Acquired Epidermolysis Bullosa
Treated with a Gold Compound

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Abstract. A 20-year-old man with acquired epidermolysis bullosa of 3 years’ duration was treated intramuscularly with gold sodium thiomalate. After a total dose of 1000 mg gold sodium thiomalate, administered over a period of 9 months, the patient has shown an almost complete remission, without any apparent side effects of the chrysotherapy.

Key words: Acquired epidermolysis bullosa; Gold sodium thiomalate

Epidermolysis bullosa includes both inherited forms and an acquired non-hereditary form—epidermolysis bullosa acquisita (EBA) (1). This group of diseases is characterized by erosions, formation of bullae with superficial mild scarring and milia formation localized to the sites of apparently slight trauma, predominantly on the elbows, hands, knees and feet. In some sub-groups, minor to severe dystrophic changes and lesions of the oral mucosa occur.

EBA is a rare form which appears in adult life and is often associated with systemic diseases such as Crohn’s disease and ulcerative colitis (8).

Many therapeutic agents, primarily corticosteroids, have been used with varying success in patients with EBA. Using a gold compound, we have successfully treated one patient who had previously responded poorly to other treatments.

CASE REPORT

The clinical, histopathological, immunological and gastroenterological findings in this 20-year-old patient with EBA and Crohn’s disease have been reported earlier (4). In 1976 he was initially treated with prednisone 40 mg and azathioprine 150 mg daily. The prednisone dose was subsequently reduced to 5 mg daily. After 14 months of therapy, the patient showed a complete remission of the gastrointestinal disorder. However, after a slight remission of the skin symptoms, the patient suffered an exacerbation of these symptoms, with numerous new bullae, and was re-admitted in February 1978 (Fig. 1). Despite an increase in the dose of prednisone, the skin lesions could not be brought under control. Treatment with gold sodium thiomalate was therefore instituted. A 10 mg test dose was given intramuscularly, supplemented by 20 mg on the 7th and the 14th day. Subsequently he received 50 mg weekly until a total dose of 1000 mg gold sodium thiomalate had been used. During this treatment there was a gradual, but slow recovery, and after 9 months of therapy an almost complete remission of the skin lesions and the lesions of the oral mucosa was obtained (Fig. 2). No side effects of the chrysotherapy have been observed.

DISCUSSION

Systemically administered corticosteroids, corticotropin and vitamin E have been used in the treatment of EBA (3, 5, 7, 8). The reported results,
however, are conflicting. In our patient, an apparently adequate dose of prednisone and azathioprine did not affect the EBA, although there was complete remission of the bowel disorder. Histological and immunochemical studies on skin biopsies from our patient have shown vasculitis with C3 deposits in the vessel walls and granular deposits of immunoglobulins and C3 in the basement membrane zone (4). Accordingly we have suggested that immune complexes might be involved in the pathogenesis of EBA. Another autoimmune disease—pemphigus vulgaris—has been treated successfully with chrysotherapy (6). The successful chrysotherapy observed in our patient may indicate a systemic effect of gold sodium thiomalate on the reticulo-endothelial system (2) involved immunological mechanisms, rather than a direct effect on the dermo-epidermal junction.

REFERENCES