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ORIGINAL ARTICLE

Living Donor Liver Transplantation for Acute Liver Failure in Pediatric Patients Caused by the Ingestion of Fireworks Containing Yellow Phosphorus

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Yellow phosphorus is a protoplasmic toxicant that targets the liver. The ingestion of fireworks containing yellow phosphorus, either by children who accidentally consume them or by adults who are attempting suicide, often results in death due to acute liver failure (ALF). We present the outcomes of 10 children who ingested fireworks containing yellow phosphorus. There were 6 boys and 4 girls, and their ages ranged from 21 to 60 months. One patient remained stable without liver complications and was discharged. Three patients died of hepatorenal failure and cardiovascular collapse, and living donor liver transplantation (LDLT) was performed for 6 patients. The patients had grade II or III encephalopathy, a mean alanine aminotransferase level of 1148.2 IU/L, a mean aspartate aminotransferase level of 1437.5 IU/L, a mean total bilirubin level of 6.9 mg/dL, a mean international normalized ratio of 6.6, a mean Pediatric End-Stage Liver Disease score of 33.7, and a mean Child-Pugh score of 11.3. Postoperatively, 2 patients had persistent encephalopathy and died on the second or third postoperative day, and 1 patient died of cardiac arrest on the first postoperative day despite a well-functioning graft. The other 3 patients were still alive at a mean of 204 days. In conclusion, the ingestion of fireworks containing yellow phosphorus causes ALF with a high mortality rate. When signs of irreversible ALF are detected, emergency LDLT should be considered as a lifesaving procedure; however, if yellow phosphorus toxicity affects both the brain and the heart in addition to the liver, the mortality rate remains very high despite liver transplantation. *Liver Transpl 17:1286-1291, 2011.* © **2011 AASLD.**

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Phosphorus means "light bearer" and is the ancient name of the planet Venus when it appears before sunrise. It was first described by the German scientist Hening Brand.[1,2] Yellow phosphorus is an allopathic type of elemental phosphorus, which is also called white phosphorus. Yellow phosphorus, an inorganic substance, does not naturally occur in the environment. This waxy, yellow, transparent, and combustible solid reacts rapidly with oxygen and easily catches fire at room temperature but is stable in water; thus, it is transported and stored in water.[1-3] Yellow phosphorus is manufactured for use in matches, pest poisons, firecrackers, fireworks, watches (for light), military ammunition, and agriculture (as a fertilizer).[3]

Round, penny-sized fireworks, which are known as *catapat* in Turkey, look like brown chocolate and

Abbreviations: ALF, acute liver failure; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ICU, intensive care unit; INR, international normalized ratio; LDLT, living donor liver transplantation; PELD, Pediatric End-Stage Liver Disease.

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contain mainly yellow phosphorus (10%), silica, potassium chlorate, iron oxide, and magnesium carbonate[2,4,5]; however, the quality and the registered production controls are poor. Although other substances in fireworks are less toxic, yellow phosphorus is very toxic, melts easily in oil and bile, and is rapidly absorbed through the intestinal tract. After ingestion, 69% to 73% of the total ingested dose accumulates in the liver within 2 to 3 hours, and this may result in acute liver failure (ALF). Additionally, smaller amounts reach the brain, heart, kidneys, and pancreas.[5-7] The estimated dose of yellow phosphorus that is lethal to the liver is 1 mg/kg, and the ingestion of that amount results in death due to ALF and cardiovascular collapse.[2,3,8] Although yellow phosphorus poisoning may result from industrial accidents in developed countries, it also occurs in adults who are attempting suicide and accidentally by oral intake in children, particularly on festival days, in developing countries.[5,7,9]

Unfortunately, the only definitive treatment for ALF due to the ingestion of fireworks containing yellow phosphorus is liver transplantation because no antidote or medical treatment is available to reverse the toxic effects on the liver. However, the impact of living donor liver transplantation (LDLT) and the survival rates of patients with ALF due to yellow phosphorus are not widely known. Thus, we report 10 patients who ingested fireworks containing yellow phosphorus, and we discuss the outcomes and survival of 6 of these patients who underwent LDLT.

PATIENTS AND METHODS

From June 2006 to October 2010, 10 patients [6 boys and 4 girls with a mean age of 44.2 ± 7.01 months (range = 21-60 months)] were admitted to Turgut Ozal Medical Center (Malatya, Turkey) because of the ingestion of fireworks containing yellow phosphorus. In some cases, emergency doctors had observed brown, penny-sized fireworks in the child's stomach while a nasogastric tube was being placed, and in the other cases, family members stated that they had seen brown firework stains around and inside the child's mouth. Six of the patients underwent LDLT because of ALF. After the study protocol was approved by the ethics committee and the institutional review board of the Inonu University Medical Faculty (reference number 2010/145), the patients' charts were reviewed retrospectively. Their ages, weights, hospital stays, laboratory tests, operation times, graft types, mean follow-up times, and preoperative and postoperative medical outcomes were recorded.

After the ALF diagnosis and the need for urgent liver transplantation were established, detailed counseling was provided to the patients' families, and the patients were put on the National Organ Coordinating Center's list for emergency cadaveric liver transplantation. Meanwhile, family members and close relatives were asked to be donors. All ALF cases and donor candidates were evaluated by the same multidisciplinary team, which included pediatric gastroenterologists, liver transplant surgeons, and radiologists. The donor evaluation was based on blood type and biochemical studies, viral serology tests, multislice spiral computed tomography, and a volumetric assessment of the liver graft. Donors were accepted for transplantation if the graft-to-recipient weight ratio was >0.8 and the fatty liver content was <30%. The donor and recipient operations were started simultaneously, and an ultrasonic dissector was used for the parenchymal transaction during the donor hepatectomy. The Pringle maneuver was not used. All hepatic graft anastomoses were performed under an operating microscope with polypropylene sutures.

A Doppler echo ultrasound examination of hepatic perfusion was performed for all patients within the first 2 days after surgery for the early detection of transplant vascular thrombosis. Anticoagulation therapy consisted of aspirin (100 mg daily). All patients also received tacrolimus-based immunosuppression. Tacrolimus blood levels were maintained between 10 and 15 ng/mL during the first month and between 5 and 10 ng/mL thereafter. Methylprednisolone therapy (10 mg/kg) was started intraoperatively, and the dosage was then tapered to reach 1 mg/kg/day at 2 weeks and 0.25 mg/kg/day at 3 months; there was a progressive switch to alternate-day therapy at 6 months, which was followed by withdrawal.

Statistical Analysis

Continuous variables are reported as means and standard deviations, and categorical variables are reported as numbers and percentages.

RESULTS

The first patient, a 4-year-old boy, was referred to our center 1 day after he was admitted to a local hospital with complaints of abdominal pain, nausea, and vomiting. His initial medical management included gastric irrigation, activated charcoal, and intravenous hydration at a local hospital 10 hours after ingestion. According to a laboratory evaluation, his hepatic aminotransferases were minimally elevated, and his electrolytes were normal. Supportive treatment, which included intravenous hydration and close monitoring, was performed in the pediatric intensive care unit (ICU) for 48 hours. The patient remained stable, and no complications occurred during his stay, so he was discharged (Table 1).

The second patient, a 5-year-old boy, was referred to our center with complaints of abdominal pain, nausea, diarrhea, vomiting, and weakness. Before he was hospitalized and transferred to our center, he had spent 3 days at home after the ingestion of fireworks. On admission, he had jaundice and apathy. Laboratory tests revealed hyperbilirubinemia and elevated aspartate aminotransferase (AST), alanine aminotransferase (ALT) and international normalized ratio (INR) levels. The patient was considered to have ALF

TABLE 1. Demographic Data and Clinical Characteristics of the Patients

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10
Age (Months)	48	60	42	48	54	21	58	42	47	22
Sex	Male	Male	Female	Male	Female	Female	Male	Male	Male	Female
Primary symptoms										
Jaundice	—	+	—	—	+	+	+	+	+	+
Weakness	—	+	—	—	+	+	+	+	+	+
Vomiting	+	+	—	—	+	+	+	+	+	+
Abdominal pain	+	+	—	—	+	+	+	+	+	+
Time from ingestion to admission (hours)	10	72	1	1	6	3	2	2	6	1
Encephalopathy grade	—	III	—	—	II	II	II	II	III	II
Time from ingestion to encephalopathy (days)	—	4	—	—	5	4	5	6	3	4
AST (IU/mL)	70	3150	40	45	1150	1625	2197	1200	754	1699
ALT (IU/mL)	55	3828	38	42	1400	1034	1051	1650	875	879
Total bilirubin (mg/dL)	1.2	10.8	0.8	1.1	7.8	3.6	3.7	13.5	8	4.9
INR	1.1	3	1	1.2	7.3	2.5	11	5.1	10.8	2.9
Prothrombin time (seconds)					76.8	26.8	79.1	53.2	103.6	30
Child-Pugh score					11	12	10	13	12	10
PELD score					36	38	37	26	47	18
Treatment	Medical	Medical	Medical	Medical	LDLT	LDLT	LDLT	LDLT	LDLT	LDLT
Hospital stay (days)		7	1	1	4	2	3	33	33	71
Follow-up (days)	670	0	0	0	0	0	0	402	90	120
Outcome	Alive	Death on day 7	Death on day 1	Death on day 1	Death on day 3	Death on day 1	Death on day 2	Alive	Alive	Alive

due to yellow phosphorus. His liver and renal function tests continued to worsen; on the fourth day after the poisoning, oliguria, severe metabolic acidosis, and grade III encephalopathy occurred, and he needed a ventilator. During that time, his father was prepared as a donor, but the patient died in the ICU because of multiorgan failure (Table 1).

The third patient was a 3.5-year-old girl, and the fourth patient was a 4-year-old boy; they had similar histories. Their families brought them to the emergency department of a local hospital with the complaint of firework ingestion. Although the physical examinations and the laboratory tests were normal, the patients were transferred to our center. Our laboratory investigations revealed normal hepatic and renal function. Supportive treatment in the pediatric ICU was prescribed for both, but the patients suddenly died because of cardiovascular collapse within 24 hours of the ingestion of fireworks, although all their vital signs and electrolytes were normal (Table 1).

The remaining 6 patients [3 boys and 3 girls with a mean age of 40.7 ± 15.8 months (range = 21-58 months) and a mean weight of 13.8 ± 3.1 kg (range = 9-18 kg)] were brought to the emergency department of a local hospital with complaints of vomiting and abdominal pain 1 to 6 hours after the ingestion of fireworks. Their main symptoms were abdominal pain, vomiting, and weakness. All the patients were hospitalized in the ICU with fluid and electrolyte repletion. Ninety-six to 168 hours after they had ingested the fireworks, all the patients became more somnolent (grade II or III encephalopathy) with elevated liver enzyme levels [mean ALT level = 1148.2 ± 311.5 IU/L (range = 875-1650 IU/mL), mean AST level = 1437.5 ± 507.3 IU/L (range = 754-2197 IU/L), and mean total bilirubin level = 6.9 ± 3.4 mg/dL (range = 3.6-13.5 mg/dL)]. Their coagulopathy gradually worsened before the operation; the mean prothrombin time was 61.6 ± 30 seconds (range = 26.8-103.6 seconds), and the mean INR was 6.6 ± 3.8 (range = 2.5-11). The mean Pediatric End-Stage Liver Disease (PELD) and Child-Pugh scores were 33.7 ± 10.2 (range = 18-47) and 11.3 ± 1.2 (range = 10-13), respectively (Table 1). Other causes of ALF were excluded by laboratory studies for all patients. The patients were listed at the National Organ Coordinating Center for emergency liver transplantation, and their parents were evaluated as potential donors because of the shortage of cadaveric donor grafts. LDLT was performed for 6 patients 128 ± 35.3 hours (5.3 ± 1.5 days) after the ingestion of the fireworks. The donors were mothers in 2 cases and fathers in 4 cases. The mean graft-to-recipient weight ratio was 1.2 ± 0.3 (range = 1.1-1.8). The mean cold ischemia time for living donor grafts was 67 ± 17.1 minutes (range = 47-105 minutes). The mean length of the surgery was 8 ± 1.1 hours (range = 7-10 hours). The patients remained in the ICU for a mean duration of 5 ± 3 days (range = 1-9 days), and the mean hospital stay was 24.3 ± 27.2 days (range = 2-71 days). The explanted livers were very hard and pale during the operation and showed signs of toxic hepatitis with extensive necrosis, balloon degeneration, and steatosis during the histopathological examination (Fig. 1).

During the postoperative period, encephalopathy persisted in 2 patients while they were receiving intravenous mannitol and oxygen, and they died on postoperative days 2 and 3 with well-functioning grafts. One of the 6 patients (16.7%) died because of sudden

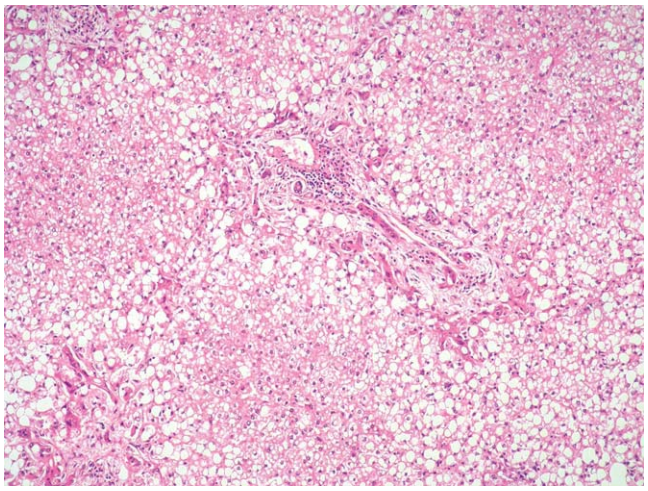

Figure 1. Histopathological examination: extensive necrosis, balloon degeneration, and steatosis.

cardiac arrest on postoperative day 1 with a well-functioning graft. Although the mortality rate due to firework toxicity was 60% for all 10 patients, the rate was 50% for those who underwent LDLT. When this article was being written, the 3 remaining children were still alive and had good graft function. The mean follow-up duration was 204 ± 172.1 days (range = 90 - 402 days). The details of the postoperative period for all the patients who underwent LDLT are presented in Table 2.

DISCUSSION

Exposure to yellow phosphorus is uncommon in developed countries, whereas acute poisoning can still occur because of suicide attempts by adults and accidental ingestion by children in developing countries.[5,7,9] Yellow phosphorus is a protoplasmic and supertoxic poison that is absorbed rapidly by the gastrointestinal tract after oral ingestion. In an experimental animal study using a lethal dose of a radioactive isotope of phosphorus, phosphorus became particularly concentrated in the liver (up to 70%) within 2 to 3 hours, and lesser amounts were found in the blood (12%), kidneys (4%), pancreas (0.4%), and brain (0.39%).[6] Studies addressing the metabolism of yellow phosphorus are very limited, and nothing is known about real enzymatic catalysis. Nevertheless, some studies have reported that this protoplasmic poison impairs glycogenesis, lipid peroxidation, and protein synthesis (prothrombin and plasma proteins are required to regulate blood coagulation) and produces fatty changes and massive necrosis in multiple organs (principally in the liver and first at the periphery of the hepatic lobules).[2,3,5,6]

The clinical effects of acute poisoning with yellow phosphorus have classically been divided into 3 stages.[3,8,10] The initial gastrointestinal stage is characterized by vomiting, nausea, diarrhea, and abdominal pain, which occur within the first 24 hours after

TABLE 2. Details of the Postoperative Period for All Patients Who Underwent LDLT

	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10
Treatment	LDLT	LDLT	LDLT	LDLT	LDLT	LDLT
Donor	Mother	Father	Mother	Father	Father	Father
Donor age (years)	58	21	54	42	47	23
Graft type	Left lobe	Left lateral segment	Left lateral segment	Left lateral segment	Left lobe	Left lobe
Graft-to-recipient weight ratio	1.2	1.1	1.1	1.2	1.8	1.1
Time from ingestion to LDLT (hours)	144	120	144	168	96	96
Explant histology	Massive necrosis and diffuse steatosis	Massive necrosis and diffuse steatosis	Massive necrosis and diffuse steatosis	Massive necrosis and diffuse steatosis	Massive necrosis and diffuse steatosis	Massive necrosis and diffuse steatosis
Postoperative complications	Encephalopathy	—	Encephalopathy	Bile leak	Hepatic atherothrombosis and small bowel perforation	Bile leak
Infection	—	—	—	*Acinetobacter*	*Acinetobacter*	—
Reoperation (n)	—	—	—	3	2	1
Hospital stay (days)	4	2	3	33	33	71
Follow-up (days)	0	0	0	402	90	120
Outcome of LDLT	Death on day 3	Death on day 1	Death on day 2	Alive	Alive	Alive

ingestion. Laboratory tests are almost normal during this period. During this stage, sudden death may occur for no known reason (as in 2 of our cases); it may be that the ingestion of a very large amount of fireworks can directly result in cardiovascular arrhythmia and collapse within the first 24 hours. Cardiac collapse may also occur because of fluid and electrolyte loss due to vomiting or diarrhea in addition to heart toxicity. Although we could not document dysrhythmias or electrolyte disturbances in our patients, repeated electrocardiograms may demonstrate myocardial damage with dysrhythmias, widened QRS complexes, or depression of ST segments, and early death is generally due to cardiac dysrhythmias secondary to electrolyte abnormalities such as hypocalcemia and hyperkalemia.[3,8,11] The second stage (1-4 days), which may last for several days, is essentially a symptom-free period, but liver enzyme levels become elevated, and toxic hepatitis begins to spread. In our first case, the initial insult was resolved spontaneously; this could be explained by the ingestion of a small amount of fireworks, or the reasons may be unknown. The third stage can end in ALF and acute renal failure with metabolic derangements, encephalopathy, coagulopathy, arrhythmia, cardiogenic shock, and abnormal liver tests. In short, this is multiorgan failure; in our patients, the third stage occurred between 4 and 7 days. Patients progress to the third stage because of the systemic effects of high-dose phosphorus after it has been absorbed.[3,7-11] If liver transplantation, which is the only treatment during the last stage, is not performed, death is inevitable.[5,7]

There is no specific medical test for the diagnosis of yellow phosphorus poisoning, and phosphorus blood levels are not useful for this either. The diagnosis mainly depends on the history provided by the children or their parents. If the history is unclear, a garlicky odor and luminescence of vomitus or stool may be helpful. Faint fumes emanating from the stool are called smoking stool syndrome.[3,4,8,12,13]

No antidote or specific medical treatment is available for patients who have ingested fireworks containing yellow phosphorus.[3,10,12,14] A laboratory investigation yields supportive evidence for the various stages of the clinical picture. Biochemistry tests can indicate metabolic acidosis, hypoglycemia, hyperbilirubinemia, and increased AST and ALT levels. Hematological abnormalities include elevations in the INR and the partial thromboplastin time and leukopenia. An electrocardiogram may demonstrate inverted T waves and changes in the QRS complex, arrhythmias, and atrial fibrillation. All these findings can be observed easily during the third stage.[3,8,11,15]

The treatment should include the removal of phosphorus and general supportive measures only during the first and second stages. The management of acute poisoning includes the removal of the toxin with gastric lavage and potassium permanganate (1:5000), which oxidizes phosphorus into relatively less toxic phosphoric acid and phosphates. Alternatively, a 0.2% solution of copper sulfate may be used as a stomach wash; this converts phosphorus into nontoxic copper phosphide. The avoidance of any oily or fatty foods is preferable because these can enhance the absorption of phosphorus.[1-3,8,13] However, whether these measures (including activated charcoal lavage) are effective has not been well demonstrated.[2,10,16] Moreover, intravenous steroids, a trial of *N*-acetylcysteine, and exchange transfusions are not useful, and their effects have not be demonstrated for the treatment of acute yellow phosphorus poisoning.[2,17] Supportive measures include the management of hypotension, hypoglycemia, hypocalcemia, seizures, coagulopathy, and arrhythmias.[1-3,8,11,13]

In the past, studies of yellow phosphorus–related poisoning have recommended only the removal of phosphorus and general supportive measures.[2,3,8,13,16] In 2009, only 2 studies reported the utility of liver transplantation for managing ALF due to the ingestion of fireworks containing yellow phosphorus. In the first study, Akman et al.[5] (Turkey) reported cadaveric liver transplantation for a 3.5-year-old girl. The second study was conducted by Santos et al.[7] (Spain), who reported cadaveric liver transplantation for a 4-year-old girl with end-stage ALF due to the ingestion of firecrackers; the results were good. They also stated that emergency liver transplantation may be lifesaving for patients with irreversible ALF due to the ingestion of fireworks containing yellow phosphorus. We have reported 6 pediatric patients who underwent emergency LDLT because of ALF due to fireworks, and their parents provided the liver grafts. The available data[2,3,5,7,8,16] and our experience indicate that patients who ingest fireworks containing yellow phosphorus and have stage III liver damage may not be saved by the removal of phosphorus and general supportive measures, which can be useful in stages I and II of phosphorus poisoning. When ALF has reached stage III (which is irreversible), emergency liver transplantation should be considered the only definitive treatment; however, if yellow phosphorus affects the brain and the heart in addition to the liver, the mortality rate is high (73%-90%)[7,9,10] despite liver transplantation. Hepatic encephalopathy can cover up all the findings of yellow phosphorus neurotoxicity during the third stage; therefore, it is not easy to determine whether encephalopathy results from a hepatic coma or yellow phosphorus neurotoxicity. Two of our patients with persisting encephalopathy but well-functioning grafts died on postoperative days 2 and 3, and 1 of the 6 transplant patients (16.7%) died because of cardiac arrest on postoperative day 1. This may have occurred because of the involvement of other organs in addition to the liver.

LDLT is an excellent option for patients with ALF due to accidental poisoning by yellow phosphorus and allows easier access to liver grafts in emergency situations. When this article was being written, 3 of our 6 transplant patients (50%) were still alive and were experiencing good graft function [mean follow-up = 204 ± 172.1 days (range = 90-402 days)].

In conclusion, the ingestion of fireworks containing yellow phosphorus may progressively lead to ALF and multiorgan failure with a high mortality rate. Because sudden death occurs mainly in the first few days, children who have ingested yellow phosphorus should be closely monitored in the ICU for the assessment of their cardiac rhythms and electrolyte levels. If the first signs of irreversible ALF due to firework poisoning are detected, emergency liver transplantation should be considered as a lifesaving procedure. However, if yellow phosphorus affects the brain and the heart in addition to the liver, the mortality rate is high despite liver transplantation.

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REFERENCES

1. Eldad A, Simon GA. The phosphorous burn—a preliminary comparative experimental study of various forms of treatment. Burns 1991;17:198-200.
2. Nikkanen HE, Ewald MB. Phosphorus. In: Flomenbaum N, Goldfrank L, Hoffman R, Howland MA, Lewin N, Nelson L, eds. Goldfrank's Toxicologic Emergencies. 8th ed. New York, NY: McGraw-Hill Companies; 2006:1486-1491.
3. Agency for Toxic Substances and Disease Registry. Toxicological profile for white phosphorus. http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=285&tid=52. Published September 1997. Accessed July 2011.
4. Makalinao IR, Maramba NPC. Watusi Poisoning Monograph: A Comprehensive Guide to Human Health Effects, Toxicity and Treatment. Manila, Philippines: National Poison Control and Information Service; 1999.
5. Akman SA, Cakir M, Baran M, Arikan C, Yuksekkaya HA, Tumgor G, et al. Liver transplantation for acute liver failure due to toxic agent ingestion in children. Pediatr Transplant 2009;13:1034-1040.
6. Ghoshal AK, Porta EA, Hartroft WS. Isotopic studies on the absorption and tissue distribution of white phosphorus in rats. Exp Mol Pathol 1971;14:212-219.
7. Santos O, Restrepo JC, Velásquez L, Castaño J, Correa G, Sepúlveda E, et al. Acute liver failure due to white phosphorus ingestion. Ann Hepatol 2009;8:162-165.
8. Brent J, Wallace K, Burkhart K. Phosphorus. In: Erdman A, ed. Critical Care Toxicology: Diagnosis and Management of the Critically Poisoned Patient. Philadelphia, PA: Elsevier Saunders; 2005:851-861.
9. Fahim FA, el-Sabbagh M, Saleh NA, Sallam US. Biochemical changes associated with acute phosphorus poisoning (in humans). Gen Pharmacol 1990;21:899-904.
10. McCarron MM, Gaddis GP, Trotter AT. Acute yellow phosphorus poisoning from pesticide pastes. Clin Toxicol 1981;18:693-711.
11. Talley RC, Linhart JW, Trevino AJ, Moore L, Beller BM. Acute elemental phosphorus poisoning in man: cardiovascular toxicity. Am Heart J 1972;84:139-140.
12. Simon FA, Pickering LK. Acute yellow phosphorus poisoning. "Smoking stool syndrome". JAMA 1976;235:1343-1344.
13. Krishnakumar N, Krishnakumar S, Dalus MR, Balamugesh T. Yellow phosphorous poisoning—smoking stool syndrome. J Assoc Physicians India 1997;45:885-886.
14. Fernandez OU, Canizares LL. Acute hepatotoxicity from ingestion of yellow phosphorus-containing fireworks. J Clin Gastroenterol 1995;21:139-142.
15. Diaz-Rivera RS, Collazo PJ, Pons ER, Torregrosa MV. Acute phosphorus poisoning in man: a study of 56 cases. Medicine (Baltimore) 1950;29:269-298.
16. Davis KG. Acute management of white phosphorus burn. Mil Med 2002;167:83-84.
17. Marin GA, Montoya CA, Sierra JL, Senior JR. Evaluation of corticosteroid and exchange-transfusion treatment of acute yellow-phosphorus intoxication. N Engl J Med 1971;284:125-128.