PP036 ACUTE INGESTION OF ZINC PHOSPHIDE WITH FATAL OUTCOME.

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INTRODUCTION

Rodenticide poisoning is relatively common poisoning in Malaysia. There are 205 total recorded case from 2006-2016 which made 0.5% of total poisoning in Malaysia.[1] Common rodenticides are Coumadin or zinc phosphide based. Zinc phosphide powder reacts with water to release phosphine gas and even more vigorously in presence of gastric juices. The three most common clinical presentations were gastrointestinal (profuse vomiting and abdominal pain:100%), cardiovascular (palpitation and sweating:80%), and respiratory (dyspnoea and tachypnoea:75%) signs and symptoms.[2] Mortality is high with ingestions exceeding 1gm.[3]

CASE HISTORY

A 39-year-old Chinese gentleman with underlying depression was brought to Alor Gajah Hospital, Melaka with severe stomach ache, profuse vomiting and diarrhoea. Initial diagnosis of acute gastroenteritis quickly revised as he later informed ingesting 8 sachets of "TIKUSAN–MICEKILLER" (Figure 1) containing 4g of zinc phosphide each, 5-hour prior to presentation.

On presentation, he was conscious with GCS of 15/15, BP of 130/80 mmHg, pulse rate 122/min, SpO_2 100% on room air, temperature of 37 C and respiratory rate of 26 breath/minute. His capillary blood sugar was 15 mmol/l and serum ketone 0.1 mmol/l. Abdomen was soft, not guarded with hyperactive bowel sounds and other systemic examinations were unremarkable.

His blood investigations revealed elevated TWBC of 21.3 $x10^9$/L, Hb of 16.4 g/dL, platelet 308 x 10^9/L. Renal profiles urea 1.97 mmol/L, creatinine 97 umol/L, Na 131mmol/L, K 3.4. mmol/L, coagulation profile showed INR of 6.66 and PT of 60.4 while his liver panel showed mildly elevated ALP of 134 IU/L, ALT 11.81 IU/L, TB 9.02 ummol/L and albumin of 47.60g/L.

Over the next hours he deteriorated and required intubation for worsening respiratory distress. Post intubation he developed hypotension and supraventicular tachycardia (SVT) that successfully terminated after four attempts of synchronized cardioversion and pharmacotherapy. His blood gas showed severe metabolic acidosis, pH of 6.92, HCO_3 7 mmol/L, pCO_2 30 mmHg, pO_2 28 mmHg, BE -28. He received a bolus of sodium bicarbonate 70 mmol and the Toxicologist on-call was consulted. Patient arrived at isolation resuscitation bay at approximately nine and half hours post ingestion and was in hypotensive state (BP 56/37, pulse rate 124) with cardiac monitor showing accelerated junctional rhythms (see Figure 2). He had received a total of three litres of normal saline and a litre of Gelafusine. Bedside echo showed global hypokinesia with distended plethoric IVC. Intravenous infusion Noradrenaline started at 0.8mcg/kg/min with another bolus of 1mmol/kg of $NaHCO_3$ given. Intravenous $MgSO_4$ 2gm over 20 mins loaded in view of prolonged QT and controlled hyperventilation targeted in view of severe metabolic acidosis. Right prior departure to ICU, he developed ventricular fibrillation (VF) and required 30 minutes resuscitation. He was defibrillated using biphasic machine 200J x 5 times, IV adrenaline 1mg x 5, IV amiodarone 300mg x1; 150mg x1, IV calcium gluconate 1 gm, IV $NaHCO_3$ 70 mmol leading to return of spontaneous circulation. High dose insulin therapy started at 1mmol/kg/h