Urinary Ascites - A Consequence of Intraoperative Injury

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Abstract
Patients with complications after surgery often pose enormous challenges to physicians. We report the case of a young lady developing fever, oliguria and intractable ascites referred to us after undergoing abdominal surgery. She was diagnosed subsequently to have a urinoma with urinary ascites masquerading as a case of sepsis. Percutaneous nephrostomy followed by definitive repair and reimplantation of ureter later led to subsidence of the symptoms.

Introduction
Postoperative complications are often mimickers of systemic illnesses and are referred to physicians. Diagnosis is often delayed and tortuous and clinical suspicion is the key. ‘Urinoma’ is one such intraabdominal space occupying lesion resulting from intraoperative injury with an incidence of 0.5- 1%.¹

Case Report
A 27 yr old lady presented with high grade fever, diarrhea and oliguria along with progressively increasing abdominal distension for last 5 days. She had undergone total abdominal hysterectomy with bilateral salpingo-oophorectomy 8 days back for benign ovarian tumour and multiple leiomyomata in some rural centre.

On admission she had pallor, tachycardia and temperature of 102°F. There was diffuse tenderness on palpation of abdomen with moderate ascites without organomegaly. Other systems were within normal limits.

Investigations revealed-
- Hemoglobin- 8.8 g/dl, total leucocyte count- 14,500/cumm, ESR- 80 mm and platelet- 1.5 lakhs/cumm. Blood biochemistry revealed- urea- 186 mg/dl, creatinine- 13.3 mg/dl, sodium- 140 meq/L, potassium-4.8 meq/L and random sugar- 84 mg/dl. Liver function tests including prothrombin time were normal. Urine routine and microscopic examination revealed- Pus cells 15-18/hpf, RBC- 5 to 6/hpf with presence of albumin and growth of Enterococcus faecalis on culture. Chest X ray was normal. Ultrasonography of whole abdomen revealed- absent left kidney, right kidney size 13.2 cm and ascites. HIV 1 and 2, HBsAg, anti-HCV antibody and antinuclear antibodies were negative.

Antibiotics were administered but urine output failed to improve even with judicious use of diuretics. Nephrology referral was done and patient received haemodialysis. Rapidly accumulating ascites warranted repeated therapeutic paracentesis. Ascitic fluid study revealed- Total count- 3040/cu mm, neutrophils- 95%, lymphocytes- 5%, Glucose- 88 mg/dl, Protein- 4 g/dl, Albumin- 2.8 g/dl Adenosine deaminase (ADA- 2.3 U/L), RBC- present, low serum ascitic albumin gradient (SAAG). Ascitic fluid c/s was positive for Enterococcus faecalis. Ascitic fluid for M cells were negative in all three samples.

After receiving focused antibiotics and four haemodialysis sessions, fever subsided but ascites and oliguria persisted. One curious finding after every episode of paracentesis was, a transient improvement in urine output followed by rapid reaccumulation of ascites.

Urological opinion was sought considering the postoperative onset of the problem. They advised a computed tomography (CT) scan of abdomen which revealed a cystic space occupying lesion (SOL) (14.5 x 7.2 cm) in the pelvis along with ascites and absent left kidney (Figures 1 and 2). Then the ascitic fluid biochemistry was re-evaluated which revealed- Creatinine- 15.1 mg/dl (serum creatinine 5.5 mg/dl) i.e- ascitic fluid creatinine/serum creatinine more than 1. The fluid from the cystic SOL was also aspirated under CT guidance which revealed a creatinine value of 22 mg/dl. On the grounds of suspicion intravesical injection of methylene blue dye through urethral catheter was done and subsequent paracentesis showed a change in colour of ascitic fluid. The course of clinical events and investigations led us to the diagnosis of intraoperative ureteric injury with a resultant urinoma and urinary ascites.

A percutaneous nephrostomy drain was introduced along with urethral catheterization (Figure 3). Ascites...
diminished, urine output improved and creatinine level gradually normalised. A definitive repair was done later after 3 weeks and ureter was reimplanted in the urinary bladder. Postoperative period was uneventful and the patient was discharged in a stable condition.

Discussion

Urinary ascites after undetected intraoperative bladder injury is uncommon.2,3 Anecdotal reports, like the occurrence of a ‘pseudo-renal failure’ after laparoscopic surgery due to urinary peritonitis are available.4

The incidence after caesarean section varies from 0.14-0.94%5 while that after abdominal hysterectomy is around 0.4 of 1000.6 Urinary peritonitis may give rise to an urinoma. Urinoma is caused by extravasation of urine. Three essential factors are required for formation of urinoma - a functional renal unit, breach in the pelvicalyceal system, and ureteral obstruction. Urine causes lipolysis and inflammation with formation of a fibrous capsule around the collected urine.7 Sometimes if ascites is massive, it may also give rise to pleural effusion by shift of fluid across the diaphragm.8

The main problem with this condition is delayed diagnosis due to its presentation mimicking acute kidney injury or sepsis.9 Treatment is surgical and with prompt delineation, complications like parapelvic granuloma, periureteral fibrosis, abscess formation and sepsis can be avoided.7

The importance of awareness regarding this post-operative complication cannot be undermined, as such cases masquerade as systemic illnesses and are referred to physicians. The key to the avoidance of this catastrophe lies in the prevention of bladder injury or ureteric injury. However if such an accident occurs, signs indicating bladder injury intraoperatively are bloody urine and the presence of gaseous distention of the urine bag indicative of a communication between the bladder and the intraperitoneal space (the catheter bag sign). Prompt intraoperative surgical repair saves many a life-threatening complication.

References