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Recent Therapeutic Approaches in Toxic Epidermal Necrolysis and Stevens - Johnson syndrome

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Abstract

Toxic epidermal necrolysis (TEN) and Stevens-Johnson syndrome (SJS) are rare but potentially life-threatening medical emergencies and involves mucous membrane characterised by widespread epidermal loss. The main cause may be drug induced by some of the frequently used drugs like Antibiotics (chloramphenicol, macrolides, penicillin, quinolones, most commonly by sulphonamides), Anticonvulsants (carbamazepine, phenobarbitone, lamotrigine, valproate, phenytoin), NSAIDs and Allopurinol. Some of the infections like Mycoplasma pneumonia may be responsible for the occurrence of TEN and SJS rarely. This article presents an overview of the aetiology, pathogenesis and clinical features of TEN and SJS and discusses the principles of management and the role of potential disease-modifying therapies.

Keywords: Toxic epidermal necrolysis; Stevens-Johnson syndrome; Antibiotics

Introduction

Toxic epidermal necrolysis (TEN) and **Stevens-Johnson syndrome** (SJS) are rare but potentially life-threatening medical emergencies and involves mucous membrane characterised by widespread epidermal loss.

The main cause may be **drug induced** by some of the frequently used drugs like **Antibiotics** (chloramphenicol, macrolides, penicillin, quinolones, most commonly by sulphonamides), Anticonvulsants (carbamazepine, phenobarbitone, lamotrigine, valproate, phenytoin), **NSAIDs** and Allopurinol. Some of the infections like Mycoplasma pneumonia may be responsible for the occurrence of TEN and SJS rarely [1,2].

Epidemiology

The incidence of TEN and SJS is 0.4–1.2 and 1.2–6 cases/ million/ year, respectively. Women are more frequently affected than males and incidence increases with age [3].

People who are at risk includes Slow acetylators, Patients treated for brain neoplasms or head injury, **Immunocompromised patients** and Patients having HIV and **AIDS**.

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There are **societies** related to ophthalmology which are serving their best to make the world aware of many things related to the eye and its disorders. One of them include **Afro-Asian Council of Ophthalmology** is one of the oldest International ophthalmological societies which is representing the major of Afro Asian countries. It promotes the advancement of all modern techniques in all aspects of Ophthalmology [4,5]. Another society called **Somali Ophthalmological Society** works with ophthalmologic societies to upgrade ophthalmic studies and to enhance access to the most elevated quality eye.

Recently an author, **Arshalooz J Rahman** presented **Causes and outcome of Stevens-Johnson syndrome in children presenting to a tertiary care center** at **9th World Dermatology & Pediatric Dermatology Congress** conducted at UK on October 10-11, 2016 which created good impact in the public [6,7].

Another eminent personality, **Marzook A M** has presented **Fluconozole induced Stevens Johnson syndrome-toxic epidermal necrolysis overlap** at **3rd International Conference and Exhibition on Pharmacovigilance & Clinical Trials** at Hyderabad, India on October 27-29, 2014 and made people aware of the new side-effects of the drugs and suggested that fluconazole should be considered as one of the risk drug capable of causing SJS/TEN [8-15].

Kendrick Co Shih Richie Chan from University of Hong Kong Hong Kong is going to present an abstract on **Management of Early and Late Ocular Manifestations of Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis** at the upcoming conference **11th Global Ophthalmologists Annual Meeting** at Dubai, UAE on September 25-27, 2017.

Clinical Features

Acute Phase

In initial stages TEN and SJS will show symptoms like stinging eyes, fever and difficulty in swallowing and later involves cutaneous involvement of presternal region of the trunk and the face palms and soles [15-30]. **Erythema** and erosions of the ocular, buccal, genital mucosa occurs in more than 90% of patients, and involving the respiratory and gastrointestinal tracts in some cases.

Most symptoms involving ocular region are acute **conjunctivitis**, eyelid edema, crusts, **erythema**, and ocular discharge, to conjunctival membrane or corneal erosion or pseudomembrane formation, and, in severe cases, to cicatrizing lesions, fornix foreshortening, symblepharon, and corneal ulceration [30-40].

Other notable symptoms includes haemorrhagic crusting of the lips, conjunctivitis, intense pain in affected areas of skin, genital soreness and erosions, **arthralgia**

Late phase

In the late phase, symptoms like hyper and hypopigmentation of the skin, nail dystrophies and ocular complications (severe dry eyes, symblepharon, **trichiasis**, visual loss, distichiasis, ankyloblepharon, entropion, corneal ulceration and lagophthalmos will occur. Mucosal sequelae involving mainly the oral and oesophageal mucosa, and to a lesser extent lung and genital mucosa are the other symptoms [41-57].

Pathogenesis

The exact sub-atomic premise of TEN and SJS still stays to be completely clarified. It gives the idea that TEN patients, who have an expanded occurrence of the haplotype HLA-B12, exhibit a failure to detoxify moderate receptive medication

metabolites. A safe reaction is then mounted against the antigenic complexes formed by the collaboration of these metabolites and host tissues [58-63]. There is additionally convincing proof that the end-purpose of TEN and SJS (ie **epidermal necrolysis**) is because of far reaching apoptosis of keratinocytes. Cytokines, for example, interleukin-6, tumor corruption calculate alpha also, the CD95 framework (Fas ligand and Fas receptor) seem to assume a part in the acceptance of this apoptosis and subsequently have moved toward becoming focuses for conceivable restorative mediations in TEN [64-75].

Diagnosis

TEN and SJS can be diagnosed by the physical and symptomatic examination and by **skin biopsy** and immunofluorescence to exclude other differential diagnosis [75-81].

Therapeutic interventions

Immediate withdrawal of the causative drug

1. **Supportive therapy:**
    - Fluid and electrolyte balance
    - Regulation of temperature to minimise heat loss
    - Topical antiseptics
    - Debris should be cleaned daily
    - Pain relief
    - Nutritional support
    - Monitoring for signs of sepsis
2. **Pharmacotherapy:**

    High dose steroids for stabilising the epithelial loss. Other therapies like cyclosporine (3–4 mg/kg/day), cyclophosphamide (100–300 mg/day), N-acetylcysteine (2 g/6 hr) and plasmapheresis, Thalidomide, which is having anti-TNFα activity and acts as anti-angiogenetic and immunomodulator has been accepted for treatment of TEN. High-dose intravenous immunoglobulins can be used apart from its potential risk factors like renal and cardiac insufficiency which can be reduced with precautions.
3. **Treatment of sequelae:**

    Ophthalmological complications like ocular sequelae, conjunctival scarring and possible blindness should be avoided by prior treatment. Antiseptic and lubricative eye drops should be used to avoid severe dryness.

Recently a case report titled **Late Sequelae in a Survivor of Stevens Johnson Syndrome in Childhood** has been published in the journal of **Pediatrics & Therapeutics** by Deniz Ozceker which presented its triggering factors and about long term sequelae. Another journal named **International Journal of Ophthalmic Pathology** is also publishing many articles related to the ocular disorders and their treatment.

Prevention

Precautions should be taken by avoiding the drug exposure and detailed history should be collected from the patient before prescribing the drug and Allergological testing should be done in the patients for identifying the drugs to which the patient is sensitive with possible re-challenging if necessary [82-100].

Conclusion

As SJS and TEN are severe and life-threatening, careful precautions and care should be taken to avoid the condition. Studies should be encouraged on the treatment of SJS/TEN to decrease the morbidity and mortality rates towards this syndrome.

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