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Unusual Presentation of Amiodarone Induced Pulmonary Toxicity

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Abstract

Pulmonary complications are estimated to occur in 1-5% of patients using amiodarone. Interstitial pneumonitis is the most common of these complications. Amiodarone induced pulmonary toxicity (AIPT) is a drastic side effect of amiodarone that usually occurs with higher doses and prolonged duration of treatment. However, rare presentations of AIPT can still occur. The pathophysiology AIPT is not yet clear; however, direct toxic injury to lung cells and indirect immune-mediated lung injury are possible mechanisms. We present a case that highlights an unusual presentation for AIPT, with acute respiratory failure occurring over week duration after two months of starting low dose of amiodarone.

Keywords

amiodarone; pulmonary toxicity; atrial fibrillation; arrhythmia

Introduction

Amiodarone induced pulmonary toxicity (AIPT) is a drastic side effect of amiodarone that usually occurs with higher doses and prolonged duration of treatment. However, rare presentations of AIPT can still occur.

Case Report

A 62-year-old man was evaluated in the emergency department for acute shortness of breath for one week. He denied associated chest pain, cough, hemoptysis, fever and weight loss

On exam, heart rate was irregular at 96 bpm and oxygen saturation of 87% on room air. Physical examination was significant for bilateral diffuse fine inspiratory crackles

His past history is significant for hypertension, coronary artery disease, atrial fibrillation diagnosed 2 months prior, for which he was started on oral amiodarone 200 mg daily and Apixaban.

Chest radiograph showed bilateral ground glass infiltrates. His laboratory work showed white blood cell count of 21 x103/mm with neutrophils of 92% and hemoglobin 12.4 g/dL. Arterial blood gas done on room air showed marked hypoxemia with PaO2 of 51 mmHg, with mildly low PaCO2 of 33 mmHg and normal PH of 7.42. Computed tomography (CT) of chest with contrast revealed ground glass opacities predominant in lower lobes, suggestive of interstitial pneumonitis. HIV test was negative. Work up for autoimmune diseases was negative as well.

Patient was started on broad spectrum antibiotic therapy and oral prednisone 60 mg daily. Amiodarone was discontinued due to suspected AIPT. Apixaban was also discontinued because of suspected underlying pulmonary hemorrhage.

Despite these measures patient's respiratory status was not improving; oxygen demand increased from nasal cannula to non-re-breather mask within four days. He further worsened and needed to be on Bi-level positive airway pressure (BiPAP), with eminent intubation. Antibiotics were stopped given absence of any evidence for infective etiology with negative blood and sputum cultures. Steroids treatment was escalated to IV methylprednisolone 40 mg every 6 hours. Over two weeks' duration, the patient responded to high dose steroid regimen with decrease of oxygen demands to nasal oxygen at 4 L/hour. Patient needed another two weeks to be off oxygen.

Discussion

Pulmonary complications are estimated to occur in 1-5% of patients using amiodarone [1]. Interstitial pneumonitis is the most common of these complications [2]. The pathophysiology of amiodarone induced pulmonary toxicity (AIPT) is not yet clear; however, direct toxic injury to lung cells and indirect immune-mediated lung injury are possible mechanisms [3]. Microscopic picture includes numerous lipid laden foamy macrophages in alveolar air-spaces. These cells can still be found in patients taking amiodarone without pulmonary toxicity, so they are not pathognomonic of AIPT [1].

The diagnosis of AIPT in our case was established based on the new onset of pulmonary symptoms, CT chest findings showing interstitial pneumonitis, recovery on discontinuation of amiodarone, along with absence of any infectious etiology and negative workup for other possible differentials. The response to steroids also was typical for AIPT. Pulmonary hemorrhage was a possible differential diagnosis; however, absence of hemoptysis and stable red blood cell count throughout the hospital stay were against that diagnosis.

AIPT has been shown to correlate with the duration and intensity of amiodarone treatment [1]. Daily maintenance and cumulative doses of amiodarone are considered independent predictors of lung toxicity especially with daily doses of more than 500 mg [1]. Patients receiving amiodarone for 6-12 months have been identified as the highest risk for developing lung toxicity [3]. Cumulative incidence of AIPT correlates with duration of treatment; where its 4.2% after 1 year, 7.8% after 3 years and 10.6% after 5 years of treatment [4]. AIPT most commonly presents as subacute illness with progressive exertional dyspnea and non-productive cough [5], which usually requires months from amiodarone initiation to become clinically significant [3]. The unusual features in our patient were the relatively low daily dose of 200 mg and acuity of his presentation after only two months from starting amiodarone.

Conclusion

This case highlights an unusual presentation for AIPT, with acute respiratory failure occurring over week duration after two months of starting low dose of amiodarone.

Figure

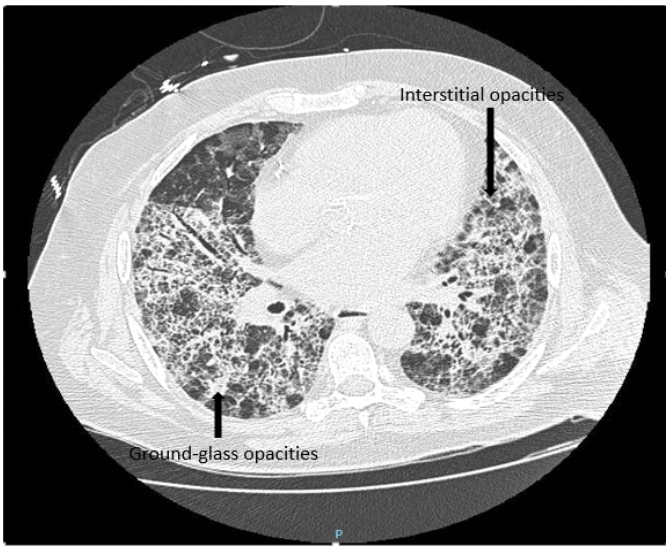

Figure 1: CT chest with contrast showing diffuse interstitial and ground glass opacities, suggestive of interstitial pneumonitis.

References

1. Papiris, S.A., et al., Amiodarone: review of pulmonary effects and toxicity. Drug Saf, 2010. 33(7): p. 539-58.

2. Martin, W.J., 2nd and E.C. Rosenow, 3rd, Amiodarone pulmonary toxicity. Recognition and pathogenesis (Part I). Chest, 1988. 93(5): p. 1067-75.

3. Ernawati, D.K., L. Stafford, and J.D. Hughes, Amiodarone-induced pulmonary toxicity. Br J Clin Pharmacol, 2008. 66(1): p. 82-7.

4. Yamada, Y., et al., Incidence and predictors of pulmonary toxicity in Japanese patients receiving low-dose amiodarone. Circ J, 2007. 71(10): p. 1610-6.

5. Dusman, R.E., et al., Clinical features of amiodarone-induced pulmonary toxicity. Circulation, 1990. 82(1): p. 51-9.

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