Case Report

Urinary incontinence associated with sabulous urolithiasis: a series of 4 cases

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Introduction

The accumulation of large amounts of sand-like sediment in the urinary bladder (sabulous urolithiasis) is a potential consequence of urinary incontinence in horses resulting from compromised bladder emptying (Holt 1997). The following report describes 4 cases of this syndrome. Two cases had detectable detrusor dysfunction shortly following the onset of urinary incontinence and one an incomplete history whereby the interval between onset of urinary incontinence and development of detrusor dysfunction was unknown. In contrast, the fourth case presented with severe cystitis 2 years following the onset of urinary incontinence and 5 years following dytocia, with detrusor dysfunction and sabulous urolithiasis developing as an end-stage problem.

In this article we describe the investigation and treatment of three cases of sabulous urolithiasis and the potential course of events in Case 4.

Case details

Case 1

A 17-year-old gelding presented with a 5-month history of urinary incontinence. The owner first noticed dribbling of urine during ridden work. Subsequently, incontinence had progressed to persistence throughout the day, with urine dripping both at rest and during motion. The owner had not observed normal micturition since initial presentation and antibiotic and anti-inflammatory medication administered by the referring veterinary surgeon failed to result in resolution of clinical signs.

Clinical examination revealed severe urine scalding on the preputial region and dorsal aspects of the hindlimbs. Neurological examination, including assessment of perineal sensation and anal tone, was unremarkable and all clinical parameters were within normal limits.

Rectal examination revealed a distended bladder extending cranially into the abdominal cavity, from which urine was easily expressed by manual pressure. Transrectal ultrasonographic examination using a 5 MHz linear probe confirmed distension of the bladder and revealed hyperechoic particles within the urine and a markedly hyperechoic line extending from caudodorsal to cranioventral within the bladder (Fig 1).

Evacuation of urine was accomplished via a 150 cm 28 Fr. catheter1, following which transrectal examination revealed a flaccid and atonic bladder. In addition, a large doughy mass was palpated within the cranial aspect of the bladder. A urine sample was collected for routine analysis (Table 1), bacterial culture and antibiotic sensitivity. Culture of the sample yielded a mixed growth with no predominant organisms.

Cystoscopic examination, performed following passage of a 1 m, 8 mm diameter human gastroscope2 via the penile urethra, revealed mild mucosal inflammation of the cranioventral...
### Post mortem examination

All abnormal findings were restricted to the urinary tract. A spherical 20 cm diameter ‘charky’ mass and approximately 100 ml sabulous material were found within the bladder (Fig 3). The bladder mucosa was thickened and haemorrhagic with mild thickening of both ureters but no gross change on their mucosal surface. Both kidneys were grossly normal.

Culture of urine collected post mortem revealed a mixed growth with a marked predominance of *Proteus* spp. Histopathological examination of the bladder wall revealed a thickened epithelium with a marked neutrophilic infiltration, congestion of the *lamina propria* and areas of fibrosis and dystrophic calcification. These findings were consistent with an active severe cystitis.

### Case 2

A 6-year-old Irish Draught gelding was presented as a cruelty case to the referring veterinary surgeon for evaluation of weight loss, quidding and urinary incontinence. The history prior to examination was unknown.

Clinical examination revealed a full bladder, easily expressed by the application of manual pressure during transrectal examination. Ancillary diagnostic tests had revealed a mild leucopenia and neutropenia, 1300 strongyle eggs/g faeces and a moderate growth of *Staphylococcus intermedius* on urine culture. No clinical improvement was noted following completion of a 5-day course of antibiotics and the horse was subsequently referred for further evaluation.

On presentation at the referral hospital, the horse was emaciated (condition score 1/5; Carroll and Huntington 1988), had a bilateral nasal discharge and was noted to persistently dribble urine during walking, resulting in urine scalding of skin over the medial hindlimbs. No abnormalities were noted during a routine neurological examination, including assessment of perineal sensation and anal tone.

Haematology, biochemistry and faecal analysis were unremarkable. Urine analysis was performed on urine collected both by free catch and catheterisation (Table 1).

Rectal examination and cystoscopy were performed as described previously. A large distended bladder was palpated *per rectum*, extending into the abdominal cavity. Cystourethroscopy revealed inflammation and focal areas of

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**Table 1: Urine analysis in 4 cases of urinary incontinence**

<table>
<thead>
<tr>
<th>Method</th>
<th>Case 1 (Catheter)</th>
<th>Case 2 (Free catch)</th>
<th>Case 3 (Catheter)</th>
<th>Case 4 (Free catch)</th>
<th>Case 4 (Catheter)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific gravity</td>
<td>1.028</td>
<td>1.030</td>
<td>1.022</td>
<td>1.025</td>
<td>1.020</td>
</tr>
<tr>
<td>PH</td>
<td>8.4</td>
<td>8.0</td>
<td>8.0</td>
<td>9.3</td>
<td>8.0</td>
</tr>
<tr>
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<td>-</td>
<td>Trace</td>
<td>+</td>
<td>Trace</td>
<td>+++</td>
</tr>
<tr>
<td>Protein</td>
<td>-</td>
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<td>+</td>
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<td>Bilirubin</td>
<td>-</td>
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<tr>
<td>Glucose</td>
<td>-</td>
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<td>-</td>
<td>-</td>
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</tr>
<tr>
<td>Acetone</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Comments</td>
<td>Occasional red, white and epithelial cells</td>
<td>Few white blood cells</td>
<td>Numerous white blood cells</td>
<td>Numerous white blood cells, occasional red cells and epithelial cells</td>
<td>Few red blood cells, leucocytes and bacteria</td>
</tr>
</tbody>
</table>

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**Fig 2:** Cystoscopic view of the yellow/white mass on the bladder floor of Case 1.

**Fig 3:** Gross post mortem view of bladder from Case 1, showing large ‘charky’ mass (M) and sabulous material (S).
granulation tissue at the bladder entrance (Fig 4) and a large volume of sabulous material within the bladder. Clear urine was noted to periodically flow through both ureteral openings, which appeared grossly normal.

Based on the clinical findings, a diagnosis of detrusor dysfunction was made. The welfare charity responsible for the case was notified of the poor prognosis for return of bladder function, and elected immediate euthanasia.

**Post mortem examination**

Diffuse marked thickening and reddening of the bladder mucosa and focal irregular areas of ulceration on the dorsal mucosal surface (0.5–5.0 cm in diameter) were present. Other regions of the urinary tract were grossly normal. Histopathological examination of the bladder wall confirmed the presence of mucosal ulceration with replacement by a thick layer of granulation tissue containing neutrophils. These findings were consistent with a severe, diffuse and chronic ulcerative cystitis. Attempts to identify the caudal mesenteric ganglion and pelvic plexus for histopathology were unsuccessful.

**Case 3**

A 5-year-old Thoroughbred gelding presented with a 2-month history of urinary incontinence. Extensive work-up by the referring veterinary surgeon had failed to find a cause for the incontinence, and the horse had failed to respond to antibiotic and corticosteroid therapy combined with placement of an indwelling urinary catheter.

Following referral, abnormal clinical findings were confined to the urinary tract. No abnormalities were noted during a routine neurological examination, including assessment of perineal sensation and anal tone. Urine was noted to frequently dribble from the penis at rest, and dried urine crystals were evident on the medial aspects of the hindlimbs. Transrectal palpation revealed a markedly distended bladder from which small amounts of urine could easily be expressed. Ultrasonography of the bladder was unremarkable and cystoscopic examination revealed an erythematous bladder mucosa and accumulation of sandy material within the cranioventral aspect. Clear urine was passed intermittently from grossly normal ureter openings. No bacteria were cultured from the urine and results of urine analysis were normal.

Despite the poor prognosis for clinical remission, the owner elected to attempt therapy. Prednisolone (1mg/kg bwt per os) and trimethoprim-potentiated sulphonamide (12.5 g sulphadiazine and 2.5 g trimethoprim b.i.d. per os) treatment was initiated. Following 4 days of unsuccessful management, the sympathomimetic drug bethanechol chloride (Myotonine) was added to the regime, initially at 0.2 mg/kg bwt t.i.d. per os. No clinical response was noted following 3 days of treatment and consequently the bethanechol dose was increased to 0.4 mg/kg bwt q.i.d., combined with drainage of urine from the bladder on 2 occasions. The latter procedure resulted in temporary improvement on both occasions, presumably by decreasing the potential for overflow. Following a poor response to 11 days of therapy, the owner elected for euthanasia.
Post mortem examination revealed a congested and thickened bladder mucosa with a semisolid mass of sabulous material towards the apex. Histology revealed a heavy infiltrate of neutrophils and lymphocytes into the bladder submucosa. Gross and histological examination of the sacral spinal cord was unremarkable.

**Case 4**

A 21-year-old Dales pony mare was referred for investigation of intermittent urinary incontinence and pollakiuria of 2 years, duration. The mare was multiparous and had last foaled 5 years previously. No infertility problems had been noted during her breeding history; however, the last parturition had required assistance. The incontinence resolved during the late winter months with intermittent and variably severe episodes over the summer months. Normal urination was maintained during these episodes but was followed by dribbling of urine. Spontaneous urine dribbling was also noted, unassociated with normal micturition. The owner perceived the incontinence to be more evident during oestrus. There had been no weight loss noted since the onset of clinical signs. Several courses of antibiotic therapy administered by the referring veterinary surgeon had resulted in only temporary resolution.

Clinical examination failed to reveal any abnormalities with the exception of urine scaling on the skin of the proximal hindlimbs. Transrectal ultrasonographic examination of the bladder revealed moderate distension and a reduction in bladder wall thickness compared with the previous examination (Fig 7). A reduction in the severity of mucosal inflammation was evident during both vaginoscopic and cystoscopic examinations; however, the mucosal surface of the bladder contained many irregular contours.

Therapy was continued for a further 3 weeks, following which the owner reported the continued presence of urinary incontinence. Upon consideration of the apparent resolution of incontinence throughout the winter, and the perceived increased severity of clinical signs during oestrus, hormonal therapy was initiated with the synthetic progestagen altrenogest (Regumate)3 (0.044 mg/kg bwt s.i.d., per os), which resulted in an increase in severity of incontinence. Subsequent oestrogen therapy (Oestradiol benzoate)4 (0.01 mg/kg bwt i.m. s.i.d. for 4 days, then every other day) partially reduced the severity of clinical signs, but failed to result in complete resolution.

The findings of a further clinical examination 3 months later were similar to those previously noted, with the exception of the detection of a markedly enlarged bladder during transrectal palpation. Ultrasonographic and cystoscopic examinations were repeated and revealed the presence of a large amount of sabulous material within the bladder. Clear urine was observed to intermittently exit each ureteral opening, both of which appeared less inflamed than previous examinations. The owner was informed of the poor prognosis for recovery. Two months following the third examination, the owners noted that the mare was having difficulty rising, and elected for immediate euthanasia. Unfortunately, a post mortem examination was not possible in this case.

**Discussion**

This report describes 4 cases of urinary incontinence ultimately associated with the development of sabulous urolithiasis, characterised by the build-up of large quantities of sabulous material within an enlarged atronic bladder (Holt and Pearson 1984; Holt and Mair 1990). Despite the fact that sabulous urolithiasis is commonly associated with urinary incontinence in the horse, there is a scarcity of information on its pathogenesis in the literature.

The cases presented included 3 geldings and one mare, aged 5–21 years. These findings support the reported absence of any apparent age predisposition for sabulous urolithiasis in horses. Other reports have similarly shown urinary incontinence associated with this syndrome to be more common in males, particularly geldings (Holt and Pearson 1984; Holt and Mair 1990; Schott and Carr 2003).

Previous history can provide vital clues to aetiology in incontinence cases (Schott and Carr 2003). Unfortunately, Case 2 had a minimal history as a result of its presentation as a cruelty case. The history of Cases 1 and 3 were unremarkable prior to presentation with no indication of prior trauma, lameness, or respiratory problems which may have supported

**Re-evaluation**

During the intervening period, the degree of incontinence varied with no obvious pattern. Re-examination revealed
an underlying neurological deficit. The history of complications during parturition in Case 4 is interesting considering that dystocia has been reported as a possible cause of neurological damage leading to urinary incontinence (Pringle and Schott 1997; Schott and Carr 2003; Sponseller 2003). Indeed, a report of 15 cases of bladder paralysis (Carr 2002) included 4 that developed post foaling; however, the authors did not indicate the time period between parturition and onset of incontinence. Although the period of 3 years between dystocia and onset of urinary incontinence in Case 4 may be considered excessive, it remains possible that trauma at the time of parturition may ultimately have resulted in urethral sphincter dysfunction (Pringle and Schott 1997). This may consequently have resulted in both urinary incontinence and a predisposition to cystitis. Alternatively, the onset of urinary incontinence may have resulted from a primary cystitis, the ultimate severity and chronicity of which may have resulted directly in detrusor muscle dysfunction.

In addition to careful consideration of historical data, information relating to the character of urination may be helpful in establishing an underlying cause. In Cases 1, 2 and 3, and ultimately in Case 4, the apparent unconscious urine overflow was consistent with complete detrusor muscle dysfunction.

With the exception of urinary incontinence and associated urine scalding on the limbs, clinical examination failed to reveal any abnormalities in any of the cases. The absence of any neurological deficits may be considered unusual, as 50% of cases in a series presented by Holt and Mair (1990) had a variety of neurological deficits, including perineal hypalgesia, various degrees of ataxia and impaired proprioception. Additionally, 7 of 15 cases reported by Carr (2002), and 10 of 34 cases reported by Schott and Carr (2003) had either neurological disease or detectable neurological lesions at post mortem examination.

Detrusor dysfunction may be caused by primary neurological disease involving the lower motor neurons (Clarke 1992; Sponseller 2003). Although the absence of other neurological signs (e.g. perineal hypalgesia) in the 4 cases presented was more suggestive of detrusor muscle dysfunction, the presence of neurological lesions could not be fully ruled out without more detailed pathological examination of the sacral grey matter and peripheral nerves. Unfortunately, attempts made in Case 2 to identify the caudal mesenteric ganglion and pelvic plexus were unsuccessful; however, examination of these structures and the sacral spinal cord in Case 3 failed to reveal any abnormalities. It is also possible that upper motor neuron bladder dysfunction, typically characterised by increased urethral tone, a variably sized tonic bladder and short episodes of urination (Clarke 1992), may secondarily result in detrusor dysfunction due to incomplete bladder emptying and subsequent accumulation of sabulous material within the bladder (Schott and Carr 2003; Sponseller 2003).

Conditions which compromise the adoption of a normal urination posture, such as lumbosacral pain, may result in incomplete bladder emptying and it has been suggested that this may also result in the accumulation of sabulous material within the bladder and secondary detrusor dysfunction due to the physical weight of the material, so-called myogenic dysfunction (Pringle and Schott 1997; Schott and Carr 2003; Sponseller 2003). It is therefore considered important to attempt to promote bladder emptying, either by catheterisation or pharmacological means, in cases where temporary urinary retention exists (Clarke 1992; Pringle and Schott 1997).

Although Cases 1, 2 and 3 had histopathological evidence of cystitis post mortem, this possibly resulted from chronic urinary stasis, a common finding in bladder dysfunction (Hodgson 1987; Clarke 1992; Pringle and Schott 1997). A variety of bacterial species have been implicated in equine cystitis, including *E. coli*, *Proteus*, staphylococci and streptococci (Hodgson 1987), all of which were cultured in the current case series. A quantitative measure of bacterial number was obtained only from Case 4. Greater than $10^5$ colony forming units/ml obtained by free catch is considered significant evidence in symptomatic human patients for a diagnosis of cystitis (Stamm 2001); however, lower numbers of bacteria ($10^2–10^4$/ml) may be indicative of infection in samples obtained by catheterisation or following recent voiding and water diuresis. The presence of pyuria alongside qualitative evidence of significant bacteruria is often considered sufficient evidence in uncomplicated human urinary tract infections to initiate empirical therapy (Stamm 2001). Case 4 had clinical findings consistent with severe cystitis prior to the accumulation of sabulous material within the bladder. Indeed, the initial presentation of incontinence may have been due to the presence of cystitis, since irritation of the bladder mucosa can result in an overactive bladder, leading to persistent dribbling of urine (Holt 1997). Furthermore, the prolonged duration of the incontinence in this case may have resulted from an insufficient duration of antibiotic administration, often required to achieve full resolution in cases of cystitis (Hodgson 1987). Alternatively, the initial episode of incontinence may have resulted from a delayed dystocia-related urethral sphincter dysfunction (Pringle and Schott 1997) with a subsequent predisposition to the development of cystitis. Although both incontinence and cystitis have been reported following ingestion of *Sorghum* species, these cases also exhibit additional neurological signs (Adams et al. 1969). Additionally, none of the cases in this report were exposed to these plants.

Despite the fact that repeated neurological examinations in Case 4 failed to reveal any deficits, the small size of the bladder when initially presented with urinary incontinence may have been consistent with an upper motor neuron lesion; particularly in view of the fact that this case was finally subjected to euthanasia due to a reported inability to stand. It would therefore have been interesting to examine spinal cord tissue post mortem for evidence of a progressive lesion. The use of pressure profilometry of the bladder and urethra may also have provided additional information regarding the pathogenesis in this case (Clarke et al. 1987; Clarke 1992). Irrespective of the initial cause of the urinary incontinence, however, it is likely that urinary retention contributed to progressive detrusor dysfunction and accumulation of sabulous material within the bladder.
Following a diagnosis of detrusor dysfunction, the prognosis for return of bladder function is grave (Holt and Pearson 1984; Holt and Mair 1990; Pringle and Schott 1997). Therapeutic options include drainage combined with repeated bladder lavage and surgical removal of sediment. However, these techniques fail to address the primary problem of impaired detrusor function. Pharmacological therapy to alter bladder muscle tone and urinary sphincter tone would seem more rational. Although parasympathomimetics have been beneficial in cases where there is temporary detrusor dysfunction (Clarke 1992; Booth et al. 2000), their success in cases associated with sabulous urolithiasis has been poor (Schott and Carr 2003). This was demonstrated in Case 3, where an increasing dosage of the muscarinic agonist bethanecol failed to resolve the bladder dysfunction, even when combined with periodic bladder drainage.

Increasing sphincter tone is of use in some cases of human and canine urinary incontinence, using either α-adrenergic agonists (e.g. phenylpropanolamine) and/or oestrogens (Aaron et al. 1996; Sullivan and Abrams 1999; Scott et al. 2002). Although in theory this effect would be less desirable in cases of detrusor dysfunction, whereby increased urethral tone may lead to increased urinary retention and increased stretching of the bladder wall, Watson et al. (1997) reported 2 cases of sabulous urolithiasis which improved following oestrogen therapy. Similarly, Madison (1984) reported a case of oestrogen-responsive urinary incontinence in an aged pony mare, although it was not stated whether sabulous urolithiasis was present. It remains possible that oestrogens alleviate incontinence by a different mechanism in horses from that in man and dogs. In Case 4, there was some mild response to oestrogen therapy, consistent with oestrogen raising the threshold for overflow of urine from the bladder (Holt 1997). Although the spontaneous improvement in severity of urinary incontinence in Case 4 throughout the winter months was initially considered to be consistent with the apparent increased severity during oestrus, suppression of oestrus via the use of progestogens actually worsened the incontinence. Alternatively, the apparent increase in severity throughout the summer may have resulted from increased water intake from the pasture exceeding maintenance requirements and resulting in an increased urine output. Such an occurrence may have resulted in the owner’s perception that the severity of incontinence had worsened.

Therapeutic protocols to decrease urethral tone would be unlikely to result in full evacuation of the bladder and may initially make the degree of incontinence worse by decreasing the threshold for overflow. They are, therefore, of limited use in sabulous urolithiasis but useful in cases of upper motor neuron bladder dysfunction, which is characterised by an increase in urethral tone (Sponseller 2003). It may be that combination therapy with muscarinic agonists and α-adrenergic antagonists (e.g. phenoxybenzamine) would be more successful in cases of detrusor dysfunction. However, it would be anticipated that any therapy must be initiated early to avoid irreversible detrusor dysfunction.

### Manufacturers’ addresses

1. Cook Veterinary Products, Bloomington, Indiana, USA
2. Glenwood Laboratories Ltd, Chatham, Kent, UK.
3. Hoechst Roussel Ltd., Milton Keynes, Buckinghamshire, UK.
4. Intervet UK Ltd., Cambridge, Cambridgeshire, UK.

### References


