FIG. 1. (a) and (b).—Aged 3 months: lytic areas in frontal region and early hyperostosis of the mandible.

FIG. 2.—Aged 7 months: mandibular hyperostosis remaining.

changes here described from the appearance of secondary neoplasm, particularly neuroblastoma, may be difficult, and the diagnosis may only become clear with evolution of more typical radiological appearances.

Eversole, Holman, and Robinson in 1957 described focal resorption of cortical bone as an early microscopical feature in the evolution of infantile cortical hyperostosis. The relative vascularity and the thinness of the cortical bone of the skull vault may be the reason why this histological change has not until recently been reported radiologically.

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R. D. H. Boyd, D. G. Shaw,* and B. M. Thomas Departments of Paediatrics and Radiology, University College Hospital, London WC1E 6AU.

*Correspondence to Dr. D. G. Shaw.

Renal Cortical Necrosis in an Infant

Renal cortical necrosis is a rare and usually rapidly fatal disease, death resulting from acute renal failure. However, in recent years with the increasing availability of improved supportive measures, including peritoneal dialysis and haemodialysis, some of the patients survive long enough to allow calcification of the necrotic renal cortex which can be detected radiologically.

Up to 1968, 17 cases of renal cortical necrosis radiologically diagnosed ante mortem had been reported in adults, but only 3 in infants (Rémy et al., 1968; Whelan, Ling, and Davis, 1967). Mauer and Nogrady (1969) reported a newborn infant in whom papillary and cortical necrosis was docu-
mented radiologically in life. We here present an infant with renal cortical necrosis associated with calcification shown radiologically, who survived after prolonged peritoneal dialysis.

Case Report

A 10-month-old male infant was admitted on account of anuria. During the previous 2 days he had been treated for diarrhoea and vomiting. On the third day of that illness he stopped passing urine, and there was fever with generalized convulsions. Hb was 7.0 g/100 ml, white blood cells were 10,000/mm³, with normal differential count. The platelets were 300,000/mm³ and the reticulocytes 0.8%. Plasma sodium was 124 mEq/l., potassium 6.2 mEq/l., chloride 81 mEq/l., HCO₃⁻ 10 mEq/l., calcium 4.0 mg/100 ml, phosphorus 4.6 mg/100 ml, and urea 225 mg/100 ml. Serum proteins were 5.7 g/100 ml, with 4.3 g/100 ml albumin, and 1.4 g/100 ml globulin. Blood pressure was 180 mmHg systolic. An abdominal x-ray film obtained at that time was normal. He was treated with blood, fluids, calcium, electrolytes, antibiotics, and antihypertensive drugs. Anuria persisted and on the 8th day peritoneal dialysis was started. The response to dialysis and the course of the disease can be seen in Fig. 1. 8 days after starting dialysis he passed a small amount of urine showing specific gravity 1013, albumin 130 mg/100 ml, glucose 2.8 g/100 ml, and some red cells. Over the following days the urinary volume increased gradually, oedema disappeared, and the blood pressure and urea became normal. Peritoneal dialysis was continued for 40 days. On the tenth day he developed pseudomonas peritonitis which was successfully treated with antibiotics and by increasing the amount of exchangeable fluids in the peritoneal cavity. Two days after peritoneal dialysis was discontinued (50th day of illness) an abdominal x-ray showed extensive diffuse bilateral renal cortical calcification (Fig. 2).

An excretory pyelogram done at that time showed a failure to concentrate the dye.

Six months later blood pressure was normal and blood urea 70 mg/100 ml; urinalysis showed specific gravity 1012, proteinuria as well as red cells and casts in the sediment. The creatinine clearance was 31 ml/min. An abdominal x-ray revealed essentially the same picture as before, though both kidneys were rather irregular and decreased in size.

FIG. 1.—Response to peritoneal dialysis, and course of disease.
Discussion

Although a definite diagnosis of renal cortical necrosis can be made only by biopsy or at necropsy (Lloyd-Thomas, Balme, and Key, 1962), the history, clinical course, and radiological findings in our case provide good evidence of this. According to McAlister and Nedelman (1961), renal cortical necrosis must be considered in acute renal failure in children, especially when diarrhoea, vomiting, and dehydration are part of the clinical picture.

The necrotic lesions are attributed to renal ischaemia and the numerous causes of the disease have been extensively reviewed (Lauler and Schreiner, 1958; McAlister and Nedelman, 1961; Black, 1967; Rémy et al., 1968). Though the first radiological manifestation is renal enlargement, recognition of this may well be difficult. Gradual decrease in kidney size, owing to shrinkage of the necrotic tissue follows, and calcification of the necrotic areas become the outstanding features.

According to Black (1967) renal calcification is of three types: (1) fine diffuse and chiefly cortical, (2) coarse and mainly medullary, and (3) localized. In our case shrinkage of the kidneys and irregularity of the renal contour became evident, and the pattern of nephrocalcinosis was of the first of the above types. Black (1967) states that the most frequent causes of this type of nephrocalcinosis are renal cortical necrosis and chronic glomerulonephritis. Lloyd-Thomas et al. (1962) considered a ‘tram-line’ pattern of calcification characteristic of renal cortical necrosis.

There must be a time relation between the onset of the disease and the x-ray appearance of renal calcification (Whelan et al., 1967). In our case nephrocalcinosis was detected radiologically on the 50th day of the illness. McAlister and Nedelman (1961) suggested that 1 to 2 months may elapse before calcification occurs.

There is no doubt that our patient survived mainly because of prolonged peritoneal dialysis. This procedure is especially applicable to infants and small children, and its application is likely to lead to an increasing number of patients with this disease surviving.

Summary

A 10-month-old infant developed renal cortical necrosis, leading to nephrocalcinosis shown radiographically. The infant survived after prolonged peritoneal dialysis.

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A. Moschos,* C. Danelatou-Athanassiadou, F. Tzortzatou, and C. Katerelos

Paediatric Clinic of Athens University, St. Sophie’s Children’s Hospital, Goudi, 608, Athens, Greece.

* Correspondence to Dr. A. Moschos.

Cytomegalovirus Infection Presenting as Acute Haemolytic Anaemia in an Infant

Cytomegalovirus (CMV) infection is now known to be common at all ages. A recent survey (Collaborative Study, 1970) in the North West of England gave an incidence of virus isolation in babies as 0·4%, rising to 5·2% at 5 years and 40–45% in women of child-bearing age. Lamb (1971) gives even higher figures for children according to their environmental conditions. The majority of infants who excrete the virus have minimal or no symptoms, so that it may be difficult to correlate virus excretion with clinical diseases in many cases (British Medical Journal, 1971). CMV infection, if congenital, commonly presents either as a glandular fever-like syndrome with hepatosplenomegaly, or as disease of the central nervous system with microcephaly and mental retardation (Weller and Hanshaw, 1962). Except in the neonatal period (Emanuel and Kenny, 1966; McInerney and Stern, 1970), acute haemolytic anaemia may be a rare presentation of acquired disease, one fatal adult case being reported by Coombs (1968) and 22 cases in children suggested by Zuelzer et al. (1966), associated with lymphadenopathy, 7 of which had a fatal outcome.

Case Report

A 4-month-old male baby was admitted with a history of rapidly increasing pallor over the previous 3 days associated with noticeable pink staining of the napkin by the baby’s urine. He had had a cough and cold
Renal cortical necrosis in an infant.

A Moschos, C Danelatou-Athanassiadou, F Tzortzatou and C Katerelos

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