Fatal case of commercial moisture absorber ingestion

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DESCRIPTION

One of the most commercially available moisture absorbers is a dehydrating agent containing calcium chloride. In humid environments such as basements, kitchens, cabinets and closets, calcium chloride provides effective moisture absorption by becoming highly concentrated calcium chloride brine. Ingestion of calcium chloride can lead to severe hypercalcaemia and can be fatal. In our case, hypercalcaemia developed from intentional ingestion of moisture absorber and the patient eventually died. To our knowledge, fatality from commercial moisture of absorber ingestion has not been reported. A 31-year-old man presented to the emergency department (ED) 20 min after intentionally ingesting 300 mL of commercial moisture absorber solution. It contained 300 g of calcium chloride (figure 1). The patient had a history of a pervasive developmental disorder. When the patient first presented to ED, he was alert. The patient's initial symptoms were nausea and vomiting. Gastric lavage was done, and activated charcoal was used. Although initial vital signs were relatively stable, initial ECGs revealed sinus bradycardia (36 bpm), with corrected QT interval of 421 ms (<430 ms) which, in subsequent study, increased to 512 ms. The results of the laboratory studies were as follows: serum total calcium 20.7 mg/dL (8.2–10.8 mg/dL), ionised calcium 3.82 mmol/L (1.13–1.32 mmol/L), blood urea nitrogen 18.9 mg/dL (7.8–22.0 mg/dL), creatinine 0.78 mg/dL (0.7–1.2 mg/dL), albumin 4.2 g/dL (3.1–5.2 g/dL), phosphorus 5.3 mg/dL (2.5–5.5 mg/dL), creatine kinase 94 IU/L (38–174 IU/L), sodium 139 mmol/L, potassium 4.0 mmol/L and chloride 107 mmol/L. Blood analysis results were pH 7.37, $paCO_2$ 45 mm Hg, paO_2 81 mmHg and bicarbonate 26 mmol/L on room air. Urinary calcium excretion was not measured. Patient was given adequate normal saline, pamidronate and then furosemide intravenously. The second arterial blood gas analysis was done 1 hour and 40 min after the patient's arrival. pH of 7.25, $PaCO_2$ 43 mm Hg, PaO_2 123 mm Hg and bicarbonate 18.9 mmol/L were measured. For the treatment of advanced metabolic acidosis, we had to manage it with haemodialysis. At that time, however, there was no continuous renal replacement therapy (CRRT) equipment available at our hospital, so the patient had to be transferred to nearby hospital. CRRT device w as applied immediately after the patient's transfer, but unfortunately symptoms. did not improve. On the fifth day of hospitalisation, the patient eventually died of multiple organ failure. Hypercalcaemia has many causes; more than 90% of cases result from primary hyperthyroidism or malignancy.[1] Other causes of hypercalcaemia include chronic lithium therapy, thiazide diuretics, pheochromocytoma, adrenal insufficiency, theophylline toxicity and vitamin D poisoning,[2] and there was a fatal case of hypercalcaemia due to calcium channel blocker ingestion. In general, treatment of hypercalcaemia is aimed at lowering serum calcium levels. Despite best efforts, the patient remained in shock with signs of renal failure while on CRRT and eventually hepatic failure ensued. The cause of death was announced as multiple organ failure due to complications of hypercalcaemia by the treating medical team.

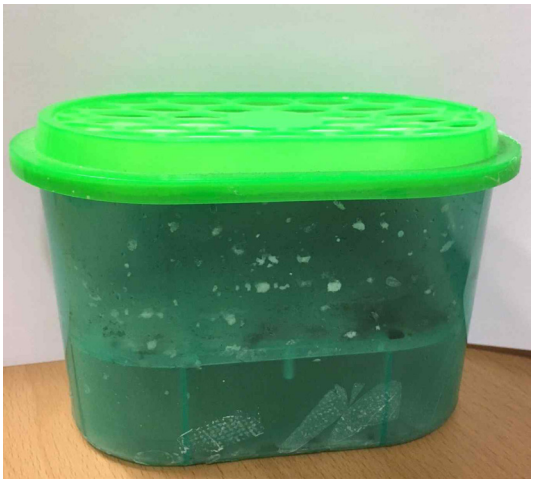

Figure 1 Ingested commercial moisture absorber.

Contributors KC, BS and HY performed history and examination, acquired diagnostic data and studies, prepared the manuscript and images and reviewed the literature. HY designed the treatment plan. HK and HY wrote and edited the manuscript and prepared the learning points. HY prepared the final version and approval of the manuscript.

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Patient consent Parental/guardian consent obtained.

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