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"GUDUCHI (*TINOSPORA CORDIFOLIA*) AND HERB-INDUCED LIVER INJURY: A CRITICAL REVIEW OF CLINICAL REPORTS, CAUSALITY ASSESSMENT AND BOTANICAL IDENTITY, WITH IMPLICATIONS FOR DRAVYAGUNA PRACTICE"

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ABSTRACT
Guduchi (*Tinospora cordifolia* (Willd.) Hook.f. & Thomson) is one of the most widely used Ayurvedic drugs and is classically described as a rasayana indicated in jaundice (kamala). Its promotion as an immune booster during the COVID-19 pandemic was followed by reports from India of acute hepatitis with autoimmune features after its use, and by a polarised debate between hepatologists and the Ayurveda community. This critical review appraises the published clinical reports, the quality of causality assessment, the question of botanical identity, mechanistic plausibility and non-clinical safety data, and interprets them from a Dravyaguna perspective. Clinical evidence comprises two Indian case series, a multicentre retrospective study of 43 patients, and individual reports from other countries. Injury appeared idiosyncratic and often immune-mediated, and was most severe in people with pre-existing chronic liver disease. Causality was graded mostly as possible or probable, without rechallenge and without species-level authentication of the consumed material. Substitution with hepatotoxic *T. crispa* is documented in trade but cannot by itself explain the reports, because most tested market samples were authentic and *T. cordifolia* also contains clerodane furanoditerpenoids. A good laboratory practice 28-day study in rats found no toxicity of an authenticated aqueous stem extract up to 1000 mg/kg/day, which is consistent with an idiosyncratic rather than intrinsic mechanism. We propose a minimum reporting dataset for suspected cases and practice cautions grounded in the classical principles of dose, duration and patient assessment.

KEYWORDS: Guduchi, *Tinospora cordifolia*, herb-induced liver injury, RUCAM, pharmacovigilance, Dravyaguna

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INTRODUCTION

Guduchi (*Tinospora cordifolia* (Willd.) Hook.f. & Thomson, Menispermaceae) is a climbing shrub whose stem is among the most frequently used drugs in Ayurveda and in Indian household medicine.[1] Charaka lists the juice of Guduchi among the four medhya rasayana,[2] and the Bhavaprakasha Nighantu describes it as bitter and astringent, hot in potency (ushna virya), sweet in post-digestive effect (madhura vipaka), balancing all three dosha, and indicated in fever (jvara), jaundice (kamala), anaemia (pandu), diabetes (prameha), skin disease (kushtha) and vatarakta.[3] The stem is an official drug of the Ayurvedic Pharmacopoeia of India.[4] Preclinical studies have reported hepatoprotective, immunomodulatory, antidiabetic and anti-inflammatory activity, although well-designed clinical trials remain few.[1]

During the COVID-19 pandemic Guduchi was promoted widely as an immune booster, and its use, often self-directed and prolonged, rose sharply.[5,6] In July 2021 a hospital in Mumbai reported six patients who developed acute hepatitis with autoimmune features after taking Guduchi.[5] The Ministry of Ayush responded that linking Giloy to liver damage was misleading and that the authors had not analysed the herb that the patients had consumed.[7] A multicentre study of 43 patients followed,[6] as did commentaries from Ayurveda researchers arguing that substitution with the hepatotoxic *Tinospora crispa* was a more likely explanation,[8,9] and reviews by hepatologists listing Guduchi among the Ayurvedic herbs most often reported in liver injury.[10] The LiverTox database of the United States National Institutes of Health now grades *T. cordifolia* as a well-established cause of clinically apparent liver injury.[11]

The debate has been conducted largely in letters and commentaries, and the two sides have tended to talk past each other. Hepatologists have emphasised clinical phenotype and temporal association; Ayurveda scholars have emphasised identity, quality and classical safety. Both are legitimate questions of evidence, and both belong to Dravyaguna, which concerns itself with the correct identification of a drug as much as with its actions. This review therefore appraises the published clinical reports, the quality of causality assessment, the evidence on botanical identity and quality, mechanistic plausibility and non-clinical safety data, and interprets these findings in the light of classical principles. Its aim is not to defend or condemn Guduchi but to set out what is known, what is not, and what would settle the question.

METHODS

This is a critical narrative review. We searched PubMed and PubMed Central, publisher websites, the LiverTox database and Government of India press releases for publications on liver injury associated with *Tinospora* species, using combinations of the terms Tinospora cordifolia, Guduchi, Giloy, Tinospora crispa, herb-induced liver injury, drug-induced liver injury, hepatotoxicity, autoimmune hepatitis, RUCAM, authentication, DNA barcoding and toxicity. We included clinical case reports, case series and cohort studies of liver injury temporally associated with Guduchi; studies on authentication and adulteration of *Tinospora* species in trade; non-clinical toxicity studies; mechanistic studies on immunomodulation; and published commentaries. Classical descriptions were taken from the Charaka Samhita[2] and the Bhavaprakasha Nighantu.[3]

For each clinical report we extracted the setting, number of patients, product form, dose and duration where available, latency, clinical phenotype, autoantibodies, histology, causality grading and outcome, and noted whether alternative causes were excluded, whether the consumed product was retained and analysed, and whether its species was confirmed. Causality grading was interpreted against the updated Roussel Uclaf Causality Assessment Method (RUCAM).[12]

GUDUCHI IN CLASSICAL DRAVYAGUNA

Classical texts know Guduchi by synonyms such as Amrita, Chinnaruha and Madhuparni, which reflect its reputation for preserving life and its ability to regenerate from cut stems.[3] The part used is the stem, which is administered as fresh juice (swarasa), decoction (kvatha), powder (churna) and the starch-rich aqueous sediment known as Guduchi satva.[4] These dosage forms differ in how much of the lipophilic and diterpenoid fraction they extract, a point that matters when comparing toxicity data across preparations but that has received little attention.

Two classical principles are relevant to the present debate. First, rasayana therapy is not prescribed unconditionally: Charaka frames it within assessment of the person and preparation of the body, and the rasayana chapters repeatedly tie benefit to

suitability.[2] Second, Charaka states that even a strong poison can become an excellent medicine when used correctly, and that a medicine used wrongly becomes a poison.[2] Classical authority therefore does not claim that a drug is safe in every person, dose and duration; it claims that safety depends on correct use. This is close to the modern concept of an idiosyncratic adverse reaction and offers common ground between the two communities.

A further classical point concerns identity. More than one *Tinospora* species is used under the name Guduchi in different regions; *T. sinensis* in particular shares the common name with *T. cordifolia.*[13] Dravyaguna has long recognised that a drug cannot be judged unless it is first correctly identified, and this applies equally to claims of safety and of harm.

CLINICAL EVIDENCE OF LIVER INJURY

The published clinical evidence is summarised in Table 1. The first case series described six patients (four women and two men, median age 55 years) seen between September and December 2020.[5] All presented with acute hepatitis after taking Guduchi as an immunity measure; four had a definite and two a probable revised autoimmune hepatitis score, and liver biopsy showed an autoimmune pattern of drug-induced liver injury. The authors proposed that Guduchi either induces an autoimmune-like hepatitis or unmasks an underlying autoimmune chronic liver disease.[5]

The largest study is a retrospective analysis from 13 centres across India covering April 2020 to July 2021.[6] Of 49 patients enrolled, 43 were analysed; 53.5% were women and the mean age was 50.4 years. Median time from first intake to symptoms was 46 days and median duration of use was 42 days. The most common presentation was acute worsening of chronic liver disease (51.1%), followed by acute hepatitis (44.2%) and acute liver failure (4.7%). Chronic liver disease was already known in 16.3% and newly diagnosed during evaluation in a further 39.5%. Antinuclear antibody was positive in 41.9%. RUCAM graded causality as probable in 67.4% and possible in 32.6%. Four patients (9.3%), all with underlying chronic liver disease, died, and two underwent liver transplantation. Of the 22 patients who had a biopsy, interface hepatitis and cholestasis were each seen in 77.3% and eosinophils in 68.2%, and none met criteria for definite autoimmune hepatitis.[6]

Outside India, a community hospital in California reported three patients with liver injury after Ayurvedic products, one of whom had taken a decoction containing only *T. cordifolia,*[14] and a case from the United States described probable injury (RUCAM score 6) after a supplement containing 900 mg of *T. cordifolia* stem extract together with quercetin and stinging nettle.[15] Earlier, a single-centre Indian series of severe liver injury associated with Ayurvedic and herbal medicines had found Guduchi as an ingredient in eight of the formulations retrieved, with autoantibodies in 29.6% of patients.[6,16] Hepatologists have since reviewed the wider Ayurvedic literature on hepatotoxicity,[10,17] and LiverTox notes more than 50 published cases, a clinical picture suggesting immune mediation, and a higher risk of severe outcomes in people with pre-existing liver disease.[11]

APPRAISAL OF CAUSALITY

RUCAM assigns points to latency, course after withdrawal, risk factors, concomitant drugs, exclusion of alternative causes, previous reports of hepatotoxicity and response to re-exposure, and grades causality from excluded to highly probable.[12] Its developers specify that it is intended primarily for prospective use, because retrospective scoring is less accurate; that it must be calculated separately for each co-administered product; and that it applies to acute liver injury rather than to pre-existing chronic liver disease.[12] Each of these conditions bears on the Guduchi literature.

The multicentre study was retrospective, and more than half of its patients presented with acute worsening of chronic liver disease, a setting for which RUCAM was not designed.[6,12] Over 40% took Guduchi within multi-herbal products, five patients took other herbal preparations intermittently, and heavy metals above permitted limits were found in two of the six retrieved products.[6] Rechallenge was not attempted, for sound ethical reasons. These features lower the certainty of attribution in individual cases.

Table 1. Published clinical reports of liver injury temporally associated with Guduchi.

Report	Setting and patients	Exposure and phenotype	Causality and identity
Nagral et al., 2021[5]	Single centre, Mumbai; 6 patients (4 women)	Self-administered Guduchi for immunity; acute hepatitis with autoimmune features; biopsy autoimmune-pattern DILI	Revised AIH score definite in 4, probable in 2; product species not confirmed
Kulkarni et al., 2022[6]	13 centres, India; 43 analysed	Pure Guduchi in 58.1%, multi-herbal in 41.9%; median latency 46 days; acute-on-chronic disease in 51.1%; 4 deaths, 2 transplants	RUCAM probable 67.4%, possible 32.6%; 6 retrieved products screened for metals and by GC/MS-MS; species-level authentication not reported
Philips et al., 2018[16]	Single centre, Kerala; severe liver injury after herbal medicines	Guduchi an ingredient in 8 retrieved formulations; autoantibodies in 29.6%	Multi-herbal products; attribution to Guduchi alone not possible
Karousatos et al., 2021[14]	Community hospital, USA; 3 patients	One patient took a decoction of *T. cordifolia* alone; acute hepatocellular injury with jaundice	Authors stress complexity of causality
Nnamani et al., 2023[15]	Single case, USA	Combination product with 900 mg *T. cordifolia* stem extract, quercetin and nettle	RUCAM 6 (probable)

On the other hand, the signal has several features that make a causal association credible at the population level (Table 2). Independent centres in different regions and countries described a consistent phenotype with a similar latency, recovery or improvement followed withdrawal in most patients, and the histological pattern of mixed portal inflammation, eosinophils and cholestasis is typical of drug- or herb-induced injury rather than of classical autoimmune hepatitis.[5,6] The most balanced reading is that an association between Guduchi use and idiosyncratic liver injury is plausible and consistent, that causality in individual cases is mostly possible or probable rather than certain, and that the incidence of injury among users is unknown because no study has a denominator of exposed people.

Table 2. Strengths and limitations of the current clinical evidence.

Features supporting a causal association	Features limiting certainty
Similar phenotype and latency reported independently from many centres and from outside India[6,14,15]	Retrospective data collection; recall-based estimates of dose and duration[6]
Improvement after withdrawal in most patients[6]	RUCAM applied in acute-on-chronic disease, outside its validated scope[12]
Biopsy features typical of herb-induced injury rather than classical autoimmune hepatitis[6]	Multi-herbal products and concomitant herbs in a substantial minority[6]
Plausible immune mechanism	Heavy-metal contamination in some retrieved products[6]
Cases with single-ingredient products[6,14]	No species-level authentication of consumed material in any report; no incidence data

BOTANICAL IDENTITY AND QUALITY OF THE CONSUMED MATERIAL

The substitution hypothesis deserves serious attention because *T. crispa* is documented hepatotoxin. A French case of toxic hepatitis after high-dose *T. crispa* pellets was confirmed by microscopy and chromatographic fingerprinting;[18] a later case led to the identification of borapetoside C

and seventeen related cis-clerodane furanoditerpenoid lactones as probable toxins, with a mechanism similar to that of other furanoditerpenoid-containing plants such as *Teucrium chamaedrys* and *Dioscorea bulbifera;*[19] and a review from China added two cases of fulminant hepatitis.[20] The Ministry of Ayush and several Ayurveda authors have argued that some Guduchi-associated cases may reflect such substitution.[7–9]

Three observations nevertheless argue against substitution as a complete explanation. First, *T. cordifolia* itself contains clerodane furanoditerpenes,[21] so the chemical class implicated in *T. crispa* toxicity is not unique to that species, a point also made by hepatologists in the debate.[6,22] Second, in a metabolomic survey of 25 commercial *Tinospora* products from different parts of India, 20 were authentic *T. cordifolia* and 5 were suspicious:[23] adulteration exists, but most products were genuine. Third, the multicentre study included patients who used home-collected plant material (27.9%) as well as products from licensed manufacturers (11.6%), so the cases did not arise from a single supply chain.[6]

The decisive point is that the identity question is now answerable. Species-specific DNA markers distinguish *T. cordifolia* from its adulterants with full sensitivity and specificity in validation;[13] microscopy combined with HPTLC identifies *T. crispa* through borapetosides B and C and *T. sinensis* through tinosineside A;[24] and high-resolution mass spectrometry with chemometric models discriminates the three species in finished products.[23] No published clinical case has yet used any of these methods on the material the patient consumed. Until they are applied routinely, neither the claim that Guduchi is hepatotoxic nor the claim that the cases were caused by substitution can be proved for any individual patient.[25]

MECHANISTIC PLAUSIBILITY AND NON-CLINICAL SAFETY

Guduchi is well documented as an immunostimulant. Early work isolated immunopotentiating compounds from the stem;[26] an arabinogalactan polysaccharide from the aqueous extract showed polyclonal B-cell activation;[27] and further active compounds and a broad range of immune effects have since been described.[28,29] An immune-stimulating drug could plausibly precipitate or unmask autoimmune-like liver injury in a predisposed person. This mechanism is recognised for drugs and other herbs,[30] and immunostimulatory supplements have been reported to activate or exacerbate autoimmune disease in susceptible people.[31] The frequent finding of antinuclear antibodies and the high proportion of patients with previously unrecognised chronic liver disease in the Indian series are consistent with this hypothesis.[6]

Non-clinical data point the same way. A good laboratory practice study following OECD guideline 407 gave an authenticated aqueous stem extract to rats at 100, 300 and 1000 mg/kg/day for 28 days and found no mortality or treatment-related changes in clinical signs, haematology, clinical chemistry or organ histology, setting the no-observed-adverse-effect level at 1000 mg/kg/day.[32] This argues against intrinsic, dose-dependent hepatotoxicity of the authentic stem extract. It does not exclude idiosyncratic injury, which standard repeated-dose rodent studies are not designed to detect, so the animal and human data are not in conflict. It should also be noted that the study was conducted by a manufacturer of Guduchi products.[32]

A DRAVYAGUNA PERSPECTIVE

Viewed through Dravyaguna, the reports describe a pattern of use that differs from classical practice in several respects. Classical use of Guduchi as a rasayana presupposes assessment of the individual and attention to suitability,[2] whereas pandemic use was population-wide, self-directed and often continued for weeks or months without any assessment.[6] Classical prescription specifies dose and duration; in the multicentre study daily intake of decoction ranged from 15 to 90 mL and use extended in some patients to almost a year.[6] Classical texts also attend to the state of the patient (avastha) and to underlying disease; the high proportion of undiagnosed chronic liver disease among affected patients suggests that the people most at risk were precisely those whom a careful physician would have identified before prescribing.

This reading does not dismiss the reports. Charaka's statement that a medicine wrongly used becomes a poison[2] is an acknowledgement that harm from a classical drug is possible. The concept of individual susceptibility encoded in prakriti and avastha parallels the modern concept of idiosyncrasy, and a drug that is beneficial in kamala in one person may still precipitate injury in another with latent autoimmune

liver disease. Dravyaguna therefore provides a framework both for taking the signal seriously and for defining safer use, rather than a reason to reject it.

RECOMMENDATIONS

Clinical practice

- Ask about known or suspected liver disease, autoimmune disease and previous drug-induced liver injury before prescribing Guduchi for more than a few weeks.
- Prefer pharmacopoeial-grade, batch-identified products from licensed manufacturers over unidentified or home-collected material.
- Prescribe a defined dose and duration, and avoid open-ended self-medication for prevention.
- Advise patients to stop Guduchi and seek review at the first sign of jaundice, dark urine, pruritus or unexplained fatigue, and consider baseline and periodic liver tests in prolonged use.

Pharmacovigilance and case reporting

Future case reports should meet a minimum dataset (Table 3) that allows both hepatologists and Ayurveda experts to evaluate them. The central requirement is that the consumed product be retained and authenticated at species level, and that RUCAM be applied to each product separately.[12,25] Cases should be reported to the national pharmacovigilance programme for Ayurveda, Siddha, Unani and Homoeopathy drugs as well as to conventional systems.

Table 3. Proposed minimum dataset for suspected Guduchi-associated liver injury.

Domain	Items
Product	Name, manufacturer, batch, licence number, form (swarasa, kvatha, churna, satva, tablet, extract), plant part, single- or multi-ingredient, source
Exposure	Dose, frequency, start and stop dates, purpose, prescriber or self-medication, concomitant drugs and herbs
Identity and quality	Retained sample; species confirmation by DNA marker, microscopy with HPTLC, or LC-HRMS; heavy metals, pesticides, aflatoxins and solvents
Patient	Age, sex, pre-existing liver or autoimmune disease, alcohol use, viral and metabolic work-up
Injury	Pattern (hepatocellular, cholestatic, mixed), autoantibodies, IgG, biopsy where performed, outcome after withdrawal
Causality	RUCAM calculated for each product separately; any unintentional re-exposure

RESEARCH

Priorities include prospective registries with authenticated samples; case–control or cohort studies that estimate incidence among users; comparison of dosage forms, since satva, decoction and solvent extracts differ chemically; studies of genetic or immunological susceptibility; and joint review panels that include both hepatologists and Ayurveda pharmacologists, so that clinical and botanical expertise are applied to the same cases.

LIMITATIONS

This is a narrative rather than a systematic review, and some reports published in regional or non-indexed journals may have been missed. Most clinical data come from India during 2020 to 2022, when use was unusually high and often unsupervised, so they may not reflect risk under classical prescription. Published cases are subject to reporting bias in both directions. Finally, we could not assess primary data, and our appraisal depends on what the original authors reported.

CONCLUSION

The published evidence supports a real but uncommon association between Guduchi use and idiosyncratic, often autoimmune-like, liver injury, with the greatest risk in people with pre-existing liver disease. It does not support the view that authentic Guduchi is an intrinsic hepatotoxin, nor does it show that the reported cases were caused by substitution, because no case has been authenticated at species level. Both the hepatology and Ayurveda communities can close this gap by adopting a shared reporting standard that combines careful causality assessment with botanical and chemical authentication. Until then, Guduchi should be prescribed as classical texts intend: for a defined purpose, dose and duration, to a person who has been assessed, rather than as an open-ended preventive.

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