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Fanconi SYNDROME

Cases of acquired Fanconi syndrome are on the rise in parts of New Zealand. **Julie Tomlinson** of Gribbles Veterinary, Auckland, outlines the presenting signs to be aware of.

DISEASES OF THE proximal tubule are fairly rare in veterinary medicine. Primary renal glucosuria, proximal renal tubular acidosis and Fanconi syndrome have been described. Primary renal glucosuria may occur as a sole reabsorptive defect, or may be the initial finding prior to development of overt Fanconi syndrome. Proximal renal tubular acidosis is characterised by reduced bicarbonate reabsorption. Fanconi syndrome is a more generalised loss of proximal tubular reabsorptive function, resulting in excessive loss of water and multiple solutes from the glomerular filtrate (Langlois et al., 2013). Glucose and bicarbonate are the most commonly affected, but loss of amino acids, phosphate, sodium, potassium, calcium, magnesium, uric acid, lactate, ketones, carnitine and protein can also occur. The condition can be congenital or acquired as a result of proximal tubule insult, and, if left untreated, can progress to renal failure (Jamieson and Chandler, 2001).

HEREDITARY FANCONI SYNDROME

The hereditary form is well described in Basenjis (10% incidence) but can be seen in other breeds including Norwegian Elkhounds, Cairn Terriers, Schnauzers, and Shetland Sheepdogs (Giger et al., 2015; Hostutler et al., 2004). It is associated with a mutation in the FAN1 gene with an autosomal recessive inheritance pattern (Giger et al., 2015; Johnson et al., 2012). Clinical signs typically develop in middle age (four to seven years) (Giger et al., 2015). One study of 60 dogs with idiopathic or hereditary Fanconi syndrome found that lifespan was not significantly reduced, and owners reported a good quality of life (Yearly et al., 2004).

ACQUIRED FANCONI SYNDROME

The acquired form can occur with any injury to the proximal renal tubular epithelium due to certain antibiotics, infectious agents, heavy metals or other toxins – see Table 1 (Riordan and Schaer, 2005; Hooper and Roberts, 2011; Reinert and Feldman, 2015). Cases of acquired Fanconi syndrome have also been described in dogs with copper-associated hepatitis (Langlois et al., 2013) and there is a single case report of transient Fanconi syndrome in a dog with primary hypoparathyroidism (Freeman et al., 1994).

In 2007, reports of acquired Fanconi-like syndrome in association with Chinese-origin chicken jerky treats began to occur.

From 2007 to 2015, the US Food and Drug Administration (FDA) received approximately 5,200 complaints of

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illness associated with consumption of chicken, duck or sweet potato jerky treats, most of which involved products imported from China. The reports involved more than 6,200 dogs, 26 cats and three people and included more than 1,140 canine deaths (FDA Annual Update). Cases have also been reported in Australia, Canada, Japan and Europe (Thompson et al., 2013; Igase et al., 2015; Carmichael et al., 2014; Hooijberg et al., 2015).

In 2013, an Australian paper provided a summary of 102 cases of kidney disease in dogs exposed to a Chinese-origin chicken jerky treat. The majority of the dogs affected were small or toy breeds, and six of the 102 died or were euthanased due to the illness. Follow-up data was available for 35 survivors, for which the time to resolution of clinical signs ranged from <two weeks to six months (Thompson et al., 2013). The underlying toxin has yet to be identified. Contamination of these products with illegal antibiotics (sulphonamides, tilmicosin and enrofloxacin) has been reported, although there is no evidence linking these contaminants to clinical cases of renal tubular injury (Sheridan et al., 2014).

CLINICAL FINDINGS

Polyuria and polydipsia are the most common presenting signs. Others may include dehydration, weakness, lethargy and weight loss. Dogs with the hereditary form may have a poor haircoat (Yearly et al., 2004). Laboratory abnormalities include normoglycemic glucosuria, a normal anion gap hyperchloremic metabolic acidosis, hypokalaemia, hypophosphatemia and aminoaciduria. Ketonuria, mild proteinuria and azotemia may also be found (Hostutler et al., 2004). Other clinical signs and laboratory abnormalities may depend on the underlying aetiology. For example, dogs that have consumed Chinese-origin jerky treats may exhibit GI signs (anorexia, vomiting and diarrhoea) and have a mild elevation in ALT (Thompson et al., 2013). Treatment involves elimination of the underlying aetiology (if possible), electrolyte/bicarb supplementation and supportive care. Prognosis depends on the underlying etiology, the degree of renal injury and response to treatment.

At Gribbles Veterinary, we have recently seen an increased number of acquired Fanconi syndrome cases in the Auckland and Northland regions. Clinical histories were available for several cases in which the dogs were fed chicken, duck or sweet potato jerky products. However, we do not have feedback to know whether the dogs improved after discontinuation of the jerky product. Please be aware of this condition, the hallmark finding of glucosuria with normoglycemia, the possible link to Chinese-origin jerky treats and the importance of performing a urinalysis and obtaining a diet history in sick patients. VS

TABLE 1:

Differentials for acquired Fanconi syndrome

Antibiotics	Gentamicin Cephalosporins High doses of amoxicillin Degraded tetracyclines*
Other drugs	Cisplatin Streptozotocin Chlorambucil (cat) Salicylates*
Heavy metals	Copper Lead Mercury Cadmium*
Other toxins	Chinese-origin jerky treats Melamine/Cyanuric acid Ethylene glycol
Infectious agents	Leptospirosis Pyelonephritis
Hypoparathyroidism	
Hypovitaminosis D	
Monoclonal gammopathies*	
Amyloidosis*	
Neoplasia*	

*Reported in humans

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