META-ANALYSIS

Poisons causing acute kidney injury in Indo-Pak region: A meta-analysis

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Abstract

Objective: To systematically review relevant literature to assess the extent of acute kidney injury problems as a result of exposure to different poisons in subcontinental settings.

Method: The Meta-Analysis was conducted in April 2025, and comprised search on PubMed, Cochrane Library, Web of Science, Embase, PubMed Central and Google Scholar databases for relevant literature related to Pakistan and India using key words and medical subjects' headings in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analysis guidelines. After removal of duplicates and irrelevant articles, quality was assessed using Newcastle Ottawa Scale. R studio was used for analysis, while forest plot was applied using both fixed and random effect models for all-group mortality and sub-group analysis for paraphenylene diamine poisoning. Publication bias was evaluated using funnel plot asymmetry, Egger's regression test, and Begg's rank correlation test.

Results: Of the 19 studies analysed, 12(63.2%) were from India and 7(36.8%) were from Pakistan. The overall patient population was 3,769. Of the total, 10(52.6%) studies only addressed paraphenylene diamine poisoning, 1(5.3%) one each focussed on paraquat, organophosphate, copper sulphate, methyl alcohol and rodenticides, and the rest comprised multiple substances. There was wide heterogeneity and publication bias among the studies [I^2 (90%)]. A pooled mortality of 20-23%, with fixed and random-effect model estimates being 0.23 (95% confidence interval: 0.22-0.25) and 0.20 (95% confidence interval: 0.12-0.28), respectively. Heterogeneity I^2 of 90% was observed.

Conclusion: A number of cheap and easily available substances could cause poisoning and systemic harmful effects, including single- or multi-organ failure. Extent of morbidity and rate of mortality varied widely with multiple co-factors, while wide heterogeneity required more focussed and detailed publications on the subject.

Keywords: Acute kidney injury, Poisons. Paraphenylene diamine, Methyl, Alcohol, Paraquat, Organophosphate.

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Introduction

Acute kidney injury (AKI) refers to the abrupt reduction of kidney functions, which is typically determined through an increase in serum creatinine or a decrease in urine volume, and is characterised by globally accepted staging paradigm systems, the most recent one Kidney Disease Improving Global Outcome (KDIGO).[1]

Aetiologies of AKI are wide-ranged, and have been traditionally divided into pre-renal, intrinsic and post-renal categories. Although hypovolaemia and sepsis are the main pre-renal diseases, intrinsic diseases are frequently encountered and are most likely secondary to an injury due to nephrotoxic agents (drug-induced nephrotoxicity, bacterial infections, or poisoning).[2]

The poisonous agents implicated in AKI are pesticides, herbicides, alcohol derivatives, heavy metals, paraphenylene diamine (PPD) and drugs in toxic doses.[3] These poisons damage the kidneys mostly by direct tubular toxicity, resulting in acute tubular necrosis, oxidative stress and free radical formation (observed with paraquat and methanol), and haemodynamic instability or shock that further impairs renal perfusion. Deposition of crystals in renal tubules is also induced by certain toxins, like ethylene glycol, PPD and organophosphates (OPs), that may induce rhabdomyolysis, which, in turn, causes secondary damage to the kidneys.[4] AKI caused by the toxin is commonly severe, oliguric, and necessitates dialysis more than other pathogen-induced conditions. The mortality rates are high, reaching up to 20% in certain cohorts, because of the coupled renal and systemic effects.[5] Recovery may not be complete even among survivors, and a high percentage still runs the risk of chronic kidney disease (CKD).

In the Pakistan-India subcontinent, epidemiology of AKI has the same complicated interplay of socioeconomic, infectious and toxicological causes. A 25-year retrospective study in Pakistan specifically highlighted a rise in toxin-related causes of AKI, such as toxic rhabdomyolysis and chemical exposures, reflecting the changing epidemiology of kidney injury in the region.[6] The subject of poison-induced AKI has become a relevant and under-reported cause. In 394 patients presenting with poisoning or envenomation into a large study done in northern India,

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among those who developed AKI, 41% were with paraquat and methanol, and more than one-half needed renal replacement therapy, while the mortality was nearly 28% with only two-thirds clinically practically regaining their kidney functions.[7] Similarly, AKI was observed in 22.3% of OP-poisoned patients in a Karachi-based study, and, also, poor timing of hospital presentation indicated a significant risk of kidney injury.[8] These results underline a significant regional burden of AKI based on poison, poor outcomes, such as high mortality, and regular dialysis requirements.

Together, AKI linked to poisoning has been disproportionately related to poor outcomes in the subcontinent, with evidence showing that AKI induced by poison often needs dialysis, has high short-term death rate, and often incomplete recovery of renal function.[6-8] With this background, emphasis should be placed on the outcome of poison-related AKI, especially in areas where the availability of renal replacement therapy is limited. The current meta-analysis was planned to integrate existing evidence on the outcomes of poison-related AKI in the subcontinent.

Materials and Methods

The systematic review was conducted in April 2025, and comprised search on PubMed, Cochrane Library, Web of Science, Embase, PubMed Central (PMC) and Google Scholar databases for original articles published in the English language. The analysis was carried out in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) guidelines.[9]

The search strategy used medical subject headings (MESH) and key words, including: ("Poisoning" OR "Toxicity" OR "Toxins" OR "chemical poisoning" OR "nephrotoxic agents") AND ("Acute Kidney Injury" OR "AKI" OR "acute renal failure" OR "kidney damage" OR "renal toxicity") AND ("Asia, Southern" OR "India" OR "Pakistan" OR "Asian" OR "Asian population"). These terms were adjusted in accordance with the requirements of each database. The results of the search were not restricted or filtered in any way. References of all relevant articles were manually reviewed by each researcher to identify additional studies suitable for inclusion in the meta-analysis. Through this cross-referencing process, further studies were identified and included.

Elimination of duplicate studies was done using Endnote X9 (Clarivate Analytics, US).

Observational studies comprising adult AKI patients with no previous co-morbidities after exposure to poisons. There was no limit in terms of time period. Outcomes in terms of all-cause mortality, complete renal recovery and partial renal recovery were recorded and analysed. Complete recovery meant serum creatinine was within normal limits (<1mg/dl), while partial recovery meant serum creatinine was >1mg/dl, but renal replacement was no more required.

Studies with no clear definition of population, AKI or poisons or toxins exposed, or with insufficient data for estimating mean difference (MD) with a 95% confidence interval (CI) were excluded, so were comments, reviews, case-control studies as well as duplicates.

Data was extracted by three researchers using a standardised tool. Discrepancies and disagreements, if any, were resolved with consensus by involving a fourth reviewer. Data collected included authors' name, year of publication, total number of patients, type of poison exposure, percentage of male and female patients, mean age, symptoms, duration of follow-up, laboratory values, requirement of haemodialysis, and outcome.

As all the included studies were observational, quality assessment was done using New Castle Ottawa scale.[10] Publication bias was evaluated using funnel plot asymmetry, Egger's regression test, and Begg's rank correlation test.

A leave one out sensitivity analysis was performed to assess the robustness of the pooled mortality estimates. Each study was sequentially excluded, and the meta analysis was repeated using a random effects model (DerSimonian Laird method)[11] for both the overall mortality analysis and the PPD subgroup. The results were presented using forest plots, showing the recalculated pooled effect after each exclusion. Risk of Bias assessment was done using The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE)[12] checklist.

Data was analysed using R studio version 4.16-2) used for retrospective studies. The combined data from multiple analyses was presented graphically as a forest plot, while funnel plots were generated for each outcome to determine if there was any publication bias. For variables, such as age and clinical features, simple descriptive statistics, such as mean ± standard deviation (SD) were used, while categorical data was expressed as frequencies and percentages. The Meta-analysis was registered in international Prospective Register of Systematic reviews (PROSPERO registration number CRD4202613222379).

Results

Of the 9,277 studies identified, 1,594(17.2%) were found through Pubmed, 28(0.3%) through Cochrane, 1,000(10.8%) through Google Scholar, and 6,655(71.7%) through Science Direct. Of them, 20(0.21%) studies were

shortlisted for full-text evaluation. Following detailed evaluation, 4(20%) studies were excluded as they either did not report outcomes for poisoning cases separately, or had insufficient data. The remaining 16(80%) studies were analysed along with 3 that were manually retrieved from cross-referencing, leaving a total of 19 studies[5,8,13,-29] for quantitative analysis (Figure 1).

Of the studies analysed, 12(63.2%) were from India and 7(36.8%) were conducted in Pakistan. The studies spanned over a period extending from1993 to 2025. All the 19(100%) studies were observational, with 14(73.7%) being retrospective, 2(10.5%) prospective and 3(15.8%) cross-sectional. The studies widely varied in terms of sample size, ranging from 13 to 1,032.[14,18] Most studies focussed on young adults with mean ages ranging between 20.50±6.2422 years and 40±8.5 years.[20,25] Poisonings other than PPD were mostly in male patients, like in a study[20] in which the

Table-1: Characteristics of the studies analysed (n=19).

Author	Country	Year	Study Design	Study Population	Male n (%)	Mean Age (years)	Surgical Technique	Duration of study (years)	Patients requiring HD/RRT
Agarwal SK[13]	India	1993	retrospective	66	N/A	N/A	Barbiturate (30), Copper sulphate (19), Mercuric chloride (9), Mandrax (4), Lithium (1), Ethylene bromide (1), Naphthalene (1), Tincture Iodine (1)	17	66 (100)
Chrispal A[14]	India	1993	retrospective	13	2 (15.4)	27.2	PPD	3.5	5 (38.5)
Gopalakrishnan S[15]	India	2020	prospective	99	51(51.5)	(19 to 46)	Rodenticide	N/A	N/A
Hanif S[8]	Pakistan	2022	cross-sectional	300	164(54.6)	28.5±6.15	Organophosphate	0.5	N/A
Iqbal J[16]	Pakistan	2019	cross sectional	42	13(31)	25.87±5.59	PPD	1	N/A
Jain PK[17]	India	2011	prospective	1020	286 (28)	27.81± 8.4	PPD	5	88 (8.62)
Khaskheli MS[18]	Pakistan	2018	retrospective	1032	350 (33.9)	22.08±8.42 (12 - 40)	PPD	6	N/A
Kondle R[19]	India	2012	retrospective	50	9 (18)	23.8±7.8	PPD	3	3 (6)
Kute VB[20]	India	2012	retrospective	91	91(100)	40±8.5	Methyl alcohol	2	91(100)
Naha K[21]	India	2012	retrospective	35	12 (34.2)	29.18±10.77	Copper sulfate	10	13 (37.14)
Nampoothiri K[22]	India	2010	retrospective	32	13(40.6)	27.8	Cleistanthus collinus	2.1	N/A
Naqvi R[5]	Pakistan	2017	retrospective	184	93(50.5)	24.37±8.3	PPD(135), methanol(8), Copper sulfate(5), OP(5), Paraquat(5),Tartaric acid(4), Phenobarbitone(3), enzodiazepines(2), Datura(2), Ammonium dichromate(1), Rat killer(1), Acetic acid(1), Arsenic(1), Boiler water(1) Multiple substances combination(9)	26	PPD: 131, methanol: 8 copper sulphate: 5, OP: 4 paraquat:5, others: 23
Naqvi R[23]	Pakistan	2015	retrospective	100	44 (44)	23.11±7.94	PPD	5	97 (97)
Perumal S[24]	India	2016	cross-sectional	46	16(34.7)	27.34± 9.51	PPD	N/A	9 (19.56)
Qureshi MMM[25]	Pakistan	2020	retrospective	24	2(8)	20.50 ± 6.24	PPD	0.3	N/A
Sweni S[26]	India	2012	retrospective	32	N/A	33	Chemical Toxins=8	1	8/8 (100)
Reddy SY[27]	India	2012	retrospective	81	43 (53)	29.58± 8.37	PPD	3.5	66 (81.4)
Tanweer S[28]	Pakistan	2018	retrospective	122	27 (22.1)	23.21±8.2	PPD	1.5	16 (43.2)
Yadla M[29]	India	2025	retrospective	400	300(75)	30.7±11.1	Paraquat	10	HD: 169(42.2) CRRT: 79(19.7) Peritioneal dialysis: 66(16.5) hemoperfusion: 76 (19)

HD=haemodialysis, RRT= renal replacement therapy CRRT= Continuous renal replacement therapy, PPD=para phenyl amine diamine, OP= organo phosphorus

Table-2: Quality assessment of the studies analysed.

S. no.	Author	Selection				Comparability	Outcome/ Exposure			Total	Quality
		Representativeness of the exposed cohort	Selection of the non-exposed cohort	Ascertainment of exposure	Demonstration that outcome of interest was not present at start of study	Comparability of cohorts on the basis of the design(type of poison) or analysis (multivariate analysis to adjust for confounders?)	Assessment of outcome	Was follow-up long enough for outcomes to occur	Adequacy of follow up of cohorts		
1	Agarwal SK[13]	*	*	*	*	*	*	*	-	7	Good
2	Chrispal A[14]	*	*	*	*	-	*	*	-	6	Fair
3	Gopalakrishnan S[15]	-	*	*	*	-	*	*	-	5	Fair
4	Hanif S[8]					Cross-sectional study					
5	Iqbal J[16]					Cross-sectional study					
6	Jain PK[17]	*	*	*	*	-	*	*	-	6	Fair
7	Khaskheli MS[18]	*	*	*	*	-	*	*	*	7	Good
8	Kondle R[19]	*	*	*	*	*	*	*	*	8	Good
9	Kute VB[20]	*	*	*	*	*	*	*	-	7	Good
10	Naha K[21]	*	*	*	*	*	*	-	-	6	Fair
11	Nampoothiri K[22]	*	*	*	*	-	*	*	-	6	Fair
12	Naqvi R (2017)[5]	*	*	*	*	*	*	*	-	7	Good
13	Naqvi R (2015)[23]	*	*	*	*	-	*	*	-	6	Fair
14	Perumal S[24]					Cross-sectional study					
15	Qureshi MMM[25]	*	*	*	*	-	*	-	-	5	Fair
16	Sweni S[26]	*	*	*	*	*	*	*	-	7	Good
17	Reddy YS[27]	*	*	*	*	*	*	*	-	7	Good
18	Tanweer S[28]	*	*	*	*	-	*	*	-	6	Fair
19	Yadla M[29]	*	*	*	*	*	*	*	-	7	Good

culprit substance was methanol, whereas PPD poisoning was reported more frequently among females, with one study reporting it very high[14] (Table 1). Quality assessment showed 8(42.1%) studies each were good and fair quality (Table 2). Risk of bias assessment showed 1(5.2%) study had poor quality, while 3(15.8%) had excellent quality (Table 3).

There was a wide range of reported decline in the amount of urine output in 7% to 92% of patients.[15,23] Similar pattern of wide variation was seen for haemoglobinuria/cola-coloured urine; in 8.6% to 94% of patients.[19,27]

Figure: Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) flow-chart.

Table-3: Risk of bias assessment of the studies analysed.

Author	Max Possible Score	Obtained Score	Adherence %	Quality
Agarwal SK[13]	21	8	38.09	Poor
Chrispal A[14]	22	12	54.54	Fair
Gopalakrishnan S[15]	24	24	100	Excellent
Hanif S[8]	25	22	88	Excellent
Iqbal J[16]	25	17	68	Fair
Jain PK[17]	27	18	66.67	Fair
Khaskheli MS[18]	21	15	71.43	Good
Kondle R[19]	24	19	79.16	Good
Kute VB[20]	25	18	72	Good
Naha K[21]	24	19	79.16	Good
Nampoothiri K[22]	22	15	68.2	Fair
Naqvi R (2017)[5]	22	16	72.7	Good
Naqvi R (2015)[23]	22	19	86.36	Excellent
Perumal S[24]	22	16	72.7	Good
Qureshi MMM[25]	21	12	57.14	Fair
Sweni S[26]	21	17	80.95	Good
Reddy YS[27]	21	15	71.43	Good
Tanweer S[28]	21	17	80.95	Good
Yadla M[29]	21	14	66.66	Fair

Cervico-facial oedema was common in PPD poisonings, ranging from 61% to 95%[27,28] of cases. Respiratory distress (dyspnoea, tachypnoea) occurred in 11% to 93% cases.[18,24] Hypotension was noted in 8% to 20%[21,28] of PPD and other poisonings. Gastrointestinal symptoms were common; vomiting in 81% to 87% patients,[15,20] abdominal pain in 31% to 52%, and gastro-intestinal bleeding in 6% to 40%. Neurological signs, such as altered sensorium and convulsions,[17] appeared in barbiturate and rodenticide cases, though convulsions were relatively rare, ranging from 2% to 23%.[13,15] Muscle aches featured in 83% to 100% of PPD cases.[6,25]

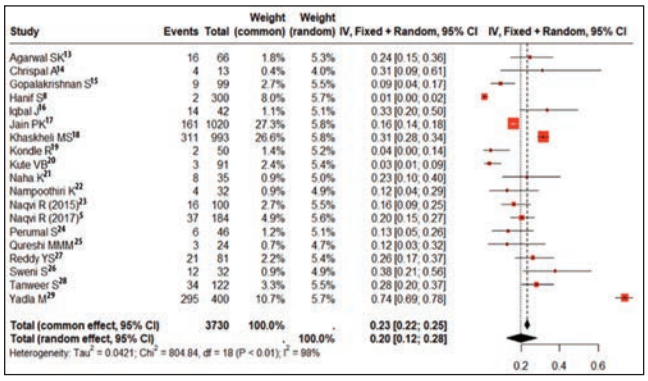

Figure-2 A: Forest plot analysis of all-cause mortality.

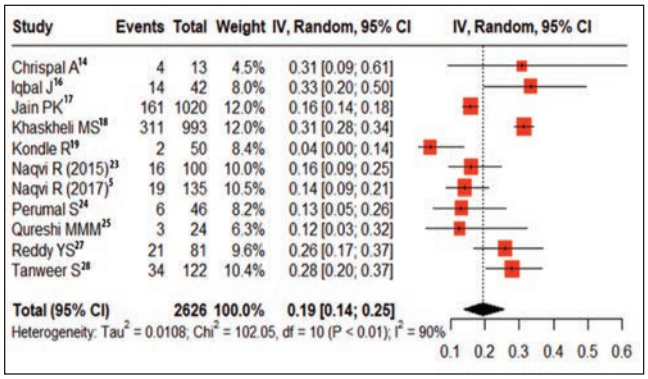

Figure-2 B: Forest plot sub-group analysis of paraphenylene diamine (PPD) mortality.

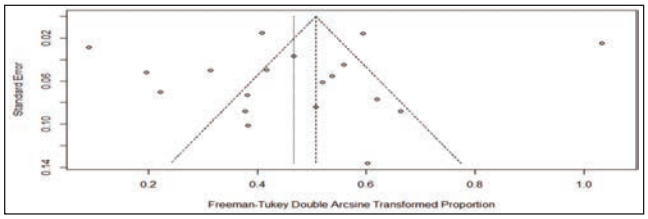

Figure-3 A: Funnel plot analysis of all-cause mortality.

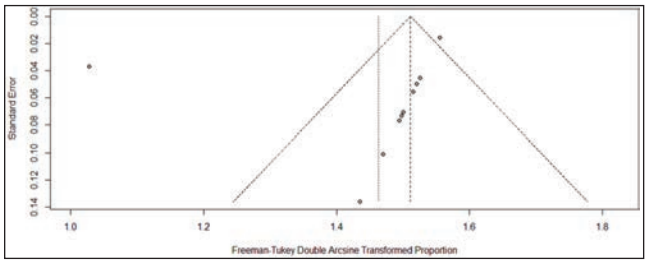

Figure-3 B: Funnel plot sub-group analysis for paraphenylene diamine (PPD) mortality.

All the studies reported an increase in serum creatinine levels, with the highest being 25.15 mg/dl in PPD poisoning, and the same study also reported the highest serum urea levels in PPD poisoning cases 496mg/dl.[5] Electrolyte derangements were frequent; with hyperkalaemia 5.4-9.3meq/L,[5] hypocalcaemia 7.2mg/dl and hyperphosphatemia 8.5mg/dl.[14] In many studies, hepatic enzymes were markedly elevated in PPD poisoning cases. Similarly, very high levels of creatinine phosphokinase (CPK) (maximum of >1 million U/L)[5,23] and lactate dehydrogenase (LDH) (maximum of 29,130U/L)[5,23] were reported in PPD poisoning cases.

Across the studies, 23% to 100% cases fully recovered and became dialysis-independent.[19,29]

Partial renal recovery ranged from 5% to 23% cases.[15,26]

Leave one out sensitivity analysis for overall mortality showed that the pooled estimate remained stable after sequential exclusion of each individual study. The recalculated pooled mortality ranged from 0.15 (95% CI: 0.11-0.19) when the study by Yadla et al.[29] was excluded to 0.21 (95% CI: 0.12-0.30) when studies by Hanif et al.[8] or Khaskheli et al.[18] were excluded. No single study significantly altered the overall effect size (Figure 2A).

In the PPD subgroup of 10(52.6%) studies, the leave one out analysis similarly demonstrated stability, with pooled estimates ranging from 0.15 (95% CI: 0.08-0.22) to 0.18 (95% CI: 0.13-0.24) after the exclusion of any single study. No study disproportionately influenced the pooled estimate (Figure 2B).

Visual inspection of the funnel plot suggested asymmetry. Egger's test confirmed significant asymmetry (t=2.49, df=17, p=0.023), while Begg's test did not reach statistical significance (Kendall's $\tau = 0.1345$, p=0.447). The funnel plot displayed the relationship between effect sizes and standard errors. The pooled effect estimate was reflected by a central vertical line around 0.5. Upon visual evaluation, the asymmetry among studies was evident, especially among those with higher standard errors, meaning small sample sizes (Figure 3).

Discussion

Poisons, regardless of their ingestion being accidental or intentional, remain a challenging issue in emergency departments (EDs) of hospitals. Intention can be either self-harm or harm to others. Overdose of medicinal substances or recreational substances may also fall in the category of

poisoning emergency. In the Pakistan-India region, in contrast to benzodiazepines and opioids commonly used in European countries, common poisons seen in emergencies are herbicides, insecticides, pesticides (organophosphorus compounds commonly used in pesticides), corrosives, ethylene glycol, methanol, PPD, copper sulphate ($CuSO_4$), Datura, rodent killers, raw fish gall bladder (icthyotoxic), arsenic, ammonium dichromate, kerosene oil, diesel, naphthalene, mercuric chloride, paraquat, turpentine oil, etc[5,13,29] Animal toxins in the region were not analysed and addressed in the present study.

Depending on dose, timing and extent of damage, as a result of exposure to any of the poisons, the affected person is dealt with in an emergency setting, or referred to high dependence unit (HDU) or tertiary care in case of organ damage. The present study analysed only studies from India and Pakistan addressing AKI managed by nephrology services after exposure to poisons. Of the 19 studies, 12 were from India and 7 from Pakistan, and together comprised a total population of 3,769. Of the total, 8 studies only addressed one poison, that is PPD, while another 3 studies discussed multiple poisons, including PPD.

While PPD is relatively new to the field of nephrology, its use as a hair-dye is quite ancient, and the first published report related to it goes back more than a century.[30] At the current tertiary care renal unit, the first AKI patient as result of PPD poisoning was seen in 2010.[23] PPD is a derivative of alanine, and its human exposure is through hair-dyes, fortified henna, photocopying, printing ink, black rubber and lithography plates. It usually produces local dermatological reactions, which may vary from mild to severe anaphylactic reactions. Upon oral ingestions, it leads within minutes to severe laryngeo-facial oedema, causing respiratory distress and difficulty in swallowing. These patients often require tracheostomy upon reaching the emergency department. Angioedema, hepatotoxicity and rhabdomyolysis occur. AKI results from both direct toxicities of tubular epithelial cells and blockage of tubular Lumina with myoglobin casts. Very high levels of CPK and LDH have been reported in published literature in PPD poisoning cases.[5,14] In a previous study conducted by the current team, 16% mortality was seen in the acute phase of AKI post-PPD poisoning, while complete renal recovery over an average of 6 weeks was reported in 77% cases.[23] An Indian study also showed renal recovery in 61.5% of patients in a small sample.[14] The pooled mortality with fixed and random effect models in the present meta-analysis was 20-30%. Subgroup analysis for mortality in PPD poisoning cases led to results that were not much different. This may be explained by the fact that PPD poisoning cases constituted the majority of overall population.

A large Indian study of 400 paraquat poisoning causing AKI cases over 10 years reported high overall mortality of 74%. However, patients who were timely subjected to hemoperfusion showed significantly lower mortality rate.[29]

$CuSO_4$ poisoning causing AKI is also a problem that has been reported for long in the region. In one study, 11 patients developed AKI after $CuSO_4$ poisoning and 5(45%) of them died during the acute phase of the illness.[31]

Methyl alcohol is an adulterated, cheap and potent liquor. Many outbreaks of methanol poisoning have occurred in Pakistan and India.[17,29] Outbreaks have been reported from both countries at least in newspapers reports, if not in scientific literature, leading to heavy mortality and morbidity.[32] Haemodialysis rapidly removes both toxic acid metabolites and parent alcohols, and plays a fundamental role in treating severely poisoned patients, and has proven to be a lifesaving therapy in the case of accidental or intentional overdose.[17]

OP anti-esterase compounds, widely used as insecticides in agricultural and domestic settings throughout the world, are easily available. They act via multiple routes, including oral ingestion, inhalation or trans-dermal, and can be used for self-harm or homicidal purposes. A study in Pakistan, with a population of 300 OP poisoning cases, reported AKI in 22.3% and emphasised the lag time between poison ingestion and arrival at hospital. However, the study did not comment on the need for Renal Replacement Therapy (RRT) or mortality.[8]

High mortality rates in cases of poisoning and AKI in the subcontinent are driven by a complex interplay of easy access to toxins, many of which are available at a very low cost, and delayed medical intervention, with many people reaching a proper healthcare facility only after organ damage has already occurred. Additionally, there are limited specialised healthcare resources.

The current analysis revealed a high heterogeneity across studies (I^2=90%). This variation can be explained by differences in toxic agents / poison used, ranging from PPD, paraquat, OP, pesticides, alcoholic derivative, heavy metals, benzodiazepines or other drugs in heavy doses. Additionally, diagnostic criteria for AKI differ, with significant differences in baseline creatinine measurements, as well as attitude towards specific renal care and support. The inconsistent reporting of lag time before presenting to proper renal service (from where the study was generated) further complicated the task of

reporting the issue in a uniform pattern.

Conclusion

In the subcontinent, comprising Pakistan and India, there is a variety of cheap and easily available substances that can cause poisoning and systemic harmful effects, including single or multi-organ failure. The exposure to these poisons can be intentional or accidental and for both suicidal or homicidal purposes. The extent of morbidity and rate of mortality varied widely based on multiple co-factors, like dosage, time lag between exposure and reaching a medical facility, and availability of services at that particular facility. Furthermore, many healthcare facilities dealing with such patients have not published such data. Therefore, the wide heterogeneity in available literature makes it more difficult to lay down proper guidelines for the management of such patients.

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Author Contribution:

AA: Data curation, formal analysis and writing initial draft (methods section).
SA: Data curation, formal analysis and writing initial draft (results section).
AA: Data curation, validation and writing initial draft (introduction section).
RN: Concept, final analysis, review of initial draft, discussion writing and editing.