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What not to expect when you're expecting – Postpartum proximal ureteric rupture: A case report

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ABSTRACT

Introduction: Spontaneous ureteric rupture is an extremely rare cause of acute abdominal pain in the intrapartum and postpartum period. We present the case of a right ureteric rupture diagnosed immediately postpartum.
Case: A 23-year-old woman in her second pregnancy (who had had a previous caesarean section) developed acute-onset right-flank pain 12 h after vaginal delivery. A contrast computerized tomography scan suggested a ureteric injury; ureteroscopy diagnosed a proximal ureteric rupture and a stent was placed.
Discussion: This case outlines an extremely rare cause of abdominal pain in the peripartum. There can be serious complications, including urinoma, abscess and sepsis, and therefore the diagnosis should not be delayed.

1. Introduction

Spontaneous ureteral injury, defined as injury which occurs in the absence of any interventional procedure, external trauma or surgery, is a rare cause of acute abdominal pain. Intrapartum rupture of the ureter is an uncommon complication and in the absence of known renal pathology is particularly rare [1]. Complications of ureteral rupture include urinoma, abscess and sepsis [1]. It is important to consider this as a potential cause of acute abdominal pain in the obstetric population so that the diagnosis is not delayed.

2. Case Presentation

A healthy 23-year-old woman with a background of one previous caesarean section (gravida 2, para 1) was admitted for induction of labour at 40 weeks of gestation because of her South Asian origin. A cervical ripening balloon catheter was inserted overnight and removed the next day. An artificial rupture of membranes was performed with the observation of clear liquor and intravenous oxytocin infusion commenced. At 4 cm dilatation the woman requested epidural anaesthesia, and an indwelling catheter (IDC) was inserted promptly.

Full dilatation was reported 6 h after the commencement of oxytocin and 1 h was allowed for passive descent of the foetal head before pushing commenced. After 10 min of active pushing the cardiotocograph became pathological, with late and prolonged decelerations, so the decision was made for an instrumental delivery. The indwelling catheter balloon was deflated prior to delivery, with rosé-coloured haematuria noted prior to removal. No significant vaginal bleeding was noted prior to delivery.

A low cavity vacuum delivery with a right mediolateral episiotomy was performed. The baby was delivered occiput-anterior with two pulls and a McRoberts manoeuvre was required to deliver the foetal shoulders. She delivered a healthy male neonate with Apgar scores of 9 and 10 at one and 5 min postpartum. The third stage of labour was complicated by postpartum haemorrhage secondary to atony and local haemorrhage at the episiotomy site. A syntocinon infusion and ergometrine were administered with good effect, and the episiotomy laceration required routine suture repair. The estimated blood loss was 600 mL. An IDC was reinserted following delivery and clear urine was noted. The epidural catheter was removed immediately following delivery. The woman remained well overnight with no concerns of pain or haemodynamic instability.

The IDC was removed on day 1 postpartum with one normal micturition taking place 2 h after removal. Fifteen hours after delivery the woman developed acute right-flank pain radiating to the right iliac fossa. The pain was constant and severe, requiring opioid analgesia. On examination she had rebound tenderness at the right iliac fossa and right renal angle. An IDC was re-inserted, draining approximately 1000 mL of clear urine, with no improvement in pain after bladder decompression. The woman became febrile (38.4 °C), tachycardic and hypotensive. Haemoglobin level was 86 (normal range 105–145), white cell count 6 (normal range 4–14.5), venous lactate 1.9 (normal range 0–2.0), creatinine 58 (normal range 45–90) and CRP 87 (normal range <3).

Early provisional diagnoses considered were uterine rupture, puerperal sepsis, renal calculi and appendicitis. Initial resuscitative procedures included IV crystalloid, ceftriaxone and metronidazole plus one dose of gentamicin. Although the urine output was >100 mL/h the woman remained hypotensive and tachycardic.

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Computerized tomography (CT) of the abdomen and pelvis with contrast was performed, looking for signs of uterine rupture. The CT scan (Fig. 1) demonstrated a small volume of free fluid in the right retroperitoneal space, involving the inferior perinephric space and extending into the lateral pelvic side wall. There were no signs of uterine rupture. The radiologist reported the fluid was simple in nature and suspected the fluid to be urine.

Bloods were repeated and haemoglobin was stable at 86, haematocrit 0.265 (normal range 0.37–0.47), CRP 76 and creatinine 50. Preliminary results of urine microscopy showed the presence of organisms. The impression at this time was likely sepsis from a urinary tract source. The fluid seen on CT was considered to be possibly be transudative fluid. A lower urinary tract injury was considered unlikely, given normal urine output and clear urine. The patient remained haemodynamically unstable, with ongoing tachycardia and hypotension. She was transferred to the intensive care department and commenced on IV metaraminol approximately 28 h after the birth. One unit of packed red blood cells was given.

The urology team was consulted regarding the CT scan results and recommended a CT IV pyelogram for further investigation of possible urinary tract injury. This was performed and confirmed contrast extravasation at the level of the right mid ureter at the level of the fourth lumbar vertebra. There was no associated hydronephrosis bilaterally. The radiologist also reported that there was no collection amenable to radiologically guided drainage.

This ureteric injury was an unexpected finding, particularly given the patient was maintaining a urine output of >100 mL/h and the urine was clear. The urology team reviewed the case immediately and an emergency cystoscopy was performed in the morning, 24 h following the initial development of the pain. Posterior bladder wall ecchymosis was noted with an otherwise normal mucosa and ureteral orifice. A guide wire could not be passed initially under X-ray guidance, so the decision was made to proceed to a right rigid ureteroscopy (Fig. 2). This demonstrated a grade III incomplete proximal ureteric rupture, correlating to the level of suspected injury on CT. This was treated with a ureteric stent, which was placed over a wire placed under vision and position confirmed on X-ray.

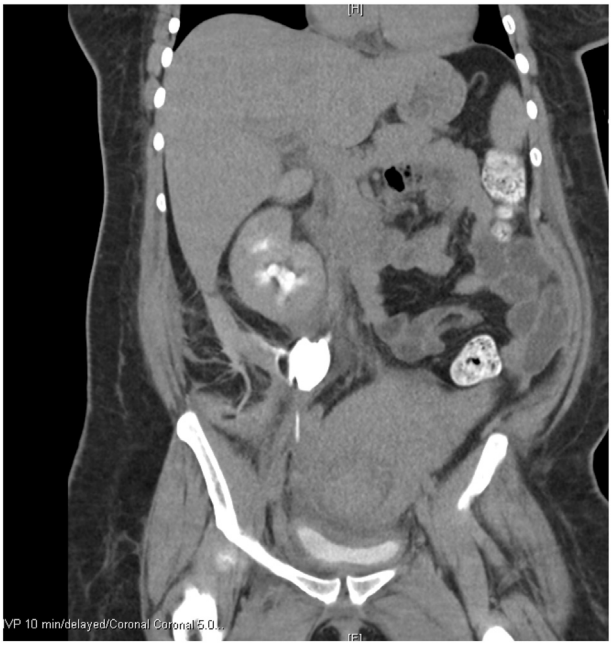

Fig. 1. CT Intravenous pyelography - Delayed phase demonstrating ureteral rupture, urinoma and extravasation of contrast.

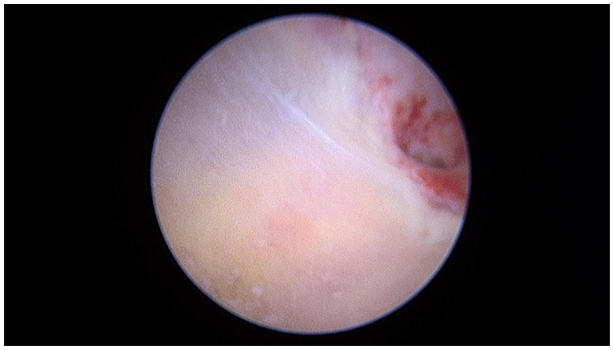

Fig. 2. Ureteroscopy image; demonstrating ureteric lumen (right) and region of laceration (left) with surrounding fibrous tissue and periureteric fat clearly seen.

Postoperatively the patient was monitored in the intensive care unit. She had persistent fevers and antibiotics were upgraded to IV piperacillin/tazobactam for five days. Urine microscopy was positive for *E. coli*. She was stepped down to ward-based care, remained well for the remainder of her admission and was discharged home 10 days after delivery.

Follow-up was planned for a cystoscopy, retrograde pyelogram and removal or exchange of the stent 6 weeks postoperatively. At this time the intra-operative pyelogram demonstrated no radiological evidence of ureteric contrast leak, so the stent was removed with no complications.

3. Discussion

Spontaneous ureteral rupture is defined as ureteral damage in the absence of external trauma, degenerative kidney disease, previous surgery or iatrogenic manipulation [2]. Ureteral rupture is caused by increased pressure in the renal collecting system [3]; when pressure exceeds a critical level, reported as 20 mmHg to 75 mmHg, rupture may occur [2]. The most common causes of increased intraluminal pressure are nephrolithiasis or ureteric strictures [3]. Rarely, malignancy, bladder retention or external compression by tumours, vascular structures, or the gravid uterus [3] can elevate pressure in the ureter, causing ureteric injury.

Renal tract injury during pregnancy or delivery, whilst very rare, has been described at the distal, mid and proximal ureter as well as at the renal calyces and the bladder. Rupture of the ureter is an exceptionally rare obstetric complication. Complications of ureteral rupture include urinoma, abscess and sepsis [1].

During pregnancy there is a physiological dilatation of the urinary collecting system, with hydronephrosis found in up to 80% of women [4]. This is attributed to both hormonal and anatomical changes in pregnancy. Mechanical compression is considered a major cause from enlarged vascular structures and the uterus. Right-sided dilatation occurs up to 80% more than on the left, due to the relationship of the right ureter to the right iliac artery and ovarian artery at the pelvic brim as well as the natural dextrorotation of the uterus by the sigmoid colon [1]. In the third trimester, dilatation is more likely due to the foetus compressing the ureter (often the foetal head) [5]. Progesterone has also been postulated to contribute to smooth muscle relaxation effects on the ureter [5]; however, studies in non-pregnant women with progesterone have been unable to show any evidence of this, and there is no relationship between progesterone levels and severity of dilatation [4].

Rupture of the urinary tract is rare in the intra- or postpartum setting, and when reported is normally in previously diseased kidneys. There have been four other spontaneous postpartum ureteral ruptures reported [1,3,6,7]. Our patient did not have any underlying renal or ureteric pathology or other predisposing factors. She had no indication for renal imaging during or before her pregnancy.

The precise aetiology of ureteric rupture in this case is unknown. We hypothesize that the increased ureteric intra-luminal pressure from physiological hydronephrosis of pregnancy in addition to rapid changes in intra-abdominal pressure while in active labour may have caused ureteral injury. It is also possible that the active stage of labour may have placed some traction on the ureter itself. Furthermore, an overly distended bladder after IDC removal could also have exacerbated the rupture (1000 mL was passed from the IDC when it was reinserted). Due to the proximal level of ureteric injury in relation to the pelvis, we consider it to be highly unlikely that avulsive force from instrumental delivery or scarring from her previous caesarian contributed to the causation of injury.

In conclusion, it is important to have a high index of suspicion for postpartum renal tract injury, as it can result in significant morbidity if not identified early. A low threshold for testing for urinary tract infection is also needed as well as attempting to avoid bladder overdistension postpartum, in case such conditions can contribute to ureteric injury.

Contributors

All authors were involved in the clinical care of the patient and contributed to the conception, drafting, review, and revision of the manuscript. All authors read and approved the final version of the paper and take full responsibility for the work.

Conflict of Interest

The authors declare that they have no conflict of interest regarding the publication of this case report.

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Patient Consent

Informed consent was obtained from the patient for publication of this work.

Provenance and Peer Review

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