
Case Report

Acute renal failure—an unusual consequence of uterine prolapse

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Introduction

Total uterine prolapse occurs when both the cervix and the fundus of the uterus completely descend outside the vaginal introitus. The entire vagina is everted and there is often an accompanying cystocele and/or rectocele, as well as enterocoele. Childbirth via the vaginal route, oestrogen deficiency, chronic intra-abdominal pressure from pulmonary disease or lifting, chronic straining during bowel movements, and neurological injury may all play role in the development of genital prolapse, and, therefore, this is more commonly a disease of multiparous, post-menopausal women [1,2]. Uterine prolapse might cause obstruction of the lower ureters in addition to disturbances in bladder and bowel function. The prevalence of ureteral obstruction varies widely from 2 to 92% in several reports [1]. We present a case of neglected uterine prolapse in a 25-year-old patient resulting in acute renal failure.

Case

A 25-year-old (gravida = 2, parity = 2) woman living in a rural village was referred to the University Hospital in January 1998 with the diagnoses of total uterine prolapse, massive eversion of the vagina and rectal prolapse, as well as renal failure. She was poorly cooperative and appeared to be mentally retarded. She had been married for 7 years and had given birth to her second child 3 years previously. One week after this vaginal delivery, her uterus had prolapsed whilst chopping wood. She had visited two or three doctors, but had not been able to afford treatment previously. Occasionally, bleeding occurred from the ulcerated lesions over the prolapsed organs; she had dysuria, pelvic pain as well as difficulty in defaecation. She had regular menses and did not have urinary incontinence. Her past and family histories were not significant.

Physical examination was unremarkable, except mental retardation. The pelvic examination revealed uterine and rectal prolapse (Figure 1). The vaginal mucosa was ulcerated and hygiene was quite poor. Laboratory studies disclosed the following results: Hb 8.5 g/dl; blood urea nitrogen 33 mmol/l; serum creatinine 451 μmol/l; albumin 29 g/l; potassium 3.7 mmol/l; chloride 95 mmol/l; calcium 3.0 mmol/l; and phosphorus 2.0 mmol/l. Urinalysis showed a pH 6.5, specific density 1030, abundant leucocytes, and was negative for protein and glucose. The urine culture revealed Staphylococcus aureus infection. Her daily urine volume was 1500–2000 ml. Transvaginal ultrasonography was performed via reduction of the prolapsed organs, and this was normal. Transabdominal ultrasonography showed bilateral hydronephrosis (Figure 2) with dilated proximal ureters. The longitudinal axis of the right kidney measured 170 mm and the left kidney measured 161 mm. Both kidneys had increased parenchymal echogenity of grade 2–3. Histopathological examination of the ulcerated vaginal and cervical mucosa revealed chronic inflammation.

As it is uncommon to observe such an advanced case of uterine prolapse in a young patient, we looked

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for joint hypermobility and thoracic kyphosis to detect a possible underlying disorder. The patient did not have joint hypermobility but had a thoracic kyphosis of 21.0° measured by the Ferguson method [3] from the lateral radiograph of the spine. We were unable to perform electromyographic (EMG) examination of the pelvic floor muscles because the patient was uncooperative and could not relax.

The patient was managed with protein restriction, intravenous fluids, urethral catheterization, oloxacin (i.v.), allopurinol (p.o.), erythrocyte transfusion and bed rest with the reduction of the prolapsed organs. Her urine volume increased up to 8 l/day. Future fertility was not desired and, 1 week posthospitalization, vaginal hysterectomy together with anterior and posterior colporrhaphy, and culdoplasty were performed. There were no complications associated with the surgery. She was discharged with a blood urea nitrogen 5.4 mmol/l and serum creatinine 80 μmol/l on the 10th post-operative day.

Discussion

Hydroureteronephrosis due to uterine prolapse was the major cause of acute renal failure in our patient. Hyperuricaemia and urinary tract infection may be other predisposing factors, and we think hyperuricaemia and urinary tract infection are related to the hydroureteronephrosis. Genital prolapse is a distressing condition as it causes discomfort with daily activities as well as with sexual activity. It interferes with bowel and bladder function, predisposes the patient to recurrent urinary tract infections and possible renal problems by ureteral obstruction [4]. Renal failure caused by uterovaginal prolapse was first described by Froriep in 1824 [1]. This is a rare condition today as health care has improved throughout the world. There are reported cases of post-hysterectomy vaginal vault prolapse causing renal failure due to chronic partial ureteral obstruction [5]. The obstruction occurs as the uterus descends, the downward traction of the uterine arteries located over the lower ureters causing the bladder trigone and lower ureters to be dragged outside the pelvis. The caudal displacement of the trigone results in compression of the ureters between the uterus and the medial borders of the genital hiatus [1]. The resulting hydroureteronephrosis is usually asymptomatic, but recurrent urinary tract infections may occur. Impairment of renal function is generally limited to neglected cases [1, 5].

The anatomical supports of the female genital organs are the pelvic cellular connective tissue and the levator ani muscle group [6]. The active basal tone of the levator ani muscles helps to maintain the urogenital hiatus closed [7]. Tearing and separation of the pelvic floor muscles, especially at the time of vaginal delivery, will weaken the pelvic floor. Childbirth also causes stretching or compression of the pelvic nerves as they traverse the pelvic floor, resulting in partial denervation of the pelvic floor musculature. Subsequently, recovery may occur as the denervated muscles are re-innervated by the surrounding intact nerves [1, 7]. Single-fibre EMG studies of the pubococcygeus muscle indicate significantly increased fibre density in patients with prolapse compared with normal control subjects [8]. Genetic factors are probably important as patients with defects in collagen synthesis are predisposed to genital prolapse. There are reports which show an association between genital prolapse and joint hypermobility which is a known clinical marker for abnormal collagen [2]. In our patient, we were unable to demonstrate such an abnormality.

The bony pelvis and the spinal curvature might have possible roles in genital support as well [6]. The rare finding of genital prolapse in the newborn is almost always associated with spina bifida and other congenital defects, including kyphosis [6]. In the study of Lind et al. performed on adult patients with uterine prolapse, the mean thoracic spinal curvature was 13.0° vs 8.1° in controls. Thoracic kyphosis of 21.0° was present in our patient. The defect in pelvic supporting tissues in this patient is due primarily to the recent vaginal delivery, with thoracic kyphosis having a possible role as well. Together with these predisposing factors, an acutely increased intraabdominal pressure due to physical exertion resulted in genital prolapse. In conclusion, acute renal failure secondary to hydrenephrosis is a rare sequela of genital prolapse.

References


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