

A month later, the boy was admitted to our paediatric department with rotavirus gastroenteritis. On admission, the mother reported that since the bronchoscopy he had been unusually "clingy" and had had a morning cough that was occasionally productive. Antibiotics and daily chest physiotherapy at home had failed to resolve the cough. Although the boy had generally been a mouth breather and snorer before the choking episode, the mother described a different type of noisy breathing at night. He had a normal voice, cry, and exercise tolerance. On examination he was well nourished, not cyanosed, and had no added respiratory sounds. Chest radiography was normal.

Under direct laryngoscopy and tracheoscopy, his larynx, subglottis, and trachea down to the carina were normal. At the carina there was granulation tissue on the right side and pus appeared from both main bronchi, especially from the left side. A piece of plastic protruded from the left main bronchus, overlying the carina. It was about 2 × 1 cm and transparent, apart from one end that was red. It was removed with a rigid bronchoscope and identified as the tear-down portion of the anti-tamper oversleeve from a baby-food jar. Reviewed after a month, his respiratory symptoms had resolved.

Our case represents an unusual combination of events. Aspiration of food was recognised and treated appropriately at the initial presentation. However, the identification of a second foreign body was not made and may have been hindered by the transparent nature of the plastic. Even at the second endoscopy, its identification would have been almost impossible but for the red tip. Endoscopists must keep in mind the possibility of a transparent plastic foreign body at the time of bronchoscopy for non-specific pulmonary symptoms.

It is ironic that this child had a severe and potentially life-threatening inhalational injury caused by a fragment of a plastic oversleeve introduced to protect infants from adulterated food. The tear-down section should be completely coloured and preferably not detach from the jar or bottle.

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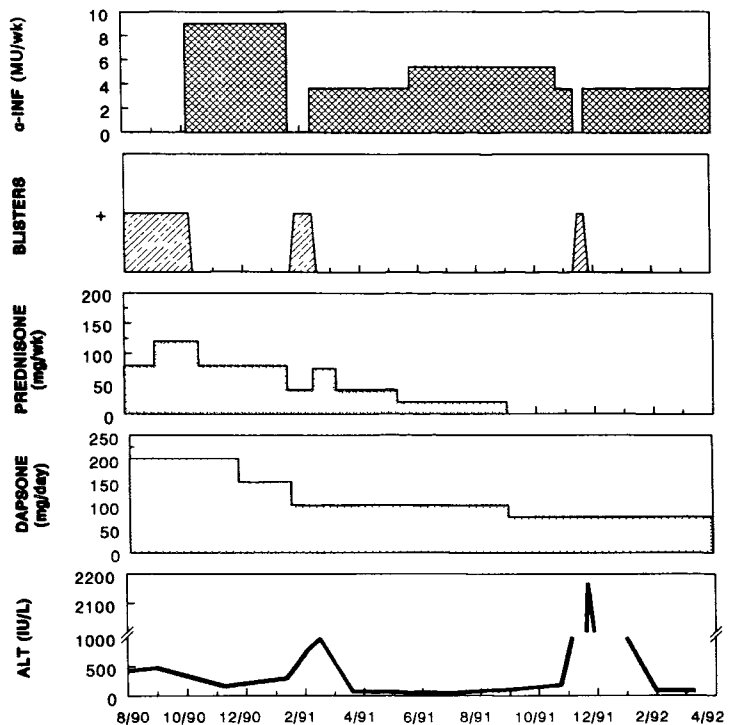
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Interferon alfa for linear IgA bullous dermatosis

SIR,—Linear IgA bullous dermatosis is an immune-mediated subepidermal blistering characterised by linear IgA deposition at the skin basement membrane zone.¹⁻³ Therapy may be difficult despite use of dapsone and prednisone.^{1,2} We have treated a patient with linear IgA bullous dermatosis and chronic hepatitis C with interferon alfa.

A 44-year-old man had a pruritic generalised eruption of papules and blisters. Biopsy revealed subepidermal blister formation and neutrophilic infiltration at the dermal papillae. Direct immunofluorescence showed linear IgA deposits at the basement membrane. Serum IgA concentration was normal. The patient had a history of factor IX deficiency haemophilia, multiple blood-product transfusions, and chronic hepatitis C (hepatitis C virus antibody positive). Initially, the patient's skin disease responded well to dapsone 100 mg daily. Over the next 16 months, however, he became less and less responsive, requiring dapsone 200 mg and up to 50 mg prednisone daily. In October, 1990, we added interferon alfa to the treatment. With this therapy, we could taper prednisone to zero and substantially reduce the dapsone dosage (figure). Discontinuation of interferon in January and November, 1991, was followed by a flare of blisters with raised serum transaminase and bilirubin. Reinstatement of the interferon led to clearing of the blisters and normalisation of serum transaminase.

Interferon alfa may restore the liver's IgA-clearing capacity reduced by injury due to chronic hepatitis.^{4,5} The down-regulating effect of interferon alfa on lymphocyte immunoglobulin promoter genes may also influence IgA concentration.⁶ However, this patient



Effects of interferon alfa on linear IgA bullous dermatosis and chronic hepatitis C.

α-INF = interferon alfa, ALT = alanine aminotransferase, wk = week.

had normal serum IgA before therapy. The immunomodulatory effects of the cytokine on granulocytes⁷ and an effect on leucocyte adhesion molecule expression⁸ is another possible mechanism. Alternatively, hepatitis C itself may exacerbate blister formation.

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Response of cyclophosphamide-resistant Wegener's granulomatosis to etoposide

SIR,—The treatment of severe connective tissue disease, especially Wegener's granulomatosis (WG), is traditionally corticosteroids with azathioprine or cyclophosphamide. A few WG patients are resistant. We report the use of etoposide to induce remission in such a patient.

A 50-year-old man presented with malaise, weight loss, and pyrexia. He had sinusitis with blood-stained nasal discharge, deafness, pleuritic chest pains, arthralgia, oral ulcers, and vasculitic lesions on his fingers. White cell count (WCC) was increased to $15 \times 10^9/l$ with 10% eosinophils, erythrocyte sedimentation rate (ESR) 60 mm/h, and pulmonary shadows on chest radiography. Arteritis was diagnosed and he responded initially to prednisolone 60 mg daily. 4 months later he developed further cutaneous lesions with the typical histological features of leucocytoclastic vasculitis.