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Pseudo-Azotemia Secondary to Bladder Injury: A Case Report with Review of Literatures

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Abstract:
Ascites, oliguria, and abdominal pain accompanied by an elevated serum creatinine level are commonly associated with acute renal failure. However, similar symptoms may arise in cases of intraperitoneal urinary leakage, leading to confusion with acute renal failure. Here, we present a 31-year-old female admitted as a case of iatrogenic bladder injury mimicking acute renal failure. She was initially diagnosed with suspected septic acute kidney injury on her 12th pot-operative day following cesarean section following progressive decrease in urine output, abdominal distension, significant abdominal pain, and diarrhea lasting for 4 days. On physical examination she had feature of sepsis with postoperative intraabdominal collection and surgical exploration was done found to have about 400 ml of urine in the peritoneal cavity and a 6cm full-thickness defect in the bladder dome. For the finding mentioned peritoneal fluid was drained and the defected bladder repaired in two layers. On 4th postoperative pseudo-azotemia resolved and the discharged with uneventful recovery. This case highlights diagnostic challenges and therapeutic considerations associated with this rare condition, discussed alongside a review of pertinent literature.

Keywords: *Pseudo-Azotemia; Ascites; Iatrogenic Bladder Injury*

Introduction

Bladder injury at the time of cesarean section (CS) is an infrequent occurrence [1-4]. During the past six 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].Here the authors report a case of a 31-year-old patient who undergone CS_2 weeks back presented with persisting abdominal pain, abdominal distension, decreased urine output and significantly elevated serum creatinine diagnosed to have ascites with abdomino-pelvic ultrasound examination. Through this observation and a review of the literature, the aim of work was to highlight the

diagnostic difficulties and the various therapeutic modalities of this pathology.

Case Summary

A 31-years old female was admitted to Emergency department of Yekatit 12 Hospital Medical College with assessment of r/o septic Acute kidney injury on her 12 postoperative day after cesarean section for an indication of previous cesarean section and postterm pregnancy presented with progressive decrease in urine output, abdominal distension with significant abdominal pain and diarrhea of 4 days duration. Otherwise no history fever or vomiting, failure to pass flatus and feces or other chronic medical illnesses. For this she went to nearby health center and referred to our hospital for better management with above impression. At presentation she was tachycardic, tachypneic and had low grade fever record. She had pale8 conjunctiva with significant abdominal distension with signs of fluid collection. She produced after catheterization 1000ml of urine.

She was investigated WBC-7000/μL, Hct-27%, peritoneal fluid analysis WBC-4000/μL with N-78%, urine analysis shows protein 2+, renal function being BUN-35, Cr-6mg/dL serum electrolyte was within normal range and serum Albumin was 3.7mg/dL. Bedside ultrasonography showed normal kidney with collapsed urinary bladder and massive intra-abdominal free fluid. Laboratory for determining Creatinine from the abdominal fluid was not available in our setup.

With an impression of Pseudo-azotemia 2ry to Iatrogenic bladder injury plus 12th postoperative day after cesarean section for an indication of previous cesarean section and postterm pregnancy and Started on intravenous antibiotics and prepared and taken to operation theatre.

Under General anesthesia, and through midline abdominal incision peritoneal cavity entered. Intraoperative finding was 4000ml urine in peritoneal cavity and there was 6cm full thickness bladder dome defect as shown on figure below with adhesion of bladder to that anterior abdominal wall. The peritoneal fluid sucked out and adherent bladder gently dissected off the anterior abdominal wall and the defect repaired in two layers and abdominal cavity lavaged and drain left in Douglas pouch and abdomen closed. Patient transferred to recovery room with stable vital sign after she is extubated. Patient transfused with two units of Packed RBC. Postoperative 2nd and 4th postoperative day Hct was 32% and 35% and Cr became 1.9mg/dL and 1.1mg/dL respectively. Drain was removed on the 4th postoperative day and she was discharged on the 5th postoperative day and she has been on follow up and she has no postoperative complication.

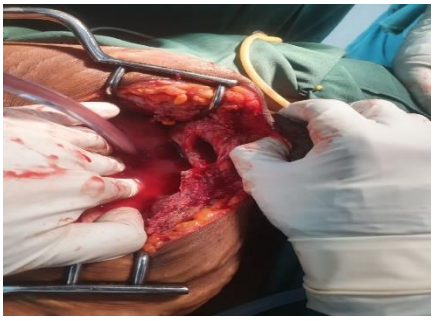

Figure 1: Arrow shows bladder dome defect

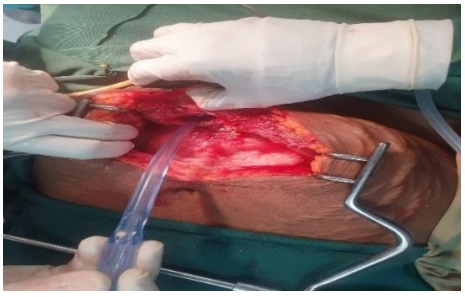

Figure 2: shows the intraoperative finding of the procedure performed

Discussion

A 31 years old female kept in medical emergency OPD with an assessment of r/o septic Acute kidney injury plus 12th postoperative day after cesarean section for an indication of previous cesarean section and post-term pregnancy presented with progressive decrease in urine output, abdominal distension with significant abdominal pain and diarrhea of 4 days duration. Otherwise no history fever or vomiting, failure to pass flatus and feces or other chronic medical illnesses. For this she went to health center and referred to our hospital for better management with above impression. At presentation she was tachycardic, tachypneic and had low grade fever record. She had pale conjunctiva with significant abdominal distension with signs of fluid collection. She produced after catheterization 1000ml of urine.

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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, 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 and signs of peritoneal irritation or peritonitis 2-4 days after bladder injury [8]. In our case, as in others, low abdominal pain may present as early as 2-4 days after surgery. 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 catheterization only draining of urine. However, when a urinary catheter was reinserted at our emergency room, 1 liter of urine was drained [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 urine output. 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 CO2 content, and development of metabolic acidosis) are consistent findings among patients with intraperitoneal bladder rupture [10]. When urine enters the peritoneal cavity, reverse auto-dialysis 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 bladder repair done 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 ultrasonography showed no hydronephrosis. The diagnosis of bladder perforation can be confirmed radiologically by extravasation of contrast from the bladder which is not done in our patient [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.

Conclusion

In summary, we herein reported a case of intraperitoneal urinary leakage resembling acute kidney injury. In this case, the serum creatinine levels markedly elevated and due to unavailability of laboratory there was difficult in determining ascitic fluid creatinine level. Hence, patients presenting with an acutely rising serum creatinine level, recurrent ascites and oliguria should be assessed for intraperitoneal urine leakage by an internal medicine specialist. 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.

Consent

A written informed consent was obtained from the patient for the operation and publication of this case report and image taken during intraoperative time.

Acknowledgment

The authors would like to thank everyone who took part in the management of the patient. Our gratitude also goes to the Yekatit 12 Hospital Medical College administration for their permission to use the patient medical records.

Conflict of interest

We, the Authors declare that there is no conflict of interest.

References

1. Kaskarelis D, Sakkas J, Aravantinos D, Michalas S, Zolotas J (1975) Urinary tract injuries in gynecological and obstetrical procedures. Int Surg 60: 40-43.
2. Rajasekar D, Hall M (1977) Urinary tract injuries during obstetric intervention. Br J Obstet Gynaecol 104: 731-734.
3. Phippus MG, Watabe B, Clemons JL, Weitzen S, Myers DL (2005) Risk factors for bladder injury during cesarean delivery. Obstet Gynecol 105: 156-160.
4. Rahman MS, Gasem T, Al Suleiman SA, Al Jama FE, Burshaid S, et al. (2009) Bladder injuries during cesarean section in a university hospital: a 25-year review. Arch Gynecol Obstet 279: 349-352.
5. Adhikary S, Mathews P, Gopalakrishnan G (2005) Renal failure and ascites after remote laparoscopy. CMAJ 173: 1323.
6. Kruger PS, Whiteside RS (2003) Pseudo-renal failure following the delayed diagnosis of bladder perforation after diagnostic laparoscopy. Anaesth Intensive Care 31: 211-213.
7. Darmon JC, Chevallier L, Diemunsch P, Saussine C, Favreau JJ, et al. (1997) Urinary complications during laparoscopy: a urachal diverticula injury. Contracept Fertil Sex 25: 385-388.
8. Vilos GA, Haebe J, Crumley TL, Maruncic MA, King JH, Denstedt JD (2001) Serum biochemical changes after laparoscopy may be indicators of bladder injury. Am Assoc Gynecol Laparosc 8: 285-290.
9. Hassan I, Lo´pez C, Gee H, Toozs-Hobson P (2009) Urinary ascites following caesarean section: an unusual presentation of bladder injury. Eur J Obstet Gynecol Reprod Biol 147: 237-238.
10. Heyns CF, Rimington PD (1987) Intraperitoneal rupture of the bladder causing the biochemical features of renal failure. Br J Urol 60: 217-222.
11. Mokoena T, Naidu AG (1995) Diagnostic difficulties in patients with ruptured bladder. Br J Surg 82: 69-70.