A Rare Survival in Celphos Poisoning

Praveen Kumar P[1], Babu M[1], Poornima Nair[1], Rajeswari[1], Sreejith V Ravi[1], Sivakumar K[2], Sanbakasree[2], Raveendran M[3]

Abstract

Aluminium phosphide poisoning releases phosphine gas which causes inhibition of cytochrome oxidase, inhibition of electron transport chain and thereby myocardial suppression. It is known to cause various electric abnormalities in the heart from ST-T depression to fatal tachyarrhythmias. Here we present a case of celphos poisoning presenting with both supraventricular tachycardia and ventricular tachycardia.

Introduction

Celphos (Aluminium Phosphide) is one of the most common suicidal poisoning agent in southern India. It can easily be bought over the counter and has no effective antidote. Its toxicity results from the release of phosphine gas when the tablet gets into contact with moisture. Phosphine gas primarily affects heart, lungs, gastrointestinal tract and the kidneys. Here we present a case of Celphos poisoning with varying electric abnormalities in the heart.

Case Report

55 year old male was referred from a private hospital with alleged history of consumption of 2 celphos tablets (kurunai marundhu). He was given stomach wash (contraindicated) and referred here. On receiving in the ER room, patient was drowsy, restless with SpO_2 95%, BP 90/60 mmHg. He was treated with initial fluid challenge with 2 litres normal saline. BP improved to 110/70 mmHg. About 500 ml of coconut oil was then instilled through the Ryles tube. Multidose Activated charcoal was then given through the Ryles tube. Patient was still drowsy and restless with altered sensorium. ECG taken showed supraventricular tachycardia. (Figure 1) Patient treated with vagal massage and initial Inj. Adenosine 6 mg. It was reverted to normal sinus rhythm. After 15 minutes patient had feeble pulse with blood pressure not recordable. Multiparameter showed Ventricular Tachycardia (Figure 2). He was given 50 J of DC shock. It reverted to sinus rhythm. Patient was started on inotropic support and iv fluids rushed. Due to hemodynamic instability and decreased responsiveness, patient was intubated and connected to mechanical ventilation. As Insulin is known to increase the cardiac contractility GIK regimen was started (100 ml 25%Dextrose + 10ml KCl + 8 U rapid Insulin). $MgSO_4$ 2 mg iv stat was given followed by 4mg in 500 ml NS over 4 hours. ECG monitor showed intermittent ventricular premature contractions with paroxysmal supraventricular tachycardia. Intravenous Amiodarone was tried but with no beneficial effect. Bedside echo was done showed Left ventricular systolic dysfunction grade II with ejection fraction 51%. No wall motion abnormality was present. After 3 hours inotropic support was tapered, Patient was weaned from ventilator after 6 hours.

By that time blood investigations were available which showed normal

[1]Junior Resident in General Medicine, [2]Assistant Professor General Medicine, [3]Professor of Medicine, Coimbatore Medical College Hospital, Coimbatore, Tamil Nadu

Received: 02.08.2016; Revised: 12.11.2016; Accepted: 31.01.2017

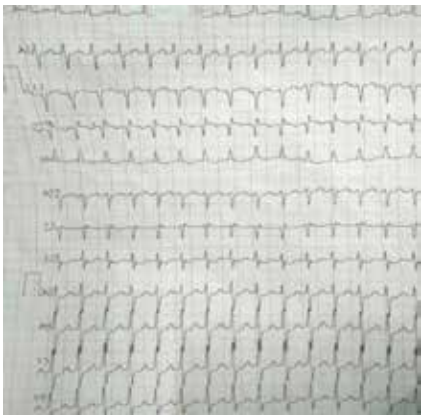

Fig. 1: Supraventricular tachycardia

hemogram with normal renal function tests and electrolytes. ABG analysis showed compensated metabolic acidosis. LFT showed Bilirubin 1.4 mg% with direct 0.6mg% and indirect 0.8 mg%. SGOT was 102 U/L and SGPT 88 U/L. CPK was 383 (normal 20-200U/L). Serum Magnesium was 1.6 mg/dl (1.5 – 2.5 mg/dl) and calcium 8 mg/dl. Patient was then treated with Inj. N Acetyl cysteine for 3 days. Liver enzymes returned to normal. Repeat ECG (Figure 3) and Echo showed normal systolic function with EF-65%. Patient was then discharged.

Discussion

Aluminium Phosphide Poisoning, a solid fumigant pesticide (for stored cereal grains), widely used in India (Quickphos, Celphos, Rice tablet). Most commonly used for suicidal deaths, in North India. Each Tablet weighs 3gm and liberates 1gm of phosphine (PH3) gas which has high dissolution and diffusion capacities. For the silver nitrate test on gastric aspirate, diluted gastric content is heated in a flask up to 50°C for 15-20 mins, keeping silver nitrate paper on the mouth of the flask. If phosphine is present then the paper will turn black due to silver phosphate. The aim of therapy is to sustain life till phosphine gets excreted from the body. Antacids 60ml per hour may reduce PH3 absorption. There is no antidote to phosphine. Magnesium Sulphate has membrane stabilizing properties. Dose 1gm iv stat then 1gm iv hourly for three hours, 1gm continuous infusion daily for 4 to 7 days. It does not have morality reduction benefits. Mortality is still high from 30 to 100% depending upon whether the tablets are fresh ones opened from new packs or old exposed tablets. The management of

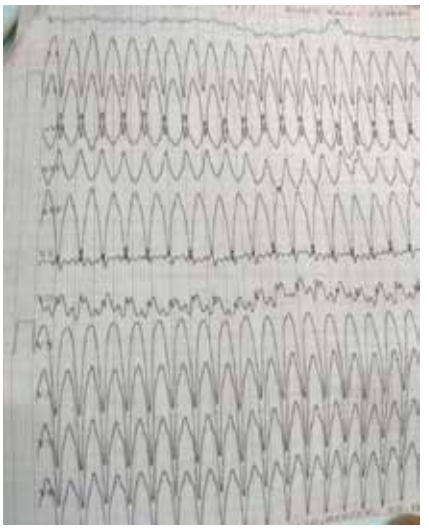

Fig. 2: Ventricular tachycardia

Celphos poisoning is still supportive therapy. After ingestion, removal of unabsorbed poison from the gut ("gut decontamination"), especially if administered within 1–2 hours, can be effective. Potassium permanganate (1:10,000) gastric lavage can decompose the toxin. The rationale behind the use of a mixture of soda bicarbonate and coconut oil in our patients is guided by the chemical reaction of AlP with moisture and HCl, liberating phosphine gas which rapidly gets absorbed through gastric mucosa. As the poison itself causes a lot of gastric mucosal damage, it exposes a lot of raw area for phosphine absorption. The mechanism by which coconut oil reduces the toxicity of phosphides is unknown but most probably it forms a protective layer around the gastric mucosa, thereby preventing the absorption of phosphine gas. Secondly, it helps in diluting the HCl and again inhibiting the breakdown of phosphide from the pellet.

While treating a patient, enquiry regarding manufacturing date of tablet and exposure of tablet prior to ingestion (old or new) guards prognosis. Secondly, duration of ingestion and arrival to hospital and start of management is important. All at admission is diagnosis confirmation by history, vial in hands of attendant and pungent smell, proceeding with ABG (severe acidosis), lactate levels will again guide for further management along with other investigations (as mentioned in manuscript). Profound shock (along with myocarditis, dysrrhythmias, MOFD), is an important

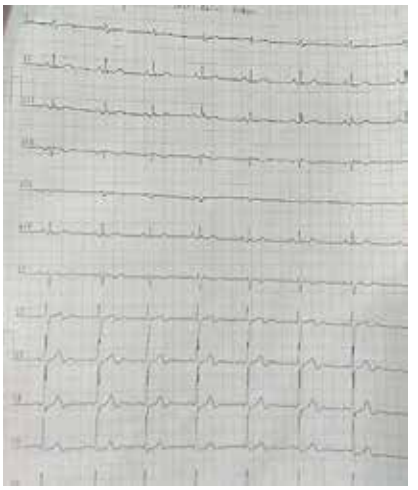

Fig. 3: Normal rhythm at discharge

cause of death as this hypotension is refractory to vasopressors. Amiodarone is a good drug, being used safely in dysrhythmias in such cases.

Refractory myocardial depression from AlP toxicity is very common and carries a very high mortality. Vascular changes may lead to marked low blood pressure that does not respond well to pressor agents. Cardiotoxicity/toxic chemical myocarditis is manifested as depressed left ventricular ejection fraction, ECG changes varying from ST segment elevation/depression, PR prolongation, broad QRS complexes, and right or left bundle branch block, supraventricular ectopics or fibrillation.

Conclusion

Since death is rapid and survival after significant poisoning is difficult, prevention is the logical option. The most effective way for prevention is to either ban or impose strict regulation on the sale of aluminium phosphide tablets. Shielding of tablets in smaller plastic with holes and spikes so that they can't be swallowed as such, is likely to reduce the incidence of Aluminum phosphide poisoning.

References

1. Chugh SN, Arora BB, Malhotra GC. Incidence and outcome of aluminium phosphide poisoning in a hospital study. *Indian J Med Res* 1991; 94:232–5.
2. Singh D, Jit I, Tyagi S. Changing trends in acute poisoning in Chandigarh zone: A 25-year autopsy experience from a tertiary care hospital in northern India. *Am J Forensic Med Pathol* 2009; 20:203–10.
3. Bogle RG, Theron P, Brooks P, Dargan PI, Redhead J. Aluminium phosphide poisoning. *Emerg Med J* 2006; 23:e3.
4. Bogle RG, Theron P, Brooks P, Dargan PI, Redhead J. Aluminium phosphide poisoning. *Emerg Med J* 2006; 23:e3.
5. Singh S, Bhalla A, Verma SK, Gill K. Cytochrome c oxidase inhibition in 26 aluminum phosphide poisoned patients. *Clin Toxicol (Phila)* 2006; 44:155-8.