Fatal yellow phosphorus poisoning in a child

***K Jagadish Kumar**[1], **M Santhosh Kumar**[1], **Divya Sankuru**[1], **Nandakishore Reddy**[1]

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Introduction

Yellow (white) phosphorus is a poison which involves the gastrointestinal, hepatic, cardiovascular, nervous and renal systems[1]. It is used in fireworks and as rodenticides[2]. Rodenticides are available as pastes containing 2 to 5% of yellow phosphorus[1]. Ratol is a commonly used rodenticide in Indian houses and contain 3% yellow phosphorus[3]. We present a case of acute yellow phosphorus poisoning in a 15 year old girl with suicidal intention that led to multi-organ failure and death.

Case report

A 15 year-old girl was brought with a history of consumption of a whole rodenticide paste tube Ratol, containing 3% yellow phosphorus. She was first admitted to a local hospital with gastrointestinal symptoms and weakness on the day of consumption and referred to our hospital after 48 hours. There was no history of altered sensorium, convulsions or oliguria.

On admission, the child was conscious (Glasgow coma scale 15/15), icteric with a temperature of 98^0F, pulse rate (PR) 122/minute, respiratory rate (RR) 32/minute, blood pressure (BP) 90/70 mm Hg and oxygen saturation (SpO_2) 96% in room air. Abdominal examination revealed a 4 cm hepatomegaly. Stomach wash was given. Inj. ceftriaxone and Inj. vitamin K were given and she was commenced on N-acetyl cysteine.

On day 2 of admission, her BP started falling accompanied by cold peripheries because of peripheral collapse. Normal saline boluses (20ml/kg) were given thrice followed by dopamine 10µg/kg/minute. Her blood sugar was monitored. She was conscious but was talking incoherently. Her BP improved and maintenance intravenous fluids were given. Her PR was 90/min, BP 94/68 mm Hg, RR 20/min, SpO_2 100% in room air, capillary filling time (CFT) less than 3 seconds, warm peripheries with IV Fluids at 5ml/kg/hr. Arterial blood gases (ABG) revealed a pH of 7.35, a bicarbonate level of 9.7 mEq/L and a base excess of -13.9. Therefore a slow bicarbonate infusion was started.

On day 3 of admission, her BP started to fall again and she developed hypotension in spite of dopamine infusion. Therefore, epinephrine infusion 0.1 µg/kg/minute was started. She was noted to be irritable, oriented, with a HR of 138/minute, RR of 32/minute, SpO_2 of 98% and BP 92/70 mm Hg. Abdominal examination revealed a 4 cm hepatomegaly. Her 2D echocardiogram was normal. Her blood sugar was maintained normally. The electrocardiogram (ECG) showed ST wave depression. Her prothrombin time (PT) and activated partial thromboplastin time (APTT) were prolonged and therefore, fresh frozen plasma was given.

On day 4 of admission, in spite of dopamine and epinephrine, her BP started decreasing and noradrenaline infusion was started. She was febrile with a RR of 34/minute and a BP of 90/48mm of Hg. Her sensorium started deteriorating (GCS 10/15). Her RR was 58/ minute, BP 84/50mm Hg while on 3 inotropes, SpO_2 88% with 5 litres of oxygen. The chest X- ray showed bilateral parenchymal infiltration. Dobutamine infusion 10 µg/kg/minute was started. She was also given IV piperacillin and tazobactum. ABG revealed hypoxia. Child was put on mechanical ventilator with PRVC mode.

On day 5 of admission, she was still on the mechanical ventilator with PRVC mode. She was having tachycardia (PR 146/minute) and BP was 80/40mm Hg while on 4 inotropes. Her adrenalin dose was increased up to 0.5 µg /kg/minute. In spite of ventilator and 4 inotropes to support the systemic circulation, child deteriorated and expired.

Investigations carried out during the 5 days are shown in Table 1.

[1]*JSS Medical College, JSS University, Mysore, India*

**Correspondence:* jagdishmandya@gmail.com

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Table 1: ***Investigations***

Investigation	Day 1	Day 2	Day 3	Day 4	Day 5
Haemoglobin (g/dL)	12	14			14
Total white cell count (/cu mm)	2700	2610			6880
Platelet count (/ cu mm)	1.052	1.53			90000
Packed cell volume	36.3	42.8			
Blood sugar (mg/dL)	109	366			
Bilirubin (Total & direct) mg/dL	1.14/0.27		3.01/1.28		3.9/3.6
Sodium (mmol/L)	128	130	133	133	132
Potassium (mmol/L)	3	2.5	2.5	2.9	3.8
Chloride (mmol/L)	96		91	91	100
Urea (mg/dL)	40	44	49		29
Creatinine (mg/dL)	0.8	1	1		0.8
Calcium (mg/dL)	7.5	8.7			
Prothrombin time (seconds) /INR	25.3/1.95	54/1.88	64/4.57		46/4.02
Activated partial thromboplastin time (seconds)	33	39.2	46		46.5
Aspartate transaminase (U/L)	47	958	755		135
Alanine transaminase (U/L)	33	659	834		292
Alkaline Phosphatase (U/L)	151				
Magnesium (mg/dL)		2.3	2.83		
CK-MB (ng/mL) (Normal 1-7)		4.86	6.47		
Troponin-T(ng/mL) (Normal 0.0001-0.0249)			0.067		
Echocardiography		Normal; Ejection fraction 60%	Normal		
ECG		Normal	Sinus tachycardia, ST wave depression	Sinus tachycardia, ST wave depression	
Chest X-RAY	Normal		Normal	Bilateral parenchymal infiltration	Bilateral pneumonia
Arterial Blood Gas					
pH	7.39	7.35	7.26	7.49	7.21
Pao2	120	85.1	70.3	93	62.4
PaCo2	22.2	17.8	23.5	22.5	25.2
Bicarbonate	13.3	9.7	10.2	16.8	9.7
Base Excess	-9.9	-13.9	-15.2	-6.1	-16.7
Lactate	5.6	3.2	5.7		7.5
Ionic Calcium	0.72	1.27	2.09	0.9	

Discussion

Yellow phosphorus is a general protoplasmic poison which causes multi-organ failure[1]. The patient passes through three stages. In the first stage, which occurs during the first 24 hours, patient is either asymptomatic or has signs and symptoms of gastrointestinal irritation. The second stage occurs between 24 to 72 hours during which the patient may be asymptomatic and be discharged prematurely. There may be mild elevation of liver enzymes and bilirubin in this stage. The third stage occurs after 72 hours with acute hepatic failure and coagulopathy, until the resolution of symptoms or death[1].

The child reported here had the classical presentation. During the first 48 hours she developed only gastrointestinal symptoms as described. She progressed to acute liver cell failure on the 3rd day of ingestion and was started on N-acetyl cysteine. A few studies have shown improvement with N-acetyl cysteine[4]. Our case succumbed to multi-organ failure. From the day of admission our patient had leucopenia and thrombocytopenia. Basheer et al reported severe leucopenia along with neutropenia secondary to selective myelosuppression in a 14-year-old girl following ingestion of yellow phosphorus but her platelet counts were normal[5]. Cardiac involvement includes hypotension, tachycardia, arrhythmias and cardiogenic shock[1,3]. Severe hypotension has been observed as one of the major clinical features[4]. Our child too had tachycardia, increased troponin levels along with ECG findings (ST wave depression) and inotropes resistant shock. In a study from Ecuador, out of 85 patients, arrhythmias were noted in only 5 of them. They observed ECG changes such as altered or inverted T waves, QRS complex changes, tachycardia,

arrhythmias, atrial fibrillation and decreased ventricular contractility[2]. Central nervous system effects include changes in mental status like confusion, psychosis, hallucinations, and coma[1]. Our patient was confused with irrelevant talk on the third day. Gonzalez-Andrade et al reported gastrointestinal symptoms in 71% of patients, jaundice and hyperbilirubinemia in 37%, increased liver enzymes in 32%, prolonged coagulation time in 18% and hypoglycaemia in 13% of patients. They concluded the bad prognostic factors as high dose, late medical attention (after 3 days), coma, hypoglycaemia and metabolic acidosis[2]. In our child the poor prognostic factors were probably high dose and metabolic acidosis.

A dose of more than 1 mg/kg of yellow phosphorus is almost invariably fatal and our child had ingested 35g of ratol[2,4]. Ratol is a cheap, easily available over the counter rat poison, commonly mistaken for toothpaste and consumed by children[3]. The best methods to overcome yellow phosphorus poisoning would be increase public awareness and ban the sale of these fatal poison containing preparations as suggested by Mohideen *et al*[3].

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