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A case of severe acute hepatitis due to oral intake of firecrackers

Çatapatın oral alımına bağlı gelişen akut ciddi hepatit vakası

To the Editor,

A firecracker is an explosive substance containing yellow and red phosphorus, potassium chloride and antimony, and it is used for celebrations during holidays in Turkey (Figure 1). We report herein the case of a paranoid schizophrenic who presented with acute severe hepatitis after ingestion of firecrackers.

A 39-year-old male paranoid schizophrenic who swallowed 15 firecrackers was brought to the emergency department by his family. He had nausea and mild abdominal pain. His physical examination was unremarkable. As he had swallowed the firecrackers one hour before, gastric lavage was done and medicinal charcoal was given. The patient was taken to the intensive care unit in order to follow the possible cardiac and metabolic side effects of these substances and to support vital functions. Blood values showed no abnormality in the first 24 hours, but the severity of nausea and vomiting increased, and severe abdominal pain started. Biochemical

tests showed an increase in the serum levels of liver enzymes and bilirubin. He had leukopenia, thrombocytopenia and an increased prothrombin time (PT) (Table 1). Plain abdominal X-rays and abdominal ultrasound were normal. Other causes that could elevate liver enzymes were eliminated. Supportive treatment with intravenous fluids, electrolytes, antiemetics, and N-acetyl cysteine was started. The long PT persisted, and liver enzymes were even higher without signs of encephalopathy. Acute severe hepatitis caused by yellow phosphorus intoxication is an indication for liver transplantation. Though schizophrenia is a relative contraindication for transplantation, the patient was referred to a transplant center and placed on the waiting list for cadaveric liver transplantation (1). The patient remained at this center for two weeks and received supportive treatment. Since PT and liver enzymes normalized and the patient was asymptomatic, liver transplantation was not attempted.

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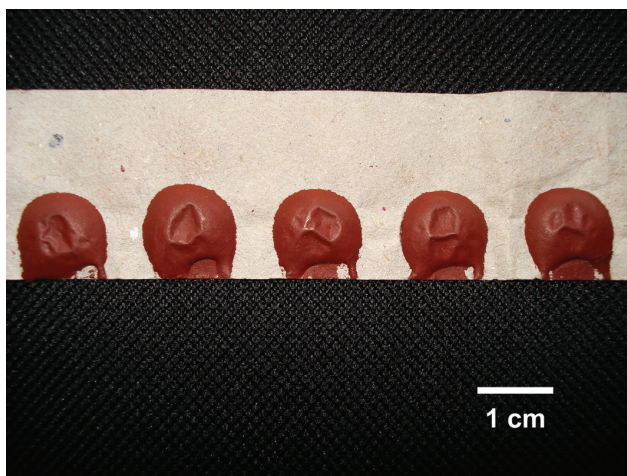
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Table 1. Laboratory results

	AST (U/L)	ALT (U/L)	Total bilirubin (mg/dl)	PT (Second)	INR	WBC (10 ³ /uL)	Hb (g/dl)	PLT (10 ³ /uL)	Creatinine (mg/dl)	Glucose (mg/dl)	Na/K/Cl (mEq/L)
Normal range	0-38	0-41	0.0-1.1		0.82-1.2	4.8-10.8	14-15	130-400	0.70-1.20	76-110	133-150/3.3-5.1/95-115
Basal Values	23	19	0.34	13.5	0.9	5100	14.7	222.000	0.85	101	139/4.9/100
6 th hour	10	5	0.3	22.9	2.18	3800	13.9	184.000	0.8	85	144/4.1/105
12 th hour	30	15	1	23	2.20	3300	13.3	164.000	0.9	89	143/4.5/105
24 th hour	173.3	60	2.2	23.3	2.23	2000	14.3	93.000	1.2	86	144/3.9/100
48 th hour	1722	637	3.4	33	3.2	1200	13.8	114.000	0.9	89	137/4.1/92
72 nd hour	3228	1510	4.1	87.7	11.72	2250	13.9	97.000	1.1	82	141/3.7/102
1 st week	1043	1045	6.62	70.8	5.79	2900	12.6	94.000	0.73	100	131/3.5/103
2 nd week	152	172	21.08	21.3	1.81	3140	14.1	56.000	0.89	98	135/4.5/95
3 rd week	118	103	9.95	13.1	1.13	5500	10.9	383.000	0.9	102	136/4.3/100
4 th week	96	101	4.75	11.9	1.06	5600	11.5	375.000	0.9	90	134/4/106

AST: Aspartate aminotransferase. ALT: Alanine aminotransferase. PT: Prothrombin time. INR: International normalized ratio. WBC: White blood cell. Hb: Hemoglobin. PLT: Platelet.

**Figure 1.** The firecrackers.

Phosphorus intoxication inhibits the oxidation-reduction process in the liver and disturbs lipid and carbohydrate metabolism, thus causing dystrophic and sclerotic changes (2, 3). Acute hepatotoxic effects of yellow phosphorus intake range from no clinical or biochemical defect to varying degrees of subclinical hepatic failure including hepatic necrosis, cholestasis and fulminant hepatic failure (4).

Phosphorus intoxication due to oral intake is a rare phenomenon. It leads to death by causing hepatic failure. Children and adults with psychiatric problems may ingest it unintentionally or as a matter of curiosity, as in our patient. Therefore, a restriction of sale should be considered during the marketing of these materials.

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