URINARY TRACT DISORDERS IN HORSES-
Advances in Diagnostics and Treatments

Thomas J. Divers, DVM, DACVIM, DAVECC
College of Veterinary Medicine
Cornell University, Ithaca, New York USA

Introduction- Drs. Lisle George and Robert Whitlock in 1976 during my residency encouraged me to develop and interest in studying urinary tract diseases in large animals. I am grateful to him for encouraging me to do so. In these notes, I will give a brief overview of equine urinary tract disorders pointing out some of the discoveries or observations made over the last 45 years.

Acute Renal failure/ Acute Kidney Injury

In most of the recent literature the term acute renal failure (ARF) has been replaced by acute kidney injury (AKI). One commonly used definition of AKI is an acute increase in serum creatinine of 0.3 mg/dl or greater and oliguria. *The switch in terms from ARF to AKI is semantic and in my mind has not improved our understanding or management of the disease.* Uremia is the clinical condition caused by renal failure and accumulation of harmful products in the blood associated with decline in kidney function.

AKI leading to renal failure and uremia can result from either intrinsic (kidney) disease/dysfunction or obstruction/rupture of the urinary tract. Acute renal failure is more common in large animals than chronic renal failure (CRF) and we can often reverse the disease process in ARF. Intrinsic causes of acute renal failure (ARF/AKI) include those disorders that cause significant functional decreases in glomerular filtration rate (GFR), mostly due to abnormal glomerular filtration pressures (common with septic shock), or morphologic nephron damage (common with nephrotoxins) or both. Effective glomerular filtration is mostly determined by the following factors:

1. Perfusion pressure at the glomerulus, which is a result of afferent renal artery blood flow along with pressure balances between the afferent and efferent arterioles.
2. Filtration efficiency of the glomerulus.

In septic, acute heart failure or hemorrhagic shock patients, ischemia is often blamed as the triggering cause for the ARF. Reduced renal blood flow (RBF) in those patients has been attributed to renal vasoconstriction secondary to systemic hypotension. However, ARF/AKI has been shown to also occur in the setting of a hyperdynamic circulation with increased RBF (*remember RBF does not equate to GFR*). In some cases of ARF the decreased GFR may be more a result of inappropriate dilation of efferent arteriole or renal vascular endothelial dysfunction and renal hyperemia may exist in those cases. I mention this because I believe it helps explain why fluid therapy, although the cornerstone of treatment for nephrotoxic renal failure, may not be a successful treatment for sepsis/shock induced ARF and patients may continue to be oliguric/anuric in spite of systemic normovolemia and normal renal blood flow. In addition, no single drug treatment is consistently effective in treating those oliguric/anuric cases; the renal pathophysiology in those cases may just be too complex for any drug to work consistently. *In many cases of sepsis/septic shock related ARF in horses, there may be minimal morphologic change on light microscopy of the kidney in spite of life-threatening functional changes!* Some of the more common diseases associated with this type of ARF in horses are; generalized sepsis (all ages but especially foals), acute severe diarrhea, life threatening hemorrhage or hemolysis and anaphylaxis.

I believe it also important to remember that in many cases of intrinsic ARF, especially those with tubular necrosis, *not every nephron is affected equally*; some nephrons may be

nonfunctional and if the basement membrane is lost they remain nonfunctional, other nephrons may be dysfunctional but recover while others may be relatively unaffected and are responsible for providing most of the renal function (hyperdynamic) during the acute phase of the renal failure. The most common site for nephron damage in horses with ARF is the proximal tubule; acute glomerulopathies are rare in horses! The tubular damage causing AKI may be from necrosis or apoptosis that may result from abnormalities in renal and peritubular blood flow, toxin accumulation, oxidative damages and tubular cast causing individual nephron obstruction. The most common toxic disorders causing tubular necrosis (ATN) are nephrotoxic drugs (e.g.. aminoglycosides, tetracycline (not minocycline or doxycycline), bisphosphonates and myoglobinuric or hemoglobinuric nephrosis. The later two disorders may also have a substantial vasomotor component to the disease. Although morbidity and mortality can be high in ATN cases, tubular injury frequently is reversible, depending on whether necrotic cells and intratubular casts are removed and sufficient renal tubular cell basement membrane remains intact. If renal cortical necrosis occurs, the prognosis is grim!

Lastly, separating prerenal azotemia from intrinsic renal disease (AKI) with azotemia is not always easy and both may be present in the same patient. Urine specific gravity and BUN:Cr ratios (both high with pre-renal azotemia), enzymuria (rarely used) and serum and urine electrolyte finding are helpful in sorting the two out. Cytologic examination of the urine can also be helpful although due to the high pH of herbivore urine, casts are not commonly seen in the urine even with tubular necrosis. . Blood values for Symmetric Dimethylarginine (SDMA) are reported in normal horses(<14 µg/dL) but this assay rarely has any practical advantage over serum creatinine as a measure of GFR and in house testing may not be available in many equine clinics. SDMA testing may be of clinical value for evaluating GFR in certain heavily muscled Quarter Horses or Warmbloods that can normally have serum creatinine values slightly outside the normal reference range. SDMA results in those horses can be helpful for purchase exams or if those horses are being considered for treatment with a nephrotoxic drug.

Key Laboratory Findings in Horses with Acute Renal Failure

- Dysfunction in two-thirds or more of nephrons will result in failure to eliminate urine waste products including urea and creatinine and inability to properly dilute or concentrate urine (1.007-1.015= isosthenuria). I*n horses with peracute renal failure the urine specific gravity may be up to 1.022 for a brief time before dropping into the classic isosthenuria range.*
- Electrolyte abnormalities are common including: hypochloremia and hyponatremia. This is likely a result of decreased tubular resorption of these electrolytes caused by tubular damage, high flow rates in the more functional nephrons and neuroendocrine influences. The hyponatremia can be dramatic at times and correction *might* need to be gradual if the sodium is < 125meq/l to prevent encephalopathy, although I have rarely, if ever, seen encephalopathy develop in those cases following rapid correction of severe hyponatremia.
- Serum potassium concentration is variable as decreases in feed intake (due to azotemia or the predisposing disease) in herbivores may offset the decline in potassium renal excretion resulting from the acute renal failure and decreased GFR. Marked hyperkalemia (>6.0 mEq/l) in ARF *might* indicate that the renal failure is oliguric or anuric or at least not just prerenal azotemia! Additionally and after treating many horses with acute colitis, some of which have hyperkalemia on presentation, I have noticed that if the hyperkalemia does not resolve after a few hours of aggressive fluid therapy this suggest that the horse has ARF and not “just” pre-renal azotemia.
- Ultrasound findings of the kidneys are often unremarkable with AKI/ARF unless obstruction is the cause. If there is marked perirenal edema or the kidneys are very large on ultrasound examination in horses with ARF the prognosis for recovery should be lowered. With

myoglobinuria nephrosis, the urine will be discolored and positive for occult blood. Proteinuria is generally minimal in most cases unless it is a rare case of acute glomerulopathy. Bacteriuria is uncommon in horses with ARF since acute septic nephritis is rare in adult horses; foals may have acute septic nephritis due to bacteremia (i.e., *Actinobacillus* spp.). Horses with ARF caused by L*eptospira sp*. (usually *L. Pomona*) infection may have pyuria without bacteriuria noted on routine cytologic urine exam.

Clinical Signs of Acute Renal Failure:

- Often the clinical signs are more associated with the predisposing disease (e.g., diarrhea, septic shock) than the renal failure per se.
- Uremia causes anorexia and depression and on occasion uremic encephalopathy, (blood ammonia is elevated). Colic signs are present in some horses with ARF likely in association with a rapid swelling of the kidney; this is not a good finding as these cases are likely to have oliguric or anuric ARF.
- Clinical findings due to obstructive renal failure or rupture of the urinary tract also include stranguria, abdominal distention, colic, and dramatic ultrasound findings.
- Clinical findings of acute septic nephritis would include fever and in some cases, enlarged kidneys are noted on ultrasound examination. Horses with ARF caused by Leptospira spp. infection are often febrile for at least a couple of days before signs of ARF (depression, anorexia) occur.

Treatment of Acute Renal Failure:

- Treat the initiating disease.
- Correct prerenal factors (dehydration).
- Determine if the patient is anuric, oliguric, or polyuric. If the patient is polyuric, the prognosis is better and management is much easier but it does not always mean that treatment will be successful in restoring renal function. For the polyuric ARF patient, fluid therapy is the “gold standard” treatment. The hope is that fluid therapy re-establishes renal blood flow and glomerular pressures, flushes out tubular debris that will further improves glomerular function, and corrects electrolyte abnormalities. Although intravenously administered saline, including hypertonic, therapy has often been a successful therapy for equine ARF, there is some concern about using large volumes of 0.9% saline fluids for several days in horses with ARF. The administration of large volumes of high chloride fluids can cause hyperchloremia and metabolic acidosis and persistent hyperchloremia could lead to renal vasoconstriction and a consequent reduction in glomerular filtration. In horses with ARF, hypochloremia is common so the avoidance of high chloride fluids may not be as important in horses with ARF as in humans. The most recent guideline for fluid treatment of humans with ARF is “saline remains the preferred solution for volume resuscitation, though chloride concentrations should be monitored”(J Clin Med. 2020 Jun;). Intravenously administered starches ie, Hetastarch, have been associated with a higher risk of ARF in humans with septic shock and although this has not been confirmed in horses it is likely best to avoid synthetic starches in horses with ARF as they could cause an osmotic nephrosis.

If the patient is oliguric or even worse, anuric, after correction of prerenal factors, the prognosis is guarded or poor and fluid therapy alone is certainly not the “gold” standard. In fact, overhydration will lead to an earlier demise! Several vasopressor drugs have been used to try to improve urine production in patients with oliguric or anuric AKI, mostly by adjusting intra-renal hemodynamics and tubular function, although *none* have reach reached statistical significance in meta-analysis reviews! Norepinephrine has been my “favorite” vasopressor drug for foals with both oliguric ARF and hypotension: if the foal is oliguric/anuric but normotensive, fenoldopam

(0.04 ug/kg/min) might be effective in increasing urine production. In adult horses with oliguric/anuric ARF that have not responded to fluid therapy, I have often administer low dose dopamine and have then used vasopressin if the dopamine therapy does not increase urine volume. I am well aware of the meta-analysis studies showing that low dose dopamine is not thought to be efficacious in humans with ARF but, the number of and sensitivity of dopaminergic receptors that affect renal vasodilation and urine production appear to be different between humans and horses as low dose dopamine and fenoldopam will increased RBF (dopamine) and urine output (both drugs) in adult horses and foals. There were no systemic effects (HR, SBP etc.) observed in healthy horses and foals with either drug at the low dose. I have had some *luck* using dopamine in adult horses with oliguric/anuric ARF. In equine practice, we do not have good, readily available options for dialysis and if a conversion from oliguria/anuria to polyuria does not occur rather quickly (24-48 hrs.) then the prognosis becomes grave. For foals with hypotension and oliguric/anuric ARF that are hypotensive in spite of adequate cardiac preload, I have often combined dobutamine with dopamine or fenoldopam. Diuretics should be used with caution in ARF as there is no evidence they will improve outcome in other species with ARF. I will sometimes try one or more doses of furosemide in refractory oliguric cases out of frustration when nothing else is increasing urine output.

Infectious causes of ARF are not common; but in horses leptospirosis should be considered. There have even been outbreaks of fever and ARF in weanlings and foals due to *Leptospira pomona* (*kennewicki*). Urinalysis often has increased WBCs but no bacteria seen on routine examination. Horses with *Leptospira* ARF will have high level of cross-reacting antibodies when tested as infection and fever precede the onset of renal failure by several days. Beta lactam or fluoroquinolones are commonly used antibiotics for treating leptospirosis. Prognosis for recovery is good if appropriate treatment provided.

Note on Prognosis: Intrinsic acute renal failure is relatively common in horses and with the above therapy our success rate has been approximately 60% for recovery and return to health. Most cases either have: complete clinical recovery (the 60%), 30% do not respond and are euthanized and approximately 10% improve but are not clinically normal due to permanent loss of >50% of nephrons causing signs of chronic renal failure (weight loss, polyuria/polydipsia, etc.). With drug-induced or myoglobin nephrotoxicity it often takes 3 days of treatment before there is a decline in serum creatinine, so don't panic when the creatinine does not change after 1-2 days of treatment.

Prevention of drug induced ARF is very important!!! - maintain hydration during aminoglycoside or tetracycline therapy, use the right drug dose and interval, provide intermittent diuresis if needed and closely monitor serum creatinine!. If the serum creatinine increases 0.3 mg/dl or more it might be time for either: a drug change, sodium chloride diuresis or if the nehrotoxic drug must be continued the administration interval should be prolonged and diuresis provided. I sometimes check trough levels of aminoglycosides to determine if toxic levels are occurring. Peak levels would be checked also to determine if we are administering enough (therapeutic levels) of the drug. The hydration status of a patient cannot be overemphasized as most of the nephrotoxic drug related ARF cases I have seen were not because a drug was overdosed but occurred because of routine drug dosing in a dehydrated patient. Although NSAIDs very rarely cause ARF by their selves, they can predispose to ARF when aminoglycosides, tetracycline, bisphosphonates are being used.

Chronic renal failure due to either glomerulonephritis or interstitial fibrosis is less common in horses than is acute renal failure. Weight loss is the most common clinical sign of CRF although polyuria and excessive dental tartar might be noted. Peripheral edema (due to hypoproteinemia) is often seen with glomerulonephritis cases. Glomerulonephritis cases may occur following streptococcal infection, intravenous allogenic stem cell administration and the ensuing immune response but in most cases the exact predisposing cause cannot be determined. Bilateral nephroliths (mostly due to NSAID therapy in dehydrated horses) are relatively common in performance horses and the stones may cause chronic renal failure due to chronic intermittent obstruction. The first case series on bilateral nephrolithiasis/ureterolithiasis causing renal failure was reported by Dr. Ehnen (VMD) et al. and historical investigative work along with literature review of a similar syndrome in people was convincing that administration of NSAIDs to dehydrated horses was likely responsible for the syndrome. It was hypothesized that renal crest necrosis occurs in association with concurrent dehydration and NSAID administration and then the necrosing renal papillae became mineralized (mostly calcium carbonate). It may be that a small percentage of horses with renal crest necrosis are predisposed to calculi formation due to dietary or more likely inherent risk factors such as abnormal urine crystal solubility factors causing mineral precipitation or a lack of inhibitors of crystal formation.

Renal dysplasia is also a relatively common condition in the horse/donkey and has been reported in neonates, foals, and young adult horses with no apparent predilection for breed or gender. Most previously reported cases had clinical and clinicopathologic findings consistent with renal failure which included depression, lethargy, and ill-thrift with associated azotemia and electrolyte abnormalities. Onset of clinical signs occurred as late as 7 years of age.

Treatment for CRF is generally symptomatic except for rare cases of chronic septic pyelonephritis in which case non-nephrotoxic antimicrobials are used. If the causative bacteria have a high antimicrobial resistance, nephrotoxic drugs like amikacin could, as last resort, be used but would require dose interval adjustment and close serum concentration monitoring. High energy diets with omega-3 fat, moderate protein, low calcium are generally recommended for CRF along with avoidance of dehydration and toxic drugs. Horses with ureterolithiasis/nephrolithiasis can sometimes be salvaged by using either shock wave lithotripsy or nephrotomy for stone removal. Unfortunately, most horses with this condition are in chronic renal failure with asymmetrically dilated or fibrotic kidneys before the initial veterinary examination (ususally for for weight loss or poor performance). Horses, unlike humans, rarely show obvious colic signs with the ureteral obstruction. This is unfortunate, as early removal of the stones would improve prognosis.

Ureteral disorders in foals. Ureteral defects in young foals include ectopic ureter, ureteral tears and hydroureters. Ectopic ureter(s) is most commonly diagnosed in fillies and causes urine scalding as the predominant clinical sign. Diagnosis of ectopic ureter can be confirmed by cystoscopic examination looking for ureteral openings into the bladder and the normal pulsile flow of urine from the ureters. Contrast imaging studies may also confirm the diagnosis. Treatments of ectopic ureter include: surgical transposition of the ectopic ureter into the bladder (Ureteroneocystostomy), a recently described cystoscopically guided ureteral ostioplasty or surgical removal of the ureter and kidney on the affected side. Although nephrectomy as part of the treatment seems unnecessary, in cases with pyelonephritis secondary to the duration of the ectopic ureter, this is often the most practical option. Assuming the correct kidney is removed (!) and the other kidney is normal as expected the serum creatinine increases approximately 0.7 mg/dl immediately following surgery but due to

hypertrophy of the opposite kidney the eventual creatinine will only be 0.2-0.3 mg/dl above the expected value with two functional healthy kidneys. Ureteral (tears) causing azotemia and accumulation of urine in the retroperitoneal space occur in neonatal foals and have identical clinical pathologic findings as found with ruptured bladder. The cause of the defect(s) is unknown but in approximately 40% of the cases the defect is bilateral. Ruptured ureter(s) initially causes accumulation of urine in the retroperitoneal space, sometimes causing stranguria and vaginal mucosal prolapse in fillies or the retroperitoneal membrane may rupture causing "classic" uroperitoneum. These can be surgically repaired but ureteral catheters are often needed for several days post-operative to prevent ureteral leakage. A third ureteral problem in neonatal foals is inhibition of ureteral urine flow into the bladder. It is unclear if this is a ureteral motility dysfunction (most likely) or an anatomic anomaly at the ureteral-vesicular junction. This syndrome causes azotemia, hyponatremia and dilated ureter(s) and kidney seen on ultrasound examination. These are very difficult to repair although creating a new ureteral opening into the bladder via cystoscopy and laser surgery or a ureteroneocystostomy might work? I would caution about making this diagnosis based upon ultrasound exam alone in a recumbent foal on intravenous fluids as ureteral distention can be seen without clinical relevance in some recumbent foals receiving intravenous fluid therapy.

Two other neonatal foal urinary conditions that I would like to mention are hypercreatinemia in foals born to mares with placentitis. A 2014 publication reported that serum creatinine concentration was higher in foals born from mares with grossly abnormal placentas than in foals born from mares with grossly normal placentas (P = 0.029). The serum creatinine may vary from 3.5 mg/dl up to a rare high of 15 mg/dl with the BUN less relative elevation (BUN is a smaller MW and diffuses across the placenta easier than creatinine). The serum creatinine will generally return to normal range within 48 hours without fluid therapy if there are no medical problems in the foal and it is nursing well. These foals may be at higher risk for having in-utero stress and associated muscle wasting which may also increase serum creatinine at birth. Affected foals are at increased risk of perinatal asphyxia . The other unusual urinary condition in foals that I would like to mention is a detrusor/sphincter dyssynergy in 1-5 day old foals. Although unproven, this disorder *may* be due to a central nervous system injury (birth hypoxia with "dummy bladder") that disrupts the micturition pathways and particularly the parasympathetic relaxation of the urethral sphincter. Placing a urinary catheter to prevent bladder rupture and supportive medical care for neonatal encephalopathy generally results in normalcy of urination within 2-7 days. Phenazopyridine (4-6 mg/kg PO q 8-12) is administered to these foals for 3 days and is often used in foals with indwelling urinary catheters to decrease bladder discomfort. I admit urinary catheters might not be easy to place in fillies or maintained in the bladder of colts but they are often necessary for proper patient care in disorders such as this and in the management of critically ill foals in order to prevent ruptured bladder or measure urine output in hypotensive foal or in foals with ARF. In hypotensive septic foals, measuring urine output has been more valuable to me in the fluid management of the foal than trying to monitor central venous pressure.

Bladder Dysfunction

Urinary tract infections and incontinence are not nearly as common in horses as in small animals but principles for both diagnosis and treatment are similar between the species. When a urinary tract infection is suspected, it is important to request a colony count on the catheterized or mid-stream voided urine sample. The colony count will help determine the most

significant organism if more than one is cultured. Geldings and stallions may develop urinary incontinence due to sabulous calculi forming in the bladder and damage to the detrusor muscle; the urine is initially sterile but will become infected following catheterization. Brood mares may develop incontinence, cystitis, sabulous cystitis or pyelonephritis following urethral injury at foaling and secondary lower urinary tract infections.

Sabulous cystitis, is a common condition in middle-aged male horses. Clinical signs include dribbling urine for a prolonged period with contact dermatitis in many cases, but otherwise the horses are in good health and seemingly undisturbed by the incontinence. Gait deficits, abnormal anal and tail tone or muscle atrophy are rare, unless it is a horse that has survived EHV-1 myelitis or has polyneuritis equi. Rectal examination of affected horses reveals a very large urinary bladder and following per rectum manual expression only a small amount of urine is voided. Ultrasound examination per rectum shows large amounts of hyperechoic material in the ventrum of the bladder. Flushing the debris from the bladder with a dilute DMSO solution followed by saline rinse will temporarily resole the urine dribbling, but signs will recur due to the chronic damage to the detrusor muscle and the chronic infection that usually develops following catheterization. Antibiotics will be needed long term. The cause of this syndrome is unknown but my theory has been that chronic back pain in male horses can lead to build up of calcium carbonate sludge in the bladder and eventually the detrusor muscle becomes damaged from the sludge. Nerve supply to the bladder muscle, hypogastric and pelvic splanchnic nerves, were normal in two cases that Dr. John Cumming (neuroanatomist, neuropathologist) examined in the 1990s. Horses can live for years with this condition but complete recovery is rare.

Rupture of the lower urinary tract- Ruptured bladder or urachus causing uroperitoneum is a common problem in foals; bladder rupture may also infrequently occur in post foaling mares. Although most bladder ruptures occur in foals at birth, they infrequently occur in older foals possibly related to urachal infections and necrosis of the bladder. Recumbent neonatal foals may also rupture their bladder unless an indwelling urinary catheter is in place. Lifting the recumbent foals when they have a distended bladder may cause the rupture. Rupture of the bladder at the apex occasionally occurs in older foals two to six weeks of age, resulting in uroperitoneum. Diagnosis of uroperitoneum is based upon clinical signs (stranguria, pollakiuria, abdominal distention and depression), elevated serum creatinine, and frequently characteristic electrolyte abnormalities (hyponatremia, hypochloremia and hyperkalemia) and ultrasound findings using a sector or linear probe. It *may* be necessary to compare the peritoneal fluid to serum creatinine ratio (>2) to confirm the diagnosis. Surgical repair of the defect should be performed after medical treatment to correct electrolyte abnormalities and hypotension. If the potassium is markedly elevated and/or the abdomen severely distended with urine, I recommend placing a catheter in the abdomen to provide adequate drainage prior to the foal being anesthetized. The drain should not be removed until immediately prior to surgery as earlier removal will result in subcutaneous accumulation of urine. Correction of hyponatremia and/or hyperkalemia should be accomplished with limited volume fluid therapy. Hyperkalemia and hyponatremia can be corrected by treating the foal with equal volume of 5% dextrose mixed with 2.5 % sodium bicarbonate; if potassium is normal 0.9 % saline can be slowly administered instead. If hyperkalemia is severe (>8.0 mEq/L) and/or there are cardiac changes on an ECG (prolonged PR interval, spiked T wave, flat or absent P wave), calcium borogluconate (30 ml) should be administered with the first ½ liter of fluids (e.g., 0.9% sodium chloride). If hyponatremia is severe (<115 mEq/L), the sodium should ideally be corrected slowly 2-3 mEq/L/hour, but this is not always feasible in equine practice and I am not sure how important it is. The urachus, umbilical vein and/or umbilical arteries are frequently infected in neonatal foals and these structures should all be routinely evaluated by ultrasound

examination. Large swelling at the urachus may be a result of hematoma, infection or rupture of the urachus with leakage of urine subcutaneously. If there is subcutaneous accumulation of urine, mild hyponatremia, hypochloremia, and azotemia may exist. Surgical removal of the "leaking" urachus is sometimes required although many can often be treated medically with indwelling catheter, antibiotics, and hot packing the affected area. Patent urachus is common in neonatal foals and may be noted within the first two days in otherwise healthy foals or may develop later, more commonly in foals with other medical illnesses. The patent urachus can be treated several times daily with topical Nolvasan or carefully cauterized by an experience person three times daily with silver nitrate. Systemic antibiotics are often recommended for foals with patent urachus. If the urachus is believed infected surgical removal should be considered. Although more common in calves, foals with chronic urachal absess may occasionally present with stranguria because the bladder is adhered to the infected urachus and body wall. Surgical removal of the infected urachus and affected portion of bladder wall is recommended.

Urethral and Bladder Stones:

Clinical signs of cystic calculi include dysuria and hematuria following exercise. Following acute urethral obstruction, stranguria, anuria, colic and signs of distress occur. Diagnosis is based upon clinical examination and sonogram or endoscopy of the urethra and bladder. Cystic calculi are palpated during rectal examination, concentrating on the pelvic floor or brim of the pelvis area. Palpation of the perineal area in horses with urethral obstruction wmight detect pulsating contractions of the urethral muscles and in some cases the stone can be felt along with a dilated urethra just proximal to the stone. The kidneys should be scanned in horses with cystic or urethral stones as these same horses may have nephroliths which increases the risk of future bladder/urethral stones. There have been many methods for removing bladder calculi and the most recent one is the use of a laparoscopic specimen retrieval pouch to allow fragmentation and removal of the stones. On rare occasion, the stone will damage the trigone area of the bladder causing partial ureteral obstruction, hydronephrosis and chronic renal failure. .

Other Causes of Hematuria

Geldings or stallions with hematuria at the end of urination most often have an ("blow out" of the dorsoproximal urethral mucosa); I have not aware of this syndrome occurring in other species. This "tear" in the pelvic, dorsal urethral mucosa causes chronic hematuria (at the end of urination) when the bulbourethral muscles contract and forces blood through the tear into the urethral lumen. Surgical procedures either perineal urethrotomy or corpus spongiotomy which involves a perineal approach as for a PU but not actually cutting into the urethra has been described. Following the procedure, the bulbourethral muscle pressure is decreased during urination diminishing the urethral bleeding which allows healing of the defect. Hemorrhagic cystitis may rarely occur from NSAID administration, bladder neoplasia or idiopathic causes. The idiopathic and NSAID induce cystitis typical cause multifocal or diffuse lesions seen on cystoscopy while neoplasia is more commonly seen as a mass. Endoscopic examination of the bladder is indicated in those cases to rule out neoplasia and to visualize: (1) The hemorrhagic lesions in the bladder and (2) to confirm that urine entering the bladder from the ureters is normal color.

Occasionally adult horses develop idiopathic renal hematuria and it may be so persistent that the kidney is removed as a last resort. Renal carcinoma should always be considered in older horses with renal origin hematuria. Theoretically, these could be removed successfully but in real life most have already metastasized before the diagnosis is made.

Psychogenic PD/PU is common in show and pleasure horses that are mostly stall

confined and without constant access to hay. It may also occur in those horses following introduction of new horses to the barn, recent shipping, following extreme hot weather, other stressors or excessive ingestion of salt. Serum electrolytes generally remain normal and urine is hyposthenuric (specific gravity < 1.008). If there is no clinical or laboratory evidence of renal disease, treatment may include: correcting psychological or dietary predisposing causes and *gradually* limiting water intake until the horse becomes very mildly dehydrated. These cases may require long periods of careful water intake adjustments due to renal medullary washout and inability to properly concentrate urine in some cases. Diabetes insipidus (DI), either central (lack of ADH) or nephrogenic (tubules do not respond appropriately to ADH) should also be ruled out in these cases. Horses with central DI often are hypernatremic if dehydrated and are hyposthenuria or isosthenuric in spite of the dehydration. Response to administration of ADH (vasopressin) can help confirm the diagnosis of central DI.

Renal tubular acidosis is rarely diagnosed in horses. There is no proven breed or sex predisposition although I have a *suspicion* that persistent or recurring RTA it is more common in the Friesian breed. Transient cases of RTA have been diagnosed following a primary renal insult. Cases are mostly mature horses with depression (due to metabolic acidosis), weight loss and partial anorexia as clinical signs. Hyperchloremic metabolic acidosis is the classic laboratory finding. Severe hypokalemia is present in some cases. Often the serum and urine findings do not allow type classification of the RTA. Treatment is bicarbonate (IV or oral) and prior correction of hypokalemia if it exist.

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