EXFOLIATIVE DERMATITIS DUE TO PARACETAMOL

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A patient having borderline lepormatous leprosy developed exfoliative dermatitis while on treatment. Provocation tests done 2 months after subsidence of the dermatitis proved erythroderma to be on account of paracetamol.

Key words: Exfoliative dermatitis, Erythroderma, Paracetamol.

It is estimated that about 5% of patients get some sort of an adverse effect attributed to the drugs.¹ Drugs like ampicillin and penicillin are notorious for producing cutaneous side effects,¹ while with other drugs like aspirin, the frequency of major side effects is not very high taking into account its large consumption,²'³ though urticaria due to aspirin is quite common⁴'⁵. Acetaminophen (paracetamol) a substitute of aspirin, is even less known to produce adverse cutaneous effects, though fixed drug eruption due to this drug has been reported.⁶ Systemic side effects with this drug are more frequent.⁶ A case of exfoliative dermatitis due to paracetamol, seen at this hospital is reported.

Case Report

A 25-year-old male having borderline lepromatous leprosy, receiving DDS, thiacetazone and INH reported with fever and morbilliform rash of one week duration. He had no other clinical abnormality. Investigations revealed slightly raised SGOT and SGPT. Urine analysis showed presence of bile salts and urobilinogen.

All the drugs which the patient had been taking, were stopped and he was put on 4 tablets of betamethasone (equivalent to 20 mg prednisolone) per day. The same evening in the ward, the patient had fever for which he was given one paracetamol tablet. By next morning, patient had developed generalized crythroderma. He had marked redness and oedema of entire skin

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with sheets of thick scales which were most marked on his face, back and flexural folds. Betamethasone dose was increased to 16 tablets per day. He was also given antacids (Mucain gel) 2 spoons thrice a day in between milk feeds. The patient made a quick recovery, the erythema and exfoliation subsided in 5 days. Corticosteroids were gradually reduced and he was discharged 2 weeks later on 2 tablets of betamethasone per day. No antileprosy drug was prescribed and the patient was told not to take any other drug.

Two months later, the patient was admitted again to find out the cause of erythroderma. For this, provocation tests^{8'9} were undertaken. Initially INH, which was least suspected to be the cause was introduced. The patient was given 50 mg of INH. This resulted in no untoward side effect over the next 24 hours. During the next four days, INH dose was gradually increased to 300 mg per day without any problem. There-after the patient was given 25 mg DDS. This too was well tolerated by the patient and was continued as such. When the patient had been on DDS 25 mg for 3 days, he had fever with chills and rigors at night. Pending investigation for the cause of fever, the patient was given one table of paracetamol. Within 4 hours of paracetamol administration, the patient had swelling of the face. By next morning, he had a hands and feet. full blown exfoliative dermatitis. As before, all the drugs were discontinued and the patient was put on corticosteroids. He recovered uneventfully during the next one week. Corticosteroids were gradually tapered off. He was subsequently put on DDS, INH and thiacetazone which he tolerated well.

Comments

Cutaneous adverse effects to drugs occur in 1-2% of the cases. 10'11 These reactions may be mild to severe and in some cases, if treatment is delayed, death may ensue. Exfoliative dermatitis, an uncommon side effect, is one of the serious reaction patterns to drugs and has been reported more often due to the drugs like thiacetazone9'10, INH9, streptomycin9, ethambutol10, chloroquine9 metallic compounds, antiepileptic drugs12 and DDS13. No such side effect to the best of our knowledge has been reported with paracetamol.

Paracetamol as the cause of erythroderma in this case, was not suspected initially. patient had come with morbilliform rash and developed exfoliative reaction after steroids and paracetamol were administered. This change of morbilliform rash to erythroderma was considered to be the natural course of development of the reaction. In fact it was considered that the reaction was on account of any of the drugs which the patient had taken before hospitalization. These drugs were DDS, INH and thiacetazone. To us, probability of thiacetazone causing erythroderma appeared more than that of DDS which in turns was more likely than that of INH. Therefore, provocation test as detailed by Pasricha¹⁴ were instituted first with INH and subsequently with DDS with no untowards reaction. When for fever, a tablet of paracetamol was given (the patient had been tolerating DDS well), sudden development of erythroderma confirmed paracetamol to be the offending agent.

The occurrence of initial morbilliform rash, with which the patient presented, is difficult to explain. It is possible that the patient may have taken some preparation containing paracetamol at home for fever.

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