

EXACERBATION OF DERMATITIS HERPETIFORMIS BY HEN'S EGG AND FLUORIDE TOOTH PASTE

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Exacerbation of skin lesions of dermatitis herpetiformis occurred after intake of hen's eggs in a young female and after use of fluoride containing tooth paste in another patient. Elimination of egg from the diet in the first case and avoidance of fluoride containing tooth paste in the second case resulted in remission of the skin lesions.

Key words : Dermatitis herpetiformis, Fluoride, Egg, Exacerbation.

Dermatitis herpetiformis is characterized by remissions and exacerbations. Its aetiology remains obscure. Many reports indicate association of a coeliac-like syndrome with dermatitis herpetiformis.¹⁻³ These patients are sensitive to gluten and pathological changes occur in the jejunal mucosa,⁴ which in turn lead to malabsorption.⁵ In most patients malabsorption may respond to a strict gluten-free diet.⁶ In some patients, skin lesions also respond to such a dietary treatment.⁷ Fry and McMinn observed that 3 of 7 patients with dermatitis herpetiformis who were dependent upon dapsone, required none and were clear of their lesions by the end of one year on gluten-free diet.³ Similar observation has been made by Frodin et al also.⁷ Adjuvant approaches in the management of dermatitis herpetiformis include avoidance of foods containing wheat, rye and barley flour—all of which contain gluten. It is also known that some of these patients show a marked sensitivity to iodides and so iodides, iodized table salts and sea fish that may contain iodides are better avoided in them. Rarely, a flare up of the disease may occur after topical application of iodine or following use of iodine-containing radio-opaque contrast media.⁸ We report two cases in which the skin lesions of dermatitis herpetiformis exacerbated following intake of hen's egg and after use of fluoride containing tooth paste.

Case Reports

Case 1

A 16-year-old female had intensely pruritic, extensive skin eruption of three months duration. There was no history of drug intake prior to the onset of the skin lesions or associated gastro-intestinal symptoms. The skin lesions consisted of grouped vesicles, bullae, papulo-vesicles and patches of erythema mainly on the extensor aspect of the forearms, posterior axillary folds, lumbo-sacral area and buttocks. A few hyperpigmented, circular macules and scars also were seen at these sites. The vesicles and bullae were tense and Nikolsky's sign was negative. The mucous membrane of the mouth and genitalia was unaffected. All other systems were clinically normal. Routine laboratory tests on blood, urine and stools were normal except for 28 mm ESR. Skiagram of the chest was normal. Cytology of the smear taken from the base of a ruptured vesicle showed many neutrophils and a few eosinophils. There were no acantholytic cells. Histopathology of an early vesicle including the surrounding erythematous area revealed subepidermal bulla containing neutrophils and neighbouring papillary tips showed neutrophilic abscesses.

A diagnosis of dermatitis herpetiformis was made and she was treated with dapsone 100 mg twice daily. She responded well to this treatment and at the end of 5 days there were no new lesions. She was discharged from the ward

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with advice to continue dapsone 50 mg thrice daily. Three weeks later, while on dapsone maintenance therapy, she developed exacerbation of the skin lesions which she attributed to the intake of hen's eggs for three days. She was readmitted in the ward and the dose of dapsone was increased to 100 mg thrice daily. Egg was eliminated from the diet. By the end of two weeks she was completely free of skin lesions and the dose of dapsone was reduced to 50 mg twice a day. While in the ward, she was given one boiled egg, daily for 3 days. On the third day, she developed a flare up of dermatitis herpetiformis and it was again controlled with dapsone 100 mg thrice a day. Later, she was on maintenance dapsone therapy, eggs having been completely eliminated from her diet. There was no relapse of skin lesions for three months of follow-up.

Case 2

A 34-year-old male diagnosed to have dermatitis herpetiformis clinically and histopathologically was maintained on dapsone 50 mg twice a day. When the disease was under control, she developed relapse of her skin lesions suddenly following use of a new tooth paste that contained fluoride. There were no associated gastro-intestinal symptoms. The dose of dapsone was increased to 100 mg thrice a day and he stopped using the fluoride containing paste. This led to remission of skin lesions. Gradually the dose of dapsone was reduced and finally maintained on 50 mg twice a day. Patch test done with 1% solution of the paste neither showed a local reaction nor caused flare up of dermatitis herpetiformis. He was again asked to use the fluoride containing tooth paste which was followed by a sudden exacerbation of skin lesions on the third day. There were no lesions in the oral mucosa. The dose of dapsone was again increased to 100 mg thrice a day and then finally maintained on 50 mg twice a day. He was advised to avoid

tooth paste containing fluoride. He is now free from skin lesions and is still on follow up.

Comments

Some substance in the hen's egg worsened dermatitis herpetiformis in our first case. Similarly, in the second case also, exacerbation of skin lesions occurred after the use of fluoride—a halogen-containing tooth paste. Patients with dermatitis herpetiformis are usually advised to avoid foods containing wheat, rye and barley flour because they all contain gluten. Most of these patients experience a flare up of the disease when given iodides and this accounts for the injunctions against sea food. Elimination of milk from diet also has been observed to cause remission of skin lesions in some cases.⁹ The exact mechanism how eggs caused exacerbation of skin lesions in our case is not known. Fluoride, just like iodide, belongs to the group of halogens and the mechanism of exacerbation of dermatitis herpetiformis by both these halogens may be the same. It is important to get a detailed dietetic and other histories in every patient with dermatitis herpetiformis because a particular food substance or a chemical may be the factor responsible for acute flare up of the disease or for lack of therapeutic response to dapsone.

References

1. Marks J and Shuster S : Dermatitis herpetiformis : Role of gluten, *Arch Dermatol*, 1970; 101 : 452-457.
2. Shuster S, Watson AJ and Marks J : Coeliac syndrome in dermatitis herpetiformis, *Lancet*, 1968; 1 : 1101-1106.
3. Fry L, Mc Minn RMH, Cowan JD et al : Gluten-free diet and re-introduction of gluten in dermatitis herpetiformis, *Arch Dermatol*, 1969; 100 : 129-135.
4. Brow JR, Parker F, Weinstein WM et al : The small intestinal mucosa in dermatitis herpetiformis, *Gastroenterology*, 1971; 60 : 355-369.
5. Johnson DP and Alpert ME : Dermatitis herpetiformis—A disease associated with intestinal malabsorption, *Amer J Gastroenterol*, 1971; 55 : 21-32.

6. Marks R and Whittle MW : Result of treatment of dermatitis herpetiformis with gluten-free diet after one year, *Brit Med J*, 1969; 4 : 772-775.
 7. Frodin T, Gothard R, Hed J et al : Gluten-free diet for dermatitis herpetiformis : Long-term effect on cutaneous, immunologic and jejunal manifestations, *Acta Dermato-Venerol* (Stockh), 1981; 61 : 405-411.
 8. Warmer J, Brooks SEH, James WPT et al : Juvenile dermatitis herpetiformis in Jamaica : Clinical and gastroenterological features, *Brit J Dermatol*, 1972; 86 : 226-237.
 9. Engquist A and Pock-Steen OC : Dermatitis herpetiformis and milk-free diet, *Lancet*, 1971; 2 : 438-439.
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