Extraordinary cases of ordinary ("harmless") products

4de Lage Landen Symposium Intoxicaties



Dr. Geert Verstegen Belgian Poison Centre

09 June 2017, Ghent

Disclosure belangen spreker

| (potentiële) belangenverstrengeling | Geen |
|---|--|
| Voor bijeenkomst mogelijk relevante relaties met bedrijven | Bedrijfsnamen |
| Sponsoring of onderzoeksgeld Honorarium of andere (financiële) vergoeding Aandeelhouder Andere relatie, namelijk | geen geen geen geen |

Don't miss the extraordinary case between the ordinary



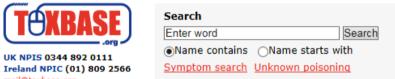
"Alle Dinge sind Gift, und nichts ist ohne Gift; allein die Dosis machts, daß ein Ding kein Gift sei."





Ethyl acetate







A service commissioned by Public Health England (PHE) on behalf of the UK Health Departments

General Info Specialist areas Chemicals Exposure in pregnancy Poisons A-Z Dosage Calculator Updated 05/2013 Printable version Ethyl acetate

The information on TOXBASE® requires expert clinical interpretation (ideally users should complete the TOXBASE® E-Learning modules; click here) and, therefore, should only be used by clinically trained medical/nursing professionals, who are responsible for the correct interpretation of the relevant clinical case history. In severe or complex cases, including multiple ingestions, and people with significant comorbidity we recommend that you discuss your case with your poisons service: in the UK NPIS 0344 892 0111, in Ireland NPIC (01) 809 2566. If your patient is pregnant please telephone the UK Teratology Information Service 0344 892 0909.

TOXBASE® entries should not be used as patient information sheets.

Type of Product

UK NPIS 0344 892 0111

mail@toxbase.org

Colourless liquid with a fruity odour. Used as an artificial flavouring agent in pharmaceuticals and foodstuffs. Solvent and extracting agent.

Synonyms

Acetic acid ethyl ester, acetic ester, acetic ether, acetidin, acetoxyethane, ethyl acetic ester, ethyl ethanoate, vinegar naphtha

UN 1173 CAS 141-78-6

Toxicity

Low by inhalation and ingestion. Irritating to mucous membranes. Prolonged skin contact may cause irritation. Prolonged eye contact with the vapour has caused corneal clouding. Rapidly absorbed and metabolised to acetic acid and ethanol. CNS depression could occur following large exposures.

Ethyl acetate: case

- A 78-year-old woman intentionally ingested 100 ml of nail polish remover containing 85% ethyl acetate and minor percentages of methylpyrrolidone, dimethylsuccinate, dimethylglutarate and dimethyladipate.
- On admittance, one hour later, she presented cardiovascular shock with pronounced bradycardia (25 beats/minute) and blood pressure of 45/30 mm Hg. Blood gas results under 40% FiO₂ were pH 6.39, pCO₂ 25 mm Hg, PO₂ 440 mm Hg, bicarbonate 6 mEq/L and a anion gap of 45 mEq/L.
- Serum ethanol was 1.2 g/L, methanol 0.15 g/l and osmolality 352 mOsm/kg. The patient was intubated, transferred to the ICU and hemodialysis was started for 3 hours.

Ethyl acetate: case

- After dialysis arterial pH was 7.40, bicarbonate 23 mEq/l with an anion gap of 29 mEq/l.
- On day 2, a marked toxic hepatic injury became apparent (aspartate aminotransferase 6339 U/L, alanine aminotransferase ALT 3733 U/L). Lactate normalised on day 4 but there was persistant hemodynamic instability. She was extubated on day 8, transferred to a medical ward on day 11 and recovered without sequellae.

Ethyl acetate: biochemistry

100 mL x 0.85 x 0.90 = 76.5 gram ethyl acetate (0.87 moles)

After metabolisation: 0,87 moles of acetic acid ~ 52,24 grams.

This corresponds to 870 mL of vinegar 6°!!!

Probably released in < 30 minutes*

^{*}Metabolism of ethyl acetate in the rat: hydrolysis of ethyl alcohol in vitro and in vivo. Toxicol Appl Pharmacol. 1975 Nov;34(2):309-13. Gallaher EJ, Loomis TA.

Ethyl acetate: discussion

- From animal data it is known that it is easily absorbed orally and can be hydrolysed by liver and plasma esterases into ethanol and acetic acid (2).
- Based on the clinical course, ethyl acetate was apparently rapidly absorbed and readily hydrolysed with a pronounced acidosis accompanied by a cardiovascular shock.
- There was severe toxic hepatic injury which has been described in animals (2) but not in humans. In the literature we found one report of fatal ethyl acetate poisoning (1). The low ratios of ethyl acetate to ethanol in post mortem tissue samples confirmed the rapid biotransformation of ethyl acetate which fits with our observations.

Conclusion:

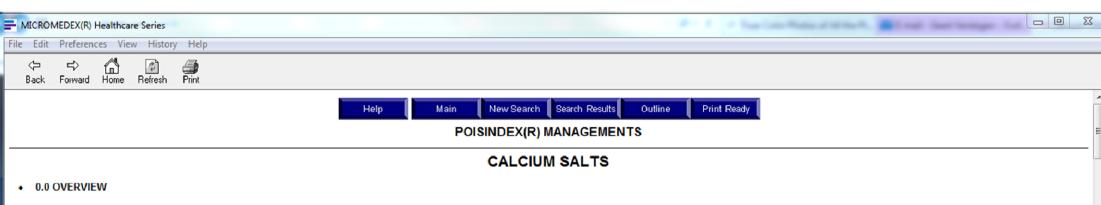
- Ingestion of small amounts of nail polish removers containing ethyl acetate is a common accident and is usually considered not toxic.
- However, if ingested in large amounts it can lead to rapid and massive release of acetic acid with a life threatening acidosis and hepatocellular damage.

^{1.} Coopman V, Cordonnier J, De Meyere C. Fatal workplace accident involving ethyl acetate: a distribution study. Forensic Sci Int 2005; 154:92-95.
2. Specifications for the identity and purity of food additives and their toxicological evaluation: some flavouring substances and non-nutritive sweetening agents. FAO Nutr Meet Rep Ser 1968; 44:1-18.



Calcium chloride





- 0.1 LIFE SUPPORT
- A) This overview assumes that basic life support measures have been instituted.

0.2 CLINICAL EFFECTS

- 0.2.1 SUMMARY OF EXPOSURE
 - A) USES: Calcium carbonate is used medically as an antacid. Calcium citrate and calcium carbonate are used as dietary supplements. Calcium chloride and gluconate are used medically for treatment of hypocalcemia, hyperkalemia, and hypermagnesemia. Calcium salts are used in manufacturing, mining and other industries.
 - B) PHARMACOLOGY: Calcium is a cation that is necessary for many physiological activities, some of which are poorly understood. It is essential for the normal functioning of organ systems including the muscles, nervous system, and cardiac function.
 - C) TOXICOLOGY: Hypercalcemia may cause abdominal pain, delirium and renal stones. Prolonged ingestion of alkaline calcium salts may cause metabolic alkalosis and hypercalcemia (the "milk-alkali syndrome").
 - D) EPIDEMIOLOGY: Exposure to calcium containing antacids is common, but significant toxicity is very rare.
 - E) WITH THERAPEUTIC USE
 - 1) ADVERSE EFFECTS: Calcium supplements may cause Gl upset or constipation. Patients with renal insufficiency may develop hypercalcemia. Extravasation of calcium chloride salts may cause local irritation or necrosis. Rapid intravenous administration of calcium salts may cause hypotension, bradycardia, syncope, and cardiac dysrhythmias.
 - F) WITH POISONING/EXPOSURE
 - 1) MILD TO MODERATE TOXICITY: Acute calcium poisoning is rare, and almost exclusively from intravenous administration. Symptoms of hypercalcemia include lethargy, muscle weakness, vomiting, nausea and constipation. Some calcium salts cause gastrointestinal irritation. Minor skin exposure to caustic calcium salts may cause dermal irritation.
 - 2) SEVERE TOXICITY: Life threatening manifestations are very rare and include complications from altered mental status such as aspiration pneumonia, and cardiac dysrhythmias.

Calcium chloride: case

- A thirsty 74-year-old man with antecedents of Chronic Obstructive Pulmonary Disease mistakenly ingests a glassful of calcium chloride solution collected from a humidity absorber device. The solution was stored in a mineral water bottle.
- Half-an-hour after ingestion, the patient complains of vomiting, diarrhoea and abdominal pain. He becomes dyspneic and he is admitted at the emergency department one hour after the onset of the symptoms. Physical examination reveals a GCS 15/15, blood pressure 70/32, heart rate 140/min regular; wheezing and bilateral crackles at lung bases; abdominal tenderness.
- Laboratory evaluation shows an acidosis pH 7.09, HCO₃⁻ 13.4 mmol/L, lactate 4.7 mEq/L, pCO₂ 49.3 mmHg, pO₂ 70.9 mmHg, saturation of oxygen 86.5%. The calcaemia is 27.1 mg/dL (nl. 8,6-10,5) ~ 6.8 mmol/L. Renal function is normal.

Calcium chloride: case

- The patient is transferred to the intensive care unit. He becomes unconscious and is intubated and ventilated. Despite intensive hydration and diuresis, he dies from intractable cardiac arrest 4 hours after ingestion.
- A calcium concentration of 220 g/L was measured in the solution collected from a humidity absorber similar of that involved in the accident → intake of ± 40 grams of CaCl₂.

Calcium chloride: discussion

- The risk of hypercalcaemia should be taken into account when evaluating a patient with a history of unusual, large Ca²⁺ ingestion.
- Safety data sheets and major textbooks insist on the irritant properties of CaCl₂ but do not (always) consider the risk of hypercalcaemia after ingestion.

Calcium Chloride New 8/2014 Printable version

TOXBASE® entries should not be used as patient information sheets.

Type of Product

Chemical agent used as a moisture absorber; found in dehumidifiers and food packaging.

Ingredients

Calcium chloride

Toxicity

Unlikely to be significantly toxic following accidental ingestion. There is a theoretical risk of hypercalcaemia following ingestion; however hypercalcaemia is unlikely to develop, even in children, following ingestion of small amounts.

ALERT BOX

All those who have been exposed to this product as a result of self-harm should be referred for assessment.

All patients who are symptomatic should be referred for medical assessment.

If this product was ingested accidentally by an adult or a child and the patient has no new symptoms since the time of ingestion, the patient does NOT need to be referred for medical assessment. Patients should be advised to seek medical attention if symptoms develop.

Features

Features are unlikely to arise following acute ingestion.

Management

Unlikely to be required.

A small glass of water (or milk or juice) may be given if there is mild gastrointestinal upset.

In symptomatic patients check serum calcium and U&Es.

"Alle Dinge sind Gift, und nichts ist ohne Gift; allein die Dosis machts, daß ein Ding kein Gift sei."







Bas-en-Basset : le quinquagénaire qui s'était étouffé en mangeant un steak frites n'a pas survécu

Vu 11200 fois | Le 28/05/2013 à 23:13 | mis à jour le 29/05/2013 à 12:05 | Réagir (5)



Dans notre édition de lundi, nous évoquions un accident peu banal : un homme, âgé de 58 ans, s'était étouffé avec deux morceaux de viande, samedi soir, alors qu'il consommait un steak frites dans une pizzeria de Bas-en-Basset.

Secouru par ses amis, puis par les sapeurs-pompiers et le SAMU42, il avait été évacué dans un état très préoccupant à l'Hôpital Nord de Saint-Étienne.

On a appris, mardi, que la victime est décédée dans la journée de dimanche.

Denture cleaner tablet



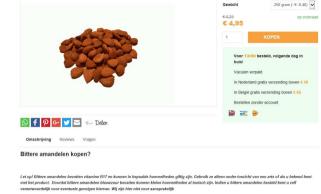
- A 95-yr-old lady swallows 2 denture cleaning tablets, containing sodium bicarbonate, citric acid and « Caroat » (= triple salts of potassium peroxomonosulphate, potassium hydrogensulphate and potassium sulphate; will result in pH ~ 2 + generation of H₂O₂).
- 4 hours later, she is hospitalised in critical condition, with major dyspnoea, peripheral cyanosis, major oedema of tongue and mouth resulting in almost complete obstruction of the oropharynx. A portion of unsolubilised tablet is found in the pharynx. A successful intubation is done and ventilation is started resulting in better oxygenation. Infiltrates are visible on RX in both lungs.
- Transfer to intensive care unit in other hospital where she dies on the next day.

Denture cleaner tablet

- Unusual exposition:
 - 2 dry tablets < > normally 1 solubilised tablet
 - Dry: locally large impact (pH + H₂O₂)
 - Aspiration versus normally swallowed

Amygdalin

Bittere amandelen kopen?



geloven dat deze noot door de aanwezigheid v en ingenomen. Driemaal daags twee bittere mygdatine. Deze stof heeft behalve medicinale hoofd, algehele zwakte, niet meer op benen de stoelgang lopen.

Let op! Bittere amandelen bevatten vitamine B17 en kunnen in bepaalde hoeveelheden giftig zijn. Gebruik ze alleen onder toezicht van een arts of als u bekend bent met het product. Doordat bittere amandelen blauwzuur bevatten kunnen kleine hoeveelheden al toxisch zijn. Indien u bittere amandelen besteld bent u zelf verantwoordelijk voor eventuele gevolgen hiervan. Wij zijn hier niet voor aansprakelijk

Deze amandel is niet zomaar een nootje om te eten, maar staat bekend om zijn medicinale werking. Zo doet men geloven dat deze noot door de aanwezigheid van B17 kanker kan helpen voorkomen. Dit is echter niet bewezen. Bittere amandelen moeten beslist niet te veel worden ingenomen. Driemaal daags twee bittere amandelen en dus maximaal 6 per dag wordt als maximum aangeraden wegens de aanwezigheid van laetrile of amygdaline. Deze stof heeft behalve medicinale eigenschappen ook bijwerkingen als er te veel van wordt gebruikt. De bijwerkingen variëren van licht gevoel in het hoofd, algehele zwakte, niet meer op benen kunnen staan, hartkloppingen, misselijkheid en buikkrampen. In zware gevallen kan men flauw vallen en laat men de stoelgang lopen.

Land van herkomst: Spanje

Published cases: amygdalin

Clinical Toxicology (2010) 48, 574–576 Copyright © Informa UK, Ltd. ISSN: 1556-3650 print / 1556-9519 online DOI: 10.3109/15563650.2010.492351

informa

LETTERS TO THE EDITOR

Child cyanide poisoning after ingestion of bitter almonds

To the Editor:

Many species in the Rosaceae family are cultivated and eaten as culinary fruits throughout the world. Although the outer fleshy part of fruits such as apricots, peaches, and numerous types of plums is indeed delicious, the stone contains toxic cyanogenic heterosides. The best known of these poisons is amygdalin. Hydrolysis of these molecules in the digestive tract of mammals can release hydrocyanic acid that can lead to cyanide poisoning.1 Several cases of severe poisoning have been reported after the ingestion of apricot stones from Prunus armeniaca that appears to be the most toxic species.23 Another widely cultivated drupe in temperate climates is the almond. There are several varieties of almond trees (Prunus amygdalus). The most common variety, P. amygdalus dulcis, produces small quantities of cyanogenic heterosides. However, the bitter almond variety (P. anvedalus amara) produces fruit with stones containing high amounts of amygdalin as apricot pits. There have been few reports describing severe poisonings after the ingestion of bitter almonds.4-6 The purpose of this report is to describe a case of severe poisoning after the ingestion of bitter almonds with confirmed ingested dose and measured cyanide blood level.

Determination of cyanides in blood

Cyanide ions were assayed by high-performance liquid chromatography linked to a fluorimeter (HPLC-Fluo). Based on the method previously described by Chinaka, 7 we developed and fully validated a rapid assay (less than 2 h), which can be performed using a small-volume whole blood sample. Briefly, a 50-μL blood sample is deproteinized by the addition of 2.5 mL of a methanol-water solution. Cyanide ions potentially present in the supernatant were derivatized by the addition of a 100-μL solution containing taurine (5 mmol/L) and naphthalene dialdehyde (1 mmol/L) (1 : 1, v/v) in the dark. Chromatographic separation (Surveyor Thermo Fisher Scientific, Waltham, MA, USA) was performed on a UP BP2 column (150 × 1 mm; 5 μm) (Interchim). The

Case report

The patient is a 30-month-old girl weighing 14 kg with no particular medical history whose grandfather gave her a bitter almond to taste in December 2009. The almond had been picked directly from an almond tree near Beziers in southern France (Languedoc region) and shelled the night before. The child liked the taste and asked for more. According to the statements by both the mother and grandfather, she ate a total of five bitter almonds. Between 10 and 15 min after ingestion, the child became pale and hypotonic with difficulty holding her head up. The concerned parents decided to take the child to the emergency room and called the emergency helpline. During the trip in the car, the girl experienced fluctuating consciousness followed by general seizures. On the road, the family met the emergency team that observed the following findings: miosis. obnubilation, rightward fixation of eyes, and tachycardia. These clinical findings were compatible with a post-seizure state. Intravenous diazepam (5 mg) was prescribed and the child was taken to the local hospital emergency room. On arrival at the emergency room (1 h and 30 min after ingestion) and after consulting the poison control center, a diagnosis of cyanide poisoning was evoked (clinical feature at this moment: drowsiness, hand tremor, hypersialorrhea, blood pressure 105/55 mmHg). After collecting a blood sample, clonazepam (0.3 mg) was administered by the direct intravenous route to prevent further seizure followed by hydroxocobalamin (70 mg/kg) in a 15-min perfusion (infusion beginning 1 h 50 min after ingestion). At the end of perfusion, neurological findings were normal and the child rapidly recovered consciousness. She was transferred overnight to the children's intensive care unit at the University Hospital Center where brain scan was carried out under sedation with midazolam showed no sequelae. During surveillance, no clinical or laboratory abnormalities were found (no acidosis observed at the intensive care unit - arterial blood gas pH 7.39 and plasmatic bicarbonate level 24 mmol/L - but these biological analyses were performed between 30 and 50 min after the antidote perfusion). The delayed biological results do not allow excluding the development of metabolic acidosis before specific treatment. The child was discharged the next morning with no additional treatment. Cvanide measurement of the pretreatment sample of whole blood based on high-performance liquid chromatography linked to a fluorimeter showed a concentration of 2.33 mg/L (toxic level > 0.5).

Acute Cyanide Toxicity Caused by Apricot Kernel Ingestion

Jeffrey R Suchard, MD Kevin L Wallace, MD Richard D Gerkin, MD A 41-year-old woman ingested apricot kernels purchased at a health food store and became weak and dyspneic within 20 minutes. The patient was comatose and hypothermic on presentation but responded promptly to antidotal therapy for cyanide poisoning. She was later treated with a continuous thiosulfate infusion for persistent metabolic acidosis. This is the first reported case of cyanide toxicity from apricot kernel ingestion in the United States since 1979.

[Suchard JR, Wallace KL, Gerkin RD: Acute cyanide toxicity caused by apricot kernel ingestion. *Ann Emerg Med* December 1998:32:742-744.]

INTRODUCTION

Ingestion of foods containing cyanogenic glycosides may result in cyanide poisoning. The last report in the United States of human cyanide poisoning due to apricot kernel ingestion was published in 1979. Despite a warning issued by the authors of that report and subsequent investigation by the US Food and Drug Administration (FDA), foods containing cyanogenic glycosides continue to be marketed for presumed healthful effects. We report a case of acute cyanide poisoning occurring after ingestion of apricot kernels purchased at a health food store.

CASE REPORT

A previously healthy, nonsmoking, 41-year-old woman chewed and swallowed approximately 30 apricot kernels (estimated total, 15 g) from an 8-ounce bag purchased as a health food. Within 20 minutes she developed generalized weakness and numbness. She telephoned a friend and

Published cases: ethanol enema

Hindawi Publishing Corporation Case Reports in Emergency Medicine Volume 2014, Article ID 191237, 3 pages http://dx.doi.org/10.1155/2014/191237





Case Report

Self-Administered Ethanol Enema Causing Accidental Death

Thomas Peterson, Landen Rentmeester, Bryan S. Judge, Stephen D. Cohle, and Jeffrey S. Jones

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Excessive ethanol consumption is a leading preventable cause of death in the United States. Much of the harm from ethanol comes from those who engage in excessive or hazardous drinking. Rectal absorption of ethanol bypasses the first pass metabolic effect, allowing for a higher concentration of blood ethanol to occur for a given volume of solution and, consequently, greater potential for central nervous system depression. However, accidental death is extremely rare with rectal administration. This case report describes an individual with klismaphilia whose death resulted from acute ethanol intoxication by rectal absorption of a wine enema.

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Don't miss the extraordinary case between the ordinary

- Sheep with 5 legs are rare, but they do exist. You see these cases once in a lifetime, but you should recognise them.
- « Unusual » cases of « harmless » products:
 - Large quantities (suicidal ingestions!)
 - Unusual way (e.g. aspiration, rectal)
 - High concentration
 - Long duration of exposition
 - ...
- Don't forget to alert the Poison Centre if you have an unusual case.



A dog has four legs, right?

