Erectile incontinence post radical prostatectomy

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A patient who underwent a nerve-sparing radical retropubic prostatectomy, who was post-operatively continent, developed urinary incontinence associated with erectile recovery. The bladder was stable and minimal distortion of the bladder neck was observable on videourodynamics. We suggest that the mechanism of incontinence is urethral traction, induced by an erect penis causing an anatomically minor, but functionally significant opening of the bladder neck/sphincter mechanism. This case highlights the importance of a stable sphincter mechanism in maintaining continence. It also emphasizes the fine line between success and failure in post radical retropubic prostatectomy continence; and is a reminder of the importance of surgical technique in achieving the goal of a cancer free, potent, continent patient.

CASE HISTORY

We report on a case of urinary incontinence associated with erectile recovery in an otherwise continent man post radical retropubic prostatectomy.

The patient (aged 76) underwent a nerve-sparing radical retropubic prostatectomy for clinically localized prostate cancer. He made an excellent post-operative recovery and regained full continence within 3 months. He suffered from impotence post-operatively, which was only partially responsive to PDE₅ inhibitor treatment. Subsequently, he was treated with intracavernosal alprostadil, with good effect. However, he experienced urinary leakage with erections, which occurred despite emptying his bladder pre-injection.

The patient underwent video-urodynamic examination prior to, and subsequently with, an induced erection. This confirmed that the bladder was stable, completely emptied, and that the patient was continent prior to having an induced erection (see Figure 1a). Alprostadil induced a moderate erection at which point significant urinary leakage was observed (see Figure 1b). Slight lengthening of the membranous urethra was observed at the time of erection,

Figure 1 (a) Videourodynamic picture demonstrating a stable bladder neck and continence with a flaccid penis. (b) Videourodynamic photograph demonstrating marked urinary incontinence associated with an erect penis in the same patient

however the bladder neck distortion induced by the erection was minimal with no observable descent. It was not felt that the incontinence could be corrected surgically and the patient is contemplating stopping erectile dysfunction treatment.

In our, single surgeon, series of 186 cases our pad-free continence rate is 96%. It is our preferred technique to leave a long intra-pelvic urethral stump prior to urethral

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division and the dorsal venous complex is divided over a wire and subsequently oversewn to avoid bunching of the complex and distortion of the sphincter. The bladder neck is carefully reconstituted with eversion of the bladder mucosa. We suggest that erectile incontinence occurs when the penis is erect due to traction being exerted on the urethra, which transmits to the vesico-urethral anastomosis, causing an anatomically minor, but functionally significant opening of the external sphincter. This is sufficient to cause urinary incontinence. This report supports the advocacy of stabilizing the sphincter by suturing it to the rear of the pubis symphysis.

A previous study¹ demonstrated that stretched penile length was reduced in 68% of patients following retropubic prostatectomy; and in 19%, this amounted to a shortening of at least 15%. Post-operative cavernosal fibrosis was suggested as a possible cause, as there was no correlation with prostate size, operative technique or potency. One possibility not suggested was that the shortening was due to urethral traction. Other factors, which have been shown to be associated with increased incontinence post radical retropubic prostatectomy are large prostates, and a membranous urethral length less than an arbitrary 12 mm, measured from pre-operative endo-rectal magnetic resonance imaging scans.² A large prostate would imply a greater distance for the bladder to be mobilized for the bladder-urethra anastomosis and possibly greater posterior traction. Similarly, a short membranous urethra implies that the posterior traction from the bladder is transmitted more directly to the sphincter and consequently may impair continence.

These studies demonstrate the fine line between success and failure in maintaining urinary continence post radical retropubic prostatectomy. Incontinence post radical retropubic prostatectomy occurs in up to 20% patients, and persists after a year in up to 10%. Impotence is reported in up to 80%. Groutz³ found the predominant cause of post radical retropubic prostatectomy incontinence was intrinsic sphincter deficiency. This was present in 88% of patients with persistent incontinence post radical retropubic prostatectomy, but occurred in isolation in only one-third. Detrusor instability, detrusor failure and reduced urethral compliance, consistent with urethral scarring, were all identified as concomitant pathologies although rarely in isolation. Three operative strategies have been suggested to maintain continence post radical retropubic prostatectomy. The first is bladder neck preservation, whereby the circular bladder neck fibres are preserved⁴. The second is preservation of the puboprostatic ligaments to maintain anterior urethra support and minimize trauma to the striated sphincter⁵. The third is neurovascular nerve preservation. All of these have been shown to improve the time to continence, but no statistical difference has been

identified in long-term continence, either individually or in combination.⁶

We believe that this case is the first reported case of incontinence being induced by erectile recovery post radical prostatectomy.

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REFERENCES

- 1 Savoie M, Kim SS, Soloway MS. A prospective study measuring penile length in men treated with radical prostatectomy for prostate cancer. *J Urol* 2003;**169**:1462-4
- 2 Coakley FV, Eberhardt S, Kattan MW, *et al.* Urinary continence after radical retropubic prostatectomy: relationship with membranous urethral length on preoperative endorectal magnetic resonance imaging. *J Urol* 2002;**168**:1032-5
- 3 Groutz A, Blaivas JG, Chaikin DC, *et al.* The pathophysiology of post radical prostatectomy incontinence: a clinical and video urodynamic study. *J Urol* 2000;**163**:1767-70
- 4 Solway MS, Neulander E. Bladder-neck preservation during radical retropubic prostatectomy. *Semin Urol Oncol* 2000;**18**:51-6
- 5 Poore RE, McCullough DL, Jarrow JP. Puboprostatic ligament sparing improves urinary continence after radical prostatectomy. *Urol* 1998;**51**:67-72
- 6 Delveliotis C, Protogerou V, Alargof E, Varkarakis J. Radical prostatectomy: bladder neck preservation and puboprostatic ligament sparing-effects on continence and positive margins. *Urol* 2002;**60**:855-8

Simultaneous presentation of myasthenia gravis and mesothelioma

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Myasthenia gravis may be a paraneoplastic phenomenon, caused by underlying cancer.¹ This case describes the first co-presentation of myasthenia gravis and pleural mesothelioma. If this is a paraneoplastic occurrence, then with the expected increase in incidence of mesothelioma,² further cases should arise.

CASE HISTORY

A man aged 71 years, and previously in good health, presented to the acute medical take with difficulty chewing and swallowing. He gave a 6-week history of variable left

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Figure 1 Computerized tomograph of thorax, pleural mass indicated

Figure 2 Thorax computerized tomograph showing pleural mass

upper eyelid drooping, slurred and nasal speech and difficulty with chewing and swallowing. Symptoms were most prominent in the evenings and it took him over an hour to eat his evening meal. There was no double vision or shortness of breath. Upper and lower limbs were unaffected. He had lost 3.5 kg in weight over the preceding month. His past medical history was unremarkable, and his only medication was a statin for hypercholesterolaemia. He was a life-long non-smoker and a retired brick-layer and car factory worker.

On examination, he was well. He was not cachectic or clubbed. Respiratory and cardiovascular examinations were normal. He had a pronounced nasal dysathria that fatigued. There was a subtle left ptosis that was not clearly fatiguable on upward gaze. Eye movements were normal and there was no diplopia. There was bilateral facial weakness with incomplete eyelid closure and a very poor lip seal whereby he could not whistle or blow his cheeks out; the forehead was spared. Palatal elevation was mildly affected. The tongue was normal. The limbs were of normal tone and

power, both proximally and distally: there was no fatigability. All reflexes were present and showed no post-tetanic augmentation. His vital capacity was 2.5 L. A chest X-ray demonstrated multiple right-sided pleural masses and a small pleural effusion.

Clinically, the diagnosis was myasthenia gravis with a predominant bulbar presentation. This was confirmed by the presence of anti-acetylcholine receptor antibodies, and supportive neurophysiological findings. There were no anti-voltage gated calcium channel antibodies or anti-MuSK antibodies detected. He responded well to anti-cholinesterase and steroid treatment.

Computerized tomography thorax confirmed the presence of pleural masses (Figures 1 and 2). There were no thymic tissues seen within the anterior mediastinum. One of the masses was biopsied and a histological diagnosis of sarcomatoid mesothelioma was made. There was no evidence of malignancy elsewhere.

COMMENT

Both mesothelioma and myasthenia gravis are rare diseases. In the UK, the prevalence of mesothelioma is 8/100 000 in men and 1/100 000 in women² and the prevalence of myasthenia gravis 15/100 000.³ Their dual diagnosis is unlikely, and this is the first reported case.

Paraneoplastic syndromes result from non-local effects of cancer and in 80% of cases they are diagnosed before the cancer.¹ Myasthenia gravis is the most common paraneoplastic neurological disease and 10% of cases are paraneoplastic.⁴ The classical association is that of thymoma and myasthenia gravis, where 30% of thymomas cause myasthenia gravis.⁴

There is one reported case of paraneoplastic cerebellar degeneration in a patient with known mesothelioma.¹ However, this is the first case report of the co-presentation of myasthenia gravis and pleural mesothelioma. If the association is more than chance, then with the expected increase in incidence of mesothelioma² further cases should be described.

REFERENCES

- 1 Candler PM, Hart PE, Barnett M, Weil R, Rees JH. A follow up study of patients with paraneoplastic neurological disease in the United Kingdom. *J Neurol Neurosurg Psychiatry* 2004;**75**:1411–15
- 2 Treasure T, Sedrakyan A. Pleural mesothelioma: little evidence, still time to do trials. *Lancet* 2004;**364**:1183–5
- 3 Robertson NP, Deans J, Compston DAS. Myasthenia gravis: a population based epidemiological study in Cambridgeshire, England. *J Neurol Neurosurg Psychiatry* 1998;**65**:492–6
- 4 Posner JB. Immunology of paraneoplastic syndromes. *Ann. N. Y. Sci.* 2003;**998**:178–86