A child with kwashiorkor misdiagnosed as atopic dermatitis

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Abstract

Although uncommon, kwashiorkor continues to occur in developed, but mainly in developing nations. It is a type of protein-calorie malnutrition that occurs in the setting of insufficient protein intake in the presence of sufficient caloric intake. Skin and hair changes should prompt a thorough dietary history and appropriate dietary intervention. We report a case of a 12-month-old girl in Belo Horizonte, Minas Gerais, Brazil, who presented with diffuse edema, desquamation, and irritability misdiagnosed as atopic dermatitis. The diagnosis was consistent with kwashiorkor as a result of severe dietary restriction. The mother had placed the child on a severely restrictive diet, consisting only of potatoes, gelatin, and juice as a consequence of the inability to breastfeed. Kwashiorkor is often under-diagnosed or misdiagnosed and if unrecognized or untreated, may be devastating. This makes it imperative that physicians consider this diagnosis, recognize potential risk factors, and be prepared to accurately assess overall nutritional status of patients.

Keywords: protein energy malnutrition, kwashiorkor, pediatric nutritional diseases

Introduction

The term kwashiorkor was coined in 1935 by Cicely Williams, a British pediatrician, in an article in the Lancet, in which she described its presence in children with maize diets [1]. Associated physical findings may include a dermatitis, protuberant abdomen, thinning hair, and "bull-dog" face. Clinical findings may also include weight loss/failure to thrive, delayed linear growth, irritability, and lethargy [1, 2].

The condition is a type of protein-calorie malnutrition most often seen in children of impoverished countries or areas of famine. Causes are controversial, but the most commonly accepted theory is inadequate dietary protein in the presence of sufficient caloric intake [1-4]. Children placed on restrictive diets by "well-intending" parents, as a result of concern for food allergies, have been reported to be previously at risk of nutritional deficiency, such as kwashiorkor [3, 4].

Case Synopsis

A 12-month-old girl presented with a 3-month history of progressive apathy and skin changes. The child's mother reported she was diagnosed as having severe atopic dermatitis, after consulting with some pediatricians and dermatologists. She had been using moisturizer cold cream, topical steroids (hydrocortisone), and tacrolimus 0.03% ointment. Despite treatment the child's skin condition did not improve. When asked about the child's feeding, the mother reported that she placed the child on a severely restrictive diet, consisting only of potatoes, gelatin, and juice, because of the inability to breastfeed. In addition, the child's mother believed that skin lesions were related to dietary protein intake (mainly milk).

Examination revealed generalized hypopigmentation with numerous erythematous and denuded patches mainly over his trunk and legs; discolored hair was also present. Desquamation in a flaking or "paint-chip" pattern was prominent on the abdominal area and lower extremities (**Figures 1, 2**).

The child's abdomen was distended with umbilical

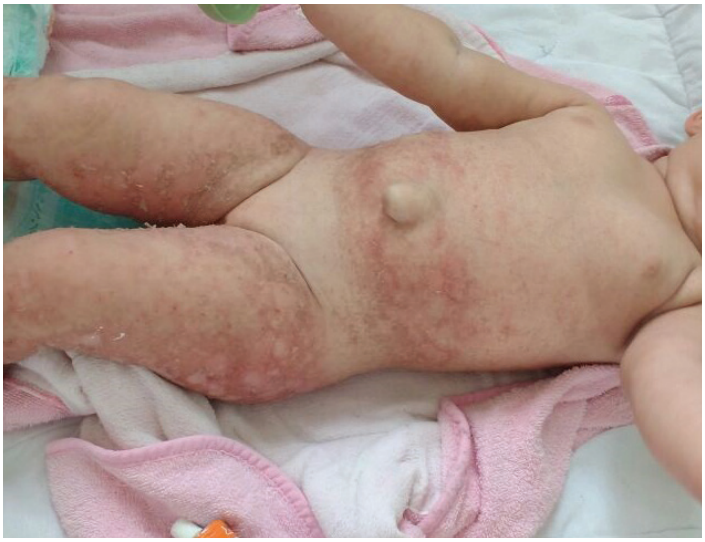

Figure 1. *Generalized hypopigmentation with numerous erythematous and denuded patches*

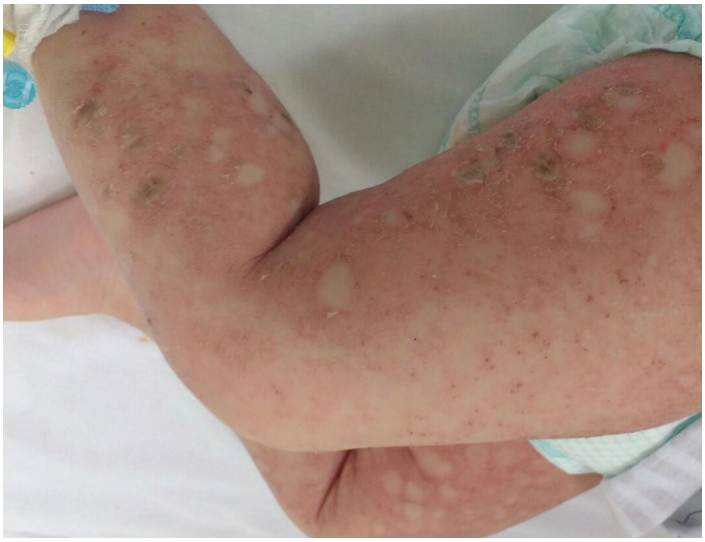

Figure 2. *Desquamation in a flaking, "paint-chip" pattern was prominent on the lower extremities*

hernia and dependent edema over the extremities. Her hair was pale yellow. Laboratory measurements were as follows: normal complete blood count; total protein, 5.4 g/dL (normal, 5.7-8.2 g/dL); albumin, 2.7 g/dL (normal, 3.2-4.8 g/dL); aspartate transaminase and alanine transaminase levels were elevated at 76U/L (normal, <11-34U/L) and 55U/L (normal, 10-49U/L), respectively. Values for zinc, vitamins A, K, and E and 1, 25-vitamin D were within normal reference. Our patient's clinical and laboratory findings were consistent with kwashiorkor secondary to dietary protein restriction. Our patient's skin changes improved rapidly within three weeks after increased dietary protein.

Case Discussion

Protein-energy malnutrition (PEM) is manifested primarily by inadequate dietary intakes of protein and energy, either because the dietary intakes of these 2 nutrients are less than required for normal growth or because the needs for growth are greater than can be supplied by what otherwise would be adequate intake [1, 2]. PEM is almost always accompanied by deficiencies of other nutrients. Historically, the most severe forms of malnutrition, marasmus (nonedematous malnutrition with severe wasting) and kwashiorkor (edematous malnutrition), were considered distinct disorders. Nonedematous malnutrition was believed to result primarily from inadequate energy intake or inadequate intakes of both energy and protein, whereas edematous malnutrition was believed to result primarily from inadequate protein intake [2]. A low plasma albumin concentration, often believed to be a manifestation of malnutrition, is common in children with both edematous and nonedematous malnutrition.

Kwashiorkor has been reported in developed and undeveloped countries families [3]. It may be a result of the use of unusual and inadequately nutritive foods to feed infants whom the parents believe to be at risk for milk allergies, and also in families who believe in fad diets [2-4]. Many cases are associated with rice milk diets, a product that is very low in protein content [5]. In addition, protein-calorie malnutrition has been noted in chronically ill patients in neonatal or pediatric intensive care units as well as among patients with burns, HIV, cystic fibrosis, failure to thrive, chronic diarrhea syndromes, malignancies, bone marrow transplantation, in born errors of metabolism [6-8].

Edematous malnutrition (kwashiorkor) can occur initially as vague manifestations that include lethargy, apathy, and/or irritability. When kwashiorkor is advanced, there is lack of growth, lack of stamina, loss of muscle tissue, increased susceptibility to infections, vomiting, diarrhea, anorexia, flabby subcutaneous tissues, and edema [1-3]. The edema usually develops early and can mask the failure to gain weight. It is often present in internal organs before it is recognized in the face and limbs. Liver enlargement can occur early or late in the course of disease. Dermatitis is common, with darkening of the

skin in irritated areas, but in contrast to pellagra not only in areas exposed to sunlight. Depigmentation can occur after desquamation in these areas or it may be generalized. The hair is sparse and thin; in dark-haired children, it can become streaky red or gray. Eventually, there is stupor, coma, and death. Infection and sepsis continue to be the main causes of death in severe acute malnutrition, although other causes include dehydration, electrolyte imbalances, and heart failure [2, 3].

World Health Organization (WHO) has developed guidelines for managing severe malnutrition [3, 9]. Death also can occur once treatment is instituted because of refeeding syndrome, which is associated with severe electrolyte and metabolic changes. The decision as to whether to treat in the hospital or community depends on the patient's clinical condition and availability of resources. WHO has formulated a three-phase management approach, in which the patient initially is resuscitated and stabilized (phase 1), started on nutritional rehabilitation (phase 2), and followed-up for recurrence prevention (phase 3).

Conclusion

This case exhibits an important theme in recent literature: food fads and unconventional dietary changes may cause nutritional deficiencies and skin changes are often key signs of the diagnosis.

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