Toluene Poisoning (Accidental Ingestion of Evostik)

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Summary

INTRODUCTION

Childhood poisoning is one of the causes of high morbidity and mortality especially among under-five children in low income countries. The home and its surroundings may harbour poisonous substances which might be ingested by adventurous children. Evo-stik glue is a modified silane (MS) polymer based high performance adhesive. It contains toluene, a colourless, sweet-smelling liquid with detrimental effects on virtually every organ in the body especially brain and kidneys. Toluene is a component of household items such as gasoline, shoe and nail polish.

AIM / OBJECTIVES

From a few reported cases of toluene poisoning worldwide there was need to create awareness on the possibility of toluene poisoning as well as its deleterious effects. This is a case report on exposure to the effects of toluene by accidental ingestion that set the platform to emphasize the importance of prompt and appropriate management of toluene poisoning.

METHODOLOGY

This case report is about a 2year old girl who accidentally ingested 15-20ml evostik glue stored in an attractive container in the home. QH was presented at the Lagos State University Teaching Hospital Paediatric Emergency Unit with a one day history of persistent spontaneous, non-projectile and non-bilous vomiting.

On admission, she was conscious, moderately dehydrated with dry *buccal mucosa*. (38.2°C) febrile, but not pale, icteric, or cyanosed. She developed seizures, recurrent hypoglycaemia, generalized peripheral edema and *Anurias*.

On the second day of admission a renal challenge was performed to which the kidneys were unresponsive. Intravenous fluid was then restricted in line with conservative management of acute kidney injury when renal challenge fails. Deranged electrolytes and hypoglycaemia were corrected. She was also placed on an antihypertensive *(Amlodipine)*. In addition, she received intravenous *Omeprazole* and *Ranitidine* on account of upper gastrointestinal bleeding. Haemodialysis and upper gastrointestinal endoscopy could however not be done on the second day of admission due to logistic reasons.

On the third day she slipped into unconsciousness and her Glasgow coma scale had dropped to 8/15. A double volume exchange blood transfusion was commenced. The patient's clinical state suddenly deteriorated during the procedure and she succumbed despite all efforts at resuscitation.

RESULTS

She developed persistent vomiting, altered sensorium and acute kidney injury within 48 hours of presentation. The case was 100% fatal. Toluene significantly inhibits the N-Methyl-D-

aspartic acid (NMDA) subtype of glutamate-activated ion channel. It remarkably reduces metabolic function in the brain increases dopamine release and the activity of dopaminergic neurons.

CONCLUSION

Toluene poisoning is associated with high mortality. Early presentation and prompt intervention may lead to a good outcome. Prevention of accidental ingestion of toxic substances at home is key in averting mortality associated with childhood poisoning.

Keywords: Toluene Poisoning, Evostik, Nigerian

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Introduction

Childhood poisoning is one of the causes of high morbidity and mortality especially among under-five children in low income countries.[1, 2]

Toluene(*Methylbenzene,Toluol, Phenylmethane*) is a hydrocarbon compound that can be surprisingly prevalent in the Nigerian household settings being present in common items like gasoline, acrylic paints, varnishes, lacquers, paint thinners, adhesives, glues, rubber cement, and shoe polish where it is used as an industrial solvent or diluent [3, 4].

Evo-stik glue is a modified silane (MS) polymer based high performance adhesive. It contains toluene as a solvent during manufacturing and its the main components [5].

Toluene is a colourless, sweet-smelling liquid, a property which can potentially confer on the containing substance, posing the risk of accidental poisoning in children.

Additionally, being a volatile *lipophilic* compound that readily crosses the blood-brain barrier to cause alterations in mental status, it is recreationally inhaled by the teenage and young adult population especially in developed countries as a substance of abuse It is also known to be a cause of significant morbidity and even mortality in the same population.

Toluene poisoning occurs primarily in three ways: inhalation, ingestion or cutaneous exposure. Pathophysiology of toluene toxicity include its effect on the central nervous system as it readily crosses the blood brain barrier and selectively affects both voltage-gated and ligand-gated ion channels.

Toluene significantly inhibits the N-Methyl-D-aspartic acid (NMDA) subtype of glutamate-activated ion channel. It remarkably reduces metabolic function in the brain. It increases *dopamine* release and the activity of *dopaminergic* neurons [6].

The direct effect of toluene on the skeletal muscles resulting in *rhabdomyolysis* and *myoglobinemia* has also been implicated as one of the causes of acute kidney injury in toluene poisoning [4]

Toluene poisoning can have detrimental effects on virtually all and everybody system. In the central nervous system, it can cause anything from euphoria to coma; in the cardiopulmonary system, it is known to cause aspiration pneumonitis and even sudden cardiac arrest deaths, following *arrhythmias*. Abdominal pain, vomiting and *haematemesis* can also follow toluene ingestion or inhalation.

As a manifestation of gastrointestinal poisoning. *Rhabdomyolysis,* dermatitis and aplastic anaemia have also been reported as musculoskeletal, cutaneous and *hematological* effects of Toluene poisoning respectively.

The renal manifestations of Toluene poisoning include:

i). Renal *Tubular Acidosis,*
ii). Hypokalemia,
ii)i. Hypophosphatemia,
iv). Hyperchloremia,
v). Azotemia,
vi). Sterile *Pyuria,*
vii). Haematuria,
viii). Proteinuria [3, 6].

In the Nigerian setting, Toluene containing substances are likely to be ingested accidentally by younger children who have access to these household items which are stored with little to no extra precaution.

CASE REPORT

QH was a 28 month old girl who presented at the Lagos State University Teaching Hospital Paediatric Emergency with a one day history of persistent vomiting which was preceded by ingestion of 15-20ml of evostik glue one hour to the onset of vomiting.

The glue had been stored in an over-the-counter medication bottle which was familiar to the child. No local remedy was administered. The vomiting was initially spontaneous, non-projectile and non-bilous, and contained evostik particles. It subsequently became post-prandial. There was no associated change in bowel habit, no reduction in urinary output and no abdominal distention. She was initially resuscitated at a private facility with intravenous fluids and parenteral anti-emetics and later referred to LASUTH when clinical condition failed to improve.

She was the third child of a 40 year old civil servant and a 36 year old seamstress, in a monogamous setting. Significant findings on systemic review at admission were the absence of seizures, cough, fast breathing, jaundice and reduction in urine volume.

On admission, she was conscious, moderately dehydrated with dry *buccal mucosa.* She was febrile (38.2ºC), but not pale, icteric, or cyanosed. She had no peripheral oedema. Her respiratory rate was 34

Figure 1: *Bottle Containing The Evostik Ingested By Toddler. The Bottle Had the Label of Baby Rex Baby Mixture Aand Diagram of Children.*

cycles/minute, with vesicular breath sounds and no additional sounds. Pulses were full volume, regular and synchronous. The heart rate was 112 beats/ minute and only the first and second heart sounds were present. Her abdomen was full, moved with respiration, and soft with no palpable organ enlargement. She was fully conscious and alert with coherent speech, normal tone in all limbs, and no signs of meningeal irritation.

She was immediately admitted to the emergency room where fluid management for moderate dehydration was commenced. She developed seizures, recurrent hypoglycaemia, generalized peripheral edema and *anuria* all on the second day of admission. Her vomitus was noticed to contain altered blood. Hypertension with blood pressure reading on the second day of 150/80mmHg was recorded. Serum electrolyte, urea and creatinine done were deranged. Serum sodium was 126mmol/L (Hyponatremia), bicarbonate was 10mmol/L (Metabolic acidosis), Urea was 105mg/dL (Azotemia) with elevated creatinine of 2.89mg/dL.

At the point of development of *anuria,* a renal challenge was performed to which the kidneys were unresponsive. Intravenous fluid was then restricted in line with conservative management of acute kidney injury when renal challenge fails. Deranged electrolytes and hypoglycaemia were corrected. She was also placed on an antihypertensive (Amlodipine). In addition, she had intravenous Omeprazole and Ranitidine on account of upper gastrointestinal bleeding. Haemodialysis and upper gastrointestinal endoscopy could however not be done on the second day of admission due to logistic reasons.

By the third day on admission, she had slipped into unconsciousness, and her Glasgow coma scale had dropped to 8/15.

A double volume exchange blood transfusion was commenced as an alternative, in view of the logistic delay in carrying out haemodialysis. The patient's clinical state suddenly deteriorated during the procedure and she succumbed despite all efforts at resuscitation.

Discussion

Accidental poisoning is a cause of significant morbidity and mortality in developing countries. Affecting predominantly the under-five demographic of any population [1, 7]

In the present case, the patient was 28 months old, falling within the most vulnerable age group. At this age increasing curiosity and propensity to explore coupled with inability to discriminate between harmful and harmless substances puts children within this age range at increased risk of accidental poisoning as demonstrated by this case. Accidental poisoning has been reported in females, although it is more common in males [7]

An additional factor that appeared to increase the risk of accidental poisoning involving household items was storage. When potentially poisonous substances are not stored in a "child-proof" container and out of reach of children, they pose the risk of accidental poisoning to children. Particularly under fives are valnurable due to the relative ease of access [7]

Often, household items that pose major risks of accidental poisoning were stored in containers that originally contained otherwise harmless substances and even food items. Unsuspecting children mistake them for the original content of the container and ingest, causing poisoning.

In the present case, the Toluene-containing domestic glue was stored in a container which previously contained paracetamol, a pleasant tasting syrup, which the patient was probably familiar with, and mistook for the same.

Toluene toxicity occurred following acute or chronic exposure. Toxicity from acute exposure, which usually happened accidentally, could occur among any age group but especially children for some reasons earlier discussed. Toluene is highly dangerous to life or health when the exposure is 500ppm its odor is discernable at a concentration of 8ppm which is 25 times less than the permissible limit of 200ppm [3].

The odor is believed to provide adequate warning of an acute hazardous concentration. Chronic exposure occurs among adults who are workers in hydrocarbon manufacturing factories, due to repeated exposures [3]

Some authors have reported cases of acute exposure to toluene in adults resulting in acute renal failure but none has however been reported in children in the past [4, 8].

The severity of clinical presentation of toluene poisoning following accidental ingestion varies depending on the amount ingested by the victim as well as duration prior to presentation.

The initial emergency clinical features also depend on the exposure route. Accidental exposure via inhalation is more likely to present with features of chemical pneumonitis such as difficulty in breathing, before features of any other complications. This current case presented with persisted vomiting due to gastrointestinal irritation as a result of the route of exposure. However, she subsequently developed other features of systemic toxicity like seizures, loss of consciousness and *oliguria*.

Hence, Clinical manifestation of systemic toxicity of Toluene Poisoning (TP) include:

1. Persistent vomiting
2. Hemathemesis
3. Seizures
4. Altered sensorium
5. *Oliguria/ Anuria*.[3, 6]

Diagnosis of poisoning is largely clinical, particularly due to unavailability of toxicology test in our environment. Complete blood count, Serum electrolyte, Urea, creatinine, liver function test as well as random blood glucose are very important investigations in the management of TP. Chest radiography and pulse oximetry or Arterial blood gas measurement were recommended for severe inhalation exposure or where pulmonary aspiration was suspected.

Management of TP is largely supportive. Fluid and elecrolytes must be monitored carefully and corrected. Bicarbonates therapy should only be given when potassium and calcium have been properly corrected. Hemodialysis and Exchange blood transfusion are modalities of management that may improve outcome if promptly instituted. TP has a fatality of nearly 100%, especially when presentation or management is delayed for any reason. However, a few cases with successful outcome have been reported in adults [4, 8].

No documented antidote for TP yet, prevention is KEY. Parental education is crucial in the prevention of accidental exposure. Parents should in turn teach young children about the dangers of poisons, beginning at an early age. Advice to the parents about the proper storage and labeling of harmful chemicals is vital.

Furthermore, they should be informed about common household products that may be dangerous and recommend steps that they can take to minimize the possibility of an accidental exposure including safe storage of hydrocarbons. Educating parents about supervision of their children, when they are in high-risk areas (eg. kitchen, garage, and laundry room) where toxic substances may be present cannot be over emphasized.

Inhalant abuse occurs in adolescents and should be discouraged. Hydrocarbons may be inhaled for recreation and as part of suicidal gestures and attempts. Treatment of the underlying causes of these behaviors might help in preventing hydrocarbon abuse.

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