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INFORMATION CENTRE OF IRELAND

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NEWSLETTER

The National Poisons Information Centre will be celebrating our 50th anniversary in 2016. To mark the occasion we will be holding a symposium at the Intensive Care Society of Ireland Annual Scientific Meeting on Saturday 11th June. Further information will be available on our website closer to the time (www.poisons.ie).

Essential oils

Essential oils have been used through the ages as perfumes and medicinal agents and they continue to be popular today as massage oils, aromatherapy oils, and room fragrances. Their widespread use may imply that they are innocuous agents when in fact there are numerous reports in the literature outlining their toxic effects. Last year we received over 200 enquiries about essential oils. 90% of them involved children aged <5 years and most cases involved less than 20mls.

Features can occur from as little as 5-10mls of concentrated oils. There may be ataxia, agitation, drowsiness, delirium, seizures, coma and respiratory depression. Acute renal failure can occur. Fulminant hepatic failure has been reported after ingestion of pennyroyal and clove oil; a 15 month old child had an ALT of 13,000 within 23 hours of ingesting 10mls of clove oil. He was treated with IV acetylcysteine and his hepatic impairment resolved after 4 days.

Aspiration of essential oils can occur if patients vomit and may result in pneumonitis, acute lung injury and acute respiratory distress.

Skin contact with undiluted oils can cause irritation, a burning sensation, and contact dermatitis. Prolonged exposure can result in dermal absorption and systemic toxicity. Exposed skin should be irrigated with copious volumes of water for at least 10-15 minutes.

Observe all patients until 6 hours post-ingestion of any amount. Treatment is symptomatic and supportive. Benzodiazepines may be useful for agitation and seizure activity. Acetylcysteine can be considered for treating hepatic failure although there are no formal studies assessing its usefulness in these cases.

Reference: Janes SE, Price CS, Thomas D. Essential oil poisoning: N-acetylcysteine for eugenol-induced hepatic failure and analysis of a national database. Eur J Pediatr. 2005 Aug;164(8):520-2. Epub 2005 May 14.

Khan AJ, Akhtar RP, Faruqui ZS. Turpentine oil inhalation leading to lung necrosis and empyema in a toddler. Pediatr Emerg Care 2006; 22: 355-357.

DNP (Dinitrophenol)

Over the past 3 years there has been a sharp increase in cases of toxicity from “fat burner” or weight-loss products that contain dinitrophenol (DNP). Dinitrophenol was first used as a diet aid in the early 1930s and quickly become a popular over-the-counter weight loss drug. Increasing reports of adverse effects ultimately lead to a ban by the FDA before it re-emerged as a weight loss product in the 1980s.

Products usually contain 100-250mg of DNP and the “recommended dose” is 1-2 tablets per day. It has a slow onset of action and the long half-life (up to 14 days) means there is a high potential for accumulation. The lowest reported fatal dose is 2.4g in divided doses of 600mg per day (McFee et al 2004). DNP is also absorbed across the skin and systemic toxicity can occur by this route.

MODE OF ACTION

- DNP interferes with the uptake of phosphate into the mitochondria thereby interrupting oxidative phosphorylation and production of ATP.
- It stimulates cellular metabolism of pyruvate to NADH with excess energy inside the mitochondria dissipating as heat.
- It also blocks lipogenic effects of insulin and increases the basal metabolic rate. Increased release of mitochondrial calcium causes muscle contraction and severe hyperthermia.

As a result, metabolic rate, glycolysis and lipolysis are increased and fat stores reduced. There is also a risk of uncontrolled thermogenesis leading to multiorgan failure (particularly with higher doses).

FEATURES Can include fever, dehydration, nausea, vomiting, restlessness, flushed skin, sweating, dizziness, headaches, rapid respiration and tachycardia. Convulsions and muscle rigidity have been reported. There can be rhabdomyolysis and acute renal failure. Yellow staining of the skin is common.

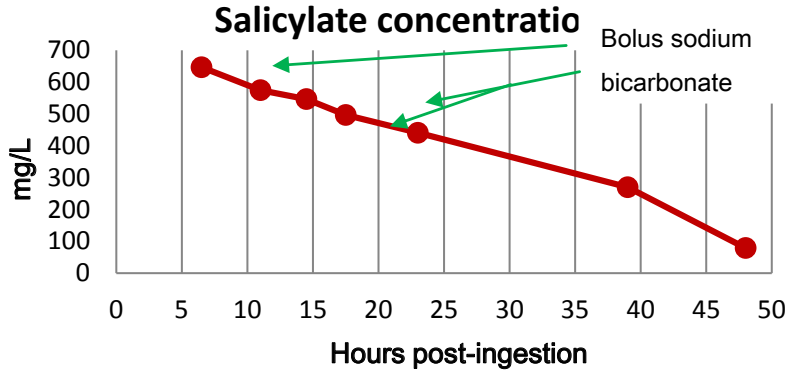
TREATMENT All patients should be observed for at least 12 hours. Monitor body temperature, cardiac rhythm and blood pressure. Treat seizures conventionally. Severe agitation may contribute to hyperthermia so sedate with diazepam if required. Treat hyperthermia with active cooling methods. Dantrolene may be useful to reduce muscle hyperactivity. If temperature cannot be controlled, sedation, paralysis and ventilation may be required.

Further Reading: Grundlingh J, Dargan PL, El-Zanfaly M, Wood DM. 2,4-dinitrophenol (DNP): a weight loss agent with significant acute toxicity and risk of death. J Med Toxciol 2011; 7(3): 205-212.

Kamour A, George N, Gwynnette D, et al. Increasing frequency of severe clinical toxicity after use of 2,4-dinitrophenol in the UK: a report from the National Poisons Information Service. Emerg Med J 2015; 32: 383-386

Salicylate toxicity

CASE REPORT A young girl presented to hospital 6 hours after ingesting a mouthful of oil of wintergreen (methyl salicylate). She was vomiting repeatedly and was drowsy, hypokalaemic (K^+ 3.1), and tachypneic. Blood results on admission showed a high salicylate concentration (647mg/L) and mixed acid-base disturbance: pH 7.41, pCO_2 2.81, Bicarbonate 16, base excess -9.9, lactate 3.5. She was given IV fluids with potassium and a bolus dose of sodium bicarbonate (1mL/Kg of 8.4%). Over the next 12 hours she maintained a good urine output but remained lethargic. Salicylate levels fell slowly and ABGs at 15hrs pi were pH 7.53, pCO_2 1.99, Bicarbonate 16, BE -9. She received 2 further bolus doses of sodium bicarbonate to enhance excretion and her salicylate level fell to 79mg/L at 48hrs pi. She was discharged clinically well on Day 3 post-ingestion.



COMMENT Salicylates cause CNS depression and significant acid-base disturbances in overdose. Stimulation of the respiratory centre leads to hyperventilation and respiratory alkalosis. Salicylates also impair ATP synthesis and interfere with oxidative phosphorylation; the resulting accumulation of lactate and inorganic acids causes an anion gap metabolic acidosis. In severe cases there can be agitation, coma, convulsions, cardiac dysrhythmias, acute non-cardiogenic pulmonary oedema and cerebral oedema. Hypokalaemia is common. Salicylate is rapidly absorbed from the stomach and the half-life in overdose is extended to ~20 hours. Because it is a weak acid, alkalinization of the urine with sodium bicarbonate can enhance excretion of salicylate.

TREATMENT Check acid/base status, U&Es, FBC, INR and blood glucose. Give fluids intravenously to replace fluid loss. Correct hypokalaemia urgently and administer sodium bicarbonate to correct metabolic acidosis. Avoid early intubation; sedation and loss of ventilatory drive in salicylate poisoned patients can result in sudden deterioration. Discuss all cases with an anaesthetist. If plasma salicylate is > 500mg/L in adults or >350mg/L in children, consider urinary alkalinisation to achieve a urine pH of 7.5-8.5; hypokalaemia must be corrected first. Haemodialysis is useful in severe cases. Monitor salicylate levels every 3 hours until there is clinical improvement.

Discuss all serious cases with the National Poisons Information Centre (Tel: 01-8092566).

Low toxicity products in winter

Artificial Coal- Contains ceramic fibre and aluminium silicate, with a coating of iron oxide, and manganese. Ingestion may cause mild nausea and vomiting but treatment is unlikely to be required.

Candles-Candle wax is of low toxicity but ingestion may cause nausea. Large amounts may cause an obstruction.

Silica Gel- Often found in shoe boxes, leather goods, cameras, and electrical goods. It is inert and considered non-toxic.

Mercury (ingestion)-metallic mercury found in thermometers is virtually non-toxic in a single acute ingestion. Symptoms are not expected and no treatment is required.

Cut Flower Food- Mainly sucrose in crystal or liquid form. Not considered toxic and no symptoms are expected. No treatment is required.

Snake bites

An increasing number of people are keeping snakes as pets and despite proper handling there is always a risk of accidental snake bite. No licence is required to keep snakes in Ireland so there is no comprehensive list of species that are kept as pets here. Experts advise us that the most common pets are the North American species (eg vipers).

Snake venom varies depending on species. It can contain a mixture of neurotoxins, haemotoxins, and cytotoxins which are responsible for the systemic features. Approximately half of people who are bitten will not develop features of envenoming but signs of toxicity can be delayed so all patients require observation for at least 24 hours. In the early stages there can be anaphylaxis requiring epinephrine. The venom of some species can cause descending paralysis and respiratory failure so intubation and ventilation may be required. Haematological effects can include systemic bleeding, platelet abnormalities, and haemolysis. Local effects around the bite can include swelling, blistering, necrosis, secondary infection and pain. The limb may appear cyanosed and cold.

Antivenom may be required for systemic features and severe local effects; please contact us for further information if patients are symptomatic. We can discuss possible identification of the snake with a herpetologist and we can assist in sourcing the relevant antivenom.

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