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Case Report

Herbicide Induced Interstitial Lung Disease in a Child: A Case Report

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Abstract: Drug induced interstitial lung disease is a common cause of acute and chronic lung disease which occurs mostly with cytotoxic and non-cytotoxic drugs. The clinical symptomatology is generally non specific and early diagnosis is important in the management of the patient. The mechanism of lung injury due to the drugs is by direct toxic action on lungs, hypersensitivity reactions and autoimmune mechanisms. The direct toxic action on lung tissue results in diffuse alveolar damage, interstitial pneumonia and organizing pneumonia. The diagnosis of drug induced lung disease depends on proper clinical suspicion, history of drug exposure and relevant radiological findings and exclusion of other causes of lung injury. Chest radiography and HRCT are most important diagnostic methods available to identify the disease. We report a case of drug induced interstitial lung disease in a 13 year old boy. The child developed acute respiratory illness-following ingestion of PARAQUAT (contact herbicide). The chest radiography and HRCT have shown the features of interstitial pneumonia. The child recovered following appropriate treatment.
Keywords: Drug Induced Interstitial Lung Disease (DILD), Paraquat, Interstitial pneumonia

INTRODUCTION

Drug induced interstitial lung disease (DILD) is mainly caused by direct toxic action on lung tissue, due to hypersensitivity reaction and by neural or humoral mechanism. The mechanism involved in cases of DILD includes direct injury to the pneumocytes or alveolar capillary endothelium and by T-cell mediated immunological reaction [1].

The clinical features of DILD include cough, dyspnoea, fever with history of drugs exposure and exclusion of other causes of lung disease [2]. The drug that commonly produce adverse reaction in the lungs include cytotoxic drugs like bleomycin, busulfan, cyclophosphamide and mitomycin etc. and non-cytotoxic drugs like amiodarone, bromocriptine, diphenyl-hydantoinetc [10]. HRCT is the main investigation to identify the various subtypes of ILD (interstitial lung disease), that develop even if the chest radiography was normal, but HRCT has an accuracy of only 45% in predicting the specific pattern [3]. KL-6 has been reported as sensitive marker of ILD [4, 5]. The mechanism of cytotoxic pulmonary injuries includes direct injury to pneumocytes or the capillary endothelium with subsequent release of cytokinins and recruitment of inflammatory cells.

The other mechanism involved in development of drug induced interstitial lung disease is by T-cell mediated immune reaction. Monoclonal antibodies also reported to produce drug induced interstitial lung disease. Non specific interstitial pneumonia (NSIP) is a most common type of drug induced interstitial lung disease other types of interstitial lung diseases include usual interstitial pneumonia (UIP), desquamative interstitial pneumonia (DIP), organizing pneumonia, eosinophilic pneumonia (EP), hypersensitivity pneumonia (HP) [1]. PARAQUAT (1,1-dimethyl- 4 , 4'–bipyridilium dichloride) is a lethal herbicide used in agriculture. The lethal dose LD50 in humans is approximately 3-5mg/kg which translated into liters as 10-15ml of 25% solution [6]. Ingestion of paraquat results in severe lung damage. Acute pulmonary edema occurs within a few hours of exposure and in some cases death results from respiratory failure and pulmonary fibrosis. The primary mechanism is associated with generation of free radicles and oxidative damage to lung tissue [7]. The patients present with cough, dyspnoea within 2-4 days of Paraquat ingestion. Many studies are reported on the toxic complications of Paraquat ingestion in the literature. Sequential Radiological and functional pulmonary changes in patients with Paraquat intoxication has been reported [8]. Xiao-lixu*et al.* [9] in their study on Paraquat poisoning in rats found the development of pulmonary fibrosis at different stages.

CASE REPORT

A13 year's old mentally deranged boy was brought to hospital by his parents with history of ingestion of 10ml of Paraquat (24%) solution. Gastric Lavage was done immediately; there was no other symptom of toxicity. However after 2 days of Paraquat ingestion the child developed progressive cough, shortness of breath and discolouration of tongue which resulted in respiratory distress for which he was ventilated. On clinical examination the child has bilateral crepitations in the lungs with broncho vesicular breath sounds. CNS and CVS examination was normal. SPO2 was 65% without oxygen and 95% with 5 liters of oxygen. Respiratory rate was 62/min. Routine laboratory investigations and serum electrolytes were within normal limits. ABG was consistently PO2<40 mgm/hg. Chest radiograph showed bilateral extensive reticulonodular opacities. There was no pleural effusion or hilaradenopathy. Heart size was normal (Fig.1).

The high resolution computed tomography (HRCT) findings – showed extensive honey combing and cystic spaces, predominantly in right upper lobe with traction bronchiolectasis. There are patchy areas of ground glass opacities with consolidation were noted in both lower lobes. Evidence of intralobular interstitial and subpleuralseptal thickening was noted in both lower lobes. The above findings are correlating with findings of both usual interstial pneumonia (UIP) and Non specific interstitial pneumonia (NSIP) (Fig. 2&3).

The child was treated with Nebulization, IV antibiotics-piptaz, Levofloxacin, Linezolide, Cefuroxime, IV Decadron for 2 weeks. The child has improved considerably and follow up Radiography shows significant resolution of lung lesions (Fig. 4).

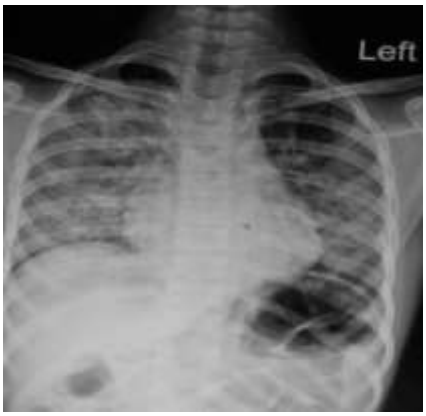

Fig. 1: Chest radiograph: Bilateral extensive reticulonodular opacities

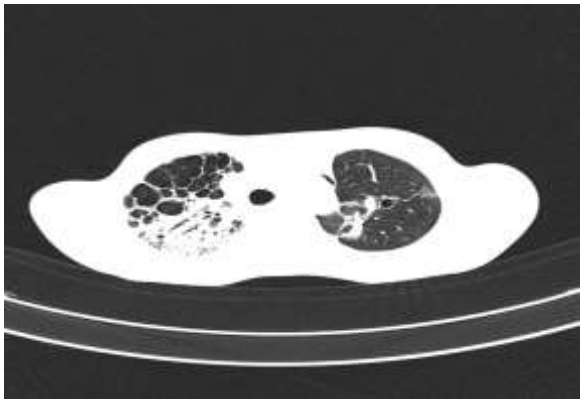

Fig. 2: HRCT Chest: honey combing, cystic spaces, predominantly in Right upper lobe with traction bronchiolectasis

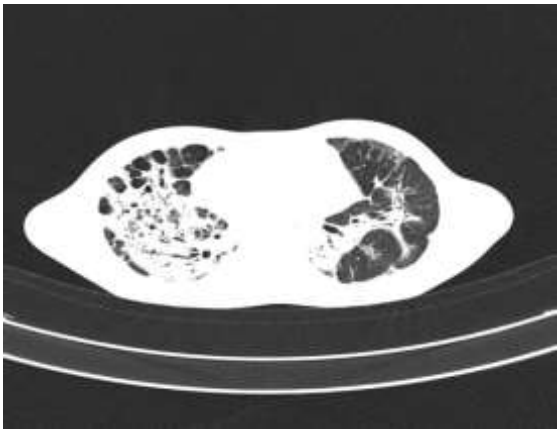

Fig. 3: HRCT Chest: Ground glass opacities with consolidation noted in bilateral lower lobes

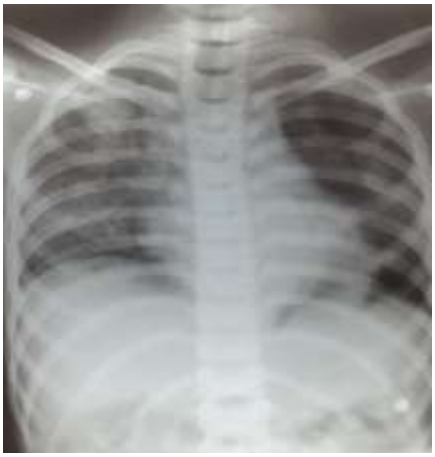

Fig. 4: Chest Radiograph: Significant resolution of parenchymal opacities

RESULTS

	Xiao-Lixu*et al*. [9]	**Inwohhuh*et al*. [8]**	**IM*et al*. (11)**	**Present Case**
GGO	3-7 days	7 days	---	7 days
Consolidation	---	2 weeks	7 days	10 days
Nodules	7 days	-------	---	10 days
Interstitial Thickening	14 days	14-28 days	----	27 days
Honey Combing	14-28 days	14-28 days	1 month	27 days

DISCUSSION

The diagnosis of drug induced interstitial lung disease is mainly by strong clinical suspicion and by exclusion of other causes. Since pesticides like Paraquat and Diquat which are highly toxic, any kind of exposure to these agents is to be analyzed carefully. Early detection of drug induced lung disease is important in preventing permanent lung damage and in the reversal of the disease process.

The drug induced interstitial lung disease can be either interstitial pneumonia or organizing pneumonia. In the present case ingestion of Paraquat has resulted in changes suggesting both Non specific interstitial pneumonia (NSIP) & usual interstitial pneumonia (UIP), some of the changes result in end stage lung disease. In some cases lung biopsy helps us to diagnose drug induced interstitial lung disease and also to exclude other causes of interstitial lung disease.

CONCLUSION

A relatively healthy child developed severe respiratory complication after ingestion of a toxic herbicide. Though the lung changes are permanent in the form of honeycombing, the child has improved considerably with appropriate treatment due to the reversal of some of the lung changes. Besides cytotoxic and non-cytotoxic drugs, ingestion of herbicides can produce severe lung toxicity resulting in permanent end stage lung damage.

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