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Case Report

Crossroads of Industrial Chemistry and Clinical Toxicology: A Rare Case of Organotin Poisoning

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Abstract

Background: Cresylic acid and tributyltin oxide (TBTO) are prevalent industrial compounds utilized in household biocides and disinfectants. Oral intake, particularly when coupled, can result in significant systemic toxicity, even though human exposure often occurs through inhalation or dermal routes.

Case Presentation: A 19-year-old woman from rural India intentionally ingested a disinfectant that contained TBTO and cresylic acid, which caused multiple organ poisoning in an unusual case. She responded to supportive therapy and eventually made a complete recovery.

Conclusions: This case emphasizes the possibly life-threatening risks associated with exposure to phenolic and organotin compounds and highlights the value of early supportive treatment and careful supervision. Because there isn't a lot of information on co-ingestion in humans, clinicians should treat such poisonings as if they were novel exposures.

Introduction

Tributyltin oxide (TBTO), a potent industrial biocide used in anti-fouling coatings, disinfectants, wood preservatives, and other applications, is one of the many organotin compounds.[1] According to reports, most cases of human TBTO poisoning happen through inhalation or skin contact.[2, 3] Symptoms that have been documented include dizziness, nasal congestion, and irritation of the upper respiratory system.[3] The majority of toxicity information comes from animal experiments because oral intake is relatively uncommon in people. Many disinfectants contain cresylic acid, a mixture of cresols that is both harmful and corrosive. Ingestion has been linked to documented systemic toxicity, with symptoms such as coma, acute respiratory distress syndrome (ARDS), and liver and renal failure.[4,5,15] TBTO retains its biocidal activity in preparations when combined with cresylic acid, which has a quick antimicrobial action. These compounds may combine to produce synergistic toxicity. To the best of our knowledge, this is the first documented instance of deliberate co-ingestion of TBTO and cresylic acid in humans.

Case Presentation

About an hour after ingesting a disinfectant that included TBTO and cresylic acid, the 19-year-old girl was wheeled to the emergency room. She was extremely restless, had excessive salivation, and had a history of multiple episodes of non-blood-stained material and non-bilious vomiting. Among her arrival vital signs were a respiratory rate of 24 breaths per minute, a pulse rate of 160 beats per minute, a blood pressure of 110/62 mm Hg, desaturation of 92% under room air, and an altered mental status—Glasgow Coma Scale (GCS) score of E3V4M5. Arterial blood gas (ABG) analysis showed a lactate level of 1.4 mmol/L, a pH of 7.29, an HCO_3 level of 18 mEq/L, a base excess of -6.4, and an ionized calcium concentration of 0.97 mmol/L. The ECG showed a heart rate of 156 beats per minute and a significant number of premature ventricular complexes (PVCs). [Figure 1] There were no indications of congestion or burns on the oral mucosa, and chest auscultation revealed extensive crackles.point of care lung ultrasound showed no B –lines , eliminating the possibility of cardiac failure.

Chest and abdominal X-rays [Figure 2] were unremarkable. Aside from a mild hypokalemia of 3.2 meq/L, the lab metabolic panel was within normal limits. [Table 1] The clinical impression was aspiration pneumonitis with altered mental status secondary to toxin intake. The supportive management strategy included administration of intravenous fluids, oxygen therapy, and continuous cardiac monitoring. 20 mL of 10% intravenous calcium gluconate was administered. Additionally, she was given intravenous boluses of sedatives, such as fentanyl and midazolam, to decrease agitation. Over twelve hours, the patient's vital indicators—HR: 80 bpm, BP: 100/60 mmHg, SpO2: 94% RA, GCS: 15/15—returned to normal. The patient's cardiac biomarkers were within normal limits. She was transferred to the medical observation unit for supervision. Before being discharged with stable vital signs for psychiatric follow-up, she had been hemodynamically stable for the previous three days of her hospital stay.

Discussion

This illustration highlights the uniqueness of the simultaneous ingestion of TBTO and cresylic acid, which has not been documented elsewhere. Despite the fact that the literature discusses each agent independently, illuminating their individual toxicodynamics, there are no treatment guidelines when ingested in combination. The importance of treating the presentation as a new exposure, which is the cornerstone of our strategy of all-inclusive supportive therapy and empirical intervention, is further emphasized by this. This index case shows the possibility of significant multi-organ toxicity after taking TBTO and cresylic acid (Tables 2 & 3). Early cardiac irritability (PVCs), gastrointestinal discomfort, and respiratory and neurological issues were all part of the clinical presentation.

The organotin compounds are digested from the gastrointestinal tract (Kimbrough, 1976). Acute toxicity of organotin compounds is strongly influenced by the length of the alkyl chains attached to the tin. Tributyltin (TBT) is generally less toxic than trimethyl- and triethyltins.[6,7] Generally, the toxicity of organotin compounds is influenced more by the alkyl substituents than the ionic substituent, which may form the rest of the molecule (for example, salicylate, acrylate, etc.). Tributyltin compounds are moderately toxic via both ingestion and dermal absorption. Reported oral LD50 values for tributyltin oxide (TBTO) range from 55 to 87 mg/kg in mice and rats. Other mucous membranes, such as the eyes and nasal passages, may also become irritated upon exposure. The degree of the absorption of tributyltin oxide is between 20% and 55%. TBTO can readily pass through cell membranes and build up in fat tissue due to its strong affinity for lipids.[8,9] By improving membrane permeability, cresyl acid may aid in this absorption. The systemic effects of both medications, which are neurotoxic [10] and hepatotoxic, can be enhanced by one another. As shown in the index case, TBTO has the potential to directly block H+/K+-ATPase in renal intercalated cells, which can cause hypokalemia and renal tubular acidity.[11,12]

Despite having PVCs, this patient's

condition may be indicative of the early cardiotoxicity of TBTO, which is linked to several factors, including oxidative stress and oxidative phosphorylation disruption [10], both of which can lead to myocardial necrosis. The patient's creatinine Kinase(CK) and lactate dehydrogenase (LDH) levels, the cardiac biomarkers, were within normal ranges. Thus, TBTO's cardiotoxicity was related to its ability to impair calcium homeostasis.[14] Calcium gluconate may help treat particular symptoms of TBTO toxicity, such as cardiac irritability coupled with hypocalcemia, which was the case in our scenario. It may strengthen the membranes of cardiomyocytes. However, it is neither a cure nor an antidote. It should be a component of a more comprehensive supportive care approach that includes monitoring, oxygen therapy, and, when necessary, critical care. In TBTO poisoning, calcium gluconate has no antidote effect, and there is no particular research on it. Remember clinical indications, such as arrhythmias or low calcium levels, while determining how to utilize it. Nevertheless, it does not mitigate the main effects on the mitochondria or endocrine system.

Conclusion

While animal toxicology data suggest both compounds are individually toxic, the absence of documented co-exposure means that potential synergistic toxic effects are unknown in clinical settings. Any confirmed case—accidental or intentional—of co-ingestion should be considered suitable for publication to fill this gap in knowledge.

Experimental models are needed to assess how cresols and TBTO interact with each other. Given the rarity of such combined exposures, this case underscores the need for heightened awareness of industrial chemical poisoning involving uncommon but highly toxic agents like organotins, especially when co-ingested with organic solvents that may amplify their effects.

Clinicians should treat co-exposures involving organotins and phenolics as novel toxicologic entities, requiring aggressive monitoring and individualized supportive care. Greater awareness of such rare poisoning profiles is essential for emergency and critical care physicians.

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Figures and Tables

Table 1. Laboratory metabolic panel of index case

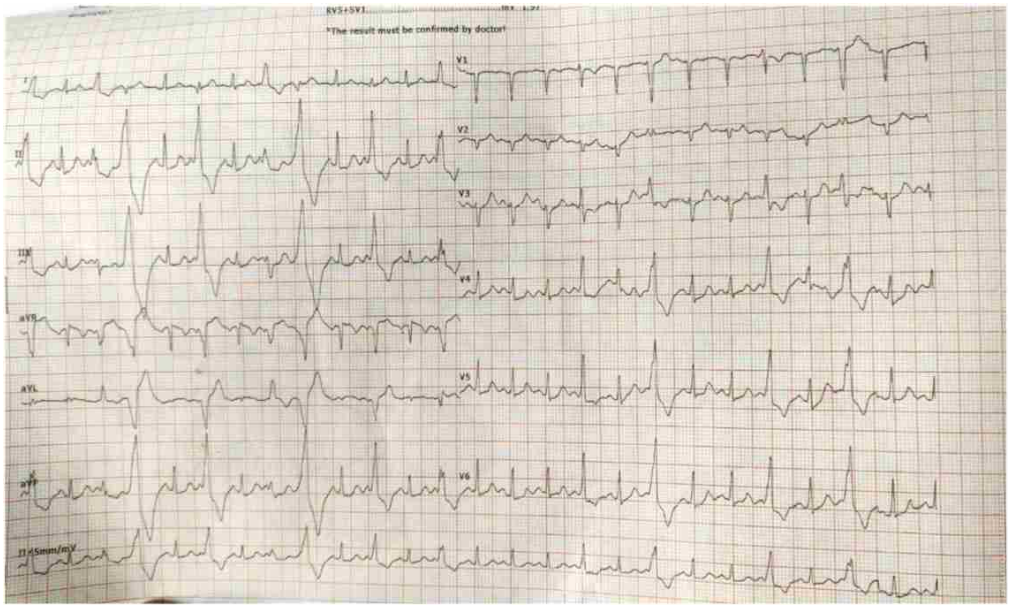
PARAMETER	VALUE
Urea (mg/dL)	25 mg/dL
Creatinine (mg /dL)	0.59 mg /dL
Sodium/potassium (mmol/dL)	144 /3.27 meq/dL
Calcium / Magnesium (mmol/dL)	9.6/2.7 meq/d
LAST /ALT / ALP(IU/L)	31/41/82
Albumin (mg /dL)	5.01
Hemoglobin	14.2 g/dL
Platelet count	5.10 lakh
cells/cummTroponin	<1.5 ng /mL
Creatinine kinase (total)	93 IU/L
LDH	377 U/L

Table 2. Mechanisms of Toxicity of Tributyltin Oxide (TBTO)

Mechanism	Description
Mitochondrial damage	Inhibits ATP synthesis by disrupting oxidative phosphorylation
Immune suppression	Induces thymic atrophy and leukopenia and reduces natural killer (NK) cell activity[7]
Endocrine disruption	Mimics or blocks hormone receptors, contributing to reproductive and metabolic toxicity[9]
Oxidative stress	Triggers lipid peroxidation and apoptosis through free radical generation
Neurotoxicity	Alters neurotransmitter levels and damages neurons, as observed in animal models

Table 3. Systemic toxicity of TBTO across various organ systems

System	Observed Effects (based on animal and human data)
Respiratory	Nasal congestion, cough (inhalation)
Reproductive	Fetal loss, testicular damage (animals)
Neurologic	Behavioral abnormalities, neurodegeneration
Hepatic/Renal	Liver enzyme elevation, nephrotoxicity
Cardiovascular	Myocardial necrosis, Vascular endothelial swelling, Infiltration of inflammatory cells in the myocardium



1. ECG of index case showing tachycardia of 156 bpm and multiple PVCs

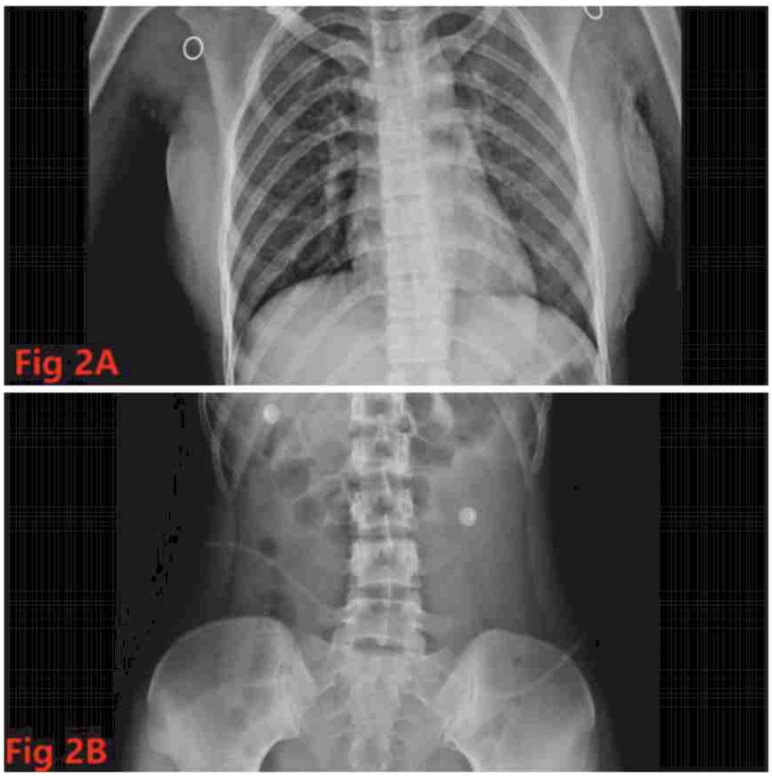


Figure 2A *shows the chest radiograph of the index case. There were no features of aspiration/pneumomediastinum/pneumothorax.*
Figure 2B *shows an abdominal radiograph. There was no evidence of pneumoperitoneum*

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