

Accidental poisoning with biodiesel preservative biocide

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Abstract

Although biodiesel fuels' use is getting more and more popular, there are only few reports in the literature of poisoning with such agents, and none referring to their preservatives: biocides. We present the management of a 49-year-old Caucasian male who was admitted, after accidental ingestion of biocide solution, in the intensive care unit of a tertiary hospital. In spite of his devastating condition upon arrival to the hospital, he had a remarkable recovery with no local or systemic sequel due to multidisciplinary and early supportive approach of his care. Hippokratia 2014; 18 (2):166-167.

Key words: Biofuels, toxicity

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Introduction

Biodiesel is a cleaner-burning replacement for petroleum diesel fuel. It is nontoxic and biodegradable. Yet, the latter makes it vulnerable to microorganism contamination. Biocides can be added to biofuels to protect them against biological infestation and growth. Unfortunately, information about biocides' toxicity comes either by laboratory or animal studies¹. A rare case of accidental poisoning with such agent in a middle-aged man and its management is presented here.

Case report

A 49-year-old male patient (weight 90kg, height 173cm, BMI 30.1kg/cm²) presented to the local health center, 30 min after accidental ingestion of 30 ml of biodiesel biocide (90% solution) containing 3,3'-Methylenebis[5-methyloxazolidine]. His past medical history included arterial hypertension and diabetes mellitus type II under medication. Surgical history and family history was non-contributory. He had no known drug allergies. After initial general assessment he was referred to the district general hospital where he was admitted to the ENT department. Clinical examination revealed diffuse redness of the oral mucosa and edema of the uvula and arytenoid cartilages. A gastroscopy attempt was unsuccessful due to the excessive oedema of the upper esophagus. Antibiotics, corticosteroids, antihistamine and gastroprotective agents were initiated. Clinical re-evaluation after 4 hours revealed a deterioration of the laryngeal oedema. Emergency induction to anesthesia and intubation were carried out due to endangered airway and the intu-

bated patient was transferred to a tertiary hospital. Upon arrival at the Emergency department, norepinephrine 0.5 mcg/kg/min continuous intravenous infusion (c.iv) was initiated due to refractory hypotension. Further evaluation of the laryngopharynx with flexible fiberscope was not possible due to the massive oedema. Chest X-ray showed air bronchogram of the right lower lobe, electrocardiography displayed sinus bradycardia, while laboratory finding revealed high anion gap metabolic acidosis (pH 7.20, HCO₃⁻ 16.4 mEq/Lt, PaCO₂ 42 mmHg, Lactate 1.4 mmol/Lt, Anion gap 18, Delta/delta ratio 0.6) and neutrophilic leukocytosis (White Blood Count 16.5 G/l with 80% Neutrophils)

The patient was admitted to the Intensive Care Unit (ICU) with SOFA (Sequential Organ Failure Assessment) score 9, and he was initiated sedation (propofol 30 mcg/kg/min c.iv), analgesia (fentanyl 2mcg/kg/min c.iv), mechanical ventilation, corticosteroids regimen (dexamethasone 4mg/iv, bd), total parenteral nutrition, antimicrobial and antiulcer prophylaxis. Epinephrine (3 mcg/min c.iv) was also initiated 3 hours later due to recurrent episodes of bradyarrhythmias. The biomarkers of myocardial injury were within normal range (state them in brackets). No previous similar cases had been reported to the Hellenic Poison Control Center; hence, further consultation with the product's manufacturer company and the European Association of Poison Centers were conducted. On his 2nd day in the ICU, an elective surgical tracheotomy was performed and a neck Computer Tomography (CT) revealed diffuse tissue oedema of the retropharyngeal muscles, hypopharynx, pallatum molle, uvula and the larynx, with fuzzification and infiltration of paravertebral

structures and muscles. CT of the thorax showed thickening of the lower third of esophageal wall with mediastinal "contamination", corrosive foci in the posterior wall of the trachea and possible aspiration at the right lower pulmonary lobe. Transthoracic echocardiography and transcranial Doppler examinations were normal.

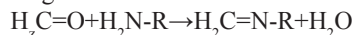
His medical condition gradually improved, the metabolic acidosis resolved, acid base status was normalized and on the 4th day inotrope infusion was discontinued. The following day, sedation was terminated; an early initial neurologic evaluation was performed without any abnormal findings and de-escalation of mechanical ventilatory support was achieved. On the 9th day of his hospitalization, endoscopic reevaluation through the tracheostome revealed reduced oedema of the arytenoid cartilages and normal tracheal anatomy. After uneventful swallowing testing for clear liquid (water) ingestion, oral administration of drugs and nutrition was initiated. Ten days later, a second CT showed full tissue recovery of the injured structures. On the 21st day, the patient was transferred to the ward and a week later he was discharged from the hospital, fully recovered. On a follow up visit 3 months later, the patient had returned back to his previous daily life, without any sequels.

Discussion

3,3'-Methylenebis[5-methyloxazolidine] (C₉H₁₈N₂O₂) is a light yellow liquid aliphatic amine used like diesel biocide^{2,3}. Safety standards for manufacturing, distributing and using these agents are very strict, thus there is scarce literature about toxicity in humans³. In most cases toxicity information comes from animal studies. Therefore, consultation with toxicologists and manufacturing companies is essential when dealing with a similar poisoning.

In the case presented, therapeutic plan was based on the fact that this particular biocide degrades to formaldehyde. Unfortunately, there is no literature about the time table and the rate of this process in human body.

Formaldehyde is a physiological intermediary metabolite in mammals and plays an important role in the 1-carbon pool as methylene tetrahydrofolate. It is almost completely bound to amino functions of proteins, thereby forming Schiff's bases:



Due to the strong irritating effect of formaldehyde and its low threshold of olfactory detection (0.5 to 1 ppm) accidental ingestion of formalin occurs rarely and ingestion of as little as 30 mL (1 oz) of a solution containing 37% formaldehyde has been reported to cause death in adults⁴⁻⁸. Ingestion may cause corrosive injury

to the gastrointestinal mucosa which is most pronounced in the pharyngeal mucosa, epiglottis and esophagus. Its systemic effects include metabolic acidosis, central nervous system depression and coma, respiratory distress, tachyarrhythmias and renal failure^{8,9}. No specific antidote is currently available.

In the aforementioned case, early intubation and early tracheostomy was the base of the therapy. Arrhythmia's treatment was also a key element, as its nature (bradyarrhythmia) was the opposite than expected. This could be explained either by an increased sensibility of the autonomic nervous system to analgesia-sedation regime or by the stimulation of vagus nerve due to the regional oedema (mass effect). The evolution of his clinical condition and the normalization of cardiac rhythm along with the resolving of tissue oedema support the second hypothesis. Finally, the early engagement of a toxicologist is considered the sine qua non of a carefully planned therapeutic strategy.

Conflict of interest

None declared.

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