Management of a Case of Toxic Epidermal Necrolysis in Intensive Care Unit

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ABSTRACT
Toxic epidermal necrolysis (TEN) is a life-threatening acute mucocutaneous syndrome with high mortality. It usually occurs because of adverse immune reactions to certain drugs. It is characterized by the necrosis of keratinocytes and the separation of the epidermis from the underlying dermis. TEN is a rare disorder with high mortality (30% of cases). A 42-year-old female with preexisting seizure disorder presented with generalized exanthema, increasing exfoliation, mucosal, and ocular involvement after intake of phenytoin as an antiepileptic agent. Clinical diagnosis of TEN was made with the involvement of around 80% of the total body surface area. Early treatment was started with dexamethasone along with cyclosporine for 10 days. Early diagnosis, management, fluid resuscitation, prevention of hypothermia, nutrition, analgesia, thromboprophylaxis, pain control, good aseptic practices, and amniotic membrane transplantation helped in quick recovery and nearly complete healing of the wounds.

Keywords: Case report, Cyclosporine, Mucocutaneous syndrome, Phenytoin, Toxic epidermal necrolysis.

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INTRODUCTION
Toxic epidermal necrolysis syndrome (TENS) is a life-threatening cutaneous condition presenting with features of epidermal loss, which is associated with extracutaneous and systemic manifestations. It is commonly precipitated by intake of drugs or changes in medications. However, other causes, such as bacterial and viral infections, have also been described. The most common drugs implicated in Stevens–Johnson syndrome (SJS)–toxic epidermal necrolysis (TEN) development include phenobarbital, phenytoin, carbamazepine, lamotrigine, nonsteroidal anti-inflammatory drugs, allopurinol cotrimoxazole, fluoroquinolones, albendazole, and fluconazole.1,2 Early recognition of TENS and stopping the causative agent is the cornerstone of treatment.3 Toxic epidermal necrolysis syndrome is seen in around 0.4–1.2 people per million population per year and carries a mortality of 20–40%.4 It can be differentiated from SJS by the extent of the total body surface area (TBSA) affected. In TENS, >30% of TBSA is affected.5 The main line of treatment is immunosuppression with dexamethasone and cyclosporine.6,7

CASE PRESENTATION
A 42-year-old female with a preexisting seizure disorder presented to our setup. She was on long-term treatment with phenobarbitone, which was changed to phenytoin 2 weeks before symptom onset for unknown reasons. Ten days later, she developed an area of erythema on her back with itching, which spread to the entire trunk and upper and lower extremities over the next 72 hours. It involved 80% of the TBSA with symmetrical, erythematous exanthema and blisters over the trunk, extremities, and face (Figs 1A and B). The Nikolsky I-sign was positive. Her oral mucosa showed erosions with a white-colored coating. The patient had severely restricted mouth opening with edema. The patient also had conjunctival congestion and sloughing. The SCORTEN score (SCoRe of Toxic Epidermal Necrolysis) was determined on admission. It is a prognostic score for TENS with one

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A case of phenytoin-induced TEN, and its management successfully with steroids and cyclosporine, is presented. The patient developed TEN 1 week after initiation of phenytoin. Immunosuppression was achieved using cyclosporine and dexamethasone. Cyclosporine inhibits activated T-lymphocytes, macrophages, keratinocytes which are involved in the disease process of TEN. It inhibits the metabolism of tumor necrosis factor (TNF). It also has an antiapoptosis action, which interrupts the desquamation and allows early reepithelialization of the affected skin and mucosa. Secondary infection should be prevented until reepithelialization. Skin lesions start healing by 2 weeks. Nonadhesive nature sterile dressing should be considered for skin lesions. Evidence is poor regarding appropriate timing of debridement of the affected skin. The dressing of the involved area can be done by biological dressing, biosynthetic dressing, or fiber dressing such as nanosilver impregnated dressing. No difference has been found between use of debridement or antishear wound care involving leaving of detached skin at place to act as biologic dressing. Recent evidence suggests use of nanocrystalline dressing instead of petroleum-impregnated dressing. Hospital-acquired infection is common in TENs and can be life threatening. Most common causative organisms are staphylococcus and pseudomonas. Good wound care and complete aseptic precautions while handling the patient helps to prevent infection. Prophylactic antibiotics do not change survival and are not recommended. Phenytoin alternatives like nonaromatic antiepileptic agents like valproic acid and topiramate are safe in patients who have developed phenytoin-induced TEN. Immunosuppression, thermoregulation, wound care, nutrition, infection control, and analgesia were the mainstays of management.

**Conclusions**

We hereby present the successful management of a rare case of TEN with extensive skin and mucosal involvement admitted to the ICU. Immunosuppression, sepsis care, wound care, nutrition, infection control, and analgesia were the mainstays of management.

**Highlights**

We present successful intensive care management of a rare case of TEN. Immunosuppression, aseptic practices, wound care, fluid management, good nutrition, and pain control are the critical aspects of management in these cases.
References


