Outcome of Stevens Johnson syndrome and toxic epidermal necrolysis treated with corticosteroids

Sir,

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are drug-induced or idiopathic reaction patterns characterized by skin tenderness, along with erythema of the skin, followed by extensive cutaneous and mucosal sloughing. They are life-threatening due to multisystem involvement and mortality ranges from 25 – 70%.[1] SJS includes cases with less than 10% epidermal detachment, mucosal lesions, and widespread purpuric lesions; SJS / TEN overlap when the epidermal detachment is between 10 and 30%; mucosal lesions, widespread purpuric lesions, and TEN when the epidermal detachment is more than 30%, and mucosal lesions and widespread purpuric lesions are present.[2] Early intervention with corticosteroids controls inflammation,[2] as corticosteroids are potent agents that target several intracellular processes, to modify almost all components of inflammatory and immune responses, hence, some favor early use of corticosteroids. Some studies suggest that systemic steroids adversely affect the outcome by increasing the risk of septicemia and gastrointestinal bleeding.[3-6]

A retrospective analysis of the records of patient's admitted in the dermatology ward with SJS,SJS-TEN overlap, and TEN, between 1997 and 2005, was performed. A detailed study of case records regarding clinical presentation, investigations, treatments, treatment outcome, and provoking factor was done.

The total number of patients admitted with SJS was 10 (41.6%), with SJS-TEN overlap was eight (33.3), and with TEN was six (23%). Mean age of the patients was 26.4 years. The mean percentage of body surface area involved was 35.5%. The patients reported to the hospital within 1.9 days of appearance of the lesions. Prodromal signs were seen in all the patients.

The drugs implicated in the decreasing order of frequency were phenytoin 8 (33.3%) carbamazapine 5 (20.6%), sulfonamides 5 (20.6%), amoxicillin 3 (12.5%), ibuprofen 2 (8.33%), and ciprofloxacin 1 (4.16%). Viral infection was seen in one case [Table 1].

The offending drug was stopped immediately. The patients were bathed daily and paraffin gauze was applied over the raw body surface area. Antibiotics, ceftriaxone, and gentamycin were given prophylactically. Twenty-two patients were started on oral or intravenous (IV) corticosteroids with doses ranging from 1 to 3 mg / kg / body weight. Oral pednisolone was given in patients who could take it orally. Dexamethasone was given IV. Corticosteroids were tapered according to the response seen. The patients, received corticosteroids for 14 – 30 days (mean 15 days). Two patients did not receive corticosteroids as one had sepsis and in the other the

Table 1: SCORTEN, drugs implicated and basic disease of patients

SCORTEN	Implicated drug	Basic disease
4	Phenytoin	Head injury
3	Amoxicillin	Fever
4	Carbamazepine	Convulsions
4	Ibuprofen	Fever
4	Co-trimoxazole	UTI*
5	Phenytoin	Convulsions
4	-	Chicken-pox
6	Phenytoin	Convulsions
3	Amoxicillin	Fever
3	Ibuprofen	Joint pain
5	Ciprofloxacin	URTI**
4	Co-trimoxazole	URTI**
5	Carbamazepine	Convulsions
6	Phenytoin, carbamazepine	Convulsions
7	Phenytoin	Head-injury
4	Co-trimoxazole	UTI*
4	Amoxicillin	URTI**
5	Carbamazepine	Convulsions
4	Phenytoin	Head injury
3	Co-trimoxazole	Fever
4	Carbamazepine	Convulsions
5	Co-trimoxazole	Fever
5	Phenytoin	Convulsions
5	Phenytoin	Convulsions

*Urinary tract infection, **Upper respiratory tract infection

etiology was of viral origin, the patient had chicken pox. A Tzanck smear was done, which showed multiple multinucleated giant cells, and serum IgM was raised above normal. Strict monitoring of the patients' vitals was done daily. Sepsis screening was done at baseline and twice weekly. All the patients recovered. The average period of stay in hospital was 19.4 days (range 7 – 41 days). All patients were kept in isolation.

The principle of symptomatic treatment are the same as that for burns and include fluid replacement, nutritional support, and antimicrobial therapy.[3] Early reporting by our patients, within 1.9 days of appearance of lesions, and the early use of corticosteroids may have favored a better prognosis. Patterson[7] reported the effectiveness of early steroid administration in the Stevens-Johnson syndrome in 41 cases.

Lesser total body surface area involvement showed a better survival rate as seen in other studies.[8] Most of our patients, 75% (18 cases), had 30% or less than 30% of body surface area involvement. Younger patients had better prognosis as seen from other studies.[9-11] The majority of our patients were between 18 and 30 years. From our analysis we have seen that steroids, when given early, preferably within 72 hours, in younger patients, with lesser body surface area involvement, without any comorbidity, and under very strict supervision, preferably in well-equipped centers, prove beneficial to the patients with SJS / TEN.

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