

## Case Report

# Single dose amoxicillin induced toxic epidermal necrolysis – A rare life threatening dermatological condition

Sunil Pathak<sup>1</sup>, Mukesh Nagar<sup>1</sup>, Mahendra Kavad<sup>2</sup>, Harshil Patel<sup>2\*</sup>, Kalpic Patel<sup>2</sup>, Maitray Patel<sup>2</sup>

<sup>1</sup>Assistant Professor, <sup>2</sup>PG Student,

Department of Pediatrics, SBKS MI & RC, Sumandeep Vidyapeeth, Vadodara, Gujarat, India

\*Corresponding author email: [hmpatel52@gmail.com](mailto:hmpatel52@gmail.com)

	International Archives of Integrated Medicine, Vol. 3, Issue 4, April, 2016. Copy right © 2016, IAIM, All Rights Reserved. Available online at <a href="http://iaimjournal.com/">http://iaimjournal.com/</a> ISSN: 2394-0026 (P) ISSN: 2394-0034 (O)	
	Received on: 12-03-2016 Source of support: Nil	Accepted on: 02-04-2016 Conflict of interest: None declared.
<b>How to cite this article:</b> Pathak S, Nagar M, Kavad M, Patel H, Patel K, Patel M. Single dose amoxicillin induced toxic epidermal necrolysis – A rare life threatening dermatological condition. IAIM, 2016; 3(4): 187-190.		

## Abstract

Toxic epidermal necrolysis is a severe cutaneous drug reaction, mainly involving skin and mucous membranes. We have presented here a case report of 16 year old female patient who was diagnosed with toxic epidermal necrolysis clinically as well as histopathologically. The patient presented with history of fever, oral cavity ulcer, skin lesions which were ill defined, erythematous macular with darker hemorrhagic centre. Skin biopsy showed full thickness necrosis of dermis, dermal-epidermal separation and paucity of upper dermal cellular infiltration. The patient was treated with intravenous immunoglobulins, low dose steroids and systemic antibiotics and the patient improved significantly. The main purpose of the case report was to emphasize that even one single dose of amoxycillin can induce a toxic epidermal necrolysis.

## Key words

Amoxicillin, cutaneous lesions, Toxic epidermal necrolysis, Skin biopsy.

## Introduction

Toxic epidermal necrolysis is severe adverse cutaneous drug reactions that predominantly involve the skin and mucus membranes. The incidence of toxic epidermal necrolysis is 0.9 to

1.4 persons per million per year in general population [1]. It is characterized by mucocutaneous tenderness and typically hemorrhagic erosions, erythema and severe epidermal detachment presenting as blisters and

area of denuded skin. Diagnosis relies mainly on clinical signs together with the histological analysis of a skin biopsy showing typical full-thickness epidermal necrolysis due to extensive keratinocyte apoptosis. Here we are presenting a 16 years old female child presented with fever followed by skin lesion all over the body after taking single dose of amoxicillin.

### **Case report**

A 16 years old female patient, 2<sup>nd</sup> out of two siblings, product of non-consanguineous marriage, admitted with history of fever, oral ulcer, for which she took single dose of amoxicillin after that she developed skin lesions all over body with in next 2 days. Lesions were ill-defined erythematous macular lesions with darker hemorrhagic centre present all over the body. Area of confluent erythema along with fluid filled bullae was present over palms (**Photo – 1**). Nikolsky's sign was positive; multiple oral and genital ulcers were also present. Lesions also involved the eye in form of conjunctivitis, blepharitis and keratitis. Except the skin lesions rest examination was normal. Laboratory investigation were Hb- 9.3 gm%, TC- 4000, DC- 80/14/3/3, platelet- 1.55 lakhs, INR- 1, serum electrolytes, renal function test, liver function test were normal. Skin biopsy showed full thickness necrosis of dermis, dermal-epidermal separation and paucity of upper dermal cellular infiltration (**Photo – 2**). Clinical and histopathological findings are suggestive of toxic epidermal necrolysis and we treated the patient with intravenous immunoglobulin 1 gm/kg/day for 3 days, low dose steroids, systemic antibiotics along with other supportive care. With treatment, Symptoms got completely relieved only healing scars were present on discharge (**Photo – 3**).

### **Discussion**

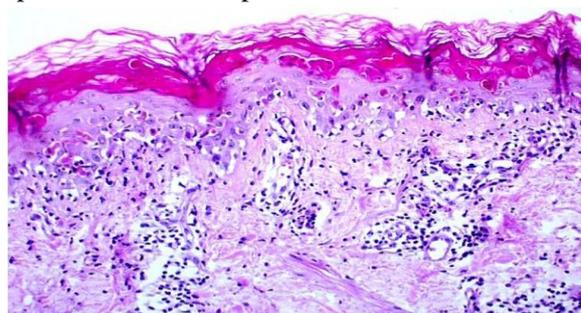
Toxic epidermal necrolysis is severe adverse cutaneous drug reactions that predominantly involve the skin and mucous membranes. The pathogenesis of TEN is not fully understood but is believed to be immune-mediated. TEN is specific drug hypersensitivity reactions in which

cytotoxic T lymphocytes (CTL) play a role in the initiation phase. In the early phase of disease, blister fluid contains mainly cytotoxic CD8+T lymphocytes [2, 3], suggesting that a major histocompatibility (MHC) class-I restricted drug presentation leads to clonal expansion of CD8+ CTLs, and the subsequent immune reaction that causes TEN. Cytotoxic T lymphocytes can induce the cascade either through the perforin/granzyme or the “death receptor”, Fas-Fas ligand (FasL) pathway [4]. Groups of drugs that cause TEN are [1] antibiotics include sulphonamides [5], penicillins, cephalosporins [2], NSAIDs [3], anticonvulsants etc.

**Photo – 1:** On admission.



**Photo – 2:** Skin biopsy shows necrosis of the epidermis and sub-epidermal blisters.



In TEN prodromal symptoms are often severe and include nausea, vomiting, high fever, malaise, headache, sore throat and painful skin. Skin lesions include severe mucous membrane with generalized epidermal sloughing. The initial lesions are ill defined, dusky erythematous macules with darker purpuric centres. Lesions appear symmetrically on face, upper trunk and

lower trunk. Nikolsky's sign is positive. There is >30% cutaneous surface epidermal detachment in TEN. Mucous membranes involvement include oropharynx, eyes, genitalia and anus, crusted lips, increased salivation, redness of eyes with photophobia. Serial ophthalmic examination required for ocular lesions. There may be synechiae between the eyelids and conjunctiva. There may be mucopurulent conjunctivitis; keratitis and conjunctival erosions have high risk of sequelae.

**Photo – 3:** On discharge.



Diagnosis is mainly based on combined approaches of clinical and histopathological findings. The skin biopsy helps in confirm the diagnosis. Routine investigation include CBC, RBS, renal function tests, serum electrolytes, liver function tests, blood culture, HIV should be done to rule out other possible infections.

The management of TEN include immediate hospitalization and treatment on an emergency basis. The main principles of treatment mainly base on symptomatic treatment include

analgesia, fluid replacement, and Anti-infectious therapy, aggressive nutritional support, warming of environmental temperature and skin care with appropriate dressings. The causative drug should be identified and discontinued immediately. Analgesic should be given to relieve pain, in fluid replacement; fluid is calculated by parklands formula. RL is used for it. Temperature should be maintained at 30-32 °C to reduce caloric losses through the skin. Broad spectrum antibiotics should be given to minimized systemic infection. Steroids should be initiated during the initial stage and rapidly tapered off as it may increase risk of infection. Intravenous immunoglobulin (IVIG) has anti-Fas (CD95) activity cause inhibition of keratinocytes apoptosis [6]. IVIG should be started within 48-72 hours of onset; dose should be 0.4mg/kg/day for 5 days. Ophthalmic care should also be taken by instillation of eye drops.

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