SUCCESSFUL THROMBOLYTIC THERAPY FOR BILATERAL RENAL INFARCTION: A CASE REPORT

Han-Ching Lin, Paul Ming-Chen Shih,1 Tu-Hao Chang, Hung-Lung Ke, Wen-Jeng Wu, and Chun-Hsiung Huang
Departments of Urology and 1Radiology, Kaohsiung Medical University Hospital, Kaohsiung Medical University, Kaohsiung, Taiwan.

Acute renal infarction is a rarely reported disease in the medical literature. Angiography, renal scintigraphy, intravenous pyelography, sonography, and enhanced computed tomography may be useful in diagnosing acute renal infarction antemortem. Therapeutic guidelines for the treatment of renal infarction have not been established. We report a case of bilateral renal infarction in an elderly woman with atrial fibrillation, which was successfully treated by thrombolytic therapy.

Key Words: atrial fibrillation, renal infarction, thrombolytic therapy

Detection of acute renal infarction is often delayed or missed due to the rarity of the disease and its unspecific clinical presentation [1]. The patient with acute renal infarction usually has acute flank pain or abdominal pain that suggests other, more common diseases, such as urolithiasis, bowel disorder, or even myocardial infarction [1]. Additionally, neither risk factors nor laboratory examinations that have a clinically acceptable specificity for acute renal infarction exist.

Because of the rarity of the disease, it is questionable if the superiority of any particular therapy can be evaluated in prospective randomized clinical trials. In 1973, Moyer et al compared the results of surgical and medical management for unilateral renal involvement and found conservative therapy to be favorable [2]. Until now, there are no available data regarding the treatment of bilateral renal embolism. We report a case of bilateral renal infarction that was treated successfully by thrombolytic therapy.

CASE PRESENTATION

A 65-year-old woman visited our emergency department because of a left-side flank pain that had persisted for about 1 hour. She had a history of atrial fibrillation, without treatment for more than 6 months. On physical examination, she had obvious left flank pain and an irregular pulse rate of 90 bpm, but was systemically well. Atrial fibrillation was noted in her electrocardiogram from 1 year previously. At first she received medical treatment but stopped 6 months ago as there was no discomfort with this problem. Urine and blood laboratory results showed positive urine dipstick test for erythrocytes and protein, and elevated C-reactive protein. No bleeding tendency and international normalized ratio (INR) of 1.1 were noted. On transthoracic echocardiography, severe mitral regurgitation and tricuspid regurgitation were recorded. No thrombus was detected in this patient. Anuria was also noted since she visited our emergency department. Abdominal sonography ruled out obstructions, and the renal structures appeared to be regular. Abdominal computed tomography (CT) (Figure 1) revealed bilateral renal infarction with minimal contrast enhancement of the lower pole in the right kidney and the posterior portion

Received: March 3, 2006 Accepted: March 31, 2006
Address correspondence and reprint requests to: Dr Wen-Jeng Wu, Department of Urology, Kaohsiung Medical University Hospital, 100 Tzyou 1st Road, Kaohsiung 807, Taiwan. E-mail: wejewu@kmu.edu.tw
Successful thrombolytic therapy for bilateral renal infarction

of the left kidney. Bilateral renal infarction due to atrial fibrillation was the first impression. Emergency angiography was performed soon after abdominal CT to identify bilateral renal artery embolism and to rule out further arterial occlusion lesions. Intra-arterial thrombolytic therapy with bolus injection of urokinase (Urokinase-GCSA, Korea) 500,000 IU into the bilateral renal artery was performed after general survey. After 24 hours of persistent infusion of urokinase at a rate of 50,000 IU/hour to the main trunk of the right renal artery, the intra-arterial catheter was removed. Revascularization of the main trunk of the right renal artery (Figure 2) and the posterior–inferior branch of the left renal artery (Figure 3) was seen on follow-up angiography.

Gross hematuria occurred in the first 12 hours after thrombolytic therapy and resolved spontaneously after 36 hours. A large volume of intravenous

Figure 1. Continuous films of enhanced computed tomography at admission (A–D, cranial to caudal). (A–C) Films show no blood supply of upper pole and middle portion of the right kidney and ventral aspect of the left kidney. (D) There is little enhanced area of the right kidney.

Figure 2. (A) Angiography of the right renal artery, pre-thrombolytic therapy. (B) Follow-up angiography, post-thrombolytic therapy. The embolus (arrow) was resolved after treatment and good vascularity of the right kidney was re-established.
fluid was given to maintain adequate intravascular volume and to prevent shock due to thrombolytic therapy-induced massive bleeding. Laboratory data showed no obvious change in hematocrit in the next 3 days. Renal function improved with an increase in urine output. Normalization of serum creatinine (<1.5 mg/mL) was achieved. Twenty-four-hour clearance of creatinine was 52 mL/minute at 2 weeks after thrombolytic therapy. Antiarrhythmia therapy with amiodarone and anticoagulation therapy with warfarin to keep INR 1.5–2.0 were given after discharge.

**DISCUSSION**

Atrial fibrillation, previous embolism, and valvular and ischemic heart disease are major risk factors for acute renal infarction [3]. However, acute renal infarction has also been associated with rare conditions: trauma-related unilateral [4] or bilateral [5] renal infarction has been described following severe blunt trauma. Both hereditary and acquired clotting disorders raise the susceptibility for renal embolism, as illustrated by several case reports describing antiphospholipid syndrome [6], sickle cell disease [7], or thrombophilia [8]. Vessel anomalies such as fibromuscular dysplasia [9], or hereditary diseases such as Marfan or Ehlers–Danlos syndrome bring about changes in the vessel wall leading to dissection, and these are the causes of renal infarction [10]. Similarly, dissecting aortic aneurysms [11] or renal artery aneurysms, and a very unusual entity—primary renal dissection, have been reported to cause renal infarction [12].

Three series have been reported about renal infarction recently. Domanovits et al described the clinical characteristics of 17 patients with renal infarction over a 45-month period, 11 (65%) of whom had atrial fibrillation and were assumed to have had an embolic event [13]. Korzets et al presented 11 cases of renal infarction in 10 patients, over a 36-month period [14]. Five of these patients had atrial fibrillation. Hazanov et al reported 44 cases of renal infarction in patients with atrial fibrillation [15].

Therapeutic guidelines for the treatment of renal embolism have not been established. Prompt recognition of acute occlusion of the renal artery is important since thrombolysis, anticoagulation, or embolectomy may minimize the loss in renal function. Moyer et al in 1973 compared the results of surgical and medical management for unilateral renal embolism and found conservative therapy to be favored [2].

Domanovits et al reported 19 cases with unilateral renal embolism successfully treated with conservative treatment [13]. All of them received systemic anticoagulation therapy and two received additional local intra-arterial thrombolysis with tissue plasminogen activator, which was shown to be effective and feasible.
In our case, bilateral renal infarction was definitely diagnosed with contrast abdominal CT within 24 hours. According to the experience of Domanovits et al [13], conservative treatment with systemic anticoagulation therapy was given to our patient first. But a rapid deterioration in renal function with anuria gave us the hint of a high possibility of dialysis therapy for the rest of her life. Embolectomy was delayed due to her heart problem. Therefore, she received intra-arterial thrombolytic therapy by urokinase bolus injection into the bilateral renal artery in addition to systemic anticoagulation therapy. A novel treatment was applied as the intra-arterial catheter was retained for 24 hours for urokinase perfusion to the main trunk of the right renal artery. Although acute complication such as gross hematuria occurred in our case, the renal function improved after revascularization, and the gross hematuria spontaneously subsided 36 hours after thrombolytic therapy. No obvious change in hematocrit and additional blood transfusion were noted in our treatment. The outcome was satisfactory due to increased urine output and there was no need for dialysis therapy.

REFERENCES

利用血栓融解療法成功治療雙側腎梗塞

林漢青¹  石明誠²  張圖豪¹  柯宏龍¹  吳文正¹  黃俊雄¹
高雄醫學大學附設醫院 ¹泌尿科 ²放射科

急性腎臟梗塞為文獻罕見疾病。血管攝影，腎臟顯像攝影，靜脈腎孟攝影，及電腦斷層為目前診斷常用的工具。但是治療的準則至今仍沒有定論。本文我們提供一個利用血栓融解療法成功治療心房顫動所引起之雙側腎梗塞的個案。

關鍵詞：心房顫動，腎梗塞，血栓融解療法
（高雄醫誌 2006;22:410－4）