Current Practice

Evaluation and treatment of children with poisoning

S S Manohari[1], *Kavinda Dayasiri[2], Michael Jansz[2]

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Acute poisoning is a common presentation seen in the paediatric emergency setting. Majority of these attendings are non-life threatening and observation alone is adequate. Most are due to accidental poisoning but in the teenage years, deliberate poisoning and experimentation with illicit substances are increasingly seen[1,2]. Any substance should be considered a potential poison, depending on the dose and duration of exposure[3].

The initial approach includes pre-hospital care, emergency care and arrangements to be made for definitive care. In the pre-hospital set-up, monitoring and intravenous (IV) access are considered the most important initial steps. In addition, supplemental oxygen, correction of hypoglycaemia, treatment of seizures with benzodiazepines, administration of naloxone and treatment of broad complex tachycardia with sodium bicarbonate should be considered, according to the available facilities and competencies of the emergency medical services (EMS) on a case by case basis. There is limited evidence for clinical efficacy of pre-hospital treatment with activated charcoal[3]. However, in reality, little is expected from EMS.

Initial management in the emergency department includes resuscitation with an ABC approach. Airway assessment should be focused on assessing patency by looking, listening and feeling for air movement. If inadequate breathing is present, the airway should be opened up with simple manoeuvres such as jaw thrust, followed by the usage of airway adjuncts. Airway oedema, leading to obstruction, may be caused by corrosive agents and plants containing calcium oxalate crystals.

Breathing should be assessed by the respiratory rate, usage of accessory muscles and peripheral oxygen saturation. If the respiratory effort is not satisfactory, support should be provided initially with bag-valve mask ventilation until a definitive airway is established, as indicated. This will be more of a consideration in children with organophosphate poisoning and central nervous system depressant overdose.

The adequacy of the circulation can be evaluated by direct measurements such as capillary refill time, heart rate and arterial blood pressure. This is also depicted by indirect measures of tissue perfusion such as altered level of consciousness, urine output, skin turgor and colour. Resuscitation with 20ml/kg of crystalloid fluid boluses for hypotension is recommended for shock. However, in beta blocker overdose, the mainstay of treatment for hypotension is High-dose Insulin Euglycaemia Therapy (HIET). Incorporation of inotropes/ vasopressors should be judicious and a toxicological opinion should be sought. Arrhythmias should be managed by correcting the precipitants such as acidosis and/or hyperkalaemia as well as the use of antidotes e.g. sodium bicarbonate in tricyclic antidepressant poisoning. Anti-arrhythmic drugs should be avoided as much as possible as they themselves can be arrhythmogenic.

Assessment of disability includes the neurological status with the Alert, Voice, Pain, Unresponsive (AVPU) score or Glasgow Coma Scale (GCS), pupillary size and their reaction to light, posturing and seizures, and blood sugar level.

The child should be exposed thereafter, while preventing hypothermia. On the other hand, teenagers can present with hyperthermia in suspected overdoses such as cocaine, sympathomimetic agents, salicylate, and anti-cholinergic agents *(Datura)*. In these circumstances, cooling with external and internal methods is recommended. There is little place for antipyretics[1].

[1]*Senior Registrar in Emergency Medicine*
[2]*Consultant Paediatrician Emergency Treatment Unit, Lady Ridgeway Hospital, Colombo*
**Correspondence:* kavindadayasiri@gmail.com

https://orcid.org/0000-0003-0438-9837

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At the end of the resuscitation, decontamination and early administration of antidotes should be considered. Gastrointestinal (GI) decontamination can be performed by several methods. Syrup of Ipecac is no longer used in clinical practise or as a home remedy. Gastric lavage, which was once a routine method of GI decontamination, is no longer routinely recommended, as it has demonstrated no clinically significant benefit. It also has the risks of serious complications such as aspiration, gastrointestinal perforation and electrolyte imbalance[2].

Activated charcoal (AC), with its adsorbing effect on the ingested substances, plays the main role in GI decontamination. A dose of 1-2 g/kg is recommended and is best given within 60 minutes of ingestion of the toxin. Multiple Dose Activated Charcoal (MDAC), acts as a mode of enhancing elimination by preventing the enterohepatic circulation of drugs such as digoxin, carbamazepine and phenobarbital. MDAC is also used in overdose of salicylates to form bezoars or concretions, and in ingestion of slow-release/ continuous release preparations. The disadvantages of using AC are that it is unpalatable, poorly accepted, and tends to be vomited out by children. The palatability can be improved by mixing it with flavoured syrup or ice cream. AC cannot be used to counter overdose of ionized substances such as heavy metals, electrolytes, acids and alkali. AC is contraindicated in patients who have ingested corrosives or hydrocarbons, in patients with bowel obstruction/perforation, in patients with a high risk of aspiration and in patients with altered levels of consciousness[4].

Urinary alkalinisation enhances elimination of drugs that are renally excreted and also weak acids. These weak acids are dissociated in alkaline urine preventing passive diffusion and reabsorption from tubules and thereby enhancing excretion. This is mainly important in moderate to severe poisoning with salicylates. Urinary pH should be maintained from 7.5 to 8.5 with sufficient sodium bicarbonate. Plasma electrolytes and acid-base status should be monitored during this process[2].

Extracorporeal elimination with haemodialysis is indicated for substances that have a small volume of distribution, higher water solubility, low protein binding and low molecular weight (salicylates, lithium, theophylline, and alcohol). Charcoal haemoperfusion can be used for overdose with theophylline, barbiturates and carbamazepine.

Risk assessment of any patient presenting with an overdose is a crucial step in initial evaluation and management. It is important to determine the toxic substances involved, the dose and route of ingestion, and time of ingestion. In addition, the risk assessment should inquire about co-ingestions, especially in teenagers, type of exposure (accidental, suicidal, therapeutic or euphoric) and past medical illnesses. The source of information may be the patient himself/herself or family members, relatives and paramedics/EMS. Although confidentiality may be breached, such actions should be taken in the best interest of the patient. Patient's clinical status and observations should be carefully assessed and most importantly, adequately documented.

Next step is to provide the patient with supportive care as required. This may differ according to the patient's clinical condition and the ingested agent. The main forms of these are appropriate fluid therapy, analgesia, sedation, glycaemic control, catheterization, nasogastric decompression, temperature control, and psycho-social support.

The first line investigations include serum electrolytes, blood urea nitrogen, serum creatinine, blood glucose, liver functions and blood gases[1,3]. In special circumstances, where unexplained metabolic acidosis is present, serum osmolality should be measured to detect the osmolar gap. Urine toxicology screen is of limited value in the initial management, as the results may take time and critical interventions cannot be delayed. Cost-effectiveness is also a barrier to its routine use. Negative predictive value of urine toxicology screen is low as it cannot detect all commonly found poisons. The time duration to sample collection also may affect the sensitivity of the results. Quantitative drug levels are mostly beneficial in acetaminophen overdose[3]. Other instances where plasma concentrations are considered include poisoning with salicylates, iron, lithium, digoxin, theophylline, ethylene glycol, methanol, carboxyhaemoglobin, methaemoglobin, and anticonvulsant overdose[3].

Plain abdominal radiographs can be considered to identify radiopaque pills such as iron, lead, mercury, cocaine, calcium, and sustained-release or enteric coated agents[3]. An electrocardiogram should be considered where conduction anomalies are suspected, such as in children with tricyclic antidepressant and oleander toxicity[1].

Antidotes should be considered early where the poison is known and their availability. Purpose of this is to neutralize the effects of the toxin as early as possible or to counteract the effects[1]. Knowledge on the possible adverse effects of the antidotes is paramount and a toxicological opinion should be sought in difficult cases.

Further care, in most cases, requires admission to a hospital ward for observation. Decision to discharge from the emergency department should be considered only after a thorough history and risk assessment has excluded the need for admission. The social circumstances, including parental level of understanding and confidence, and accessibility of emergency care in the event of an unexpected deterioration, are vital facts to consider on decisions made to discharge the patient. Any suspicion of deliberate ingestion that indicates underlying psycho-social issues or an incompatible history suggesting possible non-accidental injury should prompt admission for detailed evaluation[5].

Paracetamol (Acetaminophen)

Paracetamol (PCM) poisoning can be due to accidental exposure, deliberate self-poisoning or repeated supra-therapeutic ingestions. The main factors to consider in the initial risk assessment are drug formulation, dose, time since ingestion, serum concentration, clinical and laboratory features of acute liver injury. A toxic dose of ≥200mg/kg of acute single ingestion is an indication to start treatment with the standard regimen of N-acetyl cysteine. PCM nomogram can only be used in the acute ingestion of immediate release formulations with known time of ingestion (within the first 24 hours). In acute immediate release PCM overdose (elixir form), activated charcoal should be given within 2 hours from ingestion, and if massive ingestion (>30g) or modified release formulation, activated charcoal can be considered up to 4 hours from ingestion. Where facilities are available, PCM level should be checked and plotted in nomogram within 4-8 hours. If the value is on or above the treatment line, N-acetyl cysteine should be commenced. Currently recommended standard regimen is 200mg/kg over 4 hours followed by 100mg/kg over 16 hours. This regimen shows less adverse outcomes compared to the previous three bag regimen. If the initial PCM level is double the nomogram line, a second bag of N-acetyl cysteine 200mg/kg should be used over 16 hours. Two hours prior to completion of N-acetyl cysteine infusion, alanine aminotransferase (ALT) and PCM levels should be repeated. Alanine transaminase (ALT) levels >50 U/L or more than 10% elevation from the baseline and PCM level more than 10mg/L are indications to further continue the second bag of acetylcystine[6].

AC should be considered up to 4 hours following ingestion of modified release PCM ingestion of more than 10g or 200mg/kg. Nomogram is not applicable in these patients and treatment is guided by the PCM levels. When a toxic dose is ingested, N-acetyl cysteine should be started immediately and continued for 20 hours, irrespective of the serum levels. A massive ingestion warrants double dose of N-acetyl cysteine. If the patient has ingested a non-toxic dose, two serum PCM levels should be done 4 hours apart, the first level 4 hours after the ingestion. If either value is above the nomogram, N-acetyl cysteine should be initiated. Continuation of N-acetyl cysteine, depending on ALT levels or PCM levels, should be carried out as in acute ingestion[6].

Liquid PCM preparation ingestions of more than 200mg/kg, warrants a serum PCM level at least 2 hours post-ingestion. N-acetyl cysteine is not indicated if the value is below 150mg/L, and no further levels are needed. However, N-acetyl cysteine should be commenced if both the initial 2-hour level and the repeat level performed after 4 hours are above 150mg/L[6].

Repeated supra-therapeutic dose ingestion (200mg/kg or more over 24 hours, or 300mg/kg or more over 48 hours, or daily therapeutic dose for more than 48 hours with abdominal pain or nausea or vomiting) is an indication for PCM levels and ALT levels. If the PCM level is less than 20mg/L and ALT is less than 50U/lL, no further treatment is indicated. If levels are above that, N-acetyl cysteine should be commenced and investigations should be repeated 8 hours after initial levels. If the repeat ALT is less than 50U/L and PCM level is less than 10mg/L, N-acetyl cysteine can be stopped, unless initial ALT levels are more than 1000 U/L, which is an indication to continue N-acetyl cysteine for at least 20 hours. If the repeat levels are higher, N-acetyl cysteine should be continued till the levels reach below the treatment thresholds[6].

Cessation of N-acetyl cysteine is considered when ALT is decreasing, the International Normalized Ratio (INR) is less than 2.0, and the patient is clinically well. Additionally, modified release preparations, with initial PCM levels greater than double the nomogram line, should have a level of less than 10mg/L[6].

Oleander

Yellow oleander is a naturally occurring cardio-active steroid which has become an important health issue in rural areas of Sri Lanka. Since the time course of toxicity is variable and dose-response relationship is unpredictable, risk assessment on likelihood of toxicity is somewhat complicated. Admission for observation is usually needed, due to possible delayed onset cardiac toxicity for up to 72 hours post-ingestion [7].

Decontamination with AC is indicated for anyone with a possible toxic dose ingestion. Enhanced elimination with MDAC is important. AC should be used with caution, especially in the setting of

unprotected airway or ileus following atropine therapy. There is no established place for extracorporeal elimination[7].

Digoxin assays may cross-react with non-digoxin cardiac glycosides and may be of use in yellow oleander poisoning. However, poor correlation with toxicity limits its use[7]. Hyperkalaemia is a possible complication, although its treatment is complicated. Treatment with insulin-dextrose (50ml of 50% dextrose followed by IV short acting insulin 10 units) is of value. Exogenous calcium may lead to increased intracellular calcium leading to a 'stone heart'. Hence, current practise is to avoid calcium due to lack of evidence[7]. Bradycardia of less than 40 beats per minute can be treated with IV atropine, most likely as a bridging therapy. IV isoproterenol or oral salbutamol too may be of use until temporary pacing can be instituted, in the Sri Lankan set-up. Electrical cardioversion for malignant ventricular arrhythmias is generally ineffective, and should be done using low energies, when indicated. The definitive antidote for hyperkalaemia, renal failure, bradycardia and ventricular tachycardia is anti-digoxin fab[7].

Kerosene

Accidental kerosene oil poisoning is common among children. It is poorly absorbed from the gastrointestinal tract. Possible complications of kerosene ingestion include aspiration, central nervous system (CNS) complications such as lethargy, coma and convulsions, arrhythmias such as atrial fibrillation, ventricular fibrillation, hepatic failure, renal failure, bone marrow toxicity and haemolysis. Skin and mucous membrane contamination cause irritation[8].

Initial management includes removal of the possible source and priority provided for management of patency of the airway. Intubation may be needed in initial resuscitation if signs of severe hypoxia, respiratory distress and reduced level of consciousness are present. Supplemental oxygen should be considered if airway is patent and maintained[8]. Contaminated clothes should be removed. Gastric lavage is avoided[8]. Routine use of corticosteroids is not recommended and routine prescription of antibiotics is controversial[8].

Salicylate

Toxicity due to salicylate ingestion can be due to both acute and chronic exposure. Multi-systemic manifestations are seen with CNS involvement presenting as cerebral oedema, coma, agitation, tinnitus and seizures. Pulmonary involvement may present with hyperventilation and acute lung injury and nausea and vomiting may indicate gastrointestinal system involvement[9]. Management of salicylate toxicity is supportive as no specific antidote is available [9].

In the initial resuscitation, intubation should be considered for patients with altered level of consciousness and features of acute lung injury. IV sodium bicarbonate bolus and pre-oxygenation bag valve mask hyperventilation prior to intubation, are helpful to prevent acidosis and increased salicylate concentrations in the CNS. Maintenance of increased minute ventilation and low partial pressure of carbon dioxide (pCO_2) following intubation, help to achieve the targeted mild alkalaemia of pH 7.45 – 7.50 [9].

Gastrointestinal decontamination with AC and elimination with MDAC are indicated in toxic overdose. Initial salicylate concentration, as well as subsequent levels, should be measured if facilities are available. The Done nomogram has the disadvantage of poor reliability in interpretation of toxicity and therefore is not indicated. Blood pH should be correlated with clinical context to decide on the possible ongoing toxicity rather than the salicylate concentration alone [9].

Enhanced elimination with urine alkalinisation to maintain a pH between 7.5 -8.0 facilitates salicylate excretion. This is indicated in significant toxicity for patients having normal renal functions or in altered renal function in combination with renal replacement therapy. Clinical target is to maintain a urine output of 2-3 ml/kg/hour. Haemodialysis alone is an effective and rapid method of enhanced elimination. This has the added advantage of correction of acid-base and fluid and electrolyte abnormalities. Indications to start haemodialysis include significant CNS involvement, acute lung injury, impaired renal functions not responding to fluid therapy, significant hyperthermia, refractory acidaemia and refractory electrolyte imbalance[9]. Electrolytes disturbances should be corrected with special attention to maintaining high normal potassium levels. Supplementation of glucose is also mandatory to prevent CNS glucose depletion. Volume status should be assessed and euvolaemia is expected to maintain electrolyte as well as acid-base balance [9].

Conclusion

Poisoning in children is a cause of significantly preventable morbidity and mortality. A structured approach to the initial risk assessment of a child with poisoning is paramount. All children with significant toxicity should be assessed carefully and resuscitation, if needed, should be offered with standard ABCDE approach. Specific management depends on the type of poison, ingested dose and availability of resources. Although deliberate ingestion is uncommon in children, all such

children should be referred for psychological evaluation and their families should be offered support.

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