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A RARE CASE OF DIFFUSE ALVEOLAR HEMORRHAGE FOLLOWING ORAL AMPHETAMINE INTAKE

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Diffuse alveolar hemorrhage (DAH) is a clinical syndrome, which refers to injury to the capillaries, arterioles and venules, leading to red blood cell accumulation in the distal air spaces. It is defined by the clinical triad of hemoptysis, anemia and progressive hypoxemia. Chest radiographs reveal non-specific patchy or diffuse bilateral pulmonary consolidation. Multiple conditions are associated with DAH, of which Wegener's granulomatosis is the most frequent, and underlying disease determines the prognosis and treatment. This case describes DAH as a result of oral amphetamine abuse in a young patient of which the diagnosis was established by laboratory, clinical and radiologic findings. The patient experienced a rapid recovery without significant sequelae.

Key-word: Drugs, abuse.

Case report

A 20-year-old male patient was admitted to the emergency department because of aggressive behavior and respiratory depression with a short episode of apnea, after the administration of 20 mg of diazepam, during a Techno party. He reported the oral use of amphetamine (ecstasy). After admission there was an episode of hemoptysis and hematemesis. Arterial oxygen saturation dropped to 70%, without clinical repercussion.

Clinical findings

Lung auscultation showed discrete crepitation of the lower right lung.

Laboratory findings were a mild leukocytosis and C-reactive protein increase.

Chest radiograph (AP-view) (Fig. 1) shows a normal cardiac size and bilateral, predominantly perihilar, areas of increased opacity.

High-resolution CT examination of the lungs (Fig. 2) revealed multifocal ground glass attenuation with areas of consolidation and discrete peribronchovascular thickening, here depicted on an axial image (A). A Reformatted image in the coronal plane (B) shows the diffuse extent in both lungs. There was a normal heart size and no presence of pleural effusion.

Based on the clinical and CT findings the diagnosis of pulmonary hemorrhage was made. The presence of hemoptysis, hypoxemia, recent amphetamine use and rapid radiographic recovery supported the diagnosis.

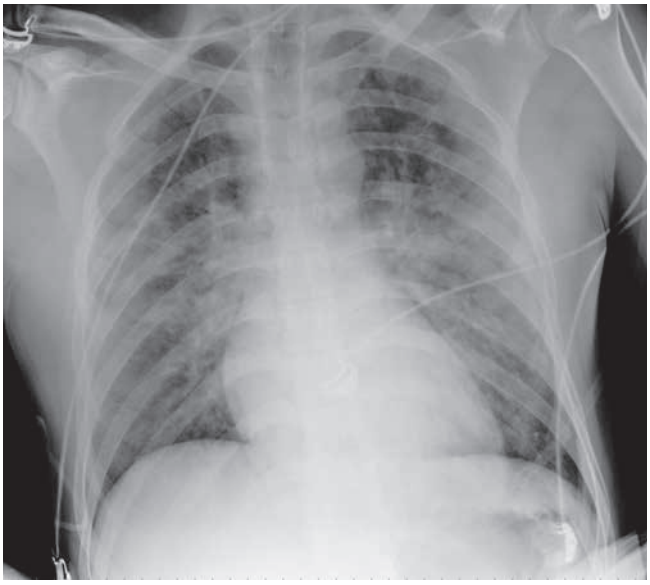

Fig. 1. — Chest radiograph (AP-view) showing normal cardiac size and bilateral, predominantly perihilar, areas of increased opacity.

The patient was admitted and treated with oxygen administration and antibiotic therapy for the risk of aspiration pneumonia. The lung opacities cleared rapidly over the course of 48 h. Laboratory results tested positive on the use of 3,4-methylenedioxymethamphetamine (MDMA, ecstasy). No other drug substances were found.

Discussion

Diffuse alveolar hemorrhage (DAH) is a clinicopathological syndrome describing the accumulation of intra-alveolar red blood cells originating from the alveolar capillaries. The classical clinical triad includes hemoptysis, anemia and hypoxemia, which can be severe (1).

On plain radiography DAH manifests as multifocal bilateral areas of increased opacity with a normal heart size. High-resolution CT shows multifocal ground-glass attenuation, which occasionally is centrilobular in distribution and associated with interlobular septal thickening.

The differential diagnosis of these radiologic findings must be broad and include acute lung injury, diffuse infection, and noninfectious inflammatory conditions (e.g. pulmonary hemorrhage and acute hypersensitivity pneumonitis).

Inhaled and intravenously abused drugs, including opiates and cocaine

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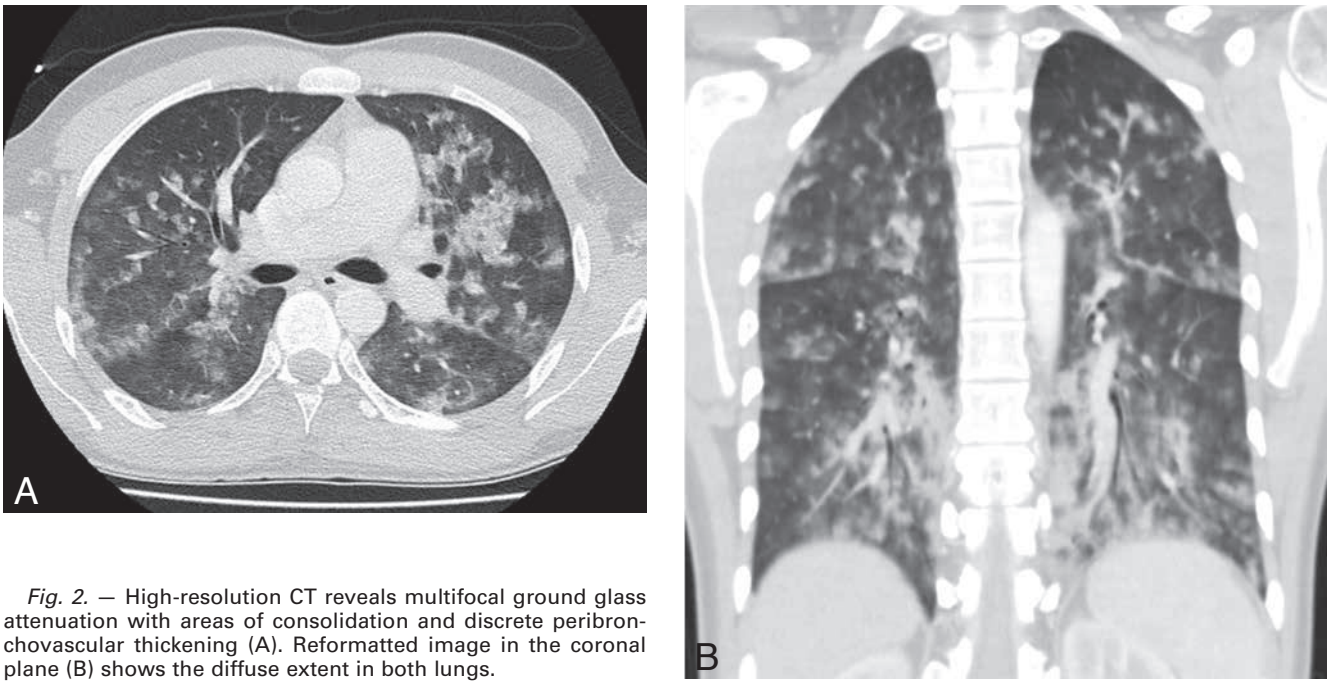

Fig. 2. — High-resolution CT reveals multifocal ground glass attenuation with areas of consolidation and discrete peribronchovascular thickening (A). Reformatted image in the coronal plane (B) shows the diffuse extent in both lungs.

or crack, are known to cause acute lung injury (noncardiogenic pulmonary edema) and DAH. The mechanism by which the increased pulmonary capillary permeability occurs in acute lung injury is unclear, but is thought to be the consequence of a pulmonary endothelial abnormality (2).

Diffuse alveolar hemorrhage and acute lung injury are radiographically indistinguishable. Because of this the development of respiratory failure with bilateral airspace consolidation that typically appear shortly after crack use and rapid recovery after the cessation of the responsible agent had been termed "crack lung". Lung biopsy of this condition has revealed diffuse alveolar damage, alveolar hemorrhage, and interstitial and intra-alveolar inflammatory cell infiltration (3).

During the past 2 decades, the acute toxic effects of MDMA have been widely investigated. Observational studies in humans and direct animal experiments had demonstrated typical acute toxic syndromes, including hyperthermia, hyponatremia, myocardial ischemia, intracerebral hemorrhage, acute hepatic failure and acute hallucinogenic psychosis (3).

Although there are a few case reports of MDMA-related acute noncardiogenic pulmonary edema to be found in the literature (4), limited work has been done concerning the pulmonary toxic effects of MDMA. The mechanism causing noncardiogenic pulmonary edema due to abuse of amphetamines, a structurally similar material with MDMA, is still not clear but may be due to direct cellular toxicity as suggested by animal studies (4).

To our knowledge there hasn't been a single case report of MDMA-induced diffuse alveolar hemorrhage after oral amphetamine intake published. Our patient provided us with an experience of uncomplicated DAH after the oral use of MDMA (ecstasy) that resolved under supportive care without significant sequelae.

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