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Hypoalbuminemia, oliguria and peripheral cyanosis in an infant with severe atopic dermatitis

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A six-month old male infant with severe atopic dermatitis was admitted with hypoalbuminemia, oliguria and cyanosis of the extremities. There was marked edema and generalized eczema with foul, yellowish exudates. The patient's major clinical manifestations were attributed to the loss of albumin through the skin. Although atopic dermatitis is a common disease in children, here we want to show that systemic disturbances may arise from such condition, describe the total care given the patient, and emphasize the wholistic approach in managing cases of severe atopic dermatitis. Intensive treatment was instituted and the patient was discharged after three weeks and remained in a stable condition.

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Atopic dermatitis is most prominently a disease of infancy and childhood. It is a common disease (1). Epidemiologic studies indicate an increasing incidence in Japan (2–4). Total care in atopic dermatitis may sometimes prove difficult and the dermatitis may be so severe as to warrant hospital admission. Problems may be multiple and varied and systemic involvement may occur (1).

In 1986, Abrahamov et al. reported a case of atopic dermatitis with the complaints of growth failure, edema and anemia, with hypoalbuminemia as the probable cause. Here, we present an infant with severe atopic dermatitis who had generalized edema, oliguria and peripheral cyanosis.

Case report

A six-month old male infant was admitted to this hospital because of generalized eczematous skin lesions and edema. The mother had a history of urticaria. His 4-year old brother had atopic dermatitis. The patient was given mixed feeding up to 2 months after which he was fed purely on soy bean milk and boiled rice. At 5 months, he was tried on orange juice but was discontinued when he had diarrhea. The total caloric intake was 400 kcal per day. The infant was apparently well until about 3 months of age when erythematous, maculopapular and vesicular lesions developed on the face. Atopic dermatitis was diagnosed by a dermatologist. The patient was prescribed skin ointments which partially improved the lesions. At 5 months of age, the eczema worsened and now involved the whole body. Edema, decreased urine volume and cyanosis of the extremities were noted at 6 months of age prompting referral to hospital. On admission, he was conscious but weak and edematous. His weight was 9.1 kg. He had generalized eczematous skin lesions from the scalp down to the lower extremities with foul, thick yellowish exudates (Fig. 1). The buccal mucosa was dry. Lungs and heart examinations yielded normal findings. His abdomen was distended, with no hepatosplenomegaly nor signs of ascites. The extremities were cyanotic, with equal but weak pulses, and no clubbing. Neurological examination was essentially normal.

The patient was hydrated and given Cefazolin 100 mg/kg/day, Ketotifen, and cyproheptadine. Albumin was given for three days. Nutritional support with a hydrolyzed milk formula was initiated. Skin care through three-times-daily bathing was instituted, with hydrocortisone, ibuprofen and gentamycin mixture for the face, betamethasone-gentamycin and ibuprofen combination for the body and betamethasone-gentamycin lotion for the head. Initial laboratory work-ups revealed leukocytosis 20.6 × 10^9 /l with neutrophilic predominance and 8% eosinophils, total protein of 26 mg/l, hypocalcemia 37

Fig. 1. Patient on admission.

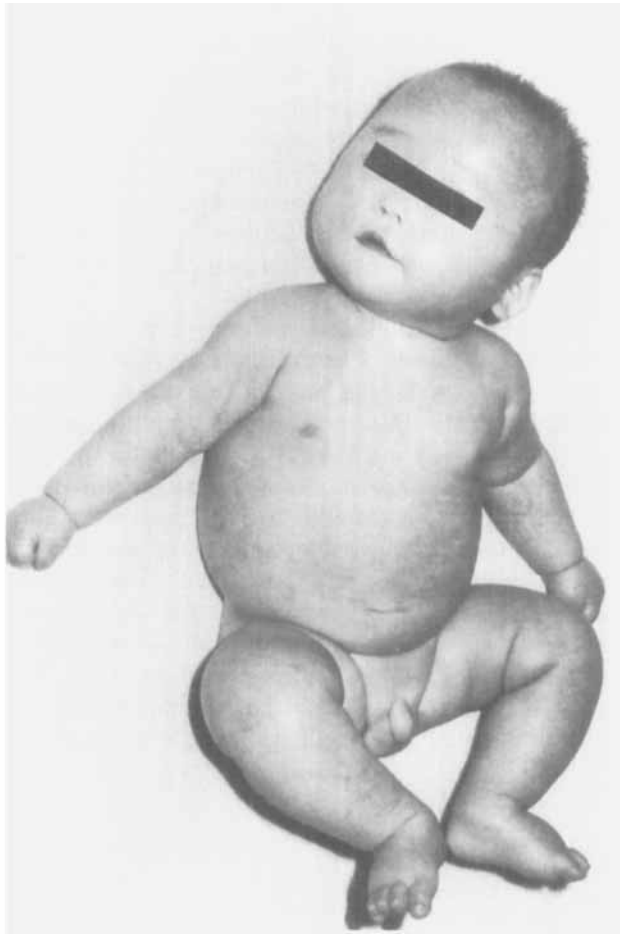

Fig. 2. Patient on his eighth day in hospital.

mg/l, elevated LDH (1500 u/l), low serum cholesterol (0.71 g/l), and blood urea nitrogen (30 mg/l). Occult blood and fat in stools were negative. Urinary amino acid profile was normal.

On the second day in hospital, oral prednisone 1 mg/kg/day was started and a single dose of Furosemide 1 mg/kg was given. Chest x-ray showed normal findings. Repeat analysis of total protein in serum revealed 30 mg/l, hypoalbuminemia of 56% (reference value: 66.6–72.8) and albumin-globulin ratio of 1.28. LDH 4 and 5 were elevated. Cultures of the skin, nose and throat yielded growth of Staphylococcus aureus. Total-IgE was 3000 kU/l with IgE antibodies against egg white, soy bean, house dust, and wheat (RAST, Pharmacia).

The edema diminished on the third hospital day and the patient's weight decreased to 9000 g. Skin condition had improved remarkably after eight days (Fig. 2) and prednisone was discontinued. Serum total proteins were normal on the 10th hospital day.

The patient was discharged improved after 24 days with a weight of 7.4 kg. The condition remained stable on subsequent follow-ups.

Discussion

Abrahamov et al. reported a case of atopic dermatitis with edema, failure to thrive and anemia (5). They attributed their findings to the patient's severe hypoproteinemia. An increased loss of protein through the skin probably increasing metabolic requirements and low food intake were suggested as reasons for the low serum albumin in their patient. They assumed that the anemia was secondary to impaired erythrocyte production. The generalized edema seen in our patient may also have been due to hypoalbuminemia.

Patients with generalized dermatitis or psoriasis may have a low serum albumin concentration but no evidence of true impairment of hepatic function (6). The SGOT was normal but the LDH isoenzymes 4 and 5 were high in our patient.

Mukai et al. noted an increased serum LDH activity, particularly LDH 4 and 5 in most cases with severe atopic dermatitis and suggested that these may be used to monitor response to treatment (7).

The hypoalbuminemia seen in our patient was possibly the result of the dilution within the plasma and

to an increased passage into the extravascular space. There was an impaired capillary permeability to albumin leading to decreased plasma albumin due to the extensive inflammation of the skin. The vasodilation may have resulted in increased total vascular capacity. Because of increased capillary permeability, some of the increased plasma volume was then lost into the tissues (6). Furthermore, patients with dermatitis have abnormal vasoconstrictor response and have a precarious hemodynamic state (6).

Our patient developed oliguria, a problem not found in the patient described by Abrahamov (5). The mechanism of oliguria may be explained by the marked decrease in the urinary excretion of sodium and potassium as was suggested by Shuster (6). The glomerular filtration rate and plasma sodium were normal, indicating that the filtered load of sodium was normal and the tubular resorption was greatly increased. If this were the case, then water absorption would be a consequence, and this would result in the passage of small volumes of dilute urine (6).

In summary, we reported a case of an infant who suffered from atopic dermatitis presenting with hypoalbuminemia, oliguria, and peripheral cyanosis. Atopic dermatitis may result in severe systemic disturbances as found in this report and that of Abrahamov (5). Prompt and aggressive treatment should be instituted and a wholistic approach to chronic management must be emphasized.

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