Introduction

Inguinal hernia and Stress Urinary Incontinence (SUI) are well-known complications of radical prostatectomy [1,2]. However, SUI development following inguinal herniorrhaphy is extremely rare. We report the case of a patient who developed SUI following inguinal herniorrhaphy after open radical prostatectomy. To the best of our knowledge, this is the first reported case of SUI development following inguinal herniorrhaphy in a man who had undergone radical prostatectomy.

Case Presentation

A 66-year-old man was diagnosed with localized prostate cancer cT2cN0M0. His past medical history was unremarkable. He underwent open radical prostatectomy in 2008 after a 9-month neoadjuvant hormone therapy. Following the prostatectomy, he developed mild SUI that gradually improved. Thereafter, he did not need a pad after 15 months of prostatectomy. Four years later, he underwent salvage radiation therapy (66 Gy) for biochemical recurrence. Subsequently, the prostate-specific antigen serum levels decreased from 0.64 ng/mL to 0.1 ng/mL. After radiation, he did not experience the difficulty in voiding and his Qmax was 21.0 ml/second and urinary incontinence did not develop. During the follow-up period, he developed left inguinal hernia, for which he underwent synthetic mesh repair (inguinal herniorrhaphy) in 2014. After removal of the urethral catheter 12 French in size, he developed severe SUI. He then needed more than three pads a day for his total incontinence while standing. Cystourethrography and cystourethroscopy detected only a mild urethral stricture, and a urodynamic study showed no detrusor overactivity. Conservative therapies along with pelvic exercise and use of several agents, such as anticholinergics and β2-stimulants, were ineffective in treating SUI. Two year later, an artificial urinary sphincter implantation was performed, and SUI disappeared. Patient’s clinical course is shown in Figure 1.

Discussion

The patient had moderate SUI for a few months after prostatectomy. Thereafter, SUI improved gradually, and he rarely needed pads. SUI recurred after inguinal herniorrhaphy. Imaging as well as endoscopic and urodynamic studies were negative for urinary incontinence. The underlying mechanisms for SUI recurrence after inguinal herniorrhaphy were unclear. Radiation to the prostatic fossa might have slightly affected the urethral sphincter; however, this was not the main cause for the development of SUI because he did not develop SUI after radiation therapy. It may be suspected that this patient with post-operative sphincter damage had developed urethral stenosis according to radical prostatectomy and/or post-operative radiation therapy which contributed
to improvement of urinary incontinence. However, placement of urethral catheter during surgery for inguinal hernia dilated the stenosis, and after removal manifested stress urinary incontinence which had been once hidden by the urethral stenosis. This story is, however, unlikely, because of the following reasons: 1) his SUI after prostatectomy disappeared 15 months surgery and he did not need a pad even after radiation, 2) he did not have the difficulty of voiding suggesting urethral stricture that could mask SUI, 3) it is hard to dilate urethral stricture with 12 French urethral catheter. Thus, we believe that herniorrhaphy was the main cause of SUI.

Abdominal pressure to the bladder might have been reduced by the dilated inguinal canal before inguinal hernia repair. However, after the herniorrhaphy, the abdominal pressure to the inguinal canal was blocked, resulting in increased abdominal pressure to the bladder; this in turn might be the main cause of SUI recurrence after the herniorrhaphy (Figure 2). To the best of our knowledge, this is the first reported case of SUI development following inguinal herniorrhaphy in a man who had undergone radical prostatectomy. Although rare in occurrence, we need to be aware of SUI after hernia repair in patients with a weak or damaged urethral sphincter.

References
