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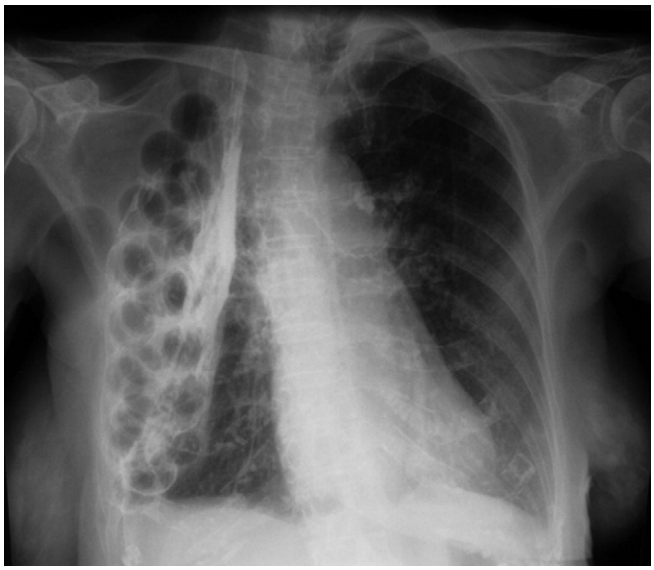

Figure 1. Chest radiograph.

Figure 2. CT contrast-enhanced scan of the lung. Used with permission of Marc Andronikof, MD, Emergency Department, Hôpital Antoine Béclère (APHP), Clamart, France.

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An 80-year-old woman was admitted to our emergency department after having fallen at home. Her medical history comprised pulmonary tuberculosis treated in 1948, mild cardiac failure, and restrictive pulmonary insufficiency, for which she was receiving nasal oxygen continuously at 1 L/minute. She complained of shortness of breath. Her temperature was 37.5°C (99.5°F), blood pressure 131/56 mm Hg, pulse rate 100 beats/min, respiratory frequency 40 breaths/min, and pulse oxygen saturation 81%. Her chest radiograph is shown (Figure 1). A computed tomographic (CT) scan was ordered to rule out a pulmonary embolism (Figure 2).

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DIAGNOSIS:

Methyl-methacrylate (Lucite) sphere in the right extrapleural space and left pleural effusion. Before the discovery in the 1950s of drugs effective against *Mycobacterium tuberculosis*, collapse therapy was the mainstream of treatment of pulmonary tuberculosis because it was shown effective in stopping the disease in an individual. In 1881, Carlo Forlanini of Turin, Italy, introduced the use of artificial pneumothorax.[1] As the method gained in popularity, many means to create and maintain the collapse were applied: artificial pneumothorax with air refills, phrenic nerve crush, thoracoplasty, and extrapleural plombage. In the latter case, many available materials were used: fat, paraffin wax, bone, gauze sponge, silk, gelatin, rubber balloons, oil and, as in our case, Lucite balls.[2] The occurrence of long-term complication of these treatments is debated.[2-4] In any case, many patients remained asymptomatic but for a certain degree of respiratory failure. Since the 1950s, the use of collapse therapy (and plombage) declined steadily. A young physician may not have encountered such a patient in his practice. Our patient eventually died in the pulmonary ward a few days later of respiratory failure.

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DIAGNOSIS:

Cresol intoxication. The urine levels of para-cresol, meta-cresol, ortho-cresol, and phenol were 10,186, 11,015, 388, and 101 mg/g creatinine, respectively, at 12 hours after ingestion. The methemoglobin level was 1.1%, with a hemoglobin concentration of 14.8 g/dL. His hospitalization course was complicated by pneumonia, gastric corruption, and liver and renal function impairment. He recovered uneventfully after intensive supportive care.

Cresol, a commonly used household disinfectant worldwide, was the original active ingredient in the American brand Lysol. It may cause gastrointestinal corrosive injury, central nervous system and cardiovascular disturbances, and renal and hepatic injury after intoxication.[1] When ingested, cresol is excreted in the urine and renal damage is possible. The black urine is a prominent feature of cresol intoxication. Other different diagnoses of black urine include hemoglobinuria, myoglobinuria, alkaptonuria, melanuria, porphyrinuria, and tyrosinuria. In addition, some medications including L-dopa, methyldopa, chloroquine, primaquine, furazolidone, metronidazole, nitrofurantoin, cascara/senna laxatives, methocarbamol, and sorbitol may cause black urine.[2,3]

Initial management includes gross decontamination, activated charcoal use, and case-based decision for gastric lavage. Medical treatment of cresol intoxication is primarily supportive to maintain cardiovascular and respiratory functions. Hemodialysis is indicated in acute renal failure.[1,4] Benzodiazepine and anticonvulsants can be used if seizures occur.

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