Current management of corrosive, alcohol, and organophosphate poisoning: Pearls and pitfalls

Dear Editor,

Poisoning is a significant global public health issue. Patients who are poisoned often receive an overdue medical evaluation. Several reasons for this exist, as the symptoms that appear are often delayed, and lack of awareness regarding cases of poisoning. This article aims to describe the initial management of the acutely poisoned patient and the potential pitfalls that often occur.

Corrosive poisoning is a chemical reaction that results in tissue injury. Strong acids and bases (pH <2 or >12) are the most frequently used corrosive chemicals. An effective technique to measure the scope of esophageal injury in cases of corrosive poisoning is endoscopy, which should not be performed on hemodynamically unstable patients. The ideal period for endoscopy is within 12–48 h.[1] The use of a nasogastric tube at the beginning of treatment guarantees patency of the esophageal lumen. Esophageal catheterization could be a nidus for contamination; hence inserting a nasogastric can increase gastroesophageal reflux and slow-down mucosal healing.

In developing countries, organophosphate (OP) ingestion cases account for most hospital admissions. The common signs and symptoms of OP poisoning are muscarinic, nicotinic, and central nervous system effects.[2] Testing should be conducted to prove the butyrylcholinesterase function in the plasma or acetylcholinesterase in the blood. The physician can offer treatment based on symptoms in cases without a predetermined baseline level. The poison management of OP includes decontamination (lavage and activated charcoal) and issuing of antidotes with the most crucial part being atropine. Atropine is the only approved treatment for OP poisoning that acts on muscarinic receptors through acetylcholine. Adults receive a starting dose of between 2 and 5 mg IV. Evidence about the use of oximes to treat OP poisoning is inconsistent, and interpretation is difficult. Studies have failed to show any benefit from activated charcoal, even from acute or delayed administration for poisoning.[3]

Acute alcohol poisoning is a dangerous condition caused by excessive alcohol consumption. It is composed of methanol, ethylene glycol, and isopropyl alcohol. Unmetabolized methanol can last up to 30–85 h before complete elimination by the kidneys or the lungs.[4] Methanol poisoning is managed based on objective and historical data. Electrocardiograms, basic metabolic panels, and acetaminophen concentrations should be obtained from patients. The measurement of serum levels of methanol in Indonesia is not routinely done. Moreover, the negative serum level does not entirely reject methanol toxicity because the level of methanol depends to some extent on the time of methanol consumption. Supportive care, fomepizole (antizole, 4-methylpyrazole, or 4MP), ethanol, dialysis, and folate are all therapeutic agents for methanol toxicity. Fomepizole is administered at a dose of 15 mg/kg intravenously within 30 min and continues with 10 mg/kg every 12 h until the methanol level reaches below 30 mg/dL. Empirical data determined the dosage as 10% of intravenous ethanol with a loading amount of 8 mL/kg given intravenously between 30 and 60 min, whereas the maintenance dosage should be 1–2 mL/kg every hour.[5]

Acknowledgment

This report is based on work supported in part by the Article Review Program Tahun 2020 Grant from Universitas Airlangga.

Financial support and sponsorship

This report is based on work supported in part by the Article Review Program Tahun 2020 Grant from Universitas Airlangga.

Conflicts of interest

There are no conflicts of interest.

Nabilah Nabilah[1], Yudith Annisa Ayu Rezkitha[2], Yoshio Yamaoka[3,4,5], Muhammad Miftahussurur[5,6]

[1]Department of Internal Medicine, Faculty of Medicine, Dr. Soetomo Teaching Hospital, Universitas Airlangga, Surabaya, Indonesia, [2]Department of Internal Medicine, Faculty of Medicine, Universitas Muhammadiyah Surabaya, Surabaya, Indonesia, [3]Department of Environmental and Preventive Medicine, Faculty of Medicine, Oita University, Yufu, Japan, [4]Department of Medicine, Gastroenterology and Hepatology Section, Baylor College of Medicine, Houston, TX, USA, [5]Department of Internal Medicine, Division of Gastroentero-Hepatology, Faculty of Medicine, Dr. Soetomo Teaching Hospital, Universitas Airlangga, Surabaya, Indonesia, [6]Helicobacter pylori and Microbiota Study Group, Institute of Tropical Disease, Universitas Airlangga, Surabaya, Indonesia

Address for correspondence:
Prof. Muhammad Miftahussurur, MD., Ph.D, Department of Internal Medicine, Division of Gastroentero-Hepatology, Faculty of Medicine, Dr. Soetomo Teaching Hospital, Universitas Airlangga, Jalan Mayjend Prof. Dr. Moestopo No. 6-8, Surabaya 60286, Indonesia.
E-mail: muhammad-m@fk.unair.ac.id

Submitted: 21-Jul-2022; **Revised:** 26-Sep-2022;
Accepted: 28-Sep-2022; **Published:** 01-Apr-2023

REFERENCES

1. Chirica M, Bonavina L, Kelly MD, Sarfati E, Cattan P. Caustic ingestion. Lancet 2017;389:2041-52.
2. Peter JV, Sudarsan TI, Moran JL. Clinical features of organophosphate poisoning: A review of different classification systems and approaches. Indian J Crit Care Med Medknow Publ 2014;18:735-45.
3. Kristi L, Koenig MF. Activated charcoal has no benefit in organophosphate overdose. NEJM J Watch 2008;2008:1-3.
4. Zakharov S, Pelclova D, Diblik P, Urban P, Kuthan P, Nurieva O, *et al*. Long-term visual damage after acute methanol poisonings: Longitudinal cross-sectional study in 50 patients. Clin Toxicol (Phila) 2015;53:884-92.
5. Pohanka M. Antidotes against methanol poisoning: A review. Mini Rev Med Chem 2019;19:1126-33.

Access this article online

Quick Response Code:

Website: www.jmsjournal.net

DOI: 10.4103/jrms.jrms_635_21

How to cite this article: Nabilah N, Rezkitha YAA, Yamaoka Y, Miftahussurur M. Current management of corrosive, alcohol, and organophosphate poisoning: Pearls and pitfalls. J Res Med Sci 2023;28:20.