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Research Letter

Urinary ascites with elevated blood creatinine following cesarean section indicating bladder injury

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Bladder injury at the time of cesarean section (CS) is an infrequent occurrence [1–4]. During the past five decades, there has been a steady increase in the incidence of CS worldwide. As a result, the complications associated with CS have gained enormous importance. Unrecognized bladder injury during CS can lead to vesico-vaginal fistula, vesical calculi, menuria without urinary incontinence secondary to a vesico-uterine fistula, and urinary ascites or uroascites, as in our case [1–4].

A 37-year-old woman underwent a repeat CS at a local hospital. Her past history included a subumbilical longitudinal incision of the abdomen as a result of multiple uterine leiomyomas with menorrhagia and dysmenorrhea 12 years before. She had also undergone a CS four years earlier owing to previous laparotomic myomectomy. This time, the morning after the CS, the patient's urinary catheter was removed, and she subsequently reported some abdominal pain. The pain gradually increased, and she complained of difficulty in voiding. The abdomen was mildly distended with audible bowel sounds. Urine (150 mL) was drained out by a single urethral catheterization. However, she continued to complain about difficulty in voiding. Intermittent catheterization was performed twice and 100 mL and 75 mL of urine were drained out, respectively. However, there was no fever or any other suggestive clinical data. On the third day, the patient complained of dyspnea, and intermittent, severe, colicky abdominal pain. On examination, there was progressive abdominal distension. Abdominal ultrasound scan showed a large amount of ascites in the pelvis. She was then transferred to our emergency room for further evaluation and management. At the emergency room, intravenous fluids were reinstated, and a urinary catheter was inserted, with drainage of 2000 mL of urine. Soon, the patient felt more comfortable and her abdomen became less distended. Blood analysis at our emergency room showed mild, normocytic anemia (11.1 gm/dL), and leukocytosis (16,200 count/μL) with neutrophils at 89%. The urinary sediment demonstrated frank hematuria and leukocytes. The biochemical analysis of the patient's blood initially revealed creatinine 7.94 mg/dL, sodium 140 mEq/L, potassium 5.5 mEq/L, and estimated glomerular filtration rate (GFR) 6 mL/minute/1.73 m^2. Emergency abdominal-pelvic computed tomography (CT) without contrast-enhancement was performed and revealed right abdominal wall swelling caused by hematoma or cellulites. No hydronephrosis was noted. Antibiotics (cefazoline and gentamycin) were administered at the emergency room. The patient was admitted to our ward the next day. The biochemical analysis of her blood revealed creatinine 0.75 mg/dL, sodium 137 mEq/L, potassium 5.6 mEq/L, and estimated GFR >60 mL/minute/1.73 m^2. The dramatic change of creatinine and estimated GFR was found after urinary catheter insertion. She was then sent to operation room for further management. First, we performed cystoscopy and a rent of about 1 × 1 cm at the dome of the bladder was noted (Fig. 1). We repaired the bladder injury via laparotomy with 3-0 chromic one layer and 4-0 vicryl one layer. After we repaired the bladder, we rechecked by methylene blue injection. Blood analysis the day after the operation showed mild normocytic anemia (10.9 gm/dL) and no leukocytosis (10,800 count/μL), with neutrophils at 82%. The biochemical analysis of her blood on day following surgery revealed blood urea nitrogen (BUN) 7.9 mg/dL, creatinine 0.78 mg/dL, and estimated GFR >60 mL/minute/1.73 m^2. On the next day, her bowels opened and she started to sip water. She recovered well after the operation and was discharged four days after surgery with a urinary catheter inserted. She returned to our outpatient clinic one week later and we removed the urinary catheter. Cystoscopic evaluation two weeks after

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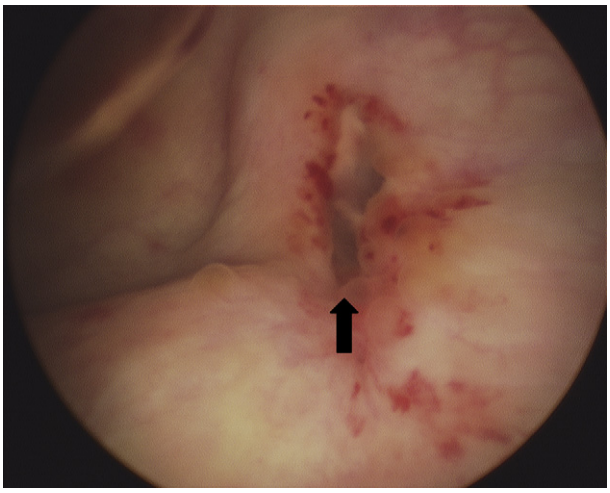

Fig 1. Cystoscopic view of bladder injury at the left bladder dome.

surgery showed intact bladder mucosa; the follow-up on the patient was uneventful.

The reported incidence of bladder injury during CS ranges from 0.14–0.94% [1,2]. Prior CS is a significant risk factor for bladder injury occurring at the time of repeat CS, amounting to a nearly four-fold increased risk over primary CS [3]. Formation of a bladder flap causes 43% of injuries, 33% occur during entry into the peritoneal cavity, and 24% occur during the uterine incision [3]. Adhesions from previous CS and other pelvic surgery also represent a significant risk factor for bladder injury during multiple CS [4]. For this case, the complexity of previous pelvic surgical history was suggestive of higher incidence of bladder injury following the CS.

Diagnosis of accidental intraperitoneal bladder injury may be delayed [5], as in our case in which the diagnosis was made on the fourth postoperative day. Cases that go unnoticed during surgery can later present serious complications that require laparotomy, or other additional major surgery. The morbidity of unrecognized bladder injury is high, and symptoms of urinary peritonitis are often nonspecific [6,7]. Patients may present with abdominal pain, low back pain, leukocytosis, and signs of peritoneal irritation or peritonitis 1–4 days after bladder injury [8]. In our case, as in others, low abdominal pain may present as early as 6–8 hours after surgery, showing improvement with the placement of a urinary catheter. She presented diffuse abdominal pain and distension first, and low urine-output symptoms that, in the immediate postoperative period, suggest either an ileus or an intraperitoneal hemorrhage. With the absence of bowel sounds and normal hematologic indices, the patient was suspected of having an ileus and managed accordingly. If bladder injury occurs, most urine drains into the peritoneal cavity, so it was reasonable for her to complain of difficulty in voiding and intermittent catheterization only draining a small amount of urine.

However, when a urinary catheter was reinserted at our emergency room, two liters of urine were drained. As Hassan et al. [9] reported previously, we believe this probably happened through a gravitational effect: a column of urine is formed in the conventional urinary drainage system which persists after the urine is drained from the patient's bladder. The weight of such a column results in the development of negative pressure in the bladder, not only emptying it but also tending to draw the bladder wall against the end of the catheter and, in this case, because of the perforation, sucking out the urine which had escaped into the peritoneal cavity. So, after the surgery, when the patient complained of difficulty in voiding with stable vital signs, intermittent catheterization resulted in only a small amount of urine. Besides, much urine was drained after a urinary catheter inserted. We must keep the bladder injury in mind.

On the other hand, profound disturbances in serum electrolytes and acid-base status (elevated serum urea, creatinine, and potassium, decreased serum sodium and CO_2 content, and development of metabolic acidosis) are consistent findings among patients with intraperitoneal bladder rupture [10]. When urine enters the peritoneal cavity, reverse autodialysis occurs. Urea and creatinine diffuse down their concentration gradients into the blood, producing a characteristic biochemical profile of pseudorenal failure. In our case, the postoperative rise in plasma creatinine dropped dramatically after an indwelling catheter was reinserted and returned to normal range. This means that the postoperative rise in plasma creatinine was probably caused by resorption rather than renal dysfunction. Additionally, the emergency abdominal-pelvic CT without contrast-enhancement showed no hydronephrosis. The diagnosis of bladder perforation can be confirmed radiologically by extravasation of contrast from the bladder [11]. Therefore, if urinary ascites as a result of bladder injury causing raised plasma creatinine mimicking acute renal failure is highly suspected, we may perform abdominal-pelvic CT with contrast-enhancement to identify the site of the bladder injury, because the postoperative rise in plasma creatinine in this situation is not caused by acute renal failure, which is a contraindication for abdominal-pelvic CT with contrast-enhancement.

The key to the diagnosis of bladder injury is awareness of the clinical entity. This diagnosis should be considered in clinically-relevant situations, such as those subsequent to CS. Urinary ascitis should be a differential diagnosis in women having abdominal distension and ileus following a CS.

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