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SEVERE ACUTE THINNER INTOXICATION*

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Thinner which contains aromatic hydrocarbons such as xylene, toluene and N-hexane is widely used in industrial plants manufacturing dyes, plastic, varnishes and glues. Chronic intoxication due to abuse of solvents, including thinner, by workers who inhale the solvent vapor is frequently encountered. Acute intoxication with ingestion of excessive amounts is relatively rare and usually fatal. It is reported that 45-50 ml of orally ingested thinner is enough to cause severe complications.

The case reported here was forced to drink 200 ml of thinner by an older friend, and presented with severe complications such as rhabdomyolysis, polyneuropathy, chemical pneumonia and coma. To the best of our knowledge this is the first case reported in the literature to survive acute thinner intoxication with such complications.
Key words: aromatic hydrocarbons, toxic polyneuropathy, toluene intoxication.

Thinner which contains aromatic hydrocarbons, such as toluene, xylene and N-hexane, is widely used in the industrial manufacturing of plastics, varnishes, paints and glues. Chronic intoxication due to abuse of solvents, including thinner, and among industrial workers who inhale solvent vapor is frequently observed[1-5]. However, acute intoxication due to ingestion of excessive amounts is unusual and often fatal. This patient, who was forced to drink approximately 200 ml of thinner by his friends, presented with severe complications related to aromatic hydrocarbons. Although the estimated lethal, oral dose of thinner is reported to be 45-50 ml, this patient survived after one month in intensive care[2].

Case Report

A 14-year-old male patient presented with severe muscle weakness, difficulty in walking, edema of the extremities and neck, and accompaning oral ulcerations. The initial examination findings were as follows: heart rate 130 bpm, blood pressure 110/70 mmHg, respiratory distress and rales on auscultation, severe muscle weakness, flaccid quadriplegia, absence of deep tendon reflexes and stupor. Fasciculation and Babinski sign positivity were not observed. Two hours after he was admitted to the hospital, he experienced a cardiopulmonary arrest. He was resuscitated and put on mechanical ventilation, since spontaneous

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respirations were absent. Laboratory investigations were as follows: hemoglobin 10.6 g/dl, reticulocytes 1.8%, leukocytes 17,600/mm^3 and platelets 113,000/mm^3. Urinalysis revealed mild proteinuria and microscopic hematuria. Serum sodium was 132 mmol/L, potassium 7.2 mmol/L, AST 2,484 IU/L, ALT 548 IU/L, lactate dehydrogenase (LDH) 8,096 U/L (150-320 U/L), creatine kinase (CPK) 121, 168 U/L (25-90 U/L), and myocardial CPK (CK-MB) 1,860 U/L (5-35 U/L). BUN, glucose and other electrolytes were within normal limits. Antinuclear antibody was negative. Chest roentgenogram revealed marked cardiomegaly, patchy infiltration, areas of atelectasis and bilateral pleural effusion. Voltages were depressed in all derivations in the ECG, indicating myocardial damage. Electromyographic features revealed a mixed (motor, sensory and otonomous), mainly axonal polyneuropathy. Sural nerve biopsy showed axonal degeneration and segmental demyelination. Biceps muscle biopsy displayed muscle fiber degeneration and an increase in connective tissue. Bone marrow aspirate was normal. The initial diagnosis was fulminant polymyositis according to the clinical and laboratory findings. On the 7th day, in an effort to clarify the etiology of this clinical course, the patient admitted that he had been forced to drink approximately 200 ml of thinner by his friends at the plant where he was working. The thinner consumed at this plant was found to contain 60 percent toluene, 25 percent N-hexane and five percent xylene. These findings completely explained the severity of the clinical course. He was initially put on mechanical ventilation and supported with vitamins B1, B6 and E, and physical therapy and rehabilitation were initiated on day 15. He was extubated on the 7th day of his admission, and by the end of the second month of hospitalization, all the pathological laboratory findings had returned to normal except for severe mixed polyneuropathy (Fig. 1).

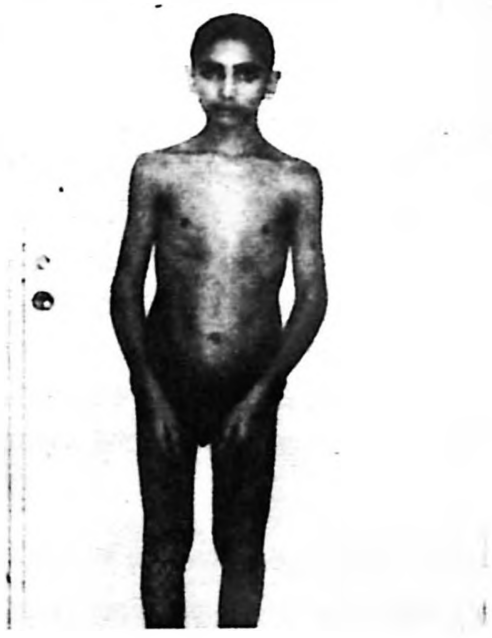

Fig. 1: The appearance of the patient with severe muscular atrophy after treatment.

Discussion

Abuse of thinner containing toluene is very common, and this solvent is the most common agent among the hydrocarbons[2, 4]. Toluene appears to have more severe, acute effects when combined with N-hexane[3]. Toluene and xylene cause generalized central nervous system depression, which may cause ataxia, convulsion, coma and respiratory arrest. Like other hydrocarbons, they may sensitize the myocardium to the arrhythmogenic effects of catecholamines. Ventricular arrhythmias and cardiac arrest may occur. Pulmonary aspiration can cause pulmonary edema and hemorrhagic alveolitis. A high dose of toluene exposure may produce rhabdomyolysis and myocardial involvement with elevated CPK and CK-MB levels[1, 2]. Acute exposure of toluene in this patient led to severe rhabdomyolysis, muscle atrophy and myocardial involvement. Hepatomegaly and centrilobular hepatic necrosis have also been described in the literature in cases of acute exposure[1]. Bone marrow depression has not been described as a result of either chronic or acute exposure to toluene, in contrast with benzene, which produces bone marrow toxicity. N-hexane rather than toluene is suggested to be chiefly responsible for polyneuropathy[3]. The renal abnormalities encountered in toluene sniffers include proteinuria, hematuria and distal renal tubular acidosis[4]. It has been reported that clinical recovery takes up to three years in the most severe cases[3, 5].

We considered this case to be interesting because, to our knowledge, there is no reported surviving case in the literature with such severe complications. We would like to warn physicians to suspect intoxication with aromatic hydrocarbons in patients presenting with rhabdomyolysis, severe polyneuropathy and coma.

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