A Case of Reverse Dialysis

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Abstract: A 28 yrs old lady underwent diagnostic laparoscopy as a part of evaluation for primary infertility. A midline cystic lesion was incidentally detected in the anterior abdominal wall. Post-op, patient developed abdominal distension, nausea and vomiting. Lab parameters showed elevated serum urea and creatinine. USG showed minimal ascites with normal kidney size and parenchyma. Ascitic fluid analysis revealed raised peritoneal fluid urea & creatinine levels than serum levels with peritoneal fluid - serum creatinine ratio of 3.9. CT cystography revealed extravasation of dye suggestive of urinary leak causing urinary ascites. She was diagnosed as intra op bladder rupture causing reverse dialysis resulting into pseudo-renal failure. Post laparoscopic repair, patient improved with normalisation of renal parameters. This case report aims to increase awareness of urinary ascites with any unexplained azotemia in the back ground of genitourinary surgical procedure.

Keywords: Reverse Dialysis, Urinary Ascites, Pseudo Renal Failure, Bladder rupture

1. Introduction

Pseudo-renal failure describes a rare condition, with biochemical abnormalities that mimic Acute Kidney Injury (AKI) in the setting of normal kidney functions (Gomerular filtration rate) without any structural abnormality. One of the causes is reverse dialysis which occurs in urinary ascites, where peritoneal membrane acts as filter and absorbs urea, creatinine and allows exchange of electrolytes. Urinary ascites occurs when upper or lower urinary tract ruptures causing intraperitoneal urinary leak followed by reverse intraperitoneal dialysis of urine (Reverse Dialysis). Biochemical parameters in urinary ascites closely mimic AKI, including elevated blood urea nitrogen, elevated serum creatinine, hyperkalemia and metabolic acidosis [1]. Surgical management to the rupture of urinary system is the key to recovery.

2. Case Report

A 28 years old lady married for last four years, being evaluated for primary infertility underwent planned diagnostic laparoscopy. Intra-op findings revealed 2 x 2 cm Cystic Lesion posterior to the anterior abdominal wall in midline below umbilicus which was resected. Procedure was uneventful and patient was comfortable. She was shifted to the ward for observation. In ward, she was unable to pass urine. There was no sensation of bladder fullness. No history of dysuria, fever, intermittent stream, loin pain or anasarca. After 7-8 hrs, patient was catheterized, drained 1000 ml of Urine. Catheter was removed next day morning. Following catheter removal she was unable to pass urine with abdominal distension. Patient was again catheterized. Urology consultation was taken for recurrent urinary retention. Patient had normal urine output and renal function tests. Catheter was removed. She was able to pass small quantity of urine. Subsequently she developed anorexia, nausea and vomiting with azotemia.

3. On Examination

Averagely built lady with normal vital parameters. No periorbital puffiness or pedal edema. JVP not raised. On examination, abdomen was soft and non tender. There was no organomegaly or ascites. Bladder was not palpable.

4. Investigations

Haemogram was normal. Serum biochemistry revealed progressive azotemia (Urea/Creatinine – 35/1.13 → 55/3.53 mg/dL) with normal electrolytes. Liver function tests revealed Bilirubin 0.6 mg/dL, aspartate aminotransferase 16 IU/L, alanine aminotransferase 17 IU/L with total serum protein & albumin being 6.8 g/dL & 3.9 g/dL respectively. Urine analysis was normal. Ultrasonography revealed normal kidney size, maintained cortico-medullary differentiation with no evidence of obstructive uropathy. Minimal ascites was noted. USG guided ascitic tap was done from a small pocket of fluid collection.

<table>
<thead>
<tr>
<th>Peritoneal fluid analysis</th>
<th>Value</th>
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<tbody>
<tr>
<td>WBC / RBC</td>
<td>NIL / 02</td>
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<tr>
<td>GLUCOSE</td>
<td>104 mg/dl</td>
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<tr>
<td>T PROTIEN</td>
<td>0.9 g/dl</td>
</tr>
<tr>
<td>ALBUMIN</td>
<td>0.5 g/dl</td>
</tr>
<tr>
<td>UREA</td>
<td>66 mg/dl</td>
</tr>
<tr>
<td>CREAT</td>
<td>12.9 mg/dl</td>
</tr>
<tr>
<td>Peritoneal Fluid: Serum Creatinine ratio</td>
<td>3.9</td>
</tr>
</tbody>
</table>

In view of grossly elevated urea & creatinine values with peritoneal fluid serum creatinine ratio 3.9, patient was suspected to have urinary ascites. Patient underwent CT Cystographic studies which revealed extravasation of dye suggestive of urinary leak causing urinary ascites.
Figure 1: CT CYSTOGRAM
(Showing leaking of dye in peritoneal space suggestive of urinary leak.)

Figure 2: CT Cystogram showing leak from bladder with peritoneal spill

Figure 3: CT Cystogram showing patent urachus. The cyst which was removed during diagnostic laparoscopy was likely urachal cyst causing urine leak leading to urinary ascites.

Diagnosis

She was diagnosed as intra op bladder rupture causing reverse dialysis resulting into pseudo-renal failure.

Management

She was taken up for emergency laparoscopic repair of the defect. Following surgery, patient recovered completely with normalisation of renal parameters over 48 hrs.

5. Discussion

Bladder rupture leading to urinary ascites is the most common cause of pseudo renal failure [2]. Bladder rupture can occur because of trauma [3], surgical procedure [4] post radiotherapy to pelvic organs [5], or even spontaneously. Gynaecological procedures, urological procedures like TURBP or abdominal surgeries are likely to injure urinary bladder. Posterior urethral valve is the most common cause of spontaneous urinary ascites in neonatal urinary ascites. [6]

Bladder perforation due to any cause will cause steady urinary leak resulting into progressive urinary ascites which is generally painless. Peritoneal membrane acts as filter and absorbs urea, creatinine and allows exchange of electrolytes [7]. This leads to rise in serum urea and creatinine levels and clinical features of uremia. Despite normal renal excretory functions (normal GFR), patient presents with progressive uraemia (pseudo-renal failure). Patient improves marginally with removal of urine, either through catheterisation or through therapeutic paracentesis. In such cases, it is found out that after every episode of paracentesis, there was a transient improvement in urine output followed by rapid reaccumulation of ascites. It requires surgical closure of the defect as definitive management.

In our case, cystic lesion was removed during diagnostic laparoscopy which was likely urachal cyst.
Urachal cyst occurs in the remnants between the umbilicus and bladder [8]. Normally, the urachus closes down towards the end of pregnancy. Defective obliteration of the urachus leads to urachal abnormalities. The incidence of urachal remnants is about 1.03% of the population [8].

Urachal cyst can get infected and usually presents with pain abdomen and fever. It may rupture leading to peritonitis or it may drain through umbilicus. It can be managed by antibiotics, percutaneous drainage and surgical removal [9].

Bladder rupture generally presents with painless progressive ascites. CT cystography is the most sensitive and accurate diagnostic test for bladder rupture. Deck et al [10] reported that the sensitivity and the specificity for diagnosing intraperitoneal bladder rupture using CT cystography were 78% and 99%, respectively. Prognosis is good if detected early. It can be fatal if gets infected. It requires definitive surgical management.

Pseudo-renal failure closely mimics AKI and hepatorenal syndrome. This diagnostic error can lead to inappropriate medications and haemodialysis.

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References