INH Induced Lichenoid Eruptions

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A 15 year old boy admitted with fever for 10 days associated with altered sensorium. Clinical findings and relevant investigations (including CSF study) were consistent with the diagnosis of tubercular meningitis (TBM). He was put on oral corticosteroid and CAT-I antitubercular drugs (ATD) (Thrice weekly dosage of INH: 600mg; Rifampicin: 450mg, Ethambutol: 1200mg, Pyrazinamide: 1500mg) [Day 1]. The patient improved clinically and was discharged on 10th post-admission day with ATD and tapering dose of oral corticosteroid.

About 6 weeks after discharge the patient presented with multiple brown black eruptions over dorsal aspects of palms and soles (Figures 1 and 2) [Day 42]. The lesions were mildly pruritic. Punch biopsy of the lesion revealed marked hyperkeratosis, papillomatosis and acanthosis of epidermis. The basal layer showed patchy vaculopathy. The dermal papillae showed prominence of blood vessels with lymphocytic infiltrate in perivascular areas. Melanin laden macrophages were also seen in the papillary dermis. The overall histopathological features were consistent with lichenoid tissue reaction (LTR) (Figure 3a and 3b).

Studies have shown that the incidence of Pyrazinamide-induced rash during treatment for active tuberculosis was substantially higher than with the other first-line ATDs. However, INH induced cutaneous adverse drug reactions (CADR) are now being increasingly reported.

As the boy was having TBM and was on the verge of completing the intensive phase of therapy we decided to continue with all the four ATDs. Pyrazinamide and Ethambutol were withdrawn after 2 weeks and the patient entered into the continuation phase [Day 56]. However, fresh lesions continue to appear and morphologically similar lesions were found over the scrotum, glans penis, peri-oral region (Figure 4) and buccal mucosa.

INH was withdrawn and Ofloxacin was added to Rifampicin [Day 77]. New lesions stopped appearing and the existing eruptions responded completely to 2 weeks [Day 91] of oral prednisone 25 mg daily, which was tapered to 1 mg over 3 months [Day 181] and then stopped.

A diagnosis of INH induced LTR was considered. The causality analysis for INH and the lesions was suggestive of adverse drug events [Probable/likely on WHO-UMC causality assessment scale and probable (total score 5) on Naranjo probability scale]. CADR is one of the commonly observed major adverse events of ATDs. It includes morbilliform rash, erythema multiforme syndrome, urticaria, and rarely exfoliative dermatitis or lichenoid eruption. LTR constitute less than 10% of the total incidence of first line ATD induced CADR. Incidence rate of INH induced CADR is about 0.98%.2

INH, though rare, can cause LTR.3,4 It still remains unclear how photosensitive LTR are induced, but allergy, including delayed type allergy may play a role. An autoimmune attack by T cells on the epidermis seems to be the primary pathological event in the development of LTR. To conclude, side effects to ATDs are common and unusual adverse effects must be recognized early, to reduce associated morbidity and mortality. Though unusual, INH may be the causative agent in different CADRs and it should always be considered while confronting drug induced cutaneous eruptions.

References


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