

Adverse Drug Reactions from Ayurvedic Medicines: A Case-Based Toxicological Review

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Abstract

Ayurvedic medicines are pharmacologically active and may cause adverse drug reactions (ADRs) through predictable dose-related toxicity, idiosyncratic susceptibility, interactions, inappropriate use, or product-quality defects. This concise case-based review integrates routine hospital pharmacovigilance with published reports of aconite cardiotoxicity, herb-induced liver injury, mercury-associated renal injury, and lead or arsenic poisoning. A one-year prospective study in an Ayurvedic teaching hospital recorded 84 adverse events, of which 52 were confirmed as ADRs; gastrointestinal and cutaneous reactions predominated and most were mild or moderate. Severe published signals included profound hypotension and bradycardia after excessive *Mahashankha Vati*, cholestatic or autoimmune-like hepatitis associated with *Ashwagandha*, *Tinospora cordifolia* and polyherbal formulations, anuric renal failure after a mercury-containing medicine, and multisystem toxicity linked to lead- or arsenic-containing products. These cases do not justify generalization to all Ayurvedic medicines, but they disprove the assumption that traditional or natural products are universally risk-free. The review proposes denominator-based surveillance in Ayurvedic hospitals, standardized product-level documentation, preservation and laboratory testing of implicated batches, structured causality assessment, and an *Agad Tantra*-led sentinel network linked to national pharmacovigilance. Safe use requires qualified prescribing, traceable manufacturing, appropriate dose and duration, patient counseling, early recognition, and non-punitive reporting.

Keywords: Ayurveda; adverse drug reaction; pharmacovigilance; *Vatsanabha*; herb-induced liver injury; heavy metals; *Agad Tantra*.

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1. Introduction

Ayurveda uses medicines of herbal, mineral, metallic and animal origin, often in complex formulations. Therapeutic activity, however, also creates the possibility of unwanted effects when dose, duration, preparation, co-medication, patient susceptibility or product quality is unfavorable. WHO therefore recommends that herbal medicines be included in pharmacovigilance systems with clear documentation of composition, quality and concomitant therapy [1]. An ADR is a harmful and unintended response for which a causal relationship with a medicinal product is at least reasonably possible [2,3].

Medicine-related harm in Ayurveda should not be treated as a single category. A reaction at a recommended dose differs from overdose, and both differ from contamination, substitution, adulteration or manufacturing failure. This distinction is central to toxicological interpretation and fair communication. The purpose of this review is to retain the principal published clinical signals, examine how causality should be judged, and propose a practical future agenda for safer Ayurvedic practice without portraying the entire system as either harmless or dangerous.

2. Scope and Toxicological Framework

A targeted narrative review of English-language human reports and authoritative guidance available through August 2026 was undertaken. Case reports, case series, analytical product investigations and hospital

pharmacovigilance studies involving identifiable Ayurvedic medicines or ingredients were prioritized. Because this was not a systematic review, the cases illustrate signals and mechanisms rather than incidence or comparative risk.

The evidence is interpreted through four overlapping pathways: intrinsic dose-related toxicity, exemplified by aconite; idiosyncratic or immune-mediated injury, particularly herb-induced liver injury (HILI); quality-related exposure to undeclared or excessive toxic elements; and use-related harm from self-medication, duplicate formulations, prolonged exposure or interactions. Each pathway demands a different preventive response.

3. Evidence from Routine Ayurvedic Hospital Practice

Ajanal et al. prospectively monitored patients at KLE Ayurveda Secondary Care Hospital, Belgaum, for one year using spontaneous and intensive surveillance [4]. They recorded 84 adverse events, of which 52 were confirmed as ADRs, giving a reported incidence of 1.14%. Among the confirmed reactions, 23 (44.23%) were related to *Panchakarma* procedures, 13 (25.00%) to herbal formulations and six (11.53%) to *Rasa Aushadhi*. The gastrointestinal system was affected in 24 cases (46.15%) and the skin in 15 (28.84%). Thirty reactions (57.69%) were moderate and 20 (38.46%) mild; most patients recovered.

This study corrects two misconceptions. First, recognizable ADRs occur in ordinary Ayurvedic clinical practice, not only in isolated poisoning reports. Second, most detected events were manageable rather than catastrophic. The correct figures are 84 adverse events and 52 confirmed ADRs, not 85 and 54. Its main limitation is that one hospital and one year cannot represent all formulations or settings, yet it demonstrates the value of active surveillance with a denominator.

The distribution also shows why surveillance must extend beyond tablets and powders. Nearly half of the confirmed reactions were associated with *Panchakarma* procedures, so safety review must capture procedure, material used, technique, timing and operator-related factors in addition to medicinal products. At the same time, the herbal and *Rasa Aushadhi* categories establish that product-specific monitoring remains necessary. A comparable protocol used across multiple teaching hospitals could distinguish local practice patterns from reproducible signals.

4. Representative Published Cases

Table 1 retains all principal case signals reviewed. The word associated is used where definitive ingredient-level causality was not established.

Exposure / report	Main presentation	Interpretation	Outcome / lesson
<i>Mahashankha Vati</i> overdose [7]	Numbness, nausea, vertigo, BP 30/10 mmHg and pulse 46/min.	Predictable aconite toxicity after excessive <i>Vatsanabha</i> exposure.	Recovered with urgent care; record cumulative dose and duplicate formulations.
<i>Ashwagandha</i> case series and synthesis [9,10]	Jaundice and pruritus with cholestatic or mixed liver injury.	Probable idiosyncratic HILI; incidence is unknown.	Most recovered after withdrawal; severe cases required intensive care or transplantation.
<i>Tinospora cordifolia</i> , six patients [11]	Acute hepatitis with autoimmune features; several had predisposition.	Autoimmune-like injury or unmasking of silent liver disease.	Five improved; susceptibility and alternative causes require assessment.

Exposure / report	Main presentation	Interpretation	Outcome / lesson
<i>Giloy</i> /polyherbal formulations, three cases [12]	Hepatocellular injury after 90-120 days; RUCAM 7-8.	Probable HILI, but one ingredient cannot be isolated in complex products.	All recovered after withdrawal; preserve the exact product and ingredient list.
Mercury-containing medicine in a child [14]	Anuria, creatinine 11 mg/dL, nephritis, hypertension and seizures.	Renal mercury toxicity supported by product analysis.	Dialysis for 17 days followed by renal recovery; pediatric exposure needs special caution.
Three lead-toxicity cases [15]	Abdominal colic, anemia and markedly elevated blood lead.	Chronic lead exposure confirmed in consumed medicines.	Improved after exposure removal and chelation; investigate the implicated batch.
Three chronic arsenic cases [16]	Systemic, neurologic and cutaneous manifestations.	Cumulative arsenic exposure; alternative sources must be excluded.	Long-latency toxicity may mimic common disease.
<i>Bhasma</i> -associated adult case [17]	Fatigue, constipation, weight loss, cognitive symptoms; blood lead 94 microg/dL.	Product contained 19,400 mg/kg lead and 1,430 mg/kg arsenic.	Biomarker and product analysis strongly supported causation.

Exposure / report	Main presentation	Interpretation	Outcome / lesson
Long-term prescribed supplements [18]	Severe multisystem arsenic and lead toxicity.	High cumulative metal burden despite prescribed use.	Prescription cannot replace batch quality assurance and clinical monitoring.

Table 1: Representative case reports and series. HILI: herb-induced liver injury; RUCAM: Roussel Uclaf Causality Assessment Method.

5. Toxicological Interpretation of the Cases

5.1 Aconite-related cardiotoxicity

Vatsanabha (*Aconitum ferox*) contains potent aconite alkaloids that persistently activate voltage-gated sodium channels, producing paresthesia, hypotension, bradyarrhythmia or malignant ventricular arrhythmia [7,8]. The *Mahashankha Vati* report involved about 70 mg of *Vatsanabha* against a cited recommended dose of 15 mg and is best classified as dose-related poisoning, not an idiosyncratic ADR [7]. Management requires stabilization, continuous ECG monitoring and treatment of hemodynamic compromise or arrhythmia; no universally accepted specific antidote exists [8]. Prevention depends on correct processing, standardized content, supervised dosing and avoidance of overlapping aconite-containing formulations.

5.2 Herb-induced liver injury

HILI is a diagnosis of exclusion. Updated RUCAM improves consistency by examining latency, dechallenge, risk factors, concomitant medicines, competing diagnoses and re-exposure [5,6]. *Ashwagandha* reports most often describe jaundice and pruritus with cholestatic or mixed injury [9,10]. *Tinospora cordifolia* has been associated with autoimmune-like hepatitis, sometimes in patients with pre-existing autoimmune susceptibility [11]. Three reports involving *Giloy Kwath*, *Manjishthadi Kwatham*, *Aragwadhadi Kwatham* and *Kanchnar Guggulu* showed hepatocellular injury, RUCAM scores of 7-8 and recovery after withdrawal [12]. These signals warrant vigilance, but multi-ingredient products and incomplete authentication limit ingredient-specific conclusions [13].

5.3 Mercury, lead and arsenic

The mercury case demonstrates target-organ injury supported by direct product testing: a two-year-old child developed anuric renal failure, tubulointerstitial nephritis, hypertension and seizures after five months of exposure [14]. Lead and arsenic reports show a different chronic pattern involving abdominal pain, anemia, constipation, neuropathy, cognitive change, skin findings or multisystem dysfunction [15-18]. The strongest evidence combines compatible symptoms, biomarker elevation and analysis of the consumed product, as in the

Bhasma case [17]. Product surveys have detected lead, mercury or arsenic in selected market samples, reinforcing the need for batch testing without permitting generalization to all Ayurvedic products [19,20].

Quality-related toxicity should be attributed to the tested product and batch, not automatically to every preparation bearing a similar classical name. Relevant questions include whether the material was manufactured under an authorized process, whether elemental content was declared, whether the consumed dose and duration were appropriate, and whether contamination or substitution occurred. This product-centered approach protects patients while preventing an analytically confirmed defect in one item from being converted into an unsupported claim about all mineral or herbo-mineral medicines.

6. Causality, Recognition and Immediate Management

A temporal association alone is insufficient. Assessment should record the exact formulation, brand, manufacturer, batch, label, source, dose, duration, *anupana*, recent escalation, duplicate products and prescription status. Patient factors, conventional medicines, supplements, alcohol, occupational exposure and alternative diagnoses must be examined. Dechallenge, inadvertent re-exposure, objective biomarkers and product analysis strengthen causality; deliberate rechallenge after a serious reaction is inappropriate.

The suspected product should be withheld while severity-based care proceeds. Aconite syndromes need ECG monitoring; suspected HILI needs liver tests, coagulation assessment, imaging and exclusion of viral, autoimmune, ischemic and obstructive causes. Unexplained anemia with abdominal or neurologic symptoms should prompt lead testing when exposure is plausible, while arsenic and mercury testing should follow toxicology guidance. Chelation depends on the measured toxicant, clinical severity and renal function, not merely on the belief that a product contains metal. The remaining medicine, container and batch should be preserved for analysis and possible medicolegal investigation.

For reporting, the event should first be classified as suspected ADR, overdose, interaction, procedural complication, medication error or quality defect; more than one category may apply. WHO-UMC categories can support general assessment, while RUCAM is useful for liver injury [5,6]. Confidence increases when chronology, objective findings, dechallenge and product analysis converge. Conversely, unclear labeling, several simultaneous formulations and absent batch testing should lower certainty and be stated transparently rather than hidden behind a definitive causal label.

7. Future Directions and Recommendations

This review proposes that the next phase of Ayurvedic drug safety move from isolated reporting to linked clinical-and-product evidence. First, teaching hospitals should establish prospective ADR surveillance with a

defined denominator, a trained coordinator and monthly review. *Agad Tantra* departments can serve as sentinel units because they connect clinical toxicology, forensic documentation, poison recognition and public health. Every suspected case should be reported even before certainty is established, because signal detection depends on aggregation [21-24].

Second, an Ayurveda-specific minimum dataset should supplement standard pharmacovigilance forms. It should include classical and commercial formulation names, complete ingredients, manufacturer and batch, dose, duration, *anupana*, processing claims, co-administered medicines, practitioner or self-medication status, chronology, dechallenge and outcome. *Prakriti*, *Agni* and other individualized variables may be recorded prospectively as exploratory factors, but should not be presented as proven predictors until validated.

Third, hospitals should retain sealed samples of implicated medicines and develop referral links with accredited laboratories for botanical authentication, toxic-element testing and adulterant screening. A digital registry should connect the clinical record, causality assessment and laboratory result to the same batch. Serious clusters should trigger rapid communication with manufacturers and regulators while maintaining chain of custody.

Fourth, multicentric prospective cohorts are needed to estimate incidence by formulation and exposure duration. Nested case-control studies can investigate patient susceptibility, while analytical studies should compare declared composition with actual batch content. Training should make ADR reporting a routine professional responsibility, not an accusation against Ayurveda. Public communication should similarly avoid both extremes: Ayurvedic medicines are not universally risk-free, yet a case linked to one patient-product-use combination cannot be generalized to an entire system.

A practical three-level model is suggested. At bedside level, the practitioner documents exposure, recognizes warning symptoms, withdraws the suspected product when appropriate and reports the event. At institutional level, an ADR committee led jointly by pharmacovigilance personnel and *Agad Tantra* reviews causality, preserves samples, coordinates laboratory testing and audits recurring patterns. At regulatory level, de-identified clinical and batch data should be pooled for signal detection, manufacturer feedback, targeted inspection and risk communication. Annual institutional reports should include both the number of ADRs and the number of exposed patients so that trends are interpretable.

Future studies should predefine outcomes such as hospitalization, organ injury, time to recovery and preventability. They should also compare prescribed use with self-medication, single products with polypharmacy, and short exposure with prolonged exposure. Where a signal recurs, pharmacognostic authentication, elemental analysis and modern chemical profiling should accompany clinical causality

assessment. Such integration would generate evidence that is simultaneously relevant to Ayurveda, toxicology and public health.

8. Discussion and Limitations

The reviewed evidence demonstrates heterogeneous harm: predictable aconite toxicity, idiosyncratic liver injury and product-related metal exposure require different prevention strategies. Case reports are valuable sentinels but lack a denominator, favor severe outcomes and often omit authentication, dose or batch testing. Even improvement after withdrawal does not eliminate every competing cause. Conversely, underreporting and failure to ask about traditional medicines may conceal genuine harm. The hospital study provides a denominator but remains a single-center observation. Therefore, this review identifies safety signals and priorities rather than the frequency or comparative risk of properly manufactured and appropriately prescribed Ayurvedic medicines.

The most useful evidence hierarchy is therefore not based on whether a medicine is labeled traditional or modern. It begins with a compatible clinical syndrome and reliable chronology, becomes stronger with exclusion of alternatives and recovery after withdrawal, and is strongest when a relevant biomarker or analysis of the consumed batch confirms exposure. Applying this hierarchy consistently would improve both patient safety and the credibility of published Ayurvedic ADR reports.

9. Conclusion

Ayurvedic medicines can be associated with clinically important ADRs and toxic syndromes, ranging from mild gastrointestinal or skin reactions to cardiotoxicity, liver injury, renal failure and chronic metal poisoning. The scientifically balanced response is neither denial nor blanket condemnation. Qualified prescribing, correct dose and duration, traceable manufacturing, batch-level quality assurance, early recognition, structured causality assessment and systematic reporting are essential. An *Agad Tantra*-led, hospital-based sentinel network linking patient findings with product analysis would convert scattered case reports into actionable safety evidence and strengthen confidence in responsible Ayurvedic practice.

Declarations

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References

1. World Health Organization. WHO guidelines on safety monitoring of herbal medicines in

- pharmacovigilance systems. Geneva: World Health Organization; 2004.
2. World Health Organization. Pharmacovigilance [Internet]. Geneva: World Health Organization; [cited 2026 Sep 6]. Available from: <https://www.who.int/teams/regulation-prequalification/regulation-and-safety/pharmacovigilance>
3. Edwards IR, Aronson JK. Adverse drug reactions: definitions, diagnosis, and management. *Lancet*. 2000;356(9237):1255-9. doi:10.1016/S0140-6736(00)02799-9.
4. Ajanal MN, Nayak SU, Kadam AP, Prasad BS. Pharmacovigilance study of Ayurvedic medicine in Ayurvedic teaching hospital: a prospective survey study. *Ayu*. 2015;36(2):130-7. doi:10.4103/0974-8520.175539.
5. Danan G, Teschke R. RUCAM in drug and herb induced liver injury: the update. *Int J Mol Sci*. 2016;17(1):14. doi:10.3390/ijms17010014.
6. Hayashi PH, Lucena MI, Fontana RJ, Bjornsson ES, Aithal GP, Barnhart H, et al. A revised electronic version of RUCAM for the diagnosis of DILI. *Hepatology*. 2022;76(1):18-31. doi:10.1002/hep.32327.
7. Panda AK, Debnath SK. Overdose effect of aconite containing Ayurvedic medicine ('*Mahashankha Vati*'). *Int J Ayurveda Res*. 2010;1(3):183-6. doi:10.4103/0974-7788.72493.
8. Tak S, Lakhota M, Gupta A, Sagar A, Bohra G, Bajari R. Aconite poisoning with arrhythmia and shock. *Indian Heart J*. 2016;68 Suppl 2:S207-9. doi:10.1016/j.ihj.2015.08.010.
9. Bjornsson HK, Bjornsson ES, Avula B, Khan IA, Jonasson JG, Ghabril M, et al. *Ashwagandha*-induced liver injury: a case series from Iceland and the US Drug-Induced Liver Injury Network. *Liver Int*. 2020;40(4):825-9. doi:10.1111/liv.14393.
10. Felemban R, Aljefri L, Shaheen Y, Sindi G, Albadri M, Bukhari JI, et al. *Ashwagandha*-induced liver injury: a systematic review of case reports. *Egypt J Intern Med*. 2026;38:97. doi:10.1186/s43162-026-00683-2.
11. Nagral A, Adhyaru K, Rudra OS, Gharat A, Bhandare S. Herbal immune booster-induced liver injury in the COVID-19 pandemic - a case series. *J Clin Exp Hepatol*. 2021;11(6):732-8. doi:10.1016/j.jceh.2021.06.021.
12. Karousatos CM, Lee JK, Braxton DR, Fong TL. Case series and review of Ayurvedic medication induced liver injury. *BMC Complement Med Ther*. 2021;21(1):91. doi:10.1186/s12906-021-03251-z.
13. Philips CA, Ahamed R, Rajesh S, George T, Mohanan M, Augustine P. Comprehensive review of hepatotoxicity associated with traditional Indian Ayurvedic herbs. *World J Hepatol*. 2020;12(9):574-95. doi:10.4254/wjh.v12.i9.574.
14. Sathe K, Ali U, Ohri A. Acute renal failure secondary to ingestion of Ayurvedic medicine containing mercury. *Indian J Nephrol*. 2013;23(4):301-3. doi:10.4103/0971-4065.114485.
15. Raviraja A, Vishal Babu GN, Sehgal A, Saper RB, Jayawardene I, Amarasiriwardena CJ, et al. Three cases of lead toxicity associated with consumption of Ayurvedic medicines. *Indian J Clin Biochem*. 2010;25(3):326-9. doi:10.1007/s12291-010-0051-9.
16. Pinto B, Goyal P, Flora SJS, Gill KD, Singh S. Chronic arsenic poisoning following Ayurvedic medication. *J Med Toxicol*. 2014;10(4):395-8. doi:10.1007/s13181-014-0389-0.
17. Breeher L, Gerr F, Fuortes L. A case report of adult lead toxicity following use of Ayurvedic herbal medication. *J Occup Med Toxicol*. 2013;8:26. doi:10.1186/1745-6673-8-26.
18. Hardin J, Seltzer J, Suhandynata R, Spiegel B, Silver R, Thomas D, et al. Severe arsenic poisoning due to Ayurvedic supplements. *Clin Case Rep*. 2023;11(7):e7733. doi:10.1002/ccr3.7733.
19. Saper RB, Phillips RS, Sehgal A, Khouri N, Davis RB, Paquin J, et al. Lead, mercury, and arsenic in US- and Indian-manufactured Ayurvedic medicines sold via the Internet. *JAMA*. 2008;300(8):915-23. doi:10.1001/jama.300.8.915.
20. Mikulski MA, Wichman MD, Simmons DL, Pham AN, Clottey V, Fuortes LJ. Toxic metals in Ayurvedic preparations from a public health lead poisoning cluster investigation. *Int J Occup Environ Health*. 2017;23(3):187-92. doi:10.1080/10773525.2018.1447880.
21. Centers for Disease Control and Prevention. Lead poisoning associated with Ayurvedic medications - five states, 2000-2003. *MMWR Morb Mortal Wkly Rep*. 2004;53(26):582-4.
22. Baghel MS. The national pharmacovigilance program for Ayurveda, Siddha and Unani drugs: current status. *Int J Ayurveda Res*. 2010;1(4):197-8. doi:10.4103/0974-7788.76779.
23. Ministry of Ayush, Government of India. Ayush Suraksha: pharmacovigilance for ASU&H drugs [Internet]. New Delhi: Ministry of Ayush; [cited 2026 Sep 6]. Available from: <https://suraksha.ayush.gov.in/>
24. Thatte U, Bhalerao S. Pharmacovigilance of Ayurvedic medicines in India. *Indian J Pharmacol*. 2008;40 Suppl 1:S10-2. doi:10.4103/0253-7613.40236.