SYMMETRICAL CORTICAL NECROSIS OF THE KIDNEYS *

REPORT OF A CASE

NEWTON EVANS, M.D., AND ELMER W. GILBERT, M.D.

(From the Department of Pathology, College of Medical Evangelists, and the Pathology Laboratory of the Los Angeles County Hospital, Los Angeles, California)

This peculiar lesion has excited interest for many years. The first case was reported in 1886 by Juhel-Rénoy [17] of France. To date 53 proved cases have been recorded, mostly by English and German observers. About 14 cases have been reported in the American literature. In 1933 Ash [1] made an extensive search, gathering 62 recorded cases, 18 of which were not definitely proved as some had had no portmortem examination, in others the individuals had recovered, and 1 or 2 cases had had a biopsy on one kidney only. In our search of the literature we have found 8 additional cases [2, 18, 25, 27, 28, 30] which we think belong to this group.

A review of the literature reveals a fairly distinct clinical picture. The patient may be male or female, but the pregnant female is by far more often the victim. The age period is wide but it is evident that the child-bearing years are the usual time of occurrence. The onset of illness is abrupt or insidious, beginning late in the last trimester of pregnancy, or even at term. In other cases symptoms make their appearance in the second trimester. The usual picture at first is that of some form of toxemia of pregnancy, *e.g.* headache, nausea, vomiting, epigastric pain, and even convulsions. Examination at this time usually reveals the absence of fetal heart tones. When the convulsions begin, spontaneous or induced delivery usually occurs shortly and a dead fetus is born. Only once were living infants born, they being twins. Almost invariably following delivery the urinary output is either markedly decreased or is nil. Such urine contains pus, red blood cells, casts and much albumin. The blood pressure is frequently elevated but about as often is normal. Nitrogen retention is common. It is remarkable that the mental state of the individual is so often clear, almost to the end, which comes generally after about 5 days of partial or complete anuria. A

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few cases have been described in which the course appeared fairly typical and the patient recovered, but in these the urinary suppression was less complete.

The kidney lesion was notably uniform in all instances. Necrosis simulating infarction involved all the cortical portion and the columns of Bertin of both kidneys, with thrombosis of the vessels therein, but without occlusion of the arteries proximal to these vessels. The average weight of both kidneys was 413 gm. Only on three occasions did typical lesions of eclampsia appear in the liver. Many times the placenta showed signs of decomposition.

The incidence of etiological factors and symptoms from cases reviewed in the literature can be summed up as follows:

INCIDENCE OF ETIOLOGICAL FACTORS

Sex	Female	45 (86%) (41 pregnant)
	Male	7 (14%)
Age	13 to 65 yrs., average 32	

PRIMARY ETIOLOGICAL CONDITIONS

Pregnancy	41 (77%)
Intravenous camphor, acute tonsillitis, scarlet fever, diphtheria, malaria, myocardial infarction, pulmonary tuberculosis, ruptured liver, carcinoma of prostate, cryptogenic.	each 1

INCIDENCE OF SYMPTOMS

	%
Anuria	100
Convulsions	34
Headache	32
Vomiting	55
Edema	55
Coma	42

A case presenting the classical symptoms of cortical necrosis of both kidneys has recently come under our observation and a brief report follows.

REPORT OF CASE

Clinical History: The patient, a Negress, was first seen at the age of 33 years, in March, 1926. During her 8½ months of pregnancy there were present, edema of the ankles, headache, vague abdominal pain, and vomiting. On admittance to the hospital the temperature was normal, the blood pressure 220/120, non-protein nitrogen 46 mg., and carbon dioxide combining power 40 vol. per cent. The urine contained considerable red blood cells, albumin, and a few hyaline and granular casts. Blood Wassermann was negative. A living child was

born. Ten days postpartum the blood pressure was 180/130 and 2 weeks later 190/120.

In October, 1930, after 6½ months of pregnancy, during which time nausea, vomiting, vertigo, headache, visual disturbance, and edema of the ankles were present, she was again admitted to the hospital. A convulsion had just occurred, but the patient was conscious. The blood pressure was 190/135. The urine contained 24 per cent albumin by volume, and numerous pus cells. Blood non-protein nitrogen was 33 mg. and carbon dioxide combining power 39 vol. per cent. After 4 days a dead macerated fetus was delivered. Ten days later the blood pressure was 140/100 and the urine albumin 7 per cent by volume.

On the morning of Sept. 30, 1932, she was brought to the hospital for the third and final admission. At this time she was 39 years of age, para V, gravida VIII, and 7 months pregnant. She was drowsy and unable to answer questions, but her husband stated that she had complained for several months of nausea and vomiting, epigastric pain, spells of vertigo, headache, and edema of the ankles. About September 24th she had had a convulsion, and another one on the day of admission.

Physical examination revealed moderate edema of the ankles. Fetal heart tones were absent. The urine obtained was of insufficient quantity for specific gravity determination, coagulated on heating, and contained many pus cells and fine and coarse granular casts. Red blood cells were 3,600,000 and hemoglobin 45 per cent. There was a third convulsion the evening of the day of admission and labor pains had begun. On the 2nd day a 3 pound, stillborn fetus was delivered. The placenta which followed was fragmented and decomposed. There was only a moderate amount of bleeding. The patient remained unconscious until death occurred on the 5th hospital day, 3 days postpartum.

The blood pressure on admission was 135/95. Following the convulsion that same evening it was 170/90. Preceding delivery it was 170/110; immediately following, 155/90 and 2 hours later it was 200/105. From that time until shortly before death it ranged between 190/100 and 140/80.

Blood chemistry determinations were made twice. On the 1st day non-protein nitrogen was 52, creatinine 3.5 and carbon dioxide combining power 25. The following day the non-protein nitrogen was 85, creatinine was not determined and the carbon dioxide combining power was 35.

The anuria appears to be the most significant feature. On the day of last entry an insufficient amount of urine for specific gravity determination was secured. The 2nd day none was recorded, the 3rd day only 2 ounces could be obtained and on the 4th and 5th days there was total anuria.

Postmortem Examination

Autopsy was performed 5 hours postmortem. The body was well nourished. There was moderate edema of the feet and ankles. The brain showed generalized congestion and a moderate amount of subarachnoid edema. The lungs together weighed 940 gm., were congested, edematous and presented a few subpleural petechial hemorrhages. The heart weighed 320 gm. and contained several areas of ecchymosis beneath the endocardium of the left ventricle.

The liver weighed 1600 gm., and in appearance was suggestive of fatty degeneration, but there were no lesions typical of eclampsia. The uterus weighed 730 gm. and was moderately firm. Numerous ecchymoses averaging 1 cm. in diameter were present beneath the serosa over the whole organ. The endometrium was foul smelling and was covered by soft, dark red decomposed blood clots. The left ovarian vein throughout its midportion contained an ante mortem thrombus 5 cm. in length. Both adrenals presented cortical hemorrhagic areas. The mucosa of the urinary bladder was hemorrhagic and 2–3 cc. of thick yellow fluid were present.

The kidneys together weighed 380 gm. and were about equal in size. The capsules stripped with ease and were slightly thickened and congested. The entire surface of each kidney was yellow except for mottling with small areas of hemorrhage. Several of these areas were depressed and fibrosed. On section (Fig. 1) practically the entire cortex of the kidneys had the characteristic appearance of classical infarction but for its distribution. The necrotic areas were light yellow with hemorrhagic borders. Almost all portions of the columns of Bertin were uniformly involved while the cortical infarction was interrupted at intervals by less necrotic tissue. The pyramids appeared normal except for evidence of congestion. There were a few petechial hemorrhages beneath the pelvic mucosa. The renal vessels presented no gross evidence of thrombosis.

Microscopic Examination

Consistent with the gross pathology, the most striking histological changes were in the kidneys. The cortical structures, including all the tissue elements, were necrotic except for minute areas adjacent to the pyramids and the narrow zone beneath the capsule, which was not of sufficient depth to include any glomeruli. The greater part of the infarcted cortical tissue had a strikingly normal appearance and arrangement, except for the acute necrotic changes. The glomeruli, though somewhat swollen, looked like normal glomeruli which had suddenly necrosed. No glomeruli could be found which had been distorted or sclerosed, as would be the case in either primary or secondary contracted kidney. The sections examined showed a few sclerotic areas corresponding to the depressed surface scars seen grossly. The complete involvement of the cortical labyrinth is em-

phasized by the fact that only the rarest glomerulus could be found which took the nuclear stains as living tissue should (Fig. 2).

The convoluted tubules were all necrosed. They appeared swollen but had, for the most part, a normal arrangement and relation to each other, the necrosed epithelial cells fused with a homogeneous material filling the lumens. There were places, however, where it was evident that a minor amount of interstitial fibrosis had occurred, but it was small in amount. In numerous areas the intertubular tissue was filled with nuclear staining granular material as though a cellular exudate had collected and then disintegrated along with the necrosis of the fixed tissue elements.

The vascular elements of the cortex showed what were perhaps the most striking features. All of the vessels in the necrotic areas were likewise necrosed, but were distended with thrombotic material looking like white thrombi composed of platelets and fibrin. The most notable appearance was made by the rounded, thin walled, distorted, interlobular arteries and their branches, the afferent arterioles. For the most part these arterioles were distended with thrombotic material, but some gave the appearance of complete necrosis, the lumens closed by hyaline swelling of the walls, leaving no room for the thrombi (Fig. 3).

Liver: Microscopically the liver sections had a surprisingly normal appearance. In a few places the interlobular arterioles gave the faintest suggestion of hyaline degeneration.

Spleen: Splenic arterioles presented an intense grade of hyaline degeneration, producing an appearance in many vessels of closure of the lumens.

Adrenal: Section of one of the adrenals presented two striking findings — extensive focal necrosis of the cortex, and in one place a minute cortical adenoma.

Discussion

The following questions are pertinent: (1) What was the real character of the final attack? Should it be looked upon as eclampsia? Was the death an eclamptic death?

That it partook of the nature of an eclampsia is supported by its association with pregnancy and by its characteristic eclamptic symptoms. That it was not a classical non-nephritic or hepatic type of eclampsia is indicated by the absence of the characteristic hepatic

lesions and by the evident changes in the kidneys at variance with the simple tubular degenerative changes and the glomerular changes of classical eclampsia described by Bell and others. Furthermore, that it was not an eclampsia with nephritis (nephritic toxemia) is indicated by the absence of histological evidence of either an acute or chronic glomerular nephritis. Nor was it an eclampsia with chronic hypertensive disease, since the absence of cardiac hypertrophy rules out persistent arterial hypertension. On the other hand, it is reasonable to conclude that death was due to the cortical renal necrosis, anuria and uremia.

(2) What was the nature of the two previous (eclamptic) attacks? Were they non-nephritic eclampsia or were they nephritic?

That they were of the nature of eclampsia seems evident, although in the first of the two attacks, convulsions are not recorded. The absence of any examination of the patient between attacks makes it impossible to say with confidence that she did or did not have a true nephritic lesion. However, the absence of definite histological evidence of chronic nephritis at autopsy would rule out such a condition.

(3) What is the pathogenesis of the cortical necrosis? What is the cause of the vascular thrombosis?

Beginning with the first reported case, various hypotheses have been advanced as to the exact pathogenesis of the infarction. Juhel-Rénoy, in his case following scarlet fever, thought it was due to multiple emboli. Others believe it is primarily due to the action of some toxic agent on the renal tissues. Ash has recently advanced the idea of angiospasm resulting in stasis and thrombosis and consequent infarction. It is believed by almost all the writers that the thrombosis is primary and the tissue necrosis secondary, and not that the thrombosis occurs simultaneously with or subsequently to necrosis of the kidney parenchyma.

Our concept of the sequence of events in this lesion is the presence of some toxic substance in the circulating blood capable of producing injury to the capillary and arteriolar walls in large areas of the kidney cortex sufficient to cause extensive thrombosis and consequent necrosis of the tissues nourished by these vessels. It is, of course, conceivable that the changes in the involved vessel walls may include either spasm, paralysis or both.

Summary and Conclusions

A brief review of the recorded cases of cortical necrosis of the kidneys is presented.

A case with autopsy findings, which is unique on account of previous eclamptic attacks, is reported.

The immediate cause of death was acute renal failure with anuria.

While the clinical syndrome is apparently that of eclampsia, it is not possible to assign it to one of the recognized pathological types of this disease.

A brief discussion of the possible pathogenesis of the kidney lesion is presented.

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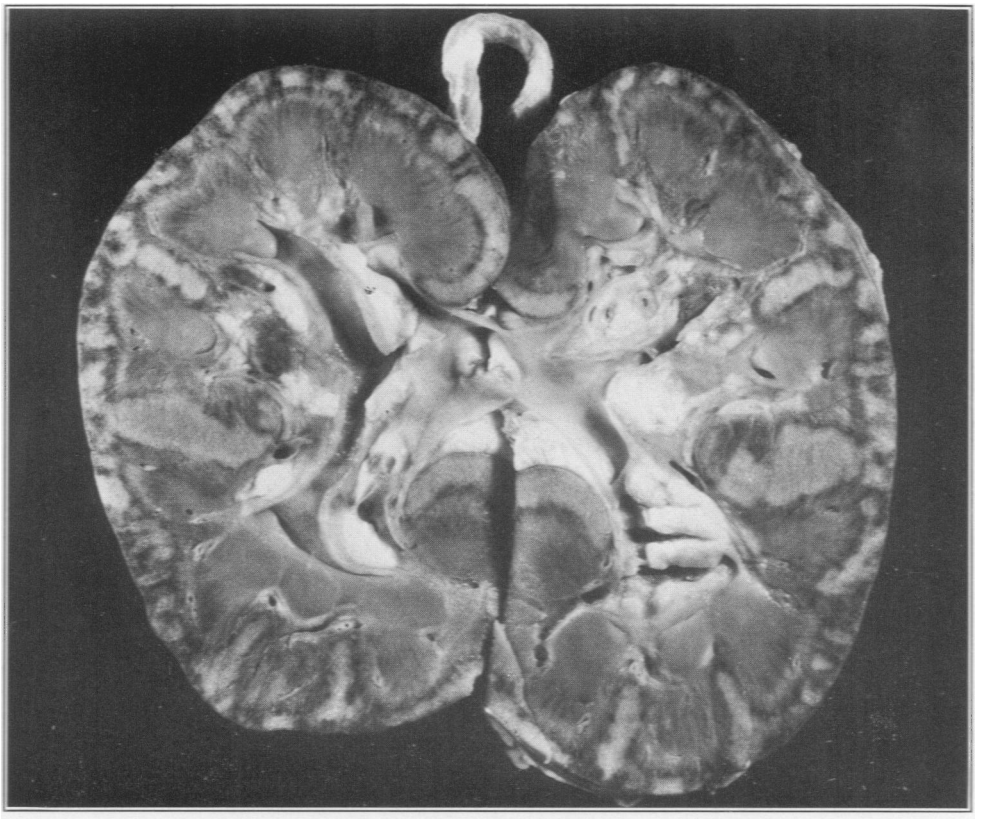
DESCRIPTION OF PLATE

PLATE 102

FIG. 1. Kidney showing practically complete necrosis of the parenchyma of cortex and columns of Bertin. The pyramids appear normal. The light colored necrotic areas have dark hemorrhagic margins.

FIG. 2. Section of cortex of kidney showing margin of infarcted area. The upper two glomeruli and tubules are not involved in the necrotic process. Below are completely necrotic tubules and glomeruli. Hematoxylin-eosin stain. × 95.

FIG. 3. Section of cortex of kidney in infarcted area. Above is a dilated interlobular artery filled with thrombus. Leading from it is an afferent arteriole distended with thrombus, and its glomerulus. Mallory's aniline blue stain. × 95.



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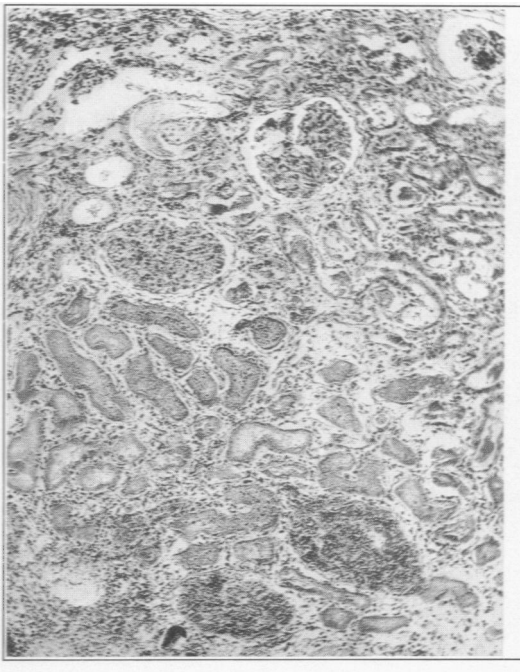

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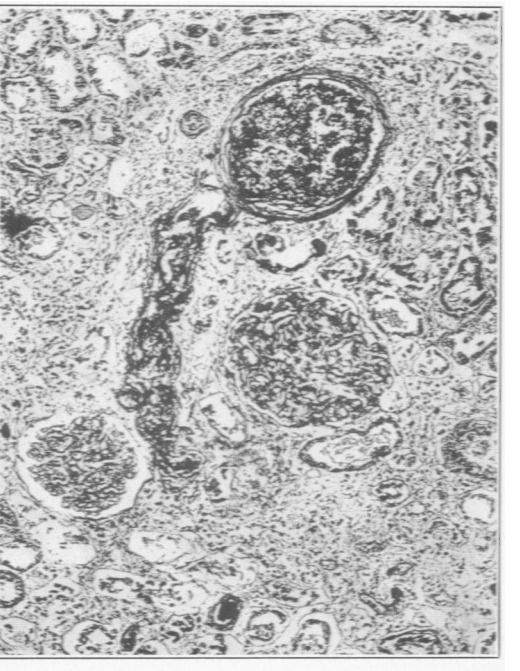

3

Evans and Gilbert Symmetrical Cortical Necrosis of Kidneys