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Olgu Sunumu

Management of Hyperacute Amiodarone-Induced Pulmonary Toxicity

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ABSTRACT

One of the major complications of amiodarone therapy is acute pulmonary toxicity (APT). Patients tend to have amiodarone-induced toxicity after Coronary Artery Bypass Surgery (CABG). For this reason, priority should be given to the treatment of this fatal complication. Acute pulmonary toxicity due to amiodarone is a treatable phenomenon if diagnosed at an early stage, for this reason, meticulous follow-up and suspicion are of great importance. We presented the treatment of a 72-year-old male who underwent 4-vessel coronary artery bypass grafting surgery but developed amiodarone-induced pulmonary toxicity immediately after amiodarone infusion.

Keywords: *amiodarone, hyperacute, pulmonary toxicity*

ÖZ

Amiodaronla Oluşan Hiperakut Pulmoner Toksisitenin Yönetimi

Amiodaron tedavisinin en önemli komplikasyonlarından biri akut pulmoner toksisitedir (APT). Koroner Arter Baypas Cerrahisi (KABC) sonrasında hastalar amiodaron kaynaklı toksisiteye eğilimi gösterirler. Bu nedenle bu ölümcül komplikasyonun tedavisine öncelik verilmelidir. Amiodarona bağlı akut pulmoner toksisite, erken tanı konulması durumunda tedavi edilebilir bir fenomendir, bu nedenle titiz takip ve şüphe çok önemlidir. Dört damar koroner arter baypas ameliyatı yapılan 72 yaşındaki bir erkek hastada, amiodaron infüzyon tedavisinden hemen sonra, amiodaron nedeniyle gelişen pulmoner toksisitenin tedavisini sunduk.

Anahtar kelimeler: *amiodaron, hiperakut, pulmoner toksisite*

INTRODUCTION

Amiodarone is a potent antiarrhythmic agent which is widely used in tachyarrhythmias. Amiodarone-induced pulmonary toxicity (APT) is the most important complication which was first reported in 1980. The incidence of APT ranges from 1 to 17% and mortality ranges from 1 to 33% [1]. Pulmonary toxicity, hypo/hyperthyroidism, corneal crystalline deposition, unspecific hepatitis, elevated liver enzymes, neurologic side effects, photosensitization of the skin are reported as adverse effects. However, pulmonary toxicity is the most dangerous adverse effect and usually seen during medium-long term therapy. There is no predictor of direct cytotoxicity or hypersensitivity reaction [2]. We report a hyperacute APT which leads to fatal interstitial pneumonitis within 72 hours after initiation of the treatment.

CASE REPORT

A 74-year-old male applied to the emergency service with sudden chest pain and shortness of breath. Non-ST-segment elevation myocardial infarction associated with increased cardiac enzymes was diagnosed and transthoracic echocardiography revealed an ejection fraction (EF) of 60% following coronary angiography, 4-vessel coronary artery bypass graft-

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ing surgery was performed. Intravenous (IV) amiodarone infusion therapy was administered in the intensive care unit for 48 hours due to rapid ventricular response atrial fibrillation in the postoperative period. On postoperative third day, even though 100% FiO_2 was applied, arterial oxygen saturation (SpO_2<90), and partial oxygen pressure (40 mmHg) decreased while partial carbon dioxide pressure (41 mmHg) increased as revealed in biochemical analysis. On physical examination, bilaterally diffuse cracks, low-grade fever, tachypnea (RR 30/min) and unproductive cough were detected. The patient's clinic got worse and demand for oxygen increased. The chest X-ray showed the new infiltration areas, especially on the right side (Figure 1A,B). Transthoracic echocardiography was repeated and no new wall motion impairment or pericardial collection was detected. Computed tomography (CT) scan showed diffuse vitreous opacities on bilateral lungs and pleural effusion on the right lung (Figure 1C). Pulmonary edema and embolism, pulmonary hypertensive episodes, fluid overload, infective pneumonia, valve dysfunction, postoperative cardiac insufficiency and pulmonary hemorrhage were excluded in the differential diagnosis of the patient. Upon suspicion of APT amiodarone therapy was discontinued and IV corticosteroid therapy was initiated (IV prednisone 2 mg/kg/day) and continuous BIPAP was required due to persistent hypoxemia. Blood, sputum, and bronchoalveolar lavage cultures were negative. Oxygen saturation and blood

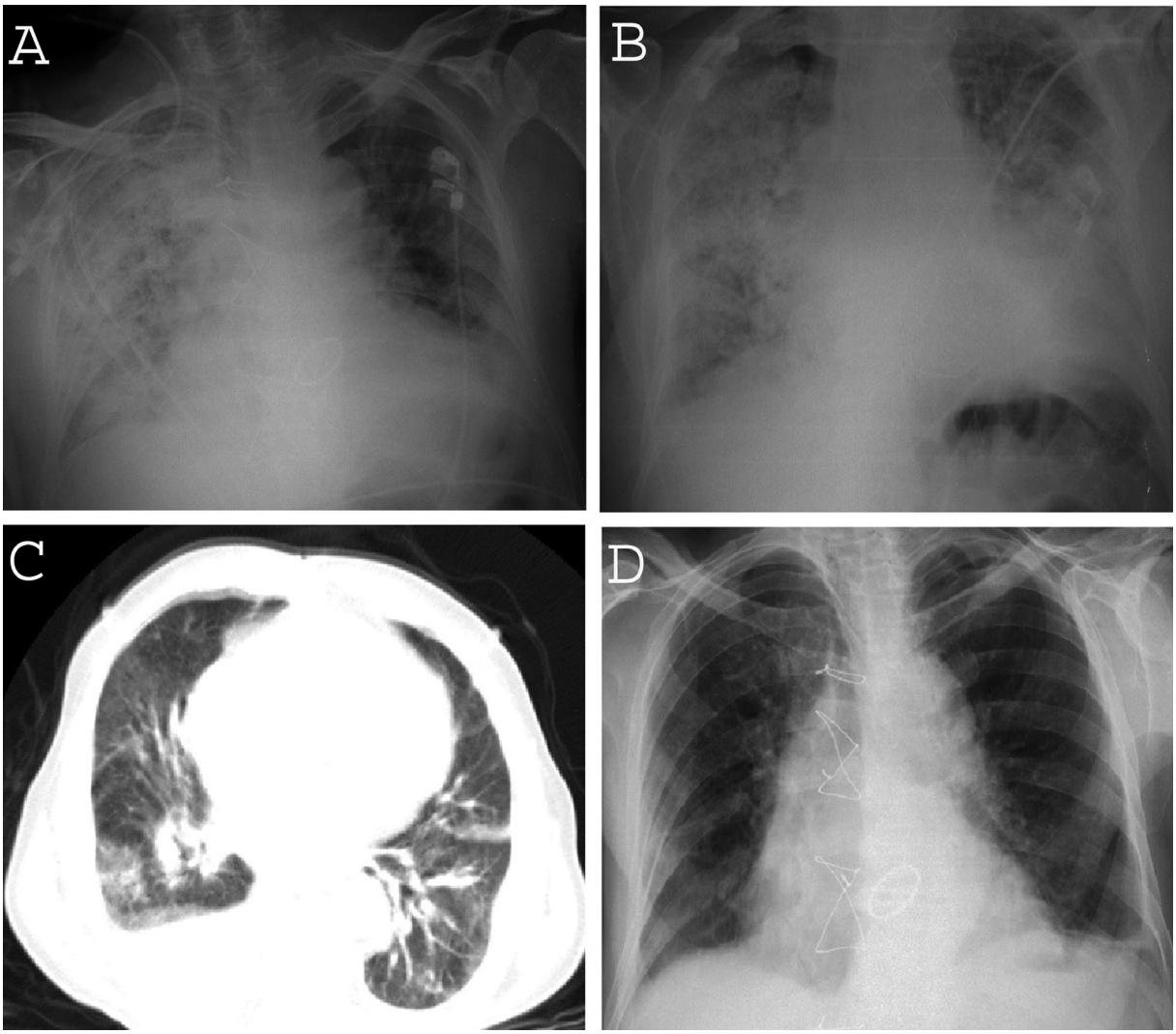

Figure 1. Chest X-ray shows the new infiltration areas especially on the right side (Figure 1A). The patient's clinic did not improve and the parenchymal opacity progressed in the chest X-ray (Figure 1B). CT scan demonstrated diffuse ground glass opacities involving bilateral lungs and pleural effusion on the right (Figure 1C). Pulmonary infiltration disappeared Chest X-ray (Figure 1D).

gas oxygen values improved. The need for high-dose steroid, and oxygen decreased and pulmonary infiltrations disappeared in chest X-ray (Figure 1d) after 24 hours. IV steroid therapy was continued for one week. He recovered with steroids and was discharged on the 16th postoperative day with sinus rhythm. The patient was doing well and did not show pulmonary symptoms after 6 months of follow-up.

DISCUSSION

Amiodarone-induced pulmonary toxicity can be presented as alveolar infiltrates, pleural thickening, pulmonary nodules or pleural effusion, which can be irreversible and lethal. Clinical symptoms and signs may be nonspecific such as fatigue, nonproductive cough, weight loss, fever, pleuritic chest pain and bilateral inspiratory crackles upon auscultation [3]. Interstitial pneumonitis, progressive pulmonary fibrosis, drugs and acute respiratory distress syndrome can also be presented. Diffuse or localized interstitial, alveolar opacities can be detected by chest X-ray or CT scan. Diagnosis is based on high clinical suspicion, patient's history, radiographic and clinical findings. APT may occur even at any time during treatment or even after cessation of treatment [4]. Applying a cumulative dose of 400 mg/day for 2 months, long duration of therapy, advanced age, and pre-existing pulmonary disease increase the risk of pulmonary toxicity. Delayed diagnosis can lead to lethal complications. The mainstay treatment of the acute toxicity is the administration of IV corticosteroid therapy, which helps to improve acute hypoxia and radiological findings [5]. Although clinical symptoms usually resolve within 2-4 weeks, radiographic findings generally improve within 3 months.

In conclusion, when we consider the comorbid factors undergoing cardiac surgery, we usually observe findings that remind us amiodarone toxicity. When we used amiodarone in this patient group, amiodarone toxicity should be always kept in mind. In addition, acute pulmonary toxicity due to amiodarone is a treatable phenomenon if diagnosed early. For this reason, meticulous follow-up and suspicion are of great importance.

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