THE RADIOLOGICAL AND PATHOLOGICAL FINDINGS IN BILATERAL RENAL CORTICAL NECROSIS: REPORT OF A CASE SHOWING RENAL CALCIFICATION

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Recent advances in the therapy of acute renal failure in bilateral cortical necrosis have resulted in the prolonged survival of some patients. Whereas previously bilateral cortical necrosis of the kidneys was of little interest to the radiologist because its rapidly fatal course was unassociated with specific radiological features, today the often lengthened period of survival has afforded sufficient time for radiologically demonstrable calcification to occur in the necrotic cortical tissue.

In 1957 Moëll[1] described the serial radiological changes in a patient who died from cortical necrosis 116 days after the onset of oliguria. He demonstrated a progressive reduction in the size of the renal shadows, although they were enlarged in the early stages. Eventually calcification of the renal cortices became manifest. None of the cases described by Sheehan and Moore[2] and Wells *et al.*[3] showed any calcification in the kidneys, probably because none of the patients lived longer than 31 days. Recently, Lloyd-Thomas *et al.*[4] drew attention to the tram-line appearance of the calcified renal cortices, which appeared to be specific for this condition and allowed it to be distinguished from the calcification involving the cortex in exceptional cases of glomerulonephritis.[5]

It is the purpose of this paper to draw attention again to the radiological features of the condition as demonstrated during life and at autopsy, and to describe the pathological findings in a case that survived for 63 days.

CASE HISTORY

Clinical Features

The patient, a 42-year-old Coloured woman, para-13 gravida-22, was known to have been severely hypertensive for at least a year before the onset of her illness. On 3 December 1962, when 30-weeks pregnant, she suffered a severe concealed accidental haemorrhage and was admitted to the Peninsula Maternity Hospital, Cape Town, in a shocked and anaemic state. Catheterization of the bladder produced 15 ml. of blood-stained urine. After transfusion and a surgical induction she was delivered of 2 macerated foetuses, and because of persistent anuria she was transferred to Groote Schuur Hospital 4 days later. Clinical examination showed that the heart was enlarged and her blood pressure was 200/130 mm.Hg. The retinal arteries were tortuous and narrowed. The blood urea on 7 December 1962 was 185 mg./100 ml., and during the 63 days of her illness she only passed about 300 ml. of urine. The basic daily fluid replacement consisted of 600 ml. of 10% invert sugar given orally, while 4% $NaHCO_3$ was added according to daily bicarbonate estimations to counteract the acidosis. After 7 days of anuria the blood urea had risen to 476 mg./100 ml. Other serum chemistry figures at this stage were: sodium 133 mEq./l., chloride 77 mEq./l., potassium 6·4 mEq./l., HCO_3 16·1 mEq./l. The first dialysis took place on 11 December 1962. The blood urea had risen to 600 mg./100 ml. before the second dialysis was performed on 18 December 1962. A third dialysis took place on 24 December 1962, after which the blood urea was reduced to 116 mg./100 ml. with sodium 138 mEq./l., potassium 4·2 mEq./l., chloride 8·6 mEq./l., and HCO_3 29·0 mEq./l. A renal biopsy done 4 days later consisted almost entirely of necrotic renal tissue. No calcification was seen. On 11 January 1963 the final dialysis was performed, the blood urea again having risen to 304 mg./100 ml. Vomiting and increasing diarrhoea preceded her death on 4 February 1963.

Autopsy Findings

The body was that of a well-covered non-European female. The right and left lungs weighed 680 and 635 G respectively. There was no pleurisy, but on section both organs were

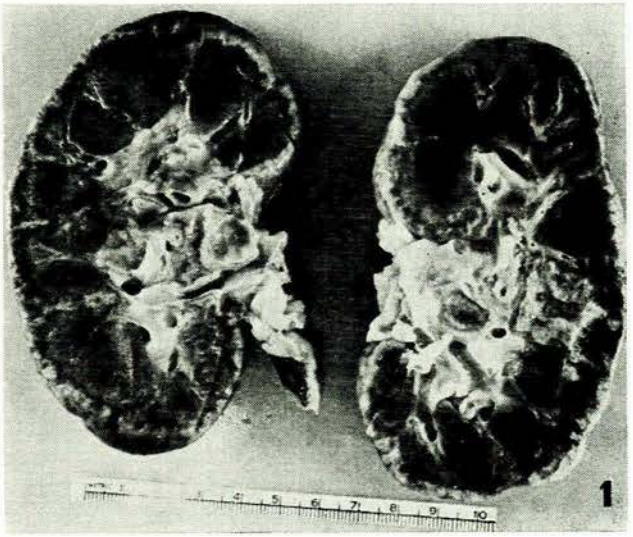

Fig. 1. The cut surfaces of the kidneys, showing the bilateral necrosis of the cortices, which are reduced in width.

diffusely oedematous. The heart weighed 382 G. The left ventricle was dilated and moderately hypertrophied. Occasional small congested foci were scattered throughout the myocardium. The spleen weighed 436 G and contained 2 small recent infarcts. The combined weight of the kidneys, which were slightly and equally reduced in size, was 280 G. Externally they exhibited a moderately coarse granularity. The appearance of the cut surfaces was that of a bilateral symmetrical cortical necrosis, which was almost complete except for a small surviving zone near one pole; it differed from the more commonly seen form in that the cortices were reduced in width (Fig. 1). The necrotic tissue was bright yellow in colour and gritty on palpation, and measured no more than 2 - 3 mm. in maximum width. A considerable loss of intermedullary cortex had also occurred. The renal pyramids were normal. The only other significant autopsy findings were focal calcification on the endometrial surface of the uterus, thrombosis of both ovarian veins, and a markedly reactive femoral bone marrow. The parathyroids were not enlarged.

Histological Examination

The presence of cortical necrosis of the kidneys was confirmed, the outlines of tubules and glomeruli still being easily discernible in the necrotic tissue (Fig. 2). A thin subcapsular zone had survived and large numbers of heavily calcified glomeruli, tubules and vessels were seen just internal to it in the necrotic tissue where the latter bordered on the viable zone. There was generally very much less calcification on the medullary aspect of the cortex though towards the hilum it was also well advanced. Areas of intense congestion were seen adjacent to the cortex, many partially necrotic glomeruli being engorged with blood, but an inflammatory reaction was inconspicuous. Afferent arterioles showed extreme hyalinization of their walls, while the lumina of interlobular and arcuate arteries were considerably reduced by concentric subintimal fibrous thickening. Occasional arteries in the boundary zone were occluded by partly recanalized thrombus.

The lungs showed oedema and intra-alveolar haemorrhages. The splenic infarcts were fresh, and several small necrotic foci were found in the substance of the organ resembling the Fleckmilz of Feitis. The congested foci in the myocardium proved to be areas of atrophy and disappearance of fibres. The parathyroids were not hyperplastic. An increase in all the normal elements was seen in the marrow. The thrombi occluding the ovarian veins contained small amounts of calcium.

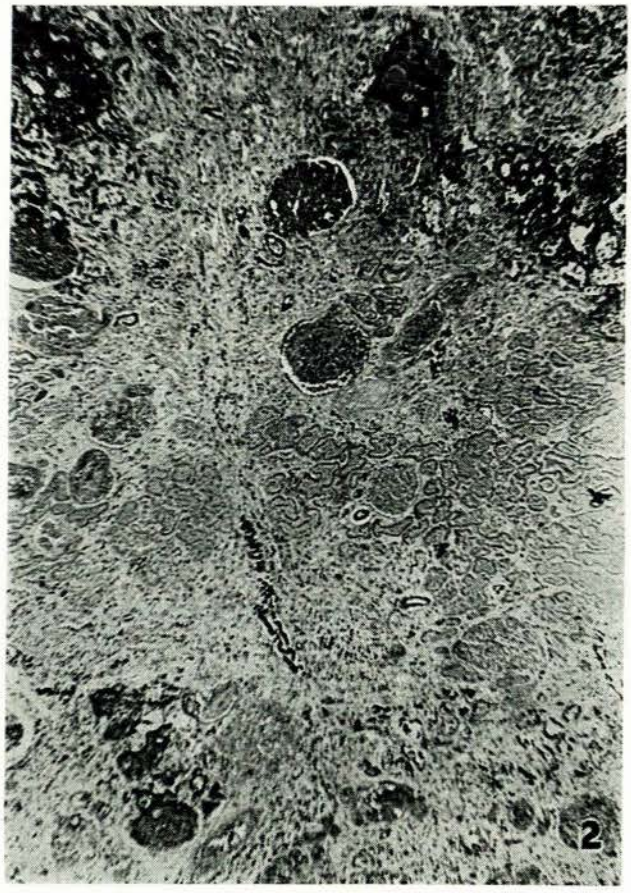

Fig. 2. A low-power view of the necrotic cortex. Calcified glomeruli and vessels are present, predominantly in the outer zones.

X-ray Findings

A tomogram of the kidneys on 21 December 1962 showed renal shadows which were thought to be larger than normal, but no calcification was visible (Fig. 3). A postmortem radiograph revealed a very widespread blotchy calcification, though only towards the hilum was there a clear tram-line effect (Fig. 4).

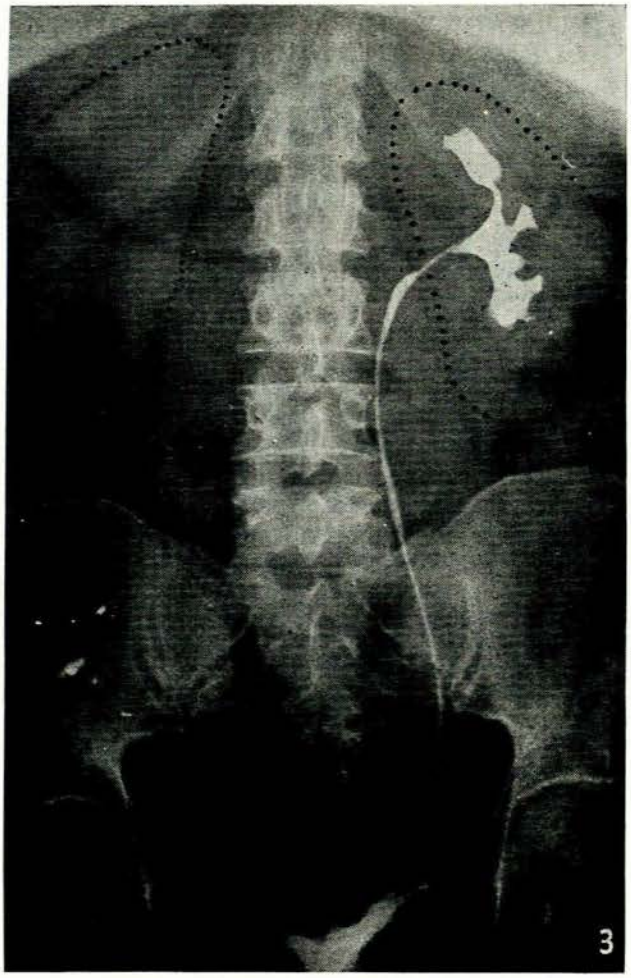

Fig. 3. The tomogram showing the dotted outlines of the enlarged kidneys.

DISCUSSION

In the early stages of bilateral renal cortical necrosis the renal shadows may be enlarged, presumably owing to oedematous swelling of the organs. This was described by Moëll[1] and McAllister and Nedelman,[6] and in the present

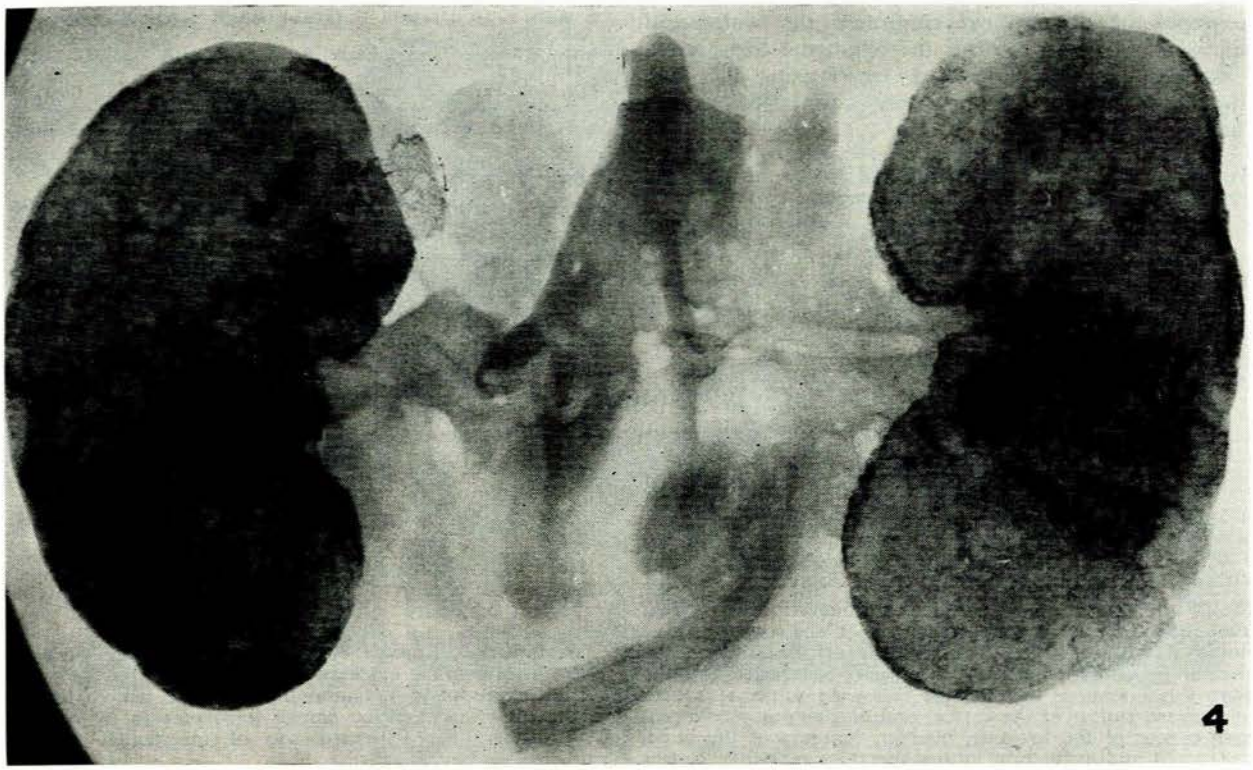

Fig. 4. The postmortem radiograph. Extensive mottled calcification is present, but only towards the hilum is the tram-line pattern easily discernible.

case the tomogram showed a similar change. At this stage a definite diagnosis depends on renal biopsy, but no idea can be gained of the extent of the lesion. The importance, therefore, of demonstrating renal calcification, as pointed out by Lloyd-Thomas *et al.*,[4] is that the extent of the necrosis can be assessed. It is upon this factor that the survival of the patient primarily depends, for those few cases that have survived are known to have usually been characterized by a patchy distribution of the necrosis. Exceptions appear to be the cases reported by Gormsen *et al.*[7] and Oram *et al.*[8] It should also be recalled that calcification in the renal cortices occurs in conditions other than cortical necrosis, and has also been described as occurring in the cortex in very rare cases of glomerulonephritis. The value of the procedure is unfortunately greatly limited by the fact that a minimum period of 1 - 2 months must elapse before calcification becomes radiologically demonstrable.[4]

Although the renal calcification in our case was very extensive, there was definitely much more of it in the outer part of the necrotic tissue than in the juxtamedullary parts of the cortex, which only towards the hilum showed appreciable calcification, the postmortem radiograph confirming this with a classical tram-line effect. It should be stressed therefore that this radiological sign, though quite possibly specific for cortical necrosis, may be incompletely developed even in a case such as ours with prolonged survival and widespread calcification. The distribution of calcification predominantly in the outer cortical zones is of some interest, for it probably implies that the subcapsular blood flow must have been much better than that on the medullary aspect of the cortex, thus permitting the various salts involved in the process of calcification to reach the necrotic cortex. This has been observed by Sheehan and Moore in reviewing one of Grasby's cases.[9] The development of a tram-line pattern of calcification as described by Lloyd-Thomas *et al.*[4] is presumably dependent on the successful re-establishment of the circulation of blood on both sides of the necrotic cortex. The factors determining this are uncertain, while it is also not clear how frequently it will occur. The importance of the circulation in the deposition of calcium has also been emphasized by Phillips.[10]

It should finally be pointed out that calcification may occur in sites other than the kidney. In the present case, as in that reported by Lloyd-Thomas *et al.*,[4] calcification was seen in the thrombi in the ovarian veins and uterus, while Alwall *et al.*[11] observed it in the myocardium in one of their cases. These findings do raise the possibility that in certain cases a tendency to more widespread dystrophic calcification may exist.

SUMMARY

Bilateral renal cortical necrosis developed in a woman following accidental uterine haemorrhage. She survived for 63 days, during which period she was dialysed 4 times. Tomograms revealed kidneys that were larger than normal.

At autopsy the kidneys showed extensive calcification in the necrotic cortices, which were much reduced in thickness, while the ovarian vein thrombi and the endometrial surface of the uterus were also partly calcified. Postmortem

radiographs of the kidneys confirmed the widespread calcification, but only towards the hilum did this present the diagnostic tram-line pattern, emphasizing that this feature may be very incompletely developed even in advanced cases.

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