

## Alteration in Pain Sensations after Cleaning Solution Ingestion in Children: A Newer Perspective

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### Abstract

Of all the cases of accidental toxins ingestion in toddlers, household cleaning solutions and detergents are quite common. "Pinesol" has been a trade name for many of these common cleaning solutions produced by different manufacturers and all these products contain varying amounts of the pine oil, other constituents of these solutions are alcohol ethoxylates, alkyl sulfonates, glycolic acid and added fragrances as per material safety data sheet.

**Keywords:** Altered pain sensation; Pinesol; Cleaning solution ingestion

### Introduction

Crude pine oil can generally have a starting tertiary alcohol including 1-alpha-terpinol [1] ranging from 20-80% by weight. The toxicity known with pine oil includes mucosal irritation and erosion, acute respiratory system injury including chemical pneumonitis and central nervous system depression, renal injury causing hematuria which may progress to renal failure. There have been previous reports of fatality attributed to pinesol ingestion, its constituents and due to its by-products of metabolism [2].

### Clinical Case

We had a 2 year old child who presented in unresponsive state to the emergency and on reviewing the history with the mother it was reported that the child had ingested an unknown quantity of pinesol in presence of his six year old sister the night prior to presentation. On the morning of presentation to the hospital, the child was wobbly, not vocalizing, and eventually fell while walking to the day care without response to his mother calling out his name. At arrival to the hospital the patient had stable vital signs on room air, no evidence of any head or body trauma. He closed his eyes in response to bright light but was not responsive to touching, calling his name or to noxious stimuli such like supraorbital pressure, squeezing of nail phalanges or to deep sternal rubbing. Neurological examination revealed pupils of small size normally reacting to the light, positive oculoccephalic reflex, normal muscle tone, deep tendon reflexes which were normal and down going plantar response. No limb or body posturing was observed in response to stimuli, modified Glasgow coma scale at presentation was assessed to be 6 (1-eye opening, 1-Verbal, 4-withdrawal on sole scratching as the best motor response). Head CT (non-contrast) done in the emergency department was normal, venous blood gas done in emergency showed PH-7.372, PCO<sub>2</sub>-43.7 mmHg, Po<sub>2</sub>-28 mmHg, HCO<sub>3</sub>-25.4 meq, lactate-1.65 mmol/L and child had an Spo<sub>2</sub> of 98% on

room air. Magnesium and phosphate levels done were normal (2.2 and 4.5 mg/dl respectively), salicylates, acetaminophen and urine toxicology for commonly used drugs (barbiturates, PCP, cocaine and methadone) were negative. B-OH butyrate levels done to rule out other metabolic disturbances was 0.261 mmol/L (N-<0.5). Metabolic profile done in the emergency room showed Na-138 meq, K-4.4 meq, CL-105 meq, glucose-83 mg/dl, blood urea nitrogen of 7 mg/dl and creatinine of 0.307 mg/dl, calcium was 9.6 mg/dl and serum ammonia done was also normal (32 umol/L). EEG done showed pattern characteristic for wakefulness, and without epileptiform discharges. He was admitted to PICU for monitoring and started on maintenance i.e. fluids, heart rate, Spo<sub>2</sub> and blood pressure remained stable on room air.

On day 2 of hospital admission the child had improvement in his sensorium in terms of intermittently opening eyes with visual attention, on being pulled into sitting position and called his name by his mother. He was able to perform simple motor tasks like manipulating a torchlight but went back to sleep soon after. The child however still did not respond to painful stimuli in the form like squeezing of nail bed, supraorbital pressure or venous blood drawn in form of a withdrawal or grimace, modified GCS was 11 at that time (4-motor, 4-eye opening, verbal response-3). There was thus dissociation in terms of improvement in his mental status however his response to pain continued to be impaired. There was also evidence of some oral lesions suggestive of shallow mucosal ulcerations and erythema on the tongue suggestive of the ingestion of solvent/irritant.

Also confounding the presentation was the multiple medications that the mother was carrying with her for bipolar disorder and included fluphenazine, benzotropine, bupropion, valproate, lithium. Levels of all these medications were negative when tested for in the blood were normal, also serum electrolyte levels repeated on day 2 of hospitalization remained normal (see Table 1 in appendix for results of laboratory testing). In view of clinical improvement in terms of his sensorium with stable vitals on room air and the ingestion happening more than 48 hours earlier, no further testing for the levels of the ingested substance was done.

	Day 0 (at admission)	Day 1 (PICU)	Day 2 (PICU)	Day 3(discharge)
GCS	6	11	14	15
Pain response	None	None	Withdrawal to lancet prick poorly localized	Withdrawal localized to location of painful stimuli (pin prick, nail bed pressure)
	(no withdrawal to sternal, supraorbital pressure)	(no withdrawal to pin/lancet prick, squeezing of nail bed, sternal pressure)		
VBG	pH-7.372, PCO <sub>2</sub> -43.7 mm, Lactate-1.65 mmol/L			
	HCO <sub>3</sub> -25.4 mmol/L			
Metabolic panel	Na-138 mmol/l, K-4.4 mmol/L, Cl-105 mmol/L, Glucose -83 mg/dl, Ca-9.6 mg/dl, BUN-7 mg/dl, Creatinine-0.307 mg/dl, Mg-2.2 mg/dl, PO <sub>4</sub> -4.8 mg/dl		Na-140 mmol/L, K-4.7 mmol/L, Cl-109mmol/L, HCO <sub>3</sub> -21 mmol/L, Glucose -96 mg/dl, Calcium -9.2 mg/dl, BUN-5 mg/dl, Creatinine-0.315mg/dl	
B OH butyrate	0.261 mmol/L			
NH <sub>3</sub>	32 mmol/L			
Acetaminophen	2 ug/ml			
LFT	AST-47 U/L, ALT-24 U/L, ALP-286 U/L, bili-0.4mg/dl		AST-33 U/L, ALT-23 U/L, ALP-250 U/L, bili-0.3 mg/dl	
Fluphenazine	<0.2 ng/ml			
Benztropine	<2 ng/ml			
Lithium	<0.2 mmol/L			
Valproate	<3 ug/ml			

**Table 1:** Depicts the change in glasgow scale score and laboratory testing done to rule out other causes of altered sensorium and pain sensations.

The child was discharged on day 3 of hospitalization and at discharge was opening eyes spontaneously, vocalizing meaningful words and was interactive, walking with a normal gait (modified GCS-15) and was grimacing, withdrawing meaningfully to previously tested painful stimuli. He was seen in neurology outpatient clinic 1 week following discharge and had been doing well as per mother who had brought the empty bottle of pinesol which the child had ingested.

## Discussion

Pine oil has is one of the essential oils which have long been used for various herbal remedies in China and middle east Asia and has been noted to be a major constituent of herbs -*Angelica sinensis* and *Aurantii fructu* [3]. Alpha-terpinol, β-pinene and γ-terpinen have been identified as major components of these herbs which are used for the extraction of essential oils. Other protective effects of these essential oils like antimicrobial properties and hypolipidemic effects have been postulated in various studies earlier [4,5].

Pine oil has been a major constituent also of the various household cleaning solutions in different quantities and thereby giving these groups of products their market name of 'Pinesol'. There has been previous case reports of fatality associated with pinesol ingestion

attributed to chemical pneumonitis causing non cardiogenic pulmonary edema and with acetone formed as a by-product from metabolism of isopropanol which is also a constituent. Alpha-terpinol, β-pinene and γ-terpinen as mentioned earlier have been proven to be a major constituent of the pine oil by gas chromatographic studies. These alcohol ethoxylates and other constituents of "Pinesol" are associated with signs of mucosal irritation and central nervous system depression seen on ingestion. Monoterpenes including alpha terpinol have been known to have various pharmacological properties like anxiolysis, antidepressant, anticonvulsant, however as per recent studies alpha terpinol has been shown to have antihypernociceptive properties [6] and can reduce the mechanical hypernociception induced by various acute phase reactants and cytokines like PGE<sub>2</sub>, IL-1 B, TNF-alpha, glutamate, nitric oxide [7] and also dopamine in mice models [8]. The cause of the discrepancy seen in improvement of sensorium this patient compared to the decreased pain perception can therefore be attributed to the alpha-terpinol which could predict clinical course in further cases of intoxication with similar solutions.

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