

Acute Renal Failure due to Bladder Injury after Cesarean Section: Case Report and Review of the Literature

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Abstract

A 31-year-old woman who had an uncomplicated cesarean section five days prior to admission presented with abdominal distention. Evaluation revealed acute renal failure and abdominal ascites. Diagnostic paracentesis showed urinary ascites, which confirmed a bladder injury. The patient was conservatively treated by placement of a Foley catheter that resulted in complete resolution of the renal failure.

Keywords: Acute renal failure, bladder injury, cesarean section

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INTRODUCTION

We report a patient with bladder perforation who presented as acute renal failure and ascites five days after an uneventful cesarean section (C/S). Pelvic adhesions caused by repeated C/Ss are considered the main cause of bladder injury. The reported incidence of bladder injury after C/S ranges from 0.14% to 0.94% (1). Intra-peritoneal urinary bladder perforation should be included in the differential diagnosis of acute oliguric renal failure soon after gynecological surgery. Biochemical features of uremia occur because of intraperitoneal extravasation of urine, which is reabsorbed through the peritoneum. In such clinical settings, early recognition and therapy are warranted. Nephrologists who are often the first to encounter these patients with presumably acute renal failure should be aware of this condition.

CASE PRESENTATION

A 31-year-old woman had a healthy baby at 39th gestational week by C/S five days prior to admission. During routine follow-up, her obstetrician discovered abdominal distension and ascites. Laboratory work revealed a high creatinine level (creatinine 3.21 mg/dL) and ++

proteinuria in urinalysis, and she was referred to the nephrology outpatient clinic. She had no history of kidney disease. Her medical history revealed three previous C/S. The last C/S had taken a long time because of pelvic adhesions caused by prior C/S operations. She did not use any medication. At presentation, she was normotensive, and abdominal examination revealed abdominal distension with shifting dullness and diffuse tenderness; no abdominal rebound tenderness was detected.

Laboratory data demonstrated elevated blood urea nitrogen (BUN) (35 mg/dL) and serum creatinine (2.66 mg/dL) levels; whereas the serum electrolytes, acid-base, and albumin levels were within the normal ranges. The 24-hour urine protein excretion was 308 mg/day. Ultrasonography showed a normal kidney size bilaterally, without evidence of hydronephrosis, and diffuse abdominal ascites. An abdominal computerized tomography (CT) scan revealed large abdominal effusion and an enlarged uterus with an uneven density that can normally be seen in the postpartum period. Diagnostic aspiration of the ascitic fluid was consistent with transudate; and spontaneous peritonitis was excluded based



Table 1. Cases of acute kidney injury due to bladder injury after cesarean section

	Age (years)	Previous C/S history	Time to Presentation after C/S (days)	Treatment
Case 1 (7)	30	No	3	Surgery
Case 2 (8)	38	No	1	Surgery
Case 3 (9)	24	Unreported	14	Surgery
Case 4 (10)	26	No	8	Foley catheter
Index case	31	Yes	5	Foley catheter

on the findings of a normal fluid cell count and negative culture results. Peritoneal fluid obtained with paracentesis revealed BUN 106 mg/dL and creatinine 17 mg/dL, which confirmed urinary ascites. Gradually, the patient's urine output decreased to 390 mg/day, and kidney function tests worsened (serum BUN 74 mg/dL and creatinine 5.32 mg/dL). A Foley catheter was inserted, and she was started on prophylactic ceftriaxone therapy. After catheter insertion, the patient had 9600 cc urine output in the following four hours. However, a subsequent CT cystourethrogram failed to show extravasation of the urine from the bladder. Two days after catheter insertion, the creatinine level rapidly decreased from 5.32 to 0.76 mg/dL. The abdominal distension began to resolve and abdominal fluid decreased in follow-up ultrasonography examinations. And after six days, the patient was discharged from hospital with a Foley catheter. Three weeks later, the catheter was removed, and she had normal renal function on consecutive follow-ups. Written informed consent was obtained from the patient.

DISCUSSION

Here, we report a patient who had developed acute renal failure due to bladder perforation after a C/S operation. She was successfully treated with the insertion of a Foley catheter and did not require additional surgery. Bladder rupture is a rare condition. In a majority of cases, extraperitoneal injuries can be managed with catheter drainage alone, while intraperitoneal injuries always require open repair (2).

The close anatomical association between the urinary bladder and uterus is well known. Injury to the bladder complicating a gynecologic procedure has long been recognized. Some of the most common causes of injuries are prolonged labor, C/S, and abdominal hysterectomy. However, blunt trauma, irradiation to the pelvis, surgical procedures, and even spontaneous cases have been described (3). Bladder injuries may cause urine to leak into the abdomen, which leads to infection such as peritonitis. Absorption of the urine by the abdominal membrane often causes a rise in the serum BUN and creatinine causing acute renal failure. Bladder rest by Foley catheter is typically employed for 7-14 days (4).

Unfortunately, 9.2% of bladder injuries are not recognized intra-operatively; and these are the cases presenting with abdominal discomfort, acute renal failure, and oliguria as a cardinal sign (4). Our patient also presented with abdominal

discomfort, swelling, and acute renal failure followed by oliguria. After evaluation and exclusion for the likely causes of acute renal failure, we suspected intra-abdominal urinary leakage. An ascites-to-serum creatinine ratio >1 is typical for urinary ascites; and this was confirmed in our patient by showing that the ascitic fluid creatinine concentration was almost six times as high as the serum creatinine level. Conventional cystography or CT cystourethrography can be used to confirm the site of injury in the bladder and has a sensitivity of 78% (5). However, CT cystourethrography can miss small leaks, and it was normal in our patient who also had the Foley catheter in place at the time of imaging. Surgical repair is the recommended treatment modality for intraperitoneal leaks; however, conservative management can be an option in selected cases. Since our patient recovered after Foley catheter placement and stayed stable even after the removal of the catheter, we did not feel the need to schedule surgery. Successful management with the placement of a Foley catheter without surgery has also been reported in other several case reports (5). The self-improvement in these cases is probably due to spontaneous closure of small perforations. The serum creatinine level has been reported to usually return to normal within 48 hours (6).

After careful review of the literature, we found only four cases of acute renal failure due to bladder injury after C/S (7-10). All of the patients presented with abdominal distention and acute kidney injury accompanied by ascites after a few days of C/S. In all cases, diagnostic paracentesis showed urine leakage that was further confirmed with cystography or CT cystourethrogram. Foley catheters were placed in all patients and were removed between days 10 and 21. None of the patients required renal replacement therapy (Table 1).

CONCLUSION

In summary, we herein report a case of intraperitoneal urinary leakage after C/S operation causing acute kidney injury. Nephrologists should be aware of this rare condition when faced with similar patients presenting with acute kidney injury, ascites, and oliguria.

Informed Consent: Written informed consent was obtained from the patient.

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