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Case Report
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Rifampicin Induced Erythroderma: A Case Report

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Abstract

Rifampicin is an antibiotic used to treat a several types of bacterial infections especially tuberculosis but rifampicin associated erythroderma is uncommon. Here we report the case of erythroderma in a patient, who was diagnosed with left pleural effusion and he had been treated with rifampicin.

Keywords: Rifampicin; Erythroderma; Exfoliative dermatitis

Introduction

Exfoliative dermatitis also known as erythroderma is an uncommon but serious skin disorder which results in generalized scaling eruption of the skin. It is usually drug induced, idiopathic, or secondary to underlying cutaneous or systemic disease. Theoretically, any drug may cause exfoliative dermatitis. Among antitubercular drugs, exfoliative dermatitis has been reported with rifampicin, isoniazid, ethambutol, pyrazinamide, streptomycin, PAS either singly or combination of two drugs in some case reports [1-3]. It usually presents with six to eight weeks of initiation of antitubercular treatment. Early recognition, prompt withdrawal of antitubercular therapy and institution of steroids, if reaction is severe, are cornerstones of its management.

Here we report a rare case of pulmonary tuberculosis with erythroderma caused by first line antitubercular drug rifampicin (Figure 1). The rifamycin is a macrocyclic antibiotics produced by Amycolatopsis mediterranei. Rifampicin inhibits the growth of most gram-positive bacteria as well as many gram-negative microorganisms such as *Escherichia coli*, *Pseudomonas*, indole-positive and indole-negative Proteus, and *Klebsiella*. Rifampicin is very active against *Staphylococcus aureus* and coagulate-negative staphylococci.

Case Report

A 59 year old male patient was diagnosed with left pleural effusion, when he was evaluated for long term dry cough. He started his treatment with first line anti tubercular drugs (Isoniazid, Rifampicin, and Pyrazinamide). After one month of the treatment he developed generalized peeling of skin with itching over hands initially followed by legs and trunk of sudden onset and progressive. He had consulted a dermatologist and was treated with corticosteroids, liquid paraffin for 20 days. Symptoms were not resolved and then he was admitted in our hospital, the physical examination revealed that the nail did not shown any change and there were complaints of generalized scaling eruption involving the scalp, trunk, palms and other extremities. After the general and physical examination it was found that there was no lymphadenopathy, and no signs of pleural effusion. Pulse rate was 82 beats/min and BP 130/90 mmHg.

Laboratory investigations done revealed that hemoglobin 12.5 g, total leucocytes count 7,100/cumm, polymorphs 73%, lymphocytes 23%, eosinophils 4%, RBC 4.2 millions/cumm, liver function tests were within normal limits. Doctor confirmed his diagnosis that the erythroderma was only due to antitubercular treatment as he was not taking any other medicines. Pyrazinamide was then withheld due to more cutaneous side effects and started with 2 combinations of antitubercular drugs (Isoniazid and Rifampicin). Erythroderma was treated with corticosteroids (Dexamethasone) and Antihistaminic (Pheniramine maleate). Isoniazid was withheld since the patient did not show any recovery from the generalized peeling of skin over palm, extremities, scalp and lower leg edema and the therapy was continued with rifampicin alone. Even then the patient did not recover from his symptoms of erythroderma and hence the therapy was switched to Streptomycin (antitubercular drug), 0.75 gm and the reaction were treated with dexomethasone hydroxyzine, pheniramine maleate, relysal ointment along with general supportive treatment. After withdrawing rifampicin the patient showed recovery from erythroderma and therefore it was suspected that the erythroderma was due to rifampicin.

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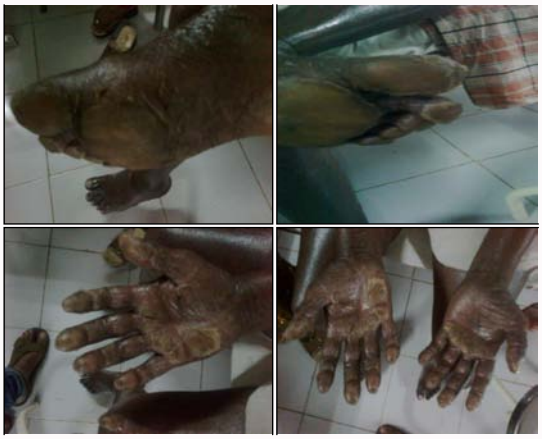

Figure 1: Pulmonary tuberculosis with erythroderma caused by first line antitubercular drug rifampicin.

Discussion

Erythroderma is the result of a dramatic increase in the epidermal turnover rate. In patients with this disorder, the mitotic rate and the absolute number of terminative skin cells are higher than normal. Moreover, the time necessary for cells to mature and travel through the epidermis is decreased. This compressed maturation process results in an overall greater loss of epidermal material, which is manifested clinically as severe scaling and shedding. Normal epidermis undergoes some exfoliation every day, but the scales that are lost contain little, if any, important viable material, such as nucleic acids, soluble proteins and amino acids. In erythroderma, however, protein and folate losses may be high [4,5].

Rifampicin generally is well tolerated. When given in usual doses, fewer than 4% of patients with tuberculosis have significant adverse reactions; the most common are rash (0.8%), fever (0.5%), and nausea and vomiting (1.5%). Rarely, hepatitis and deaths due to liver failure have been observed in patients who received other hepatotoxic agents in addition to rifampin, or who had preexisting liver disease. Hepatitis from rifampicin rarely occurs in patients with normal hepatic function; likewise, the combination of isoniazid and rifampicin appears generally safe in such patients [6,7]. Severe hypersensitivity reactions to standard antitubercular drugs are rare but they may be fatal. They usually commence after four to six weeks of therapy and must be recognized early to reduce associated morbidity and mortality.

In this case; the patient developed erythroderma reaction which is suggestive of rifampicin induced hypersensitive reaction and he showed improvement after the withdrawal of rifampicin. If the cutaneous reaction was not serious, desensitization could have been attempted, but in the case of serious reactions, reinstitution of drug cannot be attempted. So in our case he was put on a modified regimen of streptomycin and he tolerated well during continuation phase.

Conclusion

The present report concludes that the rifampicin causes hypersensitivity reaction like erythroderma. This report could alert the clinician to consider rifampicin as one of the possible agents in the causation of erythroderma and it is always preferable to confirm the cause of cutaneous reactions by clinical trials ensuring all safety measures, preferably in hospitals. Early recognition of the reaction and cessation of the causative drug can stop the progression of skin rashes and normalize the patient.

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