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SHORT COMMUNICATION

Lichen Planus Possibly Induced by Acyclovir in a Child

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Lichen planus (LP) represents an inflammatory disorder of the skin and mucous membranes, typically characterized by pruritic, purple papules and plaques (1). LP is an uncommon presentation in childhood. Acyclovir belongs to a group of synthetic drugs called nucleoside analogs used mostly for herpes simplex and zoster infection and is usually well tolerated with few cutaneous adverse effects (2). We present a child with life-long history of atopic dermatitis developing LP following the administration of a course of acyclovir. His skin eruption settled completely following cessation of acyclovir. To our knowledge, this is the first reported case of acyclovir induced LP in a child with atopic dermatitis (AD).

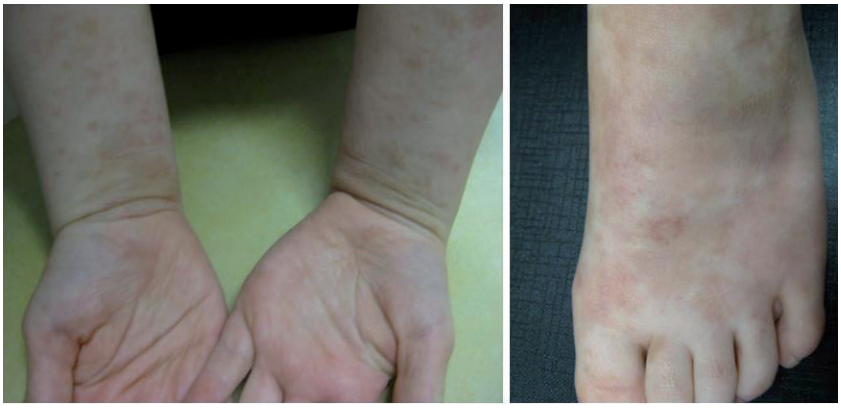

Fig. 2. Post inflammatory hyperpigmentation after clearance of lichen planus at week 8.

CASE REPORT

A 4-year-old boy with longstanding AD developed an infected exacerbation of AD. This was treated with a course of oral flucloxacillin 125 mg 4 times a day for one week and topical clobetasone butyrate twice a day. Following a lack of response after 3 weeks, he was prescribed oral acyclovir 10 mg/kg 4 times a day for 5 days by his general practitioner for possible eczema herpeticum. Seven days later, he started to develop pruritic skin eruption on his limbs and scalp (Fig. 1). This did not respond to topical betamethasone valerate and continued to get worse. Otherwise the child was well with no history of recent vaccination and was not no regular medication. At this stage, he was referred to our dermatology department for assessment.

Clinical examination showed diffuse violaceous papules and plaques on his hands, wrists feet, legs and scalp. There was no involvement of the trunk, oral cavity, nails or genital area. A clinical diagnosis of LP was made with acyclovir as a likely culprit. A skin biopsy was performed and histology results showed florid lichenoid inflammation associated with saw toothed acanthosis of the epidermis, wedge shaped hypergranulosis consistent with typical LP.

He was treated with topical mometasone furoate for two weeks. His skin cleared over the following 4 weeks with post inflammatory hyper-pigmentation (Fig. 2). The child remains symptoms free after one year with no recurrence of LP.

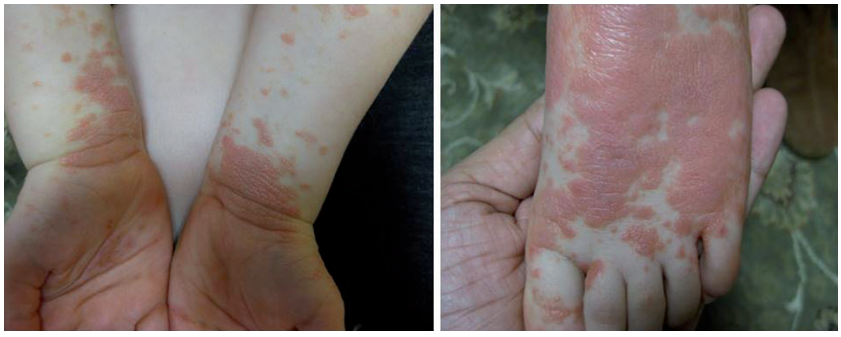

Fig. 1. Classical lichen planus with voilaceous papules and plaques on wrist and feet.

DISCUSSION

While AD is one of the most common dermatosis, LP on other hand is rare in children. Review of the literature showed no reported cases of co-existing AD and drug induced LP. LP and lichenoid drug eruption (LDE) are similar skin diseases (1), with reports of LP to be less common and more severe in children than in adults (3). Clinical and histological findings in LP can resemble LDE, the diagnosis of LDE is supported by a full drug history and resolution of symptoms following medication withdrawal (1, 3, 4). There is no good clinicopathological correlation to help distinguish LDE from LP, but histologically, apoptotic keratinocytes were observed in greater number and in clusters in LDE, as compared with LP lesions (1). LDE often spares the oral mucosa with extensive symmetrical distribution involving the limbs and the same pattern was seen in our case (4).

Most cases of LP are idiopathic, with few reports related to medication include beta-blockers, antidepressants, antipsychotics, anti-malarial, anticonvulsants and non-steroidal anti-inflammatory drugs (5). There

are no reported cases of flucloxacillin or acyclovir inducing LP in the literature but 2 cases were notified to FDA with acyclovir causing LP (2).

This child had taken flucloxacillin on a few occasions in the past with no adverse events. Although there could be a possibility that the child developed a new sensitization to flucloxacillin as there have been a few reported cases of new sensitization to penicillin (2). In our case, the suggestion of a causal role of acyclovir causing LP is mainly due to chronological timing of acyclovir. The treatment of drug-induced LP should include withdrawal of the culprit drug and symptomatic relief with topical steroids. In certain cases, it may not be possible to withdraw the suspected medication and treatment options may include systemic steroids and phototherapy which may alleviate symptoms (6). Most cases of LP reported in children have shown full clearance, while recurrence is rare as in our case (7). In conclusion we report a case of LP with acyclovir and doctors should be aware of this possible adverse effect.

The authors declare no conflict of interest.

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