Urinary incontinence in the horse: a systematic approach to diagnosis and treatment

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Abstract

Urinary incontinence is an uncommon presenting clinical sign in horses. Despite a variety of inciting causes, most horses with urinary incontinence present with chronic end-stage detrusor dysfunction, characterized by overflow incontinence, cystitis and accumulation of sabulous sediment in the bladder. In some cases, a specific underlying etiology can be identified and treated successfully. In most cases however, a definitive diagnosis is difficult to establish and treatment is symptomatic, consisting of regular catheterization to empty the bladder and the use of drugs that stimulate detrusor muscle function and/or relax the urethral sphincters. A substantial commitment from the owner is required to treat these horses, as they need meticulous nursing care to prevent secondary complications such as urine scalding. Early intervention before development of chronic overflow problems is the key to therapeutic success, as response to treatment in long-standing cases is poor.

Keywords

I. Review of the neuroanatomy and physiology of the lower urinary tract

A basic understanding of the normal neuroanatomy and physiology of the lower urinary tract is required when investigating urinary incontinence in the horse. The lower urinary tract of the horse consists of the bladder and bladder outlet (urethral sphincters and urethra). Bladder function is controlled by parasympathetic and sympathetic neurons innervating the detrusor muscle. The bladder outlet is controlled by parasympathetic and sympathetic fibers innervating the internal urethral sphincter and somatic fibers innervating the external urethral sphincter. The sympathetic nerve supply to the smooth muscle of the detrusor and the internal urethral sphincter is supplied by the hypogastric nerve, with the preganglionic fibers originating from L1-L4 and synapsing in the caudal mesenteric ganglion. Postganglionic fibers supply both the detrusor ($\beta2$-adrenoreceptors) and the internal urethral sphincter (α-adrenoreceptors). Parasympathetic innervation is provided by the pelvic nerve that originates from spinal cord segments S2-S4. The somatic nerve supply to the striated muscle of the external urethral sphincter is supplied via a branch of the pudendal nerve, which originates from spinal cord segments S1-S2.

Involuntary control of urination is a reflex mechanism. When the bladder is filling with urine, afferent signals increase sympathetic activity, inhibiting detrusor contraction and increasing internal urethral sphincter tone. When the bladder becomes full, stretch receptors in the bladder wall are activated and afferent signals result in increased parasympathetic activity. Postganglionic parasympathetic fibers stimulate muscarinic receptors in the bladder wall, causing the detrusor muscle to contract. Simultaneous inhibition of sympathetic activity causes the internal urethral

sphincter to relax. The detrusor muscle pulls the neck of the bladder open, and the bladder is emptied.

In addition, motor areas in the cerebral cortex projecting onto the midbrain, pons and medulla facilitate voluntary inhibition or contraction of the detrusor and urethral sphincters via somatic motor neurons in the reticulospinal and tectospinal tracts of the spinal cord which override the micturition reflex and enable the horse to have voluntary control over urination.

II. Pathophysiology and classification of urinary incontinence

Urinary incontinence is defined as involuntary leakage of urine which occurs when intravesicular pressure generated by the detrusor muscle exceeds outflow resistance generated by the urethral sphincters. In the horse, this may occur with both neurological and non-neurological disorders.

In the case of neurological disorders, urinary incontinence may be caused by upper motor neuron (UMN) lesions affecting the brainstem and spinal cord cranial to the lumbosacral intumescence; or they may be caused by lower motor neuron (LMN) lesions affecting the gray matter of the cauda equina (i.e. L1 to S5). UMN lesions result in increased urethral sphincter tone due to loss of inhibition of sympathetic and pudendal activity. The bladder is able to contract normally, but will only empty when pressures within the bladder exceed the resistance of the urethra. LMN lesions result in flaccid paralysis of the bladder due to damage to the preganglionic or postganglionic parasympathetic neurons, and/or in reduced contractility of the urethral sphincters as a result of damage to pre- or post-ganglionic α-adrenergic sympathetic neurons and/or the pudendal nerve. Loss of detrusor function and reduced contractility of the urethral sphincters results in overflow incontinence.

When considering neurological causes of urinary incontinence, it is important to remember that bladder paralysis and overflow incontinence that is consistent with LMN urinary dysfunction may occasionally be found in horses with neurological diseases that do not typically affect the gray matter of the cauda equina (e.g. cervical stenotic myelopathy). This is because the signs of UMN urinary dysfunction may be missed for many months, during which time secondary overflow incontinence develops. Incomplete bladder emptying leads to progressive deposition of large quantities of sabulous sediment in the bladder (Figure 1), and together with normal urine accumulation, leads to chronic bladder distension. Stretching of the detrusor muscle causes breakdown of tight junctions which prevents depolarizing waves passing from muscle fiber to muscle fiber, leading to progressive loss of detrusor function and ultimately, bladder paralysis and overflow incontinence. In this late stage of the disease, horses with UMN lesions may be indistinguishable from those with LMN lesions.
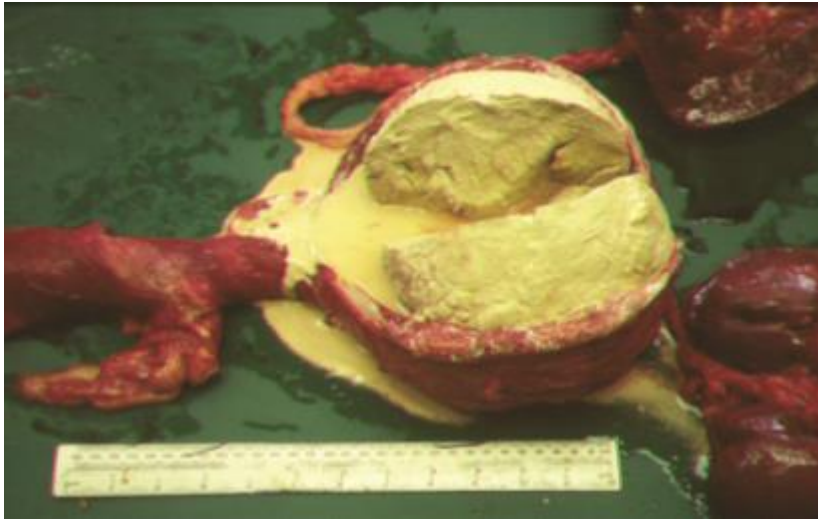
Figure 1: Post mortem view of a bladder from a horse with chronic overflow incontinence and sabulous cystitis. Notice the large accumulation of sabulous sediment in the bladder lumen. Photo courtesy of John Keen)

Non-neurological causes of urinary incontinence may also occur. These are usually associated with inflammation of the detrusor muscle leading to detrusor over activity and increased intravesicular pressure (e.g. bacterial cystitis or urolithiasis); abnormalities in the normal path for urine outflow resistance (e.g. ectopic ureters) or disorders not associated with the neurological system that prevent complete emptying of the bladder (e.g. musculoskeletal disease).

In some cases a cause of urinary incontinence cannot be established despite a thorough diagnostic investigation. Urinary incontinence in these cases is considered to be 'idiopathic', and is often complicated by sabulous cystitis[19].

III. Causes of urinary incontinence

- **Neurological disorders:**
  - **Primarily UMN lesions:** cervical spinal cord trauma, cervical stenotic myelopathy, EHV-1 myeloencephalopathy[1, 2], equine degenerative myeloencephalopathy, equine protozoal myeloencephalitis (EPM), aberrant larval migrans.
  - **Primarily LMN lesions:** polyneuritis equi, sorghum toxicity[3], iatrogenic[4], sacral vertebral trauma, osteomyelitis[5, 6] and neoplasia.
- **Non-neurological (myogenic) disorders:** cystitis[7]; urolithiasis[8,9]; trauma (e.g. bladder trauma secondary to dystocia); neoplasia[10,11]; ectopic ureter[12]; hypoestrogenism or urethral sphincter hypotonia[13,14]; musculoskeletal disease resulting in lumbar, sacral or hindlimb pain[19].
- **Idiopathic detrusor dysfunction:** no primary neurological disease identified to explain bladder dysfunction. Often chronic and complicated by sabulous cystitis[16,17,18]

IV. Diagnostic approach to urinary incontinence

The clinical manifestations of urinary incontinence in the horse depend on the underlying disease process, but by definition, all horses will have a loss of bladder control which manifests as constant or intermittent dribbling of urine from the vulva or penis (Figure 2). This can either be at rest or during exercise, and any activity that causes a sudden increase in intra-abdominal pressure may exacerbate the signs (e.g. coughing). In geldings and stallions, urine may be seen dribbling from the prepuce without protrusion of the penis. Affected horses usually smell of urine and in chronic cases, urine staining and scalding of the perineum in mares (Figure 3) and the hindlegs in gelding may be evident. In some cases the horse may be able to urinate voluntarily despite being incontinent, but the stream is usually weak (Figure 4).

Figure 2: Urinary incontinence in a gelding characterized by constant urine dribbling and penile paralysis.

Depending upon the cause, other clinical signs may also be apparent and in some cases can be used to guide the veterinarian towards a preliminary diagnosis (e.g. fever followed by sudden onset ataxia and paresis in cases of EHV-1 myeloencephalopathy). The initial assessment should be aimed at obtaining a minimum database of information that can be used to help generate a list of differential diagnoses and an appropriate diagnostic plan. An attempt should be made to differentiate between neurological and non-neurological bladder dysfunction, however it can be argued that this distinction is of academic interest only, as the treatment principles for urinary incontinence are similar, irrespective of the underlying cause. More importantly, a definitive diagnosis will enable to veterinarian to provide an accurate prognosis for the client.

Figure 3: Urine staining and scalding of the perineum. Urinary incontinence in this case was caused by post partum bladder trauma and resolved with treatment.

Figure 4: Urinary incontinence in a filly. The filly was able to urinate voluntarily despite being incontinent, but the stream was weak.

History

It is important to obtain a detailed history in cases of urinary incontinence, as the inciting cause may have occurred many months prior to presentation. If any aspect of the history points to a primary disease, then that particular aspect should be focused on during the physical examination.

There does not appear to be an association between breed and urinary incontinence, however the gender of the horse be important. Idiopathic myogenic bladder dysfunction occurs mainly in geldings[20] and oestrogen-responsive urinary incontinence has been reported in mares[13,14]. Reproductive status may also be relevant. Breeding injuries and dystocia has been reported to cause urinary incontinence in mares and owners may confuse frequent urination during oestrus as urinary incontinence. Ectopic ureters appear to be more common in females, although this may simply reflect the fact incontinence associated with ureteral ectopia is easier to recognize in females[12].

The age of the horse will help rule out certain causes of urinary incontinence. Ectopic ureters and other congenital anomalies will cause incontinence that is present from birth[12]. Remember that foals with a patent urachus will dribble urine from the umbilicus and this may be misinterpreted as incontinence in colts. Osteomyelitis of the lumbosacral vertebrae caused by *Rhodococcus equi* has been reported as a cause of neurological urinary incontinence in foals[5].

The environment and diet may also provide clues to the cause of incontinence. Ingestion of *Sorghum* spp. has been reported to cause a syndrome of ataxia and urinary incontinence, and should be considered in regions where horses have access to these plants[3].

Careful questioning may identify iatrogenic causes of urinary incontinence. Epidural administration of alcohol has been reported in show horses and the author has treated a horse with urinary incontinence secondary to a misguided attempt at medicating the sacroiliac joints. There are also

reports of iatrogenic urinary incontinence following routine theriogenological procedures[4]. Although rare, aberrant larval migrans should be considered in any horse with an inadequate deworming history.

Other aspects of the medical history may also prove useful. A recent history of trauma may allude to spinal cord injury e.g. cervical or lumbosacral fractures. Sudden onset ataxia and urinary incontinence and affecting more than one horse on the property or a history of recent exposure to EHV-1 infected horses should alert the veterinarian to the possibility of EHV-1 myeloencephalopathy[1, 2]. A history of chronic musculoskeletal pain affecting the back and hindlimbs may be significant. If a painful musculoskeletal condition limits a horse's ability to posture to urinate, complete bladder emptying may impossible. This may lead to the accumulation of large quantities of sabulous urinary sediment in the bladder, leading to detrusor dysfunction and eventual bladder paralysis[19].

Physical examination

In horses with urinary incontinence, the general physical examination is frequently non-specific and unrewarding, and there are rarely other clinical signs apart from urinary incontinence that may point towards to a specific underlying disease process. Fever may indicate an infectious process, and in such cases the veterinarian should consider bacterial cystitis, EHV-1 myeloencephalopathy and spectic osteomyelitis. Observing the horse urinate may be useful. Passage of red urine or stranguria in addition to incontinence may indicate the presence of a urolith or cystitis. Abnormal posturing may indicate musculoskeletal pain. Pain on palpation of the lumbosacral region or evidence of hindlimb lameness may also be significant and should be investigated.

A neurological examination should be performed in all horses that present with urinary incontinence. In horses with UMN urinary dysfunction, lesions in the spinal cord cranial to the lumbosacral intumescence will in most cases be associated with ataxia, paresis and dysmetria affecting the thoracic and pelvic limbs. Severely affected horses may even be recumbent. In these cases, loss of the inhibition of the micturition reflex and pudendal activity leads to increased urethral resistance despite a full bladder. On rectal exam, the bladder is turgid, and can be small, normal or large in size. Manual emptying per rectum difficult. Urine incontinence in these cases is characterized by short intermittent bursts of urine passage with incomplete bladder emptying, and often occurs secondary to increases in intra-abdominal pressure (e.g. sudden movement). In horses with LMN urinary dysfunction, lesions involving the cauda equina results in urinary incontinence in association with other signs of LMN disease, including flaccid tail paralysis, faecal retention, loss of anal sphincter tone, loss of sensation to the perineum (Figure 5) and hindlimb muscle atrophy and weakness. In geldings and stallions, the penis may also be paralyzed. LMN damage leads to loss of detrusor function and overflow incontinence. In these cases the bladder is full with relaxed urethral sphincters, which results in urine dribbling because of overflow from the bladder. On rectal exam the bladder is large and can be easily expressed using manual pressure. Urine dribbling is continuous, which helps differentiate it from UMN disease.

Figure 5: Flaccid tail paralysis, faecal retention, loss of anal sphincter tone and loss of sensation to the perineum in a horse with LMN urinary dysfunction. Polyneuritis equi was confirmed at post mortem.

Clinical laboratory tests

Blood should be collected and submitted for a complete hematological profile. Leukocytosis with and hyperfibrinogenemia indicates an inflammatory or infectious process and may be associated with septic osteomyelitis or bacterial cystitis.

A urinalysis should be performed and the sediment should be examined. Remember that crystaluria (primarily $CaCO_3$) is normal in the horse and cannot be used to diagnose urolithiasis. Haematuria may indicate the presence of a urolith, cystitis, neoplasia or bladder trauma. Pyuria indicates urinary tract infection and in these cases, a sterile urine sample via urinary catheterization should be obtained for bacterial culture and sensitivity testing.

Caution should be used when interpreting the significance of bacterial cystitis in horses with incontinence. In rare cases primary cystitis may be a direct cause of urinary incontinence[7]. Inflammation of the bladder wall in such cases leads to detrusor over activity and increased intravesicular pressure, which causes incontinence. A much more common scenario however, is the development of secondary cystitis, usually due to urinary retention and often accompanied by deposits of sabulous sediment in the bladder. Cystitis in these causes is thought to be caused by the constant irritation of the mucosa by the crystalloid material and/or ammoniagenesis by bacterial within the sediment[18]. Over time, chronic inflammation of the bladder wall leads to cicatrical pillars within the lumen of the bladder that permanently alters bladder distensibility and impairs normal detrusor function.

Endoscopy

Endoscopy is a useful diagnostic modality for horses with urinary incontinence and should be performed in all cases where a clear etiology has not been identified. Non- specific findings that are associated with most cases of chronic urinary incontinence include mucosal hyperaemia, hemorrhage and fibrin accumulation (Figure 6); often in combination with deposits of sabulous sediment. Dilated ureteral openings caused by chronic bladder distension and detrusor dysfunction may also be seen in cases with long-standing incontinence. These horses develop vesiculouretral reflux and are at risk of ascending bacterial infections.

Figure 6: Endoscopic view of the bladder of a horse with chronic urinary incontinence. Notice the mucosal hyperaemia, hemorrhage and fibrin accumulation. Photo courtesy of Derek Knottenbelt.

In some cases, a specific finding on endoscopy may enable to veterinarian to make a definitive diagnosis. Cystic calculi, mucosal hyperaemia and thickening of the bladder wall will be seen in cases of urolithiasis. Transmural bruising, mucosal hyperaemia and necrosis will be seen in cases of post parturient bladder trauma. A thickened irregular bladder wall and the presence of soft tissue masses may be seen in cases of bladder neoplasia. Ectopic ureteral orifices can be visualized in the vestibule and vagina of females and pelvic urethra of males.

Ultrasonography

Transrectal ultrasonography may be useful to rule confirm the presence of sabulous cystitis and to rule out uroliths, neoplasia or congenital anomalies, particularly if the veterinarian does not have access to an endoscope.

Urethral and bladder pressure profiles

Horses that are able to generate some degree of intravesicular pressure are more likely to respond to treatment and therefore measurement of bladder and urethral pressures to assess urinary sphincter and detrusor muscle function may have some prognostic value. Normal values for mares and geldings have been reported[21, 22]; however the equipment is usually only available in specialized centers and is therefore usually the remit of veterinarians based at referral centers or academic institutions.

V. Treatment of urinary incontinence

Treatment for urinary incontinence varies depending upon the underlying cause, but in general, the therapeutic aims are to resolve any underlying primary problems; encourage bladder emptying and to treat and prevent any secondary complications.

Once bladder paralysis occurs, sabulous sediment should be removed from the bladder via endoscopic or catheter lavage (Figure 7) and the bladder should be emptied regularly to prevent continued distension and further damage to the detrusor[18]. Emptying of the bladder is best achieved by intermittent passage of a urinary catheter, and in male horses, passage of a catheter can be facilitated by performing a temporary urethrostomy. Depending on the dedication of the client, this can be an effective long term strategy to prevent bladder distention in incontinent horses[18]. Urinary catheters should be used judiciously however, as they predispose the horse to secondary bacterial cystitis, ascending infections of the upper urinary tract and urethral strictures. Owners should also be instructed to clean the perineum and hindlegs of the horse daily and apply petroleum-based creams to the skin to prevent urine scalding.

Figure 7: Removal of sabulous sediment form the bladder via catheter lavage. A temporary urethrostomy was used to facilitate the procedure in this gelding

Administration of bethanechol chloride (0.25-0.75 mg/g PO q8-12 h) can also be used to stimulate detrusor muscle function and encourage bladder emptying. Unfortunately response to treatment is often disappointing, possibly due to an inability of the detrusor to contract normally due to chronic over stretching.

Administration of the α adrenergic blocker phenoxybenzamine (0.7 mg/kg PO q6 h) and skeletal muscle relaxants such as dantrolene or diazepam may help relax the urethral sphincter and encourage bladder emptying in cases of acute onset UMN urinary dysfunction where the detrusor is still able to function normally, but there is little indication for their use in horses with LMN urinary dysfunction or chronic end-stage urinary incontinence where the detrusor has undergone irreversible damage.

Antimicrobial treatment is usually indicated for horses with bladder paralysis, particularly if there is evidence of a urinary infection. The choice of antibiotic should be based upon the results of culture and sensitivity testing, but good empirical choices are trimethoprim sulphonamides and penicillin.

VI. Prognosis

The prognosis is largely dependent on the primary cause and the chronicity of the problem. Horses with incontinence caused by congenital anomalies, cystoliths, primary bacterial cystitis, post-parturient bladder trauma, hypoestrogenism, septic osteomyelitis, equine protozoal myeloencephalitis, EHV-1 myeloencephalopathy and cervical spinal cord trauma may all respond favorably following correction of the primary problem if treated promptly. In many cases however, the subtle neurological deficits accompanied by incontinence are not recognized by owners until months or even years after the initial damage has occurred, during which time irreversible detrusor dysfunction has occurred. A successful response to treatment in these cases is unlikely, regardless of the initiating cause. Conditions that are less likely to respond to treatment, irrespective of when the diagnosis is made, include equine degenerative myeloencephalopathy, cervical stenotic myelopathy, polyneuritis equi, sorghum toxicosis and neoplasia.

VII. References

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