Reflex Anuria: A Rare Cause of Acute Kidney Injury

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CASE REPORT

Reflex anuria: a rare cause of acute kidney injury

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Background: Acute Kidney Injury results from pre renal, post renal or intrinsic renal causes. Reflex anuria is a very rare cause of renal impairment which happens due to irritation or trauma to one kidney or ureter, or severely painful stimuli to other nearby organs.
Case Presentation: Here we present a case of acute kidney injury secondary to reflex anuria in a patient who underwent extensive gynecological surgery along with ureteral manipulation which recovered spontaneously.
Conclusion: Reflex Anuria is a rare and often not considered as cause of acute kidney injury. This case illustrates that this should be kept as a differential in potential cause of acute kidney injury in patient undergoing urogenital or gynecological surgeries.

Keywords: *acute kidney injury*; *acute renal failure*; *reflex anuria*; *post op anuria*

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Acute kidney injury causes can broadly be classified into prerenal, postrenal, or intrinsic renal categories. Reflex anuria (RA) is a very rare cause of renal impairment, which has been implicated to irritation or trauma to one kidney or ureter, or severely painful stimuli to other nearby organs. We expect this case would serve to add scant literature that might serve in better identification and definition of this entity.

Case report

An 81-year-old woman was admitted for solitary vaginal apex recurrence of cecal cancer noted on follow-up CT scan with intravenous contrast and PET scan performed one-month prior to the admission. Her medical history was otherwise notable for hypothyroidism and early-stage right breast cancer status post-lumpectomy. Cecal cancer was diagnosed 9 years ago for which she initially had right hemicolectomy with diverting ileostomy and reversal of the ileostomy. This was followed by total abdominal hysterectomy and bilateral salpingo-oopherectomy for secondary debulking 2 years ago for pelvic recurrence. She was noted to have vaginal recurrence and, subsequently, underwent radiation therapy 7 months prior to the current presentation. During this admission, she underwent low anterior resection, total vaginectomy, cytoscopy, bilateral uterolysis, and tumor debulking, and had bilateral ureteral catheter along with Foley catheter placement. In the operating room, she was adequately hydrated with normal saline and blood loss was minimal. On the day of surgery, basic metabolic panel showed blood–urea–nitrogen (BUN), 11 mg/dl, and creatinine, 0.76 mg/dl. Complete blood counts and electrolytes were within normal limit with hemoglobin, 13 g/dl; WBC, 7,600; sodium, 142 mEq/l; and potassium, 3.8 mEq/l. On post-op day 1, the patient became oliguric with urine output of 11 ml/h and creatinine increased to 1.7 mg/dl with BUN of 17 mg/dl. Review of operative report, anesthesia sheets, and surgical floor vital sign documentation did not show any episode of hemodynamic instability, and MAP was constantly over 70 mm Hg. Physical examination was unremarkable; patient was afebrile, had stable hemodynamic parameters, and had no evidence of volume depletion, including normal skin turgor, non-edematous lower extremities, and normal heart and lung exam findings. Bladder irrigation was performed, which was negative for evidence of gross hematuria and blood clots, and failed to improve urine output. Two doses of cefazolin were administered in the perioperative period as prophylaxis for the extensive surgery otherwise patient was not exposed to any nephrotoxic agents. Following day, creatinine increased to 3.8 mg/dl and BUN was 24 mg/dl. Urine analysis tested positive for microscopic hematuria, WBCs, and leukocyte esterase, but urine sediment was negative for dysmorphic RBCs or casts. There was mild hydronephrosis of right kidney with normal cortical echotexture of both kidneys and decompressed bladder on renal and bladder

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ultrasound. Urine electrolytes done on this day showed sodium of 61 meq/l and creatinine 29 mg/dl and fractional excretion of sodium of >4%. On post-op day 3, urine output started to improve without any intervention and on day 4 creatinine started to trend down. Kidney function continued to improve for the remaining course of the hospitalization. The patient was discharged seventh day post-op with normal urine output and creatinine of 0.65 mg/dl.

Discussion

Acute kidney injury results from prerenal, intrinsic, and postrenal etiology. In our patient, normal blood pressure as well as the results of urine studies was not indicative of prerenal cause. More so, the patient received adequate hydration during and after the surgery. Acute tubular injury was also unlikely in the absence of hypotension (absolute or relative) and sepsis. Although urine analysis was positive for blood and WBCs, sediment was unremarkable ruling out any glomerular pathology. Except for the two doses of cefazolin no other nephrotoxic agents were administered during this time period to explain the kidney injury. Furthermore, acute interstitial nephritis secondary to antibiotics was thought to be unlikely as the classical case would be that of non-oliguric AKI with slow stepwise increase in creatinine, which occurs after one to two weeks after exposure unless there is prior sensitization and improves slowly after removal of offending agent. Renal ultrasound showed normal cortical echotexture and mild, unilateral hydronephrosis, which was not significant to explain oligo-anuric acute kidney injury. Also, urinary bladder was noted to be decompressed without evidence of blood clots or mass leading to obstruction.

A review of literature as to other possible causes of oliguria or anuria after gynecologic or urologic procedures revealed a few case reports of anuria after ureteral catheterization; it is termed reflex anuria. This entity is not very well understood and abnormalities in autonomic nervous system and urogenital malformations are thought to increase the risk of RA (1). Two mechanisms based on the concept of intra-renal arteriolar spasm and ureteral spasm have been proposed to explain this phenomenon (1). One is the neurovascular, which can be secondary to renorenal or ureterorenal reflex. In renorenal reflex, renal parenchymal injury leads to reflex renal arteriolar vasoconstriction resulting in acute renal failure (2). Kervancioglu et al. described a patient who developed RA after renal tumor embolization. They suggested that the stimuli for the reflex could be renal ischemia, edema, or blood vessel occlusion as a result of the embolization (3). While in ureterorenal reflex, ureteral irritation or manipulation stimulates sensory fibers from the ureter which then causes reflex renal arteriolar vasoconstriction (4). The other postulated mechanism is of mechanical obstruction secondary to ureteral edema from ureteral manipulation. Sirota and Narins in 1957 made similar observation after ureteral catheterization and suggested that the oliguria could be explained by edema of the ureteral orifices, causing urinary obstruction (5). RA has since then been reported following nephrectomy (6), hepatectomy (7), hysterectomy (8), and also after acute cardiac event in a patient with history of urolithiasis (9).

In our patient, all the possible etiologies were sequentially ruled out and left us with this rare entity of 'reflex anuria', leading to acute kidney injury.

Conclusion

To sum up, we present a case of RA which, though rare with scant literature, should be considered in the differential diagnosis of acute kidney injury. This differential should be given serious consideration in patients undergoing urogenital or gynecological surgeries.

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