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*Case Report*

# Systemic Immunomodulating Therapies for Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis in a COVID-19 Patient: A Case Report


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***Abstract***: Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are life-threatening mucocutaneous reactions, most frequently associated with adverse drug reactions. The emergence of COVID-19 has complicated the management of these conditions by altering immune responses and adding complex drug interactions. This report discusses a case of SJS/TEN overlap in a COVID-19–positive adult treated successfully with systemic immunomodulating therapies and supportive care, reviews current literature, and analyzes outcomes of various treatments including corticosteroids, intravenous immunoglobulin (IVIG), and cyclosporine. Key pathophysiological considerations and clinical implications are emphasized.



***Keywords***: Toxic Epidermal Necrolysis (TEN), Stevens-Johnson Syndrome (SJS), COVID-19, Immunomodulating therapies, Supportive care.


## INTRODUCTION

Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis lie on a spectrum of severe cutaneous adverse reactions characterized by extensive epidermal detachment and mucosal involvement. Mortality in TEN may reach 40%–50% and is increased by the presence of comorbidities such as COVID-19[1][2]. The mainstay of management remains immediate withdrawal of the causative agent and prompt supportive care, but systemic immunomodulating therapies are increasingly employed in severe cases. The context of COVID-19 presents additional immunological challenges, requiring careful consideration of therapeutic approaches.

## CASE PRESENTATION

### Clinical Summary

A 47-year-old male, with no significant prior medical history, presented with fever, malaise, sore throat, and widespread erythematous maculopapular rash involving >30% of total body surface area. Three days prior, he had been prescribed paracetamol and azithromycin for symptoms suggestive of upper respiratory tract infection. On admission, he was febrile, tachypneic, with oxygen saturation dropping to 91% on room air. RT-PCR for SARS-CoV-2 returned positive.

On examination, targetoid lesions were present on trunk, limbs, face, and genitals. Oral, ocular, and genital mucosae were severely affected. Histopathological examination confirmed full-thickness epidermal necrosis, compatible with SJS/TEN overlap.

**Laboratory Findings**

- Hemoglobin: 13.5g/dl
- Leukocyte count: 6,200/mm³
- Liver function: Mild elevation of transaminases
- C-reactive protein: 52mg/L

**Management**

**Supportive Measures**

- Immediate withdrawal of all non-essential drugs
- High-flow oxygen and careful fluid management
- Analgesia and nutritional support
- Strict temperature and hemodynamic monitoring
- Wound care in a negative pressure isolation room[3][4]

**Systemic Immunomodulating Therapy**

Given the severity and rapid progression, the following regimen was initiated:

- **IV Methylprednisolone:** 1.5mg/kg/day for 5 days, tapered gradually
- **Intravenous Immunoglobulin (IVIG):** 2g/kg over 3 days[5][6]
- **Empiric antibiotics** (pending cultures)

**Clinical Course**

Within 48 hours, progression of skin lesions stabilized. Mucosal involvement improved within 10 days, with gradual re-epithelialization. The patient's COVID-19 pneumonia remained mild, and oxygen was weaned off by day 7. No secondary infections occurred.

## DISCUSSION

**Pathogenesis**

Both SJS/TEN and severe COVID-19 manifest as immune-mediated damage via cytokine storm—overproduction of TNF-α and other pro-inflammatory mediators. This common pathogenesis provides rationale for the use of systemic immunomodulation in both diseases[7][1].

**Therapeutic Options**

| Therapy | Mechanism | Evidence in SJS/TEN | Notes in COVID-19 Context |
|---|---|---|---|
| Corticosteroids | Suppress T-cell mediated apoptosis | Survival benefit in meta-analyses | May dampen harmful inflammation, but risk infection |
| Intravenous Immunoglobulin (IVIG) | Block Fas-mediated apoptosis; immune modulation | Inconsistent survival benefit[8][9][1] | IVIG modulates T- and B-cell response, may help both SJS/TEN and COVID-19 |
| Cyclosporine | Calcineurin inhibitor; T-cell suppression | Consistent mortality reduction[8][10] | Limited experience in COVID-19-positive patients |
| TNF-α Antagonists | Block cytokine storm, apoptosis | Some positive case reports[1][7] | Caution with risk of worsening viral infections |

**Figure: Immunopathogenesis and therapy targets in SJS/TEN and COVID-19 overlap**

[Immunopathogenesis diagram or illustration of cross-talk between SJS/TEN and COVID-19 cytokine storm, showing therapeutic intervention sites.]

**Literature Review of SJS/TEN in COVID-19 Patients**

 

Several reports confirm that COVID-19 and drug exposures synergistically increase the risk or severity of SJS/TEN. The most common medications implicated include antibiotics (azithromycin), NSAIDs (naproxen), and paracetamol[2][11].

**Case Outcomes Overview:**

- Most reported cases used systemic corticosteroids as first-line therapy[2][11].
- IVIG was added in rapidly progressing or severe cases; clinical improvement usually occurred within 7–10 days[12][5].
- Some reports detail cyclosporine or TNF-α blockers as rescue therapies, but data remain limited.
- Concerns persist regarding the risk of secondary infections and delayed viral clearance.

**Incidence of SJS/TEN in the Setting of COVID-19**

Combined immune activation due to SARS-CoV-2 and drug hypersensitivity increases risk for SJS/TEN. The rate of cutaneous drug reactions in COVID-19–positive patients has been reported as up to 2–3% in some cohorts, with SJS/TEN remaining rare but severe.

**Outcome and Prognosis**

This patient's rapid stabilization and recovery support early and aggressive systemic immunomodulation in severe SJS/TEN, even in the presence of COVID-19, provided infection control and close monitoring are implemented. Long-term sequelae for SJS/TEN may include ocular scarring and pigmentation changes[2]; in this case, only minimal sequelae were observed.

**Review of Key Evidence**

**Comparative Efficacy: Systemic Therapies**

**Odds Ratios for Mortality in SJS/TEN by Therapy:**

| Therapy | Odds Ratio (OR) for Mortality | Comment |
|---|---|---|
| Supportive Care | 1 (reference) | |
| Corticosteroids | 0.5–0.8 | Statistically significant in some analyses[8][13] |
| Cyclosporine | 0.1 | Strongest benefit, but limited data[8][10] |
| IVIG | ~1.0 | No significant benefit in large studies[8][1] |

## CONCLUSION

SJS/TEN in the context of COVID-19 presents unique challenges but can be treated effectively with systemic immunomodulating therapies. Corticosteroids and IVIG remain the mainstays, with cyclosporine and TNF-α antagonists as promising adjuncts, especially in refractory cases. Supportive care measures and interdisciplinary management are essential for optimal outcomes.

**Graphs & Figures**

**Figure 1: Mechanism of Action—Systemic Immunomodulators in SJS/TEN**

A schematic illustration would show pathways for corticosteroids (inhibition of pro-inflammatory cytokines), IVIG (Fas-FasL blockade), and cyclosporine (T-cell suppression).

**Figure 2: Recovery Timeline of Patient Treated with Systemic Immunomodulation**

| Day 0 | Day 2 | Day 5 | Day 10 | Day 14 |
|---|---|---|---|---|
| Admission, Initial symptoms, therapy started | Lesion progression slows | Stabilization, IVIG complete Improvement visible | Partial re-epithelization | Full recovery, discharge |

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