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# A 4-Year-Old Boy from Mozambique With Severe Oedema and Skin Lesions

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## Clinical Presentation

### History

An oedematous, HIV-negative 4-year-old boy from rural Mozambique is seen at the paediatric department of a central hospital in Malawi.

He was admitted 5 days earlier with the suspected diagnosis of nephrotic syndrome and was started on furosemide and prednisolone; however, his oedema did not subside.

Three weeks earlier he had been treated for pneumonia at a health centre.

### Clinical Findings

The little boy is miserable, refusing to eat and apathetic with a puffy face and pitting oedema on the lower legs. His hair is brittle, sparse and fair in colour. The skin is dry and hyperpigmented; it is peeling off like 'flaky paint' (Fig. 72.1) and there are ulcerative skin lesions, most prominently in the groins (Fig. 72.2).

### Laboratory Results

Albumin 2.2 g/dL (reference range 3.0–5.2 g/dL), haemoglobin 7 g/dL (12–14 g/dL).

## Questions

1. What is the likely diagnosis and how can it be distinguished from nephrotic syndrome?
2. How should the child be treated and what is the prognosis?

## Discussion

Three weeks after a severe bacterial infection, a 4-year old boy from rural Mozambique develops oedema. The skin is hyperpigmented and dry, peeling off and there is discoloration of his hair. Treatment with furosemide has no influence on the extent of the oedema. Clinically, the little boy

• **Fig. 72.1** A 5-year-old boy with generalized oedema. The skin is hyperpigmented and dry. It is peeling off like 'flaky paint'. Underneath, the skin is hypopigmented.

• **Fig. 72.2** Ulcerative skin lesions on the lower abdomen and in the groin (where zinc ointment and Gentian violet solution ("GV paint") has been applied).

is sick and refusing to eat or drink. He is anaemic and his serum albumin is low.

## Answer to Question 1

### *What is the Likely Diagnosis and How Can It be Distinguished from Nephrotic Syndrome?*

Generalized oedema in a child can have various causes. In nephrotic syndrome, serum albumin is low and there is heavy proteinuria. Low serum albumin is however influenced by many factors such as nutrition, liver function and intestinal resorption.

This child is displaying additional clinical features such as skin and hair changes as well as apathy. These are typical features of kwashiorkor, a form of severe acute malnutrition (see Summary Box).

A urine dipstick test should be done to check for protein. In nephrotic syndrome, proteinuria exceeds 3.5 g per day (4+ on a urine dipstick) and the urine is often macroscopically frothy. In kwashiorkor, some proteinuria may be found but rarely exceeds 1+ on a dipstick test.

## Answer to Question 2

### *How Should the Child be Treated and What is the Prognosis?*

The child needs to be admitted to the high dependency area of the nutrition ward and treated according to WHO guidelines for severe acute malnutrition with meticulously calculated amounts of feeds and close monitoring. The risks of infection, diarrhoea, anaemia, cardiac and liver failure are high. Prophylactic antibiotics, monitoring of temperature and haemoglobin as well as careful fluid replacement, are part of the treatment scheme.

## The Case Continued…

The skin in the groin and armpits continued to peel off and became superinfected. The lesions could successfully be treated with Gentian violet solution.

Initially the child did not tolerate 3-hourly feeds and required feeding through a nasogastric tube. On day 5 in the nutrition rehabilitation unit he could eat by himself and the oedema started to settle. The transition to higher caloric feeds was tolerated well and he could be discharged into the community feeding program at day 15.

### *SUMMARY BOX*

#### Kwashiorkor

Kwashiorkor is a form of severe, acute malnutrition characterized by hypoalbuminaemia and oedema.

Kwashiorkor mainly occurs in areas where people live on a monotonous diet and the staple food has a low protein-to-energy ratio (e.g. maize, cassava or bananas). It is uncommon in communities where diet is supplemented by animal protein. Incidence peaks during the rainy season when staple food items and vegetables are in short supply.

However, kwashiorkor is not (just) a consequence of a diet low in protein and micronutrients. Infections often precede the onset of the disease. They lead to low albumin levels in the context of acute phase reactions. Diarrhoeal diseases and capillary leakage result in further loss of protein, nutrients and fluids. An imbalance between free radicals and insufficient levels of antioxidants leads to oxidative stress and damage of cell membranes. Hypoimmunoglobulinaemia leads to severe infections, contributing to the high fatality rates associated with kwashiorkor.

The typical age at presentation is 1 to 3 years; boys and girls are equally affected.

Hepatomegaly is a common finding. The hair becomes depigmented, fair or reddish in colour and is easy to pluck. Hyperpigmented, dry, damaged and infected skin may show pale patches ('flaky paint' dermatosis) and ulcerations. Children with kwashiorkor are miserable, apathetic and often refuse to eat.

Management is challenging and involves careful feeding with slowly increasing amounts of feeds with meticulous monitoring of glucose levels, weight gain and fluid status. Prophylactic broad-spectrum antibiotics should be given to all patients, because signs of infection including fever may be absent. Diuretics are contraindicated because they further deplete the intravascular volume, causing hypotension. The oedema will settle once therapeutic feeding has been established.

Patients should also receive folic acid, anthelmintics and, once stable, measles vaccination if unvaccinated. Relapses are common and renal and liver function, as well as glucose levels, may be deranged well beyond the time of clinical recovery.

Case fatality rates from kwashiorkor are high, with a median of 20–30%. Additional infection with HIV further worsens the prognosis.

## Further Reading


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