DRUG INDUCED LUNG INJURY

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Objective:

INTRODUCTION

Although rare, nitrofurantoin is one of the commonest causes of drug-induced pulmonary disease. The acute form of the disease is more common than chronic. It is characterized by cough, dyspnoea, fever, rash and arthralgia. The CXR may reveal reticular shadowing or alveolar infiltrates. Eosinophilia in peripheral blood or in bronchoalveolar fluid may be present. A restrictive pattern on spirometry and reduced lung diffusion capacity are sometimes present.

CASE REPORT

A 50-year-old woman was hospitalized because of acute-onset cough, fever, dyspnea and respiratory failure. The patient was an ex-smoker and reported a history of allergy to house dust mite and weed pollen. She was taking nitrofurantoin approximately once a year because of recurrent urinary tract infections. She started taking a course of nitrofurantoin 8 days before admission.

On physical examination, a mild maculopapular rash was noted on patient's hands and trunk. On lung auscultation, wheezing was heard. Peripheral oxygen saturation was 88% on room air. RT-PCR test for SARS-CoV-2 was negative. The chest X-ray revealed bilateral interstitial reticular pattern. Laboratory findings were remarkable for hypereosinophilia (absolute eosinophil count 648×10^9/L, relative 45,4 %), and CRP 35 mg/L. The thoracic CT scan showed irregular consolidations of lung parenchyma in the left lower lobe and lingula, bilateral mosaic attenuation of the lung parenchyma and hypodense zones of air-trapping (Figure 1). Serology testing and stool samples for parasites were negative. Bone marrow aspiration showed 30% eosinophils. Bronchoscopy was performed, and examination of BAL fluid revealed 15% eosinophils. Lung function test results revealed mild restriction (FVC 78%, FEV1 80%, TLC 79%), and DLCO was 80%.

Nitrofurantion, the suspected cause, was ceased immediately upon admission. Due to the persistent

hypoxemia and peripheral blood eosinophilia, methylprednisolone at a dose of 32 mg was started, with gradual dose tapering. At a follow-up visit one month later, the patient reported no respiratory symptoms, eosinophil count was normal ($0,16x10^9$/L), oher laboratory tests were in the reference range, and a CT scan showed significant regression of lung parenchymal lesions (Figure 2).

CONCLUSION

In nitrofurantoin-induced lung disease, the most important step in treatment is the cessation of the causative drug. In more severe cases, corticosteroid therapy is also indicated. Thorough evaluation of the patient with hypereosinophilia and suspected drug-induced lung injury is required. Acute lung reactions to nitrofurantoin do not predispose to a chronic disease.