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CASE REPORT: SUSPECTED THIAMPHENICOL-INDUCED STEVENS-JOHNSHON SYNDROME-TOXIC EPIDERMAL NECROLISIS OVERLAP IN A CHILD – DIAGNOSIS AND MANAGEMENT

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ABSTRACT
Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are rare delayed-type hypersensitivity reactions characterized by detachment of the epidermis and mucous membranes, along with skin necrosis. While these conditions are rare in children, they are most commonly caused by antibiotics, antiepileptic drugs, and antipyretic drugs. This case report aims to highlight thiamphenicol as a rare and potentially overlooked cause of SJS/TEN in pediatric patients. A 14-year-old girl who developed painful red patches accompanied by fluid-filled blisters on almost her entire body which appeared five days after taking the antibiotic thiamphenicol. The patient also experienced red, watery eyes; blisters on the lips and oral cavity; involvement of the nipples; and was unable to swallow due to severe pain in the mouth and throat. The patient was diagnosed with SJS-TEN overlap, and showed a good response to systemic corticosteroids (methilprednisolone) and supportive therapy. Her overall prognosis was favorable, with a Severity-of-Illness Score for Toxic Epidermal Necrolysis (SCORTEN) score of 1. The management of SJS/TEN involves a multidisciplinary specialist approach, immediate withdrawal of the suspected drug, administration of corticosteroids, and comprehensive supportive care.

Keywords: steven-johnson syndrome; tiamphenicol; toxic epidermal necrolisis

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INTRODUCTION

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are rare delayed-type hypersensitivity reactions that cause epidermal and mucosal layer necrosis and separation. These two disorders are defined according to the percentage of body surface area (BSA) covered by blistering or skin detachment. Blistering or epidermal detachment affects more than 30% of the BSA in TEN, less than 10% in SJS, and between 10% and 30% in SJS-TEN overlap syndrome. (Kuijper EC, 2020)Overall, the prevalence of SJS and TEN is estimated to be 1–6 cases per one million people per year for SJS and 0.4–1.2 cases per one million people per year for TEN. A study conducted in the United States found that the juvenile population had an incidence of 6.3 per 100,000 for SJS, 0.7 per 100,000 for SJS/TEN overlap syndrome, and 0.5 per 100,000 for TEN. The highest prevalence was reported among youngsters aged 11 to 15 years. The highest death rates were reported among children aged 0 to 5 years old and those with TEN. (Antoon JW, 2018)

Drugs are a major contributing factor to the development of SJS/TEN. Sulfonamide antibiotics, aromatic antiepileptic medicines, allopurinol, oxicam-class nonsteroidal anti-

inflammatory medications (NSAIDs), lamotrigine, and nevirapine are among the high-risk pharmaceuticals most usually linked to the development of SJS/TEN. The danger is usually limited to the first eight weeks of treatment, and the triggering medicines are often those that are given continuously for 4 to 28 days before the onset of symptoms. (Mockenhaupt M et al, 2019) Children exhibit a high recurrence rate of SJS, with 1 in 5 cases indicating a potential vulnerability and possible genetic predisposition. (Finkelstein Y et al., 2011).Currently, case reports of SJS/TEN specifically induced by thiamphenicol are extremely limited; therefore, thiamphenicol-associated SJS/TEN is considered a rare occurrence.In this case report, we present one case of SJS-TEN overlap that suspected induced by thiamphenicol in a 14-year-old girl. This case report aims to enhance the understanding of SJS-TEN overlap in pediatric patients, particularly regarding its diagnosis and clinical management. Additionally, it seeks to highlight thiamphenicol as a rare and potentially overlooked cause of SJS/TEN.

METHOD

This article is a case report study that provides diagnosis, clinical management, and patient follow up care. Data from this case report were obtained through anamnesis, physical examination, and supporting examinations conducted at Adam Malik General Hospital, Medan. The data obtained were then analyzed qualitatively and presented in narrative form. This case report discusses a a 14-year-old gir who experienced SJS-TEN overlap syndrome suspected by drug-induced due to thiamphenicol. This case report will describe the risk factors found, analyze individual case, and is expected to provide insights into clinical practice, especially regarding the problem of SJS-TEN overlap syndrome in children.

RESULT

A 14-year-old girl was referred to the Department of Dermatology and Venereology at H. Adam Malik General Hospital, Medan, with the chief complaint of painful red patches accompanied by fluid-filled blisters on almost her entire body, which had appeared two days prior to hospital admission. Initially, the red patches appeared on the abdomen and chest, then spread to the face, arms, and back. These patches were accompanied by clear fluid-filled blisters, some of which had ruptured and formed erosions. The patient also experienced red, watery eyes; blisters on the lips and oral cavity; involvement of the nipples; and was unable to swallow due to severe pain in the mouth and throat. The patient had no previous history of similar complaints, no known drug or food allergies, and no history of malignancy or similar conditions in herself or her family members. Based on her medication history, the patient had taken thiamphenicol, which was administered twice daily by her mother seven days prior to admission, for a duration of five days. Other medications taken by the patient included paracetamol 500 mg three times daily, oral dexamethasone twice daily, and cetirizine once daily. A prior history of drug allergy was denied. The patient also had no known allergies to previous vaccinations. There was no family history of similar complaints.

Physical examination revealed that the patient appeared moderately ill, was compos mentis, and had a body weight of 56 kg, height of 158 cm, and a body mass index (BMI) of 11.3 kg/m². Axillary temperature was recorded at 38.9°C, pulse rate at 110 beats per minute, and respiratory rate at 18 breaths per minute. Ulcerations were present on the oral mucosa. Ocular examination showed hyperemia of the right bulbar conjunctiva accompanied by discharge. Examination of the ears, nose, and throat revealed erosions on the nasal area and auricles, hyperemia of the pharynx, and erosions on the lip mucosa covered with dark reddish crusts.Dermatological examination showed multiple erythematous macules with well-defined borders, ranging in size from millimetric to plaque-like lesions, located on the thoracic,

abdominal, and dorsal regions. A flaccid, intact bulla was observed on the mental region, with positive Nikolsky and Asboe-Hansen signs. Several bullae had ruptured, leaving erosions on the right orbital and infraorbital regions, left buccal area, and neck. Additional erosions and multiple crusts due to ruptured bullae were found on the abdomen and back, with an estimated epidermal detachment involving approximately 10–30% of the total body surface area.

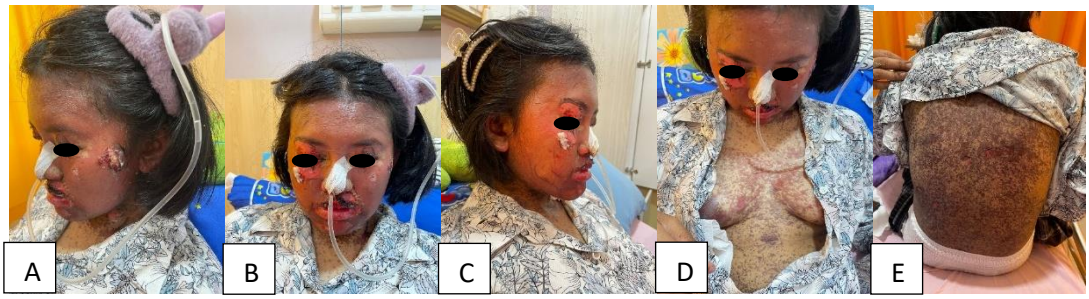

Figure 1. (A) Erosions on the orbital, left buccal, and cervical regions.(B) Bulla on the mental region and erosions on the right and left orbital, infraorbital, and oral regions.(C) Ruptured bulla on the right buccal region and erosions on the right orbital and infraorbital regions.(D) Erythematous macules ranging from lenticular to plaque size on the thoracic and abdominal regions. (E) Erythematous macules and erosions on the dorsal region.

Complete blood count revealed thrombocytosis of 530,000/µL (reference range: 150,000–450,000/µL) and mild monocytosis of 11.6% (reference range: 2.00–8.00%), while other hematological parameters were within normal limits. Liver and renal function tests, serum electrolytes, arterial blood gas analysis, albumin, and random blood glucose levels were all within normal ranges. The Algorithm of Drug Causality in Epidermal Necrolysis (ALDEN) score for thiamphenicol was 3. The patient was diagnosed with SJS-TEN overlap, suspected by drug-induced due to thiamphenicol. The SCORTEN score was 1, corresponding to a mortality rate of 3.2%. The patient was managed collaboratively with the departments of pediatrics and ophthalmology. Vital signs and fluid balance were monitored every 6 hours. A high-protein liquid diet was administered via a nasogastric tube due to the patient's inability to swallow as a result of severe oral and pharyngeal pain. Management included discontinuation of all suspected medications, intravenous fluid replacement with IVFD D5% NaCl 0.45% at 20 cc/hour, methylprednisolone injection 32 mg divided into two daily doses and tapered off according to the patient's clinical condition, ranitidine injection 50 mg every 12 hours, paracetamol injection 600 mg every 8 hours, ceftriaxone injection 1 gram every 12 hours, and OcuFresh® eye drops, 1 drop hourly in both eyes. Wound care included wet compresses with 0.9% NaCl for 15 to 30 minutes, four times daily, followed by the application of 2% fusidic acid cream twice daily on eroded lesions after compressing.

A histopathological examination was performed on the patient using a skin biopsy specimen. The macroscopic findings revealed two irregularly shaped, grayish-white pieces of skin tissue with a firm consistency, weighing 0.05 grams in total. The dimensions of the specimens were 0.4 × 0.3 × 0.2 cm and 0.3 × 0.2 × 0.3 cm, respectively. Microscopic examination showed sections of skin tissue covered by stratified squamous epithelium with mild hyperkeratosis. The nuclei displayed morphology within normal limits. In the subepithelial region, there was perivascular inflammation characterized by lymphocytic infiltration surrounding the blood vessels. A subepidermal bulla was observed, and the stroma consisted of fibrocollagenous connective tissue.

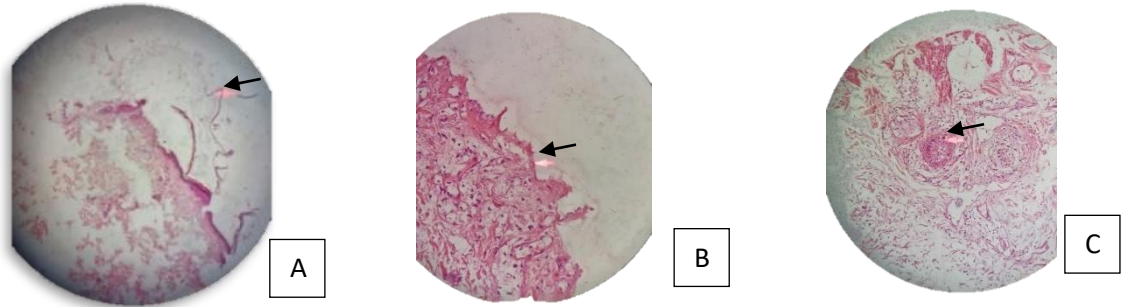

Figure 2. Histopathological findings: (A) Necrotic epidermis detached from the dermis, folded like a sheet. (B) Lymphocytic inflammatory cells surrounding blood vessels (perivascular inflammation) accompanied by hyperkeratosis. (C) Apoptosis of epithelial cells involving the hair follicles.

On the 17th day of hospitalization, the patient's rash had shown significant improvement, and the overall clinical condition had markedly improved. The lesions had begun to dry, and no new rash formations were observed. Epidermal desquamation had substantially decreased, and the patient had begun consuming solid food. Dermatological examination revealed erosions on the right orbital region, bilateral buccal mucosa, lips, and the mammary papilla. Hyperpigmented macules previously noted on the dorsal region had resolved. Laboratory blood tests performed on the 15th day of treatment showed no abnormalities. The nasogastric tube had been removed on the 12th day of hospitalization.

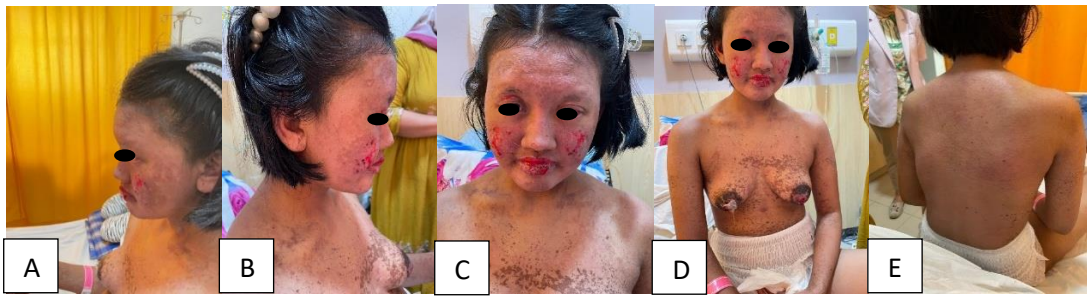

Figure 3. Day 17 of hospitalization: (A) Erosion observed on the left buccal region. (B) Erosions present on the right orbital, and right buccal regions. (C) Erosions noted on the right and left buccal regions, as well as on the lips. (D) Hyperpigmented macules observed on the thoracic region, along with erosions on the mammary papilla. (E) Previously observed hyperpigmented macules on the dorsal region were no longer present.

The patient was discharged for outpatient care on the 17th day of hospitalization. The prescribed treatment included oral methylprednisolone 4 mg three times daily for three days and tappering off based on patient's clinical impovement, followed by a tapering dose every three days; oral paracetamol 500 mg as needed for fever; and OcuFresh® eye drops, one drop every hour in both eyes. Wound care involved wet compresses with 0.9% NaCl solution for 15 to 30 minutes, four times daily, and the application of 2% fusidic acid cream to erosive lesions twice daily after the compress. The patient was advised to follow up at the Dermatology and Venereology Clinic of Adam Malik General Hospital, Medan.

DISCUSSION

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) is a life-threatening acute mucocutaneous reaction characterized by extensive necrosis and epithelial detachment (Mockenhaupt et al, 2019). SJS/TEN is a type IV hypersensitivity reaction that is caused by an immunological response to a triggering factor, most usually drugs. (Wasuwanich P et al,

2023). The primary etiology of SJS and TEN is drug exposure. Other reported causes include skin infections, although these are rare. High-risk drugs include allopurinol, carbamazepine, cotrimoxazole, other sulfonamide antibiotics, cephalosporins such as cefadroxil, macrolides, quinolones, tetracyclines, and nonsteroidal anti-inflammatory drugs (NSAIDs), among others (Mockenhaupt et al, 2019).Initial symptoms are usually non-specific and occur several days before cutaneous signs in up to one-third of cases. Early symptoms may include headaches, rhinitis, coughing, sore throat, or myalgias. Cutaneous symptoms appear quickly after a few days. Atypical targetoid macular lesions occur and frequently become confluent, advancing to vesicles and bullae, and eventually leading to full-thickness epidermal necrosis, detachment, and sloughing—all hallmarks of SJS/TEN. The injured skin is typically excruciatingly painful. The mucosal epithelium of the lips, oral cavity, oropharynx, respiratory system, gastrointestinal tract, and genitalia can become necrotic, resulting in erosions, ulcers, and detachment. (Shah et al, 2024)

Ocular involvement is common during the acute phase of SJS/TEN but may not become apparent until the later, more chronic stages. Chronic dry eyes, corneal inflammation, trichiasis, symblepharon, and keratinization of the lid edge are some of the ocular consequences. Dysosmia and dysgeusia have also been identified as consequences of mucosal involvement in SJS/TEN. Systemic organ involvement can occur through a variety of methods. Disruption of the epidermal barrier can cause homeostatic imbalance, electrolyte abnormalities, hypothermia, dehydration, and/or sepsis. Acute respiratory distress syndrome, colitis, pancreatic injury, and hepatic failure can all result from direct damage to epithelial organs. (Shah et al, 2024)Case reports or studies linking SJS/TEN to thiamphenicol use remain extremely limited. Thiamphenicol is a synthetic derivative of chloramphenicol, classified under the amphenicol group, and exhibits a broad antibacterial spectrum against both Gram-positive and Gram-negative bacteria. Unlike chloramphenicol, thiamphenicol is not metabolized into the toxic nitrobenzene compound, making it less myelotoxic.(Vasques et al, 1984) Although thiamphenicol is usually regarded safe, there have been rare instances of adverse responses to its use. Fitriana et al. reported just one instance of SJS/TEN that was thought to be caused by thiamphenicol in a pediatric patient aged 0–18 years at Dr. Soetomo General Hospital in Surabaya, between September 2016 and September 2017. (Fitriana et al., 2018). These allergic reactions are hypothesized to arise by immunological mechanisms, in which the parent chemical or its metabolites function as haptens, binding to body proteins and forming immunogenic complexes. (MIMS Indonesia, n.d.; Joint FAO/WHO Expert Committee on Food Additives, 1993). Thiamphenicol is primarily eliminated unaltered in the urine; however, about 1.5% can be converted in the liver to glucuronide conjugates. This small metabolite has the potential to cause immunological reactions in sensitive individuals. (Pirmohamed & Park, 2001).

Several research have revealed major insights into the pathogenesis of SJS/TEN. Even the particular molecular and cellular events are not totally known. The immunologic pattern in early lesions indicates a cell-mediated cytotoxic response targeting keratinocytes, resulting in extensive keratinocyte apoptosis. Immunopathological studies have revealed the presence of cytotoxic cells in early lesions, including natural killer T (NKT) cells and drug-specific CD8+ T lymphocytes; monocytes, macrophages, and granulocytes are also recruited. Three major routes contribute to keratinocyte apoptosis in SJS/TEN. The first is the interaction between Fas and Fas ligand (FasL), which causes apoptosis by attaching to particular receptors on the cell surface.(Mockenhaupt et al, 2019; Chang HC et al, 2022).The second pathway involves drug interaction with major histocompatibility complex class I (MHC-I), leading to the accumulation of cytotoxic CD8+ T lymphocytes in the bullae and the release of perforin and granzyme B, ultimately causing keratinocyte death.The third pathway involves drug-induced

activation of CD8+ cytotoxic T lymphocytes, natural killer (NK) cells, and NKT cells, which release granulysin, a potent cytolytic protein responsible for keratinocyte death (Chen KT, 2003; French LE, 2012).

No laboratory test is specific for confirming the diagnosis of SJS/TEN. However, ancillary investigations are essential for assessing severity, prognosis, and the daily management of life-threatening complications in the intensive care setting. In the present case, histopathological examination was performed. Skin biopsy for routine histologic study is highly recommended, especially when alternative diagnoses are considered. The biopsy should ideally be taken from a new lesion, preferably from the erythematous margin, rather than from a vesicle or blister, as direct sampling from blisters may result in complete separation or partial loss of the epidermis and dermis (Mockenhaupt et al, 2019).Early epidermal involvement is suggested by scattered apoptotic keratinocytes in the suprabasal layer. When the disease has progressed to the point of epidermal detachment and epidermolysis, there is subepidermal vesiculation due to severe vacuolar alterations and confluent keratinocyte necrosis. Apoptosis of epithelial cells can also impact sweat glands and hair follicles. The papillary dermis has a relatively thick mononuclear cell infiltration made primarily of lymphocytes, many of which are CD8+, and macrophages. Occasionally, eosinophilic granulocytes and varying amounts of extravasated erythrocytes may be detected. (Mockenhaupt et al, 2019).

The ALDEN score was developed to help determine drug causality in SJS/TEN. It is a retrospective tool that uses several parameters: (a) latency between initial drug intake and onset of symptoms (index day); (b) drug presence at index day; (c) prechallenge/rechallenge; (d) dechallenge; (e) drug notoriety based on incidence rates; and (f) alternative causes. ALDEN scores range from -12 to +10, categorized as follows: <0 = very unlikely, 0–1 = unlikely, 2–3 = possible, 4–5 = probable, and ≥6 = very probable (Sassolas B, 2010). In this case, the ALDEN score for thiamphenicol was 3, suggesting it as a possible culprit drug (Sassolas B, 2010).The management of SJS/TEN includes early diagnosis, immediate withdrawal of the offending drug, supportive care, and multidisciplinary treatment. Management involves protecting and restoring skin barrier function, monitoring and correcting fluid and electrolyte imbalances, treating infections, and providing nutritional support, which is crucial due to the highly catabolic state (Mockenhaupt et al, 2019; Chang HC, 2022; Siswati AS, 2024).Systemic corticosteroids in the management of SJS/TEN exhibit immunomodulatory and anti-apoptotic effects by inhibiting Fas–FasL interactions. Their anti-inflammatory properties suppress interleukin-2, tumor necrosis factor (TNF)-α, and interferon (IFN)-γ, while their immunosuppressive effects inhibit T-cell activity (Ramien M, 2020). According to the 2024 PERDOSKI guidelines, it is recommended to administer corticosteroids such as dexamethasone at an equivalent dose of 0.5–1 mg/kg/day of prednisone, adjusted based on clinical severity and total BSA involved. Pulse-dose corticosteroids may also be administered intravenously for 3–7 days, tailored to disease severity and TBSA affected (Siswati AS, 2024).A prognostic scoring system, SCORTEN, has been developed for TEN. It comprises seven independent risk factors: age >40 years, presence of malignancy, heart rate >120 bpm, skin detachment >10% TBSA, serum urea >10 mmol/L, blood glucose >14 mmol/L, and low serum bicarbonate (Mockenhaupt et al., 2019). In this patient, the SCORTEN was 1, correlating with a predicted mortality rate of 3.2%.

CONCLUSION

Stevens-Johnson Syndrome is a potentially fatal multi-organ disease with a strong etiological association with certain medications. Management involves a multidisciplinary specialist approach, immediate discontinuation of the suspected drug, administration of corticosteroid

agents, and supportive care.

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