

Case Report

Paint Thinner Ingestion-induced Stroke in a Young Male

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ABSTRACT

Previous reports of paint thinner ingestion were suggestive of rhabdomyolysis, polyneuropathy, chemical pneumonia and coma. Reports of cerebral stroke following thinner ingestion have almost never been reported.

A healthy male presented to us with unconsciousness and hemiplegia following deliberate ingestion of paint thinner. Magnetic Resonance Imaging (MRI) of head revealed cerebral infarct and multifocal oedema.

Key Words: Paint thinner; Cerebral stroke

Introduction

A paint thinner is a solvent used to thin oil-based paints or clean up after their use, besides being used as an industrial solvent for the manufacture of pharmaceuticals and several chemicals.¹ Many such thinners contain aromatic hydrocarbons such as xylene, toluene and N-hexane. Poisoning can occur due to ingestion, inhalation or transdermal absorption. Toluene abuse or “glue sniffing” has become widespread, especially among children or adolescents, because it is readily available and inexpensive. In recent decades, the organic solvent toluene (methylbenzene) has emerged as one of the best-studied neurotoxins.² BAERs (Brain stem auditory evoked responses) may detect CNS injury from toluene inhalation even at a time when neurological examination and MRI scans are normal.³

Previous reports of thinner ingestion have listed rhabdomyolysis, polyneuropathy, chemical pneumonia and

coma as common features.⁴ One report claimed methaemoglobinaemia as a consequence of thinner ingestion.⁵ We present a case of a patient who presented with cerebral stroke following thinner ingestion.

Case Report: A 35 year-old, previously healthy male was referred to us from a neighbouring district following ingestion of a thinner approximately six hours prior to admission. The exact quantity consumed could not be ascertained. He was unconscious; there were no convulsions during or prior to admission. His pulse rate was 84/minute, and blood pressure was 110/90 mmHg; respiratory rate was 26 per minute, and on auscultation, bilateral rales and rhonchi were audible. Central nervous system examination revealed gross weakness of the left half of the body (power-grade 2) compared to the right (power-grade 4), with bilateral extensor plantar response. Pupils were equal and reacting to light. In view of the weakness in the limbs and unconsciousness, an MRI (magnetic resonance imaging) of the brain was done, which revealed multifocal oedema in both cerebral hemispheres, and enhancement in both cerebral hemispheres, more marked in the right temporo-parietal lobe involving cortex, suggestive of an infarct (**Fig 1**).

The patient was managed on supportive lines. Respiratory symptoms which were evident at presentation normalized within the next few days. Transient leucocytosis with elevation of liver enzymes were the only abnormal biochemical findings (**Table 1**). Though the patient survived, there was no obvious improvement in his neurological status and higher mental functions.

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Table 1 Biochemical Investigation Results

Investigations	Day 2	Day 7
Hb (gm%)	10.3	9.8
TLC (/mm ³)	12000	5300
DLC	P83L12E2	P78L20E1
Blood urea (mg/dl)	38	20
Serum creatinine (mg/dl)	0.74	1.09
Na+ (mmol/litre)	-	139.71
K+ (mmol/litre)	-	3.32
Serum bilirubin (mg%)	1.66	.06
SGOT (IU/L)	109	114
SGPT (IU/L)	41	80
Alkaline phosphatase (IU/L)	113	192
Serum protein (gm%)	6.97	7.5
Albumin (gm%)	3.4	4.0
Blood sugar (mg%)	157	95

**Fig 1** MRI Brain Showing Multifocal Oedema with Enhancement in Both Cerebral Hemispheres - More Marked in Right Temporo-Parietal Lobe Involving Cortex**Discussion**

Long-term and intense exposure to toluene vapours in humans who abuse spray paint and related substances has led to the recognition that toluene has a severe impact on central nervous system myelin.² Chronic toluene abuse can lead to dementia.² Distribution of absorbed toluene and xylene in humans and rodents is characterized by preferential uptake in well-perfused and lipophilic tissues such as the brain, liver, lungs, and body fat.¹ There are previous reports of acute thinner intoxication leading to rhabdomyolysis, polyneuropathy, chemical pneumonia and coma.⁴ One report claimed methaemoglobinaemia as its consequence.⁵

Our patient presented with cerebral stroke and multifocal cerebral oedema. Central nervous system manifestations were a prominent feature. Other features included transient elevation in liver enzymes and leucocytosis. Respiratory symptoms were self limiting.

This case report serves to indicate that thinner ingestion has the propensity to involve mainly the central nervous system, and that the outcome is difficult to predict.

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