



October 2005

The NY State Poison Centers

# TOXICOLOGY

A Quarterly Publication • Vol. X No. 4

LETTER

## Toxicology Advice Centers • •

**Administrative Phone Numbers** - To obtain a consult in your area, call 1.800.222.1222.

**Western New York Poison Center (WNY)**

716.878.7871 • <http://wnypoison.org>

**Ruth A. Lawrence Poison and Drug Information Center  
Serving the Finger Lakes Region (FL)**

585.273.4155 • [www.FingerLakesPoison.org](http://www.FingerLakesPoison.org)

**Upstate New York Poison Center (UNY)**

315.464.7078 • [www.cnypoison.org](http://www.cnypoison.org)

**New York City Poison Control Center (NYC)**

212.447.8152

**Long Island Poison & Drug Info Center (LI)**

516.663.4574 • [www.LIRPDIC.org](http://www.LIRPDIC.org)

## Tox Trivia • •

1. The trade name of FDA approved intravenous NAC?
2. Where did our patient get exposed to diethylene glycol?
3. Can oral NAC be given intravenously?

## NYPC Tidbits • •

1. This toxin was found in elixir of sulfanilamide and toxicity related led to the passage of the Food, Drug and Cosmetic Safety Act of 1938.
2. This toxic effect appears to be permanent after diethylene glycol poisoning.
3. Ethylene glycol levels will be positive after diethylene glycol exposure? (*True/False*)

Answers on page 7

## Program Announcements • •

**FL:** Monthly conference: every 4 weeks on Thursdays starting Jan 27th (11 am to noon), and every 4 weeks on Tuesdays starting Feb 1st, 2005 (10 am-11 am).

**UNY:** Please mark your calendars for our Ninth Annual Toxicology Teaching Day to be held on November 2, 2005 at the Sheraton Hotel and Conference Center in Syracuse. A brochure can be obtained by e-mailing [fosterl@upstate.edu](mailto:fosterl@upstate.edu)

**NYC:** Consultants Case Conference • The first Thursday of the Month from 2-4pm

**LI:** Pre-Registration is required. Please contact Mr. Denis Jao at 516-663-2650 to register.

Both Telephone and Televideo broadcasts will be available.

*Target Audience:* Physicians, Pharmacists, Nurses, Physician-Assistants, Laboratory technicians, EMS staff, medical/nursing/pharmacy students and other healthcare professionals.

*Location:* New Life Conference Rooms B&C  
Winthrop-University Hospital  
259 First Street

Mineola, Long Island, New York 11501

*Times for ALL Conferences are:* 12:15 PM-1:45 PM

### LI (cont'd):

*Tuesday, September 27, 2005:* SUBSTANCE ABUSE

Mark Su, MD, ABMT, ABEM

Assistant Professor of Emergency Medicine

Director of Medical Toxicology

State University of New York - Downstate Medical Center/  
Kings County Hospital, Brooklyn, NY

*Monday, October 24, 2005:* BETA BLOCKER

TOXICOLOGY: RECOGNITION AND MANAGEMENT

Gerold Brody, MD, ABEM

Chairman Ambulatory Medicine

Winthrop University Hospital

Mineola, NY

*Tuesday, November 29, 2005:* PERCUTANEOUS

ABSORPTION AND DISTRIBUTION OF METHANOL  
IN A HOMICIDE

Joseph Avella, MS, Ph.D, FTS, ABFT

Forensic Scientist II

Sidney B Weinburg Center for Forensic Sciences

Hauppauge, NY

**Please call administrative telephone numbers for more information.**

## **Injectable Products made by Central Admixture Pharmacy Service (CAPS) of Lanham, Maryland**

FDA is notifying healthcare professionals and hospitals about a product recall involving all injectable products manufactured by Central Admixture Pharmacy Services, Inc. of Lanham, Maryland (CAPS) due to concerns regarding the sterility of these injectable products. *September 17, 2005*

## **NovoLog Mix 70/30 (70% insulin aspart protamine suspension and 30% insulin aspart injection, [rDNA origin]) NovoLog (insulin aspart [rDNA origin] injection)**

To facilitate the dispensing of the correct product, color branded labeling has been introduced for NovoLog Mix 70/30, a premixed insulin analog, and NovoLog, a rapid-acting insulin analog. *August 26, 2005*

## **Erbix (cetuximab)**

The WARNINGS and DOSAGE AND ADMINISTRATION sections have been revised to notify healthcare providers about specific recommendations on observation periods following Erbix infusion. In addition, the PRECAUTIONS and ADVERSE REACTIONS sections have been revised to discuss results seen in Erbix clinical trials regarding an increased incidence of hypomagnesemia. *September 13, 2005*

## **Herceptin (trastuzumab)**

Genentech and FDA notified healthcare professionals of updated cardiotoxicity information related to the use of Herceptin (trastuzumab), obtained from the National Surgical Adjuvant Breast and Bowel Project (NSABP) study (B-31), demonstrating a significant increase in cardiotoxicity in patients who were randomized to the Herceptin-containing arm as compared to patients who received chemotherapy alone. *August 2005*

## **Alcohol-Free Mouthwash and Hygiene Kits by Medline**

Medline and FDA notified healthcare professionals about a nationwide recall of Alcohol-Free Mouthwash and Hygiene Kits containing mouthwash because of potential contamination with *Burkholderia cepacia*. *August 29, 2005*

## **Trypan Blue 0.06% Ophthalmic Solution**

Custom RX Compounding Pharmacy and FDA notified ophthalmologists, other healthcare professionals, and consumers about a nationwide recall of Trypan Blue 0.06% Ophthalmic Solution, intended for use in the eyes during cataract surgery, because it may be contaminated with *Pseudomonas aeruginosa*. *August 26, 2005*

## **Isotretinoin - Accutane and generic isotretinoin**

FDA notified healthcare professionals and patients of the approval of a strengthened risk management program for Accutane and generic isotretinoin. *August 12, 2005*

## **Perrigo Infants' Oral Drops Containing Enclosed Syringe**

Perrigo and FDA notified healthcare professionals and consumers of the recall of all lots of concentrated infants' drops that are packaged with a dosing syringe bearing only a "1.6 mL" mark containing: acetaminophen, acetaminophen, dextromethorphan HBr, and pseudoephedrine HCl, or dextromethorphan HBr, and pseudoephedrine HCl. *August 01, 2005*

## **Counterfeit "Lipitor" Sold in the United Kingdom**

FDA alerted U.S. residents to the recent recall of a batch of counterfeit "Lipitor" (atorvastatin) sold in the United Kingdom (U.K.). *July 29, 2005*

## **Raptiva (efalizumab)**

Healthcare professionals and patients were informed about reports of immune-mediated hemolytic anemia and warnings regarding postmarketing reports of thrombocytopenia and serious infections including necrotizing fasciitis, tuberculous pneumonia, bacterial sepsis with seeding of distant sites, severe pneumonia with neutropenia, and worsening of infection. *July 15, 2005*

## **Mifeprex (mifepristone)**

Danco Laboratories and FDA have revised the BOXED WARNING and WARNINGS sections of the Prescribing Information, the Medication Guide and Patient Agreement to inform healthcare professionals of four cases of septic deaths in the United States in women following medical abortion with mifepristone (Mifeprex) and misoprostol. *July 19, 2005*

## **Natrecor (nesiritide)**

Scios and FDA notified healthcare professionals about the recommendations of an expert panel of cardiology and heart failure clinicians with regard to NATRECOR (nesiritide). The panel provided a consensus statement an educational campaign to ensure that clinicians understand when the use of NATRECOR is appropriate and when it is not appropriate. *July 13, 2005*

Continued on page 3

## **Fentanyl Transdermal (Skin) Patch**

FDA issued a public health advisory to alert health care professionals, patients and their caregivers of reports of death and other serious side effects from overdoses of fentanyl in patients using fentanyl transdermal (skin) patches for pain control. *July 15, 2005*

## **Palladone (hydromorphone hydrochloride)**

FDA issued a public health advisory to notify health care professionals and consumers that the sponsor of Palladone, Purdue Pharma, has agreed to suspend sales and marketing of Palladone (hydromorphone hydrochloride, extended release capsules), a potent narcotic painkiller, because of the potential for severe side effects if Palladone is taken with alcohol. *July 13, 2005*

## **Duragesic (fentanyl transdermal system)**

Janssen and FDA notified healthcare professionals of changes to the BOXED WARNING/WARNINGS, CONTRAINDICATIONS, PRECAUTIONS, and DOSAGE AND ADMINISTRATION sections of the prescribing information for Duragesic. *June 2005*

## **Cialis (Tadalafil)Levitra (Vardenafil hydrochloride)Viagra (Sildenafil citrate)**

FDA notified healthcare professionals of updated labeling for Cialis, Levitra and Viagra to reflect a small number of post-marketing reports of sudden vision loss, attributed to NAION (non arteritic ischemic optic neuropathy).

## **Liqiang 4 Dietary Supplement Capsules**

FDA notified consumers and healthcare professionals about the risks of taking Liqiang 4 Dietary Supplement Capsules because they contain glyburide – a drug that could have serious, life-threatening consequences in some people. *July 01, 2005*

## **Public Health Advisory: Suicidality in Adults Being Treated with Antidepressant Medications**

FDA notified healthcare professionals about the availability of updated Healthcare Professional and Patient Information Sheets for antidepressant medications that were the subject of a June 30, 2005 Public Health Advisory issued about the risk of suicidality (suicidal thinking or behavior). *July 01, 2005*

## **Iressa (gefitinib)**

AstraZeneca and FDA notified healthcare professionals of new approved labeling for Iressa that states the medicine should be used only in cancer patients who have already taken the medicine and whose doctor believes it is helping them. *June 17, 2005*



## **Quality Care Products L.L.C./Able Laboratories Inc. Drugs**

Quality Care Products, LLC, a federally licensed drug re-packer, and FDA notified healthcare professionals of a nationwide recall because of the FDA's serious concerns that they were not produced according to quality assurance standards. *June 15, 2005*

## **COX-2 Selective (includes Bextra, Celebrex, and Vioxx) and Non-Selective Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)**

FDA has requested that sponsors of all non-steroidal anti-inflammatory drugs (NSAID) make labeling changes to their products to include a boxed warning, highlighting the potential for increased risk of cardiovascular (CV) events and the well described, serious, potential life-threatening gastrointestinal (GI) bleeding associated with their use. *June 15, 2005.*

## **Children's Tylenol Meltaways - 80 Mg, Children's Tylenol Softchews - 80 Mg Jr. Tylenol Meltaways - 160 Mg**

McNeil Specialty Pharmaceuticals and FDA notified consumers and healthcare professionals about a nationwide recall of all lots and all flavors of Children's TYLENOL Meltaways 80 mg, Children's TYLENOL SoftChews 80mg, and Junior TYLENOL Meltaways 160mg. The recall addresses issues regarding the design of the blister package, information on the package, and bottle cartons for the products that may be confusing and lead to improper dosing, including overdosing. *June 03, 2005.*

# Diethylene Glycol and Neurologic Toxicity

## Case Report:

Contributed by: Michael Holland, MD, Medical Consultant, Upstate New York Poison Center, Syracuse, NY

**Case Report:** A 27 year old male with past psychiatric problems and multiple prior overdoses, presented to the Emergency Department (ED) complaining of nausea and vomiting approximately 24 hours after ingesting 16 ounces of wallpaper stripper containing an unknown percentage of diethylene glycol. He had consumed it for its presumed intoxicating effects. Upon arrival, he was awake and alert with a normal physical and neurologic examination. His heart rate was 56 beats per minute, blood pressure 168/67 mmHg, respiratory rate 16 breaths per minute with 96 % oxygen saturation on room air. Initial laboratory studies revealed an increased anion gap metabolic acidosis of 20 (normal <12), with bicarbonate level of 17 mmol/L (normal: 22-30 mmol/L), acute renal failure with creatinine of 3.7 mg/dL (normal: 0.8-1.5 mg/dL) and blood urea nitrogen (BUN) of 19 mg/dL (normal: 9-20 mg/dL). Hepatic transaminases on the day of presentation were normal.

What is diethylene glycol, and how commonly is it used in consumer products? Diethylene glycol (DEG, CAS # 111-46-6) is prepared commercially through heating ethylene oxide with glycol, forming two ethylene glycol molecules joined by an ether bond. DEG is a colorless, nearly odorless, syrupy liquid with a sweet taste. It has a molecular weight of 106 Daltons, and is soluble in water, acetone and ether, but insoluble in benzene and toluene. Because of its cheap production costs and water and solvent solubility, it has seen widespread use in industrial processes as well as in consumer products. The myriad of uses of DEG include wallpaper strippers, Sterno® liquid fuel, glass cleaners, brake fluids, theatrical fog solutions, household tints and dyes, in waxes and adhesives as a freezing point depressor, and automotive antifreeze. This ready availability, along with variable warning labels and protective packaging has led to and an unacceptably high potential for future poisonings.

## What is the historical significance of DEG poisoning?

The first experience with human poisoning occurred in the USA in 1937, when it was used as a diluent for elixir of sulfanilamide. The Massengil Company had tested the sulfanilamide, but not the DEG vehicle, as this was not required at that time. Severe poisoning resulted in the death of at least 105 people from renal failure. That tragedy has major historical significance for medical care in the USA, since it led to the passage of the Food, Drug and Cosmetic Safety Act of 1938, and greatly strengthened the regulatory powers of the FDA.

Several mass poisonings involving DEG ingestion as an accidental excipient in liquid medications have occurred since the US Massengil tragedy. Since liquid medications are produced for pediatric preparations, the overwhelming majority of victims were young children. DEG caused the death from acute renal failure of 85 children in Haiti when the glycerol diluent in an acetaminophen elixir was found to have been contaminated with DEG. DEG has no odor, and a pleasant, sweet taste, which affords no protection or warning for the unaware recipients of the contaminated medications. Similar disasters with DEG contamination of liquid medication diluents have also occurred in Argentina, India, Bangladesh, Nigeria, and South Africa.

## What are the main toxicities of DEG?

In all the mass poisoning reports, the patients developed severe metabolic acidosis and renal failure. Since these historical mass poisonings either occurred prior to the availability of hemodialysis or in countries with limited availability of this intervention, early morbidity and mortality rates were quite high and despite use of peritoneal dialysis

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in some cases, most poisoned patients died from renal failure. Some of these reports mentioned neurological injuries as well, but it appears that most victims did not survive long enough for the more severe neurological abnormalities to become apparent. In these mass poisoning exposures, renal toxicity progressing to renal failure was a consistent finding, with neurologic symptoms infrequently reported.

Neurologic complications have been described in more recent reports from developed countries, though the mechanism remains unknown.

Initial presentation of patients consisted of gastrointestinal effects with significant vomiting and abdominal pains. Patients who were more significantly poisoned experienced subsequent hepatic and renal injury. In cases where hemodialysis was promptly instituted, patients survived only to develop neurological impairments of varying severity. Cranial neuropathies have been seen, as well as progressive, diffuse demyelinating sensori-motor polyneuropathy. Patients can have EEG evidence of severe encephalopathy as well, and in fact can remain unresponsive and completely paraplegic for weeks. At this stage, their clinical prognosis can appear dismal, and the question of whether to withdraw life support should not be treated lightly. Several reports have documented good neurologic recovery in some of these cases. Since these neurologic signs begin to manifest at 5- 10 days post-ingestion, most of the victims of the early epidemic poisonings such as those in India and Jamaica never survived long enough to manifest this aspect of DEG toxicity.

## What are the toxicokinetics of DEG?

Animal studies indicate that DEG is well-absorbed after oral administration in both rats and dogs. Its volume of distribution appears to be approximately 0.8 L/kg. It was once believed that DEG metabolism involved initial cleavage into two molecules of ethylene glycol (EG), then subsequent metabolism into the toxic byproducts glycolic acid and oxalic acid. However, it is well-known that ether bonds are relatively stable and rarely undergo metabolic cleavage. More recent studies have documented that the urinary sediment from renal failure poisoning victims showed none of the expected oxalate crystals seen in patients similarly poisoned with EG. Toxicokinetic studies in animals have determined that alcohol dehydrogenase and aldehyde dehydrogenase produce the primary metabolite 2-hydroxyethoxyacetic acid (HEAA). In these test animals, more than 80% of the dose is excreted in the urine: over 50% as the unchanged parent compound, and approximately 10-30% as the HEAA metabolite.



## What initial management options should be considered for this patient?

In addition to standard poison management, immediate concerns here should be the management of his metabolic acidosis and acute renal failure. This combination should prompt laboratory investigation for toxic alcohol, specifically ethylene glycol and DEG.

## Are alcohol dehydrogenase inhibitors indicated in the management of DEG poisoning?

Since DEG is known to be metabolized into HEAA by ADH (alcohol dehydrogenase) and ALDH (aldehyde dehydrogenase), it is logical to assume that inhibition of its metabolism by ADH with either ethanol or fomepizole would prevent toxic metabolite formation. This has been shown in animal studies, and significantly reduced DEG toxicity in these animals. Although failure of this treatment has been reported by some authors, fomepizole has been used in conjunction with hemodialysis in two cases of DEG poisoning, both reporting a good outcome. However the case described by Brophy, et al had no clinical symptoms of toxicity, and the pre-dialysis serum level of DEG was only 1.7 mg/dL. Compared to known toxic levels of other toxic alcohols (methanol and ethylene glycol, toxic levels >25mg/dL), this low level may have simply represented an insignificant exposure, and presumably would have done well without any intervention. The case described by Borron et al had an anion gap acidosis and an osmolar gap at presentation, but no renal failure. Prompt hemodialysis was instituted as well as administration of fomepizole, and the patient did well.

Ethylene glycol (EG) ingestions can theoretically be managed with ADH inhibition alone, as long as acidosis has not developed and the patient's renal function remains normal. The parent EG molecule is not particularly toxic, and as long as production of the toxic metabolites is prevented, renal clearance of EG can occur. Although infrequently used in this country due to the cost of fomepizole, this management scheme is frequently utilized in other countries. However, the same scheme cannot be generalized to DEG poisoning. The parent DEG molecule itself may be directly nephrotoxic, in addition to the metabolites. Therefore, ADH inhibitors alone are not adequate treatment. All DEG ingestions with acidosis and/or renal failure should receive fomepizole and immediate referral for hemodialysis.

## What is the role of hemodialysis in DEG ingestions?

Emergent hemodialysis is critically important for management of these cases. It appears from case reports that the parent molecule is potentially toxic, as well as the metabolites. As noted above, good results were achieved in case reports of fomepizole use in conjunction with hemodialysis. Hemodialysis is the only intervention that has allowed patients with renal failure associated with DEG poisoning to survive, as peritoneal dialysis was ineffective in the Haiti and India epidemics.

In our case, hemodialysis was initiated shortly after presentation for renal failure and acidosis. Additionally, thiamine and pyridoxine were added. Quantitative analysis for ethanol, methanol, ethylene glycol and isopropyl alcohol were performed on the day of presentation and were negative. Initial renal ultrasound showed no significant abnormalities.

On hospital day 2, the patient remained alert but complained of abdominal pain and was oliguric. Hemodialysis was continued and hepatic transaminases were noted to be elevated [ALT 354 U/L (normal: 21-72 U/L) and AST 554 U/L (normal: 17-59 U/L)]

## What is the significance of the hepatotoxicity?

Several case reports have noted elevated hepatic transaminases, and most have begun N-acetylcysteine therapy. The hepatic injury appears to be less important than the nephrotoxicity, but NAC therapy is relatively benign and most experts would recommend it. Our case was treated with NAC for several days, and his transaminases returned to normal. He never had evidence of hepatic dysfunction, such as coagulopathy or hyperbilirubinemia.

## What initial signs on presentation can be used to predict neurologic toxicity?

Most recently, Alfred et al reported an epidemic poisoning where seven inmates at a correctional facility drank varying amounts of DEG. Despite rapid diagnosis and early hemodialysis, they observed delayed neurologic complications. Most developed these complications within the first week post-ingestion. These included cranial and peripheral sensorimotor peripheral neuropathies, encephalopathy, and even seizures. This was the first case series to elucidate the fact that renal injury is a marker for subsequent neurologic sequelae. Their patients with minimal exposures and no renal failure did not develop neurologic complications.

Over the next several days, our patient developed progressive lethargy, dysphonia, facial diparesis, dilated non-reactive pupils, loss of corneal and gag reflexes, and loss of visual and auditory function. By hospital day 6, he developed acute encephalopathy with moderate to severe diffuse slowing on EEG. An EMG/NCV performed at this time showed a generalized sensori-motor peripheral neuropathy with questionable evidence of demyelination.

## Are steroids useful for the neuropathy?

Several of the recent case reports with neuropathy treated with steroids have seen improvement in the neuropathy.

There are not enough cases to know whether this therapy affected the outcome, and there are certainly no controlled studies of this therapy.

## What was the outcome of this case?

Despite high-dose steroid treatment for the progressive neuropathy, our patient became quadraparetic and was intubated for respiratory failure on hospital day 12. He remained ventilator-dependent and unresponsive until hospital day 47, when he regained pupillary light reflexes. On hospital day 56, repeat EMG/NCV studies showed worsened severe demyelinating sensorimotor peripheral polyneuropathy. Renal ultrasound performed on hospital day 75 showed markedly atrophic kidneys with severe cortical thinning, indicating that he will require life-long hemodialysis (or renal transplantation). His neurologic status continued to slowly improve to the point where he could stand and walk a few steps with assistance, and he was transferred to a chronic rehabilitation center on hospital day 97. At 6 months, his neurologic function continued to improve and he was discharged to home, able to stand and walk with a walker.



Select references for case:

Hazardous Substances Data Bank, HSDB, available at: <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>

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Continued on page 7



# SPI CORNER TOPIC: **Oral N-acetylcysteine (N-AC, Mucomist®) and Intravenous (Acetadote®)**

*Contributed by: Mary Halsey-Claps, RN, CSPI, Upstate New York Poison Center, Syracuse, NY*

As the availability of products containing acetaminophen continues to flood the market, health care practitioners are faced with a need to treat overdoses in health care facilities. N-Acetylcysteine (NAC) is used orally or intravenously (IV) as an antidote for acetaminophen (APAP) toxicity.

In acute overdoses N-Acetylcysteine (NAC), especially if administered within 8 hours, prevents hepatotoxicity. It also has merit when administered within 24 hours or longer after acetaminophen toxicity.

Oral N-acetylcysteine has been successfully administered for years as an antidote, but oral dosing poses some challenges for health care practitioners. Oral N-acetylcysteine is available in 10-20% solutions it must be diluted to 5% to increase palatability. If administered by nasal-gastric tube it must also be diluted. Nausea and vomiting can occur after administration. If a dose is not retained for one hour, it is necessary for repeat dosing. Nausea can be mitigated using antiemetics such as ondansetron or metoclopramide. Dosing includes 140 mg/kg as a loading dose followed by 70 mg/kg every four hours for the duration of therapy.

Historically, patients who are unable to tolerate oral NAC, due to protracted emesis, lack of gastrointestinal tract

integrity or severe hypotension were provided oral NAC in an IV formulation. More recently, a commercially available IV antidote became available called Acetadote®. Acetadote has an advantage in causing less nausea and vomiting as well as having an approved shorter course of therapy for patients presenting before 8 hours after ingestion. The loading dose is 150mg/kg in 200ml of D5W. First maintenance dose of 50mg/kg is diluted in 500ml of D5W. This is followed by a second maintenance dose of 100mg/kg diluted in 1000ml of D5W. Careful monitoring for anaphylaxis is important. In addition, the optimal amount of D5W required for concentrated solutions in children is not established and fluid and electrolyte abnormalities can occur in children due to the large amount of D5W administered without sodium.

As a final review, N-acetylcysteine is recommended in the following scenarios; a 4 hours or more after ingestion serum level above the Rumack-Matthew, a patient with an unknown time of ingestion with a positive acetaminophen level or elevated liver function tests, or a patient with clinical signs of hepatitis. The length of treatment can be variable depending on the time of presentation and degree of hepatic damage.

**Upstate NY Poison Center  
750 East Adams Street  
Syracuse, NY  
13210**