A case of spontaneous recanalization following renal infarction

AUTHOR(S):
Hibi, Hatsuki; Itoh, Kohichi; Mitsui, Kenji; Yamada, Yoshiaki; Shimoji, Toshio

CITATION:
Hibi, Hatsuki ...[et al]. A case of spontaneous recanalization following renal infarction. 泌尿器科紀要 1993, 39(2): 159-162

ISSUE DATE:
1993-02

URL:
http://hdl.handle.net/2433/117778

RIGHT:
A CASE OF SPONTANEOUS RECANALIZATION FOLLOWING RENAL INFARCTION

Hatsuki Hibi and Kohichi Itoh
From the Department of Urology, Tosei General Hospital
Kenji Mitsui and Yoshiaki Yamada
From the Department of Urology, Aichi Medical University
Toshio Shimoji
From the Department of Urology, Nagoya University School of Medicine

A 30-year-old-male was admitted to our hospital with a tentative diagnosis of appendicitis. We found no signs of peritonitis, and therefore suspected urinary tract calculi.

Intravenous pyelography revealed his right kidney to be nonfunctional, while retrograde pyelography was normal. Angiography revealed right renal infarction. Renovascular hypertension was present, and treatment with captopril was prescribed. Two years later without medication he had normal blood pressure and plasma renin activity. Intravenous pyelography revealed a right kidney functioning well, and digital subtraction angiography (DSA) revealed spontaneous recanalization of his right renal artery.

Key words: Renal infarction, Spontaneous recanalization

INTRODUCTION

Although renal infarction is well known as a serious acute disease in the urological field, it is rarely found on routine examination. The present paper documents a spontaneous cure of renal infarction in a young male.

CASE REPORT

A 30-year-old-male presented with mild abdominal pain. His body temperature was 36.2°C, and his white blood cell (WBC) count was 10,700/mm³. He had no localized tenderness. No other blood values were abnormal, and conservative treatment was initiated with a tentative diagnosis of acute appendicitis. Two days later his WBC had decreased to 6,800/mm³, and he had no abdominal pain. Two days later he suffered another attack of abdominal pain and he was readmitted.

Blood and urine data were all within normal limits except WBC 10,500/mm³, LDH (lactic dehydrogenase) 286 IU/l and CRP (C-reactive protein) 16.3 mg/dl.

Abdominal findings again showed no peritonitic signs, and he was sent to our Department for suspicion of urinary calculi.

Renal echography gave a normal picture, but intravenous pyelography (IVP) disclosed his right kidney to be severely disturbed. No obstruction of the right ureter was noted upon retrograde pyelographic testing (RP). Renoscintigraphy showed very poor RI (radio isotope) uptake. We therefore began to suspect that his pain might have been caused by renal infarction, and initiated angiographic testing. This revealed his right main renal artery to be totally occluded, and that his collateral arteries were poorly enhanced. There were prominent dilatation of his superior mesenteric artery, irregularly curved aorta, and some irregularities in the internal wall of the aorta. All these findings suggested arteriosclerosis.

CLINICAL COURSE

Body temperature, WBC, and LDH were at their peak upon admission, and were brought to within normal limits one week later. His abdominal pain subsided with conservative treatment. However, his
blood pressure rose to 160~180 mmHg systolically, and his diastolic phase rose to 90 ~110 mmHg about 10 days after admission. His plasma renin activity (PRA) value (8.0 ng/ml/hr) lead us to a diagnosis of renovascular hypertension. We initially treated him with captopril 37.5 mg/d, and eventually increased the dose to 75 mg/d whereby his blood pressure could be controlled. The etiology of his renal infarction was unknown, although factors such as cardiac valve disorder, hyperlipidemia, and autoimmune disease could be ruled out. He was discharged from our hospital, remained well and asymptomatic for a month. Thereafter he did not consult us. Two years later, he had normal blood pressure as well as PRA values (0.1ng/ml/hr) without medication. IVP gave a normal right pyelogram except for mild atrophy and digital subtraction angiography (DSA) revealed recanalization of the right renal artery.

**DISCUSSION**

Causes of renal infarction include: cardiac disorders, post-abdominal aortic surgery, post-angiographic testing, autoimmune disease, and tumor invasion, segmental renal cortical infarction in adults during pregnancy, labor, acute inflammation, poisoning, trauma, and severe dehydration. Differential diagnosis from other acute abdominal problems is difficult, and although laboratory data may reveal leukocytosis, or high ESR (erythrocyte sedimentation rate), LDH, and GOT (glutamate oxaloactate transaminase) values may be present in cases of renal infarction, our patient was an exception. His symptoms were mild as compared with cases presented in other reports with only slight elevation of WBC and LDH.

Renal echography, IVP, RP, computed tomography (CT), and angiography are all of assistance to make a definitive diag-
nosis of renal infarction. Takeshima et al.¹ recommend employing DSA instead of arteriography as the latter may itself induce renal infarction.

While spontaneous recanalization of the renal artery has been described in the literature, Ouriel et al.², reported that functional recovery from a traumatic renal infarction could not be expected within the first 6 hours after its occurrence. When renal artery emboli are the result of arteriosclerosis or fibromuscular dysplasia, there is significantly richer collateral growth around the perirenal pelvis, periureter, and renal capsule and there have been several reports of improvement of stenosed renal arteries after treatment of hypertension or hyperlipidemia³–⁵. This was related to spontaneous cures of renovascular hypertension, Takayasu's disease and fibromuscular dysplasia. Renal cortical infarction in infants resulting from upper bronchial infections or gastroenterocolitis has, like adult infarctions, also been known to occasionally improve spontaneously⁶.

The reason for the occurrence of spontaneous recanalization may be the result of thrombolysis and/or a decrease in the size of the thrombosis. Takeshima et al.¹ reported the only previous case of spontaneous recanalization in Japan. Renal infarction has been treated with the following modalities: 1) surgical thrombectomy, 2) systemic or local anti-coagulant therapy, and 3) angioplastic balloon catheterization. The efficacy of procedures 1) and 2) has been reported to be marginal at best, and conservative treatment was the avenue of choice of Manley et al.², who rejected surgical intervention. In our case, anticoagulant therapy was too late from its occurrence. Since the result of surgical intervention has not been good, he did not hope that. Therefore we chose conservative treatment. Sadoon et al.⁸, on the other hand, related that 80% of cases of emboli caused renal failures could be helped by transluminal angioplasty.

Renal ischemia experiments revealed only minimal changes in the glomerular basement membrane (GBM) when the ischemia lasted for 3 hours⁹, and that glomerular function was completely restored when the collateral flow to the kidneys was abundant. Collateral circulation to the renal cortical vascular plexus flows from the lumbar, intercostal, inferior adrenal, gonadal and internal iliac arteries. The percolating branches of the renal cortex flow to the lateral portion of the renal cortex and Anastomose with arched branches¹⁰. Renal function in these cases can be maintained as a result of an abundant blood flow.

In conclusion, when kidney disease is not acute or complicated, conservative treatment and careful blood pressure control are the alternative of approaches.

**SUMMARY**

1) This report concerns a rare case of recanalization after renal infarction in a 30-year-old man.
2) Although he first showed renovascular hypertension, this healed spontaneously as a result of recanalization of his renal artery.
3) Renal functional recovery was complete with the exception of mild atrophy of the kidney.
4) This is the second such case report in the Japanese literature.

**REFERENCES**


(Received on July 2, 1992) (Accepted on October 27, 1992)