Toxic Epidermal Necrolysis in Patients Receiving Glucocorticosteroids

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Despite the lack of evidence of an immunologically mediated mechanism for Toxic Epidermal Necrolysis (TEN), glucocorticosteroids are frequently used for the treatment of patients afflicted with TEN. We investigated the data of 216 patients with TEN for therapy with glucocorticosteroids prior to the beginning of TEN. The data had been collected between 1984 and 1985 during the epidemiological study on severe skin reactions in West-Germany spanning the years 1981–85. 11/216 (5%) patients could be traced, who had been treated with glucocorticosteroids for at least a week prior to the first dermatological sign of TEN. Glucocorticosteroids had been administered predominantly for neurological or neurosurgical conditions. The period of treatment ranged from 1 week to several months. Doses ranging between 7.5 and 325 mg prednisolone/per day were found. There were no major differences between patients with or without steroid therapy with regard to sex, age and lethality. The observation of TEN in patients receiving therapy with glucocorticosteroids raises questions about the usefulness of treating patients with TEN with glucocorticosteroids.

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Toxic Epidermal Necrolysis (TEN) is characterized by a painful, generalized sheet-like loss of epidermis resembling a scald, mucosal ulcerations and fever. The lethality rate ranged between 30 and 34% (1–5). An association with previous medication can be established in the majority of cases, rising to 89% (1, 3, 6).

Although TEN is still a disease of unknown pathogenesis, glucocorticosteroids, which are known for their anti-inflammatory and immunosuppressive potencies (7), are frequently recommended for the treatment of TEN (3). In this study the data on patients with TEN, which had been collected during the retrospective study on severe skin reactions in West-Germany (4,5), was scrutinized to identify patients treated with glucocorticosteroids prior to the occurrence of TEN.

MATERIAL AND METHODS

The data of 216 patients with TEN were collected during an epidemiological study on the severe skin reactions "Toxic Epidermal Necrolysis and Stevens-Johnson Syndrome in the Federal Republic of Germany from 1981–85" (4,5).

TEN was defined according to the criteria of an international group of dermatologists published elsewhere (5). To qualify for inclusion in the investigation, glucocorticosteroids should have been given on a daily basis for at least 3 days before the development of the first cutaneous sign. To improve comparability the dosage of glucocorticosteroids was calculated as the dosage in prednisolone.

RESULTS

Of 216 patients with TEN, 11 (8 females, 3 males) had been receiving therapy with glucocorticosteroids when TEN developed. Lethality was 27% (3/11). The range was 29–74 years (mean = 54 years).

Most patients (7/11) were treated with glucocorticosteroids for neurological or neurosurgical conditions, other causes being septic arthritis, auto-immunhematological anemia, chronic polyarthritis and chronic bronchitis. All patients received various other drugs, ranging from acetylsalicylic acid to vincristinesulfate.

The dosage was 20-372 mg of prednisolone/day; only one patient received a dose less than 10 mg/day. Dosages for neurological-neurosurgical conditions ranged between 20 and 80 mg prednisolone/day. The highest dosage was given for treatment of septic arthritis during septic shock with hemophilus. Duration of therapy ranged between 5 days and several months (mean 4.3 weeks).

DISCUSSION

Except for a few case reports (1, 8–11) and sparse comments in the literature on TEN (12–15), this is the first investigation on a national basis of patients with TEN, aimed at antecedent therapy with glucocorticosteroids.

A small group of patients who had received glucocorticosteroids prior to the occurrence of TEN could be identified. Compared with the data of all patients with TEN, we were unable to discern any differences concerning sex, mean age or lethality. In contrast to

the few published case reports on patients suffering from immunological conditions, most of our patients received steroids for neurological or neurosurgical complaints. We cannot fully explain this observation. It may be due to differences in criteria for publishing case reports on severe skin reactions, between dermatologists and neurosurgeons.

Our results, as well as the above-mentioned case reports, show that in spite of the anti-allergic and anti-inflammatory potency of steroids (7), they cannot prevent the occurrence of TEN, even in high doses. This observation does not support an immunological mechanism in TEN and, together with the observation of an increased survival rate of patients managed without steroids (1), the basis for treating TEN with glucocorticosteroids is called into question.

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