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Case Report

A case of toxic epidermal necrolysis in a paediatric patient with mycoplasma pneumoniae infection

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ABSTRACT

Toxic Epidermal Necrolysis (TEN) is a rare, life-endangering emergency condition with a high mortality rate. While medications are recognized as common trigger factors, infections like Mycoplasma pneumoniae are also increasingly being recognized as other potential triggers, particularly in the paediatric population. A 6-year-old boy presented with fever, sore throat and tender skin lesions that rapidly progressed from erythema, macular rash to multiple vesicles, flaccid bullae and erosion, affecting more than 70% of total body surface area. Epidermal necrolysis affects more than 30% of the total body surface area including genital, oral and conjunctival mucus membranes. A diagnosis of Toxic Epidermal Necrolysis was established. Laboratory tests confirmed Mycoplasma pneumoniae infection and a chest X-ray revealed pneumonia. For management of TEN, IVIG, followed by Methylprednisolone pulses were in combination. This was combined with antibacterial therapy and supportive care. The patient made a complete recovery with no complications after 36 days under Intensive Care. This case underscores Mycoplasma pneumoniae as a paediatric TEN trigger. It also serves to highlight the efficacy of combined immunotherapy and multidisciplinary care and the importance of early intervention in minimizing complications, especially in such severe cases.

Keywords: Toxic Epidermal Necrolysis, Stevens-Johnson syndrome, Mycoplasma pneumoniae, Paediatrics, Body surface area, Critical care

INTRODUCTION

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are dermatological emergency conditions that are rare and life threatening.[1] The overall incidence rates for this condition are reported to be 1 to 10 cases in 1 million people per year; about 20% of these cases are found in children.[2] These conditions represent a spectrum of severe mucocutaneous reactions characterized by extensive epidermal removal and mucosal lesions.[3] SJS and TEN are now considered as the same disease and differ from each other by the degree of epidermal necrolysis. SJS is characterized by skin detachment less than 10% of the body surface area (BSA), SJS-TEN overlap associated with skin detachment from 10% to 30% BSA and TEN is defined as a disease with degree of epidermal necrolysis greater than 30%.[4-6]

The diagnosis of SJS/TEN mostly relies on clinical manifestations. Three phases of the pathological process are described. The prodromal phase commonly starts with nonspecific influenza-like clinical signs such as fever, headache and malaise which is followed by progressed cutaneous and mucosal impairments in second epidermolytic phase. Extensive exanthema consist of macules, target-like individual lesions without trizonal

structure (atypical target lesions), blisters, and erosions of epidermis and epithelia of mucous membranes.[6] Exanthema rapidly progress within 2-5 days (rarely takes up to 2 weeks) from a single, red tender point to a generalized skin lesion with epidermolysis.[6] Positive Nikolsky's sign (application of slight pressure on erythematous and non-erythematous areas results in epidermal detachment from underlying surfaces) is described for this disease but it is not strongly specific and can be positive for other exfoliative skin disorders.[7] Urogenital, ocular and oral mucus membranes are most frequently involved. Mucosal erosions are often very painful, resulting in reduced food intake, dysuria and photophobia. The subsequently appearing fibrinous coatings of mucosal erosions are often complicated by mucous layer adhesions that lead to function impairment. Stage of epidermal and epithelial necrosis associated with severe systemic symptoms caused by the release of large quantities of cytokines and added infection may lead to major metabolic disturbances, including sepsis, multiorgan failure, and gastrointestinal haemorrhage. Recognizing these signs early is crucial to prevent fatal outcomes. In the third phase of re-epithelization, the spread of skin detachment gradually diminishes and stops resulting in an overall improvement in the patient's condition.

Skin biopsy for conventional histological work-up and an immunofluorescent analysis to differentiate TEN from other dermatological diseases are highly recommended. There are no specific blood tests to diagnose TEN. Basic screening methods including a complete blood count, erythrocyte sedimentation rate, coagulation studies, urea and electrolytes, and liver function, blood, culture of blood, skin and mucous membranes are essential in planning antibacterial and supportive treatments and in detecting organ failure.

In general, adult cases of SJS/TEN are drug-induced whilst infections remain the main reason in the pediatric community.[4] Usually, the disease develops 1-4 weeks after drug intake. So far, the precise pathogenic mechanism is not understood. The prevailing understanding of the pathophysiology is that an immune reaction mediates the apoptosis of keratinocytes thus treatment includes immunosuppressants in addition to supportive care. The existing literature discusses several main options for treatment which includes corticosteroids (CS), intravenous immunoglobulins (IVIG) or cyclosporine although studies show conflicting results about its efficacy. In children, the literature on SJS/TEN is sparse. We present our experience with a paediatric SJS/TEN patient that responded to treatment with IVIG and corticosteroids. This case serves to demonstrate the challenges when managing and diagnosing a paediatric patient with TEN, especially when infectious and drug related triggers coexist. By bringing to attention this complex presentation and evidence-based management, we aim to contribute to the expanding understanding of paediatric TEN. Informed consent was obtained from the patient and the CARE framework was followed in the drafting of this case study.

CASE REPORT

A 6-year-old boy presented to the Regional Children's Hospital with a four-day history of fever 39-39.5°C, sore throat, malaise and a rapidly progressive rash on the skin (tender maculopapular elements that became confluent, widespread erythema, single aphthous ulcers on the oral mucosa, oedematous lips). Paracetamol has been used on an outpatient basis prior to hospitalisation with antipyretic and analgetic goals. The patient has never experienced allergic or adverse effects to any drugs. Family history of allergic diseases or dermatological conditions are absent.

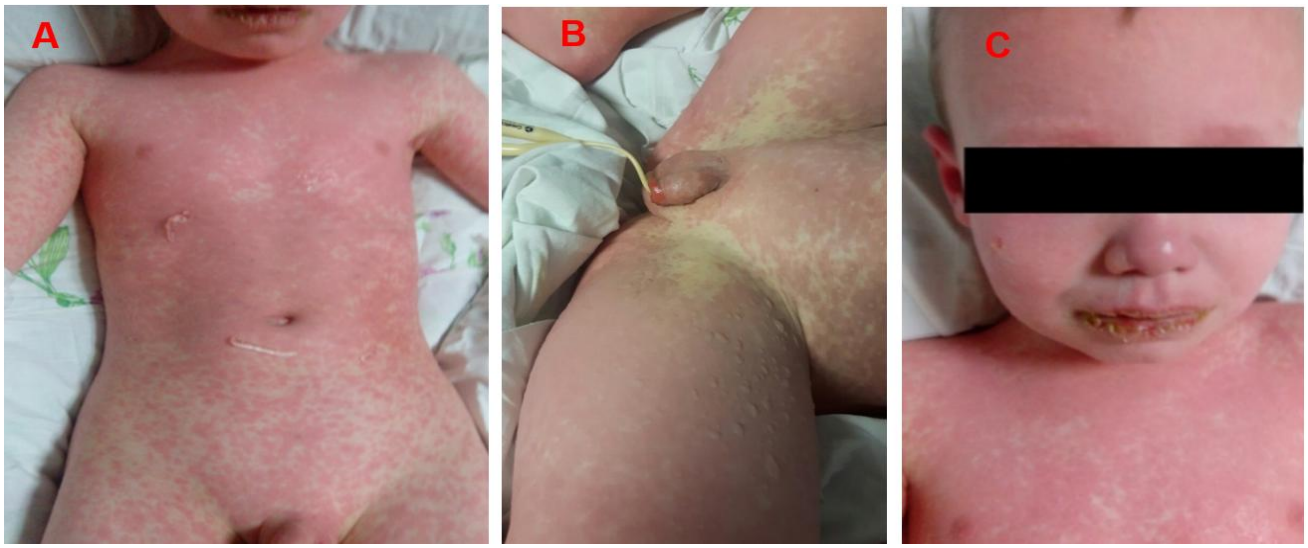

Figure 1: Mucosal and cutaneous manifestations of TEN patient diffuse erythema alongside a widespread painful, maculopapular rash on the skin of trunk, limbs (A) and genital region (B). Oedematous lips with vesicle and crust formation on them (C).

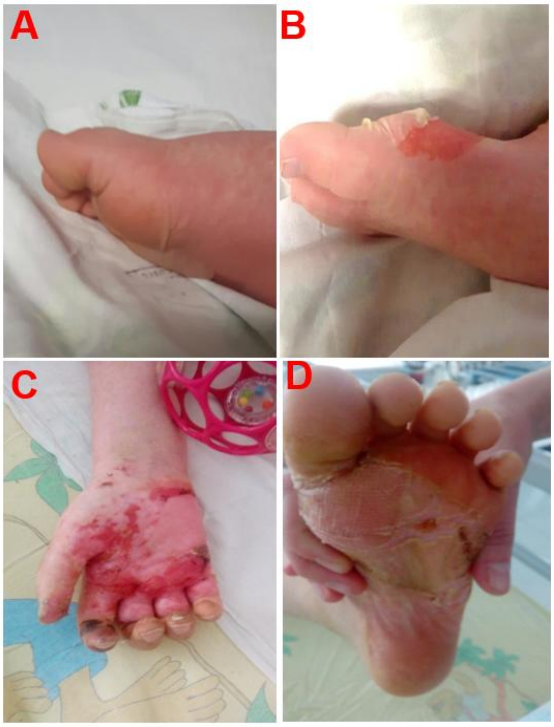

Figure 2: Bullae on the sole of the foot (A). Rupture of the blisters, leading to open, superficial erosions and epidermal detachment visible on the palms (B) and soles (C and D). Findings are consistent with >30% TBSA involvement and positive Nikolsky's sign.

On admission to the ICU the patient was conscious, oriented but distressed. The patient's initial vital signs include a fever of 38.0ºC, blood pressure of 108/55 mmHg, heart rate 150 per minute, respiratory rate 22 breaths per minute, SpO2 97% on room air and the capillary refill time was less than 3 seconds.

Physical examination revealed skin involvement about 70% of the body surface area. Skin lesions included diffuse erythema, painful macular lesions which progressed to form multiple vesicles and flaccid bullae up to 2cm in diameter. Nikolsky's sign was highly positive. Area of epidermal necrolysis was about 15-20% of BSA.[7] Ruptured bullae left painful erosions on the child's face, back and ears on (Figure 1 and 2). Upon examination of the mucous membranes, oedematous lips with crusting and small erosions, aphthous stomatitis and conjunctivitis without corneal erosions were observed. Diminished vesicular breath sounds were noted upon lung auscultation; heart sounds were clear, with rhythmic pattern, no heart murmurs. Abdomen was not distended; liver was 2 cm below the costal margin; palpation was challenging due to the skin condition. Urination was taken under control.

The main laboratory findings during the course of the disease are provided in Table 1. The patient's electrolytes, blood gas, liver function tests, creatinine, glucose levels were unremarkable. Immunoglobulin concentrations: IgG 8.69 g/l, IgM 0.91 g/l, IgA 0.6 g/l.

Table 1: The main laboratory findings the course of the disease.

	Day of hospitalization			
	1st	**3rd**	**5th**	**18th**
WBC (*10^9/l)	9.99	2.6	6.14	14.31
HB (g/l)	125	106	111	105
PLT (*10^9/l)	275	268	351	564
ESR (mm/h)	15	20	10	7
CRP (mg/l)	81.7	34.8	7.4	3.2
Procalcitonin (ng/ml)	5.2	1.3	0.3	0.25
APTT (sec/ratio)	35.3/1.18	-	24.4/0.8	23.3/0.8
INR	1.43	-	1.1	0.99
Fibrinogen (g/l)	5.03	-	2.5	1.52
D-dimer (ng/ml)	-	-	1880	-

A preliminary diagnosis of Stevens-Johnson Syndrome/ Toxic Epidermal Necrolysis (SJS/TEN) overlap disease was made based on the clinical presentation and history.

The child was put on a fluidizing air bed with a warming blanket and controlled room air temperature (Figure 3). The treatment was started with IVIG 0.9 g/kg/day, broad-spectrum antibiotic therapy (IV Ciprofloxacin 10 mg/kg/day and Linezolid 30 mg/kg/day), alongside Tramadol as an analgesic and Omeprazole with a gastroprotective goal. Infusion therapy to control hydration status with normal saline and glucose solution. Artificial tears and eye drops combined with steroids and antibiotics were prescribed.

Figure 3: Fluidizing air bed used during the treatment process.

On the 2nd day in the ICU the patient's condition showed negative dynamics. The boy was conscious but extremely anxious, any manipulations caused him intense pain. Fever was 38-38.5 ºC. Painful erosions on the oral and urethral mucosa were observed which resulted in dysuria. The patient refused oral intake of any fluid or food. The size (up to 10 cm) and number of flaccid bullae had increased considerably. Bullous elements and maculopapular lesions followed by necrosis and epidermal exfoliation were noted. Additionally, skin peeling left behind extensive, superficial erosions, with skin detachment involving around 30% of body area. The presence of eyelid oedema and crusts made it difficult for the patient to open his eyes.

In the ICU, laboratory and instrumental investigations were continued. Key haematological findings are listed in Table 1. ELISA test: *Chlamydia pneumonia* IgA 1,1 and IgM 0,1(both negative); Mycoplasma pneumonia IgA 64,1 (positive, cut off 1,1); IgM 0,7 (negative). PCR (blood) for CMV, EBV, herpes virus I, II were negative. Chest X-ray showed signs of focal right-sided pneumonia. Gram-positive cocci were found in the wound cultures. Early skin cultures were positive for *Staphylococcus epidermidis* and oral mucus membrane culture – for *Streptococcus mitis*. Later, cultures from both skin and oral mucosa were positive for multidrug-resistant *Klebsiella pneumoniae*. Blood cultures remained negative.

The diagnosis was revised to L51.2 TEN. Community acquired focal right-sided pneumonia associated with *Mycoplasma pneumonia*. Mild anaemia.

According to the child's condition, treatment was modified from the second day. IVIG 0.6 g/kg/day was continued in combination with pulses of methylprednisolone 500 mg twice a day for the next 3 days. Total dose of IVIG received was 2.2 g/kg (57g). Steroids were tapered gradually: 100 mg/day for 7 days, 2 mg/kg for 4 days, 1 mg/kg for 4 days, 0.5 mg/kg for 2 days, and then discontinued. Antibacterial and antifungal therapy was maintained: IV linezolid (30 mg/kg/day), ciprofloxacin (later replaced after 10 days with colistin 120,000 IU/hg/day) and flucanazole (6 mg/kg/day). A prolonged infusion of diazepam was started for sedation and morphine for pain management (tramadol was discontinued). To control hemodynamic status, we used dopamine 5-10 mcg/kg/min. The bladder was catheterized to control urine output and prevent mucosal adhesion. Enteral feeding via a nasogastric tube was initiated and partial parenteral feeding for a short period was given as well.

By day 5-6, in the ICU, the patient's condition had stabilized. Skin and mucosal lesions had began to regress. Epidermal detachment halted, Nikolsky's sign became negative. Small vesicles (up to 3 mm) persisted for 6-8 days more. Necrotic foci appeared on large erosive surfaces (Figure 4). Intensity of pain decreased but the need for narcotic analgesics was present for the remainder of the 2 weeks in the ICU. In next 10-12 days, small vesicles and necrotic tissues had progressed to crust formation, followed by sloughing and re-epithelialization. The patient stayed in the ICU for 21 days and then was referred to the Department of Surgery to continue wound care (Figure 5).

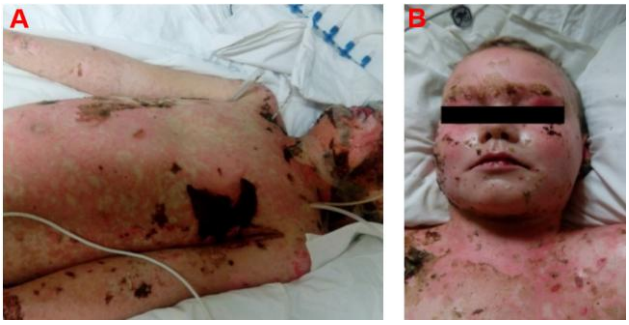

Figure 4 (A and B): Necrosis of the eroded skin surfaces. Further exfoliation of skin has already stopped, and the intensity of pain had reduced at this point in this patient.

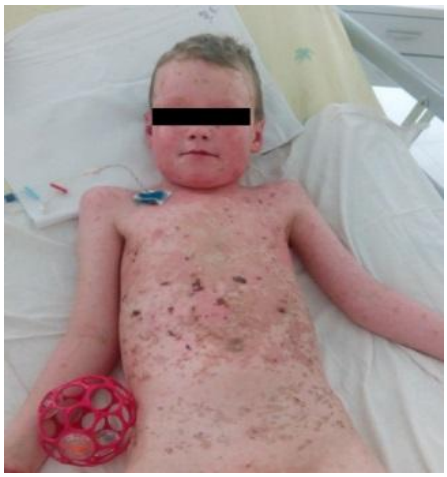

Figure 5: Overall improvement seen in the cutaneous and oral manifestations of TEN (20th day of treatment in the ICU).

Skin wound care included: sterile handling, repeated debridement of exfoliating skin, removing necrotic epidermis, using topical antiseptic agents (chlorhexidine bigluconate 0,05%) to wash denuded areas. Non-adherent petrolatum-impregnated gauze materials to cover the denuded skin to minimize infection risk (left for 3-5 days) and hydrocortisone with oxytetracycline aerosol were applied on eroded areas of skin. It should be noted that there is no standard approach for wound care. Blood crusts from ocular, nasal, and oral mucous membrane were cleaned up regularly to prevent adhesion.

The child was evaluated by an ophthalmologist from 1st day of admission. Ocular care included saline rinses, eyes drop with 0.1% of dexamethasone and broad-spectrum antibiotics (moxifloxacin hydrochloride 0.5%, gentamycin sulphate 0.3%); artificial tears and Dexpanthenol containing gel as a lubricant to enhance healing and

minimize scarring. The ocular changes regressed completely.

The patient was discharged on day 36 in a stable condition with proper instructions regarding a possible relapse. During the 1-year follow-up, there was no recurrence of symptoms, and the patient was doing well with complete resolution of the rash. He has been advised to avoid all NSAIDs.

DISCUSSION

This case report depicts the complexities in diagnosing and treating a paediatric patient with TEN, illustrating the interplay between infectious aetiologies, immunomodulatory therapy and multimodal treatment strategies for optimal care. This patient's presentation clearly aligned with the well-established triad of fever, signs of systemic toxicity and mucosal involvement together with epidermal necrolysis that defines TEN.[8]

While medications are one of the most common triggers, infectious agents such as *Mycoplasma pneumoniae* are also increasingly being recognized as triggers in paediatric patients.[3,9,10] In this case, the high levels of IgA to Mycoplasma to a recently acquired infection, likely contributing to immune dysregulation via cross-reactive antibodies or cytotoxic T-cell activation.[11] Additionally, paracetamol administered prior to hospitalization cannot be excluded with confidence as a trigger either, though the patient did not have any prior drug hypersensitivity. This illustrates the importance of thorough infectious workups in paediatric TEN patients even when drug exposure is reported.[12]

There is no proven effective pharmacotherapy so far. All the reported clinical data regarding the use of steroids, cyclosporine and VIG are mainly based on observational studies. Initial use of IVIG is in accordance with guidelines suggesting early administration to neutralize Fas-ligand-mediated keratinocyte apoptosis.[13] IVIG showed encouraging results when started within the first 2-3 days. Systemic steroids are commonly used to halt the progression of skin detachment in TEN cases. Although corticosteroid use in TEN patients is a controversial topic due to sepsis risk, when initiated early their use in preventing disease progression is supported by recent paediatric studies, particularly in patients with more than 30% body surface area detachment.[14,15]

Infection is common in such patients and may be lethal. Staphylococcal infections are the most common, followed by pseudomonas infection, which occurs after a prolonged hospitalization. In patients with burns, extensive skin detachments are associated with immunological barrier impairment and large fluid losses which results in a high risk of severe infection. Thus, our patient was initially put on aggressive antimicrobial therapy with Linezolid and Ciprofloxacin empirically due to the pneumonia and gram-positive cocci in wound cultures. However, due to the risk of contracting hospital-acquired infections as a result of epidermal loss, the antimicrobial treatment was adjusted to Linezolid together with Colistin. Procalcitonin elevation to 5.2 ng/ml and leukopenia (WBC 2.6 x 109 /l) suggests a systemic inflammation, further highlighting the need for vigilant monitoring to monitor infections in TEN patients.

The clinical presentation of TEN is characterized by signs overlapping with other conditions such as SJS, SSSS (Staphylococcal scalded skin syndrome), TSS (Toxic shock syndrome) and DRESS (Drug Reaction with Eosinophilia and Systemic Symptoms).[16] In our case, the diagnosis was made clinically based on 70% BSA involvement with more than 30% of BSA skin detachment which absent in SSSS and often minimal in TSS.[17] TSS due to staphylococcal infection was further excluded due to absence of multiorgan system involvement (GI symptoms absent, normal renal and liver function, platelet counts were more than 100,000/mm, neurological signs absent).[18] Additionally, onset of desquamation occurred within days of rash onset, unlike the 1-2 weeks of delayed peeling in TSS patients. Similarly, DRESS was excluded as the patient lacked multisystem involvement and normal eosinophil levels.[19]

CONCLUSION

This report reaffirms Mycoplasma pneumoniae alongside paracetamol as a possible TEN trigger in children and demonstrates the use of combined treatment strategies, utilizing multiple immunosuppressive therapies such as IVIG and steroids in severe cases. It further illustrates the critical position supportive care takes in children, especially when it comes to ocular and wound management, in order to reduce morbidity. The successful outcome in the patient, despite the severity of his condition emphasizes the importance of multidisciplinary care, early initiation of immunosuppressive and antimicrobial treatment as well as aggressive supportive management. Further research into this topic is needed to establish specific paediatric treatment algorithms relying on severity scoring, which would help to greatly improve prognosis of paediatric patients.

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