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RESEARCH ARTICLE

TOXIC EPIDERMAL NECROLYSIS INDUCED BY AMOXYCILLIN: A RARE CASE REPORT AND REVIEW OF LITERATURE

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ABSTRACT

Adverse drug reactions with any class of drugs are responsible for increased hospitalization. The incidence is more with antibiotics. Drugs indicated allergic reactions are labelled as IgE-mediated and non IgE-mediated hypersensitivity reactions. IgE mediated ones include angioedema, anaphylaxis, bronchospasm and urticarial including toxic epidermal necrolysis (TEN). Non IgE mediated hypersensitivity reactions include serum sickness, intersitiation nephritis, haemolytic anemia, thrombocytopenia, erythema multiforme. Amoxicillin is a broad spectrum antibiotic used to combat various infections. TEN is defined as an extensive detachment of full thickness epidermis which is a manifestation of adverse drug reaction. TEN is a rare but severe drug induced cutaneous reaction. Majority of the cases of TEN are drug induced especially anticonvulsants, antibiotics and non steroidal anti-inflammatory drugs (NSAIDS) etc. a case of TEN is reported here in a 25 years old male, complaining of sore throat for which capsule amoxicillin was prescribed. The diagnosis of TEN was made on history and clinical examination and the patient was successfully treated with parentral antibiotics, antihistamines, corticosteroids and immunoglobulins.

INTRODUCTION

Toxic epidermal necrolysis (TEN) is a rare but potentially life threatening serious disorder, characterized by more than 30% skin detachment with average 25-35% mortality[1]. TEN was originally described by Debre et al in 1939 in French as l'erythrodermiebulle uses avec epidermolyse[2]. Alan Lyell in 1956 described TEN as an eruption resembling scalding of the skin[3]. The average annual incidence of TEN worldwide is 0.4-1.3 cases per million population[4].

Case Report

A 25 years old male presented with history of sore throat for which he was prescribed capsule amoxicillin 500 mg bd x 4 days along with tablet paracetamol 500 mg x 4 day. After 2 days of medication, he developed bilateral skin lesions in the form of rashes involving both hands, which further progressed to involve neck, chest, back, abdomen, upper and lower extremities. He complained of burning sensation all over the lesions. On examination, the rashes were typical edematous, pruritic, spreading all over the body with purulent discharge, local bleeding and crusting with peeling of the skin on the back. Systemic examination and laboratory profile revealed no abnormality. Based on history and clinical examination, he was diagnosed as a case of TEN following which amoxicillin and paracetamol were stopped immediately. Patient was treated with parenteral antibiotics, corticosteroids, antihistamines, IV fluids, emollients and IV immunoglobulins 15 gm daily for 3 days. He was discharged in a satisfactory condition with advice to continue topical antibiotics and oral prednisolone in tapering doses. On follow up at 2 weeks, he was doing well and was given an Alert Card to avoid various medications in future.

DISCUSSION

Most commonly observed drug reactions are cutaneous drug reactions in 65-80% of cases[5]. Hypersensitivity reaches with the use of cephalosporin (e.g. amoxicillin) occur in 1-3% of patients. TEN is considered a severe form of erythema multiforme spectrum and is IgE mediated hypersensitivity reaction. TEN is triggered by various drugs used over a short period e.g. trimethoprim sulfamethaxzole, cephalosporins, quinolones, sulphonamide antibiotics, carbamazepine, phenytoin, valproic acid, phenobarbital, aminopenicillins, corticosteroids, allopurinol apart from other conduction including systemic lupus erythematosis, HIV, graft versus host disease, radiotherapy, bacteria, viruses, fungi, immunisation and idiopathic[6]. The pathogenesis of TEN is poorly understood. But the hypothesis is that drug induced hypersensitivity reaction causes keratinocyte cell death

occurring as a result of role played by CD8 and cytotoxic T lymphocytes, fatty acid synthetase (FAS) and fatty acid synthetase ligand (FASL). This ultimately results in epidermal detachment and sloughing[7]. Apoptosis also plays a role in keratinocyte cell death[8]. The onset of symptoms following initial drug administration varies from few hours to 3 weeks but symptoms reappear after re-challenge within 48 hours or less in an acquired immune response[9]. Once the diagnosis is made, the offending drug must be stopped immediately with an aim to reduce mortality. Higher antibiotics are used in TEN patients in view of dreadful complication of sepsis. Successful therapeutic management includes parenteral administration of glucocorticoids, cyclosporine, N-acetylcysteine, plasmophresis, thalidomide, anti-TNF alpha, hemodialysis, pentoxyfylline, IVIG[10], although none of the pharmacological agent has conclusively been shown to be beneficial as treatment modality.

CONCLUSION

TEN is rare, serious systemic disorder commonly associated with adverse drug reaction. The aim of presenting this rare case of TEN is to emphasize efficient pharmacovigilance so that occurrence of adverse drug reactions can be reduced and prevented. For this proper history must be taken before prescribing drugs like antibiotics, anticonvulsants, NSAIDS and pyrazoles. Reporting of such events is the need of hour. These offending drugs must also be avoided in family members of the patient in view of occurrence of genetic susceptibility to TEN.

References

1. French LE. Toxic epidermal necrolysis and Stevenson Johnson syndrome: our current understanding. *AllergolInt* 2006; 55: 9-16.
2. Debre R, Lemy M, Lamotte M. L'erythrodermicbulleuses area epidermolyse. *Bull SocPediatr (Paris)* 1939; 37: 231-8.
3. Lyell A. Toxic epidermal necrolysis: an eruption resembling scalding of the skin. *Br J Dermatol* 1956; 68: 355-61.
4. Abood GJ, Nickoloff BJ, Gamell RL. Treatment strategies in toxic epidermal necrolysis syndrome: where are we at ?. *J Burn Care Res* 2008; 29(1): 269-76.
5. Wolkenstein P, Revuz J. Drug induced severe skin reactions. Incidence, management and prevention. *Drug Saf* 1995; 13: 56-68.
6. Auquier Dunant A, Mockenhaupt M, Naldi L, Correia O et al. Severe cutaneous adverse reactions: correlations between clinical patterns and causes of erythema multiformemajus, Stevens-Johnson syndrome and toxic epidermal necrolysis: results of an international prospective study. *Arch Dermatol* 2002; 138(8): 1019-24.
7. Miyanchi H, Hosokawa H, Akeda T, Iba H, Asada Y. T-cell subsets in drug, induced, toxic epidermal necrolysis: possible pathogenic mechanism induced by CD 8 + T cells. *Arch Dermatol* 1991; 1276: 851-5.
8. Paul C, Wolkenstein P, Adle H. Apoptosis as a mechanism of keratinocyte death in toxic epidermal necrolysis. *Br J Dermatol* 1996; 134(4): 710-4.
9. Revuz J, Penso D, Roujeau JC. Toxic epidermal necrolysis. Clinical findings and prognostic factors in 87 patients. *Arch Dermatol* 1987; 123(9): 1160-5.
10. Paquet P, Pierard GE, Quatresaoz P. Novel treatments for drug induced toxic epidermal necrolysis (Lyell's syndrome). *Int Arch Allergy Immunol* 2005; 136(3): 205-16.

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