

Erythroderma caused by anti tuberculoid drug in pulmonary tuberculosis and HIV infected patient: A case report

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Abstract Erythroderma or exfoliative dermatitis is a cutaneous disease characterized by erythema and scaling affecting more than 90% of body surface area. This can be resulted from malignancy, drug eruption, idiopathic and underlying disease like psoriasis, atopic dermatitis and seborrheic dermatitis. Patients with HIV/AIDS are prone to drug eruption. A 50 year old man presented to our outpatient clinic with exfoliative scaly skin all over his body after consuming anti-TB therapy. He was diagnosed with lung tuberculosis (TB), human immunodeficiency virus (HIV) infection stage 3. Histopathological examination revealed mild acanthosis and spongiosis in the epidermis, dropping melanin, lymphocyte, histiocyte and perivascular polymorphonuclear (PMN) leukocytes in the dermis. We treated this patient by discontinuing his anti-TB drug, the suspected causative drug, hydrating with intravenous fluid, placing him in warm room and covering him with thin blanket. He was treated with methylprednisolone IV 62.5 mg/24 hours for 5 days, cetirizine tablet 10 mg once daily, topical vaselin applicative twice per day, gentamicin ointments applied on the erosive area twice daily and extra egg white diet 1700 kcal. Our patient's condition improved after discontinuing anti-TB drugs and receiving systemic corticosteroid as well as topical emollient. Erythroderma due to the first line antitubercular drug is uncommon. It can lead to a serious potentially life threatening problem. Therefore early recognition, early diagnosis and early treatment determine the outcome.

Key words

Erythroderma, drug eruption, pulmonary tuberculosis, antituberculosis drug, human immunodeficiency virus.

Introduction

Erythroderma or exfoliative dermatitis is a cutaneous disease characterized by erythema and scaling affecting more than 90% of body surface area. This can be resulted from malignancy, drug eruption, idiopathic and underlying disease like psoriasis, atopic dermatitis and seborrheic dermatitis.¹ Erythroderma is one of emergent cutaneous cases as it can develop to systemic complication such as electrolyte imbalance, thermoregulation, hypoalbuminemia and sepsis.²

Human immunodeficiency virus (HIV) causes decreased body immune system, known as acquired immunodeficiency syndrome (AIDS). Patients with HIV/AIDS are prone to drug eruption.³ Some drug which may result in drug eruption are trimethopim-sulfamethoxazole, anti tuberculosis drugs and antiretroviral drugs.⁴

Erythroderma can be managed by administering nutrition, correcting fluid as well as electrolyte imbalance, preventing hypothermia and secondary infection therapy.⁵ In general, drug-induced erythroderma is treated with cessation of the causative drug.⁶ This case report describes an uncommon case of antituberculosis drug-induced erythroderma in a 50 year old man with HIV.

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Case Report

A 50 year old man presented to our outpatient clinic with exfoliative scaly skin all over his body started a week prior to admission. He reported that 5 months ago and was diagnosed with lung tuberculosis (TB) as well as human immunodeficiency virus (HIV) infection stage 3. He received anti-TB drug of rifampicin®, isoniazid (I), pyrazinamide (Z) and ethambutol (E). In his second month of anti-TB therapy, reddish patches appeared, along with scaling of the skin throughout his body so that he required hospital admission with diagnosis of erythroderma. The lesion improved and he was discharged. He continued taking his anti-TB drugs for 3 weeks then the same symptoms occurred so that he was hospitalized again and diagnosed with erythroderma presumably due to anti-TB drugs. He received intravenous injections of methylprednisolone 125 mg/24 hours, ranitidine 50 mg/ 12 hours and cetirizine

tablet 10 mg/24 hours. However the symptoms persisted and was referred to our hospital.

On admission to our hospital, he looked weak and undernourished. The physical examination findings were only significant for fever and lower extremity edema. Dermatological status obtained generalized hyperpigmented patches with scaling and crusted lesion (**Figure 1**). Laboratory examination demonstrated hemoglobin 9.9 g/dl, hematocrit 29 g/dl, PT 15.8 seconds, APTT 41.9 seconds, AST 41 U/L, albumin 2.1 g/dl, blood calcium 130 mmol/L, HIV reactive, absolute CD4 163 cell/ μ l and CD4 3.19%.

Histopathological examination revealed mild acanthosis and spongiosis in the epidermis, dropping melanin, lymphocyte, histiocyte and perivascular polymorphonuclear (PMN) leukocytes in the dermis. These features resemble drug induced erythroderma (**Figure 2**).



Figure 1 Dermatological status revealed generalized hyperpigmented patches with scaling and crusted lesions.

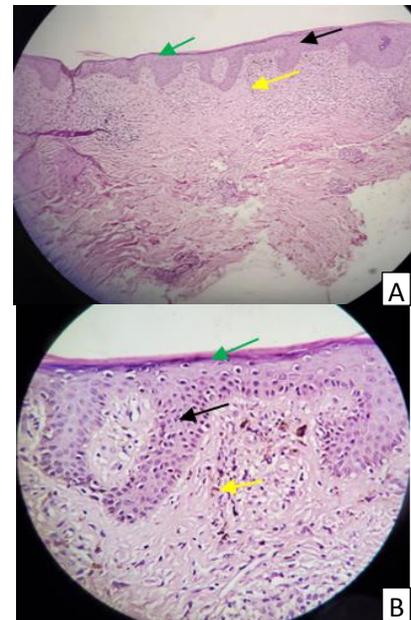


Figure 2 Histopathological examination revealed mild acanthosis and spongiosis in the epidermis, dropping melanin, lymphocyte, histiocyte and perivascular polymorphonuclear (PMN) leukocytes in the dermis A. (HE-100x) B. (HE-400x).

Based on the aforementioned finding we diagnosed this patient with exfoliative dermatitis to drug eruption.

This patient was managed by We treated this patient by discontinuing the suspected anti-TB drug causative drug, hydrating him with intravenous fluid, placing him in warm room and covering him with thin blanket. He was treated with methylprednisolone IV 62.5 mg/24 hours for 5 days, cetirizine tablet 10 mg once daily, topical vaseline, gentamicin ointment applied on the erosive area twice daily and extra egg white diet 1700 kcal.

This patient also received therapy from internist in the forms of albumin infusion 25% in 100 ml, potassium chloride 25 mEq in 500 ml normal saline, vitamin D3 1000 mg/ 24 hours and folic acid tablet 400 mcg/ 24 hours. We tapered off the intravenous methylprednisone to 31.25 mg/24 hours for 2 days then 16 mg/24 hours. After 7 days of the therapy the lesion improved, the skin became normal.

Discussion

Erythroderma is common in males, its ratio to females is 3:1. Its incidence peaks between five and six decades.⁷ Erythroderma occurring in HIV patients is mostly due to drug eruption. Anti-TB drugs may also cause cutaneous drug eruption. A study by Siripassorna *et al.* reported that first line anti-TB drugs such as pyrazinamide, rifampicin, isoniazide and ethambutol have the incidence of drug eruption by 2-5%, 1.2-2%, 1-1.2% and 0.3-1.48%, respectively.⁸ Our patient had been receiving rifampicin, isoniazid, pyrazinamide and ethambutol as anti-TB drugs before he experienced generalized exfoliative dermatitis. Although he had been diagnosed with HIV along with-TB, he has not received any medication for

HIV. Therefore we highly suspected that his condition was due to anti-TB drug reaction.

Clinical manifestation of erythroderma include erythema and scaling affecting more than 90% of body surface area. Erythroderma due to anticonvulsants, antibiotics and allopurinol develops between 2 and 5 weeks after consuming the drugs. Its typical systemic clinical features are tachycardia, fever, lymphadenopathy, hepatosplenomegaly and peripheral edema.⁹ In our case we found erythematous patches and scaling in second month of anti-TB therapy.

Histopathologically erythroderma resulted from drug demonstrated orthokeratosis, degenerative basal cells, necrotic keratinocytes, apoptotic cells, spongiosis and eosinophilic superficial infiltrates in the dermis.

In our patient the histopathological examination demonstrated acanthosis and spongiosis in the epidermal layer as well as dropping melanin, lymphocytes, histiocytes and perivascular polymorpho nuclear leukocytes in the dermal layer. All these features support the diagnosis of drug induced erythroderma.

Erythroderma due to drug eruption is managed by discontinuing the consumption of the causative drug, correcting the fluid as well as electrolyte, preventing hypothermia by placing the patient in a room with the temperature of 30-32°C, treating the secondary infection and providing high calorie high protein diet.^{5,10} Initial dose of 1-2 mg/kg body weight of systemic corticosteroid is administered continued with 0.5 mg/kg body weight until the lesion improves. In order to avoid long term effect, topical emollient and low potent topical corticosteroid are applied.¹ Our patients condition improved after discontinuing anti -TB

drugs and receiving systemic corticosteroid as well as topical emollient.

The complications of erythroderma include metabolic changes, heart failure, acute respiratory failure, and secondary infection.¹¹ Generalized skin exfoliation leads to protein loss which can result in hypoalbuminemia, thermoregulatory problem and extremity edema.¹¹ Our patient experienced hypoalbuminemia along with lower extremity edema on day 5 of follow up. Regular observation is required to monitor drug cross reactivity.

Conclusion

Erythroderma due to the first line anti-TB drug is uncommon. It can lead to a serious potentially life threatening problem. Therefore early recognition, early diagnosis and early treatment determine the outcome. In our case it is highly suspected that the causative agent is anti-TB drug. The condition improved gradually after 5 days of treatment and at 2 months of follow up his clinical condition improved significantly.

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