Amiodarone Induced Pulmonary Toxicity in a Case of Atrial Fibrillation

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Abstract

Pulmonary opacities have many causes we are presenting a case of pulmonary opacity in a patient of breathlessness; who was on amiodarone for atrial fibrillation (AF).

Introduction

Pulmonary fibrosis is known side effect of amiodarone when given for long term therapy. Some cases reported with single dose of amiodarone with idiosyncratic effect. We present a case of pulmonary fibrosis as a long term side effect of amiodarone.¹

Case Report

A 55 year man non alcoholic, non smoker, office peon by profession known case of diabetes mellitus, hypertension since 10 years presented to us with complaints of dyspnea on exertion since 3 days, heaviness of chest on right side since 3 days, nonproductive cough since 1-2 days. Patient was alright 3 days back when he started with increase in breathlessness and dry cough. He previously had mild dyspnea on exertion which is aggravated in last 3 days. There was no history of expectoration, fever with chills, abdominal pain, sweating, palpitations, giddiness, orthopnoea, paroxysmal nocturnal dyspnea, pedal edema, facial puffiness. There was history of admission for an episode of AF three months back which was controlled with pharmacotherapy with amiodarone, ecosprin, atorvastatin. There was no history of major surgical illness in past. At the time of admission he was afebrile, pulse rate 68/min regular bilaterally equal, blood pressure 160/90 mm of Hg, respiratory rate of 26/min, oxygen saturation of 85 % on room air. There was mild pallor. There was no edema, icterus, cyanosis, and lymphadenopathy. All peripheral pulses were palpable. His spine was normal, jugular venous pressure was normal, hepatojugular reflux was absent. His cardiovascular, nervous system and abdominal examination was normal. His respiratory examination revealed normal chest expansion, air entry reduced in axillary, infraaxillary, mammmary and supramammary area on right side and on left side inframammary area, percussion note was impaired in similar areas as mentioned above. There was minimal crepitations on bilateral bases and minimal wheeze present in all areas right > left tactile vocal fremitus and vocal resonance reduced on right side.

Investigations revealed hemoglobin 9.0 gm/dl, total leukocyte count of 7700/cmm out of which 80% were polymorphs, 13% lymphocytes, 3% eosinophils, 4% monocytes and 1% basophils. Peripheral blood smear show normocytic normochromic picture. Platelet count was 3, 13,000/cmm, ESR was 60, urine routine was normal, cardiac biomarkers were negative. Renal functions were: BUL 35.7, creatinine 1.40, Na: 142, K: 3.9, CL: 104 and serology was negative. ECG shows left anterior hemi-block which was there since 3 months. 2DECHO shows EF of 60%, mild concentric LVH, no RWMA, grade II diastolic dysfunction, mild PAH 35 mm of Hg and normal chamber dimensions. USG (abdomen and pelvis) showed mild hepatomegaly, Multiple GB calculi, LFT and TFT were within normal limits. Tuberculin test was negative. His chest roentgenogram shows inhomogenous opacities in right lung field and left lower zone (Figure 1).

Patient settled with diuretics and nebulization with bronchodilators. On day 3 his chest roentgenogram remains same, so we performed high resolution computed tomography of thorax. Which shows interstitial fibrosis affecting right upper and lower
lobes and to some extent middle lobe with thickening of septae and honeycombing and mild effusion on right side (Figure 2).

Clinically patient was diagnosed as amiodarone induced pulmonary fibrosis/toxicity. Amiodarone was discontinued, patient started with oral steroids. Clinical improvement was uneventful. After 7 days of treatment his saturation on room air is 97%. DOE reduced to NYHA CLASS 1 from CLASS 3. Slit lamp examinations revealed no cataract or any kind of deposits. After 15 days of treatment patient was settled completely and his chest roentgenogram was as given bellow with reduced fibrosis and no effusion (Figure 3).

Discussion

Adverse effects during long-term therapy reflect both the size of daily maintenance doses and the cumulative dose (i.e., to duration of therapy), suggesting that tissue accumulation may be responsible. The most serious adverse effect during chronic amiodarone therapy is pulmonary fibrosis, which can be rapidly progressive and fatal. Underlying lung disease, doses of 400 mg/day or more, and recent pulmonary insults such as pneumonia appear to be risk factors. Serial chest X-rays or pulmonary function studies may detect early amiodarone toxicity, but monitoring plasma concentrations has not been useful. With low doses, such as 200 mg/day or less used in atrial fibrillation, pulmonary toxicity is unusual. Other adverse effects during long-term therapy include corneal microdeposits (which often are asymptomatic), hepatic dysfunction, neuromuscular symptoms (most commonly peripheral neuropathy or proximal muscle weakness), photosensitivity, and hypo- or hyperthyroidism. Treatment consists of withdrawal of the drug and supportive measures, including corticosteroids, for life-threatening pulmonary toxicity; reduction of dosage may be sufficient if the drug is deemed necessary and the adverse effect is not life-threatening. Despite the marked QT prolongation and bradycardia typical of chronic amiodarone therapy, torsades de pointes and other drug-induced tachyarrhythmias are unusual. Diagnostic criteria for amiodarone pulmonary toxicity are as follows. Two out of seven must be there to label it.

1. New onset of pulmonary symptoms such as dyspnoea, cough, or pleuritic chest pain
2. A decrease in the DLCO of 20% from the pretreatment value, or if none is available, a value less than 80% of predicted
3. New chest radiographic abnormality such as an interstitial or alveolar infiltrate;
4. Abnormal lung uptake with gallium-67 radioisotope
5. Improvement in symptoms with drug discontinuation
6. CD8+ lymphocytosis on BAL
7. Lung biopsy with IP, BOOP, DAD or fibrosis

Conclusion

Some drugs like amiodarone, antineoplastic, antimetabolic, biologically active agents may
precipitate toxicity acutely even with single dose so always suspect drug induced pulmonary toxicity in drugs which are known to cause it.

References


