A five-year and seven-month-old, female neutered Chihuahua was presented to the R(D)SVS Internal Medicine service for investigation of a 2-month history of polyuria, polydipsia and excessive grooming of the vulva, with associated glucosuria, despite normal blood glucose. Dietary history consisted of commercial wet food, cooked chicken, as well as several treats, including daily dried duck and chicken-based treats. Physical examination revealed normal vital signs, moderate to severe obesity, dental disease and no other relevant abnormalities.

1) What are your differential diagnoses for glucosuria with normoglycaemia?

2) How would you evaluate this case further?

3) How would you treat and monitor this dog?
1. **Differential diagnosis for glucosuria with normoglycaemia**
   - Pyelonephritis
   - Leptospirosis
   - Lilies toxicity (cats)
   - Idiopathic / hereditary / congenital
     - Fanconi syndrome (with no other diseases or with renal dysplasia)
     - Primary renal glucosuria
   - Acquired (Fanconi-like syndrome) associated with
     - Gentamicin, ethylene glycol, lead or grapes/raisins toxicity
     - Ingestion of dry meat treats
     - Copper storage hepatopathy
     - Chemotherapy with chlorambucil (cats)
     - Hypoparathyroidism
     - Expired tetracycline exposure
   - False-positive urine dipstick reaction
     - Associated with amoxicillin, cefalexin or enrofloxacin exposure

2. **Further evaluation**
   Systolic blood pressure was moderately increased, though suspected to be anxiety-induced. Urine analysis confirmed marked glycosuria with no sediment abnormalities noticed on microscopy. Routine haematology, serum biochemistry, blood gases (as can become acidaeemic due to loss of bicarbonate), fructosamine and urine culture were unremarkable. Abdominal ultrasonography revealed hepatic hyperechogenicity and mildly reduced renal corticomedullary definition. Ultrasound-guided liver fine needle aspiration cytology was compatible with mild to moderate vacuolar hepatopathy. The following further diagnostics were unremarkable: thoracic radiography, leptospirosis testing (urine polymerase chain reaction and serum microagglutination) and symmetric dimethylarginine (SDMA). Urine amino acids quantification confirmed the presence of generalised proximal renal tubular dysfunction with marked loss of all the amino acids measured.

3. **Treatment and monitoring**
   The combination of history and diagnostic investigation performed led to a suspicion of either idiopathic Fanconi syndrome or Fanconi-like syndrome associated with chronic daily exposure of dry meat treats. Therefore, the treats were discontinued and the patient monitored throughout by means of urine analysis 2, 4 and 10 weeks later, as well as blood pressure, biochemistry, blood gases and SDMA at 4 weeks, which were stable. After five months of discontinuation of the treats, the glucosuria had resolved and no abnormalities were present on the remainder of the urine analysis, biochemistry, blood gases and SDMA. Absent glucosuria was re-confirmed three months later. A presumptive resolution of the Fanconi-like syndrome was therefore assumed, given that urine amino acids were not repeated for confirmation. This was due to the expense of the analysis associated with no planned change in management regardless of the result. The patient is due to visit the R(D)SVS Internal Medicine service for reassessment in two months’ time (11 months post-diagnosis).
Discussion

Fanconi syndrome is characterised by an excessive fractional excretion of many solutes, due to induction of a generalised proximal renal tubular dysfunction. The most readily identifiable loss is glucose, with subsequent osmotic diuresis and polyuria/polydipsia. Acquired Fanconi syndrome associated with the ingestion of dry meat treats has been reported several times, including in the UK. Most commonly, the exposure involves duck or chicken treats originating from China. Small breed dogs are overrepresented, perhaps due to the relative higher amount of treat consumption. Suspected cases in the UK have been encouraged to be reported through the Veterinary Poisons Information Service (https://vpisglobal.com/poisons/jerky-treats-case-registry/). Alongside polyuria/polydipsia, clinical signs may include lethargy, inappetence, vomiting, diarrhoea and weight loss. Together with glucosuria and aminoaciduria, clinicopathological abnormalities can comprise azotaemia, metabolic acidosis, hypokalaemia, hypophosphataemia, hyponatraemia, ketonuria and increased liver enzymes. Treatment is supportive, ranging from treat discontinuation alone, to need of hospitalisation, oral supplementation with electrolytes, manipulation of acid base status, and routine management of chronic kidney disease (CKD). The outcome is also variable, with complete recovery observed in most of the cases, though residual CKD and worsening progression, culminating in euthanasia, have been reported. Monitoring strategies should include periodic assessment of urine analysis, blood pressure, renal parameters, electrolytes, acid base status, liver function and screening for urinary tract infection, which is a risk due to the presence of glucosuria.

References


