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Case Report

Docetaxel induced interstitial pneumonitis: a case report

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ABSTRACT

Incidence of diffuse alveolar damage is rarely seen after administration of docetaxel used alone or in combination with other chemotherapeutic drugs in absence of radiation. Here we present one such patient who developed diffuse alveolar pneumonitis after administration of first dose of docetaxel and the patient responded to steroids rather than antibiotics and antifungal drugs confirming the cause. This rapid onset drug induced alveolar pneumonitis illustrates that no therapeutic intervention is without its intrinsic and unanticipated risks.

Keywords: Docetaxel, Alveolar pneumonitis, Toxicity

INTRODUCTION

Pulmonary toxicity associated with docetaxel based chemotherapy used as an adjuvant therapy in breast cancer patient is very uncommon. Clinical and radiologic features are nonspecific and diagnosis is made by exclusion. The rate of docetaxel induced pneumonitis depends on total dose, chemotherapy schedule and concomitant docetaxel treatment with gemcitabine and radiation.¹ Although the usual outcome is cure, it sometimes eventually progresses to pulmonary fibrosis despite steroid treatment. Hence this toxicity should be taken into account while planning treatment strategies.

CASE REPORT

A 67 years postmenopausal female was diagnosed as carcinoma left breast on biopsy from 2cm mass in upper outer quadrant in December 2012. Her pre-operative Chest X-ray & Bone scan was normal. She underwent left modified radical mastectomy. The histopathology report was infiltrating ductal carcinoma grade III with triple

negative disease. Patient was planned for adjuvant chemotherapy with Docetaxel Cyclophosphamide (750mg). First cycle was given on 13th January 2013. She tolerated the treatment well. On 15th January she developed shortness of breath and restlessness in the morning which got relieved on its own. In evening patient again developed shortness of breath and continuous, high grade fever with chills and rigors. There was no history of burning micturition, cough with expectoration or any complaints of erythematous rashes over the body. She was taken to a local hospital, where she was treated conservatively. Next day her condition deteriorated further as she complained of loose stools. On the same day patient developed chest pain which was acute in onset, non-radiating. ECG was normal. Chest Xray showed LRTI in bilateral lungs. On 18th January CECT chest was done which showed presence of parenchymal fibrous band in right lower lobe and small amount of pleural fluid bilaterally. Total leukocyte count was 7000/cu ml with eosinophilia. A diagnosis of pulmonary pneumonitis was made and she was started on antibiotics and anti-fungal coverage. Her pleural fluid was sent for cytology which showed inflammatory pathology with no evidence of malignancy. ABG analysis showed pH (7.409) with an increased pCO2 (53.1mmHg) and increased cHCO3 (32.8mmol/L). On 20th her total leukocyte count was 12700cell/cumm. Patient was kept in ICU for a week and then discharged with fever on and off and difficulty in breathing on exertion. On 08.02.13 patient again developed breathing difficulty along with cough with foul smelling yellow colored thick sputum and not associated with blood. Patient was again admitted to a local hospital. Clinically patient had grade 2 dyspnoea, vitals were stable and her O2 saturation was 90% without oxygen support, bilateral generalized crepts with bronchospasm. ECG was s/o minor ST changes in the inferior leads. Chest X-ray was s/o prominent broncho-vascular markings in both lungs. Patient was started on aggressive treatment with antibiotic and antifungal coverage but with no relief in her symptoms. Subsequently CECT Chest (on 23.03.13) was done which showed few tiny cyst in bilateral basal zones with minimal peri-bronchial thickening noted in bilateral basal zones and right middle lobe. The above investigations suggested that patient was suffering from chemotherapy induced diffuse alveolar damage. She was started on intravenous Solumedrol (1g). Following steroids patient had a dramatic response with near complete relief in her symptoms. At present she is comfortable with resolution of the pulmonary changes on her chest X-ray.



Figure 1: CT showing pneumonitis- pre steroid.



Figure 2: Complete regression- post steroid therapy.

DISCUSSION

Pulmonary toxicities associated with chemotherapeutic agents given in adjuvant settings in patients with breast cancer are rare in the absence of radiation treatment. The use of docetaxel and cyclophosphamide in combination has a favorable therapeutic index compared to anthracycline based regimens due to lower incidence of heart failure and leukemia; hence this combination is preferred more.² In adjuvant setting in lymph node negative breast cancer patients and when anthracyclines are contraindicated. Our patient was elderly and node negative and hence was started on this combination.

The largest published series of docetaxel related Interstitial pneumonitis is from Japan reporting an overall incidence of 4.6%.³ This study identified increased risk of interstitial pneumonitis in patients with preexisting interstitial changes. Our patient did not have any history of respiratory illness or any radiologic evidence of COPD or emphysema hence this was a rapid onset IP due to docetaxel.

In another study, an elder patient developed progressive dyspnoea and non-productive cough.⁴ She was found to have mild hypoxaemia and pleural and subpleural fibrotic changes which were not present on pretreatment scans. Biopsy confirmed subacute interstitial pneumonitis and patient responded well to steroids. Our patient also developed shortness of breath, cough, chest pain and fever on third day of docetaxel and had hypoxaemia and leucocytosis with eosinophilia. CT scan showed tiny cysts and peribronchial thickening confirming interstitial pneumonitis and she responded to steroids.

Many differential diagnosis have to be discussed before labeling the patient having docetaxel induced interstitial pneumonitis like lymphangitic carcinomatosis, bacterial infection, cardiogenic edema, radiation pneumonitis, pulmonary haemorrhage or allergy. Merad et al showed that in his two patients the exclusion of other diagnosis and absence of other concomitant drugs known to be associated with pulmonary toxicity and the improvement of clinical symptoms and pulmonary infiltrates following discontinuation of docetaxel supports the hypothesis that pulmonary lesions were induced by docetaxel. Similarly in our patient we ruled out other diagnosis and later patient improved with steroids and other supportive care, hence we confirmed that the pulmonary interstitial pneumonitis was docetaxel induced.

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