The Development of Hypertension after Rupture of a Kidney

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Kidney injuries have been more frequent in the last two decades, mainly because of increased automobile accidents in all countries. Less frequent causes are injuries connected with occupation and sports. Blunt trauma may result in organic change, rupture being one of the most serious. After the acute stage various complications may occur as stated by Hodges:1 “Patients who are treated nonsurgically often undergo a protracted convalescence and remain ‘renal cripples’ for weeks, months or even years following their injuries.”

This applies particularly to posttraumatic renal hypertension that may appear early within a few weeks after the injury or after months or even years. Some authors2 believe that it is the most frequent complication of the renal trauma, but others find it only sporadically. Renal arteriography enhances the possibility of precise diagnosis.

Arteriographically well-documented reports of kidney rupture followed by hypertension are rare.3,4 We were able to observe five cases of kidney rupture in approximately 600 arteriographs. Three of these five patients developed hypertension and two are briefly reported.

Case Reports

Case 1

D. J., a 21-year-old man, fell on his right flank hitting a stone. After regaining consciousness he felt severe pain in his back and right epigastrium. He found it difficult to breathe, and voided blood at the first-aid station, which he reached by “crawling on all fours.” Right-sided ballottement was positive; the right hypogastrum was intensely painful. The blood pressure was 125/70, pulse rate 84, temperature normal. The white blood-cell count was 11,300 leukocytes, per mm.3; the sedimentation rate was normal. Urine contained albumin, white cells, and erythrocytes. On the third day after injury the blood pressure was 150/100. Urography showed good excretion on both sides but meteorism prevented evaluation of the kidney shape and the calyceal systems.

The patient came to our hospital 40 days after the accident because of persistent hematuria and hypertension of 170/100. Repeat urography showed near disappearance of the right psoas and kidney contours, but renal excretion was prompt. The middle calyx was greatly deformed and dislocated downward; the ureter was bent laterally. Renal arteriography revealed transverse rupture of the middle third of the right kidney (fig. 1). The two halves were displaced, but extravasation of contrast medium was not observed. During the venous phase, the upper half was somewhat more saturated and the lower half had the smooth surface of rupture. All this indicated a complete transverse rupture of the right kidney with deformation of the middle calyx and dislocation of the ureter by hematoma. The patient was treated conservatively. Eighteen months after injury he was still hypertensive, 220/100; nephrectomy was therefore advised. The patient refused the operation.

Case 2

Ch. J., a 20-year-old man, was involved in a lorry accident. He was transported by air to the Neurosurgical Clinic and operated on for fracture of the calvarium. In addition, he had an open wound over the right eleventh rib, persisting hematuria and slight signs of peritoneal irritation. Blood pressure was 100/60, temperature normal. The blood cell count was 3.3 million erythrocytes and 6,200 leukocytes per mm.3. Urography revealed dislocation and deformation of the right lower calyx. At abdominal aortography performed 3 weeks after injury, the right artery and its branches to the upper half were well filled while the lower-half arteries were not visible. We concluded that the right kidney was ruptured and its lower half infarcted. The patient was treated conservatively but had to be hospitalized three times in the course of a year because of repeated renal colic. When last examined, 3 years after the in-

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HYPERTENSION AFTER RUPTURE OF KIDNEY

I. HYPERTENSION

AFTER RUPTURE OF KIDNEY

1. Coldblatt’s mechanism in cases of kidney ischemia when renovascular hypertension develops as a result of thrombosis, spasm, or stenosis of the renal artery or of an arteriovenous aneurysm. In most cases the hypertension is permanent, but sometimes, particularly in kidney infarction, it is only transient.

3. Other mechanisms are pyelonephritic and shrinking processes caused by infection, lithiasis, and stasis.

This brief survey shows the great variety of changes taking place when a kidney is ruptured and how difficult it is to gauge accurately qualitative and quantitative aspects of the damage.

The routine methods of x-ray examination as represented by plain urogram, pyelogram, and excretory urogram can reveal not more

- **Figure 1**

*Complete rupture of the right kidney. Selective renal arteriography 40 days after the injury-arterial phase.*

- **Figure 2**

*Appearance after rupture of kidney and infarction of the lower half with shrinkage and lithiasis. Renal arteriography 3 years after the injury-arterial phase: a small artery for the lower pole, calculus in the lower calyx.*

**Discussion**

Renal hypertension is not a constant sequel of kidney rupture. This is evidently because it is not produced only by a lesion of the parenchyma, but may be due to other factors, such as lesions of the kidney capsule, pelveocalyceal system and arterial tree. It is, essentially, a combined result of several mechanisms.

1. Page’s mechanism, producing a cellophane-type perinephritis. Hypertension in this condition is due to the compression of the kidney by a subcapsular or perirenal hematoma or to the constriction of the kidney by a fibrous thickened capsule and cyst, formed while the hematoma is healing.

2. Coldblatt’s mechanism in cases of kidney ischemia when renovascular hypertension develops as a result of thrombosis, spasm, or stenosis of the renal artery or of an arteriovenous aneurysm. In most cases the hypertension is permanent, but sometimes, particularly in kidney infarction, it is only transient.

3. Other mechanisms are pyelonephritic and shrinking processes caused by infection, lithiasis, and stasis.

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The routine methods of x-ray examination as represented by plain urogram, pyelogram, and excretory urogram can reveal not more
than 40 to 45 per cent of kidney injuries, and are not satisfactory. This is understandable, since they do not demonstrate the arterial network, so important in the development of the posttraumatic renal hypertension.

Renal arteriography is an important advance and will increase the percentage of positive findings. It was first used in late traumatic states and subsequently found to be suitable for diagnosing acute injuries even in children. It is valuable particularly in cases with nonvisualization on urography and when infection or injury to other parts of the urinary tract preclude pyelography. Another advantage of renal arteriography is the possibility of demonstrating preexisting diseases, such as tumor, hydronephrosis, or cyst, which greatly reduce the resistance of the kidney to physical stress. In our cases this method provided convincing demonstration of partial or total kidney rupture and infarction. The patients tolerated the examination quite well even a few days after their accident. It can be recommended not only for diagnosing acute kidney injuries but for elucidating the causes of posttraumatic renal hypertension, which, as a unilateral renal disease, is likely to respond to definitive treatment.

**Summary**

Two cases of kidney rupture associated with hypertension are described, and the multiformal traumatic changes causing it are surveyed. Renal arteriography is recommended not only in the early but also in the late stages of hypertension of this type, which responds to definitive treatment.

**References**


**The Enigma of Creativity**

People without historians would be as crippled as an individual with amnesia. . . .

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