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Bad for Breathing: A Pictorial of Drug-Induced Pulmonary Disease

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Drug-induced lung disease has been described with over 300 different agents, some of which are asymptomatic and may first present on imaging. These pulmonary diseases may present with variable imaging manifestations, and often overlap with other etiologies such as rejection, lymphoproliferative disorders, and infection that may be suspected in this patient population. However, there are several drugs that have classic imaging appearances, and in the proper clinical context, the radiologist should include their toxicity in the differential diagnosis, potentially expediting withdrawal of the drug and avoiding irreversible lung injury such as fibrosis.

Cocaine

The term “crack lung” refers to an acute pulmonary syndrome that occurs after the inhalation of free-base cocaine and is secondary to prolonged inflammatory pulmonary injury. Cocaine inhalation is associated with fever, hypoxemia, hemoptysis, respiratory failure, and diffuse alveolar opacities.[1] Lung tissue specimens obtained in affected patients reveal diffuse alveolar damage (DAD), alveolar hemorrhage, eosinophilic interstitial and alveolar cell infiltration, and deposition of immunoglobulin E.[1] Chest radiographs demonstrate multifocal airspace disease.[1] Computer tomographic (CT) imaging demonstrates predominantly lower lobar multifocal ground-glass attenuation with subpleural sparing, as would be expected in diffuse alveolar hemorrhage (Fig 1).[2] Recurrent episodes of hemorrhage may lead to eventual subpleural reticulation and honeycombing.[2]

Methylphenidate (Ritalin)

Intravenous use of methylphenidate has been associated with the development of lower lobe panlobular emphysema, akin to that seen with alpha-1-antitrypsin deficiency. Microscopic talc granulomata may be seen, relating to the injection base.[3,4] Chest radiographs show hyperinflation with lower lobe lucency of diffuse panlobular emphysema and CT demonstrates diffuse areas of low attenuation throughout the lungs with a paucity of vessels (Fig 2).[5]

Amiodarone

Amiodarone is a commonly used antiarrhythmic, making this a toxicity most often associated with patients with cardiac disease. Toxicity occurs in 5%-10% of patients and can present with both pulmonary and hepatic manifestations. Risk factors for developing toxicity include a daily dose greater than 400 mg and elderly status of the patient.[6] Pulmonary imaging classically demonstrates distinctive focal high-attenuation consolidations, which are most commonly peripheral in distribution (Fig 3). However, nonspecific interstitial pneumonia (NSIP) is the most commonly encountered manifestation.[6] Owing to the high iodine content and metabolism of amiodarone in the liver, this organ may also have high attenuation. The combination of high attenuation in the lung, liver, and spleen is characteristic of amiodarone toxicity. Treatment is cessation of amiodarone and administration of corticosteroids.[7]

Heavy Metals (Benzenes)

Heavy-metal pneumoconiosis results from inhalational exposure to tungsten carbide, cobalt, or diamond-cobalt. The histopathological finding of giant-cell interstitial pneumonia is pathognomonic for this disorder.[8,9] CT imaging often demonstrates patchy ground-glass attenuation and consolidation in the mid and lower lungs (Fig 4). With long-term exposure, fibrosis with architectural distortion, traction bronchiectasis, and honeycombing can occur (Fig 5).[9,10] Prognosis is favorable with early recognition of the patient’s occupational exposure, and removal of the offending environment.

Nitrofurantoin

Nitrofurantoin is an antibiotic commonly used to treat urinary tract infections. Pulmonary toxicity is uncommon with its use and

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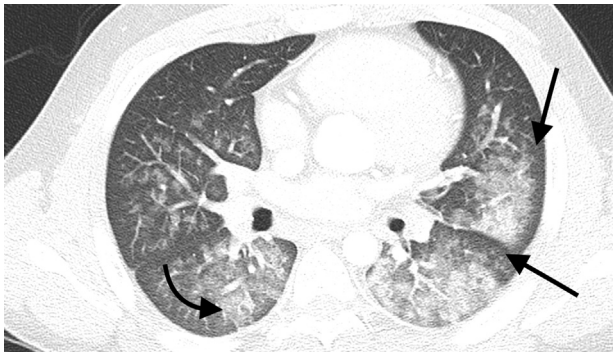

Fig. 1. Cocaine hemorrhage. Axial CT in lung window in a patient with recent cocaine abuse shows pulmonary hemorrhage and centrilobular ground-glass attenuation (curved arrow). Note the subpleural sparing (straight arrows), which is a hallmark of pulmonary hemorrhage.

Fig. 2. Methylphenidate (Ritalin) abuse. Axial CT in lung window shows emphysema with diffuse areas of low attenuation throughout the lungs with a paucity of vessels (arrows).

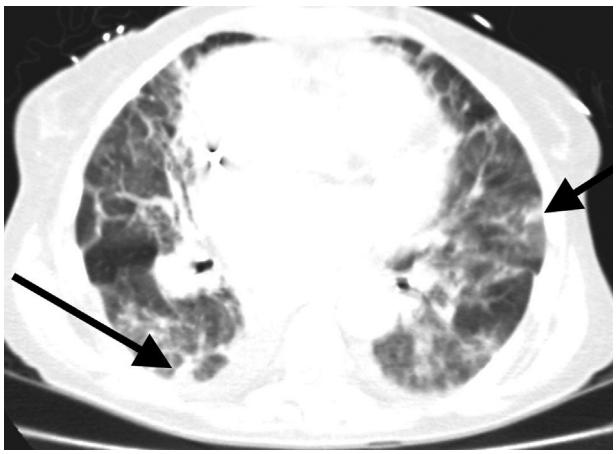

Fig. 3. Amiodarone toxicity. Axial CT in lung window shows bilateral ground-glass opacities and interstitial disease (arrows). Note cardiomegaly in the setting of long-standing cardiac disease.

occurs in less than 1% of patients; however, when toxicity occurs, it may be either acute or chronic.[11] Acute toxicity is more common, usually occurring within 2 weeks of drug initiation. Imaging demonstrates diffuse bilateral basilar-predominant heterogeneous opacities and pleural effusions. Laboratory assessment may show peripheral eosinophilia.[6] Chronic toxicity is less common and often occurs after months or years of continuous use. Imaging in chronic

Fig. 4. Heavy-metal poisoning— tungsten. Axial HRCT in a 66-year-old male shows multifocal basilar-predominant ground glass with associated traction bronchiectasis (curved arrows) and subpleural fibrosis (straight arrow). HRCT, high resolution computer tomography.

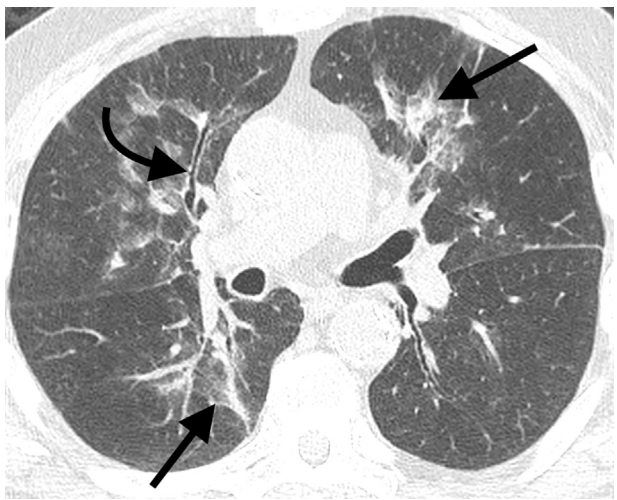

Fig. 5. Heavy-metal pneumoconiosis—benzene. Axial CT in lung window shows multifocal areas of ground-glass attenuation (arrows). There is also traction bronchiectasis (curved arrow) that can be seen with long-standing exposure.

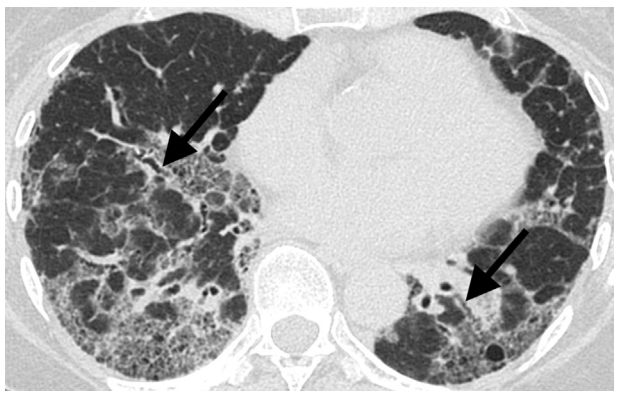

Fig. 6. Nitrofurantoin toxicity. Axial CT in chronic toxicity shows an NSIP pattern with bilateral reticular opacities with few areas of bronchiolectasis (arrows) without honeycombing.

toxicity typically demonstrates an NSIP pattern with bilateral, predominantly basilar, reticular opacities (Fig 6).[11]

Methotrexate

Methotrexate is a common chemotherapeutic agent used in the treatment of malignancies, as well as for the treatment of severe