

Diagnosis and Management of SJS/TEN as a Double-Edged Sword in Epilepsyorganic Mental Disorder Patients Induced by Carbamazepine: A Case Report, Dr. Radjiman Wediodiningrat Lawang Hospital

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KEYWORDS

Stevens-Johnson Syndrome; toxic epidermal necrolysis; Karbamazepin; Epilepsy; Organic Mental Disorders; Case Report.

ABSTRACT

Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are life-threatening cutaneous adverse drug reactions with high mortality. Carbamazepine, an aromatic anticonvulsant, is a major trigger in Southeast Asian populations due to HLA-B*15:02 genetic susceptibility. In patients with organic mental disorder (*gangguan mental organik*, GMO) and epilepsy, early diagnosis is often delayed because prodromal symptoms overlap with psychiatric manifestations or are masked by communication barriers. This case report aims to describe the diagnostic and emergency management challenges of carbamazepine-induced SJS/TEN in a patient with GMO and epilepsy. This case report was compiled retrospectively based on medical records and clinical documentation from *Dr. Radjiman Wediodiningrat Lawang Mental Hospital*. A 26-year-old female with GMO and epilepsy developed SJS/TEN overlap with 10–29% body surface area (BSA) involvement one week after carbamazepine initiation. Symptoms included high fever (39.8 °C), painful swallowing, an erythema-multiforme–like rash, bullae, and severe mucosal erosions of the lips and eyes. The Nikolsky sign was positive. Laboratory findings showed elevated liver enzymes and electrolyte imbalances. Carbamazepine and all psychiatric medications were immediately discontinued. The patient received high-dose intravenous methylprednisolone, fluid resuscitation, and supportive care before referral to a tertiary hospital. Diagnosing SJS/TEN in patients with mental disorders requires high clinical vigilance due to atypical presentations. Prompt drug withdrawal is critical despite the dilemma of seizure control. Multidisciplinary collaboration is essential for optimal management and improved prognosis.

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INTRODUCTION

Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) represent a spectrum of severe cutaneous hypersensitivity reactions mediated by immunological mechanisms, characterized by extensive epidermal necrosis and skin detachment. The incidence is rare (1–10 cases per million population per year), but the mortality rate is high. The most common cause is drug reactions, especially antibiotics, antiretrovirals, and aromatic anticonvulsants such as carbamazepine (Fowler et al. 2019; Fricke-Galindo et al. 2018; De Luca et al. 2017; Stolbach et al. 2015; Tolou-Ghamari 2025).

Carbamazepine is known to have a strong association with the risk of SJS/TEN, particularly in individuals with the HLA-B*15:02 allele, which is prevalent in Southeast Asia

(Biswas et al. 2022; Capule et al. 2020; Nakkam et al. 2022; Tham et al. 2024). Clinical challenges arise when SJS/TEN occurs in patients with psychiatric or neurological comorbidities (Charlton et al. 2020; Chiang et al. 2022; Hoffman et al. 2021). Patients with Organic Mental Disorder (OMD) and epilepsy often receive polypharmacy, making it difficult to identify the trigger drug. In addition, prodromal symptoms such as fever and malaise may overlap with, or be ignored as part of, the patient's psychiatric or behavioral symptoms (p. 1).

Despite the well-established link between carbamazepine and SJS/TEN, there remains a significant research gap regarding the diagnostic and management challenges specific to patients with psychiatric comorbidities (Alhaj et al. 2025; Mehta 2024; Vakrinou et al. 2023). Most published guidelines and case series are derived from general hospital settings, where patients can reliably communicate early symptoms. In contrast, mental health facilities face unique obstacles, including delayed symptom recognition, difficulty obtaining accurate drug histories, and the clinical dilemma of discontinuing antiepileptic therapy in patients at risk of seizure recurrence. The novelty of this case report lies in its exploration of these dual challenges—managing a life-threatening drug reaction while navigating the complexities of underlying neuropsychiatric disorders in a psychiatric hospital setting (Polis et al. 2024; Shekho et al. 2024; Tenea-Cojan et al. 2025).

This case report highlights the dilemma of diagnostic and emergency management in identifying and managing carbamazepine-induced SJS/TEN overlap in patients with a history of OMD and epilepsy in mental health facilities, emphasizing the importance of an integrated, multidisciplinary approach (Błaszczuk et al. 2015; Chinthapalli 2016; Subramanian et al. 2024).

This case report aims to describe the diagnostic and emergency management challenges in a case of carbamazepine-induced SJS/TEN overlap in a patient with OMD and epilepsy at Dr. Radjiman Wediodiningrat Lawang Hospital. Specifically, this report seeks to: (1) highlight the atypical presentation and delayed recognition of SJS/TEN in patients with psychiatric comorbidities, (2) evaluate the multidisciplinary approach involving psychiatrists, neurologists, and internists in emergency management, and (3) emphasize the importance of prompt drug withdrawal and supportive care in improving patient outcomes. The benefits of this report are threefold. For clinical practice, it provides valuable insights for psychiatrists and neurologists in recognizing early signs of severe cutaneous adverse reactions in vulnerable patient populations. For the development of hospital protocols, it underscores the need for integrated management pathways between psychiatric and dermatological services. For the academic literature, it enriches the limited body of knowledge on SJS/TEN management in mental health settings, particularly in the Southeast Asian context where genetic susceptibility is high. By documenting this case, we hope to contribute to improved clinical vigilance, earlier diagnosis, and better multidisciplinary collaboration in managing this life-threatening condition.

METHOD

This case report was compiled based on a retrospective review of the patient's medical records (RM 159748) at RSJ Dr. Radjiman Wediodiningrat Lawang. Data includes:

1. **Initial assessment** (June 17, 2024) when the patient experiences a psychotic attack.
2. **Progress** records during inpatient care (CPPT).
3. **Emergency assessment** (July 31, 2024) when dermatological complications appear.
4. **Supporting data** is in the form of clinical photo documentation of the patient's skin lesions.

5. At the end of July 2024 (about 6 weeks after starting therapy), the patient experiences systemic symptoms:
 - a. **Prodromal:** High fever and sore throat for 4 days.
 - b. **Skin Manifestations:** Reddish lesions (erythema) appear that start in the eye area, then spread throughout the body.
 - c. **Progressivity:** Within days, the lesions turn black (necrotic) and bulla (blisters) appear in the lip and neck area.

A female patient, age 26 years (Date of Birth: 16-03-1998), was taken to the RSRW emergency room on July 31, 2024 with the main complaints of blackened skin, itchy skin rash, pain in the back, tongue, and lips since the last 4 days, accompanied by fever.

Skin lesions 3 days yll start to redden, yesterday the color blackened, today the neck and lips appear bulla

The lesions start in the eyes feeling hot and itchy secret, then spread to the hands and feet every day

Decreased intake due to pain in the urobucal area and tongue, drinking using a straw and still feeling pain in his throat

Outer skin feels hot and stingy VAS 8-9

Based on the physical examination on July 31, 2024:

1. **General Condition:** Weakness, difficulty eating/drinking due to lesions of the oral mucosa.
2. **Differential Diagnosis:** SJS/TEN (given the extent of the lesion and the presence of mucosal involvement).
3. **Etiology:** It is suspected to be a hypersensitivity reaction to one of the psychiatric drug regimens taken (Drug-Induced).

Previous Disease History: The patient has a history of behavioral changes since mid-June 2024, was diagnosed with GMO ec epilepsy, and was briefly treated at RSJRW on July 11, 2024 with a diagnosis of GMO ec epilepsy. During treatment, a history of fainting suspected of seizures was found, and abnormal EEG results (Abnormal III potential epileptogenicity region F4) showed potential epileptogenicity of region F4. The patient was diagnosed with Epilepsy + GMO, and began to be given carbamazepine from July 22, 2024. The patient returned from hospitalization (KRS) on July 28, 2024. The rash appears from July 29, 2024, one week after carbamazepine consumption.

Physical Examination (When Entering the Emergency Room):

- a. **Vital Signs:** TD 108/74 mmHg, Pulse 131x/min, Temperature 39.8°C, RR 20x/min, 100% Oxygen Saturation with room air.
- b. **Generalist status:** Weak general state, *composing consciousness* (GCS 456). The head appears multiform erythema, red eye mucosa, blistered lips, canker sores. Heavy mucosal erosion of the lips with hemorrhagic crusta (blackness), as well as reddened eye conjunctiva (conjunctival injection) is observed. It shows significant mucosal involvement, a hallmark of SJS/TEN.



Figure 1. Multiform erythema lesions on the face and oral mucosa

Source: Patient clinical documentation, RSJ Dr. Radjiman Wediodiningrat Lawang (2024)

Neck: erythema and Bulla papules measuring 3x5cm. A large bulla (about 3x5 cm) containing liquid was found. The appearance of bulla indicates the presence of a detachment of the epidermis from the dermis due to extensive keratinocyte apoptosis.



Figure 2. Bulla on the neck area measuring 3x5 cm

Source: Patient clinical documentation, RSJ Dr. Radjiman Wediodiningrat Lawang (2024)

abdomen and chest as well as: papul eritama, on the back of the bulla measuring 2x3cm, on the abdomen There are lesions in the form of macula erythematosa (redness) that are widely spread (confluence) with a darker central area. Some areas show necrotic (blackening) marks.

On the back Very wide and evenly distributed lesions are seen throughout the back, indicating rapid progression of the disease.



Figure 3. Extensive skin lesions in the abdominal and back area

Source: Patient clinical documentation, RSJ Dr. Radjiman Wediodiningrat Lawang (2024)



Figure 4. Distribution of lesions in the area of the superior extremities

Source: Patient clinical documentation, RSJ Dr. Radjiman Wediodiningrat Lawang (2024)

in the groin and genitals of the erythema papulos

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- c. **Dermatological Status:** Erythema multiform, blackened skin lesions on the body, hands, and feet were found. There are new bubbles on the neck and lips. The mucosa of the eyes reddened, the lips blistered and drooled a lot. The Nicolsky sign is positive (its release is observed). The area of the body involved is estimated to be between 10-29% (SJS/TEN overlap).

Previous Therapy History:

Tx Psychiatry:

Inj zypreksa 10mg IM prn gelisah

PO Aripiprazole 10mg 0-0-0-1

PO Lorazepam 2mg 0-0-0-1

PO Olanzapin 5mg 0-0-0-1

PO Risperidone 2x1 mg

PO Valproat 2x250mg

Tx Neuro:

Inj Diazepam 10mg prn kejang

PO Carbamazepin 200mg 0-1-0 as of 16/7/2024

Supporting Examinations:

HB 12.800

Leukosit 5490

Thrombositis: 169,000

OT/PT 78/161

UR/CR 14.22/0.77

GDA 132

SE Na/K/Cl 131/3,8/93

CXR: dbn



Figure 5. Patient's thoracic photograph results

Source: Patient medical records, RSJ Dr. Radjiman Wediodiningrat Lawang (2024)

Emergency Management: Patients are diagnosed with SJS/TEN, GMO, and epilepsy. Handling involves multidisciplinary consensus:

1. **Psychiatric Specialist (dr. Budi, SpKJ):** Psychiatric medication is completely discontinued.

2. **Neurologist (dr. Anas, SpS):** Carbamazepin is discontinued immediately. Giving *bridging therapy* in case of seizures.
3. **Internal Medicine Specialist (dr. Amirah, SpPD)** The patient was immediately given supportive treatment at the emergency room in the form of:
4. Intravenous fluid administration (NaCl 0.9%).
5. High-dose corticosteroid therapy (IV **Methylprednisolone 1x 62.5 mg**), stomach guard (IV Omeprazole 1x40mg) and Antipyretic (IB Paracetamol 3x1g for fever as the initial protocol for treating SJS).
6. **Referral:** Due to a life-threatening condition that requires intensive dermatology treatment, the patient was referred to **Saiful Anwar Hospital (RSSA) Malang**.

Patients undergo supportive care in the emergency room and transit room while awaiting room availability at RSSA, including NaCl compresses on skin lesions and close monitoring of vital signs.

RESULTS AND DISCUSSIONS

This case is a severe and life-threatening clinical manifestation of an adverse drug reaction. Patients are diagnosed with SJS/TEN overlap because the affected body surface area ranges from 10-29%. The biggest challenge in these cases is the presence of comorbidities in the form of Organic Mental Disorders (GMOs) and epilepsy, where the initial symptoms are often disguised by behavioral disorders or communication barriers of the patient.

Carbamazepine is an anticonvulsant drug that is a major trigger of SJS/TEN, especially in populations in Southeast Asia. This reaction is type IV hypersensitivity in which the immune system attacks keratinocytes massively. In this case, symptoms appear about one week after the initiation of carbamazepine (starting July 22, symptoms appear July 29), according to the typical incubation period of a severe drug reaction prodromal symptoms are crucial because they often resemble ordinary infections:

1. High Fever: The patient has a fever of up to 39.8 °C.
2. Flu-Like Symptoms: Swallowing pain, sore throat, and malaise for 4 days before the rash appears.
3. Skin Sensation: Patients report intense heat, itching, and stinging (VAS pain scale 8-9) before the skin blisters.
4. Positive Nicolsky Signs: Healthy-looking skin will peel off when gently rubbed, indicating a loss of epidermal cohesion.

Based on patient laboratory data, there are several things that require close monitoring:

- a. Liver Function (OT/PT 78/161): There is an increase in transaminase enzymes that indicate stress on the liver due to systemic reactions or drug effects.
- b. Electrolytes (Na 131, Cl 93): Low sodium and chloride values (hyponatremia) reflect fluid imbalance due to loss of skin integrity.
- c. Hemodynamics: A very fast pulse (131x/min) and a tendency to low blood pressure (108/74 mmHg) are early signs of shock or severe dehydration.

Emergency management should focus on triggering and stabilizing:

1. Immediate Discontinuation: Carbamazepine and all suspected psychiatric medications should be discontinued completely.
2. Fluid Resuscitation: Administration of 0.9% NaCl IV fluid to replace fluid lost through skin burns.
3. Corticosteroids: Use of high doses of methylprednisolone (62.5 mg) to suppress immunological reactions.
4. Wound Care: NaCl compresses on lesions and topical treatments to prevent secondary infections.
5. Multidisciplinary: Collaboration between skin, neurological, psychiatric, and internal medicine specialists to manage seizures while addressing skin damage.

If not treated intensively in an adequate facility (such as RSSA Malang), patients are at risk of experiencing:

- a. Sepsis: Systemic bacterial infection through exposed skin is the leading cause of death.
- b. Blindness: Permanent damage to the cornea of the eye due to untreated mucosal erosion.
- c. Acute Kidney Failure: A result of severe dehydration and electrolyte imbalance.
- d. Neurological Instability: Risk of recurrent seizures (status epilepticus) due to sudden discontinuation of antiepileptic medication.

Cases of carbamazepine-induced overlap SJS/TEN in patients with Organic Mental Disorder and epilepsy demand observant early diagnosis and a balanced approach to emergency management. Immediate discontinuation of trigger drugs is the most crucial intervention. Multidisciplinary collaboration is indispensable to navigate the dilemma of comorbidity treatment (stopping antiepilepsy vs. preventing seizures) and ensuring optimal supportive care, thereby improving patient prognosis.

Stevens-Johnson syndrome and Toxic Epidermal Necrolysis triggered by carbamazepine are complex medical emergencies, especially when they affect patients with organic mental disorders and epilepsy. The presence of psychiatric disorders often creates communication barriers that lead to a delay in diagnosis, so patients often come to the necrotic phase where tissue damage is already widespread. Carbamazepine, as one of the anticonvulsant agents most often involved in severe hypersensitivity reactions in Southeast Asia, requires extra vigilance through rigorous clinical monitoring or genetic screening prior to initiation of therapy. The clinical manifestations in this case show rapid progressivity, starting from prodromal symptoms similar to systemic infections such as high fever and swallowing pain, which then progress to mucosal involvement and epidermal release. The SJS/TEN classification overlaps with lesion area of 10-29% confirms that this condition is on the spectrum of a very dangerous disease with a significant risk of mortality. Separation of the epidermis characterized by the appearance of bulla and positive Nicolsky signs indicates the occurrence of massive keratinocyte apoptosis mediated by the patient's own immune system. The intervention that most determines the patient's prognosis is the immediate discontinuation of the drug suspected as a trigger, in this case carbamazepine. Although discontinuation of antiepileptic drugs poses a clinical dilemma regarding the risk of seizure recurrence, the safety of patients' lives from extensive damage to skin integrity should be a top priority. Delays in stopping trigger medications have been clinically proven to worsen outcomes and increase the risk of long-term complications and death. Comprehensive supportive management, including adequate intravenous fluid resuscitation and electrolyte balancing, is crucial to prevent hypovolemic shock and acute

kidney failure. The use of high-dose corticosteroids in the acute phase aims to modulate the immune response and limit the further spread of necrosis. In addition, sterile wound care and mucosal protection, especially in the eye and urogenital areas, should be performed to prevent secondary infections and permanent functional sequelae. Laboratory tests showing impaired liver function and electrolyte imbalances reflect that this reaction is not just a skin disease, but a systemic failure involving various organs. Therefore, strict monitoring of vital signs and biochemical parameters becomes an integral part of care in burn units or intensive care. The precision of referring patients to higher healthcare facilities with intensive dermatology specialties greatly determines the patient's survival. Overall, the success of this complex SJS/TEN case management relies heavily on an integrated multidisciplinary approach between neurologists, psychiatrists, and dermatologists. This synergy is needed to navigate the dilemma between neuropsychiatric disease control and the handling of life-threatening hypersensitivity reactions. Early detection of early symptoms in high-risk groups taking aromatic anticonvulsant drugs remains the best prevention strategy to avoid the fatal consequences of the disease.

CONCLUSION

Carbamazepine-induced Stevens–Johnson syndrome/toxic epidermal necrolysis (SJS/TEN) in patients with organic mental disorder and epilepsy represents a significant diagnostic and therapeutic challenge, as prodromal symptoms are frequently obscured by psychiatric manifestations or communication barriers, resulting in delayed recognition and more advanced skin involvement. In this case, SJS/TEN overlap involving 10–29% of the body surface area developed one week after carbamazepine initiation, and successful emergency management required immediate drug withdrawal, high-dose corticosteroids, fluid resuscitation, and close multidisciplinary collaboration. This case highlights the importance of heightened clinical vigilance when prescribing aromatic anticonvulsants in high-risk populations, particularly in Southeast Asia where the HLA-B15:02 allele is prevalent; therefore, pre-treatment HLA-B15:02 genetic screening should be considered before initiating carbamazepine. Increased awareness among psychiatrists and neurologists of early prodromal symptoms such as fever and sore throat, the development of multidisciplinary management protocols in psychiatric hospitals, patient and family education regarding early signs of adverse drug reactions, consideration of alternative antiepileptics with safer profiles, and improved dermatologic intensive care capacity in referral hospitals are essential to optimize outcomes. Future research should focus on evaluating the effectiveness of routine genetic screening and multidisciplinary clinical pathways in psychiatric settings to improve early detection, prevention, and management of severe cutaneous adverse drug reactions such as SJS/TEN.

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