Case Report

Alkyl Succinate Poisoning

M Raj*, DKS Subrahmanyam**, A Agrawal***, KR Sethuraman+

Abstract
A case of alkyl succinate poisoning is being reported. Oral ingestion of this compound led to gastrointestinal tract involvement and central nervous system manifestations suggestive of parkinsonism. The patient recovered completely following conservative management without any sequelae. ©

INTRODUCTION

Pesticide poisoning is a common mode of poisoning in our country and is the commonest poisoning we encounter in our hospital. With the increasing number of agricultural poisons, consumption of relatively unknown compounds is becoming a therapeutic challenge as there is a paucity of literature. Here we report a case of alkyl succinate poisoning which to the best of our knowledge has not been reported earlier in the literature.

CASE REPORT

A 45 years female presented to us six hours after consuming 100 ml of an unknown chemical compound used for agricultural purpose. Soon after ingestion she developed repeated vomiting and three hours later developed watery diarrhea along with diffuse abdominal pain.

Physical examination revealed her to be conscious, cooperative with normal build and nourishment. There was no pallor, icterus, cyanosis, clubbing or edema. Her blood pressure was 120/80 mm Hg and pulse rate of 100 per min. Examination of cardiovascular, respiratory and central nervous system were normal. Abdominal examination showed a soft abdomen with mild epigastric and lower abdominal tenderness.

The empty bottle which was brought to us by the relatives revealed it to be containing an antitranspirant by the brand name of SALUTE with active ingredients: Alkyl succinate 20% and biologically derived hydrolysed proteins 20% and rest inert carriers. There was no mention of symptoms of toxicity or antidote on the cover. An extensive literature search could not yield any useful data. The only thing we could find out was that antitranspirants are substances that minimizes water loss from plant leaves (transpiration), and they do so either by inhibiting the metabolism or by acting as a mechanical barrier. Although the compound is considered toxic to animals, no human cases of poisoning have been yet reported. As there is no antidote, she was treated with stomach wash followed by activated charcoal and IV fluids, and was kept under observation.

She had 10 to 12 episodes of diarrhea throughout the night along with vomiting. By next morning she started having bloody diarrhea. Her vitals remained stable and fluid replacement was continued along with IV proton pump inhibitors. There were no other bleeding manifestations.

Investigations at that time showed hemoglobin 12 gms/dl, TLC 21,000/cmm, with polymorphs 72% and lymphocytes 28% and platelets 1,50,000. Her blood urea was 23 mg%, serum creatinine 1 mg%, sodium 138 mmol/L, potassium 4.6 mmol/L. Liver function tests were normal. Prothrombin time was 16 seconds (control 15 s). Her bleeding and clotting time were normal.

By the third day the diarrhea stopped and she was started on oral feeds. But on the 4th day, she became drowsy and couldn’t stand with out support. There was no history of diplopia, swallowing difficulty or other symptoms of cranial nerve involvement. There was no weakness or sensory symptoms. There were no bowel or bladder symptoms. At neurological examination she was drowsy but power was normal in all the four limbs. There were tremors at rest in both hands and tone was increased in all four limbs with cog wheel type of rigidity. Deep tendon reflexes were normal and plantars were bilateral flexor. Superficial reflexes were normal and there were no release reflexes. Sensory system examination was normal. There was no nystagmus or cerebellar signs. There were no meningeal signs. Fundus examination did not reveal any abnormality. CT scan of brain was normal. Repeated blood biochemistry did not reveal any abnormality.

*PG Student, **Associate Professor, ***Professor, +Professor and Head, Department of Medicine, JIPMER, Pondicherry. Received : 8.8.2005; Revised : 9.1.2006; Accepted : 11.9.2006
A clinical diagnosis of toxin induced parkinsonism was made and she was started on trihexyphenydyl 2 mg three times a day. On second day of starting treatment, rigidity and tremors reduced significantly and she was able to walk with support. Trihexyphenydyl was continued in the same dosage and she was discharged on day thirteenth. At the time of discharge she had only mild tremors in her hand and she was able to walk unaided. Two weeks later when she came for follow up, she was normal without any tremors or rigidity and trihexyphenydyl was gradually tapered over one week. When last seen she was doing well without any sequelae.

**DISCUSSION**

Environmental toxicants and, in particular, pesticides have been implicated as risk factors in Parkinson’s disease.\(^1\) Case reports in humans and animal studies have postulated possible role for herbicide paraquat, fungicide maneb, and trichlor ethylene.

To the best of our knowledge there are no reports linking alkyl succinate and parkinsonism in literature. Alkyl and dialkyl succinic acid are listed as toxic compounds under the class of surfactants and are key ingredients in antitranspirants. They are mainly used to make the plants more resistant to drought and have no pesticidal properties. There are studies on animal toxicity of this compound but information on human toxicity couldn’t be found despite extensive search.

Succinic acid and its salts has been found to inhibit liver enzyme acetyl-CoA carboxylase and phosphodiesterase in animals.\(^2\) Animal studies in rats have shown that oral ingestion of salts of succinic acid can cause severe GI irritation, hemorrhage of the gastrointestinal tract, diarrhoea and death in high doses.\(^3\) Dermal toxicity has also been described. There were no reports on CNS toxicity.

Our patient was apparently normal prior to and during the first few days of consuming the compound except for the GI irritation manifesting as bloody diarrhea. She subsequently went on to develop CNS manifestations suggestive of parkinsonism. The fact that her symptoms of giddiness, resting tremor and cog wheel rigidity were temporally related to the toxin consumption and that they improved dramatically with trihexyphenydyl, prompted the author to make a diagnosis of toxin induced parkinsonism. But a key element of parkinsonism, akinesia, was absent.

**CONCLUSION**

There are no reports of human toxicity for this compound. Our patient showed features of gastrointestinal toxicity, CNS toxicity and leukocytosis and she recovered completely with conservative management. The causal link between this product and parkinsonism is only suggestive and not conclusively proven beyond doubt. Further studies are required in this regard to elucidate the exact toxicity profile of alkyl succinate and related compounds.

**REFERENCES**


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