ISSN (P) : 2589-9120 / (O) : 2589-9139
PubMed-National Library of Medicine - ID: 101773527

International Journal of Medical Science and Applied Research (IJMSAR)

Available Online at: https://www.ijmsar.com

Volume – 4, Issue –2, April – 2021,, Page No. : 49 - 54

An Unexpected Acute Kidney Infarction Following Cystoscopy - A Case Report

[1]Dr. Surendra Sapkota, MD, Department of Medicine, Saint Agnes Hospital, Baltimore, MD, USA

[2]Dr. Anisha Dave, St. Matthew's University, West Bay, Cayman Islands

[3]Dr. Anas Bizanti, MD, Department of Medicine, Saint Agnes Hospital, Baltimore, MD, USA

[4]Dr. Fawaz Araim, MD, Department of Surgery, St Agnes Hospital, Baltimore, MD, USA

[5]Dr. Ahmad Al-Abdouh, MD, Department of Medicine, Saint Agnes Hospital, Baltimore, MD, USA

Citation of this Article: Dr. Surendra Sapkota, Dr. Anisha Dave, Dr. Anas Bizanti, Dr. Ahmad Al-Abdouh, "An Unexpected Acute Kidney Infarction Following Cystoscopy - a Case Report", IJMSAR – April – 2021, Vol. – 4, Issue - 4, P. No.49-54.

Corresponding Author: Dr. Ahmad Al-Abdouh, MD, Department of Medicine, Saint Agnes Hospital, Baltimore, MD, USA

Type of Publication: A Case Report

Conflicts of Interest: Nil

Abstract

Acute kidney infarction is a rare and severe condition. The prevalence of the disease is unknown as autopsy review series have found discordant rates. Acute kidney infarction can occur secondary to various etiologies including embolism, thrombosis, dissection, or iatrogenic causes. The array of clinical symptoms gives acute kidney infarction a nimbus presentation. We report a case of a 75-year-old African American male with a history of atrial fibrillation (not on anticoagulation), who presented shortly after a cystoscopy procedure complaining of left-sided abdominal pain and was found to have an acute kidney infarction.

Keywords

Kidney infarction; vascular disease; cystoscopy

Introduction

Acute kidney infarction is a rare condition associated with high rates of morbidity and mortality.[1] The incidence of acute kidney infarction has been reported to range from 0.004 to 0.007% in case series studies.[2,3]Patients usually present with symptoms that mimic other conditions such as renal colic or pyelonephritis. Hence, the autopsy review series has found higher rates.[4]

The etiologies of acute kidney infarction include embolism, dissection, thrombosis, or iatrogenic causes after vascular interventional procedures.[5] We review a case of acute kidney infarction in a patient with

persistent atrial fibrillation occurring shortly after a cystoscopy procedure.

Case Presentation

A 75-year-old African American male with a history of hypertension, atrial fibrillation (not on anticoagulation), and history of subdural hematoma following motor vehicle accident while on warfarin, requiring right frontal-parietal temporal craniotomy with evacuation of subdural hematoma four years before this presentation, who presented to the emergency room complaining of left-sided abdominal pain. The pain started a day prior subsequent undergoing cystoscopy to evaluate recurrent urinary tract infections (UTIs). He had four UTIs over the last year, and the most recent occurred two weeks earlier. The pain was 10/10, constant, sharp in nature, radiated to the left flank, and was not associated with any specific aggravating or alleviating factors. After the procedure, the urine color turned bright red but without dysuria or urine retention. He had nausea and two vomiting episodes on admission, but he denied chest pain, shortness of breath, fever, hematemesis, diarrhea, hematochezia, or urinary retention. His home medications include pravastatin, digoxin, diltiazem, clopidogrel, and doxazosin.

In the emergency department, his temperature was 98.7F, pulse rate 74 beats per minute, respiratory rate 16/minute, blood pressure 155/79 mmHg on the left arm, and pulse oximetry 97% at room air. He appeared in mild distress due to pain. Tenderness was noted in the left lower quadrant and costovertebral angle. Bowel sounds were present in all quadrants. Examination of the other systems was within normal limits.

Laboratory findings on admission revealed: hemoglobin, 13.5 g/dL; white blood cells (WBC), 9.5 × 109/L; platelets, 177 × 106/L; serum creatinine, 1.5 mg/dL (baseline 1.1 mg/dL); serum sodium level: 140 mEq/L; serum potassium level: 4.2 mEq/L; lactic acid 2.2 mmol/L; lactate dehydrogenase:296 U/L(normal range, 100–220 U/L); prothrombin time: 11.3second; activated partial thromboplastin time: 24seconds; urine leukocyte esterase +1; urine protein +1; urine red blood cells: 11-20/hpf; and urine white blood cells: 11-20/ hpf.

An electrocardiogram on admission showed atrial fibrillation with an estimated ventricular rate of 81 beats per minute. Based on his history of atrial fibrillation and his current presentation of abdominal pain and hematuria, possible diagnoses included acute mesenteric ischemia, aortic dissection, or acute kidney infarction. Computed tomography (CT) angiographyof abdomen showed acute left renal artery thrombosis and left kidney infarction **(Figure 1).**An echocardiogram showed significant dilation of the left atrium (6cm), and the left ventricular ejection fraction was 55%. Head CT was done on admission to monitor his previous subdural hematoma and didn't reveal any acute hemorrhage or infarction.

Vascular and urology services were consulted, and both recommended conservative treatment with anticoagulation. With his prior history of subdural hematoma, the risk and benefits were explained to the patient regarding anticoagulation and high risk of bleeding. He agreed to begin anticoagulation and was started on warfarin bridged with unfractionated heparin. His abdominal pain improved over the next few days and his hematuria resolved. His kidney function improved after an initial worsening, creatinine peaked at 3.3 mg/dL and then dropped to 2.6 mg/dL**(Figure 2)**. The Head CT was repeated a few days before the discharge and did not reveal any new intracranial hemorrhage.

Discussion

Acute kidney infarction can occur due to various etiologies; in a multicentral case series including 438 patients with acute kidney infarction by Oh et al., it was found that cardiogenic etiologies were responsible for 55.7% of cases, renal injuries accounted for 7.5% of cases, a hypercoagulable state contributed to 6.6% of cases and the remaining 30.1% of patients had idiopathic causes.[7]Other possible causes include trauma, instrumentation, vasculitis, sepsis, and sickle cell disease.[8]Although acute kidney infarction occurred shortly following cystoscopy procedure, we still believe that this could be a coincidence and a cardiogenic etiology better explains the etiology of acute kidney infarction in our patient based on his history of atrial fibrillation, elevated CHA_2DS_2-VASc score[9] of 3 (2 points for the age and 1 point for hypertension), and not being on anticoagulation.

Patients with acute kidney infarction could present with abdominal or flank pain, hematuria, fever, nausea, or vomiting. They may also have flank or abdominal tenderness on physical exams. Typically, patients present at an old age except in idiopathic etiologies; in a case series including 27 patients by Bolderman et al., patients with idiopathic etiologies presented at a median age of 45 while patients with other etiologies presented at a median age of 75 years.[10]The differential diagnosis of acute kidney infarction is quite board; it includes causes of acute abdomen like mesenteric ischemia, pyelonephritis, appendicitis, nephrolithiasis, ruptured abdominal aortic aneurysm, diverticulitis, incarcerated hernia, testicular or ovarian torsion, intestinal obstruction, or intestinal perforation. Given the vague presentation and broad differential, the diagnosis is regularly delayed. Hazanov et al. case series reported a delayed diagnosis (after admission) in 25 patients out of 42 (60%).[11]

Several abnormal laboratory findings suggest acute kidney infarction; urinalysis revealing hematuria, elevated serum lactate dehydrogenase, creatinine,andleukocytosis are commonly noticed.[10] Lactate dehydrogenase is abundant in the kidney, and an ischemic insult to the kidney will lead to an elevation in this enzyme. However, it is still unknown if its level correlates with the severity of the disease.[12]The diagnosis is usually made by a diagnostic image; angiography, renal isotope, or CT angiography. In Hazanov et al. case-series, angiography was positive in all cases, renal isotope scan was positive in 97%, and CT angiography was diagnostic in 80% of cases. Angiography is the gold standard modality, but it is an invasive procedure. Hence CT angiography is the most commonly used diagnostic tool, especially that it is widely available in emergency departments.[11] Many of these diagnostic tools were positive in our patients and helped eventually in making the diagnosis; these include hematuria, elevated lactate dehydrogenase, and CT angiography showing the acute kidney infarction of the left kidney.

The treatment of acute kidney infarction is controversial; conservative treatment with anticoagulation only is the most used modality. Indefinite warfarin therapy bridged initially with heparin has been recommended.[5,1] The goal international normalization ratio (INR) is 2.0–3.0 unless the patient developed thrombosis while at that goal, then a higher target of 2.5–3.5 could be reasonable. Chronic or new-onset hypertension mediated through the increase in renin secretion due to renal artery occlusion is best treated with angiotensin-converting enzyme inhibitors or an angiotensin receptor blocker.[1] Thrombolysis and

thrombectomy were evaluated in case reports and case series. Ouriel et al. revealed that revascularization in acute embolic occlusion relieved hypertension. In thrombotic occlusion, both blood pressure reduction and the restoration of renal function were successful with revascularization. However, patients with traumatic renal artery occlusion did not benefit from revascularization despite early intervention.[13] Silverberg et al. suggested that catheter device thrombolysis is safe and should be attempted for kidney salvage, even in prolonged ischemia.[14]On the other hand, Blum et al. included fourteen patients with embolic etiology treated with local low-dose thrombolysis; revascularization was successful in 13 of 14 patients but did not restore the kidney function. Based on that, it did not recommend revascularization after 90 minutes of presentation which is the estimated ischemic tolerance of the kidney.[15]The major subsequences of acute kidney infarction are loss of kidney function and persistent or uncontrolled hypertension, but high clinical suspicion leading to an early diagnosis would be most effective in preventing these complications.[11]

Figure 1: CT scan of the abdomen showing left renal artery occlusion

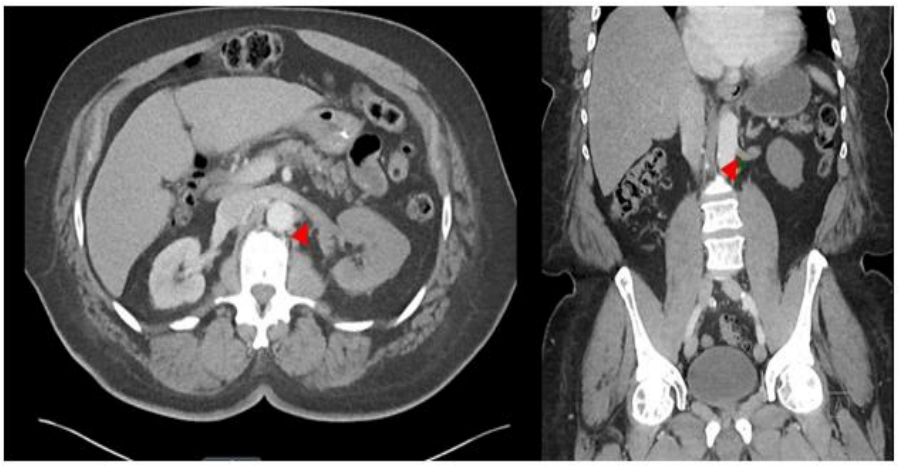
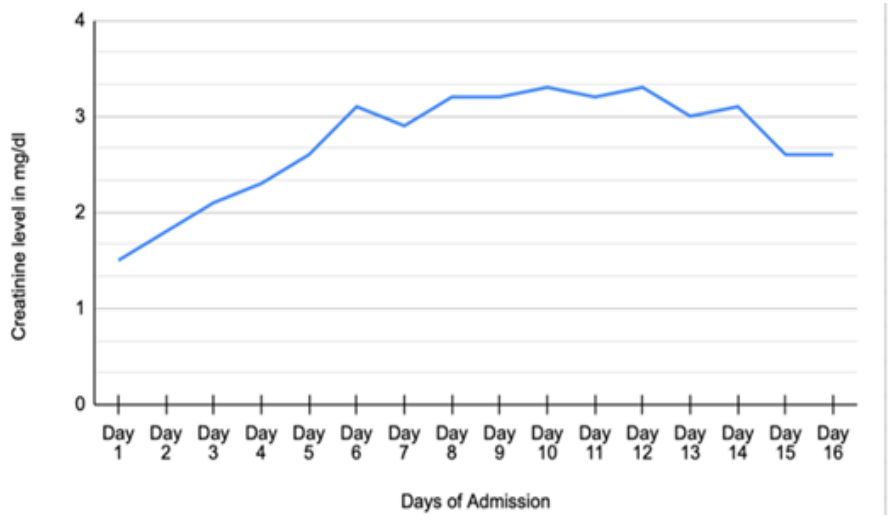


Figure 2: The change of the serum creatinine value during the hospital stay



Conclusion

In conclusion, acute kidney infarction is a rare and severe condition. A timely diagnosis is needed to start the treatment immediately, including anticoagulation and possible revascularization. There is a need for well-designed randomized controlled trials to evaluate the best treatment modality.

References

1. Paris B, Bobrie G, Rossignol P, Le Coz S, Chedid A, Plouin P-F. Blood pressure and renal outcomes in patients with kidney infarction and hypertension. *J Hypertens*. 2006; 24(8): 1649 - 1654. doi:10.1097/01.hjh.0000239302.55754.1f
2. Domanovits H, Paulis M, Nikfardjam M, et al. Acute renal infarction. Clinical characteristics of 17 patients. *Medicine (Baltimore)*. 1999;78(6):386-394. doi:10.1097/00005792-199911000-00004
3. Huang C-C, Lo H-C, Huang H-H, et al. ED presentations of acute renal infarction. *Am J Emerg Med*. 2007;25(2):164-169. doi:10.1016/j.ajem.2006.06.010
4. Paris B, Bobrie G, Rossignol P, Le Coz S, Chedid A, Plouin P-F. Blood pressure and renal outcomes in patients with kidney infarction and hypertension. *J Hypertens*. 2006;24(8): 1649-1654. doi:10.1097/01.hjh.0000239302.55754.1f
5. Domanovits H, Paulis M, Nikfardjam M, et al. Acute renal infarction. Clinical characteristics of 17 patients. *Medicine (Baltimore)*. 1999;78(6):386-394. doi:10.1097/00005792-199911000-00004
6. Huang C-C, Lo H-C, Huang H-H, et al. ED presentations of acute renal infarction. *Am J Emerg Med*. 2007;25(2): 164-169. doi:10.1016/j.ajem.2006.06.010
7. Hoxie Hj, Coggin Cb. Renal Infarction: Statistical Study Of Two Hundred And Five Cases And Detailed Report Of An Unusual Case. *Arch Intern Med*. 1940; 65 (3): 587- 594. Doi:10.1001/Archinte.1940.00190090124007
8. Korzets Z, Plotkin E, Bernheim J, Zissin R. The clinical spectrum of acute renal infarction. *Isr Med Assoc J*. 2002;4(10):781-784.
9. Stav K, Leibovici D, Goren E, et al. Adverse effects of cystoscopy and its impact on patients' quality of life and sexual performance. *Isr Med Assoc J*. 2004;6(8):474-478.
10. Oh YK, Yang CW, Kim Y-L, et al. Clinical Characteristics and Outcomes of Renal Infarction. *Am J kidney Dis Off J Natl Kidney Found*. 2016;67(2):243-250. doi:10.1053/j.ajkd.2015.09.019
11. Wong WS, Moss AA, Federle MP, Cochran ST, London SS. Renal infarction: CT diagnosis and correlation between CT findings and etiologies. *Radiology*. 1984;150(1):201-205. doi:10.1148/radiology.150.1.6689761
12. Mason PK, Lake DE, DiMarco JP, et al. Impact of the CHA2DS2-VASc score on anticoagulation recommendations for atrial fibrillation. *Am J Med*. 2012;125(6):603.e1-6. doi:10.1016/j.amjmed.2011.09.030
13. Bolderman R, Oyen R, Verrijcken A, Knockaert D, Vanderschueren S. Idiopathic renal infarction. *Am J Med*. 2006;119(4): 356.e9-12. doi:10.1016/j.amjmed.2005.06.049
14. Hazanov N, Somin M, Attali M, et al. Acute renal embolism. Forty-four cases of renal infarction in patients with atrial fibrillation. *Medicine (Baltimore)*. 2004;83(5):292-299. doi:10.1097/01.md.0000141097.08000.99
15. Sakati IA, Devine PC, Devine CJJ, Fiveash JGJ, Poutasse EF. Serum lactic dehydrogenase in acute

renal infarction and ischemia. *N Engl J Med*. 1968;278(13):721-723. doi:10.1056/ NEJM 196803282781308

16. Ouriel K, Andrus CH, Ricotta JJ, DeWeese JA, Green RM. Acute renal artery occlusion: when is revascularization justified? *J Vasc Surg*. 1987;5(2):348-355. doi:10.1067/mva.1987.avs0050348
17. Silverberg D, Menes T, Rimon U, Salomon O, Halak M. Acute renal artery occlusion: Presentation, treatment, and outcome. *J Vasc Surg*. 2016;64(4):1026-1032. doi:10.1016/j.jvs.2016.04.043
18. Blum U, Billmann P, Krause T, et al. Effect of local low-dose thrombolysis on clinical outcome in acute embolic renal artery occlusion. *Radiology*. 1993;189(2):549-554. doi:10.1148/ radiology. 189.2.8210388