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Amiodarone pulmonary toxicity : A CASE REPORT.

Abstract

2 Amiodarone is a class III antiarrhythmic agent widely used in clinical practice; however, its use may be complicated by potentially severe pulmonary toxicity. We report the case of a 66-year-old man receiving long-term amiodarone therapy for atrial fibrillation who was admitted for progressive dyspnea.

Physical examination revealed diffuse bilateral crackles. Laboratory tests showed leukocytosis with elevated C-reactive protein, and arterial blood gas analysis demonstrated severe hypoxemia. Chest computed tomography revealed bilateral multifocal pulmonary consolidations associated with diffuse ground-glass opacities.

The lack of improvement under empirical antibiotic therapy led to suspicion of amiodarone-induced pneumonitis. Discontinuation of amiodarone combined with systemic corticosteroid therapy resulted in rapid clinical improvement and near-complete radiological resolution on follow-up imaging.

Amiodarone-induced pulmonary toxicity should be considered in any patient presenting with unexplained dyspnea while receiving this medication. Early diagnosis and prompt drug withdrawal are essential determinants of prognosis.

Keywords: Amiodarone; Pulmonary toxicity; Drug-induced pneumonitis.

Introduction

Amiodarone is a commonly prescribed antiarrhythmic agent used in the management of cardiac rhythm disorders. Although highly effective, its use may be associated with several adverse effects, among which pulmonary toxicity represents one of the most serious complications.

The incidence 1 of amiodarone-induced pulmonary toxicity (APT) ranges from 5% to 15% and depends primarily on the administered dose, cumulative exposure, patient age, and the presence of pre-existing lung disease.

Diagnosis remains challenging due to the absence of a specific diagnostic test and the wide heterogeneity of clinical and radiological presentations. Early detection is crucial to reduce the risk of severe complications (1).

Case présentation:

A 66-year-old man with a history of paroxysmal atrial fibrillation at embolic risk and arterial hypertension, treated with long-term amiodarone therapy, was admitted to the pulmonology department for progressive dyspnea occurring with minimal exertion and at night. No history of allergy, occupational exposure, or systemic disease was identified.

On admission, physical examination revealed mild hepatojugular reflux without other signs of overt heart failure. Pulmonary auscultation detected diffuse bilateral inspiratory crackles.

Laboratory investigations showed: leukocyte count of 13,700/mm³ with neutrophil predominance (87%), C-reactive protein of 188 mg/L, BNP of 223 ng/L, normal renal and hepatic function, and normal thyroid function and serum electrolytes. Arterial blood gas analysis on room air revealed severe hypoxemia (PaO_2 < 60 mmHg) with normocapnia, requiring transient oxygen therapy.

Echocardiography demonstrated preserved left ventricular systolic function, moderate right ventricular dysfunction, and mildly elevated filling pressures.

Non-contrast chest computed tomography revealed bilateral multifocal pulmonary consolidations surrounded by diffuse ground-glass opacities, without a marked apico-basal gradient. Triple-vessel coronary calcifications were noted. No pleural or pericardial effusion was observed.

Empirical antibiotic therapy combined with oxygen supplementation was initiated, without clinical improvement. Given the lack of response, amiodarone-induced pulmonary toxicity was suspected. The drug was discontinued, and systemic corticosteroid therapy with gradual tapering was started.

Clinical evolution was favorable and rapid. Dyspnea significantly improved, and oxygen therapy was progressively reduced, being maintained only during exertion.

One and a half months after amiodarone withdrawal, the patient reported near-complete

resolution of dyspnea. Resting SpO_2 was 95% and did not decrease below 92% during exertion. Pulmonary auscultation was normal. Follow-up CT imaging showed near-complete resolution of consolidations and interstitial abnormalities.

Corticosteroid therapy and oxygen supplementation were discontinued without complications, and the patient was referred for outpatient follow-up.

Figure 1:

Axial chest CT scan (lungwindow) demonstrating bilateral multifocal pulmonary consolidations surrounded by diffuse

ground-glass opacities, without a significant apico-basal gradient.

Figure 2:

Axial chest CT scan (lung window) showing near-complete resolution of the previously described alveolar consolidations and most bilateral interstitial abnormalities. No newly unmasked suspicious pulmonary opacity and no pleural effusion are observed.

Discussion

Amiodarone, a class III antiarrhythmic agent, remains effective in the treatment of refractory arrhythmias; however, its use is limited by the risk of severe pulmonary complications. Amiodarone-induced pulmonary toxicity affects approximately 1–17% of patients, with increasing prevalence depending on [1] cumulative dose, duration of therapy, advanced age, and reduced baseline diffusion capacity for carbon monoxide (DLCO) (2).

Major risk factors include high cumulative dosage, prolonged treatment duration, pre-existing lung disease, and prior thoracic surgery or angiographic procedures (3).

The pathophysiology [1] of amiodarone-induced pulmonary toxicity involves two principal mechanisms: a direct cytotoxic effect on pneumocytes leading to phospholipid accumulation and parenchymal injury, and an immune-mediated hypersensitivity reaction

(4). These mechanisms may result in diverse clinical manifestations, including interstitial pneumonitis, organizing pneumonia, pulmonary nodules, alveolar hemorrhage, pleural effusion, and, more rarely, acute respiratory distress syndrome (5).

Clinical symptoms are often nonspecific and include progressive dyspnea, dry cough, pleuritic chest pain, and general malaise. Laboratory abnormalities are inconsistent but may include leukocytosis or elevated lactate dehydrogenase levels. Diagnosis relies primarily on careful clinical assessment confirming drug exposure and excluding infectious, allergic, autoimmune, or cardiac causes.

Radiologically, the most common presentation is a subacute interstitial pneumonitis characterized by bilateral, diffuse, often asymmetric ground-glass opacities or alveolar infiltrates, sometimes with basal predominance. A nodular presentation is possible but uncommon. Increased attenuation in the lung parenchyma, liver, or spleen on CT imaging strongly supports the diagnosis due to iodine accumulation (6).

Bronchoalveolar lavage (BAL) rarely provides specific diagnostic information. It may reveal increased neutrophils or lymphocytes, occasionally CD8+ T lymphocytes, but may remain normal in approximately 20% of cases. The presence of foamy macrophages reflects pulmonary accumulation of amiodarone but is not pathognomonic. In the present case, BAL could not be performed due to severe hypoxemia (7).

Pulmonary function tests typically demonstrate a restrictive ventilatory defect associated with reduced diffusing capacity.

Management of amiodarone-induced pneumonitis primarily consists of discontinuation of the drug in coordination with the cardiologist. When clinical, radiological, or functional impairment is significant, systemic corticosteroid therapy may be initiated. Dosage and duration depend on severity and may extend from several weeks to several months, given the long half-life of amiodarone and its metabolites (8).

Clinical and radiological outcomes are generally favorable; however, chronic respiratory failure may develop in approximately 15% of patients. Corticosteroid tapering must be gradual and carefully monitored, as relapses—sometimes more severe and difficult to

control—may occur.

Conclusion

Amiodarone-induced pneumonitis is a frequent and potentially serious complication in patients receiving prolonged therapy. Its clinical and radiological features are variable and nonspecific, requiring a high index of suspicion.

Management relies primarily on immediate discontinuation of amiodarone, supplemented when necessary by systemic corticosteroid therapy. In most cases, the condition is reversible, particularly when treatment is initiated promptly.

Regular pulmonary follow-up, including clinical evaluation, thoracic imaging, and pulmonary function testing, is essential before and during therapy to prevent complications and improve prognosis.

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