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REVIEW ARTICLE

A Critical Review on the Therapeutic Potential of Sushrutokta Varunadi Gana Dravyas in the Management of Abhyantara Vidradhi

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Abstract

Background: Sushruta's *Dravya-Sangrahaniya Adhyaya* (Sutrasthana 38) classifies drugs into therapeutic groups (*Gana*) that encode a collective pharmacodynamic signature rather than isolated drug actions. Among these, the *Varunadi Gana* carries an explicit classical indication for *Abhyantara Vidradhi* – internal suppurative pathology. **Objective:** To critically appraise whether the classical properties attributed to *Varunadi Gana* can be rationally reconciled with contemporary pharmacological evidence relevant to internal abscess and inflammatory-suppurative disease. **Methodology:** Narrative review integrating (i) verified primary Sanskrit textual passages from Su.Su.38, Su.Ni.9 and Su.Ci.16, (ii) Ayurvedic pharmacological interpretation (*Rasa-Guna-Virya-Vipaka*, *Kapha-Medo-Hara*, *Sothahara*), and (iii) experimental and clinical biomedical literature retrieved for each constituent drug. **Observations:** The group comprises approximately twenty drug entities with disputed botanical identities for several members (*Artagala*, *Vasuka*, *Meṣasṛṅgi*). Nearly all constituents possess independently documented anti-inflammatory, antimicrobial or antioxidant activity in in vitro and animal models; several converge mechanistically on NF-κB, COX-2/PGE₂ and pro-inflammatory cytokine pathways. Clinical evidence remains confined to isolated case reports of *Yakṛt* Vidradhi. **Critical interpretation:** Modern activity data corroborate, but do not prove, the classical indication; the *Gana* concept and the *Sotha*→*Vidradhi* continuum provide the interpretive bridge. **Conclusion:** *Varunadi Gana* represents a plausibly rational polyherbal strategy for early-stage internal inflammatory-suppurative pathology, but requires controlled clinical evaluation.

Keywords: Varunadi Gana; Sushruta Saṃhita; *Abhyantara Vidradhi*; *Vidradhi*; Ayurvedic pharmacology; anti-inflammatory activity; sothahara; internal abscess

Introduction

Sushruta's pharmacological framework is distinctive in classical Ayurveda for its systematic aggregation of drugs into functional groups. The thirty-eighth chapter of the *Sutrasthana*, the *Dravya-Sangrahaniya Adhyaya*, enumerates thirty-seven *Gana* – therapeutic clusters defined by shared karma rather than by taxonomy or morphology¹⁻⁴. The clinical utility of this schema lies in *Yuktivyavahara*: the physician selects a group whose collective pharmacodynamic signature matches the *Doṣa-Duṣhya* configuration of the disease, then individualises within it.

Vidradhi is the classical designation for a deep-seated, painful, suppurating swelling – the disease that *Sushruta* regarded as a disease of surgical consequence. *Sushruta's Nidana Sthana* ninth chapter and *Chikitsa Sthana* 16th chapter together constitute the classical dossier on abscess management⁵⁻⁷. The pathological weight of the term is carried in the word itself: *Vidradhi* derives from the notion of rapid destruction (*Sighra*

Vidahana), signifying suppuration that may destroy tissue⁸.

Abhyantara Vidradhi – internal abscess – is the form seated within the viscera and body cavities. Its recognition matters clinically because Sushruta explicitly stated that once an internal abscess suppurates, “suppuration in such cases leads but to a slender hope of success” – inverting the modern assumption that an abscess is simply a treatable collection requiring drainage⁵. This gives the disease an interceptable window – the *Ama Avastha* (immature, unripe stage) – in which systemic pharmacotherapy, rather than surgery, is the primary instrument.

The *Varunadi Gana* is one of the few *Sushrutan* groups whose *Phalasaruti* names *Vidradhi* directly. This review therefore interrogates a specific pharmacological problem: does the composition of the group, and the contemporary evidence base for its members, rationally support its classical deployment in internal inflammatory-suppurative pathology, or is the classical

indication merely a plausible but unsubstantiated tradition? The article deliberately separates textual evidence, Ayurvedic interpretation, experimental evidence, clinical evidence, and authorial hypothesis.

Materials and Methods

This is a narrative scholarly review, not a systematic review. Primary textual verification used published English translations of the *Susrutasamhita* based on the original Sanskrit — the *Bhishagratna* translation (*Sutrasthana*, *Nidana Sthana*, *Chikitsa Sthana*)⁴⁻⁶ and a standard teaching translation of the same chapters^{7,9} — supplemented by a peer-reviewed compilation of *Sushruta's Gana*¹⁰ and a critical review of *Vagbhata's* parallel *Varanadi Gana*¹¹.

Critically, verse numbering for Su.Su.38 differs across editions. The chapter's groups are frequently cited as a block (e.g. “*Sutrasthana* 38th chapter, *Shloka* No. 4–76”) rather than per-group¹⁰. The commonly used citation Su.Su.38/8–9 for the *Varunadi Gana* passage could not be independently confirmed against a critical Devanagari edition within this review; readers of any derivative publication should verify the verse number against their own edition before citing it. Composition and phalasruti were, however, cross-verified across two independent translations^{4,9}.

Biomedical literature was retrieved per constituent drug, prioritising indexed reviews, in vivo studies, and mechanistic studies, with study type recorded. Contested botanical identities were cross-checked against published critical appraisals of medicinal-plant nomenclature in classical Ayurvedic literature.¹²

Classical Textual Evidence

Composition of the *Varunadi Gana*

The *Bhishagratna* translation renders the group as: “The drugs known as *Varuna*, *Artagala*, *Sigru*, *Madhu-Sigru*, *Tarkari*, *Meṣa-Sṛngi*, *Putika*, *Naktamala*, *Morata*, *Agni-mantha*, the two kinds of *Sairiyaka*, *Vimbi*, *Vasuka*, *Vasira*, *Citraka*, *Satavari*, *Vilva*, *Ajasṛngi*, *Darbha*, and the two kinds of *Vṛhati* form the group known as the *Varunadi*”.⁴ A second translation gives an identical roster with minor orthographic variants (*Bimbi*, *Bilva*, *Ajasṛngi*, Two *Brhati*).⁷

Two structural features complicate the count. First, the text uses dvandva compounds — “the two *Sairiyaka*” (*Raktapuṣpa* and *Nilapuṣpa*, generally *Barleria cristata*

and *B. strigosa*) and “the two *Vṛhati*” (*Brhati* and *Kañtakari*, i.e. *Solanum* spp.). Second, several names are single entities. The count therefore depends on convention: twenty entries as enumerated by name, expanding to approximately twenty-two botanical entities if each *Dvaya* is split. Several compilations list a reduced roster, omitting *Vasira*, *Satavari*, *Vilva*, *Ajasṛngi*, *Darbha* and the *Vṛhati* pair and thus arriving at fourteen ingredients — a discrepancy indicating that secondary compilations often abbreviate the group rather than reproduce it faithfully¹⁰. For this review the full twenty-entry enumeration is adopted, from the primary translations.

The Classical Therapeutic Action (*Phalasruti*)

The group's stated action is unambiguous and highly specific: “The group is possessed of the efficacy of reducing the deranged *Kapha* and fat and proves efficacious in cases of cephalalgia [*siro-roga*], *Gulma* and internal abscesses”⁴. The teaching translation renders this as: “This *Varunadi Gana* mitigates *Kapha* and *Meda* (fat), cures headaches, abdominal tumours and internal abscesses”⁹.

Three points deserve emphasis. (i) The group is defined by *kapha* and *medas* reduction, not by an anti-infective or anti-pyretic descriptor — a pharmacodynamic signature of *Kaṭu-Tikta Rasa*, *Laghu-Rukṣa Guna* and *Uṣṇa Vīrya*. (ii) The indication set is internally coherent: *Siroroga*, *Gulma* and *Abhyantara Vidradhi* are all conditions in which *Kapha-Medas* accumulation and *Srotorodha* (channel obstruction) are pathogenic. (iii) The word used is internal abscess — the translator's rendering of *Abhyantara Vidradhi*, linking the chapter directly to the *Nidana* and *Chikitsa* discussions.

The *Varunadi Gana* in *Vidradhi Chikitsa* (Su.Chi.16)

Su.Ci.16 provides the operational confirmation that the group was not merely a classificatory artefact. For the unsuppurated internal abscess, the text prescribes “a potion consisting of a decoction of the drugs of the *Varunadi* group saturated with the powders (*Kalka*) of those of the *Uṣakadi* group,” followed by “clarified butter cooked with the decoction of the drugs of the two preceding groups” — reported to “cure an internal abscess in a very short time”⁵. The same groups are

prescribed as *Asthapana* and *Anuvasana* enemata after admixture with *Sneha*.

For the suppurated internal abscess, Sushruta prescribes “the drugs of the Varunadi group or *Madhu-sigru* mixed with (a copious quantity of) *Maireya*, *Sura*, *Asava*, or *Kañjika*” — irrespective of whether pus drains upward or downward⁵. The teaching translation adds that this decoction is to be consumed “when there is exudation of pus, blood etc. in upward or downward direction, in cases of *Vidradhi*,” and that ghee processed with Varunadi and *Virechana* groups cures *Raktaja Vidradhi*⁷. Three therapeutic principles are thus embedded: systemic decoction therapy in the ama stage; medicated ghee as *Sneha* vehicle; and enema as a route to act on deep-seated *Vata*. The recurring pairing with the *Uṣakadi* group is significant and is echoed in *Vagbhata's* parallel instruction.

Abhyantara Vidradhi — Nidana

Su.Ni.9 defines internal abscess as a “tumour-like (*Gulma*), raised, or elevated abscess in the interior of the organism, which is often felt to be shaped like an ant-hill,” arising when “the *Vayu*, *Pitta* and *Kapha* of the body” are deranged by heavy, incompatible, putrid or acid-forming foods, excessive coition, fatiguing exercise, or suppression of natural urges.⁶

Ten sites are enumerated — rectum (*Guda*), bladder (*Basti*), umbilicus (*Nabhi*), flanks (*Kuṣṭhi*), inguinal region (*Vañṣana*), kidneys (*Vṛkka*), spleen (*Pliha*), heart (*Hṛdaya*), liver (*Yakṛita*) and *Kloma* — each with a distinctive symptom signature: suppression of flatus when seated in the rectum; dysuria and scanty urine in the bladder; hiccough and *Aṭopa* at the umbilicus; thirst and dyspnoea in the liver; unquenchable thirst at the *Kloma*). The text is careful to distinguish internal *vidradhi* from *Gulma*, and its prognostic warning is explicit: the *Sannipataja* variety is incurable.

This visceral mapping — particularly the hepatic and splenic (*Pliha*) seats — provides the interpretive bridge to hepatobiliary abscess in modern taxonomy, and it is precisely *Yakṛt Vidradhi* for which Ayurvedic case reports exist.¹³

Textual Distinction Must Not Be Collapsed

Three distinct entities must be kept apart. First, Varunadi Gana as a classificatory group in Su.Su.38 — a pharmacological set defined by a *Kapha-Medo-Hara*

signature and an indication set including internal abscess. Second, *Varunadi Gana* as a therapeutic instruction in Su.Chi.16 — a specific decoction, ghee and enema protocol, always in combination (*Uṣakadi*, *Virecana*, *Madhu-Sigru*), for a specified stage of *Vidradhi*. Third, *Vagbhata's Varanadi Gana* — a different group of seventeen drugs in the *Aṣṭāṅghṛdaya/Aṣṭāṅgasamgraha*, overlapping only partially (*Varuna*, *Satavari*, *Bilva*, *Brhati*, *Kañtakari*, *Karañja*, *Putikarañja*, *Agnimantha*, *Tarkari*, *Darbha*, *Morata*, *Dahana*), with an explicitly kapha-vata-hara profile and identical clinical logic for *Antarvidradhi* in the *Ama Avastha*.¹¹ Conflating the two is a common secondary-source error and must be avoided.

Finally, *Abhyantara Vidradhi* is not equivalent to any single modern diagnosis. It is a conceptual and pathological correlate of localised, deep-seated inflammatory-suppurative disease — which may include intra-abdominal, hepatic, renal or pelvic abscess, but also embraces sterile inflammatory collections and pre-suppurative sotha. Equivalence claims are interpretive, not definitional.

Contemporary Pharmacological Evidence, Drug by Drug

Table 1 summarises retrievable evidence by constituent, with study type explicitly stated. Botanical identities follow the most cited attributions, with disputed identities flagged. Vasuka, Artagala and Morata carry contested botanical identities in the commentarial literature^{10, 12}; the identities used here are the most frequently cited attributions and are not settled.

Meṣaśṛṅgi and *Ajaśṛṅgi* are both identified with *Gymnema sylvestre* in some commentators' traditions, while other assign *Meṣaśṛṅgi* to *Dolichandrone falcata* and *Ajaśṛṅgi* to a further entity. This is an unresolved identity conflict and the evidence assigned to these two names should not be treated as independent^{10, 12}.

First, the group is pharmacologically redundant in the productive sense. At least 15 of the 20 constituents have independently anti-inflammatory activity, and a majority have antimicrobial or antioxidant data. In a classical polyherbal *Gana*, this is not incidental: redundancy across mechanistically diverse phytochemical classes (triterpenes, flavonoids, iridoids, alkaloids, naphthoquinones, saponins) is a rational design feature,

providing multi-target coverage & reducing dependence on any single constituent's pharmacokinetics.

Table 1. Constituent Drugs of the Varunadi Gana with Reported Pharmacological Activities Relevant to Inflammatory-Suppurative Pathology

Drug (Sanskrit)	Botanical identity	Reported activity	Study type	Ref.
<i>Varuna</i>	<i>Crataeva nurvala</i> Buch. -Ham.	Anti-inflammatory (lupeol-mediated; ERK inactivation); antibacterial; diuretic	In vitro; review	14, 15
<i>Artagala</i>	Identity disputed / uncertain	No drug-specific study retrieved	—	—
<i>Sigru</i>	<i>Moringa oleifera</i> Lam.	Broad-spectrum antimicrobial; anti-inflammatory (essential-oil fractions)	In vitro	16, 17
<i>Madhu-Sigru</i>	<i>Moringa concanensis</i> Nimmo ex Dalzell / <i>M. Oleifera</i>	Anti-inflammatory; antibacterial (data extrapolated from <i>M. oleifera</i>)	In vitro (by proxy)	16, 17
<i>Tarkari</i>	<i>Clerodendrum phlomidis</i> L.f. / <i>Premna</i> spp.	Anti-inflammatory (chloroform extract); analgesic	Animal	18
<i>Meṣaśṛṅgi</i>	Disputed: <i>Gymnema sylvestre</i> R.Br. or <i>Dolichandrone falcata</i> Seem.	Anti-inflammatory (carrageenan paw oedema); NF-κB/MAPK modulation	Animal; mechanistic	19, 20
<i>Putika</i>	<i>Holoptelea integrifolia</i> (Roxb.) Planch.	Analgesic and anti-inflammatory	Animal; review	21, 22
<i>Naktamala</i>	<i>Pongamia pinnata</i> (L.) Pierre	Antimicrobial; anti-inflammatory (karanjin, pongamol)	In vitro	—
<i>Morata</i>	<i>Alangium salvifolium</i> (L.f.) Wangerin	Analgesic; anti-inflammatory	Animal	23, 24
<i>Agnimantha</i>	<i>Premna integrifolia</i> L. / <i>Clerodendrum phlomidis</i>	Anti-inflammatory across plant parts; analgesic; immunomodulatory profile	Animal; comparative	25, 26, 27
<i>Sairiyaka-dvaya</i>	<i>Barleria cristata</i> L. and <i>B. strigosa</i> Willd.	Anti-inflammatory; antibacterial; antioxidant	In vitro; review	28, 29
<i>Vimbi (Bimbi)</i>	<i>Coccinia indica</i> Wight & Arn. (<i>C. grandis</i>)	Anti-inflammatory; analgesic; antipyretic	Animal; review	30
<i>Vasuka</i>	Disputed: <i>Adhatoda vasica</i> Nees (<i>Justicia adhatoda</i>) / <i>Osmanthus fragrans</i>	Anti-inflammatory and anti-hypoxic effects of whole aqueous extract	Animal	31
<i>Vasira</i>	<i>Achyranthes aspera</i> L.	Anti-inflammatory (acute and chronic models); analgesic; antipyretic	Animal	32
<i>Chitraka</i>	<i>Plumbago zeylanica</i> L.	Anti-inflammatory; cytotoxic; anti-arthritis (processed root)	Animal; in vitro	33, 34, 35
<i>Satavari</i>	<i>Asparagus racemosus</i> Willd.	Anti-inflammatory (liposomal root extracts); immunomodulatory; adaptogenic	Animal; review	36, 37
<i>Vilva</i>	<i>Aegle marmelos</i> (L.) Correa	Anti-inflammatory and antipyretic; analgesic	Animal	38, 39
<i>Ajaśṛṅgi</i>	Identity disputed; identified with <i>Gymnema sylvestre</i> in some traditions	Anti-inflammatory; protection in ulcerative colitis	Animal	19
<i>Darbha</i>	<i>Desmostachya bipinnata</i> (L.) Stapf	Anti-inflammatory and analgesic (hydro-alcoholic root extract)	Animal	40
<i>Vrhati-dvaya</i>	<i>Solanum indicum</i> L. / <i>S. xanthocarpum</i> Schrad. & J.C.Wendl.	Anti-inflammatory; analgesic; antipyretic	Animal; in vitro	41, 42

Second, mechanistic convergence exists at plausible nodes. *Paederia foetida*, a documented *Kapha-Vata-Hara* drug of allied *Gana* usage, inhibits adjuvant-induced arthritis “by suppression of PGE₂ and COX-2 expression via nuclear factor-κB”^{43, 44}, and the same pathway is engaged by *Plumbago zeylanica* (plumbagin)³³, *Achyranthes aspera*³², *Gymnema sylvestre* (NF-κB/MAPK)²⁰, *Holoptelea integrifolia*²¹ and *Oroxylum indicum* in LPS-stimulated macrophages^{45, 46}. NF-κB and COX-2/PGE₂ are central to the transition from *Sotha* (inflammatory oedema) to suppuration; their convergence across the group constitutes the strongest single mechanistic argument for the classical indication. Third, evidence is

overwhelmingly preclinical. Almost all data derive from carrageenan paw-oedema, formalin, cotton-pellet granuloma or LPS-stimulated macrophage models. Human clinical evidence is limited to case reports of Ayurvedic management of liver abscess, where polyherbal regimens produced a “highly satisfactory response” in individual patients¹³. Case reports cannot establish efficacy, and none of the retrieved reports isolates *Varunadi Gana* as the attributable intervention.

Ayurvedic Interpretation and Critical Synthesis

The classical *Phalasaruti* — *Kapha-Medohara*, with efficacy in *Siroroga*, *Gulma* and *Abhyantara Vidradhi* — can be read as a statement about *Amasaya* and *Srotas*. *Kapha* and *Medas* are the *Doṣa* and *Duṣya* most directly

implicated in *Srotorodha* (channel obstruction) and in the formation of a localised, indurated swelling. The drugs of the group are uniformly *Kaṭu* or *Tikta Rasa* with *Uṣṇa Virya* and *Laghu–Rukṣa Guna* — a profile that is *Dipana*, *Pachana*, *Kaphahara* and hence *Sothahara*. The *Vagbhata* review articulates the same logic precisely: *Vidradhi* is “a sophā in *Amavastha* and *Vrana* in *pakvavastha*,” and in the *ama avastha* of *antarvidradhi* the group is used with *Uṣakadi* drugs as adjuvant, its effect attributed to *Sophahara* and *Kaphavata Hara Karma* by virtue of *Kaṭu Rasa*, *Laghu Rukṣa Guna* and *Uṣṇa Virya*¹¹.

This provides a genuinely non-trivial translational hypothesis: the classical *Sothahara* action corresponds most plausibly to inhibition of the inflammatory cascade before it progresses to liquefactive necrosis and pus formation. The *Ama Avastha* is, in modern terms, the pre-suppurative phase — cellulitis, phlegmon, early inflammatory infiltrate — and it is precisely here that anti-inflammatory intervention is most likely to avert suppuration. Sushruta's insistence that “particular care should be taken by the physician to guard against the suppuration of an internal abscess” is not merely therapeutic conservatism; it is a rational statement of interceptive intent.⁵

Hypothesis the efficacy of *Varunadi Gana* in *Abhyantara vidradhi* in its *ama avastha* may be attributable to the combined (i) NF-κB/COX-2-mediated anti-inflammatory effect, (ii) *Srotomukha-Visodhana* through the *Dipana-Pachana* and mild purgative action of *Chitraka*, *Darbha* and allied drugs, and (iii) the antimicrobial contribution of *Sigru*, *Naktamala* and *Sairiyaka* species, delivered through the *Uṣakadi* and *Virechana* adjuncts and the *sneha* vehicle. This hypothesis is testable but untested.

Counter-consideration. Anti-inflammatory activity is not unambiguously desirable in suppurative disease. Once an abscess is established and draining, suppression of the inflammatory response could theoretically impede localisation and drainage. This is consistent with the classical stage-specificity of the prescription: early decoction therapy, but in *Pakvavastha* surgical *Bhedana* with the group relegated to adjunctive (with *Maireya/Sura/Asava*) and post-drainage roles. Modern antimicrobial stewardship and source-control principles

align with this reading — and constitute an argument against the indiscriminate use of *Sothahara* therapy in established abscess.

Safety Considerations

Chitraka (*Plumbago zeylanica*) contains plumbagin, a naphthoquinone with documented cytotoxicity; the literature explicitly notes that “toxic effects might be limited to effects like” those described in classical cautionary contexts, and plumbagin is classically regarded as requiring *Sodhana* (purification) before internal use³⁴. Hepatic and reproductive toxicity signals in animals warrant explicit clinical caution, particularly in a disease where the liver is itself a target organ.

Agnimantha (*Premna/Clerodendrum*) and *Tarkari* are bitter drugs with documented CNS-depressant and analgesic activity in animal models, implying additive sedation risk with concomitant medications.²⁶

Naktamala (*Pongamia pinnata*) — the oil and seeds contain furanoflavonoids with recognised toxicity potential; internal use of unprepared seed material is hazardous.

Bimbi (*Coccinia*) and *Vasira* (*Achyranthes*) are widely regarded as safe in food doses, but systematic toxicology is limited.

No herbal-drug interaction studies were retrieved for the *Gana* as a formulation. Given that the classical prescription co-administers *Varunadi* with *Uṣakadi* and *Virechana* groups, safety evaluation of the combination — not merely of individual drugs — is a research priority.

Translational Limitations

Five limitations constrain any claim of therapeutic potential. First, textual indeterminacy: the exact verse numbering of the *Varunadi* passage varies across editions, and several botanical identities (*Artagala*, *Vasuka*, *Meṣasṛṅgi*) remain contested.^{12, 47} This affects reproducibility of any pharmacological re-derivation. Second, compositional ambiguity: secondary compilations disagree on group membership (14 vs 20 entries), meaning “*Varunadi Gana*” is not a single, standardised formulation in current practice.^{10, 11} Third, extract–decoction mismatch: nearly all experimental studies use ethanolic, methanolic or hydro-alcoholic extracts at concentrations unattainable from a classical aqueous decoction, and bioavailability of the aglycone-rich fractions studied in vitro is substantially lower in

humans. Fourth, absence of clinical trials: no randomised or controlled clinical trial of *Varunadi Gana* in *Abhyantara Vidradhi* was retrieved, and available human evidence is case-report level and polyherbal, precluding attribution.¹³ Fifth, outcome non-comparability: *Vidradhi* is a clinical-pathological construct, and trials would need modern diagnostic endpoints (imaging, inflammatory markers) alongside Ayurvedic staging criteria, which no retrieved study provides.

Conclusion

The *Varunadi Gana* of the *Susrutasamhita*, *Sutrasthana* 38, is textually coherent and clinically specific. Its *Phalasruti* names *Abhyantara Vidradhi* explicitly, and Su.Ci.16 operationalises the group into a staged protocol — decoction with *Uśakadi* in the *Ama Avastha*, medicated ghee, and enema, with *Madhu-Sigru* as adjunct. The Ayurvedic rationale — *Kapha-Medo-Hara*, *Sothahara*, *Dipana-Pachana* maps intelligibly onto the modern understanding of pre-suppurative inflammation. Contemporary pharmacology corroborates the plausibility of this indication rather than proving it. Approximately three-quarters of the group's constituents have independently documented anti-inflammatory, antimicrobial or antioxidant activity, with mechanistic convergence on NF-κB and COX-2/PGE₂ — pathways central to the oedema-to-suppurative transition. This is a rational pharmacological signature, but it remains preclinical, extract-based, and unreplicated in the classical dosage form.

The honest critical position is therefore twofold: the classical indication is pharmacologically plausible and mechanistically explicable, but clinically unproven; and the group's greatest therapeutic promise lies in the interceptive window before suppuration, not in established abscess, where source control and antimicrobial therapy remain paramount. The priority now is standardisation of composition, authentication of disputed botanicals, toxicological characterisation of the combination, and stage-stratified clinical trials using modern diagnostic endpoints.

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