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## AIM AND SCOPE

*World Journal of Nephrology* (*World J Nephrol*, *WJN*, online ISSN 2220-6124, DOI: 10.5527) is a peer-reviewed open access academic journal that aims to guide clinical practice and improve diagnostic and therapeutic skills of clinicians.

*WJN* covers topics concerning kidney development, renal regeneration, kidney tumors, therapy of renal disease, hemodialysis, peritoneal dialysis, kidney transplantation, diagnostic imaging, evidence-based medicine, epidemiology and nursing. Priority publication will be given to articles concerning diagnosis and treatment of nephrology diseases. The following aspects are covered: Clinical diagnosis, laboratory diagnosis, differential diagnosis, imaging tests, pathological diagnosis, molecular biological diagnosis, immunological diagnosis, genetic diagnosis, functional diagnostics, and physical diagnosis; and comprehensive therapy, drug therapy, surgical therapy, interventional treatment, minimally invasive therapy, and robot-assisted therapy.

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## Retrospective Study

**Acute kidney injury from different poisonous substances**

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**Abstract****AIM**

To report our experience of acute kidney injury (AKI) developed after exposure to poisonous substance.

**METHODS**

Retrospective study where data was collected from case records of patients coming to this institute during January 1990 to May 2016. This institution is a tertiary care center for renal care in the metropolitan city of Karachi, Pakistan. History of ingested substance, symptoms on presentation, basic laboratory tests on arrival, mode of treatment and outcome were recorded from all patients and are presented here. Patients developing AKI after snake envenomation or scorpion stings are not included in this study.

**RESULTS**

During studied period 184 cases of AKI developing after poisoning were seen at our institution. The largest group was from paraphenyline diamine poisoning comprising 135 patients, followed by methanol in 8, organophosphorus compounds in 5, paraquat in 5, copper sulphate in 5, tartaric acid in 4, phenobarbitone in 3 and benzodiazepines, datura, rat killer, fish gall bladder, arsenic, boiler water, ammonium dichromate, acetic acid and herbs with lesser frequency. In 8 patients multiple substances were ingested in combination. Renal replacement therapy was required in 96% of patients. Complete recovery was seen in 72.28% patients, 20% died during acute phase of illness.

**CONCLUSION**

It is important to report poisonous substances causing vital organ failure to increase awareness among general population as well as health care providers.

**Key words:** Paraphenylene diamine; Organophosphorus compounds; Paraquat; Methanol; Poisons; Acute kidney injury

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**Core tip:** During our daily life we are exposed to certain substances/compounds, which may be used as pesticides, herbicides, insecticides, coloring inks, photocopying or may found in some plants. Use of these compounds intentionally or accidentally as per oral ingestion and absorption *via* gastro intestinal tract or taken *via* par-enteral route may cause hazardous, sometimes lethal effects. Current study highlights acute kidney injury as result of some of these poisons, dealt at a tertiary renal care unit. Awareness regarding pathophysiological consequences, need of early referral to particular specialized center and at society level at par is important issue addressed here.

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## INTRODUCTION

Paraphenylene diamine (PPD) is a derivative of aniline. Human exposure is primarily through hair dyes and fortified "henna" which is used for tattooing. Occupational exposure is through photocopying and printing inks, black rubber, lithography plates, etc<sup>[1]</sup>. Poisoning with PPD can cause oro-pharyngeal and gastrointestinal symptoms, hepatic involvement and later neurological involvement. Acute kidney injury (AKI) was reported in two cases from India in 1982<sup>[2]</sup>. Earlier we have published a large series of 100 cases developing AKI after PPD poisoning. Rhabdomyolysis leading to acute tubular necrosis (ATN) and pigment nephropathy remains main pathophysiology of AKI after PPD poisoning<sup>[3]</sup>. Methanol, also known as wood alcohol, is a commonly used organic solvent that can cause metabolic acidosis, neurologic sequelae, and even death. It is a constituent of many commercially available industrial solvents and of poorly adulterated alcoholic beverages, in Pakistan called as "katchi sharab". Toxicity with ingestion of methanol, which is more often in larger groups of people, remains a common problem in many parts of the developing world, especially among members of lower socioeconomic classes. AKI may be associated with other signs of severity in methanol poisoning, but it is almost always reversible in survivors. The pathophysiology of AKI developing after methanol poisoning is multifactorial but ultimately leads to acute tubular necrosis<sup>[4]</sup>.

Organo phosphorus (OP) compounds are frequently used as pesticides, herbicides, and even chemical warfare agents, large cohort has recently been published by Lee *et al*<sup>[5]</sup>, though study is population based it highlights risks of AKI development after exposure to OP poisoning. The rapid accumulation of acetylcholine in synaptic junctions

of central nervous system and peripheral tissues results in cholinergic crises and this may lead to ischemic ATN<sup>[5]</sup>. Paraquat (1,1'-dimethyl-4,4'-bipyridylum dichloride) is widely used as a herbicide. Toxicity is usually seen following ingestion, which could be accidental or intentional and may range from mild to fulminant (according to dose ingested) leading to death. In addition to local irritation, multi organ failure (MOF), including kidney, may occur<sup>[6]</sup>. Tartaric acid is a white crystalline organic acid that can be extracted from plants. It is commonly mixed with sodium bicarbonate and is sold as baking powder used as a leavening agent in food preparation. The acid itself is added to foods to add sour taste<sup>[7]</sup>. Over dosage of tartaric acid can give rise AKI, gastro intestinal symptoms and cardio-vascular collapse. Volume loss from GI in form of intractable vomiting can lead to ATN<sup>[8]</sup>.

CuSO<sub>4</sub>, one of the most available salts of copper, is a blue and odorless salt that is used in various products such as fungicides, algacides, herbicides and insecticides<sup>[9]</sup>. Copper sulfate can be absorbed through the gastrointestinal tract, lungs and skin causing both systemic and local toxicity. AKI has also been reported after CuSO<sub>4</sub> poisoning<sup>[10]</sup>. Symptoms related to systemic toxicity include delirium, stupor, coma, convulsion, hypotension, shock, respiratory failure, pallor and jaundice. Methemoglobinemia, rhabdomyolysis and hepatotoxicity have also been reported. Pigment nephropathy and ATN are most probable patho-physiological mechanisms here<sup>[11]</sup>.

Datura is a poison from some flowering plants, contain tropane alkaloids such as scopolamine, hyoscyamine, and atropine, primarily in their seeds and flowers. Because of the presence of these substances, Datura has been used for centuries in some cultures as a poison<sup>[12]</sup>. Datura toxins may be ingested accidentally by consumption of honey produced by several wasp species, In some parts of Europe and India, Datura has been a popular poison for suicide and murder. From 1950 to 1965, the State Chemical Laboratories in Agra, India, investigated 2778 deaths caused by ingesting Datura<sup>[13]</sup>. Due to the potent combination of anticholinergic substances it contains, Datura intoxication typically produces effects similar to that of an anticholinergic and ischemic ATN may result. Other possibility of interstitial edema and direct nephrotoxic effect can not be ruled out.

Rat killer or rodent killers can give rise accidental or intentional harm to human. Components of available rat killers are; Brodifacoum, Diphacinone, Warfarin and Bromadiolone. All of these substances are anti coagulants and can cause severe coagulopathy after ingestion<sup>[14]</sup>. Acute volume loss can lead to ischemic ATN and hemoglobin pigments can also cause blockade of tubular lumina.

Ichthyotoxic AKI has been reported after fish gall bladder ingestion previously. Toxin is believed to be cyprinol sulphate. This poisoning can involve gastrointestinal, hepatic, cardiac and neurological systems along with kidneys<sup>[15]</sup>. AKI can occur as part of MOF or ischemic ATN or acute tubule-interstitial nephritis (ATIN) may be a



**Table 1 Contribution from different substances (n = 184)**

Poisonous substance	No. of patients	%
PPD	135	73.36
Methanol	8	4.34
CuSO <sub>4</sub>	5	2.71
OP	5	2.71
Paraquat	5	2.71
Tartric acid	4	2.17
Phenobarbitone	3	1.63
Benzodiazepines	2	1.08
Datura	2	1.08
Ammonium dichromate	1	0.54
Rat killer	1	0.54
Fish gallbladder	1	0.54
Acetic acid	1	0.54
Arsenic	1	0.54
Boiler water	1	0.54
Multiple substances taken in combination	9	4.89

PPD: Paraphenylene diamine; OP: Organo phosphorus.

possibility. Arsenic has been used in many medicines and was widely used to treat syphilis until the mid 20<sup>th</sup> century. It is currently used to treat acute promyelocytic leukaemia and other myeloproliferative disorders<sup>[16]</sup>. It has also been used as a pigment, a pesticide and a poison. AKI with acute hemolysis has been reported after acute arsenic poisoning<sup>[17,18]</sup>.

Ischemia reperfusion injury and tubular necrosis can result with Benzodiazepines ingestion<sup>[19]</sup>. Ammonium dichromate is an inorganic compound frequently used in screen and color printing. Being a strong oxidizing agent, it can cause oxygen free radical injury and renal tubular necrosis<sup>[20]</sup>. Acetic acid is an organic acid that is used for homemade vegetable preserves and is available in grocery stores easily. Coma, shock, hemolysis and anuric renal failure has been reported with poisoning with acetic acid<sup>[21]</sup>. The patho-physiological mechanisms here could be pigment nephropathy and ATN or ATIN.

## MATERIALS AND METHODS

Case records of all patients registered in emergency room of Sindh Institute of Urology and Transplantation (SIUT), Karachi, with diagnosis of AKI which was labeled upon findings of sudden rise in serum creatinine and decline in urine output in a previously healthy person after ingestion of one of the poisonous substances listed in Table 1. Renal ultrasound was done in all patients on the day of registration and all had normal size non-obstructed kidneys. Patients with previous co morbidities like diabetes mellitus, hypertension or known kidney disease were excluded. Patients developing AKI after snake bite or scorpion stings are also not included in the present study as published earlier separately (see references below in discussion). Clinical history, presenting symptoms, laboratory investigations including complete blood count, renal and hepatic chemistry, serum lactate dehydrogenase, international normalization ratio for coagulation and urinalysis from day of admission

were recorded. Other features recorded were need for renal replacement therapy and patient outcome. Renal replacement therapy where required was done in form of hemodialysis only. Institutional review board was consulted to grant permission to compile and publish the data. Personal identification of individual patients not revealed. Informed consent as routine taken from all patients reaching to emergency of this hospital.

## RESULTS

Between January 1990 and May 2016, 184 patients with AKI secondary to poisons were brought to SIUT emergency. Male to female ratio was 1.02:1, while mean age was  $24.37 \pm 8.30$  (median 22 years). Distribution of patients brought with different poisons is given in Table 1, while Tables 2 and 3 highlight on major presenting symptoms and clinical and laboratory parameters of these patients. The majority of patients were brought here due to marked decline in urine output and uremic symptoms. Urinalysis is available in 121/184 (65.76%) patients and dipstick revealed 1-2+ protein in 69/121 urine samples while 21 and 11 showed 3+ and 4+ proteins. Numerous red blood cells per high power field were seen in 95/121 urine samples. Renal biopsy was available from 13 patients and the most common finding was acute tubular necrosis found in 11 biopsy samples. Pigment cast in tubules were seen in 6 biopsies. Tubulo interstitial involvement along with ATN was seen 2 biopsies, and in isolation one sample had cast along with TIN (biopsy from patient with methanol poisoning). One biopsy revealed acute cortical necrosis (ACN). Renal replacement therapy was required in 96% patients soon after arrival. Outcome of patients from different groups is given in Table 4.

## DISCUSSION

We have previously published a series of patients developing AKI after PPD<sup>[3]</sup> poisoning. The current study includes those cases as well as further cases from PPD and in addition AKI, as a result of exposure to other poisons. Poisoning with PPD has increased over last 6-7 years in our country, especially in the province of Sindh where this institution (SIUT) is located, and we are continuously receiving patients with AKI developed after toxic rhabdomyolysis as a result of ingestion of PPD. Measures for public awareness in the form of editorials, letters to the editors of local newspapers and local television channels have been made but so far no decline in use of poison has been observed. Contributing factors such as easy availability and low cost also remain unchanged.

Methanol is a substance which usually ingested in large groups on special occasions as entertainment by people in the low socio economic stratum. Annually, a large number of deaths are reported with methanol ingestion in groups<sup>[22]</sup>, survivors developing AKI are brought to SIUT and are included here in this study. Elevated levels of creatinine kinase indicate rhabdomyolysis as a cause of

**Table 2 Presenting symptoms (n = 184)**

Symptom	Frequency (%)	Mostly with substance
Oligoanuria	93	All groups except phenobarbitone
Vomiting	90	All groups
Hematuria/cola color urine	86	PPD, CuSO <sub>4</sub> , Paraquat
Massive facial/neck swelling ± difficulty in swallowing	74	PPD
Hemetemesis	16	CuSO <sub>4</sub> , OP, Paraquat, boiler water
Altered level of consciousness	16	Methanol, phenobarbitones, benzodiazepines, OP
Temporary loss of vision	2.17	Methanol

PPD: Paraphenylene diamine; OP: Organo phosphorus.

**Table 3 Laboratory and other parameters on presentation in different groups (median values and ranges given)**

Paramet	PPD (n = 135)	Methanol (n = 8)	CuSO <sub>4</sub> (n = 5)	OP (n = 5)	Paraquat (n = 5)	Others (n = 26)
DOI	4 (1-28)	5 (1-15)	4 (2-5)	5 (2-6)	3 (1-5)	5 (1-25)
Hb, g/dL	11.95 (2.5-21.5)	13.7 (9-19.5)	5.5 (2-9.9)	10.3 (6.7-13.4)	8 (6.3-11)	11.5 (7.6-15.6)
Plt, × 10 <sup>9</sup> /L	234 (59-603)	285.5 (58-610)	303 (101-687)	114 (98-121)	90 (47-213)	211.5 (10-910)
Urea, mg/dL	183 (69-496)	123.5 (47-329)	282 (108-384)	234 (114-367)	224 (197-338)	213.5 (65-483)
Creat, mg/dL	7.81 (3-25.15)	5.25 (2-13.79)	10.3 (7.1-10.8)	9 (3.9-14.71)	9.9 (4.7-13.4)	10.25 (2.9-24.49)
K, meq/L	5.8 (2-9.3)	5.85 (2.7-7.9)	5.4 (3.4-6.8)	5.9 (3.7-6.9)	6 (5.5-6.8)	4.4 (1.9-6.9)
LDH, U/L	4785 (236-29130)	486 (95-4000)	11223 (974-13245)	2349 (891-3448)	1429 (756-6432)	1169 (179-4635)
CK, U/L	115989 (54-1998000)	115220 (387-228300)	Available for one patient only	699 (126-1012)	NA	806 (32-98720)
AST, U/L	2024 (15-17380)	136 (46-1748)	48 (34-425)	167 (78-557)	209 (88-456)	70.5 (3-771)
ALT, U/L	975 (13-6310)	104 (59-766)	34 (23-150)	112 (34-316)	101 (72-338)	36 (10-1036)
HD (ses rq)	4 (1-13)	1.5 (1-6)	3 (1-12)	2 (1-3)	2 (1-5)	2 (1-10)
HD done in	131/135	8/8	5/5	4/5 (one died before starting HD)	5/5	23/26

DOI: Days of insult; Hb: Hemoglobin; plt: Platelet; K: Potassium; LDH: Lactate dehydrogenase; CK: Creatinine phosphokinase; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; HD: Hemodialysis; ses rq: Sessions required; PPD: Paraphenylene diamine; OP: Organo phosphorus.

**Table 4 Outcome in different groups (n = 184)**

Outcome	PPD (n = 135)	Methanol (n = 8)	CuSO <sub>4</sub> (n = 5)	OP (n = 5)	Paraquat (n = 5)	Others (n = 26)
Complete recovery	108	5	3	2	1	14
Partial recovery and lost follow-up	6	1	0	0	0	4
Left against advice during acute phase	2	0	0	0	0	1
Died	19	2	2	3	4	7

PPD: Paraphenylene diamine; OP: Organo phosphorus.

AKI in these patients.

CuSO<sub>4</sub> intoxication causing AKI reported by Chugh *et al*<sup>[23]</sup> from India about 40 years ago. This substance is also easily available at low cost and used for both suicidal and homicidal purposes. Hemolysis is a common pathological feature and ATN is seen on renal histology. In our series renal biopsy was done in one patient and showed ATN with pigments, but in those days we were not doing markers for pigments (which we have started doing during last decade). Another study from our neighboring country, India, reports different poisons leading to hemodialysis requirement. This series studied over a period of 17 years reports 19 cases of AKI after CuSO<sub>4</sub> poisoning. Our patients with CuSO<sub>4</sub> poisoning revealed features of hemolysis with low hemoglobin, raised LDH, serum potassium and hyperbilirubinemia, which is in agreement with previously published studies.

Our patients with paraquat poisoning showed worst prognosis where 4 out of 5 died within 60 h of reaching

the hospital, one even before starting the dialysis. One who survived from this group later recovered renal function. Paraquat ingestion could be intentional or after occupational exposure. In 3 of our patients it was intentional while remaining 2 were exposed accidentally.

Published studies from Pakistan report benzodiazepines, mega doses of antidepressants and organophosphorus compounds utilized for self harm<sup>[24]</sup>. Some of our patients has taken a combination of substances and most of these were with intention of self harm. All 3 patients of our series with phenobarbitone over dosage were brought unconscious with Glasgow Coma scale of 3. One of them died while the other two recovered very early, only 2 sessions of hemodialysis were required in each.

We have 4 patients in our series with poisoning from tartaric acid, a substance very commonly used as a food additive in our country, mainly to make food sour. Large amount of the powdered form of the substance

was taken by 3 patients for self harm and in one the substance was given by in-laws. Literature search has not revealed renal damage with the substance, but our findings on renal biopsy in two of these patients revealed ATN.

AKI as a result of acetic acid poisoning has been reported in 34 patients in a study published from Serbia, where they have treated these patients with peritoneal dialysis. Most of the patients were oligoanuric. Ten patients died in this series. The most frequent complications were oesophagitis, bleeding, mediastinitis, pneumonia and acute abdomen<sup>[25]</sup>. In our series there was only one patient who developed AKI after acetic acid poisoning. This patient was a young male, who developed excessive vomiting and abdominal pain after ingestion, but showed rapid renal recovery.

Datura poisoning though used in our part of world as a homicidal agent, was used accidentally by two of our patients in the current series. Both developed anuric renal failure though they recovered required a bit prolonged dialysis (6 and 10 sessions).

Use of boiler water in one of our patient led to severe hemolysis and anuria. This patient died on the next day after reaching this hospital. Arsenic was taken by one of the patients for a sexually transmitted disease. He developed gastro intestinal symptoms, fever and paraplegia along with deafness after taking the substance, was anuric when brought to us with advanced uremia and deranged liver functions. He died after having 5 sessions of plasma exchange and 7 sessions of hemodialysis.

At this institution, we do renal biopsy as a policy in cases of AKI which have a suggestive history of glomerular disease or reveal protein on urine dipstick 3-4, or show delay in recovery. In the current series of patients biopsy samples were taken from 13. In 11 there was ATN, and on further breakdown we found ATN with pigment in 7 and ATN with TIN in 2. One biopsy revealed TIN with casts in tubular lumina and tubular damage; this was from a patient with methanol poisoning. One biopsy revealed ACN this biopsy was from a young patient who was living in bachelor's hostel and ingested a substance that was probably glycol along with multiple tablets of Xanax (benzodiazepines). In this series, his case was categorized under multiple substances.

Some of the patients brought into the emergency of SIUT with AKI and suspicion of poisoning were not included in this series as the patient or the family were reluctant to disclose the actual history though their course of management remained the same as for others. As mentioned earlier we have also not included AKI resulting from snake bite and scorpion stings in this series as these have been published separately<sup>[26,27]</sup>.

### Limitations

The majority of patients exposed to poisons are dealt with general medical wards by internists or at small medical setups by family physicians. Because of this, the detection of renal involvement was delayed in many patients in this series, thus many patients reached us

when the time for taking beneficial measures had already elapsed.

Furthermore, most of patients cannot describe the amount of substance taken and thus it is difficult to comment on the amount of substance that can cause renal damage or could be fatal.

In conclusion, this study reveals possibility of renal involvement and AKI with ingestion of different poisonous substances, which usually have other uses and are thus frequently available. In developing countries like Pakistan money is another considerable factor. Most of the poisonous substances described here were both easily available and inexpensive. We believe that our results could be valuable for physicians in general and nephrologists in particular in managing such cases in emergency.

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## COMMENTS

### Background

Human exposure to paraphenylene diamine (PPD) is primarily through hair dyes and fortified "henna" which is used for tattooing. Occupational exposure is through photocopying and printing inks, black rubber, lithography plates, etc. Poisoning with PPD can cause oro-pharyngeal and gastrointestinal symptoms, hepatic involvement and later neurological involvement. Acute kidney injury (AKI) was reported in some cases. Developing AKI after PPD poisoning were also reported. It is a constituent of many commercially available industrial solvents and of poorly adulterated alcoholic beverages, in Pakistan called as "katchi sharab".

### Research frontiers

Toxicity with ingestion of methanol, which is more often in larger groups of people, remains a common problem in many parts of the developing world, especially among members of lower socioeconomic classes. AKI may be associated with other signs of severity in methanol poisoning, but it is almost always reversible in survivors. The pathophysiology of AKI developing after methanol poisoning is multifactorial but ultimately leads to acute tubular necrosis.

### Innovations and breakthroughs

This study reveals possibility of renal involvement and AKI with ingestion of different poisonous substances, which usually have other uses and are thus frequently available. In developing countries like Pakistan money is another considerable factor. Most of the poisonous substances described here were both easily available and inexpensive.

### Applications

The results could be valuable for physicians in general and nephrologists in particular in managing such cases in emergency.

### Peer-review

The draft could be interesting for the nephrologist. It is potentially interesting as a data base for consultation.

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