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# Diagnosis of Urinary Tract Disease in the Horse

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**Abnormal urination and weight loss are the most common presenting complaints in horses with urinary tract disease. This issue of *Large Animal Veterinary Rounds* discusses urinary tract diseases and describes the causes and diagnoses of polyuria/polydipsia and hematuria in horses.**

## Polyuria / Polydipsia

Polyuria/polydipsia (PU/PD) is an important clinical sign indicating a failure of normal homeostatic mechanisms to control water balance. Polydipsia is an excessive thirst manifested by excessive water intake.[1,2] Polyuria is defined as the formation and excretion of large amounts of urine.[1,2] It is not uncommon for owners to mistake pollakiuria (increased urination frequency) with polyuria.[2] One subjective way of assessing urine output is a wet stable, if the horse is kept on sawdust or shavings rather than straw.[2,3] To confirm that the horse is polyuric/polydipsic, the water intake or urine production should be measured for one or more 24-hour periods. It is usually easiest to measure water intake.

Horses obtain water from three primary sources; drink, food, and metabolism. Water intake varies with age, diet, workload, environment, and gastrointestinal water absorption. Maintenance water requirements for horses are 40-60 mL/kg body weight (BW)/day.[4] Horses that are exercised heavily, stabled in hot climates, or have diarrhea may have increased water consumption, yet produce a normal amount of urine.[3] In normal horses, glomerular filtration rate exceeds 1000 L per day, but 99% of this water is reabsorbed by the kidneys.[3] Normal urine production is around 15-30 mL/kg BW/day. This urine is usually 3-4 times more concentrated than plasma (urine osmolality of 900-1200 mOsm/kg, specific gravity 1.025 to 1.050).[3]

### *Causes of primary polydipsia*

#### *Psychogenic polydipsia*

This is one of the most common causes of PU/PD in mature stabled horses.[2] Psychogenic polydipsia is a behaviour often seen in young animals and is associated with boredom, possibly induced by changes in diet or environment.[4-6] Other predisposing factors include excessive salt or dry matter intake.[4-6]

Affected animals are in good body condition, have urine with a very low specific gravity (SG<1.005), and are not azotemic. Polyuria/polydipsia is usually more severe than is found in chronic renal failure or Cushing's disease.[5] Owners report that horses with PU/PD drink 2-3 times more water than their stable mates and their stables are often flooded with urine.[3] The response to water deprivation depends on the chronicity of the disease; if PU/PD is not long-standing (several weeks), affected horses usually concentrate their urine. In long-standing cases, the urine is not concentrated.[2,3] This is probably due to renal "medullary wash-out," where the normal osmotic gradients between the renal interstitium and the tubular lumen are reduced.[5,6] As a result, water cannot be reabsorbed from the renal tubules and tubules do not respond to the antidiuretic hormone (ADH) that is present at high concentrations.[2] Horses with "medullary washout" and psychogenic polydipsia usually concentrate their urine in response to the gradual restriction of water, as practiced in the modified water deprivation test.

Case management of psychogenic polydipsia includes efforts to reduce boredom, frequent feeding with increasing amounts of forage, more exercise, and alteration of the management routine. Water availability can be restricted, but should always be sufficient to meet maintenance (ie, 50 mL/kg/day), work, and environmental needs of the horse.[2,3]

#### *Excessive thirst*

Humidity and high ambient temperatures usually induce thirst, particularly if associated with dry feed consumption;[2] in these circumstances, urine output is normal.

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### *Compulsive salt intake (excessive salt consumption)*

This is an uncommon cause of PU/PD. Affected horses may "attack" salt blocks, particularly if they contain palatable ingredients such as glucose, and ingest large pieces of the block.[2] The history and reasons for this habit are usually unclear. The fractional excretion of sodium and chloride is often increased and affected horses are usually able to concentrate urine in response to the water deprivation test.[3,7] Treatment consists of preventing access to salt and limiting water intake to maintenance, 50 mL/kg/day.[3]

## *Causes of primary polyuria*

### *Diabetes mellitus*

Both insulin and noninsulin dependent diabetes mellitus are reported in horses. Peripheral insulin resistance secondary to Cushing's disease is the only common cause.[3] Diabetes mellitus cases have consistent hyperglycemia resulting in glucosuria and osmotic diuresis.[2,3] Treatment is usually supportive, although in cases of the insulin dependent type, insulin supplements could be helpful.[3]

### *Cushing's syndrome*

Cushing's syndrome is a common disorder of older horses. The syndrome is caused by a pituitary adenoma and is associated with other signs such as hirsutism, recurrent laminitis, and an abnormal distribution of fat in the body. PU/PD may be due to destruction of the neurohypophysis leading to decreased ADH secretion, hyperglycemia resulting in osmotic diuresis, or to increased cortisol secretions that can directly increase the glomerular filtration rate. Diagnosis is usually based on clinical signs and can be confirmed with a dexamethasone suppression test.[8] For this test, a blood sample is withdrawn in a heparinized tube, between 4-6 pm, and the cortisol level is measured. After blood withdrawal, dexamethasone (40 μg/kg, IM) is administered. A second blood sample is withdrawn around 19 h later and the cortisol level is measured. At the second sampling, normal horses have cortisol concentrations ≤ 1 μg/dL, horses with Cushing's disease have higher values.[8]

Treatment is mainly supportive and includes feeding good quality hay, hoof care to manage or prevent laminitis, and haircoat care including clipping in summer. Different drugs have been used, but the dopamine agonist, pergolide, at a dose of 1.7 to 5.5 μg/kg, PO, once daily, is most effective.[9]

### *Diabetes insipidus*

Two types of diabetes insipidus have been identified in horses: central and nephrogenic. Central diabetes insipidus is a failure of ADH production; production failure has also been reported in Cushing's syndrome and viral encephalomyelitis.[2] The nephrogenic type is due to a lack of kidney tubule response to ADH.[2] It is reported as a familial condition or secondary to acute bacterial infection of the kidney.[2] Horses with diabetes insipidus are unable to concentrate urine (SG < 1.025) in response to the water deprivation test.[2,3] ADH replacement therapy may be helpful for central diabetes insipidus, however this treatment may be cost prohibitive.[3] Nephrogenic diabetes insipidus can also be treated by restrictions of sodium and water intake.[3] Administration of thiazide diuretics is also reported to be beneficial, but the mechanism is poorly understood.[3]

### *Renal insufficiency or failure*

Acute renal failure in horses is initially associated with anuria or oliguria; if the horse survives, it may then develop polyuria due to renal tubular damage.[6,10] Renal failure can be caused by exposure to nephrotoxic agents (aminoglycosides, nonsteroidal anti-inflammatory drugs, myoglobin, and vitamin $K_3$ or D) or vasomotor nephropathy (hypoperfusion or ischemia).[8] Acute renal failure should be suspected in horses that have more marked depression and anorexia than expected for the primary diseases process.[8] Affected horses are usually azotemic with abnormal urinalysis (hematuria, proteinuria, casts, or decreased specific gravity) and other findings dependent on the primary disease process.[8]

Chronic renal failure in the horse can be caused by proliferative glomerulonephritis (immune mediated), chronic interstitial nephritis (consequent to vasomotor nephropathy, exposure to nephrotoxic agents, renal papillary necrosis, urinary tract obstruction, or renal hypoplasia), or pyelonephritis (result of ascending infection or urolithiasis).[8] Horses with chronic renal failure are usually azotemic and have isosthenuria (urine SG = 1.008-1.012). Other clinical signs include weight loss, dental tartar, decreased appetite, and oral ulcers.[8] Urinalysis abnormalities vary according to the primary cause of renal failure.

In cases of chronic renal failure due to infectious causes (pyelonephritis), antibiotic treatment based on urine culture and sensitivity can be helpful. In general, treatment for horses with polyuria due to chronic renal failure is mainly supportive.[8] Water should be available at all times and salt should be offered as long as no edema is seen. The forage component of the diet should be changed from legume (alfalfa or clover) to grass, to reduce calcium intake and decrease the risk of hypercalcemia. Body condition can be maintained by increasing grain intake and adding fat (corn oil gradually increased up to 450 mL/day) to the diet.[8] Dietary protein intake should meet requirements for maintenance and increased urinary protein loss, but should not be excessive.

### *Iatrogenic*

Fluid therapy, $\alpha_2$ agents (xylazine and detomidine), or corticoid administration can lead to polyuria.[2,3]

## *Diagnostic work up for PU/PD*

It is important to confirm the presence of PU/PD. This can be achieved by confining the horse to a stall and measuring urine output using collection devices, or more conveniently, water intake.[2] The procedural steps to work-up a case of PU/PD are outlined in Figure 1.

### *Water deprivation test*

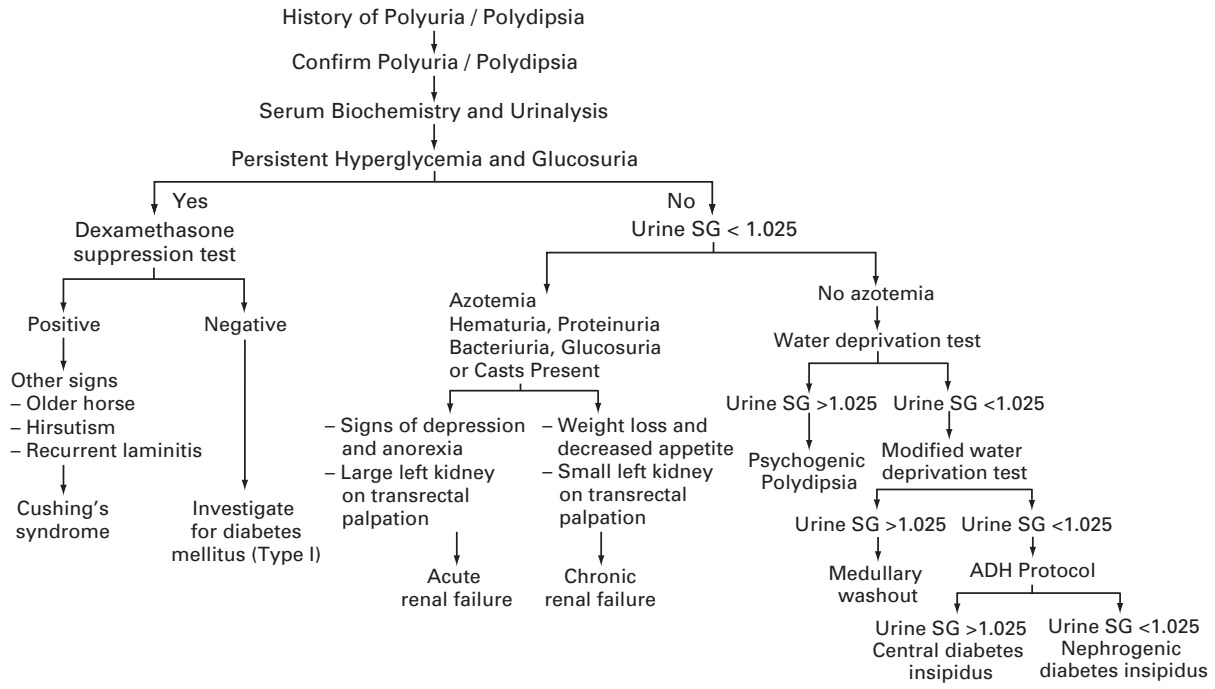
This test should only be performed in the absence of azotemia, dehydration, or hyperglycemia. The test assesses the ability of the kidneys to respond to ADH. A urine sample is taken and the horse is weighed. Water is withdrawn and urine SG is measured frequently during the test period (12 -24 h).

### *Modified water deprivation test*

This test is indicated if urine concentration fails to occur with the water deprivation test. Water intake is restricted to 40 mL/kg BW/day for 3-4 days.[2] Psychogenic PU cases with "medullary washout" should respond to this test by increasing the urine SG above 1.025. If no response is elicited (SG < 1.025) after 4 days, the next diagnostic step is to administer ADH to determine if the horse has diabetes insipidus (Figure 1).

In both tests, horses should be monitored closely for signs of dehydration because horses with polyuria (particularly, diabetes insipidus) may keep excreting water in spite of water deprivation and become severely dehydrated.[3] The tests should also be

**Figure 1: A flowchart for the diagnostic work-up in horses presenting with polyuria/polydipsia**




discontinued if the urine SG rises above 1.025, the horse loses 5% of its body weight, or the horse becomes azotemic.[3,4,6,11]

### *ADH test protocol*

If the urine is not concentrated after 3-4 days of the modified water deprivation test, ADH is administered to differentiate between central and nephrogenic diabetes insipidus. Three injections of 60 IU of synthetic ADH, IM, are given at 6-hour intervals. If the urine concentrates (SG >1.025), central diabetes insipidus is diagnosed. If the urine does not concentrate (urine SG < 1.025) and renal disease and Cushing's disease can be ruled out, the horse may have nephrogenic diabetes insipidus.

## Hematuria

Red to brown urine discolouration can be due to the presence of intact red blood cells (hematuria), hemoglobin, or myoglobin (the latter two are called pigmenturia) in the urine. In the case of hematuria, centrifugation of the urine sample reveals a layer of erythrocytes covered by clear urine. The urine remains discoloured if the urine contains hemoglobin or myoglobin. It is important to realize that normal equine urine contains pyrocatechine, an oxidizing agent that may result in red to brown discolouration of the urine after exposure to air or contact with snow.[12,13] The urine can also be discoloured by certain medications: orange with rifampin and dark brown to black with doxycycline.[14] In addition, urine colour can change to brown if stored in the refrigerator for several days.[14]

Examination of horses with hematuria should include transrectal palpation, ultrasonography, endoscopic examination of the urinary tract, urinalysis, and urine culture. Complete blood count, serum biochemistry, and coagulation tests can be helpful. Coagulation testing is particularly important if the other tests fail to identify the cause of hematuria.[12] A midstream 'free catch' urine sample is most desirable for urinalysis.[12] If hemorrhage originates from the kidneys, ureters, or bladder, hematuria will be noticed throughout urination. Hemorrhage from the distal part of the urethra, and the bladder neck or proximal urethra results in hematuria at the beginning and the end of urination, respectively.[3] A suggested diagnostic work-up can be seen in Figure 2.

### *Causes of hematuria*

#### *Urethral rent or defect*

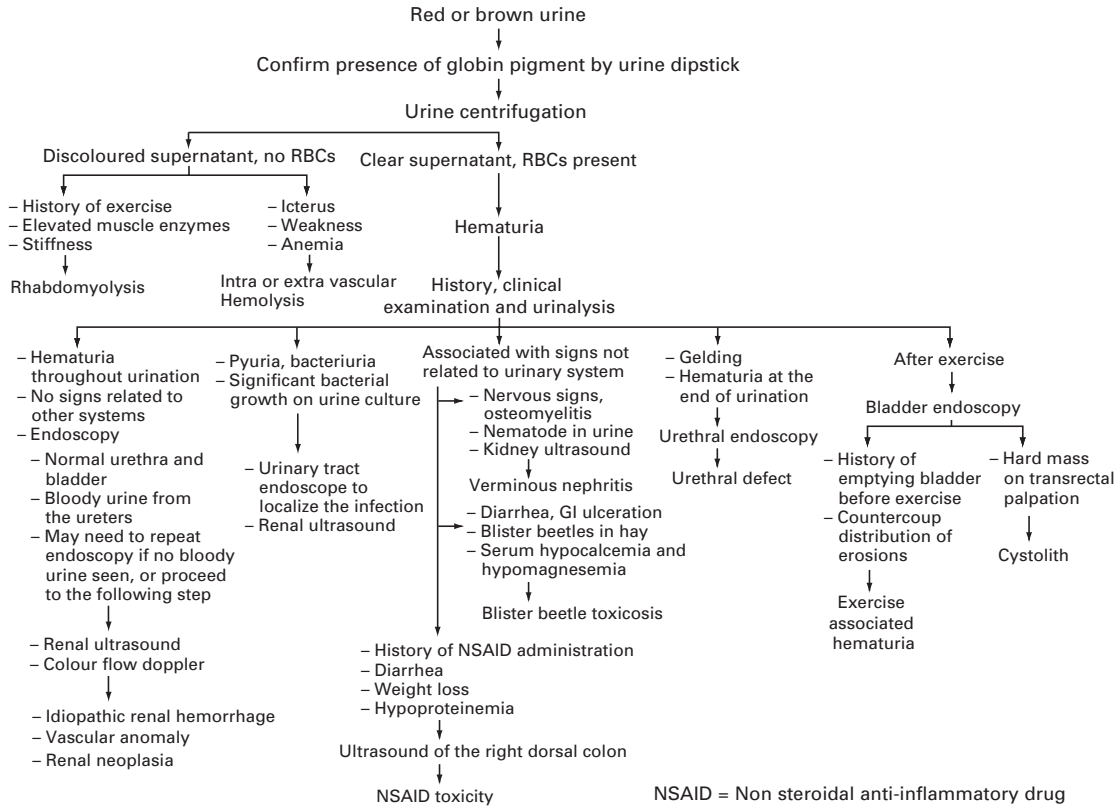
This defect occurs on the convex surface of the urethra at the ischial arch level and causes hematuria in geldings and hemospermia in stallions.[12] Interestingly, the most affected horses are Quarter Horses or Quarter Horse crosses.[3] Hematuria is typically at the end of urination and the exact cause is unknown. It has been suggested, however, that contraction of the bulbospongiosus muscle during ejaculation or urination results in a "blowout" of the corpus spongiosum penis (vascular tissue surrounding the urethra) into the urethral lumen.[3] Often, urethral rents heal without treatment.[12] However, if hematuria persists for more than a month, or if the gelding becomes anemic, surgical treatment should be employed.[3,12] Two surgical approaches to treat urethral rent are available: temporary ischial urethrotomy, or making a vertical incision that extends into the corpus spongiosum penis, but leaves the urethra intact.[12] Hematuria should resolve in 1 week after surgical treatment.[3]

#### *Urinary tract infection*

Urinary tract infections are mainly ascending in origin;[3] however, in foals, septic nephritis can be caused by septicemia. Mares appear to be at a higher risk for developing urinary tract infection than geldings and stallions, because of their shorter urethra.[3]

***Urethritis:*** Primary bacterial urethritis is undocumented in horses.[3,12] On endoscopic examination of the urethra, the prominent vasculature and cavernosal spaces can be mistaken

**Figure 2: A flowchart for the diagnostic work-up in horses presenting with hematuria**




for inflammation or even hemorrhage of the urethra.[12] Traumatic, parasitic (habronemiasis), and neoplastic diseases of the urethra are known to occur.[3,12] Repeated catheterization and cystolithiasis can result in urethritis.[12] *Habronema* larvae infestation causes granuloma of the urethral process and consequently, hematuria.[12] Treatment of urethritis consists of dealing with the underlying cause. Urethral infestation with *Habronema* can be treated with systemic or topical administration of ivermectin and corticosteroids.[12]

***Cystitis:*** Cystitis commonly occurs secondary to urinary flow disturbances caused by urolithiasis, bladder paralysis or tumour, anatomical defect, or iatrogenic trauma (catheterization or endoscopic examination).[3] Vaginitis and repeated urinary catheterization are also risk factors.[12] Signs include hematuria, pollakiuria, stranguria, pyuria, or urine scalding of the perineum in mares or the front of the hind limbs of male horses.[3] Pathological perineal urine scalding should be differentiated from pollakiuria and vaginal discharge during normal estrus activities in mares.[3] Diagnosis should be based on physical examination, transrectal palpation, cystoscopy, ultrasonography, urinalysis, and culture. Transrectal examination is helpful in case of cystolithiasis or bladder neoplasm. Definitive diagnosis is made by urine culture with >10,000 colony forming units/mL in a sample collected by catheterization or from a midstream urine flow.[3]

Antibiotic treatment should be based on urine culture and sensitivity, but a trimethoprime-sulfonamide combination is the initial choice.[3] Bladder lavage is particularly important in cases of sabulous urolithiasis. This is an accumulation of large amounts of crystalloid material in the bladder, usually secondary to bladder atony or paralysis, commonly seen in horses with a caudal spinal cord lesion.[3] Dietary supplementation with 50-75 g of salt or administration of a urine-acidifying agent (eg, ammonium chloride 20-40 mg/kg/day by mouth) has been recommended as part of the treatment for cystitis.[3]

***Pyelonephritis:*** Pyelonephritis has been associated with urolithiasis, cystitis, and bladder paralysis.[3,15] Hematuria, pyuria (rather than stranguria and pollakiuria), weight loss, fever, anorexia, or depression are also signs of pyelonephritis in horses.[3,15] Diagnostic work-up is similar to that for cystitis. Ureteral catheterization can be performed to assess if one or two kidneys are affected.[3] Treatment is mainly with systemic antibiotics. In unilateral unresponsive cases, surgical removal of the affected kidney and ureters may be attempted.[3,16,17]

***Idiopathic hematuria:*** Idiopathic hematuria is associated with sudden onset, unilateral or bilateral renal hemorrhage and is often life threatening.[3,18] Often, no other signs of disease are reported even with histological examination of affected kidneys.[12] It is usually diagnosed after exclusion of other causes of hematuria and >50% of affected cases are Arabians.[3] Treatment is supportive and may include blood transfusion and, if unilateral and persistent, nephrectomy.[3]

***Exercise-associated hematuria:*** Commonly, hematuria associated with exercise is microscopic and not observed; however, in severe cases, visible hematuria may occur. It is thought to be traumatic in origin and produced by the abdominal contents pounding the bladder against the pelvis during exercise.[3] It may be more common in horses that empty their bladder before exercise. Diagnosis is based on a

history of exercise and the presence of erosions or ulcers with a countercoup distribution on endoscopy.[3]

***Verminous nephritis:*** Hematuria can be associated with renal damage produced by *Halicephalobus deletrix* (previously *Micronema deletrix*) or *Strongylus vulgaris*.[12,19] Renal infection with *H. deletrix* is often associated with concurrent infection of the nervous system (meningoencephalomyelitis) and bones (maxilla or mandible causing osteomyelitis).[12] The nematode causes granulomas in the affected kidneys and may be found in the urine.[12]

Treatment consists of anthelmintics and anti-inflammatory drugs.[12] In cases with unilateral renal involvement where little residual renal function is left, nephrectomy can be considered.[12]

***Urinary tract neoplasia:*** Urinary tract neoplasia is a rare cause of hematuria.[12] Lymphosarcoma and adenocarcinoma are the most common kidney tumours.[12,20] Adenocarcinoma is more likely to cause hematuria than lymphosarcoma. Ultrasonography of the kidneys is very important in antemortem diagnosis.[12,20] Squamous cell carcinoma is the most common bladder tumour.[12,21] Affected cases present due to hematuria and/or stranguria, and palpable masses are felt on transrectal examination.[12] Urinalysis to identify neoplastic cells, endoscopy, and biopsy are also important diagnostic tools.

***Blister beetle toxicosis:*** Blister beetle toxicosis or cantharidin poisoning (the toxic component of blister beetles) can be associated with hematuria. Other clinical signs include diarrhea and ulceration of the gastrointestinal tract. Hypocalcemia and hypomagnesemia are seen on serum biochemistry. Diagnosis is based on clinical signs, the presence of beetles in the hay, and cantharidin in the urine. Treatment is by removing the source and symptomatic therapy.

***Drug toxicity:*** Nephrotoxic drugs, including the nonsteroidal anti-inflammatory drugs (NSAIDs) particularly phenylbutazone, can be associated with renal failure and hematuria. Other clinical effects of NSAID toxicity include right dorsal colitis, diarrhea, weight loss, and hypoproteinemia. Diagnosis is based on clinical signs, history of drug administration, and ultrasonography of the right dorsal colon. Treatment is primarily by discontinuing drug administration and supportive therapy.

***Vascular anomalies:*** Vascular anomalies of the urinary tract can be associated with hematuria and can be congenital or acquired.[12] Both arteriovenous and arterioureteral fistulas have been reported.[22] Diagnosis is based on ultrasonography (colour flow Doppler).[12]

***Urolithiasis:*** Urolithiasis is typically a disease of adult horses and accounts for 7.8% of the diagnoses of urinary tract disease in the horse.[23] Males, especially geldings, appear to be predisposed, and young racehorses may be at higher risk because of NSAID use.[3,24] The urinary bladder is the most common location for calculi (60%), followed by the urethra (24%), kidneys (12%), and ureters (4%).[23] As many as 10% of affected horses can have urolithiasis in >1 location. Signs of urolithiasis depend on the location and degree of obstruction.[3] Incomplete obstruction is usually associated with dysuria, urinary incontinence, and mild abdominal pain. Complete obstruction results in moderate-to-severe signs of pain. If the bladder or urethra ruptures, signs of pain subside and are replaced by depression, anorexia, abdominal distention, or enlargement of the penis and/or prepuce.[3] Equine calculi are mainly composed of calcium carbonate and 90% of them are yellow-green in colour, spiculated, and fragment easily. Ten percent are gray-white, smooth, difficult to fragment, and contain phosphate in addition to calcium carbonate.[3]

Renal calculi and ureteroliths can be unilateral or bilateral. Renal calculi usually occur at the level of the renal pelvis;[8] they can lead to hydronephrosis or pass down into the ureters causing blockage.[8] Unlike humans, colic is rarely a clinical sign.[8] The presence of damaged tissue acting as a nidus is a prerequisite for calculus formation. Bilateral obstruction of the upper urinary tract leads to chronic renal failure. In such cases, weight loss, polyuria, or poor performance are the most common clinical signs. Unilateral obstruction is very challenging to diagnose because signs are mild-to-nonexistent,[8] unless the calculus can be detected in the ureters. Renal or ureteral calculi are usually associated with microscopic hematuria, unless they are passed to the bladder or urethra.[8] Occasionally, they can be associated with intermittent or persistent hematuria.[8] If azotemia is not present, treatment of unilateral obstruction consists of removal of the affected kidney and ureter. If available, electrohydraulic lithotripsy is the preferred technique for ureteral calculi removal.[8]

Cystolithiasis is the most common form of urolithiasis in the horse. Hematuria following exercise is a common clinical sign; other signs are pollakiuria, stranguria, urine incontinence, or dysuria. Diagnosis is achieved by transrectal examination, ultrasonography, and endoscopy. Evaluation of horses with cystolithiasis should include urinalysis, culture, and sensitivity because it can be associated with urinary tract infection.[8]

Treatment is by surgical removal of the calculus and postoperative systemic antibiotics. Cystotomy, perineal urethrotomy after fragmentation using a lithotriptor or, in mares, urethral sphincterotomy have been advocated.[8] In mares, manual distention of the urethra after sedation and local or epidural anesthesia, allows several fingers or a small hand to pass into the bladder and retrieve the calculus with or without fragmentation.[8] Changing from legume to grass or oat hay is recommended to reduce calcium intake and prevent recurrence.[3,8] Administration of a urine acidifier (ammonium chloride 50-200 mg/kg/day PO or ammonium sulfate 200-300 mg/kg/day PO) is unpalatable and must be administered 2-3 times daily to be effective.[8] A more practical treatment is the daily addition of 60-120 g of salt to the feed to increase water intake.[8]

Urethral calculus is primarily a problem for male horses. It is usually caused by a small cystolith that passes from the bladder and lodges in the urethra, often at the level of the ischial arch in the area of natural urethra narrowing.[3] Affected horses present with signs of colic, frequent posturing to urinate, extension of the penis, dribbling of small amounts of urine, and sometimes blood at the end of the urethra. Unresolved complete obstruction can be followed by urinary bladder or urethral rupture. Diagnosis is based on clinical signs and transrectal palpation findings of a distended bladder and a pulsating urethra. In suspected cases, a urinary catheter can be passed to empty the bladder, followed by repeat transrectal examinations. Urethroscopy and careful palpation of the penis for a firm mass may also be diagnostic.[3]

A calculus lodged at the level of the ischial arch can be removed by perineal urethrotomy, which can be left to heal

by second intention. A calculus lodged in the distal urethra can be removed by gentle transurethral crushing with the hand or forceps.[3] Removal of a calculus lodged distal to the ischial arch and not palpable in the distal portion of the penis is achieved under general anesthesia with the horse in dorsal recumbency.

## Summary

Polyuria/polydipsia and hematuria are common presenting complaints for urinary tract disease in the horse. Accurate history, a thorough physical examination and work-up are very important. Objective confirmation of PU/PD should be employed before further investigation. The most common causes of PU/PD are psychogenic polydipsia and Cushing's syndrome. Hematuria should be differentiated from pigmenturia before further diagnostics are performed. The main diagnostic procedures for hematuria are urinalysis, endoscopy, and ultrasonography. Treatment of PU/PD or hematuria depends on the underlying cause and the prognosis varies with causation.

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