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MITRAL STENOSIS COMPLICATED BY RENAL ARTERY EMBOLISM

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ABSTRACT

Six patients with mitral stenosis complicated by renal artery embolism studied prospectively are presented. In five patients there was unilateral and in one patient bilateral renal artery involvement. The diagnosis was confirmed by intravenous pyelography, retrograde pyelography, renal angiography, renal scan or a combination of these. All had atrial fibrillation, proteinuria and elevated LDH levels. Two had developed hypertension at the initial presentation. All patients were treated with anticoagulants and other supportive measures. One developed reversible acute renal failure and the others had an uneventful hospital course. Subsequent I.V.P. and scans revealed nonfunctioning shrunken kidney in just one patient with complete obstruction of the main renal artery. We conclude that anticoagulation is an adequate substitute to embolectomy. The main role of surgery is in occasional patients who remain anuric despite medical therapy and in those whose angiography reveals complete occlusion of the main renal artcry.
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CASE REPORTS

CASE 1

A 48-year-old woman was admitted because of sudden severe left flank pain associated with vomiting and profuse sweating. Physical findings included irregular pulse, signs of mitral stenosis, and left flank tenderness. Laboratory tests revealed: WBC 14300/mm^3,urinalysis: 2^+ proteinuria, 16-18 WBC/HPF, 8-10 RBC/HPF, LDH: 2000 international unit (normal 80-225), BUN: 16mg%, creatinine: 1.4mg%. I.V.P. revealed non-functioning left kidney with normal right kidney. Retrograde pyelography was normal. Heparin was started followed by an oral anticoagulant. Three months later she had no complaint and was normotensive. Repeated I.V.P. showed bilateral normal excreting kidneys. The left kidney was slightly smaller than the right kidney.

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CASE 2

A 24-year-old woman who was a known case of mitral stenosis was admitted due to sudden abdominal pain radiating to left flank. Physical examination revealed BP 150/110 mm Hg, irregular pulse, and signs of mitral stenosis. There was generalized abdominal tenderness with no rebound tenderness. Laboratory tests showed WBC: 33900/mm^3, urinalysis: 3^+ proteinuria, many RBCs/HPF, 6-7 WBC/HPF, BUN: 22 mg%, creatinine: 1.2 mg%, serum LDH: 2220 I.U.

I.V.P. revealed delayed excretion by the right kidney and a non-functioning left kidney. Renal angiography showed occlusion of branches of both renal arteries (Figure 1). She was oliguric. Heparin was started. BUN and serum creatinine rose gradually up to 90 mg% and 8 mg%, respectively. One session of hemodialysis was done and urine output gradually increased. Two months later she was normotensive with normal kidney function tests. Repeated I.V.P. was normal.

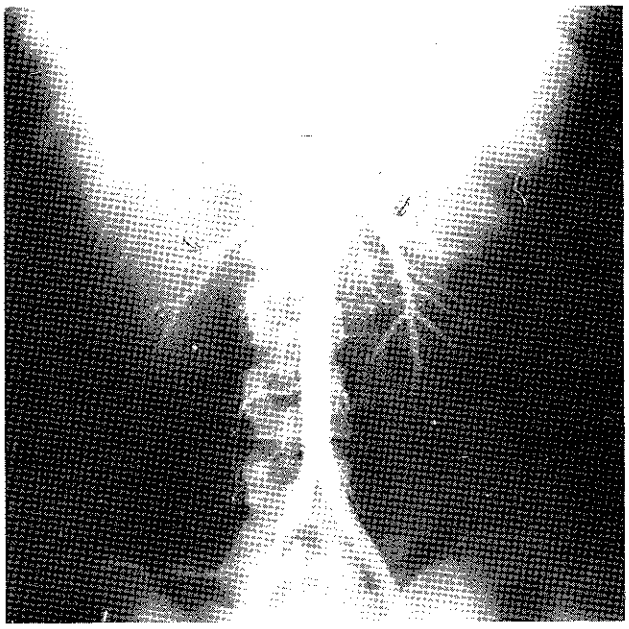

Fig 1. Renal angiography of case # 2 demonstrating bilateral renal artery obstruction (arrow).

CASE 3

A 33-year-old woman who was a known case of mitral stenosis was admitted with a two day history of night flank pain associated with fever and nausea. She had a normal vaginal delivery one week prior to admission. Positive physical findings included T: 38.8°C, tachycardia with irregular pulse, signs of mitral stenosis, and right flank tenderness. Laboratory tests revealed WBC: 14500/mm^3, urinalysis: 2^+ proteinuria, BUN: 17 mg%, creatinine: 1.1 mg%, serum LDH: 787 I.U. Blood and urine cultures were negative. I.V.P. revealed non-visualization of the right kidney (Figure 2). Renal angiography showed occlusion of a small branch of the right renal artery. Renal scan was performed three weeks after admission. Dynamic images at five-minute intervals showed diminished and delayed flow to the smaller right kidney. Posterior static imaging showed decreased concentration of radionuclide in the same kidney. The patient was discharged with anticoagulants. Mitral valve replacement was advised.

CASE 4

A 50-year-old man, a known case of mitral stenosis, referred with sudden severe right flank pain radiating to the right testis, associated with bloody urine. Positive physical findings included fever, irregular pulse, signs of mitral stenosis, and right flank tenderness. Laboratory tests revealed WBC: 13900/mm^3, urinalysis: 3^+ proteinuria, many RBCs/HPF, BUN: 24mg%, serum creatinine: 1.4mg%, LDH: 923 international units. I.V.P. showed a non-functioning right kidney.

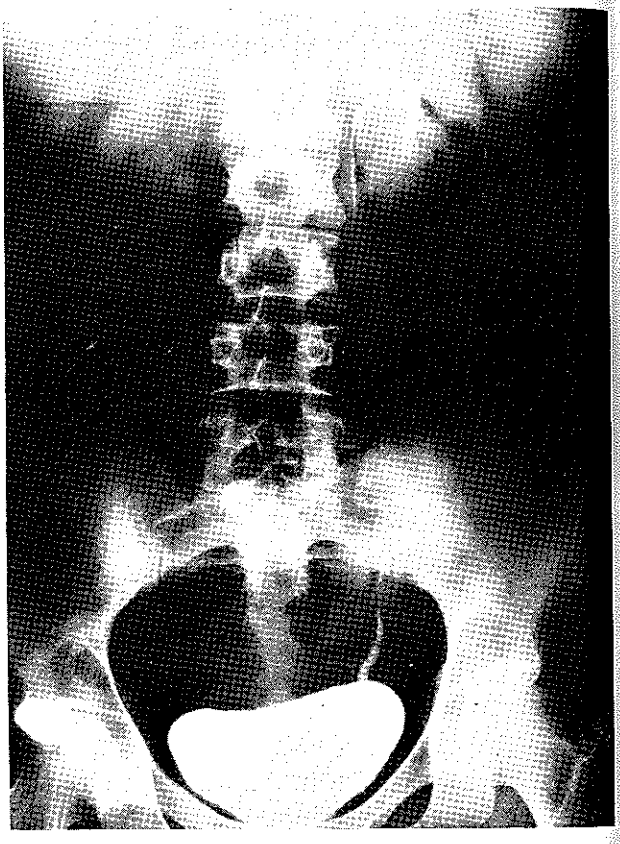

Fig 2. I.V.P. of case # 3 revealing non-functioing right kidney.

Fig 3. Selective right renal angiography of case # 4 demonstrating embolic obstruction.

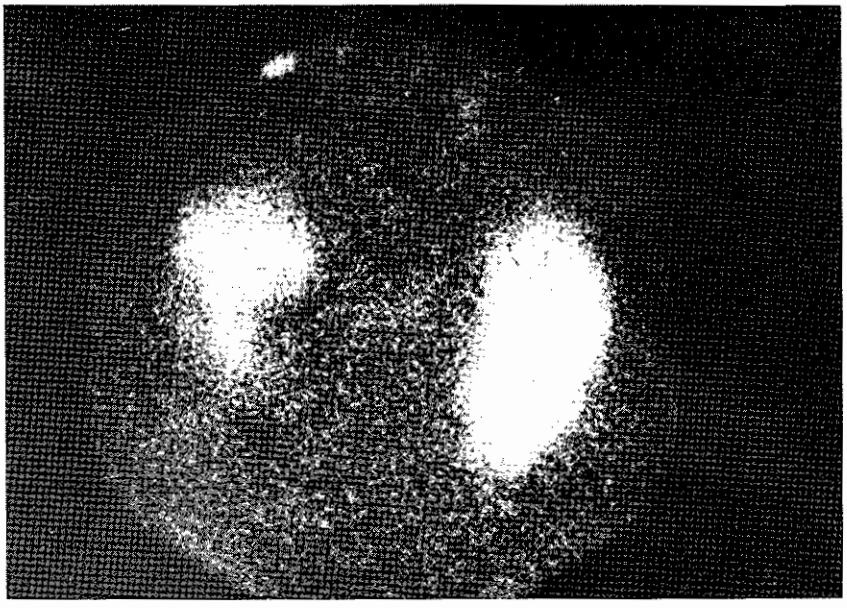

Fig 4. Static image of case # 4 one hour after injection, revealing decreased renal tracer uptake by the right kidney.

Selective renal angiography showed an embolus in one of the branches of the right renal artery (Figure 3). Heparin therapy was started. Dynamic and static renal scans showed decreased perfusion and decreased tracer uptake by the right kidney (Figure 4). Three months later he had no complaints. Repeated I.V.P. showed a 3cm decrease in the size of the right kidney with thin and irregular borders in the cortex at the site of infarction.

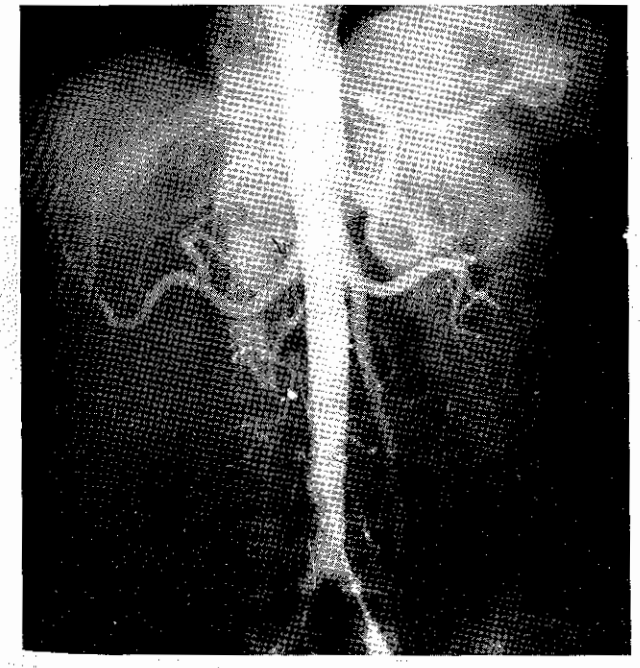

Fig 5. Angiography of case # 6 which shows complete obstruction of the main right renal artery.

CASE 5

A 57-year-old man, a known case of mitral stenosis, referred with a two day history of dyspnea. On physical examinations he had BP: 240/120 mm Hg, PR: 180/min irregular, respiratory rate: 50/min, bilateral basilar rales and diffuse abdominal tenderness. Diazoxide and furosemide were given intravenously. He gradually became normotensive. Heparin also was added in addition to digoxin. Laboratory results were: WBC: 24700/mm^3, urine was grossly bloody with 2^+ Proteinuria, serum LDH: 700 I.U., BUN: 17 mg%, creatinine 2.3 mg%.

Dynamic and static renal scans revealed loss of blood flow and decreased renal tracer uptake by the upper pole of the right kidney. Two months later I.V.P. revealed normal excretion of the dye by the smaller right kidney.

CASE 6

A 47-year-old woman presented with sudden right flank pain associated with fever and vomiting. Her physical exam revealed T: 39°C, irregular pulse, signs of mitral stenosis and right flank tenderness. Lab data showed WBC: 32000/mm^3, urinalysis: 3^+ proteinuria, many RBCs/HPF, 8-10 WBC/HPF, LDH: 1200 I.U., urine culture: negative. Echocardiography was compatible with mitral stenosis. I.V.P. showed a non-functioning right kidney. Ultrasound showed bilateral normal-size kidneys. Renal angiography revealed complete obstruction of the right renal artery (Figure

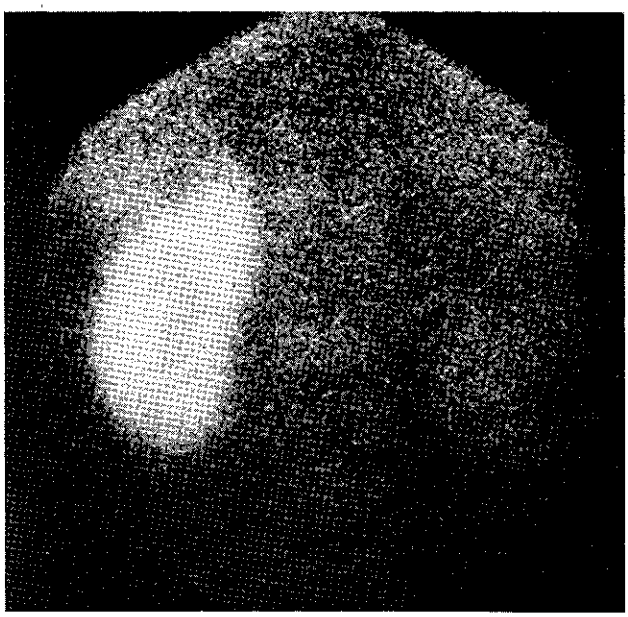

Fig 6. Posterior static image of case # 6 demonstrating complete absence of renal tracer uptake by the right kidney.

5). In dynamic renal scan there was no perfusion to the right kidney and static image failed to visualize it (Figure 6). The patient was given digoxin and heparin. Embolectomy was suggested. The patient declined and left the hospital with consent. Oral anticoagulants were continued. Two months later a second I.V.P. revealed a shrunken non-functioning right kidney with enlarged normal functioning left kidney.

DISCUSSION

In patients with rheumatic heart disease the brain is the most common site of embolization. Distal branches of the aorta and kidney are the next sites of involvement. The incidence has been reported to be 39,26, and 18 percent respectively.[1] There are two main sources for renal artery embolization:

1- Cardiac diseases are the most common cause and include mitral stenosis, ischemic heart disease, and bacterial endocarditis. Intracardiac defects associated with venous bed thrombosis (paradoxical embolism) have also been rarely reported.[2]

2- Atheromatous cholesterol crystals dislodged from different sites of the systemic circulation, particularly from the aorta. Recently, intentional infarcton of the kidney has been utilized in the treatment of both malignant and benign kidney diseases such as renal cell carcinoma and end stage renal failure with intractable hypertension.[3,4] In this procedure renal artery occlusion is induced either by an obstructing balloon or

Table I. Urinalysis, urine culture and serum LDH in six cases of renal artery embolism.

	Proteinuria	RBC/HPF	WBC/HPF	Urine Culture	Serum LDH (international units)
Case # 1	2^+	8-10	16-18	Negative	2000
Case # 2	3^+	Many	6-7	Negative	2220
Case # 3	2+	---	---	Negative	787
Case # 4	3^+	Many	---	Negative	923
Case # 5	2^+	Many	---	Negative	1700
Case # 6	3^+	Many	8-10	Negative	1200

emboli of shredded gelatin foam or blood clots. Hoxie and Coggins in 14,411 autopsies found 205 cases of renal infarction, of which 76% were found to have cardiac disease. Of these only two had been diagnosed antemortem.[5] Thurlbeck and Castelman in another analysis of autopsies in a group of patients who had been subjected to abdominal aortic operation found atheromatous emboli and renal infarction in 77% of patients,[6] Atheromatous plaques may spontaneously dislodge from an arteriosclerotic arterial tree and block the renal artery or its divisions.[7,8] Rarely, aortic angiography may cause renal artery obstruction by dislodging an atheromatous plaque.[9]

The clinical manifestations of acute renal artery embolism are well recognized. Its diagnosis can be readily established if it is borne in mind. Sudden appearance of flank pain is the most important symptom. It is often colicky in nature associated with nausea, vomiting, and sweating. It gradually diminishes in intensity and disappears in a week. In patients with rheumatic heart disease complicated by atrial fibrillation, the appearance of sudden flank pain makes renal artery embolism the first diagnostic consideration. Associated fever is probably due to tissue necrosis. Hypertension, occasionally malignant, has been reported with renal artery embolism.[10,11] It has been attributed to the release of renin from the ischemic renal parenchyma. In our patients, two cases had developed hypertension which disappeared with treatment. There is flank tenderness over the involved kidney, and a bruit may be heard.

Anuria and azotemia suggest either embolization to both kidneys or to a solitary kidney.[12] Leukocytosis and proteinuria are the constant findings; however, pyuria and microscopic hematuria are also common. All of our six cases had proteinuria and elevated LDH levels. Five had hematuria and two had pyuria. All urine cultures were negative (Table I). The most sensitive serum enzyme abnormality associated with

Table II. Paraclinical findings in six cases of renal artery embolism.

	I.V.P.	Renal Angiography	Kidney Scan	Retrograde Pyelography
Case # 1	Non functioning left kidney.	---	---	---
Case # 2	Non functioning (left) poor functioning (right)	Bilateral renal artery emboli	---	---
Case # 3	Non functioning right kidney.	Right renal artery emboli	Perfusion defect	---
Case #4	Non functioning right kidney.	Right renal artery emboli	Perfusion defect	---
Case # 5	---	---	Perfusion defect	---
Case # 6	Non functioning right kidney	Complete obstruction of right main renal artery	Perfusion defect	---

renal artery embolism is the rise of LDH level.[13] Elevation of SGOT and SGPT are other indicators.[13,14] Hemoglobinuria following renal infarction has been mentioned by Libman and Fishberg.[15] Hemoglobin is liberated in the infarcted area by autolysis. Teplick and Yarrow have stated that a non-functioning kidney of normal size in conjunction with a normal retrograde pyelogram can be diagnostic for acute renal artery embolism.[16] However, this condition may also be seen in other situations such as acute tubular necrosis and cortical necrosis.

In I.V.P. embolization of a segmental branch of the renal artery causes non-visualization or poor functioning of the involved kidney. Long-standing segmental infarction will produce local distortion with irregularity and thinning of the cortex. Occasionally, calcification may occur at the site of infarction. A thin dense rim may be seen around the involved kidney during the nephrographic phase of I.V.P. which corresponds to the viable rim of cortex produced by collateral circulation from the renal capsule and may occur in total obstruction of the main renal artery. This is called the "cortical rim sign."[17] The radiologic feature of a long-standing total renal infarction is a small kidney of regular contour usually non-visualized in intravenous pyelogram. Retrograde pyelography in this condition often reveals a normal collection system in miniature. Selective renal angiography is believed to be the best means of obtaining an accurate diagnosis with the precise definition of the anatomic picture of the renal artery. It may be hazardous to institute surgical therapeutic measures without this information. It demonstrates the exact location and size of embolus within the renal artery and the extent of renal parenchyma deprived of blood flow. The use of isotopically labeled material to visualize organ perfusion has been made with short lived radionuclide 99mTc and the rapid sequence gamma camera. By this method renal vascular perfusion can be easily observed and ischemic and infarcted areas delineated.[18] Static and dynamic renal scans are the best non-invasive methods for detection of renal infarction.

In different studies the results obtained by renal scan and angiography have had good correlations as in our study (Table II).[19] The therapeutic guidelines for the management of patients with renal artery embolism are not well defined. In an experimental study, total renal arterial occlusion for more than four hours resulted in irreversible necrosis[20] but it seems that this does not hold true for the human kidney. There are case reports in which the kidney was saved with complete obstruction of the main renal artery even after 39 days following obstruction.[21] The remarkable reversal of anuria and azotemia following renal artery embolectomy has encouraged many surgeons to prefer an aggressive operative approach. However, a careful review of the operative and nonoperative experiences with renal artery embolism in the modern surgical era has raised questions about the value of operative approach. In a review of the literature about the mortality of patients with either surgical or medical approach, Moyer showed that mortality rate of patients with embolectomy (24 patients) was 25% and those managed medically (15 patients) was 13%.[22] The difference in the therapeutic results between the operated and non-operated group is most apparent in patients with unilateral kidney involvement. The rate of kidney loss has been much higher in the operated group (56%) than that of non-operated group (23%). Higher mortality rate in the operated patients seems to

be also related to the underlying cardiovascular disease. Regarding this review and successful results of medical therapy of our six cases, it appears that medical therapy should be considered in the first line of treatment. Other supportive measures such as hemodialysis, stabilization of cardiovascular status and correction of hypertension are of extreme importance. We believe that all patients with unilateral renal artery embolism and even those with bilateral renal artery involvement who are not oliguric should receive medical treatment. Embolectomy may be considered in patients who remain anuric and show no evidence of reversal of kidney function towards normal, and also in occasional patients with complete obstruction of the main renal artery. If embolectomy is deemed necessary in an anuric patient, careful medical treatment prior to surgery is preferred to immediate intervention.

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