Bullous dermatosis of childhood induced by gemfibrozil

Sir,

Linear IgA bullous dermatosis (LABD) is an acquired, auto-immune, sub-epidermal, vesiculobullous disease caused by the linear deposition of IgA in basement membrane zone (BMZ).[1] It may be idiopathic or drug-induced, although its pathology is not clearly understood.[2] Its manifestations are similar to those of other blistering diseases, such as dermatitis herpetiformis or bullous pemphigoid. In contrast, these blistering diseases are commonly induced by drugs.[3] LABD represents bimodal age of onset. However, it is more common in the adult because this group is often being treated for multiple medical conditions.[4]

There are several reports describing the association of LABD with various drugs.[5] Drug-induced LABD should be suspected when the drug eruption resembles erythema multiforme, dermatitis herpetiformis, or bullous pemphigoid, and vancomycin is one of the more common inducers of LABD.[6] Other drugs include lithium, captopril, penicillins, cephalosporins, nonsteroidal anti-inflammatory drugs (diclofenac and naproxen), oxaprozin, and so on.[6]

A 13-year-old girl with no previous history of skin disease was treated for 3 weeks with gemfibrozil for familial hypertriglyceridemia. 3 weeks earlier, some circumferential vesicles and tens bullae as annular erythema were symmetrically noted on the areola of breasts [Figure 1].

The lesions were pain-free. Personal and familial history was unremarkable, except for familial hypertriglyceridemia. The nails, mucosae, and hair were normal. All laboratory data were within normal limits. A biopsy specimen revealed sub-epidermal bullae, with the presence of neutrophils in the underlying dermis and few eosinophils. Direct immunofluorescence revealed linear deposits of IgA and IgG at the BMZ. Based on the clinical and immunological results, a diagnosis of drug-induced LABD was made.

We suspected gemfibrozil to be the causative drug. Therefore, administration of gemfibrozil was discontinued, and we started the treatment with administration of oral prednisone and anti-histamine (15 mg/day). Lesions remitted 1-month after discontinuing gemfibrozil. A 1-year follow-up revealed neither signs and symptoms nor recurrences.

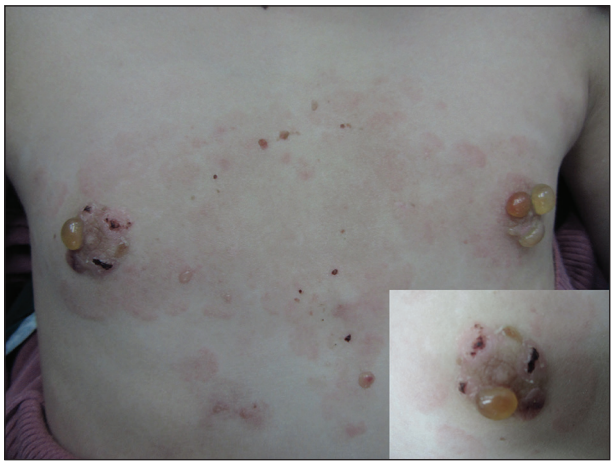

Figure 1: Linear IgA bullous dermatosis. The circumferential tens bullae with erythematous annular background in the areola of breasts

In the case of these medications, they may stimulate the immune system to produce IgA antibodies in a predisposed individual.

Drug-induced LABD usually remits within 2-6 weeks of cessation of the drug.[6] In our case, remission completely occurred after 4 weeks. To the best of our knowledge, there is no report on gemfibrozil-induced LABD.

Drug-induced LABD may be an immunological response to a drug. Drug-induced type tends to show the transient nature of cutaneous symptoms, lack of mucosal or conjunctival lesions, and rapid improvement after discontinuation of medication and lack of circulating IgA antibodies.[7]

It should be treated by discontinuing the suspected drug, but more follow-up may be required.

AUTHOR'S CONTRIBUTION

BA, MP and HS designed the study and were responsible for the overall study management. HS, NA and MS prepared the manuscript. BA and MP conducting the study and revising the draft. All authors approved the final version of the manuscript, and agreed for all aspects of the work.

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