Prolonged postpartum urinary retention: A case report and review of the literature

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Postpartum urinary retention (PUR), which is defined as difficulty in emptying the bladder completely after delivery, may be clinically pronounced or silent. The incidence differs according to the definition. Although many risk factors for this disturbance are identified in the literature, every patient at risk does not necessarily present with PUR. There is no consensus in the literature regarding management.

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Postpartum urinary retention (PUR) is defined as inability to empty the bladder completely after delivery.[1] The detailed overt and covert classification by Yip *et al.*[2] has been widely used. Overt urinary retention is defined as the inability to void spontaneously within 6 hours of delivery, and covert urinary retention is defined as a post-void residual bladder volume of ≥150 mL after spontaneous micturition. The incidence of PUR ranges between 0.05% and 37% as a result of variable definitions based upon different parameters.[3] There are only a few reports describing the prolonged form with an incidence of 0.05 - 0.06%,[4,5] described as clinical presentation lasting >7 days.[4] We report a case of prolonged PUR with long-term sequelae and review the literature.

Case report

A 28-year-old primipara who had given birth to a term 3 350 g infant in a maternity hospital was admitted to our outpatient clinic on day 4 post partum, complaining of abdominal pain, urinary incontinence and inability to void adequately. Her history revealed that her first stage of labour had lasted about 6 hours and the second stage only half an hour. Neither vacuum nor forceps application was needed during delivery, and the only intervention was mediolateral episiotomy.

At admission, gynaecological examination revealed that the episiotomy scar was intact and there was no sign of periurethral or clitoral laceration or infection. Physical examination of the abdomen revealed a palpable and painful mass. Abdominal ultrasonography showed a very large (20 × 18 × 15 cm) distended bladder and right urethrohydronephrosis. The results of laboratory blood tests were as follows: creatinine 1.69 mg/dL, blood urea nitrogen 28.73 mg/dL, haemoglobin (Hb) 7.9 g/dL, and white blood cells (WBCs) 10.3 × 10^3/μL. Urine analysis revealed the following: protein 100 mg/dL, leucocytes 75/μL, 7 WBCs/high-power field (HPF), and 8 red blood cells /HPF. Culture of the urine was negative for any micro-organisms. The patient was catheterised immediately with a 16F nelaton catheter and 3 000 mL clear urine was drained. Her pain was instantly relieved.

The urinary catheter was removed 24 hours later, but the patient was unable to void spontaneously. After consultation with the urology and nephrology departments, she was catheterised for a second time for 10 days. Meanwhile, with appropriate hydration, renal function tests returned to the normal range within 24 hours. Ten days later, attempts at spontaneous micturition failed for the second time, and the patient was catheterised again. Urodynamic tests and pelvic magnetic resonance imaging (MRI) were scheduled and clean intermittent self-catheterisation (ISC) was suggested as the next step. MRI excluded possible neurological problems such as spina bifida. Urodynamic tests revealed that bladder capacity was 650 mL without any urge for micturition and the maximum voiding phase detrussor pressure was 44 cm H_2O. Uroflowmetry performed 10 days after removal of the catheter showed a maximum flow rate (Qmax) of 7 mL/s, a mean flow rate (MFR) of 5 mL/s, urine volume of 150 mL and residual volume of >100 mL.

Renal ultrasonographic findings returned to normal within 2 weeks. The patient was followed up by ISC (after spontaneous voiding four times daily) and uroflowmetry intermittently. When the residual volume was less than 100 mL, ISC was stopped (about the second month post partum). At the fourth month post partum, the patient still had some voiding dysfunction (Qmax 15 mL/s, MFR 9 mL/s, post-void residual bladder volume <50 mL at uroflowmetry).

Discussion

Despite incontinence related to pregnancy or labour having been widely researched, mechanisms of disturbance resulting in PUR have not been fully explained. The reasons why this condition does not occur in all patients with predisposing risk factors have yet to be elucidated.

Changes during pregnancy, such as detrussor muscle hypertrophy, perineal or pudendal nerve damage during delivery and mucosal oedema after vaginal delivery, may result in voiding dysfunction.[6-8] The most important predisposing risk factors for covert PUR are instrumental delivery and prolonged labour (>700 minutes).[9,10] Regarding PUR, tissue oedema and bladder neck obstruction,[11] detrussor muscle injury,[12] catheterisation during labour,[13] epidural anaesthesia[14] and postpartum morphine[15] have also been implicated. In our case, the only predisposing risk factor for PUR was nulliparity, which according to the literature is the least

significant of the accepted risk factors.[1] In this case, prepartum unknown dysfunction of bladder or accompanying factors other than those that have previously been proposed may therefore have contributed to PUR.

There is no standardised management protocol for PUR, although this clinical condition may be seen in up to 37% of patients after vaginal delivery.[16] Since a single episode of bladder distention may result in irreversible damage of the detrusor muscle, follow-up of voiding function in postpartum women is crucial.[17] Early diagnosis of the urinary retention may prevent further distention and possibly longer periods of voiding dysfunction.

A suggested first treatment step, after excluding infection, consists of analgesics, mobilisation and adequate patient privacy.[18] The second step involves urinary catheterisation for a recommended 24 - 48 hours; this time may be prolonged if the voiding function does not return to normal. Suprapubic catheterisation is another option, but a more invasive method for a woman who has to take care of her newborn baby. Our patient was catheterised for a short period of time, but since she could not void adequately, indwelling catheterisation for 10 days was performed, which resulted in a better voiding performance although it was still not adequate. Urodynamics and MRI were performed to exclude any comorbid aetiology. Uroflowmetry, which is less invasive and easy to perform, was used for follow-up of the patient. Previously, in the follow-up of 55 patients with PUR, 10.4% stress incontinence, 8.3% overactive bladder and 6.3% voiding difficulties were reported with totally normal urodynamic evaluation.[10]

Duration of the voiding dysfunction varies widely in the literature. In one review, it was postulated that most patients recover within 2 weeks of the failed trial of voiding.[3] An investigation of overt PUR revealed that resolution time was 48 hours in 45.0% of patients and 72 hours in 29.4%, and 25.5% had required ISC for up to 45 days.[19] The duration of PUR was also no longer than 8 weeks in cases reported by Humburg *et al.*[4,18] Among the prognostic factors in cases with PUR, a high volume of urine at the time of diagnosis is of concern. Urinary volumes greater than 700 - 750 mL have been known to result in extended duration of catheterisation.[20]

In our case, 3 000 mL of urine drained at the time of admission is the only remarkable risk factor for such prolonged clinical symptoms. This patient had a very long duration of overt PUR. Spontaneous micturition was not possible before 25 days, and ISC had to be continued until the third month post partum. As far as we know, our patient has the longest duration of voiding dysfunction reported to date.

In conclusion, PUR remains a matter for debate, since the aetiology and management have not yet been clarified. Also there appears to be an urgent need for longitudinal prospective studies to establish its long-term consequences.

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