

Usefulness of Serum D-Dimer Level in Acute Renal Infarction

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Abstract

Acute renal infarction (ARI) is a rarely encountered disease in emergency services, but is of particular importance owing to higher mortality and morbidity rates in the absence of early diagnosis and intervention. On the other hand, urolithiasis cases are admitted to emergency departments very frequently with the complaint of pain. ARI with non-specific symptoms or urolithiasis-like pain would increase the likelihood of being omitted in crowded emergency rooms. Previous studies reported supportive diagnostic role in ARI of increased serum lactate dehydrogenase and C-reactive protein levels and white blood cell count in the presence of hematuria; however, none mentioned D-dimer as a likely diagnostic or prognostic marker. We hereby present 2 case reports where a contrast-enhanced tomographic scan performed on the basis of suspicions raised by high serum D-dimer levels which established the definitive diagnosis ARI. Our aim was to emphasize that serum D-dimer may be used as a criterion for supporting or excluding the thromboembolic events, such as renal and mesenteric infarction.

Key Words: Abdominal pain, acute renal infarction, emergency department, renal artery thrombosis, renal colic, urolithiasis, d-dimer

Introduction

Acute renal infarction (ARI) is defined as renal parenchymal injury due to sudden cessation of renal blood flow. ARI is a clinical condition with an incidence of around 0.007% to 1.4% in the current literature¹⁻³. Two main reasons show up in the etiology of ARI, one of which is occlusion of renal artery by an embolus originating from the heart and aorta. Embolic occlusion accounts for 48% of all ARI cases. The other important reason is renal artery thrombosis secondary to either traumas or percutaneous interventions. Among the other relatively rarer etiologies are renal artery dissections, fibromuscular dysplasia, and hypercoagulation states. On the other hand, previous reports suggested the exact etiology was not detected in 20% to 59% of all ARI cases⁴. This article presents two ARI cases, who were admitted to our emergency service with persistent left flank pain mimicking urolithiasis and were eventually diagnosed with ARI based on clinical suspicion raised by high serum D-dimer levels.

Case Presentation

Case 1: 59-year-old male presented to our emergency department due to sudden and severe pain in the left abdomen. It was learnt that the patient had been admitted to a county-side hospital with the pain beginning about three hours

ago and his pain had not relieved despite intramuscular administration of a non-steroid anti-inflammatory (NSAI) drug. His past medical history revealed a therapy for acute rheumatic fever for 4 years at the age of 22.

During physical examination, left costovertebral angle tenderness was noted. In abdomen, severe defense was detected on the left upper quadrant. A diastolic rumble of 1/4 grade was heard during cardiac auscultation. Electrocardiography showed normal sinus rhythm. Physical examination findings were as follows: blood pressure (BP): 160/95mmHg, temperature: 37°C, pulse oximeter: 96%, respiration rate: 18/min and no BP difference in both arms. Pre-diagnosis was in favor of resistant pain due to urolithiasis. Routine biochemistry, hemogram, urinalysis and D-dimer test were ordered.

Intramuscular 75 mg diclofenac sodium and intravenous 20 mg Hyoscine N-Butylbromide in 500 cc 5% dextrose was administered. However, pain did not subside, although 45 minutes passed after the treatment. Therefore, 100 mg tramadol in 150cc saline solution was administered intravenously, which reduced the pain, but did not cease completely. Laboratory results revealed: urea: 56 mg/dL (n=16.6-48.5), creatinine: 1.25 mg/dl (n=0.7-1.2) and D-dimer: 1.10 mg/dl (N <0.55 mg/dl) (Table 1). Urinalysis was normal. Since complete relief was not achieved and D-dimer test was high, an aortic dissection, or a renal or mesenteric thromboembolism was suspected. Contrast-enhanced tomographic scan was performed, and total occlusion of the left renal artery as detected (Figure 1).



Figure 1

The patient was consulted with a cardiovascular surgeon and a urologist and was put on 0.6 mg twice daily subcutaneous enoxaparin treatment. Due to lack of interventional radiology unit in our hospital, the patient was referred to higher center where he underwent a combination therapy comprising intra-arterial fibrinolytic administration and a subsequent stent implantation in the renal artery with complete reflow. Trans-esophageal echocardiography revealed no thrombus in the heart chambers and left atrial appendix, however, grade-2 spontaneous echo contrast was observed within the left atrium together with a mild-to-moderate rheumatic mitral stenosis.

Case 2: A 58-year-old female presented to our emergency room with left flank pain. She stated that the pain started 2.5 hours ago, and she took a NSAID drug, which however was not effective. Her past medical history revealed a previous surgery due to osteoarthritis on the right knee 4 months ago and a hepatic artery thromboembolism one month after the operation. She said that she had been on rivaroxaban treatment but did not take the medication for the last three days.

Her BP was 170/95 mmHg, heart rate was 88/min, respiratory rate was 20/min and the body temperature was 37.5°C. In physical examination, a marked defense was noted on her left flank. Cardiac and pulmonary examinations were normal. Electrocardiography showed normal sinus rhythm. In the light of these findings, urolithiasis was considered as pre-diagnosis, and blood tests and urinalysis were ordered (Table 1). A 75mg dose of diclofenac sodium was administered intramuscularly. Since her complaint did not subside, D-dimer test was requested to rule out such probable thromboembolic event as renal or mesenteric infarction. Moreover, serum D-dimer level was 4.66 mg/dl, which prompted us to perform a contrast-enhanced abdom-

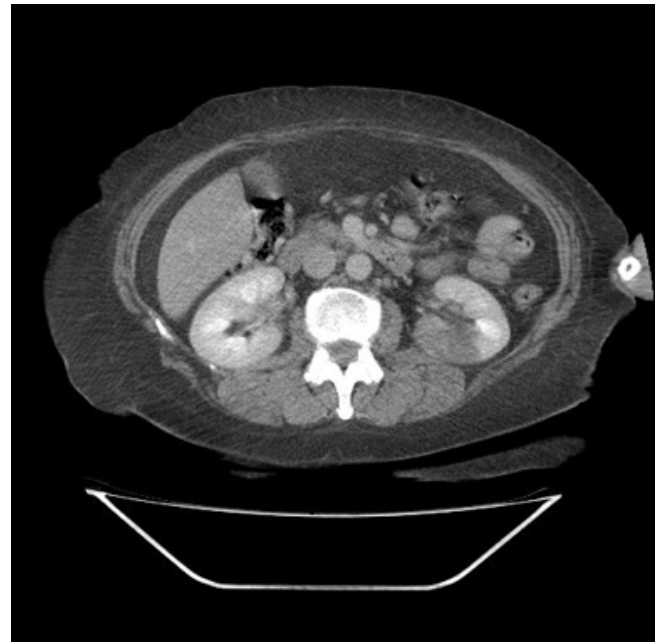


Figure 2

inal tomographic scan that showed partial occlusion of the left renal artery (Figure 2). Subsequently, enoxaparin 0.6 mg was administered subcutaneously. After consultation with a cardiovascular surgeon and a urologist, the patient was transferred to another center for further treatment.

Discussion

Diagnosis of ARI is frequently mistaken due to clinical resemblance to other more common disorders. Timely diagnosis is of paramount importance, since shortening of the time interval in which a kidney exposes to ischemia may reduce morbidity and mortality. Number of the elderly admitted to emergency departments shows a progressive escalation each year, a patient group with greater risk for thromboembolic event. This necessitates the emergence of new markers and new approaches in the differential diagnosis of scarce diseases from more common ones. We consider that serum D-dimer is likely to serve as such a new marker of differential diagnosis in patients with severe flank pain.

D-dimer is a fibrin degradation product and is especially useful in the exclusion of venous thromboembolic events if its serum level is within normal range. More recently, serum D-dimer level has been frequently utilized in the differential diagnosis or exclusion of arterial thromboembolic events. This especially holds true when it comes to dissection of aorta and mesenteric thromboembolism where marked elevation in serum D-dimer was reported^{5,6}.

Previous studies on ARI focused more on hematuria, and elevated serum levels of lactate dehydrogenase (LDH), C-reactive protein (C-RP) and white blood cell count (WBC)

Table 1. Laboratory findings of the cases.

	Case 1	Case 2	Normal Range
White blood cell count (10 ³ u/L)	13,72	5,54	(4,5-11)
Hemoglobin (g/dL)	14,2	9,8	14,0-17,5
Platelet (10 ³ u/L)	196	324	130-450
Protrombin time (second)	11,2	11,9	10-13,5
APTT (second)	18,2	21,3	23-32
INR	0,97	1,04	0,8-1,2
Glucose (mg/dL)	132	113	74-109
Ure (mg/dL)	56	20	16,6-48,5
Creatinine (mg/dL)	1,21	0,53	0,7-1,2
AST (U/L)	24	55	5-40
ALT (U/L)	14	39	5-41
GGT (U/L)	33	111	10-71
ALP (U/L)	66	181	35-135
C-reactive protein (mg/dL)	0,9	0,14	0,015-0,50
Urine eritrosite (hematuria)	1	1	0-3
D-Dimer (mg/L)	1,10	4,66	<0,55

as supportive diagnostic markers of ARI^{1, 4, 7, 8}; however, none put forth serum D-dimer as a supportive or exclusion marker for ARI. We consider that serum D-dimer would be more precise in the differential diagnosis of ARI compared with LDH, CRP, WBC, as D-dimer is more specific to thromboembolic and atherosclerotic occlusions. Likewise, hematuria would be far less specific marker of ARI, since it appeals more to the physician's consideration as a frequent sign of urolithiasis. Furthermore, there are some previous case presentations reporting normal serum LDH levels despite a sizable ARI⁹. Thereby, serum D-dimer is likely to be more sensitive in the differential diagnosis of ARI.

The limitation of our case series is the small number of cases. It should be supported by more case series. Larger case series or controlled studies are warranted to support the diagnostic role of D-dimer in ARI.

Conclusion

Serum D-dimer may prove very useful in the differential diagnosis of ARI in patient with a resistant flank pain and relatively increased risk for thromboembolism.

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