Asymptomatic large bladder diverticulum

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Summary

The authors report a case of a 61-year-old woman diagnosed with large bladder diverticulum. Diagnosis was performed only after a series of investigations carried out for the occasional finding of hypercreatininaemia. Although the significant volumes of post void residual (PVR) and the relevant urine stagnation in the diverticulum, subjective symptomatology was absent and urinalysis and urine culture were negative. The scheduled therapeutic plan consisted of fosfomycin three grams every ten days for six months, self-catheterization twice a day, voiding on a time schedule, and adequate fluid intake. The monthly scheduled follow-up at one year showed good general health, good compliance with the therapy, no urinary tract infections, a decrease in creatininemia to 1.2 mg/dl, and regression of nephrohydrosis to a mild stage. In conclusion, the absence of symptoms and negative urinalysis or urine culture allows expectant management despite the considerable size of the bladder diverticulum.

Key words: Bladder diverticulum; Hypercreatininaemia; Post void residual; Self-catheterizations.

Introduction

Bladder diverticula are projections of the mucous membrane through hernia openings in the bladder muscular coat (false diverticula) or, more rarely, projections of all the layers of the bladder wall (true diverticula) [1]. The causes are due to weakness of the bladder wall and/or to the increased pressure inside the organ, both due to urethral obstructions or to neurogenous alterations in bladder voiding. Bladder diverticula are generally classified into two groups as primary (congenital) and secondary (acquired). Primary diverticula develop as a result of congenital weakness of Waldeyer's fascia sheath, without bladder outlet disorder, whereas secondary diverticula usually occur as a result of neurogenic bladder or infravesical obstructions, such as in the posterior urethral valve. Stage *et al.* [2] described iatrogenic diverticula occurring as a result of ureteral reimplantation, suprapubic cystostomy, or after closure of the rectovesical fistula as a third group. In general bladder diverticula develop from congenital detrusor muscle defect and frequently present with urinary tract infection, which occurs as a result of urinary stasis in the diverticula. Different clinical presentations, such as bladder outlet obstruction, cyanosis of the lower extremities, intestinal obstruction, ureteral obstruction (which may occur due to direct diverticular compression), and peritonitis due to spontaneous rupture of the diverticula, were previously reported. In contrast, "urethral stenosis" involves the narrowing of the urethral channel (diameter) usually due to fibrotic processes resulting from lesions with many different causes (inflammations, infections, neoplasias) and responsible for alterations in bladder voiding [3-7].

Case Report

A nulliparous 61-year-old woman, 24 BMI (height: 168 cm, weight: 67 kg), suffering from Hashimoto thyroiditis and gastroesophageal reflux, was in good health. At 38 years an abdominal subtotal hysterectomy with ovarian conservation was performed because of menorrhagia due to uterine fibromatosis, with regular postoperative period and no complication. Five months later, surgical removal of cervical stump was performed because of moderate vaginal haematogenous bleeding. The patient experienced urinary retention in the second postoperative day at the time of bladder catheter removal; therefore, the catheter was repositioned. At catheter removal 48 hours later, the patient still experienced difficult urination characterized by a slow flow, difficulties in voiding the bladder together with significant volumes of post void residual (PVR). Nonetheless, as urinalysis results were negative and no fever was reported, the patient was discharged and prescribed intermittent self-catheterization. About three months later, the patient on her own initiative gradually suspended intermittent catheterization and regular urination was re-established. Since then, the patient was in good health for about 23 years. At 61 years, she began to suffer from abdominal pain referred to a colitis; the general practitioner prescribed routine blood chemistry analyses and accidentally diagnosed hypercreatininaemia (2.2 mg/dl). Therefore, kidney and abdominal ultrasound were performed and showed: moderate bilateral nephrohydrosis with anterior-posterior diameter of the renal pelvis of 37 mm on the right and 32 mm on the left (such values remained unchanged in the post void examination); a distal ureteral ectasia on both sides in the juxta-vesicular area; bladder overdistension with significantly thickened walls and small projections of the false diverticula. The picture was compatible with stress bladder, therefore the patient was referred to the present urogynaecological department.

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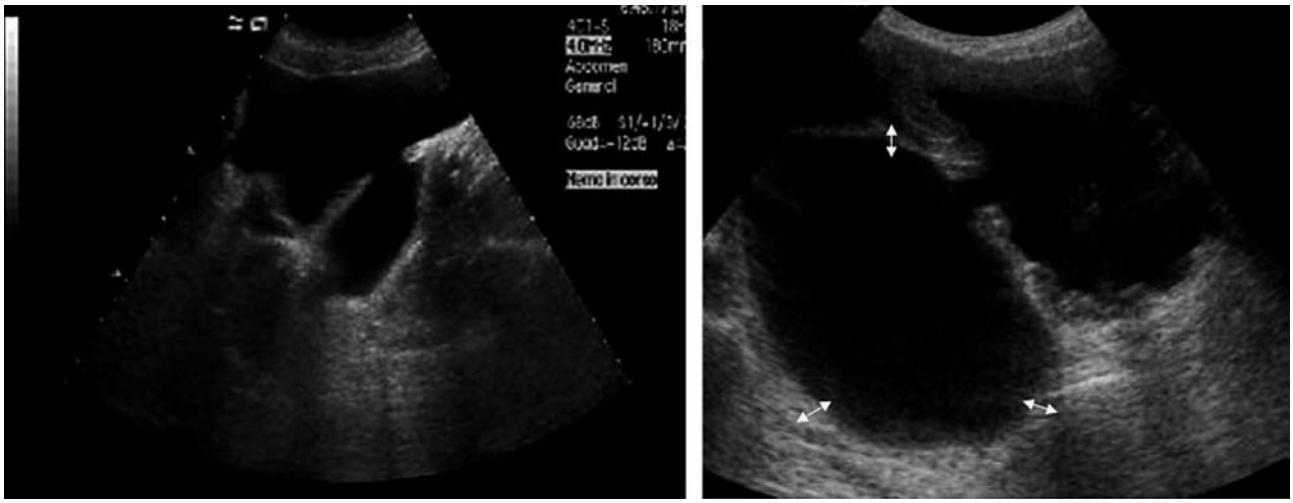

Figure 1. — Ultrasound scan of the bladder diverticulum

The clinical assessment showed: soft vagina seven cm in length, no prolapse, no urethral hypermobility or pudendal neuropathy, good and preserved exteroceptive and proprioceptive sensitivity. The bladder diary over three days was within the physiological ranges (daytime urinary frequency: eight to nine times; night time frequency: once; voided volumes: 250 +/- 30 cc; daily fluid intake: 1,500 cc; no signs of urge or stress incontinence, nor urinary disorders reported). The uroflowmetry showed a continuous and slow flow with a maximum quantity of nine ml/sec (maximum flow), voiding time of 63 seconds, and voided volume (VV) of 300 ml. An ultrasound examination showed a large bladder diverticulum (Figure 1) [8, 9], compatible with a significant PVR: an extemporaneous catheterization with a Nelaton 8 catheter showed a residual volume of 1,200 cc. At cystoscopy, the insertion of the cystoscope was difficult due to a proximal urethral stenosis, requiring dilation of the urethra; the examination confirmed the presence of a large bladder diverticulum in the anterior wall with no endoluminal neoplasms and displaying a regular wall structure. Finally, an urodynamic assessment confirmed a continuous and slow flow as in case of urethral obstruction [10-13].

The prevalence of bladder/urethral outlet obstruction in women is unknown and has probably been underestimated. Moreover, no standard definitions are available for the diagnosis of bladder outlet obstruction in women. Usually, bladder outlet obstruction is defined as a persistent, low, maximum "free" flow rate of <12 ml/s in repeated non-invasive uroflow examinations, combined with high detrusor pressure at a maximum flow (pdet.Qmax > 20 cm H2O) during detrusor pressure–uroflow examinations. In conclusion, in the present case report the diagnosis was bladder diverticulum associated with obstructive urinary difficulties due to urethral stenosis.

As the etiopathogenesis of hypercreatininaemia was clear, self-catheterization twice a day was prescribed, as well as fosfomycin three grams every ten days for six months, voiding on a time schedule, and adequate fluid intake. Moreover, the patient was asked to fill in a precise bladder diary, to perform urine culture, and creatininemia every two weeks, to undergo ultrasonographic examination. The monthly scheduled follow-up at one year showed good general health, good compliance with the therapy, no urinary tract infections, a decrease in creatininemia to 1.2 mg/dl, negative urinalysis, and regression of nephrohydrosis to a mild stage. The scheduled pharmacological and life-style intervention plan will continue. Urethral dilatations will be performed if during follow-up urethral stenosis worsens or obstructive symptoms arise; diverticulectomy will be performed if PVR increases or becomes uncontrollable by self-catheterizations, and/or cause worsening bilateral nephrohydrosis.

Discussion

Bladder diverticula represent a herniation of the bladder urothelium through the muscularis propria of the bladder wall, resulting in the typical finding of a variably sized, thin-walled, urine-filled structure adjacent to and connecting with the bladder lumen through a narrow neck, or ostium. Bladder diverticula may be classified as either congenital or acquired, with different pathophysiology, presentation, and imaging. Congenital diverticula usually present during childhood, with a peak incidence in those less than ten years old, are usually solitary, occur most commonly in males, and are located lateral and posterior to the ureteral orifice, often in association with vesicoureteral reflux [14, 15]. The primary causation in those without coexisting lower urinary tract conditions appears to be a congenital weakness at the level of the ureterovesical junction and not bladder outlet obstruction. Congenital bladder diverticula have been noted in association with congenital connective tissue disorders (such as Menkes syndrome, Williams syndrome, Ehlers-Danlos syndrome); whether there is a genetic predisposition to the formation of bladder diverticula in individuals without congenital syndromes is unclear [16].

Acquired diverticula are often multiple, located most commonly at the ureterovesical hiatus but also occur elsewhere in the bladder, and associated with bladder outlet obstruction (anatomical or functional), infection, and

iatrogenic causes. Bladder diverticula in females are uncommon and quite rare in the absence of obstruction [16]. Bladder diverticula may also be iatrogenic: inadequate closure of the muscular layers of the bladder wall following a cystotomy for any indication may result in formation of a bladder diverticulum at a weak point of the suture line. In the present case report, the patient had undergone abdominal hysterectomy. A potential relationship between bladder diverticulum and hysterectomy has been suggested since bladder injury can occur during hysterectomy and diverticula may develop on sites of occult bladder wall injury; on the other hand, urethral stenoses can appear as a result of micro-traumas leading to phlogistic processes without obstructive symptoms. Unfortunately, in the present case it is not clear whether the obstructive urinary disorders were present before the first surgery but not well detected, or if they appeared later. Indeed, the patient never reported hyperthermia associated with dysuria and positive urinalysis or urine culture.

The most common symptom is urinary tract infection, which occurs as a result of urinary stasis in the diverticula due to inadequate quantity of muscle on the diverticular wall. A narrow pedicle of the diverticulum is another factor affecting urinary stasis. Additionally, hematuria and vesicoureteral reflux are common clinical presentations. Less commonly, urinary retention (bladder outlet obstruction), intestinal obstruction, and ureteral obstruction may occur due to direct diverticular compression; finally, ureteral obstruction may develop due to the inflammation secondary to diverticulitis. The relevant feature of the present case report is the complete absence of symptomatology despite the significant volumes of PV), and the negative results at urinalysis and urine-culture despite the relevant urine stagnation in the diverticulum. Such clinical data led to a conservative treatment including pharmacological and life-style intervention: self-catheterization twice a day, fosfomycin three grams every ten days for six months, voiding on a time schedule, adequate fluid intake, monitoring the kidney function as far as nephrohydrosis, creatininemia, and urinary tract infections. Incidentally found congenital or acquired bladder diverticula, even large, may require no further therapy unless associated with persistent symptoms, recurrent infections, obstruction, stones, malignancy, or other complicating factors such as ipsilateral vesicoureteral reflux. Indications for surgical interventions are worsening urethral stenosis or arising obstructive symptoms (urethral dilatations), increased /uncontrollable PVR or worsening bilateral nephrohydrosis (diverticulectomy). In conclusion the absence of symptoms and negative urinalysis or urine culture allows expectant management despite the considerable size of the bladder diverticulum.

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