ISSN:3139-0935(Online)

Intemed: International Journal of Research in Integrated Medical Sciences (IJ-RIMS)

Journal homepage: www.intemed.in

Vol.: 2 | Issue: 8 | August 2026

Article History: Received: 05-08-2026 Accepted: 9-08-2026 Published:30-08-2026

Review Article

Visha Vega and Modern Toxicokinetics: A Comparative Review of Poison Progression

Dr. Sangeeta B. Bhosale[1], Dr. Sheetal Prakash Mirajkar (Koparde)[2], Dr. Shubham Ramnarayan Dhoot[3]

[1] Assistant Professor, Department of Agadtantra evam Vidhivaidyak, Sai Ayurveda College, Hospital & Research Center, Sasure, Vairag, Dist-Solapur, Maharashtra, India - 413402.

Email: drsangitadeshkukh@gmail.com

[2] Ph.D. Scholar, HOD & Associate Professor, Department of Agadtantra evam Vidhivaidyak, Government Ayurved College, Dharashiv, Maharashtra, India - 413501.

Email: drkopardesheetal@gmail.com

[3] BAMS, MD (Agadtantra evam Vidhivaidyak), MUHS, Nashik, Maharashtra, India - 422004.

Email: sdhoot1008@gmail.com

Abstract

Poisoning changes over time. A patient may first have local or gastrointestinal symptoms and later develop neurological, cardiovascular, respiratory, hepatic or renal injury. Modern toxicology explains this progression through toxicokinetics, which describes absorption, distribution, metabolism and excretion, and toxicodynamics, which describes the biological effects produced at target tissues. Ayurvedic toxicology, or *Agada Tantra*, describes the evolving manifestations of poison through concepts such as *Visha Guna*, *Visha Gati* and *Visha Vega*. This narrative review compares these frameworks without treating them as identical. Classical descriptions from the major Ayurvedic texts were considered alongside current toxicology literature on disposition, overdose, target-organ injury and elimination. The comparison suggests that *Visha Vega* is best understood as a qualitative, stage-based clinical account of changing poisoning, whereas toxicokinetics is a quantitative account of how a toxicant moves through the body. Toxicodynamics provides an important bridge because many manifestations used to recognize a *Vega* reflect tissue effects rather than movement of the toxicant alone. Directly equating individual *Vega* with separate ADME steps is not scientifically justified. Future research should use prospective, time-linked observations that record classical symptom clusters together with toxicant concentrations, metabolites, physiological measurements and organ-specific biomarkers.

Keywords: *Visha Vega*; *Agada Tantra*; toxicokinetics; toxicodynamics; poisoning

Introduction

A poisoned patient does not usually develop every manifestation at the same moment. The clinical picture changes as the toxicant enters the body,

reaches tissues, undergoes metabolism and is removed. The timing and severity of these changes depend on the substance, dose, route, formulation, patient and treatment. Recognizing progression is therefore central to clinical toxicology.

Ayurvedic toxicology addresses the same practical problem through a different system of observation. The *Charaka Samhita*, *Sushruta Samhita*, *Ashtanga Hridaya* and *Ashtanga Sangraha* describe poisons, their qualities, their movement and changing clinical manifestations[1-4]. Explanations of *Visha Guna* in later *Dravyaguna* and commentarial literature help clarify why classical authors viewed poison as rapid, spreading and capable of affecting successive tissues[5-7].

The concept of *Visha Vega* is especially relevant. *Vega* may be understood as a stage or wave in the clinical progression of poisoning. Recent Ayurvedic reviews have examined stage-wise manifestations, stage-related treatment and comparison with modern toxidromes[8-10]. These works are useful, but similarity in clinical questions should not be mistaken for biological equivalence. A *Vega* is not a pharmacokinetic compartment, and it cannot be assigned automatically to absorption, distribution, metabolism or excretion.

Modern toxicology separates two related ideas. Toxicokinetics describes what the body does to a toxicant through absorption, distribution, metabolism and excretion. Toxicodynamics describes what the toxicant does to the body through receptor interaction, enzyme inhibition, membrane damage, oxidative stress and other mechanisms. Standard toxicology and clinical-poisoning texts use both to explain why symptoms change over time[11-17]. This review compares the classical and modern frameworks while preserving their conceptual boundaries.

Literature Search Strategy

A narrative comparative approach was used. Classical information was drawn from the *Charaka Samhita*, *Sushruta Samhita*, *Ashtanga Hridaya*, *Ashtanga Sangraha* and relevant commentarial literature. Modern sources were searched in PubMed and Google Scholar using combinations of "Visha Vega," "Agada Tantra," "toxicokinetics," "toxicodynamics," "poisoning," "ADME," "overdose pharmacokinetics," "toxicant metabolism" and "enhanced elimination." Textbooks, reviews and clinically relevant studies were included when they explained time-dependent poison progression, disposition, target-organ effects or treatment-related changes. The comparison was conceptual; classical stages were not assigned to molecular mechanisms unless supporting evidence existed.

The Classical Framework of Poison Progression

Visha Guna and *Visha Gati*

In Ayurveda, the behaviour of a poison is linked to its inherent qualities or *Visha Guna*. Terms such as *Ashukari* (rapid action), *Vyavayi* (quick spread before complete digestion) and *Sukshma* (capacity to enter subtle pathways) express a clinical impression of speed, dissemination and penetration. They are qualitative descriptions, not measured pharmacokinetic variables. Their importance lies in reminding the clinician that some poisons can progress quickly and may not remain confined to the site of exposure.

Visha Gati describes the course or movement of poison. Classical accounts consider the source of the poison, route of entry, dose, patient strength, *Dosha* involvement and the tissues or *Dhatu* affected. *Sthavara Visha* and *Jangama Visha* may show different sequences and manifestations. A single universal timetable for all poisons would therefore be inconsistent with both classical observation and modern toxicology.

Visha Vega as a clinical stage

A *Vega* is recognized through a cluster of manifestations rather than through a measured blood concentration. Early stages may show local, digestive or mild systemic features; later stages may include more severe neurological, cardiovascular, respiratory or multisystem involvement. Different texts and categories of poisoning do not always describe the same number or sequence of stages. The safest

interpretation is therefore clinical: *Visha Vega* organizes changing signs and severity over time.

Table 1: Careful comparison of classical observations with modern toxicology

Classical observation	Modern concept useful for comparison	Important limitation
Entry of *Visha*	Exposure and absorption	Classical descriptions do not specify a molecular absorption process
Ashukari	Rapid onset	Onset depends on both kinetics and dynamics
Vyavayi	Systemic dissemination	It is not the measured volume of distribution
Sukshma	Tissue or barrier penetration	The classical concept remains qualitative
Progressive *Vega*	Time-dependent evolution of poisoning	Stages are not ADME compartments
Organ-system manifestations	Target-organ toxicity	Effects may arise from the parent toxicant or metabolites
Advanced *Vega*	Progressive toxicodynamic injury	No fixed concentration can define every stage

Modern Toxicokinetics

Toxicokinetics links an external exposure with the internal dose present in blood and tissues. It uses concentration-time data and parameters such as bioavailability, volume of distribution, clearance and half-life. At therapeutic or low experimental doses, a substance may follow relatively predictable behaviour. During poisoning, however, absorption, protein binding, metabolism or elimination can become saturated. Toxicokinetic values derived from ordinary dosing may then be misleading[18,19]. General pharmacokinetic texts provide the mathematical framework for interpreting these changes[20,21].

Absorption

Absorption is movement from the site of exposure into the systemic circulation. Oral absorption depends on dissolution, gastric emptying, intestinal motility, formulation and first-pass metabolism. Inhaled substances may enter quickly through the lungs, while dermal absorption depends on skin integrity, area and chemical properties. In overdose, delayed gastric emptying, sustained-release formulations, concretions or impaired circulation can prolong absorption. The first symptoms may therefore appear before peak concentration, or toxicity may worsen after an apparently stable interval.

Distribution

After entering the circulation, a toxicant distributes according to blood flow, protein binding, lipid solubility, ionization and tissue affinity. Highly perfused organs may be exposed early. Lipophilic substances may later move into fat or cross the blood-brain barrier. In severe poisoning, acidosis, low albumin, organ failure and altered perfusion can change the free concentration and tissue distribution. These measured processes are useful when thinking about systemic spread, but they should not be labelled as *Vyavayi* or *Sukshma* without evidence.

Metabolism

Metabolism can reduce toxicity, but it can also create a more reactive or harmful product. Cytochrome P450 enzymes and other pathways vary with genetics, age, disease, co-exposures and enzyme induction or inhibition[22-24]. Acetaminophen is a familiar example: its disposition and formation of a reactive metabolite help explain delayed liver injury and the time-sensitive value of acetylcysteine[25-27]. This example shows why clinical deterioration may continue even when the parent compound is falling.

Excretion

The kidneys, bile, lungs and other routes remove toxicants and metabolites. Renal clearance depends on filtration, secretion, reabsorption, urine pH and kidney function. A long half-life can reflect a large distribution volume, slow metabolism, renal failure or continuing absorption. Table 2 summarizes how each ADME process can shape the clinical course.

Table 2: Toxicokinetic processes and their clinical implications

Process	What it describes	Why symptoms may change over time
Absorption	Entry from the exposure site into circulation	Delayed or prolonged absorption can postpone peak toxicity
Distribution	Movement between blood and tissues	Early organ exposure and later redistribution can change the clinical picture
Metabolism	Chemical conversion by enzymes and other pathways	Detoxification or bioactivation may alter the type and timing of injury
Excretion	Removal of parent toxicant and metabolites	Reduced clearance can prolong or intensify toxicity

Toxicodynamics and the Clinical Picture

Toxicokinetics does not by itself explain every symptom. Toxicodynamics describes the relationship between the concentration at a target and the resulting biological effect. A toxicant may block receptors, inhibit enzymes, disrupt ion channels, damage mitochondria or trigger inflammation. Effects may appear quickly, accumulate gradually or persist after the blood concentration has fallen.

This distinction is essential when comparing modern toxicology with *Visha Vega*. Most *Vega* are recognized from manifestations: altered consciousness, vomiting, breathing difficulty, cardiovascular instability or other signs. Such findings are usually consequences of toxicodynamic injury. They are influenced by toxicokinetics, but they are not simply measurements of chemical movement. Toxidrome-based assessment likewise uses symptom clusters to guide urgent recognition when the exact agent is not yet known[28-30].

Table 3 separates the three levels that are commonly mixed in comparative writing. Keeping them distinct makes the Ayurveda-modern comparison more precise.

Table 3: Distinguishing Visha Vega, toxicokinetics and toxicodynamics

Framework	Main question	Typical evidence
Visha Vega	How is poisoning presenting and progressing clinically?	Stage-related symptom clusters and classical examination
Toxicokinetics	What happens to the toxicant over time?	Serial concentrations, metabolites, clearance and half-life
Toxicodynamics	What biological effect is produced at the target?	Physiology, biomarkers, organ injury and concentration-effect data

Clinical Relevance of Time and Stage Recognition

The most useful common ground between the two systems is the importance of time. Early recognition may allow removal of ongoing exposure, appropriate decontamination, antidote administration or supportive care before severe injury develops. Later management may focus on organ support, correction of physiological disturbance and selected methods of enhanced elimination. These interventions are substance- and patient-specific. Activated charcoal, urine alkalinization and extracorporeal treatment each have defined indications, limits and risks[31-35]. They should not be presented as universal measures.

For an integrative academic discussion, classical stage recognition may be studied alongside contemporary monitoring, but it must not delay evidence-based emergency care. A person with suspected poisoning requires prompt assessment and treatment through an appropriate poison service or emergency facility. The present comparison is a research and educational framework, not a treatment protocol.

Sources of Variation

The same dose does not produce the same course in every person. Age, body composition, pregnancy, genetics, nutrition, liver or kidney disease, enzyme activity, tolerance and co-exposure can alter toxicokinetics or toxicodynamics. Route and formulation also matter. Environmental toxicology and risk-assessment literature therefore treats internal dose as a link between exposure and effect rather than as a fixed consequence of external dose[36-38].

Ayurvedic texts similarly consider the strength of the poison, patient strength, route, season and other contextual factors. This is a meaningful similarity in

clinical reasoning, but the variables are defined and measured differently. Comparative research should preserve those differences instead of forcing a shared vocabulary.

Research Gaps and Future Directions

Direct empirical study of *Visha Vega* remains limited. Current literature is largely textual or conceptual. There are no widely accepted operational definitions, validated assessment tools or prospective datasets that link individual *Vega* with measured toxicant concentrations. Without these elements, claimed equivalence with modern toxicokinetics remains speculative.

A useful study should focus on one well-defined poisoning rather than combine unrelated toxicants. At predetermined times, investigators could record a structured classical assessment, contemporary clinical observations, blood concentration where measurable, metabolites, physiological measurements and organ-specific biomarkers. The minimum reporting framework is outlined in Table 4.

Table 4: Proposed framework for time-linked research on Visha Vega

Domain	Information to collect	Purpose
Exposure	Toxicant, dose estimate, route, formulation and time	Defines the starting event
Classical assessment	Predefined *Visha Lakshana* and proposed *Vega*	Makes the Ayurvedic assessment reproducible
Clinical status	Vital signs, ECG, neurological and respiratory findings	Documents changing severity
Toxicokinetics	Serial parent-toxicant and metabolite concentrations	Measures disposition over time
Toxicodynamics	Acid-base status and organ-specific biomarkers	Separates tissue effect from chemical movement
Interventions	Timing and type of supportive care, antidote or elimination method	Accounts for treatment-related change
Outcome	Recovery, organ injury, intensive care and mortality	Links stages with meaningful clinical endpoints

Such work would allow correlations to emerge from data rather than be imposed theoretically. It would also show whether stage boundaries are reproducible across observers and whether they add information beyond established clinical toxicology measures.

Discussion

The classical idea of *Visha Vega* shows that Ayurvedic toxicology viewed poisoning as an evolving process. Its attention to speed, spread, changing manifestations and stage-related severity remains clinically interesting. Modern toxicokinetics offers a more quantitative explanation through ADME and concentration-time relationships, while toxicodynamics explains how those exposures produce organ dysfunction.

Several cautious comparisons are possible. *Ashukari* draws attention to rapid action, *Vyavayi* to dissemination, *Sukshma* to penetration, and sequential *Vega* to changing manifestations. However, *Vyavayi* is not volume of distribution, *Sukshma* is not membrane permeability, and a *Vega* is not an ADME compartment. Direct translation would simplify both systems.

A more defensible interpretation is that *Visha Vega* is a qualitative clinical model of progression. Toxicokinetics is a quantitative model of disposition. Toxicodynamics provides a bridge because the manifestations used to recognize a stage are usually effects at tissues and organs. This framing respects classical observation while keeping scientific claims testable.

Conclusions

Visha Vega is an important classical framework for describing the progressive nature of poisoning. Modern toxicokinetics explains how a toxicant is absorbed, distributed, metabolized and excreted, while toxicodynamics explains the resulting biological injury. The systems address related clinical questions but use different kinds of evidence.

The most useful comparison concerns timing, systemic spread, changing organ involvement and severity. A direct one-to-one correlation between individual *Vega* and ADME is not scientifically justified. Future prospective research should document classical stages alongside serial toxicant concentrations, metabolites, physiological

measurements and organ-specific biomarkers. Until such data are available, *Visha Vega* should be presented as a classical clinical model that can generate research questions, not as an ancient version of pharmacokinetics.

Declarations

Ethics approval and consent to participate

Not applicable. This narrative review did not involve human participants, human data, human tissue or animals.

Consent for publication

Not applicable.

Availability of data and materials

No new datasets were generated or analyzed. All information discussed in this narrative review is available in the cited sources.

Competing interests

The author(s) declare that they have no competing interests.

Funding

No specific funding was received for this study.

Acknowledgments

Not applicable.

References

[1] Agnivesha. Charaka Samhita. Revised by Charaka and Dridhabala, with Ayurveda Dipika commentary of Chakrapanidatta. Acharya YT, editor. Varanasi: Chaukhambha Surbharati Prakashan; 2016. Chikitsa Sthana, chapter 23.

[2] Sushruta. Sushruta Samhita, with Nibandhasangraha commentary of Dalhana. Acharya YT, Acharya NR, editors. Varanasi: Chaukhambha Sanskrit Sansthan; 2017. Kalpa Sthana.

[3] Vagbhata. Ashtanga Hridaya, with Sarvangasundara commentary of Arunadatta and Ayurveda Rasayana commentary of Hemadri. Paradakara HS, editor. Varanasi: Chaukhambha Surbharati Prakashan; 2018. Uttara Sthana, Visha Pratishedha.

[4] Vagbhata. Ashtanga Sangraha. Srikantha Murthy KR, translator. Varanasi: Chaukhambha Orientalia; 2016. Uttara Sthana, Visha Pratishedha.

[5] Sharma PV. Dravyaguna Vijnana. Vol. 1. Varanasi: Chaukhambha Bharati Academy; 2013.

[6] Shastri K, Chaturvedi G. Charaka Samhita of Agnivesha. Part II. Varanasi: Chaukhambha Bharati Academy; 2015. Chikitsa Sthana, chapter 23.

[7] Shastri AD. Sushruta Samhita. Part I. Varanasi: Chaukhambha Sanskrit Sansthan; 2016. Kalpa Sthana.

[8] Gusain T, Rachna, Tomar BS, Chawla SK. A conceptual study of Charakokta Visha Vega and Chikitsa. Ayushdhara. 2024;11(3):153-161. doi:10.47070/ayushdhara.v11i3.1577.

[9] Dudhe PA, Ukey JS. Chaturvinshati Upkrama and its role in management of poisoning. J Ayurveda Integr Med Sci. 2023;8(5):147-151. doi:10.21760/jaims.8.5.23.

[10] Kushre H, Inchulkar SR. Toxidrome through the lens of Agad Tantra: an Ayurvedic interpretation of toxic syndromes: a conceptual review. Int J Ayurveda Pharma Res. 2026;14(7):117-122. doi:10.47070/ijapr.v14i7.4255.

[11] Klaassen CD, editor. Casarett & Doull's Toxicology: The Basic Science of Poisons. 9th ed. New York: McGraw-Hill Education; 2019.

[12] Krishnan K. Toxicokinetics. In: Klaassen CD, editor. Casarett & Doull's Toxicology: The Basic Science of Poisons. 9th ed. New York: McGraw-Hill Education; 2019.

[13] Gupta PK. Principles and basic concepts of toxicokinetics. In: Gupta PK, editor. Fundamentals of Toxicology: Essential Concepts and Applications. London: Academic Press; 2016.

[14] Hodgson E. A Textbook of Modern Toxicology. 4th ed. Hoboken: John Wiley & Sons; 2010.

[15] Timbrell JA. Principles of Biochemical Toxicology. 4th ed. New York: Informa Healthcare; 2009.

[16] Olson KR, Anderson IB, Benowitz NL, Blanc PD, Clark RF, Kearney TE, et al. Poisoning & Drug Overdose. 7th ed. New York: McGraw-Hill Education; 2018.

[17] Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS, editors. Goldfrank's Toxicologic Emergencies. 11th ed. New York: McGraw-Hill Education; 2019.

[18] Sue YJ, Shannon M. Pharmacokinetics of drugs in overdose. Clin Pharmacokinet. 1992;23(2):93-105. doi:10.2165/00003088-199223020-00003.

[19] Roberts DM, Buckley NA. Pharmacokinetic considerations in clinical toxicology: clinical applications. Clin Pharmacokinet. 2007;46(11):897-939. doi:10.2165/00003088-200746110-00001.

[20] Rowland M, Tozer TN. Clinical Pharmacokinetics and Pharmacodynamics: Concepts and Applications. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2011.

[21] Shargel L, Yu ABC. Applied Biopharmaceutics & Pharmacokinetics. 7th ed. New York: McGraw-Hill Education; 2016.

[22] Zanger UM, Schwab M. Cytochrome P450 enzymes in drug metabolism: regulation of gene expression, enzyme activities, and impact of genetic variation. Pharmacol Ther. 2013;138(1):103-141. doi:10.1016/j.pharmthera.2012.12.007.

[23] Guengerich FP. Cytochrome P450 and chemical toxicology. Chem Res Toxicol. 2008;21(1):70-83. doi:10.1021/tx700079z.

[24] Almazroo OA, Miah MK, Venkataramanan R. Drug metabolism in the liver. Clin Liver Dis. 2017;21(1):1-20. doi:10.1016/j.cld.2016.08.001.

[25] McGill MR, Jaeschke H. Metabolism and disposition of acetaminophen: recent advances in relation to hepatotoxicity and diagnosis. Pharm Res. 2013;30(9):2174-2187.

[26] Prescott LF. Paracetamol overdosage: pharmacological considerations and clinical management. Drugs. 1983;25(3):290-314.

[27] Heard KJ. Acetylcysteine for acetaminophen poisoning. N Engl J Med. 2008;359(3):285-292. doi:10.1056/NEJMct0708278.

[28] Holstege CP, Borek HA. Toxidromes. Crit Care Clin. 2012;28(4):479-498.

[29] Mokhlesi B, Leiken JB, Murray P, Corbridge TC. Adult toxicology in critical care: part I: general approach to the intoxicated patient. Chest. 2003;123(2):577-592.

[30] Megarbane B. Toxidrome-based approach to common poisonings. Asia Pac J Med Toxicol. 2014;3(1):2-12.

[31] Juurlink DN. Activated charcoal for acute overdose: a reappraisal. Br J Clin Pharmacol. 2016;81(3):482-487.

[32] Chyka PA, Seger D, Krenzelok EP, Vale JA. Position paper: single-dose activated charcoal. Clin Toxicol (Phila). 2005;43(2):61-87.

[33] Proudfoot AT, Krenzelok EP, Vale JA. Position paper on urine alkalinization. J Toxicol Clin Toxicol. 2004;42(1):1-26.

[34] Roberts DM, Buckley NA. Enhanced elimination in acute barbiturate poisoning: a systematic review. Clin Toxicol (Phila).

2011;49(1):2-12. doi:10.3109/15563650.2010.550582.

[35] Ghannoum M, Roberts DM, Hoffman RS, Ouellet G, Roy L, Decker BS, et al. A stepwise approach for the management of poisoning with extracorporeal treatments. Semin Dial. 2014;27(4):362-370. doi:10.1111/sdi.12228.

[36] Flack S, Nylander-French LA. Toxicology and human environments. Prog Mol Biol Transl Sci. 2012;112:21-38.

[37] Robinson L. Toxicokinetics. In: A Practical Guide to Toxicology and Human Health Risk Assessment. Chichester: John Wiley & Sons; 2018. doi:10.1002/9781118881880.ch4.

[38] US Environmental Protection Agency. Toxicokinetics overview [Internet]. Washington, DC: US EPA; 2025 [cited 2026 Aug 14]. Available from: https://www.epa.gov/chemical-research/toxicokinetics-overview

.