

Letter to the Editor

Activated charcoal bronchial aspiration

Broncoaspiração de carvão ativado

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To the Editor:

A 20-year-old female patient, who had attempted suicide twice, was admitted to our facility one hour after having ingested an unknown quantity of lead shot. The patient was agitated. She was sweating and had sialorrhea. Examination revealed normal vital signs, diffuse rhonchi, and miosis. She was treated with atropine and 50 g of activated charcoal (AC) administered in 400 mL of mannitol through a gastric tube, the position of which was confirmed by auscultation. A few hours later, the patient vomited, as well as experiencing a drop in the level of consciousness and agonal respiration. She underwent bronchoscopy, and an abundant quantity of blackish material mixed with food debris was obtained. She was placed on mechanical ventilation. Chest X-ray was normal. The patient developed hypotension refractory to fluid resuscitation and was started on noradrenaline. The response was satisfactory. On the second day, the patient became feverish. Chest X-ray revealed bilateral alveolar opacification (Figure 1A). The $\text{PaO}_2/\text{FiO}_2$ ratio was below 200, mechanical ventilation having been set at positive end-expiratory pressure and an FiO_2 of 100%. On subsequent days, during respiratory therapy maneuvers, there was clearance of blackish material. In order to treat pneumonias that were likely to occur over the course of her hospitalization, various antibiotic regimens were successively used. On postadmission day 12, a tracheostomy was performed. The patient developed acute kidney failure requiring hemodialysis. A chest X-ray taken on postadmission day 17 showed a pattern consistent with ARDS (Figure 1B). A chest CT scan (Figure 1C) taken on postadmission day 30 revealed a massive left pleural effusion and an interstitial pattern that was irregular and diffuse, with ground-glass areas alternating with areas of hyperinflation; there was no structural deterioration.

The patient's condition improved slowly. A transnasal fiberoptic laryngoscopy performed when the tracheostomy tube was removed revealed no abnormalities. On postadmission day 78, the patient was discharged on diazepam and fluoxetine; chest

X-ray revealed no parenchymal changes (Figure 1D). At the six-month follow-up evaluation, the patient had no respiratory complaints, and a chest CT scan (Figure 1E) revealed the following: thoracic asymmetry favoring the left chest; an asymmetric, diffuse pattern (of little significance) on both sides, with ground-glass areas alternating with hypertransparent areas; scar-like lines at right angles to the pleural surface in the upper and middle lobes; and only a few areas exhibiting a tree-in-bud pattern. The patient was uncooperative during the spirometry test; peak flow was normal (380 L/min).

Ingestion of lead shot is the second most commonly used method of suicide in Brazil.⁽¹⁾ Lead shot is illegal and usually contains carbamate. It is used as a rodenticide, being also used in agriculture. It acts as an organophosphorus compound, inhibiting cholinesterase and acetylcholinesterase activity and, therefore, hydrolysis of acetylcholine. Lead shot poisoning is treated with atropine and single or multiple doses of AC in order to reduce lead absorption.

Acute poisoning is treated with AC. Complications resulting from AC use are rare, and it is not known whether pulmonary changes are caused by AC use, by vomiting, or by ARDS.

In the present case, bacterial complications were indisputably present over the course of the condition. The initial event, one day after the normal chest X-ray, might have been determined by pulmonary edema due to an AC-elicited increase in capillary permeability,⁽²⁾ by hydrochloric acid, or, remotely, by pneumonia. Bronchial obstruction by food debris can be excluded because of the extent of the opacities and the absence of lung retraction. Aspiration of AC for days confirms the slow clearance. The CT findings six months later reflected bronchiolar obstruction, which has been described in cases of aspiration of AC.

An experimental animal study⁽³⁾ found, at 2-4 weeks, randomly dispersed AC particles in the lumen and submucosa of bronchioles and alveolar ducts; AC-laden bronchioles showed evidence of moderate nonspecific inflammation and epithelial

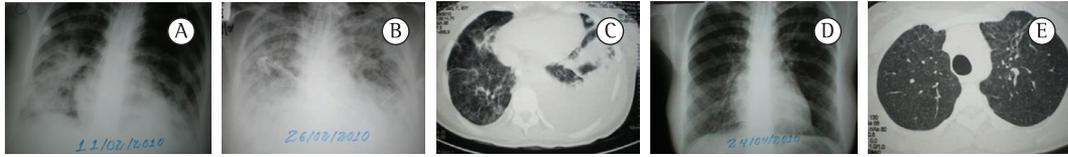


Figure 1 - In A, a chest X-ray taken on postadmission day 2, revealing alveolar opacity in the lung bases. In B, a chest X-ray taken on postadmission day 17, showing a pattern consistent with ARDS. In C, a chest CT scan taken on postadmission day 30, revealing pleural effusion. In D, a chest X-ray taken at discharge, showing no significant changes. In E, a chest CT scan taken six months after discharge, revealing a tree-in-bud pattern.

injury, and there was vascular and interstitial edema. At 12-20 weeks, there was epithelial sloughing and replacement by cuboidal cells in the AC-laden bronchioles; in the submucosa, there was granulation tissue; progressive intraluminal infiltration by inflammatory cells, fibroblasts, and fibrotic tissue resulted in varying degrees of bronchiolar obstruction; there were diffuse areas of atelectasis and alveolar thickening in the parenchyma; and AC-free sites were normal.

Granulomas⁽⁴⁾ and bronchiolitis⁽⁵⁾ have been found years after AC administration. A profile consistent with diffuse alveolar damage was found in a female patient who died 19 days after ingestion of AC (alveolar spaces, diffuse interstitial fibrosis with AC deposition in the interstitium, and proliferation of type II pneumocytes).⁽⁶⁾

A mass in the right lower lobe, mimicking a neoplasm, was found in a male patient after instillation of AC into the right mainstem bronchus; histopathological examination revealed a black mass of 7 × 7 × 4 cm with small satellite nodules; histology showed that the mass was infiltrated by carbonaceous material and was surrounded by granulomatous inflammation; the nodules also contained carbonaceous material.⁽²⁾

We can therefore conclude that AC is not inert, because it can produce an acute pulmonary reaction, causing acute respiratory failure and histopathological lesions that increase in size over time. It is of note that these lesions occur only in AC-laden areas, AC-free areas remaining normal.

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