A 43-year old female from rural area was admitted in a tertiary care hospital complaining of sudden development of rash all over the body since last four days. Initially rash developed over abdomen and chest followed by spreading to rest of the portion of the body including palm and soles. Skin rashes were initially erythematous and maculopapular and extremely itchy followed by painful blister formation, affecting mucous membrane of mouth [Table/Fig-1], anal canal, and vaginal area also. There was denudation with raw ulceration at places over face following rupture of blisters during hospital stay [Table/Fig-1,2]. Patient was unable to eat due oropharyngial painful ulceration and crusting over lips. Then purpuric macules or blisters appeared over trunk, abdomen and back [Table/Fig-3]. There was no history of similar sort of episode in the past.

On examination, the patient was alert and appeared dehydrated. Her body temperature was 38°C, blood pressure was 114/70 mm Hg, pulse rate was 96 beats per minute, and respiratory rate was 20 breaths per minute. Nikolsky’s sign was positive. The body surface area involvement of the patient at the time of presentation was 9%. Patient was suffering from multiple joint pains since last one month for which she was taking allopurinol 100 mg thrice daily for last ten days prior to development of skin rash. Patient’s body temperature was also increased to 39.4°C intermittently which diminished on paracetamol administration following three days after admission.

The patient’s complete blood count was within normal limits except slight decreased haemoglobin concentration 10.4 g/dL. Erythrocyte sedimentation rate (ESR) was normal. Her C-reactive protein was increased to 7.6 mg/dL. The serum levels of total protein, albumin, blood urea nitrogen, creatinine and elevated random blood glucose were within normal limits. Serum total IgE and ECG were normal. The sodium and potassium levels were slightly decreased. There was no evidence of eosinophilia and levels of liver enzymes and serum bilirubin were within the normal range. Serum total IgE and ECG were normal. The sodium and potassium levels were slightly decreased. There was no evidence of eosinophilia and levels of liver enzymes and serum bilirubin were within the normal range. Blood culture was negative. The serological titers for HSV was within normal limits, exclude the possibilities of HSV infection in etiopathogenesis in this particular case. Skin biopsy was advised in order to rule out differential diagnoses such as autoimmune blistering diseases like pemphigus vulgaris, bullous fixed drug eruption, erythema multiforme acute generalised exanthematic pustulosis and TEN.

Treatment was started conservatively with oral antihistaminic 25 mg, antipyretics, intravenous dexamethsone and intravenous fluid therapy. The patient was started antibiotic inj. cefotaxime 1gm i.v. to reduce the risk of secondary infection, skin lesions were treated with topical therapy and local anaesthetic creams. Chlorhexidine rinse was given to maintain good oral hygiene and white-soft paraffin was applied on the lips to relieve pain. The lesions gradually diminished and healed up in about 15 days [Table/Fig-4] and resolved with mild hypo/ or hyperpigmented spots. The causality assessment as per the Naranjo algorithm [1] revealed the ADR to be Probable (Naranjo score 7). Assessment of causality by using the algorithm of drug causality for epidermal necrolysis (ALDEN) [2] was ‘very probable’.

Severity-of-illness and prognosis was assessed by using the SCORTEN criteria. We did not seek to test the effects of re-challenge in our patient. Patient and relatives was properly counselled.
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PARTICULARS OF CONTRIBUTORS:

1. Post Graduate Trainee, Department of Clinical & Experimental Pharmacology, Calcutta School of Tropical Medicine, Kolkata, West Bengal, India.
2. Post Doctoral Trainee, Department of Clinical & Experimental Pharmacology, Calcutta School of Tropical Medicine, Kolkata, West Bengal, India.
3. Post Doctoral Trainee, Department of Clinical & Experimental Pharmacology, Calcutta School of Tropical Medicine, Kolkata, West Bengal, India.
4. Professor & Head, Department of Tropical Medicine, Calcutta School of Tropical Medicine, Kolkata, West Bengal, India.
5. Professor & Head, Department of Clinical & Experimental Pharmacology, Calcutta School of Tropical Medicine, Kolkata, West Bengal, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sukanta Sen,
Post Doctoral Trainee, Department of Clinical & Experimental Pharmacology,
Calcutta School of Tropical Medicine, 108 C.R. Avenue, Kolkata, West Bengal, India.
E-mail: drsukant@gmail.com

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