CORTICAL NECROSIS OF KIDNEYS IN AN INFANT

BY

MIRGHANI YOUSIF ALI, D.M.S.K.

Resident Assistant Pathologist, Mile End Hospital, London.
Formerly Pathology Resident to the Institute of Pathology,
and Teaching Fellow, University of Pittsburgh School of
Medicine

Bilateral cortical necrosis of the kidneys is very uncommon, and is particularly rare in infants. The condition has been typically described by various authors in association with pregnancy. Where the disease was not associated with pregnancy the age distribution in reported cases covered a very wide range, but only a few cases were recorded in children below the age of 3 months. Since the 9-weeks-old case reported by Campbell and Henderson in 1949, only two cases have been published (Zuelzer *et al.*, 1951). Those two cases occurred in infants aged 5 days and 12 hours respectively. In the three above-mentioned cases some essential features were different from those found in the following case, both clinically and in the gross pathological appearance. The rapid clinical course, the rarity of the condition in this age group, and the gross anatomical appearance of the kidneys justify the recording of this case.

Case Record

A 3-weeks-old male infant was admitted to the Springfield City Hospital, Ohio, on January 25, 1954, with respiratory difficulty. The mother stated that he refused feedings.

The infant was born in the Springfield City Hospital on December 31, 1953. The mother, a 1-gravida, was delivered after the 35th week of gestation. The infant, although weighing 5 lb. 11½ oz. (2.6 kg.), was considered premature. The delivery was normal and spontaneous under ether anaesthesia. Physical examination of the mother gave no positive findings, and the past history was non-contributory. After birth the infant's breathing was delayed, and when he began to breathe it was laboured; oxygen was administered and he was admitted to the hospital nursery. Four days after birth frontal and lateral x-ray views of the chest suggested evidence of an alveolar atelectasis. Seven days later the x-ray examination was repeated and the report revealed full expansion of the lungs with slight prominence of the bronchovascular pattern in the central lung areas. The baby progressed satisfactorily for twelve days and was discharged from the nursery in good condition.

About a week later the infant was readmitted to hospital. The mother stated that he refused feedings for several hours before readmission, had respiratory difficulties, became pale, and had a distended abdomen.

On examination the baby was dyspnoeic, pale, and lethargic. The temperature was 103° F. (39.4° C.). The mouth, tongue, and throat were healthy. The abdomen was not distended. The liver and spleen were palpable. Physical examination of the respiratory system was negative. Radiological examination of the chest revealed fine diffuse granular infiltration which seemed to be associated with the bronchovascular markings of both lungs. There was a patch of what appeared to be localized infiltration in the right base, and the markings were a little heavier in the right upper lobe than elsewhere. There was no evidence of any lobar consolidation. No abnormality of the cardiovascular system was detected, and no lymphatic nodes were palpable. Blood examinations showed: red cells, 2,420,000 per c.mm.; Hb, 57% (8.05 g./100 ml.); white cells, 36,500 per c.mm. (polymorphonuclears 63%, stab nuclears 5%, eosinophils, 1%, lymphocytes 31%). Blood Kahn reaction was negative. Blood culture showed no growth.

The temperature dropped to normal on the day after admission. The urine was scanty, was pink in colour at first, and then became blood-stained.

The baby became slightly cyanotic and irritable, and had muscular twitchings. Some petechial haemorrhages were noticed in the scalp. After only one day in the hospital the infant died following a period of respiratory distress.

For treatment, penicillin and chloramphenicol palmitate were given, and a blood transfusion and intravenous fluids were also administered.

Necropsy

The body was that of a male infant measuring 18½ in. (47 cm.) in length. There was no oedema, palpable adenopathy, or cyanosis. The right side of the diaphragm was at the level of the sixth interspace and the left side at the level of the seventh interspace. The right lung weighed 33.5 g. and the left 30 g. The lungs on section were grey-red in colour and showed many airless collapsed areas, especially

FIG. 1.—Kidneys showing extensive subcapsular haemorrhages. Cut surfaces show haemorrhage in substance of kidney. (Scale indicated in inches.)

FIG. 2.—Cortical necrosis of kidneys. Extensive necrosis of cortical structures and parenchymal haemorrhage. (× 112.5. Haematoxylin and eosin.)

FIG. 3.—Haemorrhagic cystitis. Replacement of epithelial lining by fibrin, pus cells, and red blood cells. (× 112.5. Haematoxylin and eosin.)

in the lower lobes; these areas were firm in consistency. The thymus weighed 3 g. and showed many subcapsular petechial haemorrhages. The stomach was congested and showed many petechial haemorrhages in its mucosal surface. The kidneys together weighed 146 g. They were about equal in size, were globular in shape, and their capsules stripped easily to reveal extensive subcapsular haemorrhages and lobulated dark-red cortical surfaces (Fig. 1). On section both kidneys showed haemorrhage in their substance, which gave them a dark purple-red colour, and they were moderately soft. The pelvis and calices of both kidneys were normal in size and contained some clotted blood. The urinary bladder was small and contracted, was lined by roughened haemorrhagic mucosa, and had thickened rigid walls.

Histopathology of Organs.—Many sections were taken from both kidneys; on microscopical examination these showed extensive necrosis of the cortex and marked suffusion of all the surrounding tissue with haemorrhage. Extensive search was made throughout the tissues for evidence of thrombi, but none could be found. There was considerable infiltration of the tissues immediately around the necrotic areas by polymorphonuclears (Fig. 2). Sections of the bladder showed marked thickening of the wall due to inflammatory oedema of the tissues, increase of fibrous tissue, and a thick layer of fibrinous exudate and haemorrhage replacing the mucosa. The mucosal covering was necrotic, and was covered by fibrin infiltrated with polymorphonuclears, lymphocytes, and haemorrhages (Fig. 3). Sections of the lungs showed areas of atelectasis.

Comments

The infant was affected by an acute fulminating condition, presumably a respiratory infection to which premature infants are particularly prone. The clinical course of this case was very short and pre-mortem diagnosis of the condition was almost impossible, as the condition simulated various diseases; the clinical impressions, however, were those of bronchopneumonia, meningitis, or septicaemia. The detection of oliguria, together with haematuria in the late stage of the disease, when the infant was almost dying, was the only indication of renal damage.

The gross anatomical findings presented two unique features; the massive subcapsular haemorrhages in both kidneys, and marked haemorrhagic cystitis. The complete absence of thrombi in serial sections from both kidneys was another distinct feature.

The presence of acute infection was a common feature with the other cases reported, but the clinical course in this case was too short to produce all the damage caused to both kidneys and to the urinary bladder. Unfortunately, the case was under observation for only one day, and thus many clinical tests could not be completed.

Discussion

Renal Lesions

There are various theories to explain the pathogenesis of bilateral cortical necrosis of the kidneys. Most authors agree that the underlying cause is a prolonged vasospasm of the smaller arteries and arterioles, leading to ischaemia of the cortex. Some authors regard thrombosis as the trigger mechanism, but, according to Dunn and Montgomery (1941) and Duff and Murray (1941), the thrombosis is far less extensive than would be required to explain the widespread stasis. In the present case extensive search failed to reveal any thrombi, although cortical necrosis was very advanced. The cortical necrosis produced in rabbits by staphylococcal toxins (De Navasquez, 1938) has been attributed to vasodilatation in arteries and arterioles.

There seems to be no agreement concerning the action on renal arteries and arterioles. Some authors have emphasized that vasospasm is initiated by bacterial toxins. In support of this theory Trueta *et al.* (1947) described a vascular shunt mechanism in the kidneys that diverted the blood from the

renal cortex to the medulla. Whatever the mechanism may be, there seems to be an initial agreement that some bacterial toxin starts the change which usually advances to cortical necrosis of both kidneys. The case here recorded was one in which intense toxæmia could be present following a possible acute respiratory infection.

Lesions of Other Organs

(a) *Associated Lesions.*—These are the lesions that could be produced by the same mechanism as that affecting the kidneys. The haemorrhagic cystitis described in this case was evident both grossly and microscopically. A review of all the available literature failed to reveal any mention of a similar associated condition in the previously recorded cases. The petechial haemorrhages in the scalp, the thymus, and the adrenal glands are the usual findings associated with cases of toxæmia in children. Herzog (1913) described haemorrhagic and necrotizing lesions in the alimentary tract as associated features. The necropsy findings in the present case revealed intense congestion of the stomach mucosa, with areas of haemorrhage but no evidence of necrosis.

(b) *Predisposing Conditions.*—In the present case it is important to mention delayed respiration and persistence of atelectasis as predisposing to respiratory infection. The post-mortem examination revealed no other concomitant disease or malformations.

Summary

A case of bilateral cortical necrosis and subcapsular haemorrhages of the kidneys associated with severe haemorrhagic cystitis in a 3-weeks-old infant is described and reviewed. The extent of the subcapsular haemorrhages of the kidneys and the haemorrhagic cystitis were two outstanding features not mentioned in any of the previously recorded cases. The histopathological examination of sections from both kidneys showed no evidence of thrombi in cortical vessels.

I am indebted to Professor E. Von Haam, professor of pathology, Ohio State University, for reviewing the microscopical slides. I am thankful to Dr. M. P. Hunter, former head of the pathology department of the City Hospital, Springfield, Ohio, for permission to publish this case, and to Mr. Jack Vetter and Mr. Alvin Lee for preparation of the photomicrographs.

REFERENCES

- Campbell, A. C. P., and Henderson, J. L. (1949). *Arch. Dis. Childh.*, **24**, 269.
De Navasquez, S. (1938). *J. Path. Bact.*, **46**, 47.
Duff, G. L., and Murray, E. G. D. (1941). *Amer. J. med. Sci.*, **201**, 428.
Dunn, J. S., and Montgomery, G. L. (1941). *J. Path. Bact.*, **52**, 1.
Herzog, G. (1913). *Beitr. path. Anat.*, **56**, 175.
Trueta, J., Barclay, A. E., Franklin, K. J., Daniel, P. M., and Prichard, M. M. L. (1947). *Studies of the Renal Circulation*. Blackwell, Oxford.
Zuelzer, W. W., Kurnetz, R., and Charles, S. (1951). *Amer. J. Dis. Childh.*, **81**, 2.

The new administrative headquarters in Waterloo Road, S.E.1, of the London Ambulance Service was opened on January 20. Formerly a fire station, the new premises, comprising four floors and a large garage space, have been entirely remodelled, so that not only is the L.C.C.'s ambulance service accommodation greatly improved, including a completely redesigned control room, but for the first time it has been found possible to house under one roof both the headquarters ambulance station and the administrative headquarters of the whole service. The London Ambulance Service is divided into an accident section, which deals with emergency calls, and a general section which provides vehicles for the conveyance of patients to and from hospital where less urgency is required. The accident section answers 250–300 emergency calls daily and the general section removes more than 3,500 patients a day. The average time an ambulance takes to reach a street accident is seven minutes. A central system of control enables the resources of the entire service to be used to the best advantage by diversion of work from one station to another according to the fluctuating pressure.