

An unusual cause of acute kidney injury

Introduction

Acute kidney injury is a global health problem of diverse aetiology and a leading cause of inpatient hospital mortality (Singbartl and Kellum, 2012). The prevalence of acute kidney injury is estimated to be 23.4% and up to 100 000 deaths in secondary care are associated with acute kidney injury (Wang et al, 2012).

This article reports an unusual cause of acute kidney injury in a 70-year-old man who presented with increasing confusion secondary to syndrome of inappropriate ADH secretion-related hyponatraemia (SIADH).

Discussion

An artificial urinary sphincter is widely regarded as the gold standard for the treatment of post-prostatectomy urinary incontinence, the incidence of which is estimated to be 8.4% after prostatectomy (Ricci et al, 2008). The artificial urinary sphincter was first introduced in 1973 and over the years there have been several design changes resulting in different models of the device (Vakalopoulos et al, 2012). The most common complications of the artificial urinary sphincter include urinary incontinence as a result of malfunctioning or failure of the sphincter, infection, urethral atrophy and mechanical failure (Lai et al,

Table 1. Baseline investigations

Test	Result	Reference value
Serum sodium	123 mmol/litre	132–146 mmol/litre
Serum potassium	4.4 mmol/litre	3.5–5.0 mmol/litre
Urea	3.6 mmol/litre	2.5–6.7 mmol/litre
Creatinine	61 µmol/litre	45–104 µmol/litre
Thyroxine	21 pmol/litre	10–22 pmol/litre
Thyroid-stimulating hormone	0.80 mU/litre	0.30–5.5 mU/litre
Cortisol	757 nmol/litre	>200 nmol/litre
Serum osmolality	255 mmol/kg	280–300 mmol/kg
Urine osmolality	451 mmol/kg	300–1000 mmol/kg
Urine sodium	71 mmol/litre	

2007). Age is not a contraindication to the use of the artificial urinary sphincter provided the patient is cognitively intact and can use a scrotal deactivation mechanism to operate the device.

This patient developed acute kidney injury secondary to obstructive uropathy as he was not able to deactivate his artificial urinary

sphincter because of his acute confusion. The fluid restriction was not thought to have contributed to the renal failure as the patient remained euvolaemic. There are no previous case reports of acute kidney injury resulting from failure of artificial urinary sphincter deactivation secondary to an acute confusional state. With increasing use of

CASE REPORT

A 70-year-old man was referred to hospital with a 2-week history of increasing confusion and agitation. His only past medical history was a radical prostatectomy for localized prostate cancer 15 years ago. He was not taking any regular medications. On examination his Abbreviated Mental Test Score (AMTS) was 9/10, blood pressure was 160/96 mmHg and the rest of the examination was unremarkable.

Baseline investigations (*Table 1*) revealed hyponatraemia and paired osmolalities were suggestive of syndrome of inappropriate ADH secretion (SIADH). He was placed onto 1 litre fluid restriction.

On day three of admission his creatinine level jumped from 75 to 410 µmol/litre. Owing to his ongoing confusion it was difficult to ascertain his urine output. On questioning, he reported it was 'impossible to urinate' for him as 'it was just too difficult'. On examination the

patient was euvolaemic and a bladder scan demonstrated 1100 ml residual volume. A per rectal examination was normal but a small prosthetic structure in the patient's scrotum was detected which had not been documented in the case notes. It transpired that following his radical prostatectomy he had had an iatrogenic bulbar artificial prostatic sphincter placed, which neither the patient nor his family or GP records had reported. The artificial prostatic sphincter was released by means of a scrotal deactivation mechanism, allowing free flow of 1100 ml residual urine with rapid improvement in kidney function (*Figure 1*).

With concerns about his ability to manage the artificial prostatic sphincter, he had a temporary suprapubic catheter placed until his mental health improved enough for him to be able to self-manage his artificial prostatic sphincter again.

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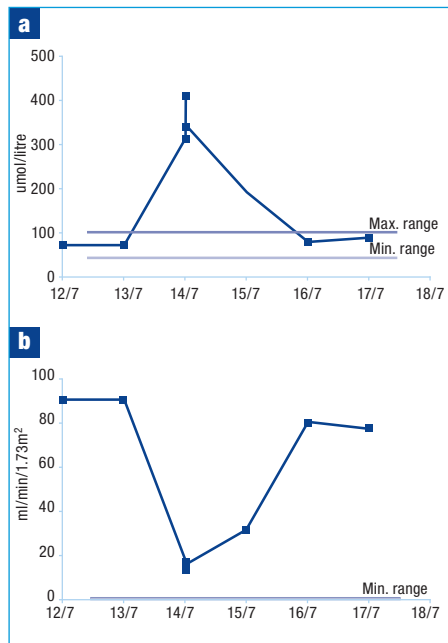
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artificial urinary sphincters in the elderly population this must be considered while establishing the underlying aetiology of acute kidney injury and ruling out common causes.

Figure 1. a. Serum creatinine level and (b) estimated glomerular filtration rate during the episode of acute kidney injury.



Conclusions

Failure to deactivate an artificial urinary sphincter leading to urinary retention may be an easily overlooked cause of acute kidney injury. With growing use of artificial urinary sphincters in the elderly population this must be considered. Awareness of the artificial urinary sphincter is needed as it can cause urinary obstruction and acute renal dysfunction. A simple click deactivates the device and allows free flow of the urine. **BJHM**

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LEARNING POINTS

- Failure to deactivate an artificial urinary sphincter leading to urinary retention may be an easily overlooked cause of acute kidney injury.
- With the growing use of artificial urinary sphincters in the elderly this must be considered in the differential diagnosis of acute kidney injury, especially when these patients present with acute confusion, unconsciousness or limb weakness secondary to acute stroke.
- A high degree of awareness about artificial urinary sphincters is necessary because they can cause urinary obstruction and acute renal dysfunction.
- Management requires a simple click to deactivate the device allowing free flow of urine.
- The authors recommend that patients should carry a wallet card or wear medical identification to tell health-care providers that they have an artificial sphincter. The presence of these prostheses and interventions must be documented in the clinical notes.

Images in Medicine

Lingual tumour in a 6-year-old boy

A 6-year-old patient, who was undergoing chemotherapy for lymphoblastic leukaemia, presented with a 1-month history of an asymptomatic, 3 cm x 2 cm well-demarcated whitish nodule on the posterior portion of the tongue (*Figure 1*). Histopathological examination confirmed the diagnosis of chronic hyperplastic candidiasis of the tongue. The lesion disappeared after antifungal therapy.

Oral candidiasis is the most frequent mucocutaneous mycosis of the oral cavity. It is found in the oral cavity of 50% of the general population as a common commensal organism (Farah et al, 2010). The pathogenesis of candidiasis depends on several factors, of which immunosuppression and poor oral hygiene are the most common. Candidiasis is often present in its pseudomembranous form; the tumour form is unusual and can be misleading. In an immunocompromised

patient, the acute appearance of a tongue tumour should be treated with antifungal therapy followed by skin biopsy. **BJHM**

Farah CS, Lynch N, McCullough MJ (2010) Oral fungal infections: an update for the general practitioner. *Aust Dent J* **55**(Suppl 1): 48–54

Figure 1. A rough-surfaced 3 cm x 2 cm whitish nodule on the posterior portion of the tongue.



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