A Rare Case Report: Triethylamine Poisoning and The Pharmaceutical Care

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Abstract

Background: Triethylamine is an important intermediate reactant and is widely used in industry and manufacturing. So far, cases of triethylamine poisoning timely saved are rarely reported.

Methods: A 29-year-old male worker from a chemical plant after taking a small sip of triethylamine was presented in ICU. Subsequently, he experienced repeated vomiting, abdominal pain, gastrointestinal bleeding and other related injuries. Owing to no significant improvement in local hospital, he was transferred to Jinshan hospital, Fudan University for better medical treatment. He received hemostasis, PPIs for gastrointestinal mucosal recovery, blood purification for poison removal, nutritional and electrolyte supplement after admission. The author participates in treatment and analyzes clinical results in order to share treatment methods and experience for similar poisoning events in future.

Results: His gastrointestinal bleeding was timely controlled, his pain and other poisoning symptoms were eliminated. And he was discharged home safely.

Conclusions: Triethylamine can break the gastric mucosal barrier, the administration of proton pump inhibitors (PPIs) had a great importance. The timely blood purification for him helped eliminate the residual chemical poison inside his body, so his further liver injury by triethylamine was prevented. His recovery is good.

1. Introduction

Triethylamine is an important intermediate reactant, a chemical substance with moderate toxicity and strong chemical odor, which is widely used in industry and manufacturing. It may have a potential threat to human health and the natural environment. Its occupational exposure or deliberate ingestion may be causes of poisoning. Triethylamine has been considered to be a possible carcinogen. Though its acute and chronic toxicity to animals, mutagenicity and embryotoxicity were reported, there are few studies on human toxicity of triethylamine. Easy to be spotted due to its special ammonia odor, there are still no reports of treatment of triethylamine poisoning. Here we report a patient of its poisoning and his successful treatment after accidental exposure of triethylamine.

2. Methods And Case Presentation

2.1 Methods

Triethylamine poisoning is rare in clinical practice. This article describes a case of triethylamine poisoning who was received PPIs for gastrointestinal mucosal recovery, hemostasis, nutritional and electrolyte supplement, blood purification for poison removal after admission without a specific antidote and no remedial measures for reference. The patient's liver, kidney function, coagulation and other laboratory indicators are monitored. And treatment experience and methods are available for sharing, hoping to save the lives of patients.

2.2 Case Presentation

X J, male, 29 years old, a chemical plant worker in Nantong City, Jiangsu Province, China, ingested a mouthful triethylamine because of a bad mood and suffered occupational exposure from operation at 1.0 pm on Jan. 13, 2019. About 10 minutes later, a burning sensation was presented in his mouth, accompanied by chest tightness, chest pain, multiple nausea and his discomforts worsened with vomiting. His vomitus was dark brown or bright red-colored, about 600 ml. He had no digestive illness before and had not taken any other medicine recently.

X J was first sent to a local hospital for basic medical care and was initially diagnosed as triethylamine poisoning. He was administered pantoprazole, almagate suspension for 6 hours without significant improvement. Then he was transferred to the affiliated hospital of Nantong University at 7.0 pm. Physical examination showed that his blood pressure (BP) was 130/86 mmHg, heart rates (HR) 104 beats/minute, respiration (R) rates 20 breaths/minute. His laboratory dates were as follows: oxygen saturation (SpO_2), 100%; hemoglobin, 170 g/L; platelets, $182 \times 10^9/\text{L}$; alanine aminotransferase (ALT), 30 μL ; aspartate aminotransferase (AST), 76 μL ; blood urea nitrogen (BUN), 4.9 mmol/L; creatinine, 64 $\mu\text{mol/L}$. white blood cells (WBC) count, neutrophils (N) were significantly elevated at $20.2 \times 10^9/\text{L}$, 91.8% respectively, and his occult blood of vomit was positive. He had a swelling tongue and mouth ulcers, and no other abnormalities. Computed tomography (CT) scans revealed exudative lesions in the right lower lung, thickened edema of the esophagus wall, cholecystitis, left kidney stones, and decreased liver parenchymal density—possibly fatty liver, and he may suffer from chemical-induced liver injury. He was diagnosed as triethylamine poisoning.

For better medical treatment. at 0:30 am on Jan. 14, 2019, he was transferred to the emergency department (ED), Jinshan Hospital, Fudan University - the nuclearization treatment center of the Yangtze River Delta Area, China, which is famous for the treatment of nuclear and chemical injury. His physical examination revealed that axilla temperature was 37.7 degree Celsius ($^{\circ}\text{C}$), BP 126/76 mmHg, HR 90 beats/minute, R 21 beats/minute, and SpO_2 98%. He was conscious. He had coarse sounds on bilateral lung and no abnormalities in others. He was diagnosed as triethylamine poisoning and upper gastrointestinal bleeding. He received femoral vein catheterization, blood purification (Technical parameters: continuous veno venous hemodiafiltration (CVVHD) and hemoperfusion (HP), blood flow 200 ml/min, dialyzer area 1.4 m^2 , low molecular heparin calcium injection 4100 AxalU, combined with heparin sodium injection 12500 units for anticoagulation, replacement fluid is hemodialysis replacement fluid, replacement rate of 0 ml/h, flow rate of 3000 ml/h), omeprazole (80 mg, ivgtt, once a day) and supplement with normal saline.

He was hospitalized in the intensive care unit (ICU) after multi-disciplinary consultation. He was conscious and cooperative with a physical examination, his abdomen was soft with scattered pain, no rebound pain, no skin yellowing and ecchymosis. His BP was 98/44 mmHg, and HR 73 beats/minute, R 20 breaths/minute, and axilla temperature 38.5 $^{\circ}\text{C}$. Arterial blood gas analysis (ABG) values were as follows: pH 7.527; SpO_2 , 98.5% (under oxygen uptake by

Nasal catheter of 3 L/min); partial pressure of carbon dioxide (PCO_2), 3.42 Kpa; arterial partial pressure of oxygen (PO_2), 13.3 Kpa; potassium, 3.70 mmol/L; sodium, 135 mmol/L; chlorine, 109 mmol/L; lactic acid, 1.2 mmol/L; serum chemistries and coagulation values were: prothrombin time (PT), 13.70 seconds (s); activated partial thromboplastin time (aPTT), 25.1 s; thrombin time (TT), 15.5 s; international normalized ratio (INR), 1.21; D-dimer, 0.898 mg/L; amylase, 113 U/L; procalcitonin, 0.33 ng/ml; total bilirubin (TB), 32 $\mu\text{mol/L}$; combined bilirubin, 7 $\mu\text{mol/L}$; ALT, 64 U/L; AST, 26 U/L; urea, 5.0 mmol/L; uric acid, 290 $\mu\text{mol/L}$; creatinine, 61 $\mu\text{mol/L}$. His liver chemistries were mildly elevated with no other abnormalities. He had smoked for 10 years and drank occasionally. He took no medication before and had no other illness. His diagnosis was triethylamine poisoning, acute upper gastrointestinal bleeding, chemical esophagitis (triethylamine poisoning). He was fasting at first. He received hemostasis, symptomatic treatment and adjuvant therapy (as in Table 1). On Jan. 15, he was allowed to take food (only cold fluid) and received heparin-free blood purification treatment. The next day he had melena (paste-like, without blood, about 300 ml) and had abdominal pain, his blood purification was suspended. Lab data were showed in Table 2. The previous treatment was continued. On Jan. 18, he was unable to eat due to sore throat, and was diagnosed as a chemical injury in the throat and pharynx diagnosed by an ent doctor. One week later, he had no obvious abnormalities, no obvious gastrointestinal bleeding and discomfort, indicating that the treatment results were good. On Jan. 22, his PT was 15 s, aPTT 28.6 s, and TT 13.8 s. The clotting time was still prolonged.

On Jan. 23, a little blood was found in his sputum, he was suggested continuing his treatment. He requested to discharge home due to medical costs and other reasons and was discharged. The final diagnosis was: triethylamine poisoning, acute upper gastrointestinal bleeding, chemical esophagitis. His medical orders suggested having more rest, keeping warming, taking foods with rich vitamin k, taking tests of blood routine and fecal occult blood tests, and avoiding possible potential poisoning and gastrointestinal bleeding. His medications were omeprazole enteric-coated capsules (20 mg, bid, orally); Clostridium caseinate tablets (40 mg, tid, orally). He had a good recovery by telephone query on Jan.12, 2020, and left that plant.

3. Results

His gastrointestinal bleeding was timely controlled, his pain and other poisoning symptoms were eliminated. And he was discharged home safely.

4. Discussion

4.1 Physical and chemical properties of Triethylamine

Triethylamine is a kind of aliphatic amine and a colorless transparent oily liquid with a vapor pressure of 53.33 mPa at 15.7 °C and with a strong ammonia-like pungent chemical odor as an organic solvent. It is volatile, flammable, explosive, highly corrosive, and weak alkaline. Its molecular formula is (CH_3CH_2)₃N (structural formula shown in Fig. 1), its density is 0.7255 g/mL, molecular weight is 101.19, and the boiling point is 89.5 °C. Over the last century, triethylamine has been extensively used in the industry as a catalyst, a solvent or a rust inhibitor for organic polymerization, mainly used in the synthesis of chemical drugs, pesticides and dyes, such as for ampicillin, amoxicillin, cefoxitin, and penicillamine synthesis, and also used in the production of additives, emulsifiers, perfumes, high-energy fuels, liquid rocket propellants, and etc^[1-4]. Because of the advantages of high production efficiency, precise size, energy-saving, and low gas output of cold-core making procedure of triethylamine, it has been widely used in the foundry industry in recent years^[5]. Therefore, it has a wide range of occupational exposure and maybe as a potential environmental pollutant, which can cause various health problems.

4.2 Characteristics of Triethylamine poisoning

Triethylamine is a moderately toxic compound, and has a strong irritating effect on human skin and mucous membranes. In addition to being directly toxic to corneal epithelial cells, it also has an irritating effect on the upper respiratory tract. It can cause itchy skin and tachycardia, and low blood pressure^[6-8]. Studies shows that triethylamine can be completely biodegraded by a microorganism of the genus *Arthrobacter* protozoa (R4 strain) in the natural environment, and Some metal ions (Cu^{2+} , Mn^{2+} , Zn^{2+} , Co^{2+} , Ni^{2+} and Ag^+) can strongly inhibit the degradation of triethylamine^[9]. Its metabolism in human body is still unclear. There is no specific antidote and detoxification method for triethylamine poisoning, so it is different from organophosphorus pesticides poisoning. Industrial poisoning is mainly caused by respiratory inhalation. The main clinic symptoms are irritations to the eye and upper respiratory tract, including blinking, eye closing, tearing, runny cough, sneezing^[10, 11], and even visual disturbance^[12]. Poisoning by oral ingestion has not been reported previously. Because triethylamine's strong ammonia odor easy to be detected, it is unlikely to cause severe intoxication. Literature search revealed no reports of death from acute poisoning with triethylamine.

4.3 Experience of treatments

After triethylamine ingestion, X J suffered repeated vomiting and melena, and was initially given pantoprazole and almagate suspension in the local hospital, with no obvious improvement. In the ICU, he received hemostasis, blood purification and fluid replacement, and nutritional support. Though no severe abnormalities of liver function in Table 1, further damage to the liver and other organs could present if he did not receive timely and proper treatments. His treatment in ICU was necessary. Proton pump inhibitors (PPIs) can effectively inhibit gastric acid secretion, and help repair gastric mucosa^[13]. They have a healing effect of over 90% for duodenal ulcer, gastric ulcer and reflux esophagitis for 4-6 weeks, superior than H_2 antagonists. Megadoses can treat refractory peptic ulcers and gastrinoma^[14].

Coagulopathy generally occur after gastrointestinal (GI) bleeding or other hemorrhage which is not common, and may respond to vitamin K_1 if the PT or INR is prolonged. X J had a definite exposure to triethylamine accompanied by repeated vomiting and melena and he had no history of gastrointestinal diseases. So he suffered from acute chemical gastrointestinal bleeding. X J's coagulation function was normal, the objective of treatment is to eliminate the residual triethylamine inside his body, repair damage, avoid possible gastrointestinal bleeding, and even the possibility of hemorrhagic shock. The use of PPIs and tranexamic acid are suitable.

Hyperthermia and hyperventilation produce increased insensible water loss, repeated vomiting can easily promote GI fluid losses leading to electrolyte metabolism disorders even severely depletion of fluid volume. Therefore, promethazine, prochlorperazine, and ondansetron were selected as antiemetics^[15]. Ondansetron is most commonly used because of its low cost and slight side effects. Depending on acid-base balance and net fluid and electrolyte intake and output, serum sodium and potassium concentrations may be normal, elevated, or decreased. X J's hypokalemia and alkalosis on admission were timely corrected. Vitamin C, potassium chloride, 5% glucoside injection, etc, were administered to maintain the normal internal environment and blood volume. Reduced glutathione and cyclophosphine adenosine glucosamine were given to nourish the heart and liver, to prevent and reduce toxic related heart and liver injury. X J's temperature was normal, his WBC count was increased, and methylprednisolone sodium succinate combined with aerosolized inhalation preparation was given as anti-inflammatory treatment for the possible inflammatory reaction when he entered the ICU.

Clinically, emetic lavage, drainage, and detoxification are routinely used as pre-treatment for drug or organophosphorus poisoning. Based on X J's condition and contraindications, the delayed injury should be considered for respiratory tract, GI tract and other organs because toxic substances absorption can have potential effect. Alkalinization of the urine and blood purification can generally enhance the excretion of toxicants may be beneficial after the ingestion of toxic substances. Blood purification was necessary for him because the toxic chemical had been absorbed into blood, especially for mega doses, strong toxicity, and unknown intake of toxicants, it can replenish body fluids and balance electrolyte. Timely blood purification treatment can certainly improve survival rate and quality of life^[16, 17]. Studies showed that hemoperfusion can effectively eliminate initial plasma high concentrations of paraquat (≥ 200 ng/ml) from the plasma in patients, while in patients with low initial plasma paraquat concentration (< 200 ng/ml), the perfusion clearance effect was not high valued. The overall clearance rate of hemoperfusion on plasma paraquat may be improved by increasing hemoperfusion time yet the elimination efficiency of hemoperfusion was decreased, while it was helpful against the rebound phenomena by repeated hemoperfusion treatment^[18]; Zou et al showed that paraquat intoxication frequently caused death due to respiratory and kidney failure, hemoperfusion could significantly improve the clearance rate of paraquat poisoning, protect key organ's function and inhibit systemic oxidative stress response^[19]. For males, a true healthy normal ALT was 29~33 IU/l, and levels above this is unhealthy and should be assessed^[20]. XJ's ALT was 52 IU/l (see Table 1), indicating the presence of potential liver injury. The timely blood purification for him helped eliminate the residual chemical poisons inside his body, and his further liver injury by triethylamine was prevented. Therefore, his ALT was kept at a low level. His liver toxicity may be exacerbated if there was no blood purification. Thus, timely blood purification treatment is necessary and significant for his treatment of poisoning. In summary, earlier initiation of treatment for poisoning patients is the most important solution for survival, and organ protection is the cornerstone of treatment, blood purification is a key tool for poison removal. Simultaneously, the regulation of duration time and the speed of blood purification are equally important.

For X J, earlier initiation of the treatment, hemostatic, PPI for acid-suppression and stomach protection, blood purification for poison removal is the important rescue firstly, and it is necessary for fluid and nutritional supportive treatment due to vomiting.

4.4 Pharmaceutical Care

After admission, pharmacists inquired X J's previous medical history and medications in detail to provide clinical treatment assistance. X J presented symptoms of gastrointestinal bleeding. After careful evaluation, heparin-free blood purification treatment was advised. Nonsteroidal anti-inflammatory drugs and glucocorticoids could bring harm to digestive tract, and their use must be restricted. And he should take pantoprazole for six weeks let the gastric mucosa repair.

5. Conclusions

Triethylamine is a commonly used chemical reagent in industrial production. It mainly destroys the gastrointestinal barrier after occupational exposure or deliberate ingestion, causing bleeding and related organ damage. The treatment after poisoning is a challenge to medicine. This article reports a case Clinically rare triethylamine poisoning, clinical pharmacists summarized and analyzed the treatment process from the perspective of drug treatment and gave pharmaceutical care, hoping to provide reference and experience for the treatment of related poisoning in the future.

Abbreviations

Ivgtt = intravenously guttae; iv = intravenous injection; qd = once a day; bid = twice a day; q8h = every 8 hours; TB = total bilirubin; CB = conjugated bilirubin; ALT = alanine aminotransferase; AST = aspartate aminotransferase; ALP = alkaline phosphatase; GGT = gamma-glutamyl transferase; Alb = Albumin; PA = Prealbumin; INR = international normalized ratio; aPTT = activated partial thromboplastin time; TT = thrombin time; Hb = hemoglobin; Scr = serum creatinine.

Declarations

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Availability of data and materials

All datasets presented in this study are included in the article.

Ethics approval and consent to participate

Written informed consent was obtained from the patient to participate to this case report and any accompanying images.

Competing Interests

The authors declare that there are no conflicts of interest.

Consent for publication

The manuscript is approved by all authors for publication. I would like to declare that the work described was original research that has not been published previously, and not under consideration for publication elsewhere.

Authors' contributions

1. Lin Yanhua is mainly responsible for collection and sorting of the entire medical data, the query of medication data, and the writing of original manuscript.
2. Jiang Huaqiao is responsible for the text review and proof reading of the manuscript.
3. Fang Zhonghong provided valuable medical records, and was responsible for literature search and review of the final paper.

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Tables

Table 1
Summary of drug treatment during X J’s hospital stay

Time	Medications and their dosage and usage
Jan. 14–22	10% potassium chloride 10 mL dissolved in glucose sodium chloride injection 500 ml (ivgtt,qd)
	Vitamin C 3 g and vitamin B6 0.2 g dissolved in 0.9% sodium chloride injection 500 ml (ivgtt,qd)
	Alanylglutamine injection 50 ml dissolved in amino acid compound injection 250 ml (ivgtt,bid)
	Reduced glutathione 1.2 g dissolved in 0.9% sodium chloride injection 20 ml (iv,bid)
	Ambroxol hydrochloride 60 mg dissolved in 0.9% sodium chloride injection 20 ml (iv,bid)
	Cyclophosphine adenosine glucosamine 150 mg dissolved in 5% glucose injection 250 ml (ivgtt,qd)
	Tranexamic acid sodium chloride injection 100 ml (ivgtt,qd)
Jan. 14–16	Lansoprazole 30 mg dissolved in 0.9% sodium chloride injection 100 ml (ivgtt,bid)
Jan. 14–17	Methylprednisolone sodium succinate 80 mg dissolved in 0.9% sodium chloride injection 250 ml (ivgtt,qd)
Jan. 15–23	Ambroxol hydrochloride 30 mg, terbutaline sulfate injection 0.5 mg, and budesonide suspension 1 mg dissolved in 0.9% sodium chloride injection 5 ml (spray,bid)
Jan. 16–23	Omeprazole for injection 40 mg (iv,q8h)

Ivgtt = intravenously guttae; iv = intravenous injection; qd = once a day; bid = twice a day; q8h = every 8 hours;

Table 2
Laboratory values during hospitalization

Time (Jan.)	TB	CB	ALT	AST	ALP	GGT	Alb	PA	INR	aPTT	PT	Hb	Scr
	μmol/L	μmol/L	U/L	U/L	U/L	U/L	g/L	mg/L		%	s	g/L	μmol/L
14	26	0	52	21	45	15	34	/	1.24	82	14.1	147	59
15	13.2	3.4	33	17	49	15	34	172	1.14	80	12.9	136	54
16	12	0	43	19	50	16	32	/	1.14	94	12.9	141	58
17	16	0	44	17	51	19	34	/	/	/	/	146	60
18	15	0	50	17	47	19	35	/	1.19	96	13.5	149	69
20	15	0	54	16	46	18	32	/	1.24	91	14.1	145	56
22	7.7	2.2	45	19	47	18	33	198	1.32	93	15	146	57
Normal Range	5~21	0~3.4	7~40	13~35	35~135	7~45	35~52	200~400	0.77~1.25	75.6~113.4	9~12.5	130~175	57~111

TB = total bilirubin; CB = conjugated bilirubin; ALT = alanine aminotransferase; AST = aspartate aminotransferase; ALP = alkaline phosphatase; GGT = gamma-glutamyl transferase; Alb = Albumin; PA = Prealbumin; INR = international normalized ratio; aPTT = activated partial thromboplastin time; TT = thrombin time; Hb = hemoglobin; Scr = serum creatinine.

Figures

Figure 1

Triethylamine chemical structure