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Chronic kidney disease in horses

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Chronic kidney disease (CKD) in the horse may be divided by clinical and pathologic findings into two broad categories: primary glomerular disease (glomerulonephritis [GN]) and primary tubulointerstitial disease (chronic interstitial nephritis [CIN]). However, pathology in one portion of the nephron usually leads to altered function and eventual pathology in the entire nephron such that CRF is an irreversible disease process characterized by a progressive decline in glomerular filtration rate (GFR). More importantly, the rate of decline in GFR is variable in horses with CRF making the short-term (months to a few years) prognosis guarded to favorable while the long-term prognosis remains poor.

Clinical signs and laboratory findings: The most common clinical sign observed in horses with CKD is weight loss. A small plaque of ventral edema, usually between the forelimbs, is another frequent finding. Moderate polyuria and polydipsia (PU/PD) are also usually present at some stage of the disease process. Accumulation of dental tartar, especially on the incisors and canine teeth and oral ulcers are other findings that may be detected in horses with CKD. Decreased performance may be an early complaint in competitive horses while growth is generally stunted in young horses with renal hypoplasia, dysplasia, or polycystic kidney disease.

Most horses with clinical signs of CKD have moderate to severe azotemia (creatinine concentration [Cr] usually 5 mg/dL [440 μmol/L] or greater at initial evaluation) unless early CKD is detected during evaluation for another clinical problem. Mild hypomagnesemia and hypochloremia are sometimes found in horses with CKD. Hypercalcemia, with serum concentrations sometimes exceeding 20 mg/dL (5 mmol/L), appears to be a laboratory finding that is unique to horses with CKD. The magnitude of hypercalcemia is dependent on calcium content of the diet. Acid-base balance usually remains normal until CKD becomes advanced but metabolic acidosis may be found in horses with end stage disease. Many horses with CKD are moderately anemic (packed cell volume 25-30%) as a consequence of decreased erythropoietin production. Horses with GN may have hypoalbuminemia and hypoproteinemia while horses with advanced CKD of any cause may also have mild hypoproteinemia associated with intestinal ulceration.

Urine collected from horses with CKD is relatively devoid of normal mucus and crystals making samples transparent. A hallmark of CKD is urine specific gravity in the isosthenuric range (1.008 to 1.012), although heavy proteinuria in an occasional horse with GN may produce values up to 1.020. Quantification of urine protein concentration is required to accurately assess proteinuria. Horses with CIN usually do not have significant proteinuria.

Diagnosis of chronic renal failure: A diagnosis of CKD is most commonly made in horses with azotemia and isosthenuria that present with a complaint of weight loss or decreased performance. Detection of hypercalcemia also strongly supports CKD.
Determining the inciting cause of CKD can be difficult because the disease has often advanced to end stage kidney disease when horses are initially presented for evaluation. Detection of moderate to heavy proteinuria without hematuria provides support for primary glomerular disease. Horses with pyelonephritis, as well as those with ureteral calculi, often have enlarged ureters that can be palpated as they course through the retroperitoneal space. Although kidneys of horses with CKD are often small with an irregular surface, these changes are not always apparent on palpation of the caudal pole of the left kidney. Ultrasonographic imaging is useful for evaluating kidney size and echogenicity and may reveal fluid distention (hydronephrosis, pyelonephritis, or polycystic disease) and presence of nephroliths. Horses with significant renal parenchymal damage and fibrosis usually have increased echogenicity of renal tissue that may be similar or even greater than that of the spleen.

**Management of chronic renal failure:** Treatment of horses with CKD is most likely to produce improved renal function if there is an acute, reversible component exacerbating CKD (acute on chronic syndrome). If an acute component is detected, it should be corrected rapidly with the goal of minimizing further loss of functional nephrons. Treatment of horses with stable CKD consists of supportive care: providing sufficient fluids (water), electrolytes, and nutritional support. In addition to Cr, serum electrolyte concentrations and acid-base balance should be measured regularly. Although no adverse effects of hypercalcemia in horses with CKD have been documented, decreasing calcium intake (replacing alfalfa or other legume hays with grass hay) may result in a return of serum calcium concentration to the normal range. NSAIDs are best avoided in horses with CKD.

As CKD progresses partial anorexia and lethargy lead to more rapid loss of body condition. Thus, nutritional management aimed at maintaining body condition is probably the most important aspect of supportive care of horses with CKD. Increasing carbohydrate (grain) intake and adding fat to the diet are recommendations to increase caloric intake. Fat can be added by feeding a vegetable oil (up to 500 mL per day) or a commercial fat supplement. Fat supplements rich in omega-3 fatty acids are likely preferable over those rich in omega-6 fatty acids as diets rich in the former have been shown to delay progression of CKD in small animals. Over the past couple of decades restricting dietary protein intake by human and veterinary patients with CKD was thought to have beneficial effects; however, the current recommendation is to provide an adequate amount of dietary protein and energy to meet or slightly exceed predicted requirements while maintaining a neutral nitrogen balance. Finally, an important but often overlooked aspect of nutritional management of horses with CKD is provision of a highly palatable diet. Feeding smaller meals more frequently and varying the diet (e.g., offering various types of concentrate feeds as appetite may vary from day to day) are helpful methods to increase food intake.

The progressive loss of nephron function that is characteristic of CKD precludes successful long-term treatment in horses. However, many horses with early CKD may be able to continue in performance or live as a pet for months to a few years. As long as Cr remains <5.0 mg/dL [440 μmol/L], affected horses often maintain a good attitude, appetite, and body condition. However, once Cr exceeds 5.0 mg/dL [440 μmol/L], the rate of progression of CKD appears to accelerate and signs of uremia (anorexia, poor hair coat, and loss of body condition) become more apparent over a period of a few weeks to months.