

Three Cases of Renal Infarction : Special Reference to Etiology, CT findings and Therapy

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Three cases of renal infarction are presented. Case 1 presented left renal infarction associated with mild mitral stenosis, moderate degree of mitral regurgitation (MR) and tricuspid regurgitation (TR). A small thrombus was detected in the posterior wall of the left atrium and left auricle. Case 2 presented right renal infarction complicated with hypertrophic cardiomyopathy (HCM) associated with severe TR and mild MR. Case 3 presented multiple infarction of the left kidney without any complication.

Renal infarction in two cases was possibly induced by small clots in the atrium or blood stream, which might had been driven from atrial fibrillation.

Introduction

It is generally accepted that renal infarction is caused by embolism or thrombus, and secondary to various surgical procedure^{1),2)}. Particularly, mitral stenosis is a common disease inducing general thromboembolism. Moreover, DCM (dilated cardiomyopathy) is enumerated as a causative factor of renal infarction³⁾. However, renal infarction with HCM (hypertrophic cardiomyopathy) appears to be very rare. We report three cases of renal infarction, discuss the pathogenecity, therapy of renal infarction, and their CT findings.

Case 1 : 53-year-old female was admitted to the hospital on October 28th, 1992 because of left flank pain and lumbago. Cardiac auscultation revealed opening snap and low pitched, small systolic murmur at apex and lower left sternal border. Electrocardiogram revealed tachycardiac atrial fibrillation. Two dimensional echocardiography revealed mild mitral stenosis (Fig. 1). A small piece of mural thrombus was attached to the posterior part of the left atrium and the left auricle (Fig. 2). Colour flow imaging showed MS jet, moderate TR and mild MR. The TR was caused by mild loss of coaptation.

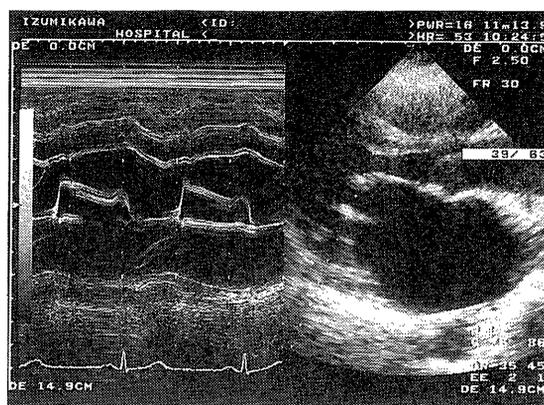


Fig. 1 : Echocardiography of case 1. Long axis view shows mild dilation of the left atrium and mild thickening of mitral leaflets(right figure). M-mode echocardiography shows mild decrease of DDR(left figure).

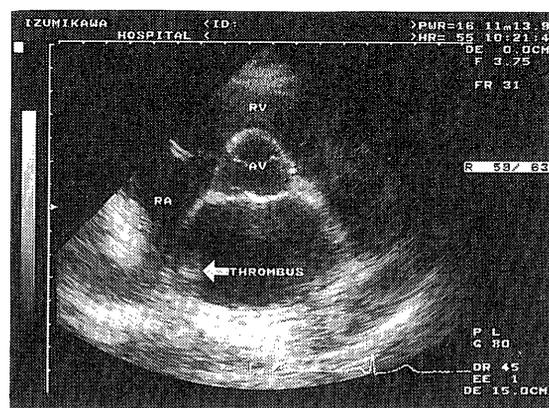


Fig. 2 : Short axis view at the aortic valve level of case 1. A small piece of thrombus is attached to the posterior wall of the left atrium(arrow).

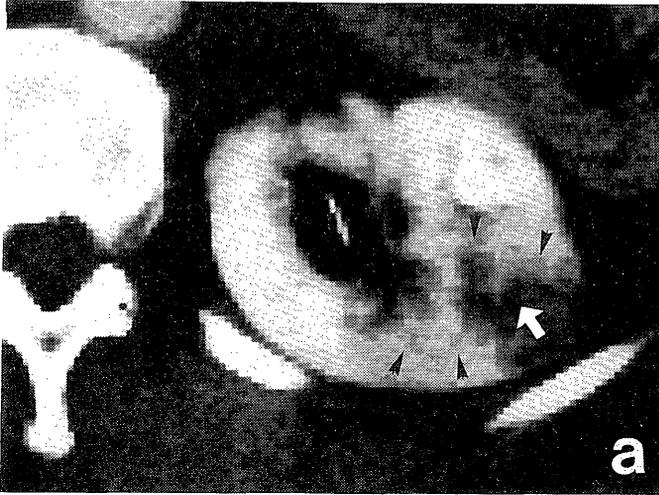


Fig. 3-a : Enhanced CT findings of case 1 reveals wedge-shaped deep-low density lesion (arrow), which is surrounded by the faintly mottled lesion (arrowhead).



Fig. 3-b : Follow up enhanced CT findings of case 1. The infarcted lesion shows recession and faintly mottled density (arrowhead). Minute deep-low density areas are dispersed in the mottled lesion (arrow).

Left renal infarction was detected by enhanced abdominal CT. A wedge-shaped, low density area was detected focally in the middle pole of the left kidney, which was surrounded by faintly mottled area inducing ischemia (Fig. 3-a). Bolus infusion of 60,000 IU urokinase was administered intravenously with intravenous infusion 2500 U of heparin for two days. Moreover, 240,000 IU of urokinase was added intravenously the next day. Thereafter, Heparin sodium of 5,000 U was instilled intravenously for 24 hours with intravenous infusion of 60,000 IU urokinase for 2 days. Then warfarin potassium was given orally without heparin and urokinase intravenously.

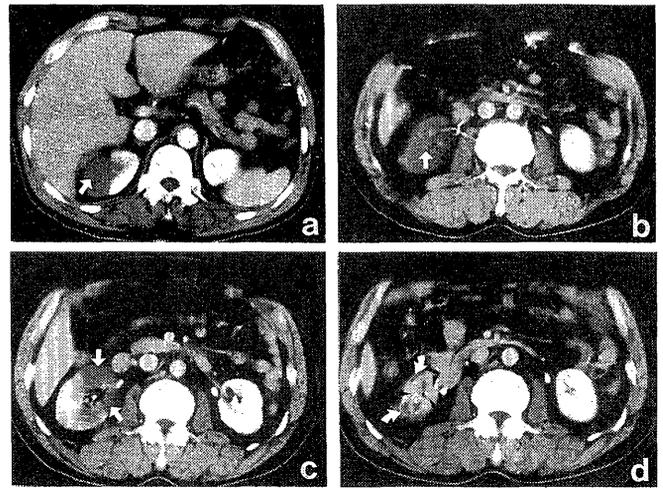


Fig. 4-a : Enhanced CT findings of case 2 reveals wedge-shaped infarction in the upper pole of the right kidney, whose density is deep-low (arrow).

Fig. 4-b : Lower pole section of case 2 reveals global infarction with swelling in the right kidney (arrow).

Fig. 4-c : Middle pole section of case 2 reveals deep-low density areas in more than 50 % of the ventral part of right kidney (arrow).

Fig. 4-d : Follow up enhanced CT study of case 2 reveals focal infarction and shrinkage of lower pole of the right kidney. Focal deep-low density areas (arrow) are scattered in the faintly mottled lesion (arrowhead).

Serum LDH level reached up to 1460 U at maximum.

Follow up CT scan revealed mottled ischemic lesion with minute deep-low density in the previous infarcted lesion of the left kidney (Fig. 3-b). Although left atrial thrombus was not dissolved by anticoagulant therapy, the patient made favorable course and EEG showed sinus rhythm one week after admission.

Case 2 : 48-year-old male was admitted to the hospital on Sep. 22, 1992 because of severe abdominal pain associated with nausea and radiating pain in the back. ECG showed atrial fibrillation and neagtive T in precordial lead V3 through V6. Abdominal CT scan disclosed a wedge-shaped infarction in the upper pole (Fig. 4-a), and global infarction with swelling in the lower pole of the right kidney (Fig.4-b). More than 50 % of the ventral part of the right kidney was also involved in the infarction (Fig. 4-c).

Bolus intravenous infusion of 60,000 IU urokinase was administered and 10,000 U of heparin sodium was dripped intravenously for 24 hours for 3 days. Then, warfarin potassium was given orally. Serum LDH value reached to 2985 U at maximum. The diameter of middle to lower interventricular septum was 25-32 mm by echocardiography. Mild loss of coaptation of tricuspid valve was

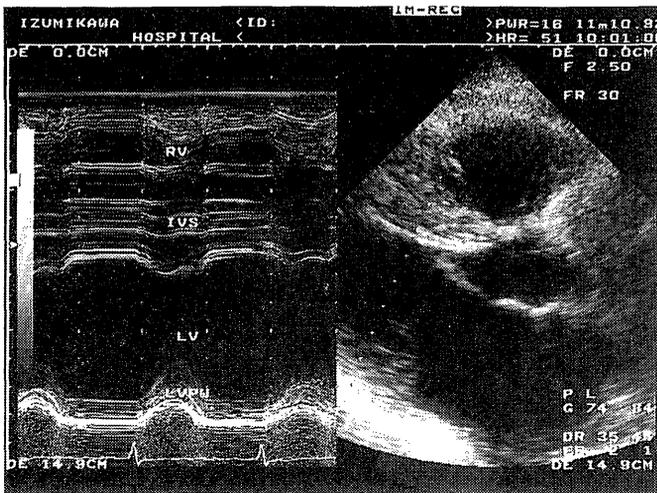


Fig. 5 : Echocardiography of case 2. Interventricular septum is markedly thick.
 RV : right ventricle, IVS : interventricular septum,
 LV : left ventricle, LVPW : posterior wall of left ventricle

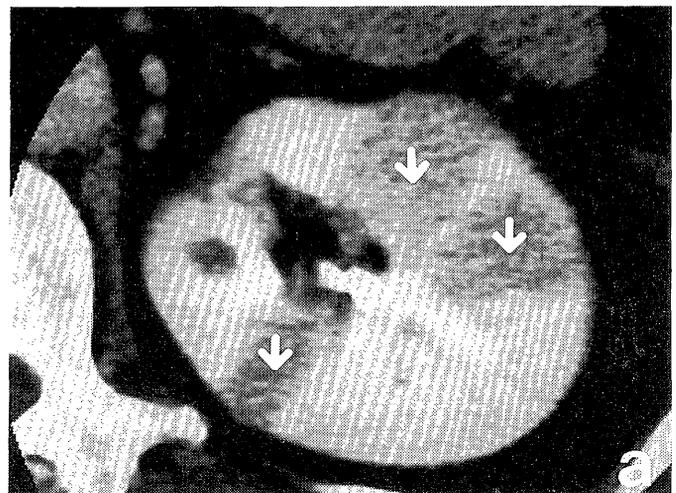


Fig. 6-a : Enhanced CT findings of case 3 reveals three wedge-shaped infarction in the left kidney, whose density is faint and mottled (arrow) compared with that of case 1 and 2.

detected. Colour flow imaging revealed mild mitral regurgitation and severe tricuspid regurgitation. Abdominal CT one month after onset revealed shrinkage of the lower pole of the right kidney and recession of infarcted lesion (Fig. 4-d). The patient also made a favorable course.

Case 3 : 42-year-old female was admitted to hospital on Nov. 15, 1992 because of the left flank pain and high fever which had been continued for five days. Abdominal CT revealed three lesions with wedge-shaped, low mottled infarction in the middle pole of the left kidney. Two were in the ventral part, while another was in the posterior part of the left kidney. Neither urokinase, heparin and warfarin was not administered for one week because hemorrhagic infarction was suspected on admission. It became apparent that contrast media used in pyelography was misinterpreted as hemorrhage. The administration of ticlopidine hydrochloride was begun from Nov. 24. Follow-up CT scan revealed recession of the infarcted lesion and cortical thinning of ventral part of the left kidney (Fig. 6-a). The patient followed an uneventful course after two weeks without consistent elevation of serum LDH, GOT and GPT.

Discussion

Renal infarction can easily be detected by the recent development of ultrasonography, doppler ultrasound⁴⁾, scintigraphy, angiography, computed tomography^{1), 5), 6), 7), 8), 9), 10), 11)} and MR1. Fever, hamaturia, leukocytosis and an elevated serum value of LDH, GOT, GPT, ALP are present

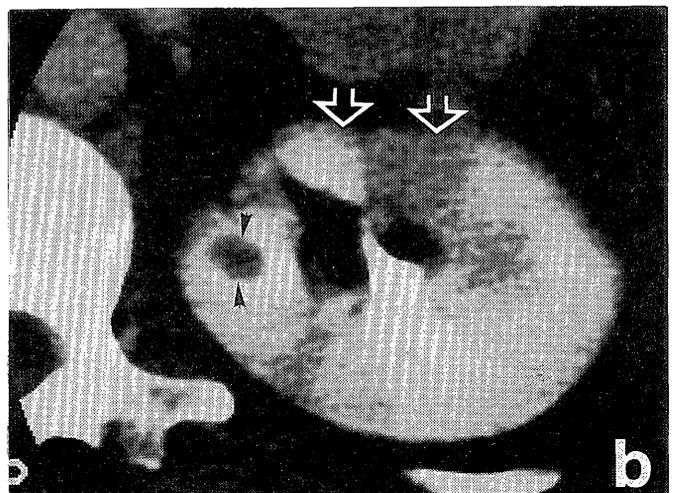


Fig. 6-b : Follow up CT findings of case 3 reveals recession of infarcted lesion and cortical thinning of the ventral part of the left kidney (open white block arrow). A solitary renal cyst is located in the posterior part of the left kidney (arrowhead).

in most case^{1), 2), 6), 10), 12), 13)}. We could not establish the correct diagnosis solely on the basis of physical examination until CT was performed.

42 % of renal infarction are due to emboli⁹⁾. An embolus or thrombus to renal artery may result from rheumatic valvular disease with atrial fibrillation, atherosclerotic debris from plaque or aortic aneurysm, left ventricular thrombus following acute myocardial infarction, bacterial^{8), 11)} or candida albicans endocarditis¹³⁾, secondary to surgical operation¹⁾. Other causative factors are sickle cell

disease⁹⁾, systemic lupus erythematosus⁹⁾, and hyperthyroidism with atrial fibrillation¹⁾.

A left atrial thrombus was supposed to contribute to the renal infarction in case 1. However, in case 2, there is some possibility that a small clot triggered by atrial fibrillation was formed in the cardiac cavity or arterial stream, finally occluded the renal artery. The exact cause of the case 3 remains unclear.

It has been reported that dilated cardiomyopathy (DCM) can be easily associated with mural thrombus in the ventricular cavity and induce the chance of embolism¹⁶⁾. A case of renal infarction following DCM has been reported¹⁶⁾. Renal infarction associated with HCM is very rare compared with DCM associated cases²⁾. Inoue et. al.¹⁶⁾ reports two cases of HCM combined with TR and MR. MR is often detected in HCM, but TR is not. In HCM, MR is attributed to two causes. One is prolapse of the posterior mitral leaflet into the left atrium because of a decrease in endosystolic volume. Another is loss of coaptation of the anterior mitral leaflet induced by SAM (systolic anterior movement). In our case, MR and TR were possibly induced by a loss of coaptation of valves and by atrial fibrillation.

Intraventricular thrombus of DCM can be formed by marked dilation of ventricular cavity, contraction failure, blood stasis, endocardial changes in the lesion of attached thrombus, fibrosis of ventricular wall, and arrhythmia¹⁷⁾. Not only blood stasis but also myocardial fibrosis and atrial fibrillation may be the source of thromboembolism in the present case.

Conservative therapy is suggested in the case of arterial renal infarction^{18), 19)}, on the contrary, nephrectomy is needed in venous renal infarction since it is associated with sepsis. Transcatheter embolectomy, vascular dilation using balloon catheter and infusion of anticoagulant agent systemically or locally are introduced currently. Contractor et. al.²⁰⁾ and Fischer et. al.²¹⁾ tried selective, intra-renal artery infusion of streptokinase successfully. Arai et. al.²²⁾ reports three cases of renal infarction, two of which was treated by 1,200 U of urokinase into renal artery. Systemic infusion of thrombolytic agent is associated with side effects including spontaneous hemorrhaging, bleeding and oozing from puncture sites, acute cerebrovascular accidents, shock etc²⁰⁾. Two cases presented here followed an uneventful course in spite of systemic intravenous infusion of urokinase. Fischer²¹⁾ concluded that selective, intra-arterial fibrinolytic therapy may obviate the need of surgical embolectomy in poor risk patients in whom operative mortality rates appear unacceptably high. Selective intra-arterial infusion of urokinase may be more effective and preferable to systemic intra-venous infusion.

The exact occluded lesion of the renal artery is not clear since arteriography was not performed in our series. The occluded lesion might not be in the main truncus of renal artery but in its branches or peripheral in three cases, which is inferred from CT findings. In our series infarction

was not detected by plain CT, but was detected easily by contrast enhancement. Cortical rim sign^{6), 9), 10), 11)} was not observed in all of our cases. In case 1, focal infarction of the left kidney indicates obstruction of the segmental artery or more peripheral branch. Only one vessel might have been involved in the renal infarction. In contrast, case 2 revealed almost global infarction of the middle and lower pole, and segmental infarction of the upper pole of the right kidney. Therefore, either the ventral or dorsal branch of the right renal artery was possibly obstructed, or some segmental arteries perfusing into upper, middle and lower pole might have been obstructed at the same time. In case 3, three segmental branches or more smaller branches were possibly obstructed. According to the experimental observation by Glazer et. al.¹⁰⁾, cortical rim sign is significant one week after renal infarction and gradually decreases. The rim sign is supposed to appear after ligation or complete obstruction of relatively large vessels, particularly the ventral or dorsal artery, and the main truncus of the renal artery¹⁰⁾. Therefore, two reasons we could not detect rim sign in our three cases can be enumerated. One is that we did not perform a CT examination one week after the onset of renal infarction, another is that the obstructed vessels were possibly to be peripheral.

The deep-low density in the infarcted lesion of case 1 and case 2 may reflect complete or partial cellular necrosis. On the other hand, faintly mottled lesion surrounding the deep-low density area in case 1 and the mottled lesion in case 3 may reflect not cellular necrosis but ischemic state. The faintly mottled lesion can be detected in the healing process of renal infarction, which possibly suggests ischemia with mild reperfusion.

Oral anticoagulant agent should be administered following surgical embolectomy, intra-arterial or intravenous infusion of urokinase to avoid secondary thrombosis.

Moreover, it is advisable to administer anticoagulant in advance in those patients who have mitral stenosis, DCM and HCM with atrial fibrillation as a prophylactic measure in order to avoid systemic thromboembolism.

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