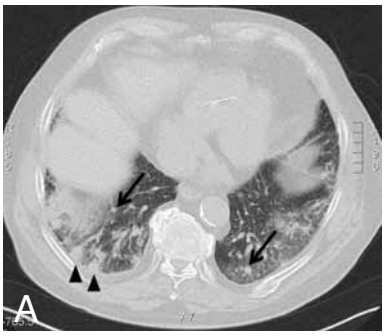
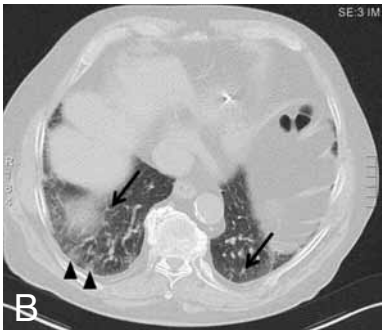
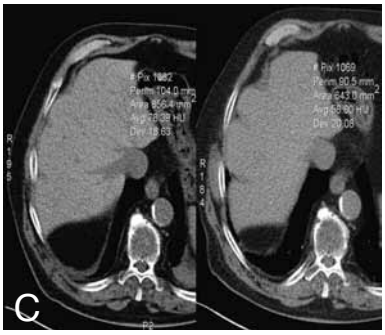
JBR–BTR, 2012, 95: 109.

IMAGES IN CLINICAL RADIOLOGY







Amiodarone induced pulmonary toxicity mimicking malignancy at initial presentation

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An 81-year-old man with an extensive cardiac history was admitted to the cardiology department because of increasing shortness of breath. His medical history included melanoma and prostate adenocarcinoma 7 and 4 years ago respectively. His medication consisted of antihypertensive, anticoagulant and anti-arrhytmic drugs. Non-enhanced computed tomography (CT) of the chest revealed bilateral zones of consolidation (arrowheads) and multiple nodules of varying size (arrows) with predominant localization in the lower lobes (Fig. A). The lowest axial slices through the liver showed an increased attenuation of the liver parenchyma suggestive of amiodarone-induced liver disease (Fig. C(1), ROI: 78 HU). In view of his oncological history the multiple nodular lesions were primarily suspicious for metastasis. Combined pulmonary and liver imaging findings also raised the possibility of amiodarone induced pulmonary toxicity (AIPT). The patient – however – refused further diagnostic testing. Therefore await-and-see policy was implemented and amiodarone was withdrawn. Follow-up CT of the chest 4 months later showed marked regression of the lesions (Fig. B) and a decreased attenuation of the liver parenchyma confirming the diagnosis of AIPT (Fig. C(2), ROI: 56 HU).

Comment

Amiodarone is one of the most frequently prescribed anti-arrhythmic agents in Europe. It effectively reduces and prevents (supra)ventricular tachyarrhythmias. However, amiodarone also has multiple potentially life-threatening adverse reactions. The most important one is AIPT often starting within months after initiating therapy. Known risk factors are a high cumulative dose, a daily dose exceeding 400 mg/day, pre-existing lung disease and age. Symptoms are nonspecific and include a nonproductive cough and dyspnea. Pulmonary function testing unveils a restrictive or mixed obstructive-restrictive pattern and often a diminished lung diffusion capacity of more than 20%.

Several forms of pulmonary disease can occur among patients treated with amiodarone. Nonspecific interstitial pneumonia (NSIP), cryptogenic organising pneumonia (COP formerly known as Bronchiolitis Obliterans Organising Pneumonia (BOOP)) and acute respiratory distress syndrome are radiologically the most frequently encountered presentations. The combination of bilateral consolidations and multiple nodular lesions such as in our case, are typical features of COP. In the appropriate clinical context, malignancy is essential in the differential diagnosis of these nodules. Extrapulmonary involvement such as an increased attenuation of the liver or spleen on non-enhanced CT is an additional argument for possible amiodarone induced toxicity.

The exact mechanism of the pulmonary toxicity is still highly debated but several mechanisms have been proposed. Cell injury can result from a cytotoxic reaction of the drug but an indirect immunological reaction has also been suggested.

Other causes of respiratory failure such as pulmonary embolism, congestive heart disease or infection need to be ruled out in order to initiate the correct therapy.

When amiodarone is considered the cause of the pulmonary toxicity, withdrawal of the drug is essential often in combination with low-dose corticosteroids. Resolution of the lesions on imaging can range from 2 to 24 months.

Reference

1. Papiris S.A., Triantafillidou C., Kolilekas L., Markoulaki D., Manali E.D.: Amiodarone Review of Pulmonary Effects and Toxicity. *Drug Saf*, 2010, 33: 539-558.

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