Renal Infarction Caused by Spontaneous Renal Artery Dissection After Playing Golf: A Case Report

전주예수병원 신장내과[1]

정승희[1], 오주환[1], 조아영[1], 선인오[1], 이광영[1], *이하은[1]

Introduction: Renal infarction is a rare and caused by cardiac diseases, a hypercoagulability and renal artery dissection. Renal artery dissection occurs in patients with connective tissue disorder following trauma. Spontaneous renal artery dissection(SRAD) is extremely rare and there are few cases occurring after exercise. Herein, we present a case of SRAD after playing golf, which was managed through medical treatment.

Case report: A 59-year old man presented to the emergency room complaining of sudden onset severe left loin pain after playing golf vigorously. The patient's vital signs were unremarkable. He was diagnosed with hypertension and diabetes, treated with medications. Laboratory tests showed an elevated C-reactive protein, while complete blood cell counts, creatinine, and urinalysis were unremarkable(Table1). The kidney CT showed about 20% volume of renal infarction in the left kidney. Furthermore, it revealed dual left renal arteries with left upper renal artery dissection. There was also a 1.5cm aneurysmal dilatation(Figure1A). The patient exhibited normal sinus rhythm. Echocardiogram revealed no thrombi or vegetation. No abnormalities were found in the laboratory tests assessing hypercoagulability(Table2). He had no clinical features of connective tissue disorder. He had no history of trauma or endovascular procedures. Finally, we concluded that SRAD was the cause of acute renal infarction. We initiated anticoagulation therapy with enoxaparin(1mg/kg every 12hours), transitioning to oral warfarin. Due to the stable blood pressure and renal function, we decided to maintain the medical treatment without intervention. After one month of anticoagulation, a follow-up kidney CT showed no progression of renal artery dissection with a reduction in the extent of the infarction(Figure1B).

Conclusion: We report a non-traumatic SRAD, which appears to be associated with golf. It is important to suspect renal infarction and SRAD in patients with acute loin pain, especially having golf activities. While there is no standardized treatment for SRAD with renal infarction, patients with stable blood pressures and renal function may be managed with medical treatment.

Fig. 1

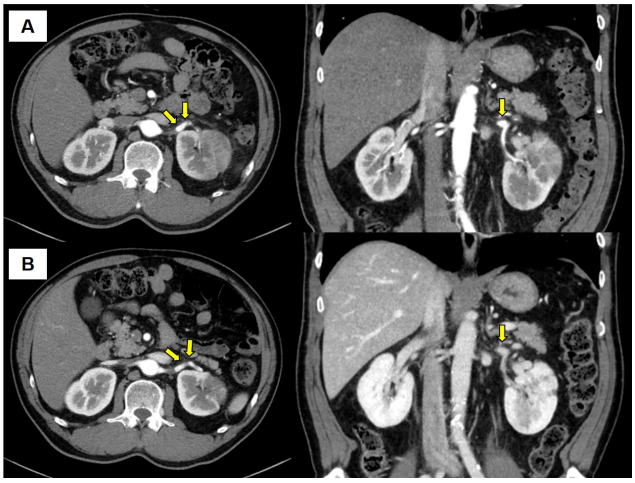

Table 1. Laboratory findings

	On admission	Outpatient (1 month)	Reference
Urinalysis			
pH	5.0	5.0	4.8-7.5
Protein	Negative	Negative	Negative
Occult blood	+	+++	Negative
Protein to creatinine ratio	0.09	0.09	<0.2
RBC (/HPF)	1-4	20-29	<5
WBC (/HPF)	1-4	<1	<5
Nitrite	Negative	Negative	Negative
Complete blood cell counts			
Hemoglobin (g/dl)	14.5	15.5	13.0-18.0
Hematocrit (%)	43.1	45.5	40.0-54.0
Platelets (10^9/L)	215	255	150-450
White blood cells (10^9/L)	4.69	4.58	4.0-10.0
Serum			
Urea nitrogen (mg/dl)	13.83	21.0	7.0-20.0
Creatinine (mg/dl)	1.2	1.0	0.6-1.2
C-reactive protien (mg/dl)	5.3	0.2	0.01-0.47
Renal function			
eGFR (ml/min/1.73m^2)	69.7	86.7	CKD-EPI

Abbreviation: CKD-EPI, chronic kidney disease epidemiology collaboration; eGFR, estimated glomerular filtration rate; HPF, high power field.

Table 2. Laboratory investigation of hypercoagulability

	On admission	Reference
Prothrombin time (sec)	8.7	10.0-13.5
INR	0.99	0.8-1.2
Activated partial thromboplastin time (sec)	33.2	25.0-35.0
D-dimer (μg/mL)	<0.02	<0.5
Protein C activity (%)	100	70-130
Protein S activity (%)	102	77-143
Anti-nuclear antibody	Weakly positive	Negative
Lupus anticoagulant antibody	Negative	Negative
Anti-cardiolipin antibody	1.8	<10.0
Anti-phospholipid antibody	1.3	<10.0
Factor V (%)	53	66-126
Factor VIII (%)	121	60-150
Antithrombin III (%)	93.4	80-120