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Case report

Acute unintentional intoxication with paraffin in a 25-year old patient – Clinical case report

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ABSTRACT

"Fire-breathing" or "fire-eating" is a special kind of street art where the acts are always stunning, spectacular and amazing. People exhibiting this kind of show are professionals, not rare amateurs, who use different kind of fuels, usually hydrocarbons, in order to produce a pillar of fire. Intoxications caused by ingestion or inhalation of liquid paraffin, used as a fuel while performing, are numerous and various. We present a clinical case report of a 25-year old, previously healthy, amateur "fire-breather". During October, 2010 this young men arrived at the Emergency Unit of the University Clinic for toxicology and Urgent Internal Medicine in a severe clinical condition, after his unsuccessful attempt to perform real "fire-breathing". He had fever, strong headaches, mild abdominal and chest pain and he also had difficulties breathing and persistent dry cough. The patient was extremely dyspneic with peripheral cyanosis and shortness of breath.

"Fire-breathers" must be viewed as a population at risk of paraffin-induced pneumonia, which has low mortality rate, but still is related with numerous and various chronic complications. Our patient was first in a life threatening, extremely serious clinical condition which was urgently treated with appropriate vigorous and effective therapy. This therapeutic protocol led to successful full recovery of these young men, who luckily didn't exhibit any chronic complications.

1. Introduction

"Fire-breathing" or "fire-eating" is a special kind of street art where the acts are always stunning, spectacular and amazing. There are different words describing this particular art including blowing, aspirating and spitting. "Fire-breathers" are street performers who usually practice extensively to demonstrate this magnificent show. To produce a pillar of fire they use two components: fuel and a flame source, which at the start sounds and looks very simple, but controlling the fuel's direction and consistency is a technique that requires a lot of time to improve. Performers use different kind of chemical substances as a fuel, but most often they utilize a hydrocarbon known as kerosene. It is a petroleum distillate, also known as liquid paraffin and it can be found on the market under its commercial name "liquid wax". Intoxications caused by ingestion or inhalation of liquid paraffin, used as a fuel while performing, are numerous and various. Usually we see different kinds of burns, skin irritation, stomach ulcers and gum diseases. But the condition that performers are quite prone to is a clinical entity called hydrocarbon pneumonitis, also known as "fire-breathers or fire-eaters lipoid pneumonia", which can often lead to acute respiratory distress and several severe respiratory complications.[1,2] Initial symptoms usually include fever, chest pain, dry cough, shortness of breath with unilateral or bilateral nonspecific alveolar infiltrates manifesting on plain chest radiographs.[3]

We report about a rare case of an amateur fire-breather who tried to perform a fire-breathing show without any previous experience.

2. Case report history

Our patient is a very adventurous, healthy 25-year old male student. One night, in the late hours, during October 2010, he was admitted to the Emergency Unit at the Clinic of Toxicology and Urgent Internal Medicine, after his unsuccessful attempt to perform a real "fire-breathing" or "fire-blowing" act. During his performance he used paraffin oil, also known as kerosene, as a fuel, which is a flammable, volatile and toxic substance. After putting some

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quantity of the fuel in his mouth he immediately tried to blow it in a pillar of fire, but during the event he unknowingly, inadvertently and unintentionally swallowed and aspirated a small amount of it. He did not have any immediate symptoms following this accident, so he felt quite well for a while. Unfortunately, two days after he got fever, felt mild abdominal and strict chest pain, with difficult breathing and dry cough. When he arrived at our emergency unit initially he complained on strong headaches, dizziness and persistent dry cough with hemoptysis. He was febrile (38.9 °C), extremely dyspneic with peripheral cyanosis and shortness of breath. We immediately put him on an intensive care monitor which measured blood pressure of 130/65 mmHg and heart rate of 75 bpm. He was conscious, contactable and well orientated. On inspection his chest was normostenic and symmetrical. Despite the severe pain, he could move his chest with regular and balanced respirations. Immediate pulmonary auscultation revealed rhonchi over the lower, basal part of the left lung and bilateral basal rales.

Laboratory test on admission showed normal findings despite the leucocytosis (Neu: 92.6%) and hyperglycemia. Also we measured fibrin degradation products and they showed secondary activated fibrinolysis (1917 ng/ml). His blood gas analysis showed hypoxia with partial respiratory insufficiency. The patient was hospitalized at the Intensive care unit and he was put on a continuous oxygen-therapy on mask. After installing an intravenous line we first administrated pain relievers, antipyretics and bronchodilatators. Due to the severe clinical condition and suspected pulmonary thromboembolism we asked for an urgent chest X-ray. It revealed bilateral perihilar and basal infiltrates with low transparency. The heart was normal and there were no pleural effusions (Fig. 1).

His initial treatment consisted of:

- Continuous oxygenotherapy on mask.
- Intravenous fluids (NaCl-0.9% 1000 ml/12 h).
- Double antibiotic therapy (2 g of ceftiaxone + 800 mg of ciprofloxacine/24 h in the first five days, after that we added 1500 mg of metronidasole/24 h with excluding ciprofloxacine from the therapy).
- Corticosteroids (8 mg of dexamethasone/24 h).
- Bronchodilatators (500 mg of aminophyline).
- Vitamin K (10 mg fitonadione).
- Anticoagulant (40 mg of enoxiparine/24 h).
- Ranitidine 300 mg.
- Vitamins.

This therapy was administered parenterally during the first 10 days. After that the patient continued receiving these drugs by mouth, for 15 days.

Because the diagnosis remained unclear we continued with extensive diagnostic procedures to maintain the right diagnosis. Electrocardiography examination showed a typical pattern of a right bundle branch block (RBBB). Echocardiography (heart ultrasound) revealed a mild reduction of left ventricular function, mild mitral regurgitation and paradoxical septal motion, due to the RBBB. The abdominal ultrasound was with normal findings. He also told us that during the accident he swallowed some amount of the fuel and that he felt abdominal discomfort for the whole time, so we also wanted to examine the condition of the upper gastrointestinal tract with upper endoscopy (esophagogastroduodenoscopy), with a flexible endoscope. We found a superficial erosions in the lower esophagus and gastric cardia; some superficial, narrow erosions in the antropyloric and posterior regions of the stomach and various duodenal lesions. His hemoculture was sterile. At the same time, during the next three days, the patients' condition worsened and he was in a very critical condition. He continued receiving the same aggressive therapy protocol, in which we added antacids and protein pump inhibitors to protect the gastrointestinal mucosa. When 10 days passed he exhibited slightly improvement in his clinical condition. Control chest X-ray, which was made 10 days after commencement of treatment, showed bilateral basal consolidations of the pulmonary parenchyma, with pleural adhesions on the left. The heart shadow remained normal (Fig. 2).

The patient received corticosteroid therapy for 15 days and it was reduced day by day; 8 mg of dexamethasone were given during the first 10 days and after that during 3 days we continued with 4 mg of dexamethasone and finished with 2 mg od dexamethasone during the last 2 days.

On the 17th day he was in a good condition so he was transferred to a regular stationary ward.

Where we continued with oral antibiotics, intravenous fluids and vitaminotherapy. Luckily, after 4 weeks the patient was fully recovered and he left our clinic in a good and improved clinical condition. Due to the seriousness of the intoxication this patient was monitored in the next three months and was found to be in an excellent condition. A final X-ray performed 5 months later showed complete regression of the pulmonary changes.

The diagnosis of this patient was acute aspiration pneumonia, known as "fire-breathing" pneumonia, or also found in the literature under the name "fire-eaters" lung or "fire-eaters" lung irritation. Of the main importance is the fact that completion of right diagnosis was a little delayed because aspiration history was not shown by the patient from the first moment. We reached the diagnosis after identifying the chemical agent used as a "fuel", with detection of its chemical structure and possible toxic effects and also detecting organic damages that it caused in the patient.

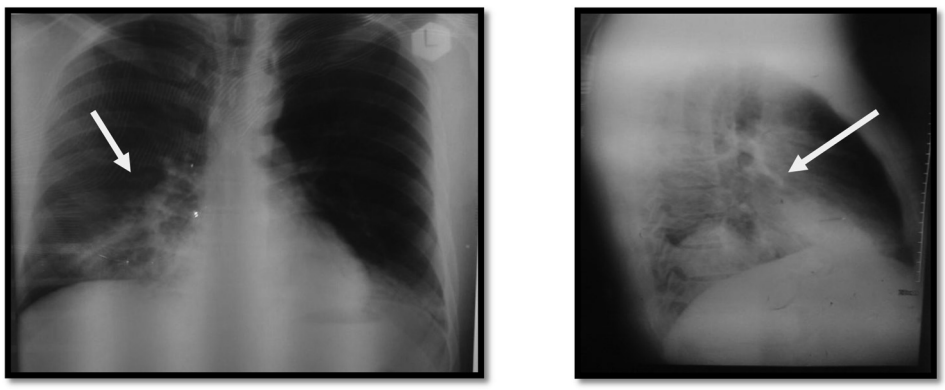

Fig. 1. Chest X-ray on admission (Areas marked with arrows show basal and perihilar infiltrates with low transparency).