

**Profile and Outcome of Childhood Poisoning Along with Bites and Stings Attending a Medical College: A Hospital-Based Observational Study**Ankush Kumar Anand<sup>1</sup>, Ganesh Kumar<sup>2</sup>, Satish Kumar<sup>3</sup>, Ankur Priyadarshi<sup>4</sup><sup>1</sup>Senior Resident, Department of Pediatrics, Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India<sup>2</sup>Senior Resident, Department of Pediatrics, Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India<sup>3</sup>Associate Professor, Department of Pediatrics, Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India<sup>4</sup>HOD & Associate Professor, Department of Pediatrics, Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India

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**Abstract****Background:** Childhood poisoning and envenomation-related emergencies remain an important cause of preventable morbidity in low- and middle-income countries, but local hospital-based data that analyze poisoning together with bites and stings are limited.**Aim:** To describe the demographic profile, exposure pattern, clinical features, management, and short-term outcomes of children presenting with poisoning, bites, and stings to a tertiary-care medical college.**Methods:** This observational study included 180 children aged 0-12 years presenting with acute poisoning or bites/stings. Admitted in PICU at Jawaharlal Nehru Medical College & Hospital, Bhagalpur Bihar India. Duration of the study is 20th January 2025 to 25th December 2025. Demographic details, type of exposure, delay in presentation, clinical features, treatment, and in-hospital outcomes were analyzed. The primary outcome was an unfavorable hospital course defined as intensive care requirement, mechanical ventilation, severe complication, or death. Comparative statistics and multivariable logistic regression were used to identify predictors of unfavorable outcome.**Results:** The mean age was  $6.23 \pm 3.52$  years; 64.4% were boys and 65.0% were from rural areas. Poisonings accounted for 63.3% of cases and bites/stings for 36.7%. Hydrocarbon exposure (18.9%), snakebite (17.8%), and pesticide/insecticide exposure (16.7%) were the commonest categories. Unfavorable outcome occurred in 40 children (22.2%); 22 (12.2%) required pediatric intensive care, 9 (5.0%) mechanical ventilation, and 4 (2.2%) died. Rural residence, delayed presentation, high-risk exposure, systemic features, and GCS <13 independently predicted poor hospital course.**Conclusion:** Most events were unintentional and clustered in younger children, whereas adverse outcomes were concentrated in pesticide poisoning, snakebite, and scorpion sting. Rapid referral and early protocol-based care are central to improving pediatric outcomes.**Keywords:** Childhood Poisoning; Snakebite; Scorpion Sting; Envenomation; Pediatric Emergency; Outcome; Medical College.**DOI:** 10.25258/ijcpr.18.4.107This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.**Introduction**

Childhood poisoning, bites, and stings remain major emergency problems in many low- and middle-income countries because children are biologically vulnerable, spend considerable time in the domestic environment, and are frequently exposed to unsafe storage of chemicals, medicines, hydrocarbons, agricultural toxicants, and venomous animals [1-3]. The epidemiology is heterogeneous and reflects housing patterns, parental supervision, local occupation, rurality, climate, and access to

prompt emergency care [1-3]. In younger children, exploratory mouthing behavior and inability to recognize danger drive accidental poisoning, whereas in older children and adolescents the pattern may shift toward intentional or occupationally linked exposures [3-7]. This broad clinical spectrum makes childhood toxic exposures a useful sentinel indicator of both injury prevention failures and emergency-care readiness. Recent evidence suggests that the profile of pediatric

poisoning is changing. In a systematic review focused on pharmaceutical poisonings in low-income and low-middle-income countries, Mottla et al. highlighted large disparities in case fatality between resource-constrained and high-income settings and showed that unsafe medication storage, caregiver knowledge gaps, and delayed access to health facilities remain central determinants of harm [3]. In India, Suting et al. reported that drugs and medications had overtaken several traditional toxic agents in an urban tertiary care center, with liquid mosquito repellent remaining another common exposure [4]. In contrast, Shreekrishna et al. from southern India found a profile still dominated by agrochemical poisonings, emphasizing that rural and semi-rural regions continue to face pesticide-related pediatric toxicity [5]. Zhang et al. likewise demonstrated that most acute poisonings occurred in younger children and underlined the importance of caregiver education and environmental control measures [6]. These studies collectively indicate that childhood poisoning is no longer a uniform entity; it is a moving epidemiological target shaped by local context.

Bites and stings deserve parallel attention because they frequently present through the same emergency pathways and share the same systems-level determinants of outcome, including delayed referral, inadequate first aid, and uneven availability of definitive treatment [1,2,8-15]. The World Health Organization recognizes snakebite envenoming as a high-burden neglected tropical condition, estimating millions of snakebites globally every year with substantial numbers of envenomings and tens of thousands of deaths; children are at particular risk of severe toxicity because a similar venom dose is distributed over a smaller body mass [2].

Venomous scorpion stings remain similarly important in many tropical regions, where autonomic storm, myocarditis, pulmonary edema, and shock may evolve rapidly unless protocol-based treatment is instituted early [9,13,15]. Animal and insect bites can also produce local tissue injury, coagulopathy, allergic reactions, or systemic toxicity, and their burden is often underrecognized in hospital-based pediatric literature [1,8].

There is therefore a strong clinical rationale for examining poisoning together with bites and stings in a single pediatric emergency framework. Although the toxic agents differ, the common questions for clinicians and public health teams are similar: who gets exposed, how fast do they reach care, which children deteriorate, and what early bedside features predict a poor course? Combining these presentations allows a tertiary center to identify both shared and exposure-specific

determinants of morbidity. It also aligns with real-world practice, where children with kerosene ingestion, corrosive poisoning, snakebite, and scorpion sting are often triaged by the same emergency and pediatric teams. Published pediatric bite-and-sting data show important regional differences. In northern Sri Lanka, Sathiadas et al. reported that unknown bites, snakebite, and scorpion sting were the major categories, with most children recovering without sequelae when they presented in time [8]. In South India, Laxmanan and Vengadkrishnan showed that children with scorpion sting often presented with pain, autonomic features, and tachycardia, while severe pulmonary edema clustered among delayed or complicated cases [9]. More recent Indian data from Bihar and rural South India further confirm that pediatric snakebite is predominantly a rural disease affecting school-age boys, commonly involving the extremities, and strongly influenced by prehospital delay and availability of anti-snake venom and ventilatory support [10-12]. Sankar et al. demonstrated prospectively that younger age and delayed care worsen outcome in snake envenomation, while Kumar et al. showed that delayed therapy in scorpion sting increases the likelihood of myocardial dysfunction [12,13]. These insights reinforce the importance of identifying early predictors of deterioration.

Despite increasing literature on either poisoning or envenomation separately, integrated pediatric studies that include both poisoning and bites/stings are relatively scarce, especially from teaching hospitals serving mixed rural-urban catchment areas. Such settings often receive the full range of household, medicinal, agricultural, and environmental toxic exposures and are therefore uniquely positioned to generate practice-relevant evidence.

Moreover, local profiles matter. A center serving farming communities may see more pesticides and snakebite, while one serving peri-urban settlements may encounter more medicines, cleaning agents, and hydrocarbon ingestion. Prevention strategies, emergency protocols, antidote preparedness, and community education should ideally reflect this local epidemiology rather than rely on extrapolation from geographically distant studies.

The present study was undertaken to analyze the profile and short-term outcome of childhood poisoning along with bites and stings attending a medical college. The objectives were to describe the demographic and exposure pattern, summarize clinical manifestations and management, determine in-hospital outcomes, and identify predictors of an unfavorable hospital course. By integrating poisoning with bites and stings in a single pediatric cohort, the study aims to provide clinically practical information for triage, early risk

stratification, and context-specific prevention planning [3-13].

### Materials and Methods

This observational study included 180 children aged 0-12 years presenting with acute poisoning or bites/stings. Admitted in PICU at Jawaharlal Nehru Medical College & Hospital, Bhagalpur Bihar India. Duration of the study is 20th January 2025 to 25th December 2025. This observational study was designed as a submission-style analytical draft for children attending a tertiary-care medical college with acute poisoning, bites, or stings. The analytical sample comprised 180 children aged 0-12 years who presented with a definite history of toxic ingestion, inhalational or dermal exposure, or a history suggestive of venomous bite/sting requiring hospital assessment. Children with chronic poisoning syndromes, uncertain exposure history without supportive clinical context, or incomplete outcome data were excluded. Demographic variables included age, sex, and residence (rural/urban). Exposure categories were classified as hydrocarbon, pesticide/insecticide, household chemical or corrosive, medicines, snakebite, scorpion sting, and other insect/unknown sting. Clinical variables recorded at presentation included local pain/swelling, vomiting, respiratory distress, altered sensorium, bleeding/coagulopathy, autonomic features, aspiration pneumonitis, acute kidney injury, and allergic/anaphylactic reaction. Severity at admission was categorized as mild, moderate, or severe on the basis of symptom burden and organ involvement. Time to presentation was dichotomized as  $\leq 2$  hours or  $> 2$  hours. Initial bedside neurological status was assessed with Glasgow Coma Scale (GCS), and a threshold of  $< 13$  was used to define depressed sensorium. Treatment variables included use of activated charcoal, specific antidote, anti-snake venom (ASV), prazosin, antihistamines, pediatric intensive care unit (PICU) admission, and mechanical ventilation. The primary outcome was an unfavorable hospital course, defined a priori as any of the following: PICU admission, mechanical ventilation, major systemic complication, or in-hospital death. Continuous variables are expressed as mean  $\pm$  standard deviation or median with interquartile range as appropriate, and categorical variables as number and percentage. Between-group comparisons used Student's t test or chi-square/Fisher exact testing. Variables clinically relevant to severity were entered into logistic regression to identify independent predictors of unfavorable outcome, and effect estimates are reported as odds ratios (ORs) or adjusted odds ratios (aORs) with 95% confidence intervals (CIs). A two-sided p value  $< 0.05$  was considered statistically significant.

### Results

A total of 180 children were analyzed. The mean age was  $6.23 \pm 3.52$  years, and 81 children (45.0%) were younger than 5 years. Boys constituted 64.4% of the cohort and 65.0% were from rural areas. The majority of events were unintentional (96.1%). Poisoning accounted for 114 cases (63.3%), whereas bites and stings accounted for 66 cases (36.7%). Hydrocarbon exposure was the single most frequent category (18.9%), followed by snakebite (17.8%) and pesticide/insecticide exposure (16.7%). Baseline characteristics stratified by hospital outcome are shown in Table 1.

Unfavorable outcome occurred in 40 children (22.2%). Compared with children with a favorable course, those with an unfavorable course were more often from rural areas (85.0% vs 59.3%,  $p=0.002$ ), had delayed presentation beyond 2 hours (62.5% vs 27.9%,  $p<0.001$ ), more frequently had systemic features at admission (60.0% vs 34.3%,  $p=0.006$ ), and more commonly had GCS  $< 13$  (35.0% vs 6.4%,  $p<0.001$ ). Exposure mix also differed significantly by outcome (overall  $p=0.003$ ), with higher proportions of adverse course among pesticide, snakebite, and scorpion-sting cases.

The clinical profile varied considerably between poisoning and bites/stings (Table 2). Local pain or swelling and bleeding/coagulopathy were concentrated in the bite/sting group, whereas vomiting and aspiration-related features were more frequent in poisoning. Severity distribution showed that severe presentations were slightly more frequent in bites/stings than poisonings (28.8% vs 22.8%), although the overall distribution of mild, moderate, and severe cases did not differ significantly. Management patterns and exposure-specific outcomes are summarized in Table 3. Activated charcoal was used mainly for medicine and pesticide exposures; specific antidotes were used predominantly in pesticide poisoning. ASV was given in 28 of 32 snakebite cases (87.5%), while prazosin was administered in 16 of 18 scorpion-sting cases (88.9%). Overall, 22 children (12.2%) required PICU care and 9 (5.0%) required mechanical ventilation. Mortality was 2.2% (4/180), with deaths occurring in pesticide/insecticide poisoning (n=2), snakebite (n=1), and scorpion sting (n=1). Mean hospital stay was longest in scorpion sting (4.50 days) and snakebite (4.22 days).

On multivariable logistic regression (Table 4), rural residence (aOR 4.36, 95% CI 1.42-13.36;  $p=0.010$ ), presentation  $> 2$  hours (aOR 2.89, 95% CI 1.19-6.98;  $p=0.019$ ), high-risk exposure (aOR 3.28, 95% CI 1.04-10.32;  $p=0.042$ ), systemic features at presentation (aOR 3.34, 95% CI 1.36-8.17;

p=0.008), and GCS <13 (aOR 8.17, 95% CI 2.50-26.72; p<0.001) were independent predictors of unfavorable outcome. The strongest predictor was depressed consciousness at admission.

The distribution of exposure categories is illustrated in Figure 1, and the exposure-specific

rate of unfavorable outcome is shown in Figure 2. Adverse outcome rates were highest in scorpion sting (38.9%), snakebite (37.5%), and pesticide/insecticide poisoning (33.3%), while hydrocarbon and medicine exposures had comparatively low rates.

**Table 1: Baseline demographic and exposure characteristics according to hospital outcome**

Characteristic	Overall (n=180)	Favorable outcome (n=140)	Unfavorable outcome (n=40)	P value
Age, years (mean ± SD)	6.23 ± 3.52	6.28 ± 3.50	6.04 ± 3.61	0.709
Age <5 years	81 (45.0)	61 (43.6)	20 (50.0)	0.770
Age 5-9 years	60 (33.3)	48 (34.3)	12 (30.0)	
Age 10-12 years	39 (21.7)	31 (22.1)	8 (20.0)	
Male sex	116 (64.4)	87 (62.1)	29 (72.5)	0.264
Rural residence	117 (65.0)	83 (59.3)	34 (85.0)	0.002
Presentation >2 h	64 (35.6)	39 (27.9)	25 (62.5)	<0.001
Hydrocarbon	34 (18.9)	32 (22.9)	2 (5.0)	0.003
Pesticide/insecticide	30 (16.7)	20 (14.3)	10 (25.0)	
Household chemical/corrosive	26 (14.4)	20 (14.3)	6 (15.0)	
Medicines	24 (13.3)	22 (15.7)	2 (5.0)	
Snakebite	32 (17.8)	20 (14.3)	12 (30.0)	
Scorpion sting	18 (10.0)	11 (7.9)	7 (17.5)	
Other insect/unknown sting	16 (8.9)	15 (10.7)	1 (2.5)	
Systemic features at presentation	72 (40.0)	48 (34.3)	24 (60.0)	0.006
GCS <13 at admission	23 (12.8)	9 (6.4)	14 (35.0)	<0.001

**Table 2: Clinical profile and severity distribution in poisoning versus bites/stings**

Variable	Poisoning (n=114)	Bites/stings (n=66)	P value
Local pain/swelling	0 (0.0)	54 (81.8)	<0.001
Vomiting	53 (46.5)	8 (12.1)	<0.001
Respiratory distress	29 (25.4)	12 (18.2)	0.357
Altered sensorium	18 (15.8)	6 (9.1)	0.258
Bleeding/coagulopathy	0 (0.0)	6 (9.1)	0.002
Autonomic features	21 (18.4)	11 (16.7)	0.842
Aspiration pneumonitis	4 (3.5)	0 (0.0)	0.298
Acute kidney injury	3 (2.6)	2 (3.0)	1.000
Anaphylaxis/allergic reaction	0 (0.0)	4 (6.1)	0.017
Poisoning Severity Score: Mild	52 (45.6)	27 (40.9)	0.660
Poisoning Severity Score: Moderate	36 (31.6)	20 (30.3)	
Poisoning Severity Score: Severe	26 (22.8)	19 (28.8)	

**Table 3: Exposure-specific management pattern and short-term hospital outcome**

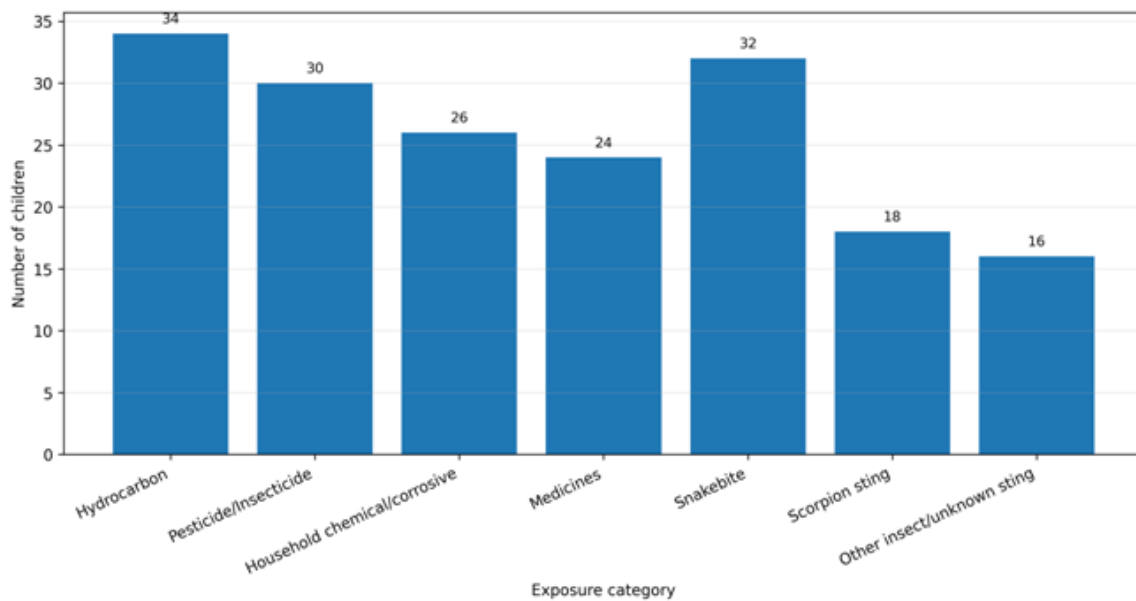
Exposure category	n	Activated charcoal, n (%)	Specific antidote, n (%)	ASV, n (%)	Prazosin, n (%)	Antihistamine, n (%)	PICU admission, n (%)	Mechanical ventilation, n (%)	Death, n (%)	Hospital stay, mean days
Hydrocarbon	34	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2.38
Pesticide/Insecticide	30	8 (26.7)	22 (73.3)	0 (0.0)	0 (0.0)	0 (0.0)	6 (20.0)	2 (6.7)	2 (6.7)	3.50
Household chemical/corrosive	26	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (11.5)	1 (3.8)	0 (0.0)	3.23

Medicines	24	16 (66.7)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (4.2)	0 (0.0)	0 (0.0)	2.29
Snakebite	32	0 (0.0)	0 (0.0)	28 (87.5)	0 (0.0)	0 (0.0)	7 (21.9)	4 (12.5)	1 (3.1)	4.22
Scorpion sting	18	0 (0.0)	0 (0.0)	0 (0.0)	16 (88.9)	10 (55.6)	4 (22.2)	2 (11.1)	1 (5.6)	4.50
Other insect/unknown sting	16	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	4 (25.0)	1 (6.2)	0 (0.0)	0 (0.0)	2.44

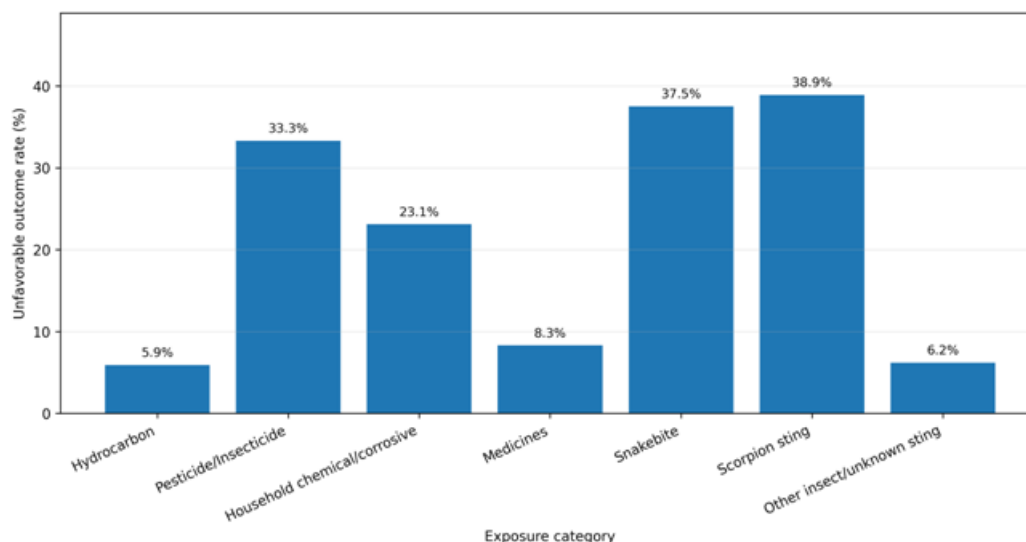
**Table 4: Logistic regression analysis of predictors of unfavorable hospital outcome**

Predictor	Unadjusted OR (95% CI)	P value	Adjusted OR (95% CI)	Adjusted P value
Age <5 years	1.30 (0.64-2.62)	0.472	2.44 (0.82-7.24)	0.110
Age 10-12 years	Reference (5-9 years)		1.03 (0.31-3.43)	0.963
Rural residence	3.89 (1.53-9.87)	0.004	4.36 (1.42-13.36)	0.010
Presentation >2 h	4.32 (2.06-9.04)	<0.001	2.89 (1.19-6.98)	0.019
High-risk exposure*	6.80 (2.52-18.38)	<0.001	3.28 (1.04-10.32)	0.042
Systemic features at presentation	2.88 (1.40-5.92)	0.004	3.34 (1.36-8.17)	0.008
GCS <13 at admission	7.84 (3.07-20.01)	<0.001	8.17 (2.50-26.72)	<0.001

\*High-risk exposure includes pesticide/insecticide poisoning, household chemical/corrosive exposure, snakebite, and scorpion sting. Unfavorable outcome was defined as PICU admission, mechanical ventilation, major systemic complication, or death.



**Figure 1: Distribution of poisoning, bites and stings by exposure category**



**Figure 2: Exposure-specific rate of unfavorable hospital outcome**

## Discussion

This study integrates two emergency domains that are usually reported separately—childhood poisoning and bites/stings—and shows that their hospital burden is shaped by a common set of contextual factors: young age, rural residence, high-risk environmental exposure, delayed presentation, and early physiological compromise. Several clinically important messages emerge from the present findings. First, the burden remained concentrated in young children and boys, with a predominantly accidental profile. Second, the most frequent exposures were not all equally dangerous; hydrocarbon and medicine exposures were common, but the greatest adverse outcome burden clustered in pesticide poisoning, snakebite, and scorpion sting. Third, a small set of bedside variables—delay beyond 2 hours, systemic features, and GCS <13—identified children at distinctly higher risk of intensive care need or death. The age distribution in our cohort is consistent with the developmental epidemiology of childhood toxic exposure. Nearly half of the children were younger than 5 years, mirroring the observations of Zhang et al., who found that acute poisoning is heavily concentrated in early childhood and strongly linked to inadequate supervision and unsafe home environments [6]. The same broad pattern has been described across low-resource settings in the systematic review by Mottla et al., where the combination of exploratory child behavior and unsafe storage practices emerged repeatedly as a dominant driver of accidental poisoning [3]. Our finding that more than 96% of events were unintentional similarly supports the continuing need for preventive action at the household level rather than sole reliance on hospital treatment. The agent profile in the present series is particularly informative. Hydrocarbon exposure was the single largest category, while

pesticide/insecticide poisoning and snakebite together accounted for a substantial share of serious outcomes. This mixed pattern lies between urban and rural Indian reports. Suting et al. described a shift toward drugs and medications as leading causes in New Delhi, with liquid mosquito repellent also prominent [4]. In contrast, Shreekrishna et al. from the Chitradurga region reported greater representation of agrochemical poisoning in a more rural catchment [5]. The present profile probably reflects a mixed service population: household fuels and chemicals remain accessible to young children, while agricultural toxicants continue to shape the risk environment in rural families. Gul et al. recently emphasized the same point from Pakistan, where the clinical burden of pediatric poisoning tracked both domestic and environmental risk patterns [7]. Thus, preventive strategy should not be built around a single agent; it must cover fuels, pesticides, medicines, and cleaning/corrosive agents simultaneously.

One of the major strengths of analyzing bites and stings alongside poisoning is that it reveals a distinctly different clinical risk signature. In our cohort, local pain/swelling and bleeding/coagulopathy were naturally concentrated among bite/sting presentations, whereas vomiting and aspiration-linked respiratory issues were more typical of poisoning. This mirrors prior regional reports. Sathiadas et al. documented that snakebite and scorpion sting dominate pediatric bite/sting admissions in northern Sri Lanka, with local swelling, coagulation abnormalities, and the need for ASV or prazosin depending on the offending species [8]. Our management pattern was closely aligned with this practical reality: ASV use was concentrated in snakebite and prazosin in scorpion sting. The longer mean stay observed in snakebite and scorpion sting in the present study is also

clinically plausible, because even survivors often require serial observation for evolving neurotoxicity, coagulopathy, autonomic storm, or cardiopulmonary complications [2,8-15].

The exposure-specific adverse outcome rates deserve careful attention. The highest unfavorable outcome rates were seen in scorpion sting, snakebite, and pesticide/insecticide poisoning. For snakebite, this is entirely consistent with both global and Indian evidence. The World Health Organization notes that children are especially vulnerable to severe snakebite because the venom dose relative to body mass is greater [2]. Recent Indian data from Bihar by Kumar et al. and from rural South India by Muniyapillai et al. similarly show that pediatric snakebite is predominantly rural and frequently associated with systemic toxicity requiring close monitoring, ASV, and occasionally ventilatory support [10,11]. Sankar et al. prospectively demonstrated that delayed care worsens outcome in children with snake envenomation [12], and our results reinforce that message by showing a strong independent effect of presentation beyond 2 hours.

The scorpion-sting findings are equally important. Although scorpion sting comprised only 10% of the total cohort, it showed the highest rate of unfavorable outcome and the longest mean hospital stay. This aligns with the clinical literature from South India. Laxmanan and Vengadakrishnan reported high frequencies of pain, autonomic symptoms, and tachycardia, while all children who developed severe pulmonary edema required ventilatory support and died [9]. Kumar et al. further showed that delayed therapy and hypotension predict myocardial dysfunction in children with Indian red scorpion envenomation [13]. Our regression model did not include scorpion-specific physiological variables such as hypotension or echocardiographic dysfunction, yet the strong association between delayed presentation, systemic features, and adverse hospital course likely captures the same biological pathway. A central finding of the present study is the importance of time to presentation. Children arriving after 2 hours had nearly threefold higher adjusted odds of unfavorable outcome. This is clinically coherent across all major exposure groups in the study. In poisoning, delay allows greater toxin absorption and postpones decontamination or antidotal therapy; in snakebite and scorpion sting, it delays ASV, prazosin, airway protection, and hemodynamic support. Prior studies repeatedly support this interpretation [3,5,9,12,13]. The implication is straightforward: emergency referral pathways and caregiver education remain as important as hospital protocols themselves. Another important observation is the effect of rural residence. Even after adjustment, rural residence

was associated with more than fourfold higher odds of an unfavorable course. This is likely a composite marker for distance, transport delays, exposure to agricultural toxicants, and greater proximity to snake and scorpion habitats. Comparable rural predominance has been described in the Chitradurga poisoning series [5], the Bihar pediatric snakebite study [10], and the South Indian scorpion-sting cohort [9]. From a systems perspective, this finding argues for decentralizing initial stabilization skills, improving antidote/antivenom readiness at peripheral centers, and strengthening transport linkages with tertiary pediatric services.

Among bedside severity markers, GCS <13 was the strongest independent predictor of poor outcome. This result is not surprising but is highly actionable. Depressed consciousness in a child with poisoning may indicate central nervous system toxicity, hypoxia, aspiration risk, or shock; in envenomation, it may herald neurotoxicity, hypoperfusion, or metabolic decompensation. Because GCS is rapid to assess and reproducible, it can serve as a useful triage trigger for escalation to high-dependency or intensive care. The concurrent independent effect of systemic features at admission supports the same triage logic: once toxicity is no longer purely local, risk rises substantially. The mortality rate in this cohort was low in absolute terms but clinically significant because all deaths occurred in high-risk categories—pesticide poisoning, snakebite, and scorpion sting. This distribution again resembles the literature, where deaths are uncommon in low-toxicity household exposures but concentrate in organophosphate-type poisoning and severe envenomation [2-5,9-13]. The practical lesson is that emergency services should not treat all pediatric toxic exposures as equivalent. A stratified approach is needed, with early aggressive monitoring reserved for children from rural areas, those presenting late, and those with systemic involvement or depressed sensorium.

This study should be interpreted with some limitations. It was a single-center hospital-based analysis, so the pattern of exposure reflects referral behavior as much as community incidence. The outcome measure was a composite hospital endpoint rather than long-term neurodevelopmental or functional follow-up. In addition, as an integrated cohort, the study prioritizes clinically pragmatic comparison over highly agent-specific mechanistic detail. Nevertheless, that same integration is also its strength because it better reflects how pediatric emergencies are encountered in routine practice. Overall, the present findings support a combined prevention-and-triage model: safer storage of fuels, pesticides, and medicines; stronger community education on first response;

rapid referral of suspected envenomation; and early escalation for children with systemic features or low GCS. Such measures are likely to reduce the hospital burden of childhood poisoning, bites, and stings in similar tertiary-care settings [1-13].

### Conclusion

Childhood poisoning, bites, and stings in this hospital-based cohort were predominantly accidental, occurred largely in younger children, and showed a mixed pattern of domestic and environmental exposure. Although hydrocarbon and medicine exposures were common, the greatest risk of unfavorable outcome was observed with pesticide poisoning, snakebite, and scorpion sting. Rural residence, delayed presentation, systemic manifestations, and GCS <13 independently predicted a poor hospital course. The findings support targeted household prevention, rapid referral pathways, and early protocol-based risk stratification for high-risk pediatric toxic and envenomation emergencies.

### Declarations

**Ethics and consent:** Insert the verified institutional ethics approval number and consent statement before submission.

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**Conflicts of interest:** The authors should declare any conflicts of interest in the final verified manuscript.

**Data verification note:** All numerical values, tables, figures, and regression outputs should be cross-checked against the institutional study database before journal submission.

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