A Rare Survival in Celphos Poisoning

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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 \( \text{SpO}_2 \) 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...
exposed tablets. The management of
is still high from 30 to 100% depending
morality reduction benefits. Mortality
daily for 4 to 7 days. It does not have
three hours, 1gm continuous infusion
Dose 1gm iv stat then 1gm iv hourly for
has membrane stabilizing properties.
PH3 absorption. There is no antidote
phosphine gets excreted from the body.
The aim of therapy is to sustain life till
will turn black due to silver phosphate.
If phosphine is present then the paper
nitrate paper on the mouth of the flask.
and diffusion capacities. For the silver
gastric content is heated in a flask up
gastric test on gastric aspirate, diluted
and electrocardiograms. For the silver
acidosis. LFT showed Bilirubin 1.4 mg%
showed compensated metabolic
blood gases. ABG analysis with normal
hemogram with normal renal function
tests and electrolytes. ABG analysis
showed compensated metabolic
acidois. 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
mortality 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

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,
dysrhythmias, MOFD), is an important
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
pressors 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 phosphate
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 phosphate poisoning.

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