

CASE REPORT

Marie-Dominique Piercecchi-Marti,¹ M.D., Ph.D.; Claude Louis-Borrione,² M.D.; Christophe Bartoli,¹ M.D.; Alain Sanvoisin,¹ M.D.; Michel Panuel,³ M.D., Ph.D.; Anne-Laure Pelissier-Alicot,¹ M.D., Ph.D.; and Georges Leonetti,¹ M.D., Ph.D.

Malnutrition, a Rare Form of Child Abuse: Diagnostic Criteria

ABSTRACT: Infantile malnutrition is often difficult to diagnose as it is rarely observed in industrialized countries. It may be associated with physical violence or occur in isolation. The essential clinical sign is height and weight retardation, but malnutrition also causes a variety of internal and bone lesions, which lead to neuropsychological sequelae and death. We report a rare case of death by malnutrition in a female child aged 6½ months. The infant presented height and weight growth retardation and internal lesions related to prolonged protein–energy malnutrition (fat and muscle wasting, thymic atrophy, liver steatosis) resulting in a picture of marasmus or kwashiorkor. We detail the positive and negative criteria that established the diagnosis of abuse, whereas the parents had claimed a simple dietary error.

KEYWORDS: forensic science, abuse, malnutrition, kwashiorkor, marasmus

Child abuse by neglect is difficult to diagnose when it is not associated with physical violence, especially as neglect may take on a variety of forms: improper hygiene, lack of medical care, or food restriction (1–3). It is rarely observed in industrialized countries and is sometimes presented as the involuntary consequence of ethnic or religious eating habits or economic necessity by the parents who are asked to account for the child's condition (4). The essential clinical sign is height and weight retardation, but malnutrition causes a variety of internal and bone lesions, which lead to neuropsychological sequelae and death (5). We present a fatal illustration concerning a female infant aged 6½ months who presented, apart from growth retardation and cachexia, severe lesions of the liver, pancreas, brain, and bone that were related to prolonged inadequate protein, vitamin, and energy intake. This was presented by the parents as a dietary error due to economic necessity, after infant formula was replaced by cow's milk.

Case Report

A female infant aged 6½ months was found dead by her mother at about 07:00 hours, in the bouncer chair where she had been put for a sleep 5 h earlier. The body, clothed only in a sleepsuit, was cold when the emergency services arrived, and the existence of a medicolegal case was recorded in the death certificate because of the child's cachectic state.

¹Laboratoire de Médecine Légale, Faculté de Médecine, 27 boulevard Jean Moulin, 13385 Marseille Cedex 5, France.

²Service de Chirurgie Pédiatrique, CHU Timone-Enfants, 264 rue Saint-Pierre, 13385 Marseille Cedex 5, France.

³Service de Radiologie, CHU Nord, Chemin des Bourrellys, 130016 Marseille.

Received 10 Jan. 2005; and in revised form 12 March 2005; accepted 12 Oct. 2005; published 21 April 2006.

Examination of the body showed a rectal temperature of 15°C for a room temperature of 15°C, and because of the presence of very pale posterior lividity and very little stiffness of the upper part of the body, the death of this thin, lightly clothed child was estimated to have occurred in the early afternoon.

On autopsy, height and weight were found to be markedly retarded. The child weighed 3166 g for a length of 59.5 cm, with a head circumference of 41.5 cm for an expected weight of about 7000 g and length of 68 cm (2.5th percentile) (Fig. 1).

Bodily hygiene was poor, with ingrained dirt in the armpits, groin, and legs, as well as weeping dermatitis of the buttocks and excoriations of the inside of the thighs (Fig. 2). The nails were soft. There were signs of major dehydration such as sunken eyes and hypotonia, marked skin folds, and depressed anterior fontanelle. The tongue was dry but there were no signs of glossitis. The abdomen was not distended. When the cavities were opened, there were no serous effusion or internal malformations, but we were surprised to observe atrophy of the thymus (weight 3 g for an expected weight of 10 g for the child's weight at the time of death) and also the buff color of the liver, suggesting lipid overload, whereas there was no subcutaneous fat layer and muscle mass was slight. The stomach was empty of food debris. The bladder and small and large intestines were empty. No skin, bone, or internal lesions suggesting physical violence were observed. Radiographs of the child revealed specific metaphyseal abnormalities known as Harris lines and delayed bone maturation. Death was attributed to severe dehydration related to denutrition. Malnutrition was suspected.

Microscopic examination of the internal organs confirmed massive liver steatosis without inflammatory changes or glycogen overload (negative PAS stain), suggesting a prolonged protein deficiency of kwashiorkor type (Fig. 3). The pancreas also exhibited minor acinar distension. In the brain tissue, the neurons of the mamillary tubercles and the pontine nuclei were slightly enlarged



FIG. 1—Severe cachexia with dehydration.

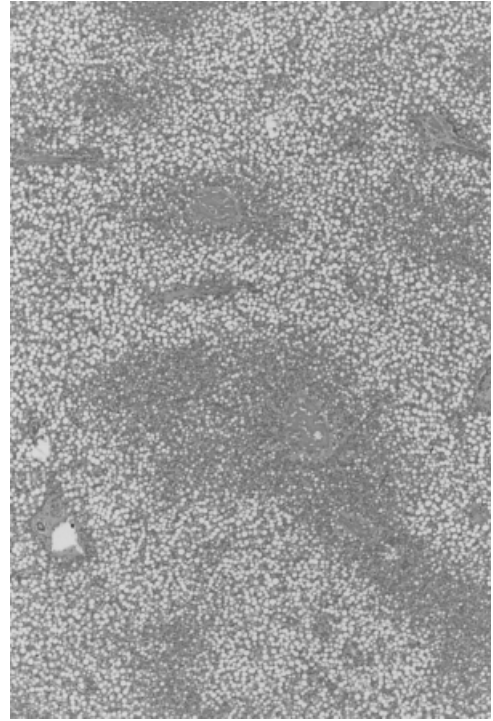


FIG. 3—Massive liver steatosis (original magnification $\times 100$).

with diffuse lesions of chromatolysis, suggesting an associated vitamin deficiency

The intestinal villi were not altered, excluding an immunologic absorption disorder, especially as the mother had not described any symptoms of diarrhea.

Laboratory tests were not requested by the examining magistrate because the quantity of blood that could be sampled was small and the magistrate had given greater attention to toxicological analysis. In addition, because of ocular hypotonia, the vitreous humor could not be sampled in sufficient quantity and without contamination. No toxic substances were present, neither ethyl alcohol nor complex organic substances, but blood and bile contained high levels of acetone. Blood acetone was 20 mg/L (normal value 3.1 mg/L), suggesting prolonged fasting.

Background

The girl was the third child of a North African couple who had been living in France for several years. The baby was born in France, at term after a normal pregnancy. Birth weight was 2830 g and length 47.5 cm, with a head circumference of 33.5 cm.

The mother did not breastfeed her and she was given infant formula. At the age of 2 days, she was transferred to the pediatric department of a public hospital because of feeding difficulty. No anomaly was detected during the 48 h she was under observation, but the pediatric nurses reported anxiety about the mother-child relationship and feared the child was rejected by the mother, but rejection was not confirmed by the psychologist who was consulted. Mother and child were discharged from the maternity unit at 8 days after a normal medical examination. The usual obliga-



FIG. 2—Weeping dermatitis of the buttocks.

tory screening tests were carried out (hyperphenylalaninemia and hypothyroidism). At discharge the baby's weight was 2900 g. Follow-up by the child welfare services (maternal and child health, MCH) was requested at discharge.

However, the child only had one medical consultation at the age of 4½ months for chickenpox on the occasion of a home visit for her siblings. At the time, the doctor noted a weight of 5000 g (small but not abnormal, and height was not recorded). The vaccination program had not been started and the parents had not responded to the follow-up letters of the MCH services, as they had not for the other two children who, although in good health, did not attend school regularly and had no regular medical follow-up. The two elder brothers were aged 5 and 3 years. They weighed 22 kg for a height of 112 cm and 16 kg for 98 cm, respectively, when they were examined for possible ill-treatment when the judicial investigation was opened.

No other abnormal event was described by the mother. She had apparently observed that from the age of 5½ months her daughter had lost weight but had not thought it necessary to see a doctor, especially as the baby was reported to be a calm, smiling infant who slept a lot. This loss of weight was described by the mother as occurring at the same time as her diet was changed for economic reasons (the family had a low but regular income), infant formula being replaced by full-fat cow's milk when the infant was 5 months old.

It was difficult to obtain details on the diet the mother gave her child, in particular the quantity and frequency of bottles. It should be noted that the mother was not mentally deficient in any way; she had obtained her high school diploma and declared that she had brought up her first two children in the same way. She was questioned on several occasions and her answers regarding the quality and quantity of food varied. A typical day appeared to consist of two to three bottles of whole cow's milk diluted with water for a total quantity of between 180 and 210 g, to which about 10 teaspoonfuls of semolina was added at about the age of 6 months, given alternately with puréed vegetables and four jars of

fruit baby food. The child finished all her meals but sometimes missed a feed in the afternoon because she was sleeping. Stools were not daily but there was no diarrhea.

On the day the baby died, the mother declared she had given her child a bottle of milk with added semolina and a fruit compote, which the child had finished entirely a few minutes before she was put back in her bouncer chair to sleep.

Discussion

In developed countries, malnutrition is often the consequence of cult diets or the deliberate exclusion of certain foods (6). A disturbance of the mother-child relationship may however be implicated (6) and it is this faulty relationship that seems to underlie the voluntary food restriction of the mother in this case, compounded by the lack of essential hygiene and health care, as there had been no change in economic or family circumstances since the birth of the second child.

This rare case of death because of abusive malnutrition is a reminder that the diagnosis is based on clinical, anthropometric, radiological, anatomopathological, and biological arguments, with in this case lesions identical to those observed in children in developing countries who suffer famine after weaning.

Malnutrition in the child occurs when food intake is of inadequate quantity and/or quality to meet the protein and energy needs of the organism (5). These needs are particularly high and specific because of growth, and insufficient intake and energy is reflected in delayed length and weight gain, probably further retarded by associated mineral deficiencies (6). Anthropometric curves are therefore indispensable tools for monitoring the child's nutritional status. Although in France monthly check-up visits for infants are obligatory if social allowances are not to be lost, there is little that social services can do apart from making out an official complaint of abuse to the authorities. The MCH services cannot compel families to present their child. A social enquiry is nevertheless started but the judicial authorities are not informed, in the early stages, if no objective evidence of neglect is reported.

Radiological findings are also objective evidence for diagnosis as they reveal Harris lines and delayed bone maturation (7). Harris lines, or growth arrest lines, have been considered as a nonspecific marker of stress undergone at a given moment in time. On X-ray, they appear as dense trabecular bone transversal to cancellous bone, whereas normal trabeculation is longitudinal. They result from temporary arrest of biological processes, when cartilage is not mineralized and hypertrophic cartilage is no longer transformed into calcified cartilage (7).

Protein-Energy Malnutrition (PEM)

Inadequate protein and energy supply is responsible for PEM. Classically, two clinical forms of severe PEM can be distinguished: marasmus and kwashiorkor. However, there are many intermediate forms and it is now accepted that these two clinical syndromes lie at extremes of the same continuum (5-8). Our case is an illustration of this, as the clinical signs were in favor of marasmic malnutrition, whereas the marked liver steatosis that we observed is more typical of kwashiorkor (8). Without treatment, death inevitably occurs within a few weeks (8-9).

Marasmus is the end result of a global energy deficiency that is prolonged but remains balanced in terms of protein/energy ratio (8). Even at an advanced stage there are few signs other than weight loss, and biological abnormalities are rare and late. Inversely, the main features of kwashiorkor are low serum albumin

and edema, as the deficiency is predominantly related to the low protein content of the diet (9). Weight loss is much less marked than in marasmus. The other clinical signs are inconstant, mainly skin changes with the development of exsudative lesions of the orifices or skinfolds. These are of poor prognosis and they were observed in this child.

If intake is inadequate, protein and energy stores must be mobilized and consumed. In parallel, energy expenditure is reduced to order to adapt to the energy deficiency. This "economy" involves reduction of physical activity and slowing of growth, which is an important item of energy expenditure in the child. Growth failure usually occurs a few weeks after slowing of weight gain, the first clinical sign of malnutrition (6). Unfortunately, as in our case no follow-up of the child is available, we cannot identify when the break in the growth curve occurred. In marasmus, muscle amino acids are mobilized to maintain the metabolism of the internal organs, in particular of the liver (9). The level of ketone bodies increases in blood (acetonemia was seven times higher than normal in this child) (8).

PEM has repercussions on the entire organism, but these vary depending on whether the clinical picture is closer to marasmus or to kwashiorkor. In the present case, the features observed were in favor of an intermediate form, so several organs showed changes. Unfortunately, in this case we have no laboratory test results to reflect these modifications. In particular, lipid levels (seeking low triglycerides) and protein levels (low serum albumin) (6) are lacking because the examining magistrate had sufficient evidence of abuse by food deprivation to bring a charge against the parents. In the liver, steatosis develops but this is present above all in kwashiorkor, glycogen is then normal or even low and the pancreas develops disorganization of acinar structure followed by atrophy (9). The major effect of severe PEM is atrophy of the thymus, lowering immune defenses and increasing the likelihood of opportunist infections (10). Renal function is also impaired with decreased ability to concentrate urine, reflected in polyuria which accounts for dehydration, but this can also be accelerated by inadequate fluid intake in the days before death (8). PEM leads to decreased bone mass with mineral loss and delayed ossification (development of Harris lines) (7). In the brain, brain tissue may be atrophic, there may be neuronal damage and altered mental function (11,12). Anorexia is frequent and also psychomotor disturbances such as irritability or apathy. In marasmus, the child sleeps a lot and gives up living. In the present case, the mother described her child as being particularly calm.

Because of decreased metabolic activity, the malnourished child shows little or no reaction to changes in temperature and is at risk of hypothermia (10). Liver glycogen reserves are particularly low in marasmus and the risk of fatal hypoglycemia is always present. In the absence of a progressive infectious disorder, in the case described, it is impossible to determine the terminal pathological factor which led to death: dehydration, hypoglycemia, or hypothermia may be debated (13).

Was Malnutrition Due to Inappropriate Diet Alone?

It is now common knowledge that as the concentration of anabolic elements in the milk of each species is proportional to growth velocity, cow's milk, by definition, is not adapted to the needs of the human infant, a mammal whose growth is particularly slow and protracted (14). Even if the water content and energy value are similar, the concentrations in sodium, potassium, calcium, phosphorus, protein, and casein of cow's milk are three to seven times higher than those of breastmilk or infant formula (14).

In addition, the lactose content of cow's milk is nearly 50% lower than that of infant formula, and the iron, essential fatty acid, vitamin E, vitamin C, and folic acid content are markedly inadequate in cow's milk.

In infants, a diet consisting exclusively of cow's milk results in marked protein deficiency because of intestinal malabsorption of cow's milk proteins, and in fluid and electrolyte imbalance with the onset of dehydration and mineral and vitamin deficiencies (14). This malabsorption leads to a diarrhea syndrome. In our case the child did not suffer from diarrhea, suggesting insufficient energy intake rather than an inappropriate diet.

While it is not difficult to demonstrate that there was at the very least an error of appreciation of the nutritional value of cow's milk compared with infant formula, it is less easy to determine the true quantity of food given each day to the child. There were however suspicions as to the amount of milk actually given, as the mother had no clear idea of the quantity, which varied during the various questionings, while a mother noticing that her child was obviously losing weight would rapidly consult a doctor.

The duration of food deprivation could not be estimated in our case with the precision suggested in the literature by the use of equations or curves (10,15) as it is probable, taking into account the time that the child survived, that food was indeed given, even if in very inadequate quantities, and even if not on a daily basis. Nevertheless, the liver lesions observed reflected prolonged inadequacy of intake and the autopsy observations make it possible to discount the mother's statement as to the quality and quantity of food that she said she had given the child before putting her to bed. In fact, estimation of the time of death as in the early afternoon (heat loss being rapid in small children, especially as the room was cold and the child lightly clothed and cachectic) (16) and the observation that all the digestive lumens were empty are not compatible with the mother's statement. In addition, the blood acetone level, which was interpretable postmortem, suggested prolonged fasting (17).

The mother and father were found guilty of deprivation of care and food leading to the death of a minor, caused by a parent. In France, child abuse carries heavier penalties if committed by the lawful parents, whether natural or adoptive, and they were sentenced to 20 years' imprisonment.

References

1. Davis JH, Rao VJ, Valdes-Dapena M. A forensic science approach to a starved child. *J Forensic Sci* 1984;29:663-9.
2. Berkowitz CD. Fatal child neglect. *Adv Pediatr* 2001;48:331-61.
3. Kloiber LL. Does the expert witness fit the crime? Injury to a child by starvation—a dietitian's testimony. *J Forensic Sci* 2004;49:108-10.
4. Roberts IF, West RJ, Ogilvie D, Dillon MJ. Malnutrition in infants receiving cult diets: a form of child abuse. *Br Med J* 1979;1:296-8.
5. Colomb V. Dénutrition de l'enfant. *Rev Prat* 2003;53:263-7.
6. Melchior JC. Diagnostic et dépistage de la dénutrition. *Rev Prat* 2003;53:254-8.
7. Resnick D. *Diagnosis of bone and joint disorders*. Philadelphia: WB Saunders Company; 1995:3353-5.
8. Zazzo JF. Physiopathology and consequences of malnutrition. *Rev Prat* 2003;53:248-53 (French).
9. McClave SA, Mitoraj TE, Thielmeier KA, Greenburg RA. Differentiating subtypes (hypoalbuminemic vs. marasmic) of protein-calorie malnutrition: incidence and clinical significance in a university hospital setting. *J Parenter Enteral Nutr* 1992;16:337-42.
10. Nagao M, Maeno Y, Koyama H, Seko-Nakamura Y, Monma-Ohtaki J, Iwasa M, et al. Estimation of caloric deficit in a fatal case of starvation resulting from child neglect. *J Forensic Sci* 2004;49:1073-6.
11. Piercecchi-Marti MD, Pellissier-Alicot AL, Leonetti G, Tervé JP, Cianfarani F, Pellissier JF. Pellagra: a rare disease observed in a victim of mental and physical abuse. *Am J Forensic Med Pathol* 2004;25:342-4.
12. Tan H, Onbas O. Central pontine myelinolysis manifesting with massive myoclonus. *Pediatr Neurol* 2004;31:64-6.
13. Bonet AM, Martinez RA, Pujals FJM, Vall CO. Kwashiorkor as a symptom of abuse and neglect in Barcelona. *An Esp Pediatr* 2001;54:405-8; Spanish.
14. Melchior JC. Dénutrition-malnutrition. In: Godeau P, editor. *Traité de médecine*. Paris: Flammarion; 1997:1478-85.
15. Meade JL, Brissie RM. Infanticide by starvation: calculation of caloric deficit to determine degree of deprivation. *J Forensic Sci* 1985;30:1263-8.
16. Saukko P, Knight B. Neglect, starvation and hypothermia. In: *Knight's forensic pathology*. 3rd ed. London: Arnold; 2004:412-20.
17. Steinhauer JR, Volk A, Hardy R, Konrad R, Daly T, Robinson CA. Detection of ketosis in vitreous at autopsy after embalming. *J Forensic Sci* 2002;47:221-3.

Additional information and reprint requests:

Marie-Dominique Piercecchi-Marti, M.D., Ph.D.
 Laboratoire de Médecine Légale, Faculté de Médecine
 27, Boulevard Jean Moulin
 F-13385 Marseille Cedex 5, France
 E-mail: marie-dominique.piercecchi@mail.ap-hm.fr