Case Report

Paracetamol induced fixed drug eruptions: case report

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INTRODUCTION

Paracetamol also known as acetaminophene is one of the most popular and most commonly used analgesic and antipyretic drugs around the world and available without a prescription, both in mono- and multi-component preparations. It is a drug of choice in patients in whom application of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are contraindicated, e.g., in the case of gastric ulcers, hypersensitivity to aspirin, impairments in blood coagulation, in pregnant women, nursing mothers and children with fever accompanying a disease.¹ The central analgesic action of paracetamol is like aspirin, i.e. it raises pain threshold, but has weak peripheral anti-inflammatory component.² Paracetamol may act by inhibiting COX-3 in CNS, which is involved in pain perception and fever but not in inflammation. Another explanation for its lack of anti-inflammatory action is that it poorly inhibits COX-1 in the presence of superoxides which are generated at inflammatory sites by leukocytes.³ Paracetamol is generally well tolerated at recommended dose. Prolonged daily use can leads to serious side effects like kidney or liver damage,⁴,⁵ stomach bleeding.⁶ Hypersensitivity⁷,⁸ (anaphylaxis⁹,¹⁰ and fixed drug eruptions¹¹) and Dermatological side effect like erythematous skin rashes associated with paracetamol have been reported but are rare. We have reported a case of bullous fixed drug eruption which ulcerates after two days.

CASE REPORT

2 year male child male child wt. 12 kg, presented to paediatrics OPD with complain of fever and cold. He was prescribed with paracetamol syrup. Next day child presented again with complain of severe itching. Itching began from evening after second dose over both the hands and chest followed by burning sensation and the subsequent development of multiple fluid filled purplish lesions. He also developed erosions over the lips. He had no significant past medical or surgical history. There was no history of any other oral drug intake. His physical examination revealed multiple purplish-livid bullae over
the fingers of both the hands and over the chest. Erosions with crusting were evident over his lips. He was afebrile and his vitals were within the normal range. There were no typical viral rashes and systemic examination was normal. The diagnosis of bullous fixed drug eruption was made on clinical finding and histopathological findings were consistent with those of fixed drug eruption with interface dermatitis along with vacuolar changes and Civatte bodies. The dermal inflammatory infiltrate consisted of eosinophils, a few neutrophils along with dermal oedema. Along with this he had past history of a similar but milder reaction to the drug 6 months back which he had taken for upper respiratory tract infection. Our provisional diagnosis was supported by literature review which revealed the presence of fixed drug eruption due to paracetamol. On reintroduction of paracetamol, eruptions increases. He had no genital lesions. The FDE was managed with syrup prednisolone tapered over 10 days, I.V cefotaxime, for fever syrup mefanemic acid, I.V fluids and fusidic acid + betamethasone cream. The assessment showed probable (Score-6), moderate (Level-4) and preventable type of ADR as per Naranjo algorithm, Hartwig scale and modified Schumock and Thornton scale respectively.

**DISCUSSION**

A drug-induced reaction should be considered in any patient who is taking medications and who suddenly develops a symmetric cutaneous eruption. Fixed drug eruptions may account for as much as 16-21% of all cutaneous drug eruptions. FDE represents a unique CDR pattern characterized by skin lesion(s) that recur at the same anatomic site(s) upon repeated exposures to an offending agent. Most commonly, the skin lesion is a dusky erythematous macule and is usually found on the extremities, lips, genitalia and perianal areas, although any skin or mucosal surface may be involved. The skin lesions may be associated with a burning sensation and may be present in multiple numbers or progress to the development of central vesicles and bullae, particularly after the repeated use of an agent. The offending drug is thought to function as a hapten that preferentially binds to basal keratinocytes, thereby releasing lymphokines and antibodies thus damaging the basal cell layer. The exact pathogenesis of FDE is unknown. According to one hypothesis FDE is classified as a type IVc immunologic reaction because of latent cytotoxic T cells in the lesions, which may become reactivated. There is also an association with HLA class I antigens, suggesting that there may be a genetic predisposition to these reactions. There are very few cases of fixed drug eruption due to paracetamol are reported. One strongly suspected case (Savin, 1970) was reported from St John’s Hospital. This was a patient who developed a fixed drug rash after taking a chloromezalone-paracetamol combination. However, no other reports of a fixed drug rash which was proved to be due to paracetamol have been found.

**CONCLUSION**

Paracetamol is widely prescribed by physician as well as also a popular OTC drug. Physician must suspect if such reaction occurs during therapy involving paracetamol and should carefully evaluate drug-associated reaction. It is important that skin reactions are identified and documented in the patient record and patient should be explained properly not to use that drug so that their recurrence can be avoided in future.

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