha-*Meda*-dominant disorders. (Cha chi 4/5) Recent analytical profiling of *Lekhaniya Mahakashaya* documented a chemically diverse matrix with more than two thousand metabolomics features and validated marker estimation for curcumin and berberine. *Basti* is a major Panchakarma procedure with a strong classical Vata-oriented rationale, and modern rectal-delivery science confirms that ano-rectal administration can provide local or systemic exposure depending on formulation, site, retention and physicochemical properties. A 2025 randomized comparative pilot study in 64 obese participants reported that Lekhan *Basti* alone and in combination with *Navak Guggul* improved anthropometric and lipid-related outcomes, although only 50 participants completed the trial and the open-label pilot design limits certainty. A separate 2025 clinical comparison of two Lekhana *Basti* schedules reported improvement in weight, body measurements and lipid profiles in both groups, while a 2026 protocol demonstrates that more rigorous assessor-blinded controlled evaluation in dyslipidemia is now being pursued. This comprehensive review integrates classical Ayurvedic reasoning, recent clinical evidence, phytochemical and pharmacological literature, rectal-delivery science and contemporary dyslipidemia concepts to evaluate the plausible therapeutic role of *Lekhaniya Gana Basti* in *Sthaulya*. The available evidence supports biological and clinical plausibility but remains insufficient for a definitive claim that a specific *Lekhaniya Gana Basti* protocol provides established lipid-lowering efficacy equivalent to guideline-directed pharmacotherapy.

KEYWORDS: *Sthaulya*; *Lekhaniya Gana*; *Lekhaniya Mahakashaya*; *Lekhana Basti*; *Basti Karma*; *Meda Dhatu*; *Medoroga*; Dyslipidemia; Obesity; *Ayurveda*

1. INTRODUCTION

Obesity is now recognized internationally as a chronic and relapsing disease that arises from complex interactions among biological, behavioral, environmental and societal determinants [1]. The World Health Organization reports that about 890 million adults were living with obesity in 2022 and that adult obesity prevalence has more than doubled since 1990 [1]. A 2024 WHO update also emphasized that approximately one in eight people worldwide were living with obesity, highlighting the population scale of the condition [33]. The Global Burden of Disease 2021 comparative risk assessment places high body mass index within the wider burden attributable to modifiable metabolic and behavioral risks [35]. A pooled analysis of 3,663 population-representative studies involving 222 million participants demonstrated the scale and geographic breadth of the global transition toward higher obesity prevalence between 1990 and 2022 [2]. The clinical importance of obesity extends beyond body weight because excess and dysfunctional adipose tissue contributes to insulin resistance, dyslipidemia, type 2 diabetes, fatty liver disease and atherosclerotic cardiovascular disease [6]. Consequently, a review focused on *Sthaulya* should examine both anthropometric manifestations and metabolic risk markers rather than treating body mass as an isolated endpoint [3].

The most characteristic lipid phenotype associated with increased adiposity is often an atherogenic pattern of elevated triglycerides, reduced HDL cholesterol, increased non-HDL cholesterol, increased apolipoprotein B and a higher concentration of small dense LDL particles [3]. This pattern helps explain why cardiovascular risk in obesity may be underestimated when clinical attention is restricted to LDL cholesterol concentration alone [3].

The 2026 multisociety dyslipidemia guideline accordingly emphasizes broader evaluation of atherogenic lipoproteins, selective apolipoprotein B testing and lifetime lipoprotein(a) measurement in contemporary risk assessment [4]. The same guideline retains lifestyle therapy as a foundation but recommends evidence-based lipid-lowering pharmacotherapy according to cardiovascular risk rather than relying on nonspecific metabolic improvement [5]. Any Ayurvedic intervention proposed for obesity-associated dyslipidemia should therefore be evaluated as an adjunctive or integrative strategy and should not be represented as a substitute for indicated cardiovascular risk management [4].

Ayurveda conceptualizes *Sthaulya* within a functional framework that includes Dosha, Dhatu, Agni, Srotas, dietary qualities and behavioral causes rather than defining the condition by a single numerical threshold [11]. Charaka places *AtiSthaulya* among the Ashta Nindita Purusha and describes characteristic problems such as reduced life expectancy, restricted activity, excessive sweating, excessive hunger and excessive thirst in the obese phenotype [11]. The same classical discussion links the disorder to excessive *Meda* and *Mamsa* accumulation, with disproportionate development of the abdomen, buttocks and breasts and impaired vigor relative to body size [11]. *Santarpana*-dominant foods and habits are repeatedly discussed in classical literature as drivers of Kapha and *Meda* aggravation, providing a therapeutic rationale for *Langhana* and *Lekhana*-oriented measures [12]. These concepts do not create a literal equivalence between *Meda* and circulating cholesterol but offer a systems-level interpretive framework for excess nourishment, altered tissue metabolism and obstructive pathology [12]. *Lekhaniya Mahakashaya* is one of the therapeutic drug groups enumerated in Charaka Samhita and is classically associated with *Lekhana* action [13]. The group is commonly described as containing *Musta*, *Kushtha*, *Haridra*, *Daruharidra*, *Vacha*, *Ativisha*, *Katurohini*, *Chitraka*, *Chirabilva* and *Haimavati*, although botanical identification of *Haimavati* varies across modern secondary sources [22]. A recent analytical dataset authenticated raw materials and characterized the combined formulation using HRLC-MS/MS Orbitrap, HPTLC and FTIR methods, demonstrating a complex phytochemical basis suitable for future quality-control research [22]. The presence of measurable curcumin and berberine in the analytical profile is notable because both compounds have independent contemporary evidence for effects on metabolic or lipid-related outcomes when studied as oral interventions [25]. Such constituent evidence can support plausibility but cannot be directly converted into proof of efficacy for a rectally administered polyherbal *Basti* because dose, extraction, matrix, absorption and pharmacokinetics differ substantially [9].

Basti occupies a central place in Ayurvedic Panchakarma and is classically emphasized for disorders in which Vata is central to the disease process or therapeutic strategy [14]. In *Sthaulya*, a *Basti* approach is frequently justified by the interaction of Vata with Kapha and *Meda* and by the objective of providing Shodhana without relying only on oral Shamana medicines [16]. Modern pharmaceutical science confirms that rectal drug administration can produce local and systemic exposure, but absorption is influenced by rectal fluid volume, formulation composition, solubility, mucosal contact, venous drainage and retention time [10]. Accordingly, mechanistic explanations for *Basti* should distinguish classical Ayurvedic reasoning from modern pharmacological hypotheses rather than presenting speculative analogies as established facts [9]. This distinction is especially important in a review intended for scholarly publication because integrative interpretation is strongest when each knowledge system is described accurately before points of convergence are proposed [16].

The evidence base for *Lekhana Basti* has expanded beyond descriptive case reports, although it remains small compared with conventional obesity and dyslipidemia research [17]. The 2025 randomized pilot study by Munshi and colleagues enrolled 64 obese participants across four groups and incorporated anthropometry, lipid markers, quality-of-life measures and expression of selected obesity-related genes [17]. The study reported significant BMI reduction in the active groups except lifestyle-only control, with maximal lipid reduction in the *Lekhan Basti* and combination groups and differential expression of UCP2, ADIPOR1, PPAR-gamma, FTO, ghrelin and leptin-related transcripts [17]. A 2025 comparative study involving 40 participants reported that both *Yoga Basti* and *Chaturbhadra Kalpa Basti* schedules produced improvements in *Sthoulya* outcomes and lipid profiles, with greater improvements reported for the *Chaturbhadra* schedule [18]. A 2026 randomized assessor-blinded protocol comparing two *Lekhan Basti* formulations with atorvastatin indicates movement toward larger and more methodologically explicit trials, but protocol publication does not constitute evidence of efficacy [19].

The purpose of the present review is therefore not to declare a settled therapeutic effect but to critically integrate the classical rationale, formulation evidence, clinical findings and contemporary metabolic context [16]. Particular attention is given to the distinction between *Sthaulya* and dyslipidemia, because one is a classical Ayurvedic disease construct and the other is a laboratory-defined category of lipoprotein abnormality [3]. The review also examines whether the properties and known constituents of *Lekhaniya Gana* provide a coherent pharmacological hypothesis for lipid and weight-related outcomes [22]. Clinical evidence is interpreted according to study design, sample size, comparator choice, follow-up, safety reporting and the degree to which outcomes were objectively measured [17]. The resulting synthesis is intended to guide future standardized clinical research rather than to encourage unsupervised therapeutic use of *Basti* or potent herbal ingredients [19].

2. Aim and Review Objectives

The primary aim of this review is to evaluate the therapeutic rationale and available evidence for *Lekhaniya Gana Basti* in *Sthaulya* with special reference to dyslipidemia [16]. The first objective is to clarify the classical Ayurvedic pathogenesis of *Sthaulya* and its relationship with *Meda*, *Kapha*, *Agni*, *Vata* and *Medovaha Srotas*

[11]. The second objective is to describe dyslipidemia using current lipoprotein science and to identify the areas where cautious conceptual comparison with *Meda-Dushti* may be academically useful [3]. The third objective is to examine the ingredients, botanical identities, Ayurvedic properties and contemporary phytochemical evidence related to *Lekhaniya Mahakashaya* [22]. The fourth objective is to review the pharmacological and clinical evidence relevant to obesity, lipid metabolism, inflammatory pathways and *Basti* administration without extrapolating beyond the studied formulation or route [17]. The final objective is to identify methodological gaps and propose a clinically meaningful research framework for future randomized trials of standardized *Lekhaniya Gana Basti* [19].

3. REVIEW METHODOLOGY

This article was prepared as a comprehensive narrative review with structured searching because the clinical literature on the exact intervention is too heterogeneous and limited for a reliable quantitative meta-analysis [17]. The review framework uses a comprehensive narrative structure integrating classical Ayurvedic sources with contemporary peer-reviewed evidence relevant to *Lekhana Basti*, obesity and dyslipidemia [16]. Contemporary evidence was prioritized from PubMed-indexed literature, peer-reviewed Ayurveda journals, major guideline sources and recent analytical publications, with emphasis on 2022 to 2026 evidence where available [4]. Older studies were retained when they represented primary clinical evidence directly evaluating *Lekhana Basti* or when they provided foundational rectal-delivery evidence not replaced by a more relevant contemporary source [20]. Classical evidence was drawn from standard descriptions of *Charaka Samhita*, *Sushruta Samhita* and related authoritative Ayurvedic literature used to interpret *Sthaulya*, *Lekhaniya Mahakashaya* and *Basti* [13].

Search concepts included *Sthaulya*, *Medoroga*, *Lekhaniya Gana*, *Lekhaniya Mahakashaya*, *Lekhana Basti*, *Medohara Basti*, obesity, dyslipidemia, lipid profile, rectal drug delivery, adipose inflammation, gut microbiota and bile-acid metabolism [16]. For modern metabolic context, preference was given to recent guidelines, expert reviews, systematic reviews, randomized trials and mechanistic reviews that directly addressed obesity-related dyslipidemia or adipose dysfunction [3]. For Ayurvedic clinical evidence, eligible reports included randomized or comparative studies, prospective clinical studies, protocols and case reports that clearly identified the *Basti* intervention and reported anthropometric, symptomatic or lipid outcomes [17]. Preclinical or constituent-level evidence was included only to explain biological plausibility and was not treated as equivalent to clinical efficacy of the whole formulation [22]. Commercial health claims, unverifiable web material and numerical outcomes that could not be traced to an identifiable publication were excluded from the evidence synthesis [19].

Evidence extraction focused on study design, sample size, participant characteristics, intervention composition or schedule, comparator, duration, outcome domains, principal findings, adverse events and important methodological limitations [17]. Clinical interpretation gave greater weight to controlled human studies than to case reports and greater weight to objective lipid or anthropometric measurements than to unsupported mechanistic statements [17]. Recent constituent research was evaluated separately because oral studies of curcumin, berberine or *Cyperus* extracts cannot establish the efficacy of the same constituents when incorporated into a *Basti* formulation [27]. The review also distinguishes a published protocol from a completed efficacy trial because planned methods and target sample size do not provide treatment outcomes [19]. This hierarchy was used throughout the Results and Discussion sections to prevent the common error of combining classical rationale, preclinical findings and clinical outcomes as though they represent the same level of evidence [16].

The requested one-sentence-one-citation format was applied throughout the narrative so that each substantive sentence is linked to a single source or source category [4]. Where a sentence represents interpretation rather than a direct empirical claim, the citation identifies the principal source from which that interpretation is derived [16]. Numerical results are limited to values reported in the cited studies or directly calculated from those reported values [17]. No synthetic patient dataset, hypothetical lipid response or invented treatment percentage is presented in this review [19]. The resulting article should be read as an evidence-informed scholarly synthesis rather than as a clinical practice guideline or a treatment prescription [4].

3.1 Evidence Sources and Search Framework

Evidence domain	Preferred sources	Key search terms	Role in review
Classical Ayurveda	Charaka Samhita, Sushruta Samhita, Ashtanga Hridaya	<i>Sthaulya</i> , <i>Meda</i> , Santarpana, <i>Lekhaniya</i> , <i>Basti</i>	Classical disease and therapeutic rationale
Clinical Ayurveda	Peer-reviewed Ayurveda and integrative medicine journals	<i>Lekhana Basti</i> , <i>Lekhan Basti</i> , <i>Medoroga</i> , lipid profile	Human efficacy and safety evidence
Modern metabolic science	PubMed, guideline publications, major expert reviews	obesity dyslipidemia, apoB, triglycerides, adipose inflammation	Contemporary disease context
Phytochemistry and constituent pharmacology	PubMed, analytical datasets, pharmacology journals	<i>Lekhaniya Mahakashaya</i> , curcumin, berberine, <i>Cyperus rotundus</i>	Biological plausibility and standardization

Drug-delivery science	Pharmaceutics and pharmacokinetic reviews	rectal drug delivery, enema absorption, rectal formulation	Route-related plausibility and limitations
-----------------------	---	--	--

4. Ayurvedic Concept of *Sthaulya*

Sthaulya is described as a state in which excessive accumulation of *Meda* and *Mamsa* produces disproportionate enlargement of specific body regions and diminished functional capacity [11]. Charaka includes *AtiSthaulya* among eight constitutionally undesirable states, indicating that the concern is not cosmetic appearance but vulnerability to disease and impaired physiological resilience [11]. The classical description emphasizes that the excessively corpulent person may have reduced Ayu, impaired Java (Loss of libido), difficulty in physical activity, excessive perspiration, excessive hunger and excessive thirst [11]. These features allow *Sthaulya* to be interpreted as a multisystem condition in which body composition, appetite regulation, exertional tolerance and metabolic function are simultaneously disturbed [11]. Such a formulation is broader than contemporary BMI classification and therefore requires careful mapping when modern outcomes are used in Ayurvedic clinical research [3].

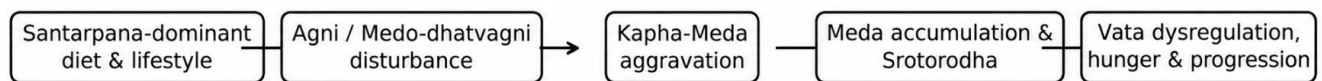
The etiological framework of *Sthaulya* prominently includes excessive intake of *Guru* (Heavy to digest), *Snigdha* (Unctuous) and *Madhura* (Sweet taste)-dominant foods, frequent eating, reduced exercise, daytime sleep and a lifestyle with low physical demand [11]. *Santarpana* is the broader classical construct for pathological over-nourishment and provides a useful etiological context for Kapha and *Meda* accumulation [12]. Classical texts also recognize constitutional or hereditary predisposition through Beeja-related considerations, showing that *Sthaulya* was not understood solely as a behavioral failure [11]. This multidimensional causal model is compatible with the modern recognition that obesity results from interactions among genetic susceptibility, neurobiology, food environment, physical activity, sleep and social determinants [1]. The overlap should be presented as a similarity in multidimensional causation rather than as evidence that the two explanatory systems are identical [16].

Agni is central to Ayurvedic explanations of transformation and tissue nourishment, and disturbance of *Agni* provides a functional link between dietary excess and abnormal Dhatu formation [12]. In *Sthaulya*, the clinical paradox of excessive appetite despite excessive tissue accumulation is explained through altered Vata movement associated with obstruction by *Meda* [11]. This creates a self-perpetuating cycle in which increased intake and impaired tissue-level regulation contribute to additional *Meda* accumulation [11]. The concept of *Medo-dhatvagni* is therefore useful for describing qualitative abnormality of *Meda* metabolism rather than simply the absolute quantity of adipose tissue [12]. Contemporary research similarly distinguishes metabolically dysfunctional adipose tissue from adipose mass alone because inflammation, ectopic lipid flux and insulin resistance influence disease risk [32].

Kapha and *Meda* share several classical qualities, including heaviness and unctuousness, which makes Kapha-*Meda* aggravation a coherent therapeutic target in *Sthaulya* [12]. Lekhana-oriented treatment is selected to oppose pathological accumulation through qualities and actions such as *Laghu*, *Ruksha*, *Tikshna*, *Deepana* and *Pachana* when clinically appropriate [13]. The principle is not equivalent to physically scraping fat from tissues, and the term *Lekhana* should be interpreted within Ayurvedic pharmacodynamic language rather than through a literal mechanical analogy [16]. A scientifically cautious translation is that Lekhana denotes a therapeutic direction intended to reduce pathological excess or accumulation within the classical framework [13]. This interpretation permits modern study of weight, waist measurements and lipid markers without claiming that the classical concept predicted a specific molecular mechanism [17].

Srotorodha is an important component of the classical pathogenesis because excessive *Meda* is described as interfering with normal movement and distribution within bodily channels [11]. The functional consequence of obstruction includes altered Vata behavior, reduced physical capacity and perpetuation of abnormal appetite and tissue deposition [11]. *Srotas* terminology should not be translated directly into arteries, lymphatics or any single anatomical conduit because its classical scope is functional and context-dependent [16]. Nevertheless, the concept is clinically useful for organizing symptoms and choosing *Shodhana* or *Srotoshodhana*-oriented therapy within Ayurveda [14]. In integrative research, *Srotorodha* may serve as a hypothesis-generating construct but should not be used as a substitute term for atherosclerotic plaque or microvascular disease [3].

The symptomatic assessment of *Sthaulya* should include classical complaints together with measurable anthropometry because the disease construct extends beyond body weight [17]. Useful contemporary outcomes include weight, BMI, waist circumference, waist-to-hip ratio and, where feasible, body composition measures that distinguish total mass from central adiposity [3]. Metabolic assessment is especially appropriate when the review focuses on dyslipidemia because atherogenic lipid abnormalities may persist despite modest changes in body weight [3]. Quality-of-life measures are also relevant because obesity can limit activity, social function and perceived health even when laboratory changes are small [17]. A balanced Ayurvedic trial should therefore combine validated classical symptom scores with objective anthropometric, biochemical and safety endpoints [19].



Conceptual Ayurvedic sequence synthesized from classical descriptions of Sthaulya and Santarpanajanya disorders

Figure 1. Conceptual Samprapti sequence of Sthaulya synthesized from classical descriptions of AtiSthaulya and Santarpanajanya disorders [11,12].

5. Meda Dhatu, Medovaha Srotas and Metabolic Interpretation

Meda is one of the seven Dhatus and contributes to lubrication, energy reserve, structural support and other physiological functions when present in an appropriate qualitative and quantitative state [11]. Ayurvedic pathology distinguishes normal *Meda* from excessive or vitiated *Meda*, which is important because the therapeutic goal is restoration of balanced tissue function rather than indiscriminate depletion [12]. *Meda*-Dushti therefore encompasses disturbance of tissue quality, quantity, distribution and metabolic processing within the classical system [12]. This distinction resembles the modern observation that adipose tissue can be metabolically healthy or dysfunctional depending on inflammatory state, ectopic lipid spillover and insulin sensitivity [32]. The resemblance is conceptual and does not imply that *Meda* Dhatu is anatomically or biochemically identical to adipose tissue [16].

Medovaha Srotas provides an Ayurvedic framework for the production, transport and functional integrity of *Meda*-related processes [12]. Factors that aggravate *Meda* or disturb Agni are therefore relevant not only to accumulation but also to the quality of tissue nourishment and metabolic distribution [12]. The resulting pathology can be interpreted through *Srotodushti* patterns such as obstruction or abnormal flow depending on the clinical context [14]. Modern obesity research likewise recognizes altered trafficking of free fatty acids and triglyceride-rich lipoproteins between adipose tissue, liver and peripheral tissues as central to metabolic dysfunction [3]. These two descriptions should remain parallel explanatory models rather than being collapsed into a single terminology [16].

Contemporary adipose biology demonstrates that expansion of adipose tissue can be accompanied by macrophage infiltration, inflammatory signaling, oxidative stress and impaired insulin action [7]. Inflammation in adipose tissue is also linked to dysregulated lipid metabolism and contributes to metabolic diseases including atherosclerosis, insulin resistance and fatty liver disease [32]. These findings provide a plausible modern explanation for why excess adiposity can produce systemic consequences even before severe hypercholesterolemia appears [3]. Ayurvedic descriptions of *Meda* excess similarly emphasize functional impairment and disease susceptibility rather than treating increased body size as an isolated physical trait [11]. The convergence supports combined anthropometric and metabolic assessment but does not validate one framework by simply translating terms from the other [16].

Agni and Ama are frequently invoked in Ayurvedic discussions of metabolic disease, but their use in scientific writing requires precise contextual definition [12]. Agni can be described as the classical principle governing digestion and transformation, while Ama denotes incompletely processed or pathological material in specific Ayurvedic contexts [12]. Neither concept has a validated one-to-one biomarker in contemporary laboratory medicine [16]. Accordingly, inflammatory cytokines, oxidized lipoproteins or insulin resistance should not be labeled as Ama unless the statement is clearly presented as an interpretive hypothesis rather than a demonstrated equivalence [6]. This distinction improves academic rigor and prevents apparently scientific language from masking an unsupported translation [16].

6. Dyslipidemia: Contemporary Perspective

Dyslipidemia refers to abnormal concentrations or distributions of circulating lipids and lipoproteins and includes phenotypes involving LDL cholesterol, triglycerides, HDL cholesterol, remnant particles, apolipoprotein B and lipoprotein(a) [4]. The 2026 multisociety guideline broadened the terminology from blood cholesterol management to dyslipidemia management in order to reflect cardiovascular risk associated with atherogenic lipoproteins beyond LDL cholesterol alone [4]. The American College of Cardiology release accompanying the 2026 guideline likewise emphasized updated risk assessment and contemporary lipid-management recommendations [34]. This is particularly relevant in obesity because LDL cholesterol may rise only modestly while triglyceride-rich lipoproteins, remnant cholesterol, apolipoprotein B and small dense LDL particles become more abnormal [3]. A review centered on *Sthaulya* should therefore avoid defining dyslipidemia only as high total cholesterol [3]. Complete lipid evaluation also provides a more meaningful safety and efficacy endpoint for trials that claim metabolic benefit [19].

Increased adiposity can promote hepatic overproduction of very-low-density lipoprotein, altered triglyceride handling, reduced HDL cholesterol and accumulation of atherogenic remnant particles [3]. Insulin resistance amplifies these processes by increasing free-fatty-acid flux from adipose tissue to the liver and altering lipoprotein lipase-dependent metabolism [6]. Adipose inflammation can further impair insulin signaling and contribute to a cycle of glucose-lipid dysregulation [7]. The cardiovascular significance of this phenotype lies in the increased

burden of apolipoprotein B-containing particles that can enter and be retained in the arterial wall [4]. For this reason, improvement in body weight alone cannot be assumed to normalize cardiovascular risk [3].

Weight reduction of at least five percent is commonly associated with improvement in triglycerides, while larger reductions may improve HDL cholesterol and broader cardiometabolic risk [3]. The effect of weight loss on LDL cholesterol is often smaller and more variable than its effect on triglycerides and HDL cholesterol [3]. This pattern is useful when interpreting Ayurvedic obesity trials because a meaningful anthropometric response may not produce parallel changes in every lipid fraction [17]. Investigators should therefore prespecify primary lipid endpoints rather than selectively emphasizing whichever fraction improves after treatment [19]. ApoB and non-HDL cholesterol are particularly useful in future studies when triglycerides are elevated or when residual atherogenic particle burden is a concern [4].

The current standard of care for clinically significant dyslipidemia remains risk-based lifestyle and pharmacological management according to guideline recommendations [5]. Statin therapy continues to be the pharmacological foundation for many individuals at elevated atherosclerotic cardiovascular risk, including patients with persistent hypertriglyceridemia when the primary objective is ASCVD risk reduction [4]. Additional therapies are selected according to LDL cholesterol, triglycerides, risk category, comorbidity and treatment response [5]. Consequently, evidence for *Lekhaniya Gana Basti* should be framed as potential adjunctive metabolic evidence unless adequately powered trials demonstrate clinically important cardiovascular or lipid outcomes [19]. No current clinical study of *Lekhana Basti* establishes equivalence or non-inferiority to comprehensive guideline-directed lipid management [17].

7. Ayurvedic Interpretation of Dyslipidemia

Dyslipidemia has no exact classical disease label because modern lipoprotein measurements were not part of the diagnostic framework of Ayurveda [16]. Ayurvedic authors often interpret abnormal lipid profiles through *Meda*-*Dushti*, *Medoroga*, Kapha predominance, *Santarpana*, *Agnimandya* and *Srotodushti* when the patient phenotype supports those features [20]. This interpretive approach is more defensible than stating that serum cholesterol is simply *Meda* Dhatu [16]. *Meda* is a tissue concept with physiological and pathological roles, whereas cholesterol is a specific molecule carried within multiple lipoprotein particles [3]. Preserving this distinction allows classical reasoning to be used clinically without making a false biochemical equivalence [16].

The association between *Sthaulya* and dyslipidemia is also incomplete because some people with obesity have relatively normal routine lipid values and some people with dyslipidemia are not obese [3]. A study population should therefore confirm both the Ayurvedic diagnosis and the modern lipid abnormality when the research question specifically concerns *Sthaulya* with dyslipidemia [19]. This dual case definition improves interpretability and avoids assuming that every patient with *Sthaulya* has the same metabolic phenotype [3]. It also allows subgroup analysis according to triglyceride-predominant, LDL-predominant or mixed dyslipidemia in future adequately powered trials [4]. Such stratification may reveal whether a *Basti* intervention influences body composition and lipoprotein metabolism through partially independent pathways [17].

From an Ayurvedic therapeutic perspective, the combination of Kapha-*Meda* aggravation with Vata dysregulation offers a rationale for selecting a *Basti* that incorporates *Lekhana*-oriented drugs [16]. The intended treatment direction includes reduction of pathological heaviness or accumulation, support of Agni, improvement of Srotas function and normalization of Vata according to the classical framework [13]. These actions remain traditional pharmacodynamic concepts and should not be relabeled as specific molecular targets without experimental confirmation [16]. Modern mechanistic hypotheses can instead be tested separately through lipidomics, inflammatory markers, insulin sensitivity, microbiome analysis or pharmacokinetic studies [22]. This two-layer model preserves classical authenticity while allowing rigorous contemporary testing [16].

8. Lekhaniya Gana: Classical Basis and Composition

Charaka Samhita describes *Lekhaniya Mahakashaya* as a group of ten drugs selected for *Lekhana*-oriented therapeutic action [13]. The commonly cited list includes *Musta*, *Kushtha*, *Haridra*, *Daruharidra*, *Vacha*, *Ativisha*, *Katuohini*, *Chitraka*, *Chirabilva* and *Haimavati* [22]. Recent analytical research at the All India Institute of Ayurveda used authenticated raw materials and contemporary instrumental methods to characterize the combined formulation [22]. The study identified 1,712 metabolomics features in positive ion mode and 322 in negative ion mode, illustrating the chemical complexity of the polyherbal matrix [22]. HPTLC marker estimation in the same dataset reported measurable berberine and curcumin, which provides useful quality-control anchors but does not define the activity of the entire formulation [22].

The classical rationale of *Lekhana* is commonly associated with predominance of *Katu*, *Tikta* and *Kashaya* tastes together with *Laghu*, *Ruksha* and *Tikshna* qualities where applicable to the individual drug [13]. These attributes are traditionally considered antagonistic to excessive Kapha and *Meda* and are therefore used to explain the group-level therapeutic direction [13]. *Deepana* and *Pachana* actions are relevant because *Sthaulya* is not interpreted solely as a storage disorder but also as disturbed transformation and tissue metabolism [12]. *Srotoshodhana* is invoked to address obstruction and impaired functional movement within the classical Srotas framework [14]. The complete therapeutic effect of a polyherbal *Basti* cannot be predicted by adding isolated *Rasapanchaka* descriptors because preparation method, dose, adjuvants and *Basti* schedule influence the clinical intervention [16].

Botanical authentication is a major research requirement because several classical names have regional or textual synonyms and modern sources may assign different botanical identities [22]. Haimavati is a particularly important example because some contemporary lists identify it as an *Iris* species while some Charaka-oriented electronic resources use Haimavati as a synonym of *Vacha* or *Acorus calamus* [30]. A formulation that contains both *Vacha* and Haimavati therefore requires explicit pharmacognostic documentation rather than reliance on an unqualified secondary list [22]. The issue is not merely taxonomic because variation in species changes phytochemical exposure, toxicological profile and reproducibility [22]. Future clinical studies should preserve voucher specimens or authenticated reference samples for every ingredient in the *Basti* formulation [19].

8.1 Composition and Research-Relevant Identity

Drug	Common botanical identity in recent LMK literature	Part commonly used	Key Ayurvedic orientation	Research note	Ref.
<i>Musta</i>	<i>Cyperus rotundus</i> L.	Rhizome	Lekhana; Kapha-Meda relevant	Recent RCT evidence exists for a standardized oral extract in obesity; route and extract differ from <i>Basti</i> .	[22,28]
<i>Kushtha</i>	<i>Saussurea costus</i> (Falc.) Lipsch. / <i>S. lappa</i>	Root	Katu-Tikta; Deepana-oriented	Recent pharmacology reviews describe sesquiterpene-rich phytochemistry; direct obesity trials are limited.	[22,24]
<i>Haridra</i>	<i>Curcuma longa</i> L.	Rhizome	Katu-Tikta; Ruksha; Kapha-Pitta relevant	Curcuminoids have contemporary metabolic trial evidence, but oral curcumin data cannot establish <i>Basti</i> efficacy.	[23,27]
<i>Daruharidra</i>	<i>Berberis aristata</i> DC.	Stem/root	Tikta-Kashaya; metabolic relevance	Berberine is a marker compound with RCT/meta-analysis evidence for lipid effects.	[22,25]
<i>Vacha</i>	<i>Acorus calamus</i> L.	Rhizome	Katu-Tikta; Tikshna; Kapha-Vata relevant	Species and beta-asarone content require safety-oriented quality control.	[22,31]
<i>Ativisha</i>	<i>Aconitum heterophyllum</i> Wall. ex Royle	Root	Tikta-Katu; Deepana-Pachana orientation	Authentication is essential because <i>Aconitum</i> genus substitutions can materially alter toxicity.	[22,31]
<i>Katurohini</i> / <i>Katuki</i>	<i>Picrorhiza kurroa</i> Royle ex Benth.	Rhizome/root	Tikta; Pitta-Kapha oriented	Often discussed for hepatometabolic relevance; direct human lipid evidence for the LMK context is limited.	[22]
<i>Chitraka</i>	<i>Plumbago zeylanica</i> L.	Root	Katu; Ushna; Deepana	Plumbagin-containing material is	[22,31]

				pharmacologically active and demands dose standardization.	
<i>Chirabilva</i>	<i>Holoptelea integrifolia</i> (Roxb.) Planch.	Stem bark	Lekhana/Bhedana orientation	Preclinical hypolipidemic findings are cited in recent LMK analytical literature.	[22]
<i>Haimavati</i>	Identity varies in secondary sources; Iris species are used in some LMK publications	Root/rhizome depending on identity	<i>Lekhaniya</i> group member	Botanical identity should be explicitly authenticated because modern sources are inconsistent.	[22,31]

9. Pharmacological Evidence Relevant to *Lekhaniya* Gana

Constituent-level pharmacology can strengthen the biological plausibility of *Lekhaniya* Gana, but it cannot substitute for testing the complete *Basti* formulation in the intended patient population [22]. Polyherbal preparations contain multiple classes of alkaloids, terpenoids, phenolics and other compounds whose extraction and bioavailability depend on the vehicle and preparation process [22]. The 2026 multi-analytical dataset confirms this complexity by documenting thousands of detectable features across complementary analytical platforms [22]. Such chemical diversity makes a single-compound mechanism improbable and supports a multi-target hypothesis that still requires experimental validation [22]. Standardization should therefore combine botanical authentication, chromatographic fingerprinting, marker quantification and batch-to-batch acceptance criteria [29].

Haridra is one of the best-studied members of the group because curcumin and related curcuminoids have been evaluated in numerous metabolic clinical trials [27]. A 2025 systematic review and meta-analysis of randomized trials of *Curcuma longa* preparations across metabolic-syndrome-related conditions reported effects on several anthropometric, glycemic, lipid, inflammatory and oxidative-stress outcomes, although formulations and populations were heterogeneous [27]. A 2026 Ayurveda-focused review specifically revisited Haridra within *Lekhaniya Mahakashaya* and highlighted contemporary evidence relevant to obesity and dyslipidemia [23]. These findings justify Haridra as a pharmacological research focus but should not be used to infer the magnitude of effect of Haridra when delivered as one component of a large-volume *Basti* [23]. Future formulation work could quantify curcuminoid recovery in the final *Basti* liquid and examine whether measurable systemic exposure occurs after administration [22].

Daruharidra is pharmacologically relevant because *Berberis* species contain berberine and the 2026 LMK dataset quantified berberine as a marker in the combined formulation [22]. A systematic review and meta-analysis of 18 randomized placebo-controlled trials involving 1,788 adults found that berberine modestly reduced LDL cholesterol, total cholesterol, triglycerides and apolipoprotein B while slightly increasing HDL cholesterol [25]. A later meta-analysis of metabolic-syndrome components also reported reductions in triglycerides and waist circumference, supporting a broader metabolic research rationale for berberine-containing botanicals [26]. These data primarily concern orally administered berberine products and therefore cannot establish the pharmacokinetics or clinical effect of *Daruharidra* in *Basti* [9]. Nevertheless, berberine provides a measurable quality-control marker and a plausible candidate for mechanistic studies when present in a standardized preparation [22].

Musta has gained recent direct clinical interest because a standardized *Cyperus rotundus* extract was evaluated in a randomized double-blind placebo-controlled 90-day obesity trial [28]. That 2025 trial enrolled 96 participants and reported significant reductions in body weight, BMI, waist and hip circumference with the standardized extract plus piperine compared with placebo under a diet and exercise program [28]. The trial is relevant to ingredient plausibility but differs from *Lekhaniya* Gana *Basti* in extract standardization, oral administration, coadministration of piperine and treatment duration [28]. The evidence, therefore supports research interest in *Musta* but not a direct claim that *Musta* contributes the same magnitude of effect within a *Basti* matrix [28]. A mechanistic study of the full formulation should examine whether *Cyperus*-derived compounds are present in the administered decoction and biologically available after rectal exposure [22].

Kushtha or *Saussurea costus* contains a chemically rich profile that includes sesquiterpene lactones and has been reviewed for antioxidant, anti-inflammatory and diverse pharmacological activities [24]. The 2024 comprehensive review of *Saussurea costus* emphasizes substantial preclinical pharmacology but does not establish a specific clinical lipid-lowering effect in *Sthaulya* [24]. This illustrates an important limitation of ingredient-based reasoning because broad pharmacological activity in vitro or in animals does not predict efficacy in a complex human intervention [24]. The plant is also conservation-sensitive in some regions, making authenticated and legally sourced raw material important for reproducible research [24]. Future studies should document species identity and supply-chain quality rather than using the traditional name alone [22].

9.1 Summary of Ingredient-Level Evidence

Ingredient marker	Evidence type	Relevant reported finding	Major limitation for this review	Ref.
<i>Curcuma longa</i> / <i>curcuminoids</i>	Systematic review and meta-analysis of RCTs	Metabolic, anthropometric and lipid-related effects across heterogeneous metabolic populations	Mostly oral standardized supplements; not <i>Basti</i> -specific	[27]
<i>Berberis</i> / <i>berberine</i>	Systematic review and meta-analysis of placebo-controlled RCTs	Modest reductions in LDL-C, total cholesterol, triglycerides and apoB	Oral berberine products; cannot infer rectal polyherbal exposure	[25]
<i>Cyperus rotundus</i>	Randomized double-blind placebo-controlled trial	Reductions in body weight, BMI and circumferences with standardized extract plus piperine	Different extract, route and cointervention	[28]
<i>Saussurea costus</i>	Comprehensive pharmacology review	Broad phytochemical, antioxidant and anti-inflammatory evidence	Clinical obesity/dyslipidemia evidence remains limited	[24]
Whole <i>Lekhania</i> Mahakashaya	HRLC-MS/MS, HPTLC and FTIR dataset	2034 metabolomics features and quantified marker compounds	Analytical evidence demonstrates composition, not clinical efficacy	[22]

10. Concept of *Basti* Therapy and Relevance to *Sthaulya*

Basti is a major Panchakarma intervention described in classical Ayurveda and is traditionally regarded as especially important in the management of Vata-related pathology [14]. Ashtanga Hridaya also preserves the central therapeutic place of *Basti* within Panchakarma while emphasizing selection according to the patient and clinical context [15]. The procedure includes decoction-based Niruha or Asthapana approaches and oil-dominant Anuvasana approaches used according to patient strength, disease, digestive status and therapeutic objective [14]. Clinical protocols frequently sequence these forms rather than using a single enema composition repeatedly [17]. The 2025 randomized pilot study used three 15-day cycles containing six Niruha and nine Anuvasana *Basti* administrations per cycle with rest intervals between cycles [17]. This schedule demonstrates why the term Lekhan *Basti* should not be treated as a uniform exposure unless formulation and timing are explicitly reported [17].

The classical therapeutic rationale for *Basti* in *Sthaulya* includes management of Vata while addressing Kapha-Meda predominance through appropriately selected drugs and vehicles [16]. This is relevant because excessive Meda is classically described as producing obstruction that alters Vata movement and contributes to excessive appetite and disease progression [11]. A Lekhana-oriented *Basti* therefore aims to combine Shodhana with a formulation designed to oppose pathological accumulation rather than merely inducing bowel evacuation [16]. The clinical effect cannot be reduced to laxation because *Basti* protocols differ in retention, composition, oil content and sequence [17]. Future studies should report evacuation time, retained duration, procedural tolerability and adherence to distinguish pharmacological exposure from nonspecific bowel effects [19].

Modern rectal-delivery science provides a plausible route-based framework but should not be presented as a complete explanation of classical *Basti* [9]. The rectum can support both local and systemic delivery, and absorption depends on solubility, lipophilicity, formulation, mucosal contact and venous drainage [9]. Liquid formulations such as enemas may release dissolved compounds more rapidly than solid rectal dosage forms, although systemic bioavailability can be variable [10]. Partial avoidance of hepatic first-pass metabolism can occur for drugs absorbed from the lower rectum, but the extent depends on the site of absorption and the compound [10]. These principles justify pharmacokinetic investigation of *Basti* but do not prove that every herbal constituent reaches systemic circulation in an active concentration [9].

The gastrointestinal tract also interacts extensively with bile acids, microbial metabolites and host metabolic signaling, creating additional hypotheses for rectally delivered interventions [8]. Bile acids function not only in lipid digestion but also as signaling molecules that interact bidirectionally with the gut microbiota and influence metabolic pathways [31]. Obesity and chronic metabolic disease are associated with changes in gut microbial ecology and bile-acid signaling, although causality and interindividual variability remain major research challenges [8]. It is therefore reasonable to study whether a retained *Basti* modifies local microbial or metabolite profiles, but current Lekhana *Basti* trials do not establish such a mechanism [17]. Microbiome explanations should

remain explicitly hypothesis-generating until direct pre-treatment and post-treatment measurements are available [8].

11. Rationale for *Lekhaniya Gana Basti* in *Sthaulya* with Dyslipidemia

The therapeutic rationale for *Lekhaniya Gana Basti* is strongest when expressed as a combination of classical formulation logic and testable modern hypotheses [16]. At the classical level, *Lekhaniya* drugs are selected for a therapeutic direction that opposes Kapha-*Meda* excess and supports Deepana, Pachana and Srotas-oriented correction according to the properties of individual ingredients [13]. *Basti* adds a procedure-based strategy traditionally centered on Vata regulation and Shodhana [14]. The combined approach is therefore coherent for a *Sthaulya* phenotype in which Kapha-*Meda* excess coexists with Vata disturbance and abnormal appetite or functional obstruction [11]. The logic remains diagnosis-dependent and should not be generalized to every patient with an abnormal lipid report [16].

At the contemporary level, several components of the formulation contain compounds with reported effects on lipid metabolism, inflammation or anthropometric outcomes when studied in other dosage forms [22]. Berberine provides the clearest lipid-related human evidence among the identified marker compounds, while curcumin provides a broader metabolic and inflammatory evidence base [25]. Musta also now has a recent randomized obesity trial using a standardized oral extract [28]. These findings support a multi-component metabolic hypothesis but cannot establish the pharmacologically active dose delivered by *Basti* [9]. A properly designed mechanistic trial would therefore quantify the administered formulation and measure systemic or local exposure to selected marker compounds [22].

The most clinically meaningful hypothesis is that *Lekhaniya Gana Basti* may improve a cluster of outcomes including *Sthaulya* symptoms, BMI, central adiposity and selected lipid parameters when used with standardized lifestyle management [17]. This hypothesis is more defensible than claiming a direct cholesterol-scraping effect because contemporary lipid physiology involves multiple particle classes and regulatory pathways [3]. It also accommodates the observation that *Basti* trials may show changes in BMI and symptoms without uniform changes in every lipid fraction [17]. The 2025 pilot study provides preliminary support for this multidomain outcome model but its small sample and open-label design require confirmation [17]. The 2026 controlled trial protocol provides a useful template for more rigorous comparison with standard therapy but results are not yet available in the cited protocol publication [19].

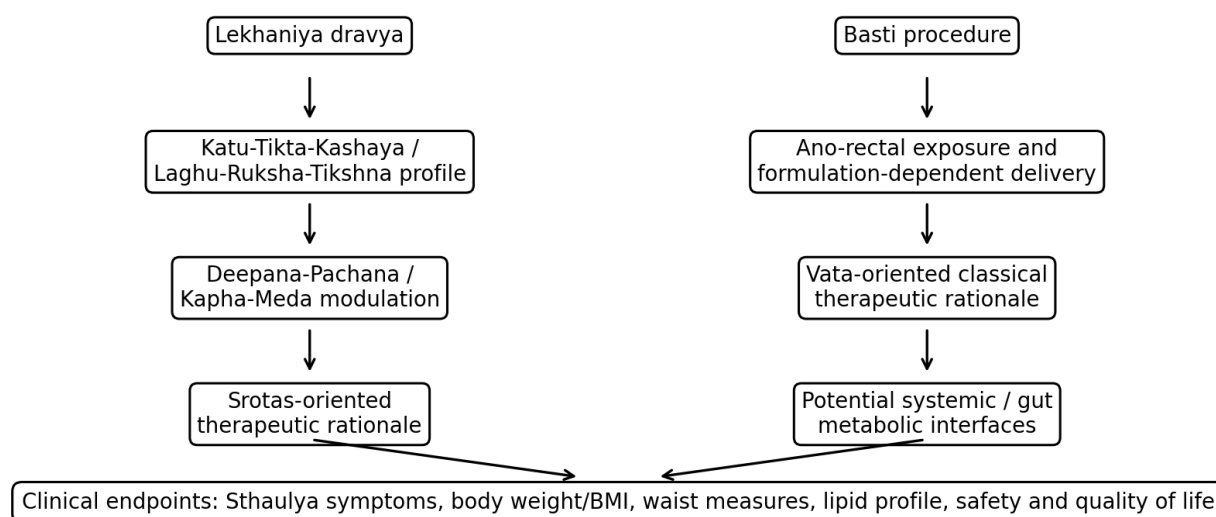


Figure 2. Proposed integrative research framework for *Lekhaniya Gana Basti*; classical actions and modern route/metabolic hypotheses are shown as parallel rather than equivalent explanations [9,13,14,22].

12. Possible Contemporary Mechanisms: Evidence and Hypotheses

The first plausible contemporary mechanism is modulation of lipid metabolism by phytochemicals present in the formulation, particularly compounds related to curcumin and berberine [22]. Human meta-analytic evidence indicates that berberine can reduce several circulating lipid measures when taken orally, providing a specific metabolic signal worthy of investigation in Daruharidra-containing formulations [25]. Curcuma longa interventions have also been studied across metabolic disorders with reported effects on lipid, inflammatory and anthropometric outcomes [27]. The central uncertainty is whether the *Basti* preparation delivers these compounds in sufficient concentration and bioavailable form to reproduce any of those effects [9]. Pharmacokinetic sampling is therefore more informative than assuming that the presence of a marker compound in raw material guarantees systemic activity [22].

The second plausible mechanism involves inflammatory and oxidative pathways associated with adipose dysfunction [32]. Obesity-related adipose inflammation can disrupt insulin signaling, increase free-fatty-acid flux and worsen glucose-lipid metabolism [7]. Several *Lekhaniya* ingredients have preclinical antioxidant or anti-inflammatory activity, but such findings are mechanistically supportive rather than clinically confirmatory [24]. A future trial could measure high-sensitivity C-reactive protein and selected adipokines as secondary endpoints while retaining standard lipid and anthropometric outcomes as the primary clinical measures [19]. Biomarker interpretation should be prespecified because multiple testing in small pilot studies can generate unstable associations [17].

The third hypothesis concerns appetite and energy-regulation pathways because *Sthaulya* classically includes abnormal hunger and the 2025 pilot study explored genes related to ghrelin, leptin and energy metabolism [17]. That study reported increased expression of UCP2, ADIPOR1 and PPAR-gamma and decreased expression of FTO, ghrelin and leptin-related transcripts immediately after active interventions compared with baseline patterns [17]. Gene-expression findings from peripheral blood are exploratory and do not prove that transcriptional change mediates the clinical effect [17]. Replication in larger samples with correction for multiple comparisons and correlation with measured metabolic endpoints would be needed before these markers could be considered mechanistic evidence [17]. The gene data are nevertheless valuable because they demonstrate a feasible route for integrating molecular outcomes into Ayurveda clinical research [17].

The fourth hypothesis involves gut-microbiota and bile-acid signaling, both of which have substantial contemporary relevance to obesity and metabolic disease [8]. Bile acids interact bidirectionally with intestinal microbial communities and act through receptors that regulate lipid, glucose, energy and inflammatory pathways [31]. Ano-rectal administration creates direct colonic exposure that makes local ecological or metabolite effects biologically plausible [9]. No cited *Lekhana Basti* clinical trial has yet demonstrated a microbiome-mediated effect, so this mechanism should not be stated as established [17]. Future research could pair stool metagenomics or targeted metabolomics with standardized dietary control to test whether changes exceed normal temporal variability [8].

The fifth hypothesis concerns rectal absorption and partial first-pass avoidance for selected compounds [10]. Modern reviews confirm that rectal administration can yield clinically useful systemic concentrations for some drugs, but the extent of absorption varies widely by formulation and molecule [9]. Herbal decoctions contain compounds spanning a broad range of molecular weights, ionization states and lipid solubilities, making uniform absorption unlikely [22]. The *Basti* matrix may also contain oil, honey, salts or other excipients that alter dissolution, mucosal interaction and retention [16]. A formulation-specific pharmacokinetic study is therefore necessary before modern systemic-delivery claims can be made for *Lekhaniya Gana Basti* [9].

13. RESULTS OF EVIDENCE SYNTHESIS

An earlier clinical study of 30 patients with dyslipidemia evaluated *Lekhana Basti* against serial lipid-profile measurements and reported statistically significant improvement after treatment and follow-up. The study is directly relevant because its primary focus was dyslipidemia rather than obesity alone. Its methodological reporting reflects an earlier period of Ayurveda clinical research and does not provide the same level of design transparency expected from contemporary randomized trials. The result should therefore be treated as supportive historical clinical evidence rather than as a high-certainty estimate of effect. A 2024 case report combining *Lekhana Basti* with *Udvardana* provides additional descriptive evidence but cannot separate the contribution of each intervention or control for natural variation.

The 2026 protocol by Satone and colleagues plans an assessor-blinded three-arm superiority trial with 129 participants divided equally among *Lekhan Basti* with Triphala Gana, *Lekhan Basti* with Ushakadi Gana and atorvastatin control. The planned outcomes include lipid profile, BMI and weight, with *Basti* administered for 16 days and atorvastatin administered for 48 days according to the published protocol. The use of an active standard-control arm is methodologically valuable because it places the Ayurvedic intervention within a clinically interpretable dyslipidemia framework. Differences in treatment duration, route and blinding remain important design considerations for later interpretation. Because the cited publication is a protocol, no efficacy or safety result should be attributed to this study until outcome data are published.

The evidence synthesis also identified a 2025 scholarly review of *Lekhana Basti* and a 2026 analytical dataset on *Lekhaniya Mahakashaya* that strengthen the contextual and formulation evidence. The analytical dataset is particularly important because clinical reproducibility depends on ensuring that the intended classical ingredients are chemically and botanically consistent across batches. Its identification of 2,034 metabolomics features highlights the challenge of reducing the intervention to a single active constituent. The same complexity supports the need for predefined marker compounds and manufacturing specifications in future trials. Taken together, the literature suggests a credible research signal but a substantial gap between traditional rationale, constituent pharmacology and confirmatory clinical evidence.

13.1 Clinical Evidence Table

Study	Design	Sample	Intervention / comparator	Key outcomes	Reported result	Main limitation
-------	--------	--------	---------------------------	--------------	-----------------	-----------------

Munshi et al., 2025	Randomized open-label comparative pilot	64 enrolled; 50 completed	Lekhan <i>Basti</i> , Navak Guggul, combination, lifestyle-only; all with lifestyle context	BMI, WHR, symptoms, IWQOL, lipids, gene expression	Active groups improved BMI; lipid reduction strongest in <i>Basti</i> and combination groups; exploratory gene changes reported	Small pilot, open label, attrition, multiple outcomes
Gayakwad et al., 2025	Comparative clinical study	40	Lekhana <i>Basti</i> in Chaturbhadra Kalpa schedule vs Yoga <i>Basti</i> schedule	Weight, BMI, circumferences, lipid profile, symptoms	Both schedules improved outcomes; Chaturbhadra schedule reported as more pronounced	Small single-center study; limited reporting in abstract
Pooja et al., 2015	Clinical study	30	Lekhana <i>Basti</i>	Total cholesterol, TG, HDL, LDL, VLDL	Statistically significant lipid-profile improvement reported after treatment and follow-up	Older design/reporting; limited contemporary control methodology
Mulay & Jain, 2024	Case report	1	Lekhana <i>Basti</i> plus Udvartana	Sthoulya clinical and anthropometric response	Clinical improvement described	No control; combined intervention; single case
Satone et al., 2026	Randomized assessor-blinded protocol	129 planned	Lekhan <i>Basti</i> with Triphala vs Ushakadi vs atorvastatin	Lipid profile, BMI, weight	No outcomes yet because publication is a protocol	Pending results; unequal treatment duration across arms

14. Critical Discussion

The available evidence supports a coherent Ayurvedic rationale for *Lekhaniya Gana Basti* in selected *Sthaulya* phenotypes, but coherence of theory is not equivalent to proof of clinical efficacy. Classical descriptions link *Sthaulya* with excessive *Meda*, Kapha-promoting causes, altered Vata behavior and impaired function, making a Lekhana-oriented Shodhana strategy internally consistent within Ayurveda. Recent clinical studies provide preliminary human signals in the same direction by reporting reductions in BMI, weight-related measures and lipid parameters after Lekhana *Basti* protocols. The central scientific question is whether these changes are reproducible, clinically meaningful and specifically attributable to a standardized *Basti* intervention beyond lifestyle and contextual effects. Current evidence does not yet answer that question with high certainty.

The 2025 randomized pilot is a major advance because it used computer-based randomization, a lifestyle-only comparison group, biochemical outcomes and exploratory molecular measures. Its design nevertheless remained open label and the final group sizes were small, which increases vulnerability to performance bias, attrition bias and unstable estimates. The immediate BMI reduction in the combination group was notable, but partial rebound at two months indicates the importance of durability and long-term weight maintenance. The lipid findings were encouraging but not uniformly significant across all fractions and time points, which argues against describing the intervention as a proven lipid-lowering therapy. Future confirmatory studies should designate one or two primary endpoints and power the sample accordingly rather than distributing inference across many exploratory measures.

The 2025 *Basti*-schedule comparison introduces another important issue because the clinical effect may depend on sequencing of Niruha and Anuvasana procedures rather than on the drug list alone. If schedule, dose, decoction concentration, oil preparation or adjuvants vary between studies, pooling the interventions under the single label Lekhana *Basti* can conceal clinically meaningful heterogeneity. A standard reporting checklist should therefore document ingredient identity, raw-drug quantity, extraction ratio, final volume, excipients, temperature, administration rate, retention, evacuation time and complete schedule. Without these details, even a positive trial

can be difficult to reproduce or translate into practice. The same principle applies to dietary advice because lifestyle cointerventions can materially change weight and triglyceride outcomes.

Formulation identity is a particularly important issue for the title of this review because *Lekhaniya Gana* and *Lekhana Basti* are not automatically synonymous. Published *Lekhana Basti* formulations may contain Triphala, honey, salts, oils, cow urine or other *Gana* combinations that differ from the ten-drug *Charaka Lekhaniya Mahakashaya*. A review titled *Lekhaniya Gana Basti* should therefore specify whether the intended *Basti* is prepared directly from the *Charaka Lekhaniya* group or whether it refers more broadly to a *Lekhana-purpose Basti*. This distinction affects both classical accuracy and interpretation of clinical evidence because studies using different formulations cannot be assumed to test the same product. The present synthesis uses *Lekhana Basti* studies as supportive related evidence while explicitly recognizing that they may not be formulation-identical to a standardized *Lekhaniya Gana Basti*.

The phytochemical evidence adds plausibility but also exposes the limitations of simple mechanism narratives. Curcumin and berberine are measurable and clinically studied compounds, yet their known oral effects cannot be transferred quantitatively to a rectal polyherbal intervention. *Cyperus rotundus* now has a recent obesity RCT, but the tested standardized extract included piperine and was administered orally under a defined regimen. *Saussurea costus* has extensive pharmacological literature but limited direct human obesity evidence. These examples demonstrate why whole-formulation trials remain essential even when individual ingredients are biologically promising.

Despite these limitations, the direction of research is encouraging because the field is moving from descriptive rationale toward randomized designs, molecular endpoints and analytical characterization. The 2026 LMK metabolomics dataset provides an unusually detailed platform for batch standardization and future reverse-pharmacology work. The 2026 dyslipidemia trial protocol demonstrates that larger controlled comparisons against standard pharmacotherapy are feasible in an Ayurveda research setting. The next methodological advance should be multicenter replication with prespecified endpoints and standardized intervention manufacturing. Such work could determine whether the preliminary clinical signal translates into a reproducible adjunctive therapy for *Sthaulya* with dyslipidemia.

The attached doctoral protocol is methodologically relevant because it proposes a substantially larger two-group sample, a defined 15-day *Basti* sequence, serial follow-up and simultaneous Ayurvedic, anthropometric and lipid outcomes. However, the synopsis states that ethics permission and CTRI registration will be obtained, so the study should not be cited as a registered or completed efficacy trial unless those identifiers and final results are subsequently verified. The protocol would also benefit from explicit reporting of the randomization sequence, allocation concealment, handling of missing data, adherence to the diet-exercise control and prespecified statistical tests for repeated measurements.

15. Safety, Contraindications and Clinical Governance

Basti should be performed only after appropriate clinical assessment because classical selection depends on disease state, *Bala*, *Agni*, *Kosha*, age and procedural suitability [14]. The procedure should be delivered by trained practitioners in settings that can maintain hygiene, monitor retention and evacuation and manage intolerance or complications [16]. Patients with unstable cardiovascular disease, severe uncontrolled metabolic illness, acute gastrointestinal disease or other major contraindications require individualized medical assessment rather than routine enrollment in a metabolic *Basti* program [17]. Concurrent conventional lipid-lowering therapy should be documented and modified only under appropriate clinical supervision [4]. The review therefore does not support self-administration of *Lekhaniya Gana Basti* for weight loss or high cholesterol [16].

Raw-drug safety begins with authenticated identity because botanical substitution can change both therapeutic chemistry and toxicity [22]. This concern is especially relevant to *Ativisha*, *Vacha* and *Haimavati* because species identity or chemotype can materially affect risk assessment [22]. Batch testing should include organoleptic and pharmacognostic identification together with chromatographic or spectrometric fingerprinting appropriate to the formulation [29]. Microbial quality, heavy metals, pesticide residues and other relevant contaminants should also be controlled according to applicable pharmacopeial and regulatory standards [22]. Clinical trials should retain samples from each batch so unexpected events can be investigated retrospectively [19].

Procedure-related adverse-event monitoring should capture abdominal cramping, rectal irritation, bleeding, dizziness, dehydration, electrolyte disturbance, constipation, diarrhea and inability to retain the formulation [9]. The presence of giddiness and gastrointestinal events in the 2025 pilot shows that tolerability reporting is necessary even when serious toxicity is not observed [17]. Rectal drug-delivery literature also recognizes local irritation and variable absorption as formulation-dependent concerns [10]. A standardized adverse-event form should be completed after every *Basti* administration rather than only at the end of a treatment cycle [19]. This approach will allow frequency, severity and relationship to *Niruha* versus *Anuvasana* components to be analyzed separately [17].

16. Research Gaps and Future Directions

16.1 Proposed Framework for a Future Confirmatory Trial

Domain	Recommended design element	Reason	Key source
Population	Adults with both clinically defined <i>Sthaulya</i> and laboratory-confirmed dyslipidemia	Ensures the trial addresses the title rather than obesity alone	[3,19]
Intervention	Authenticated ten-drug <i>Lekhaniya Gana Basti</i> with fixed manufacturing and <i>Basti</i> schedule	Improves reproducibility and interpretability	[22]
Comparator	Standard lifestyle program plus appropriate guideline-directed lipid care; consider an additional Ayurveda comparator	Protects cardiovascular care and allows incremental-effect estimation	[4,5]
Primary endpoint	One prespecified endpoint such as triglyceride or non-HDL-C change at a defined time point	Reduces multiplicity and powers the study appropriately	[4,19]
Secondary endpoints	BMI, waist circumference, WHR, symptom score, quality of life, apoB, glycemic markers	Captures both Ayurveda and cardiometabolic domains	[3,17]
Safety	Active adverse-event surveillance, rectal tolerability, liver/renal tests and discontinuation criteria	Builds reliable benefit-risk evidence	[9,17]
Mechanistic substudy	Marker pharmacokinetics, metabolomics, inflammatory markers, microbiome or bile acids	Tests hypotheses without replacing clinical endpoints	[8,22]
Follow-up	At least one post-treatment assessment beyond the active <i>Basti</i> cycle	Determines durability and rebound	[17]

17. CONCLUSION

Lekhaniya Gana Basti has a coherent classical rationale for selected patients with *Sthaulya* because the formulation is directed toward Lekhana while *Basti* provides a Shodhana strategy with strong Vata-oriented therapeutic significance. The classical model of *Sthaulya* incorporates Kapha-Meda excess, Agni disturbance, Srotorodha and altered Vata behavior, creating a multidimensional therapeutic target rather than a simple weight-reduction objective. Dyslipidemia can be studied as an important associated modern metabolic abnormality, but it should not be equated directly with *Meda* Dhatu or assumed to be present in every patient with *Sthaulya*. Recent phytochemical work strengthens formulation science by demonstrating a complex and measurable *Lekhaniya Mahakashaya* chemical profile that includes curcumin and berberine markers. Constituent-level evidence for curcumin, berberine and *Cyperus rotundus* further supports biological plausibility while remaining insufficient to prove the effect of the complete rectally administered formulation.

Clinical evidence is promising but preliminary. The 2025 randomized pilot reported improvements in BMI and lipid-related outcomes with *Lekhan Basti* and combination therapy, while the 2025 schedule-comparison study reported benefits with two *Basti* sequences. An older dyslipidemia study and a recent case report provide supportive but lower-certainty evidence, and a 2026 assessor-blinded controlled protocol shows that more rigorous testing is underway. No current evidence justifies claiming that *Lekhaniya Gana Basti* is an established substitute for guideline-directed dyslipidemia therapy or that it prevents cardiovascular events. The appropriate conclusion is that the intervention warrants standardized confirmatory research as a potentially useful integrative strategy for *Sthaulya* with metabolic abnormalities.

The next generation of research should unite Ayurvedic diagnostic rigor with modern trial methodology, authenticated pharmacognosy, chemical fingerprinting, complete lipid assessment, active safety monitoring and longer follow-up. A fixed definition of *Lekhaniya Gana Basti* is particularly important because published Lekhana

Basti formulations and schedules are heterogeneous. Mechanistic studies of rectal pharmacokinetics, adipose inflammation, gene expression, microbiota and bile-acid signaling can be valuable when they are prespecified and clearly separated from clinical efficacy claims. Such an approach can preserve the conceptual integrity of Ayurveda while generating evidence that is interpretable within contemporary clinical science. Until that evidence is available, therapeutic use should remain individualized, professionally supervised and integrated with established cardiovascular risk management when dyslipidemia is clinically significant.

REFERENCES

1. World Health Organization. Obesity and overweight. Geneva: WHO; updated 8 Dec 2025.
2. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. *Lancet*. 2024;403(10431):1027-1050. doi:10.1016/S0140-6736(23)02750-2.
3. Bays HE, et al. Obesity, dyslipidemia, and cardiovascular disease: a joint expert review from the Obesity Medicine Association and the National Lipid Association 2024. *Obes Pillars*. 2024. PMID: PMC11066689.
4. American Heart Association. 2026 Guideline on the Management of Dyslipidemia. *Professional Heart Daily*. Updated 13 Mar 2026.
5. Blumenthal RS, Morris PB, Gaudino M, et al. 2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of dyslipidemia. *Circulation*. 2026. doi:10.1161/CIR.0000000000001423.
6. Varra FN, Varras M, Varra VK, Theodosios-Nobelos P. Molecular and pathophysiological relationship between obesity and chronic inflammation in the manifestation of metabolic dysfunctions and their inflammation-mediating treatment options. *Mol Med Rep*. 2024;29(6):95. doi:10.3892/mmr.2024.13219.
7. Yan K. Recent advances in the effect of adipose tissue inflammation on insulin resistance. *Cell Signal*. 2024;120:111229. doi:10.1016/j.cellsig.2024.111229.
8. Zhu Z, Xu Y, Xia Y, et al. Review on chronic metabolic diseases surrounding bile acids and gut microbiota: what we have explored so far. *Life Sci*. 2024;338:122384. doi:10.1016/j.lfs.2023.122384.
9. Rath R, Sanshita, Kumar A, et al. Advancements in rectal drug delivery systems: clinical trials, and patents perspective. *Pharmaceutics*. 2022;14(10):2210. doi:10.3390/pharmaceutics14102210.
10. Hua S. Physiological and pharmaceutical considerations for rectal drug formulations. *Front Pharmacol*. 2019;10:1196. doi:10.3389/fphar.2019.01196.
11. Agnivesha. Charaka Samhita, revised by Charaka and Dridhabala. Sutra Sthana, Ashtaninditiya Adhyaya (chapter on undesirable constitutions). Standard Sanskrit commentary edition. Varanasi: Chaukhambha publications.
12. Agnivesha. Charaka Samhita, revised by Charaka and Dridhabala. Sutra Sthana, Santarpaniya Adhyaya. Standard Sanskrit commentary edition. Varanasi: Chaukhambha publications.
13. Agnivesha. Charaka Samhita, revised by Charaka and Dridhabala. Sutra Sthana, Shadvirechanashatashritiya Adhyaya; *Lekhaniya* Mahakashaya. Standard Sanskrit commentary edition. Varanasi: Chaukhambha publications.
14. Sushruta. Sushruta Samhita. Chikitsa Sthana; *Basti*-related chapters. Standard Sanskrit commentary edition. Varanasi: Chaukhambha publications.
15. Vagbhata. Ashtanga Hridaya. Sutra and Kalpa/Siddhi sections relevant to *Sthaulya* and *Basti*. Standard Sanskrit commentary edition. Varanasi: Chaukhambha publications.
16. Mangal G, Sharma L. A review of Lekhana *Basti*: Ayurvedic management of obesity and metabolic disorders. *Int J Ayurveda* 360. 2025;1(6):456-463. doi:10.63247/3048-7390.vol.1.issue6.4.
17. Munshi R, Panchal F, Kumbhar D. Effect of Lekhan *Basti* and Navak Guggul on lipid markers and transcriptional effects of selected genes in obese patients: a single-centre, open-labelled, randomized, comparative, pilot study. *J Ayurveda Integr Med*. 2025;16(3):101129. doi:10.1016/j.jaim.2025.101129.
18. Gayakwad A, Toshikhane S, Dash GK. A comparative clinical study to evaluate the efficacy of Lekhana *Basti* in Chaturbhadra Kalpa *Basti* Krama and Yoga *Basti* Krama in the management of Sthoulya with special reference to obesity. *Int J Ayurvedic Med*. 2025;16(2):448-454. doi:10.47552/ijam.v16i2.5323.
19. Satone S, Parwe S, Sawarkar P, Nisargandha M. Comparative efficacy of Lekhan *Basti* with Ushkadi Gana versus Triphala Gana as against standard control in dyslipidaemia (Medoroga): a research protocol for a randomised controlled trial. *J Clin Diagn Res*. 2026;20(4):JK01-JK05. doi:10.7860/JCDR/2026/78390.22706.
20. Pooja BA, Bhatted S, Bhojani MK. Effect of Lekhana *Basti* in the management of dyslipidemia: a clinical study. *Int J Ayurveda Pharma Res*. 2015;2(7).
21. Mulay M, Jain S. Role of Lekhana *Basti* and Udvartana in the management of Sthoulya (obesity): a case study. *J Ayurveda Holist Med*. 2024;11(12). doi:10.70066/jahm.v11i12.1131.
22. Singh N, Upadhyay A, Chakraborty D, Patil G, Yadav P, Galib R, Prajapati PK. Multi-analytical dataset on *Lekhaniya* Mahakashaya: HRLC-MS/MS Orbitrap profiling, HPTLC fingerprinting with marker estimation, and FTIR spectroscopy. *Data Brief*. 2026;65:112464. doi:10.1016/j.dib.2026.112464.
23. Mansoria P, Singh SB, Singh BP, Sisodia BS. Revisiting Haridra of *Lekhaniya* Mahakashaya: a classical herb with contemporary evidence for the management of obesity and dyslipidemia. *J Ayurveda Integr Med Sci*. 2026;11(1). doi:10.21760/jaims.11.1.45.

24. Kumari R, Negi M, Thakur P, et al. *Saussurea costus* (Falc.) Lipsch.: a comprehensive review of its pharmacology, phytochemicals, ethnobotanical uses, and therapeutic potential. *Naunyn Schmiedebergs Arch Pharmacol.* 2024;397(3):1505-1524. doi:10.1007/s00210-023-02694-0.
25. Blais JE, et al. Overall and sex-specific effect of berberine for the treatment of dyslipidemia in adults: a systematic review and meta-analysis of randomized placebo-controlled trials. *Drugs.* 2023;83. PMID:36941490.
26. Efficacy and safety of berberine on the components of metabolic syndrome: a systematic review and meta-analysis of randomized placebo-controlled trials. 2025. PMID:40740996.
27. Kehinde SA, Qaisrani ZN, Pattanayaiying R, et al. Clinical potential of *Curcuma longa* Linn. as nutraceutical/dietary supplement for metabolic syndrome: systematic review and meta-analysis of randomized controlled trials. *Foods.* 2026;15(1):60. doi:10.3390/foods15010060.
28. Majeed A, Majeed S, Devarajan TV, Narasinga Rao SSVV, Gudimallam S, Ramanujappa M, Thazhathidath S, Mundkur L. Efficacy and safety of *Cyperus rotundus* extract on weight management in obese individuals: a randomized, double-blind, placebo-controlled study. *Medicine (Baltimore).* 2025;104(47):e45666. doi:10.1097/MD.00000000000045666.
29. Rajput S, Mata S, Dei LP, Harisha CR, Shukla VJ, Donga S. Pharmacognostical and preliminary physico-chemical analysis of contents of *Lekhaniya Mahakashaya* Siddha Taila: an Ayurvedic formulation. *Ann Ayurvedic Med.* 2016;5(3-4):104-107.
30. Charak Samhita Research, Training and Skill Development Centre. Herb database entries for Musta, Haridra, Daruharidra, Vacha, Chitraka, Chirabilva and Haimavati; references to *Lekhaniya Mahakashaya*. 2021-2026.
31. He S, Li L, Yao Y, Su J, Lei S, Zhang Y, Zeng H. Bile acid and its bidirectional interactions with gut microbiota: a review. *Crit Rev Microbiol.* 2024;50(5):684-701. doi:10.1080/1040841X.2023.2262020.
32. Xu S, Lu F, Gao J, Yuan Y. Inflammation-mediated metabolic regulation in adipose tissue. *Obes Rev.* 2024;25(6):e13724. doi:10.1111/obr.13724.
33. World Health Organization. One in eight people are now living with obesity. News release. 1 Mar 2024.
34. American College of Cardiology. ACC/AHA release new clinical guideline for managing dyslipidemia. 13 Mar 2026.
35. GBD 2021 Risk Factor Collaborators. Global burden of 88 risk factors in 204 countries and territories, 1990-2021: a systematic analysis for the Global Burden of Disease Study 2021. *Lancet.* 2024;403:2162-2203.