MARASMUS IN SOUTH AFRICAN BANTU CHILDREN

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The commonest of the diseases of malnutrition in Bantu children, kwashiorkor, is caused mainly by a deficient intake of good-quality protein.[1-3] Another disease that usually has a nutritional origin is known as marasmus (athrepsia, infantile atrophy).[3] Marasmus is characterized by severe cachexia, and its basic cause in the cases of nutritional origin appears to be a deficient intake of calories as well as of certain important nutrients, including protein.

It has been suggested that there is a basic relationship between kwashiorkor and marasmus.[4] While there are undoubtedly cases that are clinically intermediate between these two conditions, it is our opinion that the clinical and biochemical differences between typical cases of kwashiorkor and typical marasmus cases are sufficiently clear-cut to justify regarding the two conditions as separate entities. In contradistinction to kwashiorkor there is, in marasmus, little or no oedema; skin lesions are absent or minimal; and the serum-protein concentration is only moderately depressed.[5] Marasmic children are usually more severely underweight for their age than children suffering from kwashiorkor. On the average marasmus cases fall into a younger age group (Fig. 1) than cases of kwashiorkor and respond much more slowly to treatment. Lastly, the incidence of concomitant diseases or anomalies is much greater in marasmus than in kwashiorkor and such abnormalities, when present in marasmus, are usually of primary aetiological significance in the development of the syndrome.

In this report a resumé is given of our experience with 27 cases of marasmus, 5 of which are reported in detail.

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CLINICAL MATERIAL

All the patients in the series of 27 cases (12 male and 15 female) were Bantu children admitted over a period of 2 years to the same ward of the Pretoria General Hospital. Only Bantu children are admitted to this ward, so that, although marasmus occurs in all races, the material for this study was drawn from only Bantu children. A few patients admitted during this period were excluded from the series because their clinical records were incomplete.

Age. The ages of the children ranged from 1½ months to 5 years. Nine were younger than 6 months and 18 were older. The mean age was 10·5 months.

Weights. The weights of the children ranged from 2 kg. (4 lb. 6 oz.) to 7·6 kg. (16 lb. 11 oz.). The weights of all the children were under the 3rd percentile line for average North American children.[6] In Fig. 1 the age/weight rela-

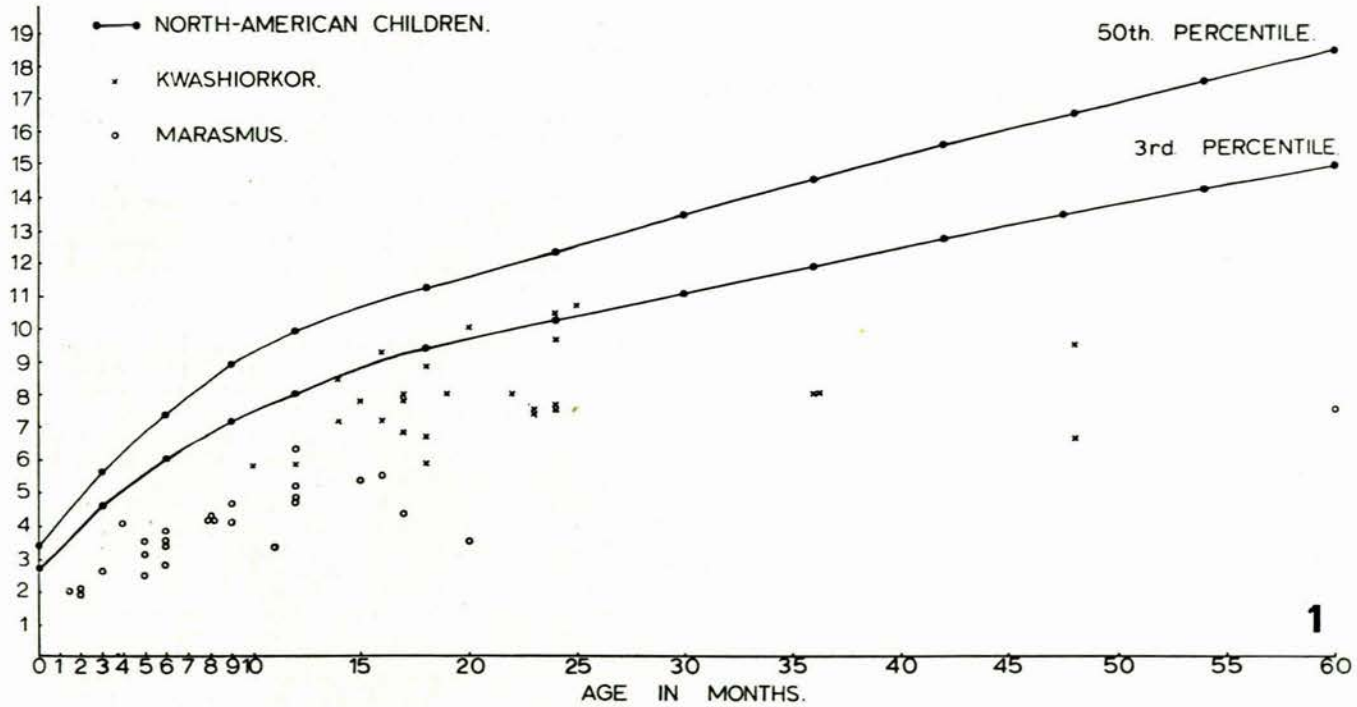

Fig. 1. Age/weight relationship of patients with marasmus (circles), kwashiorkor (crosses) and North American children (solid lines). The ordinate figures (1 - 19) represent weight in kg. The percentile lines representing North American children taken from Nelson.[6]

tionship of these children is compared with that of Bantu kwashiorkor patients admitted to the same ward and with average North American children. Even if they were compared with the kwashiorkor patients who have lost their oedema (i.e. the lowest weight recorded during recovery) the marasmic patients would still be noticeably more underweight.

History. The main complaint on admission, as given by the mother, was usually diarrhoea, vomiting or coughing. In a few cases the child was brought to hospital because the mother had herself noticed that it was not gaining weight.

Diet. All the children, with one exception, had been weaned from the breast by the time they were admitted to hospital. In most cases it was not possible to obtain a satisfactory dietary history from the mother.

Clinical picture. All the children were very small and underweight for their age. The typical picture (Fig. 2) was that of a small, thin child with a loose skin lying in folds and with little subcutaneous fat. The anterior fontanelle was often depressed and the eyes sunken. The chin and cheekbones were prominent, and so were the ribs. The abdominal wall was thin and peristaltic movements could often be seen. Muscle tone was usually poor, although opisthotonos was observed in a few cases. It is often difficult to decide whether or not dehydration is present, because the classical picture in marasmus is in many respects similar to that of dehydration.

Severe anaemia was not a characteristic finding. The haemoglobin concentration was below 9 G. per 100 ml. in only 5 cases, in all of which the morphological picture of the red blood cells resembled that found in hypochromic anaemia. The serum-albumin concentration was usually normal (for Bantu children) or only moderately decreased.

An associated disease or anomaly was found in 11 cases. Details are given in Table I.

TABLE I. CONCOMITANT DISEASES FOUND IN A SERIES OF 27 CASES OF MARASMUS

Patient	*Age in months*	*Sex*	*Weight in kg.*	*Associated diseases*
1	8	M	2·2	Bronchopneumonia
2	5	M	3·5	Cellulitis
3	6	M	3·4	Primary tuberculous complex
4	9	M	4·8	Cerebral and pulmonary venous thrombosis and haemorrhage
5	18	M	5·9	Pyelitis
6	6	F	3·5	Shigella enteritis infection
7	1·5	F	2·6	Horseshoe kidney
8	5	M	3·2	Salmonella infection, pyelonephritis, atrophic kidney
9	3	M	2·6	Shigella enteritis infection
10	12	F	4·7	Pneumatosis cystoides intestinalis
11	60	M	7·5	Brain tumour

Clinical improvement and increase in weight took place slowly in most of the patients who recovered. The average duration of stay in hospital of the patients who recovered was 10 weeks. One child spent 6 months in hospital before he could be discharged. The younger patients characteristically underwent episodes of hypothermia. This was of serious prognostic importance, for 6 of the 8 children who developed hypothermia subsequently died.

Ten (37%) of the patients died, 6 deaths occurring in the group of 11 cases where an associated illness was found and only 4 in the group of 16 patients with no concomitant disease.

CASE REPORTS

Case 10

R.M., a Bantu girl about 1 year old, was admitted on 28 July 1959 because of frequent attacks of vomiting, during which she brought up a thin black fluid. Constipation and occasional abdominal distension. She had progressed poorly since birth and had recently become very thin.

On examination. No abnormalities were found apart from marked emaciation (weight 4·7 kg.).

Special investigations. Hypochromic anaemia was found to be present. The following results were obtained per 100 ml.: Haemoglobin 8·2 G.; total serum protein 5·8 G. (serum albumin 2·4 G.); fasting blood sugar 62 mg.; serum calcium 9·4 mg.; serum inorganic phosphate 4·5 mg. Urine normal.

Progress. The patient was originally regarded as a case of nutritional marasmus. Feeds of full-strength milk were at first well taken but after about a week she began to vomit after feeds. As the vomitus contained blood a hiatus hernia was suspected, but could not be confirmed by X-ray after a barium meal. The vomiting ceased and the weight increased when the feeds were thickened with 'nestargel'. About 1 month after admission, however, the patient collapsed suddenly, with periodic respiration and bradycardia (pulse rate 40 per minute). ECG at this stage showed a sino-atrial block and a prolonged Q-T period. Cortisone was given, but the patient died shortly afterwards.

Postmortem examination (Dr. W. J. Pepler). The valve at the cardia of the stomach was incompetent and there was a polypoid cystic appearance along the greater curvature. The muscle layer in the region of the pylorus was markedly hypertrophied and the pyloric canal was very narrow. Microscopic examination of the affected part of the gastric wall showed pneumatosis cystoides. Microscopic examination of the myocardium showed a non-specific chronic myocarditis.

Comment. The vomiting in this case was caused by the pyloric stenosis, which was also responsible for the marasmic condition. The pneumatosis of the stomach wall was apparently also secondary to the pyloric stenosis. Most cases of pneumatosis cystoides reported in the literature have been secondary to obstructive lesions of the gastro-intestinal tract.[7] In spite of the pyloric stenosis, however, the child improved after the feeds were thickened, and death was apparently due to the myocarditis.

Case 11

S.M., a 5-year-old Bantu boy, was admitted on 8 August 1960 for chronic cough for 2 months and progressive loss of weight. A month before admission sores had begun to develop on the buttocks, arms, legs, and neck.

On examination. Severely emaciated; weighed only 7·5 kg. Numerous bedsores. Scattered crepitations on both sides of the chest. No other abnormality found.

Special investigations. ESR 10 mm. in first hour. Blood count showed no abnormality; blood sugar and urea and the serum cholesterol, proteins, calcium and alkaline phosphatase all within normal limits. Serological tests for syphilis negative. Mantoux test twice negative, but lung X-ray showed mottled shadows in right lower lobe and lingula, and possibly enlarged hilar glands. Fasting gastric contents repeatedly negative for tubercle bacilli. Urine normal. Urinary 17-ketosteroids 0·18 mg. per 24 hours (normal values 0·5 - 2·5 mg.) and 17-ketogenic steroids 0·11 mg. (normal values 4 - 20 mg.). X-ray of skull normal.

Progress. The child was treated for tuberculosis; improvement was rapid and the bedsores healed. About 3 weeks after admission he suddenly became comatose but recovered con-

sciousness after 2 hours. Thereafter a steady weight loss occurred in spite of a reasonably good appetite. About 2 months after admission the circumference of the skull was 20½ inches as compared with 20 inches on admission. No papilloedema, but pressure of CSF on lumbar puncture was 300 mm. of water. On chemical analysis the fluid was normal. X-rays of skull now showed very definite signs of increased intracranial pressure; small areas of calcification visible in region of hypophysis. The patient's condition was now very poor, and he died a few hours after a catheter was inserted into a lateral ventricle to relieve the pressure. Permission for autopsy refused by parents.

Comment. When marasmus occurs in older infants the presence of underlying disease must be considered to be very likely. In a recent series of 80 children over the age of 6 months suffering from 'failure to thrive', underlying diseases were present in 70 (87·5%).[8] Cerebral lesions were the commonest abnormality, being regarded as the cause of the illness in 26 cases. Cerebral tumours, especially those in the region of the third ventricle, are an important cause of the marasmic picture.[9] It is essential to bear in mind in every case of marasmus the possibility that cerebral disease may be present.

Case 12

S.N., 22 months, was admitted on 8 Desember 1960. She had lost weight progressively for 6 months. Occasional loose green stools. She was breast fed until 2 months before admission but since she was 7 months old the mother's breast milk had been insufficient and supplements of goat's milk and maize-meal porridge had been given.

On examination. Very emaciated and dehydrated; weighed only 3·4 kg. (Fig. 2). No other abnormality found.

Special investigations. The following results were obtained per 100 ml.: Total serum protein 6·9 G. (albumin 3·6 G.); serum cholesterol 165 mg.; serum calcium 8·6 mg.; serum inorganic phosphate 2·2 mg.; serum alkaline phosphatase 7 KA units; blood urea 26 mg.; haemoglobin 6·9 G. Urine normal. Neither salmonella nor shigella could be cultured from a rectal swab. Serological tests for syphilis negative. Mantoux tests negative, but chest X-rays showed shadows in the paracardiac region and hilar-gland enlargement. X-rays of skull and long bones normal. The sweat test for fibrocystic disease of the pancreas was negative.

Progress. Feeding was begun with half-skimmed milk, followed after a few days by full-strength milk. When the appetite improved the usual ward diet was given in addition. Weight unchanged for first 10 days but thereafter increased rapidly. The haemoglobin remained low, in spite of the administration of iron, and a transfusion of 120 ml. of whole blood was given, after which it rose to 10·4 G./100 ml. Anti-tuberculosis drugs were given. She progressed well and when discharged after 6 months she weighed 8·4 kg. (Fig. 3).

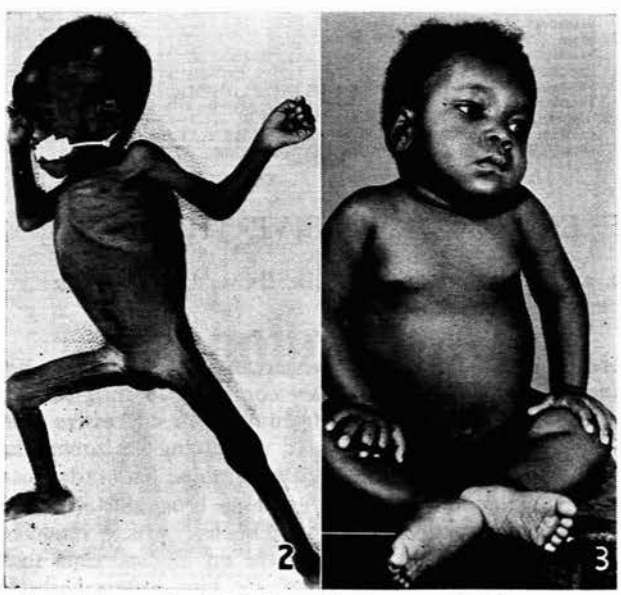

Fig. 2. Photograph of S.N. (Case 12) taken on admission.
Fig. 3. Photograph of same child as in Fig. 2 taken after recovery.

Comment. This was probably a case of primary nutritional marasmus. The mother's milk supply was insufficient from an early stage and the supplements given were most likely also inadequate. The attacks of diarrhoea with loose green stools ('starvation stools') might be a sign of undernutrition. It is possible that primary lung tuberculosis was present and played a part in the development of the clinical picture.

Case 7

E.M. was 6 weeks old on admission to hospital on 10 March 1961. She had had diarrhoea with up to 12 green watery stools per day since the age of 7 days. The birth had been normal after a full-term pregnancy, with a threatened abortion at about the 6th month.

On examination. A small and emaciated infant (weight 2·6 kg.) with the typical appearance of marasmus. No other abnormality found on clinical examination.

Special investigations. The systolic blood pressure 55 mm.Hg ('flush' method). No anaemia. Urine normal. Total serum protein 8·4 G./100 ml. (albumin 3·1 G.), serum cholesterol 165 mg./100 ml. and serum potassium 6 mEq./litre. The serum sodium and chloride normal. Blood urea 55 mg./100 ml. An intravenous pyelogram revealed a horseshoe kidney.

Progress. The child's general condition did not improve after admission; no increase in weight. The body temperature fell repeatedly to subnormal levels, and 7 weeks after admission the infant died. No postmortem examination.

Comment. The incidence of horseshoe kidney in children has been reported to be 1 in 270 of all cases brought to postmortem.[10] It is commonly associated with other congenital anomalies. Cases usually, but not always, present with hydronephrosis, infection, or renal calculi, and great care is necessary if cases are not to remain undiagnosed. It is a serious condition and the mortality rate is high although operative treatment may be successful. That the presence of congenital renal lesions should be suspected in all cases of marasmus is well illustrated in this case, where the only indication of renal disease was a moderately raised blood urea.

Case 8

A male Bantu infant of 5 months was admitted with a history of a cough and diarrhoea for 3 days. No previous illnesses, and nothing of interest in the family history. The birth had been normal after a full-term, normal pregnancy. The child was still breast fed.

On examination. A small thin baby with the typical appearance of marasmus. Weight 3·2 kg. Mild dehydration. There was a white discharge from the penis. The systolic blood pressure was 80 mm.Hg.

Special investigations. In the urine 4+ albumin and 4+ pus cells were found. Haemoglobin 10·9 G./100 ml. Total serum protein 6·2 G./100 ml. An intravenous pyelogram showed the right kidney to be atrophic. A salmonella of group B (Kaufmann-White) was cultured from a rectal swab.

Progress. After administration of the appropriate antibiotic the diarrhoea ceased, and after several weeks the pus cells and albumin disappeared from the urine.

Comment. This case and the previous one illustrate well the importance of renal lesions in the causation of marasmus. Urinary infection was reported to be responsible for 0·8% of all the admissions to a children's hospital in America.[11] Pyelonephritis is one of the most important forms of renal infection. Obstruction of the urinary tract is frequently the underlying cause and is usually congenital in origin in young children.

DISCUSSION

Patients showing the clinical picture of marasmus may belong to one of two groups, namely those in which the

condition is apparently of purely nutritional origin and those in which it is secondary to an underlying abnormality. Whatever the aetiology, marasmus is a serious condition with a high mortality rate, and convalescence is long and difficult for those who recover.[12,13] Resistance to infection seems to be lowered and attacks of hypothermia tend to occur.

In every case a thorough search must be made for any possible underlying disease or congenital abnormality. If a complete history and a thorough clinical examination, including routine urine and haematological examination, reveal no specific defect, more specialized tests should be carried out. A good review article on the subject has been published by Stevenson.[14] Radiological examination of the skull, heart, lungs, kidneys and gastro-intestinal system may all be necessary. Tumours of the brain, especially in the region of the 3rd ventricle, are an important cause of the condition and may be difficult to diagnose.[9] Atrophy of the cerebral cortex has been named as another cause.[15] Chronic infections such as syphilis and tuberculosis and repeated attacks of acute infection should be borne in mind as possible causes. Inborn errors of metabolism are now more often diagnosed than formerly, and biochemical tests designed to discover these defects should be carried out whenever indicated. Examples are conditions such as galactosaemia, congenital renal acidosis, and fructose intolerance.

Where no basic organic abnormality can be found the case may be regarded as primarily nutritional in origin. The role of emotional factors should not, however, be overlooked. Although the mechanism is obscure, there is no doubt that the personal attention and love of a mother or mother substitute are necessary if a child is to thrive, and that lack of it may bring about both physical and psychical retardation.[16]

The precise nature of the nutritional deficiency which leads to marasmus is not yet clear, but it would seem that it follows an inadequate intake of all the important nutrients, proteins and calories included.[1,4,5,17,18] Much has still to be learnt about the biochemical and physiological abnormalities that are present in marasmus.

The treatment of marasmic cases is beset with difficulties. Their nutritional requirements seem to be high, but diarrhoea and vomiting may be precipitated by too rapid an increase in caloric intake. Regulation of body temperature is imperfect and great care is necessary to prevent hypothermia. Attacks of hypoglycaemia may occur, requiring administration of glucose.

Vigorous measures should be taken against any concomitant disease that may be present.

SUMMARY

Our experience with 27 cases of marasmus is presented. We considered that marasmus and kwashiorkor are seperate conditions with distinguishing characteristics. Marasmus may be a primary nutritional condition or may be secondary to an underlying disease or anomaly. It is a serious condition, being difficult to cure and having a high mortality rate. The importance of a thorough and complete examination is emphasized and mention is made of problems that arise in the treatment of these cases.

SAMEVATTING

Die skrywers bespreek hul ondervinding met 27 gevalle van marasmus. Marasmus en kwashiorkor word as afsonderlike toestande met kenmerkende eienskappe beskou. Marasmus mag primêr nutrisioneel van aard wees of sekondêr wees tot 'n onderliggende siekte of afwyking. Dit is 'n ernstige toestand wat moeilik is om te genees en het 'n hoë sterftesyfer. Die belangrikheid van 'n deeglike en volledige ondersoek word benadruk en probleme in verband met die behandeling word genoem.

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