CASE REPORT

Diagnostic Challenge in Distinguishing Steven Johnson Syndrome/ Toxic Epidermal Necrolysis from Dengue in Endemic Areas

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ABSTRACT

This case report discusses the clinical course of a female in her mid-20s with a history of seizure disorder who presented with a fever and rapidly evolving dermatological symptoms, later diagnosed as Stevens-Johnson Syndrome (SJS) with Toxic Epidermal Necrolysis (TEN) overlap. Initially presenting with high-grade fever, cough, hypotension, and tachycardia, the patient's recent travel to a dengue-endemic area led to initial suspicion of an infectious etiology, including dengue fever. However, negative tropical disease tests and the subsequent development of erythematous and painful maculopapular rashes, coupled with mucosal involvement, shifted the diagnosis towards a severe drug reaction. The recent addition of phenytoin to her antiepileptic regimen was identified as the likely trigger for SJS/TEN. With a SCORTEN score of 4, the patient received prompt treatment, including intravenous hydrocortisone and later oral prednisolone along with supportive care.

Keywords: Dengue, Drug-induced rash, Stevens-Johnson Syndrome, Toxic Epidermal Necrolysis, Phenytoin

INTRODUCTION

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are severe drug-induced skin reactions, often presenting with fever and a rapidly progressing rash. Given the overlap in symptoms with other infectious diseases like dengue fever, it is crucial to differentiate these conditions for appropriate management. This case report highlights the challenges in distinguishing between these conditions in a region endemic to dengue, with a particular focus on the role of anticonvulsant medications like phenytoin as a trigger for SJS/TEN.

CASE

A young adult woman in her mid-20 with a history of seizure disorder came to our emergency department with a three-day history of high fever and cough. She did not have chills or rigors, but by the time she reached us, her fever had spiked to 106.6°F (41.44°C), and she was tachycardic with a pulse of 160 beats per minute. Her blood pressure was worryingly low at 80/50 mm Hg. She had been taking levetiracetam for her seizures for a year, but because her seizures persisted, phenytoin was added to her treatment 28 days before her admission. She had recently traveled to a region known for dengue fever, which put us on high alert for a tropical disease. Initial blood tests showed that her white blood cell and platelet counts were low, but other routine lab results were normal. Despite her symptoms and travel history, tests for dengue, scrub typhus, leptospirosis, and

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COVID-19 were negative. Suspecting sepsis, we moved her to the ICU. Before the transfer, she was given a test dose of intravenous ceftriaxone, which triggered a rash near the IV site, so she was treated with hydrocortisone and pheniramine.

In the ICU, her condition remained concerning, with continued fever, low blood cell counts, and hypotension. By day 4, she developed widespread red rashes. Suspecting dengue, we repeated the tests, but they remained negative. On day 5, her rash worsened, becoming dusky and painful, spreading to her face and upper body. A dermatologist diagnosed her with Stevens-Johnson Syndrome with Toxic Epidermal Necrolysis (TEN) overlap, likely triggered by phenytoin. With a SCORTEN score of 4, indicating a serious prognosis, we started her on intravenous hydrocortisone, later switching to oral prednisolone.[1] Throughout her ICU stay, we carefully managed her fluids and electrolytes and took measures to prevent infection.

Table 1: Percentage of Body Parts Involving TEN

Body Involvement	
Face	2%
Chest anterior	9%
Abdomen	9%
Palms	2%
Back	18%
Perineum	1%
TOTAL	41%

Further testing revealed worsening anemia, leukopenia, thrombocytopenia, and liver enzyme abnormalities, along with thickening of the gallbladder wall, indicating potential liver involvement. Additionally, the patient experienced persistent symptoms of nausea, vomiting, and abdominal pain, which contributed to a marked decrease in appetite and overall nutritional intake. By Day 7, the bullae on her hands and feet had noticeably enlarged and become increasingly painful, necessitating fluid aspiration under strict aseptic conditions, followed by coverage with vaseline gauze to prevent secondary infection. Despite the persistent low total leukocyte count, her platelet count began to show a gradual increase, and her overall condition started to improve steadily by Day 10. Subsequently, the bullae and maculopapular rashes showed significant resolution, and her laboratory parameters, including liver function tests, began to normalize. Following a comprehensive and thorough medical review, the decision was made to transfer her to the isolation ward on Day 13 for continued monitoring and care.

DISCUSSION

In Nepal, where dengue is endemic, healthcare providers frequently face challenges in differentiating between dengue fever (DF) and Toxic Epidermal Necrolysis (TEN) due to similarities in their clinical presentations. Both diseases are characterized by fever, a symptom shared by many infectious and systemic conditions, making it difficult to immediately identify the underlying cause. However, recognizing the subtle differences in how these conditions present can play a decisive role in making an accurate diagnosis and guiding appropriate treatment.

Dengue fever usually begins abruptly with a high fever, accompanied by intense headaches, pain behind the eyes, and muscle and joint aches, often referred to as "breakbone fever." The rash typically appears around the third day of illness, starting as an erythematous, maculopapular eruption that can progress to petechiae or purpura. It often begins on the face, chest, and limbs before potentially spreading to other parts of the body. In more severe forms of dengue, such as dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS), the rash may present with signs of bleeding, including bruising and spontaneous hemorrhage. A hallmark of dengue is a low platelet count, often below 100,000/μL, which signals an increased risk of bleeding.[2] The disease is transmitted by the *Aedes* mosquito, and Nepal's tropical climate, especially during the monsoon season, provides ideal conditions for mosquito breeding and seasonal outbreaks.[3-4]

Toxic Epidermal Necrolysis (TEN), while sharing some initial similarities with dengue, presents in a distinct manner. Both conditions may begin with fever and general discomfort, but the rash in TEN evolves differently. It starts as a red rash that quickly develops into large, fluid-filled blisters and areas of necrotic skin that peel off, typically affecting more than 30%

of the body. A key feature of TEN is the involvement of mucous membranes, such as the eyes, mouth, and genital areas, which is uncommon in dengue. While the rash in TEN can appear suddenly, it progresses rapidly to painful skin loss and is often linked to recent medication use or infections.[5] In Nepal, common drugs triggering TEN include antibiotics like penicillin and sulfonamides, anticonvulsants, or NSAIDs, which can overlap with cases of dengue, especially when drug use or secondary infections are involved. An important laboratory finding in TEN is a normal platelet count, in contrast to the marked thrombocytopenia seen in dengue.

Although both dengue and TEN can present with fever and rashes, differences in the progression, rash features, and additional symptoms are critical for distinguishing between the two. In dengue, the rash generally improves with supportive care, whereas in TEN, the rash rapidly deteriorates, leading to significant skin loss and potential life-threatening complications. TEN is further differentiated by the lack of platelet abnormalities and the presence of severe blistering and mucosal involvement, which are not typical in dengue.[5]

CONCLUSION

Clinicians must also consider the patient's recent history, as exposure to certain medications is a key indicator for diagnosing TEN. In regions like Nepal, where both diseases may occur simultaneously, careful attention to these clinical signs is vital for accurate diagnosis and appropriate treatment. While both conditions require swift and targeted interventions, their management approaches differ significantly, underscoring the importance of early identification.

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