Case 13: Primary hypoparathyroidism and renal insufficiency in a 6 month old Chow Chow

A 6 month old, male entire Chow Chow was presented for acute onset seizure activity. The dog had a two week history of pawing at the face, tremor and photophobia. Chronic polydipsia was also reported. The dog’s diet had consisted of commercial puppy food supplemented with chicken. On presentation, the dog was bright, alert and responsive. Body condition score was 4/9, at a weight of 15.4 kg. Physical examination revealed mild hyperthermia (rectal temperature 39.4°C) which resolved upon admission. Ophthalmic examination revealed bilateral lenticular cataracts. There were no other abnormalities. Doppler systolic blood pressure was 117 mmHg.

Haematology revealed a mild normocytic, normochromic, non-regenerative anaemia (PCV 0.31 l/l, ref 0.39-0.55 l/l), and a mild mature neutrophilia (17.3 x 10⁹/l, ref 3.6-12 x 10⁹/l). Serum biochemistry revealed azotaemia, with urea 9.1 mmol/l (ref 1.7-7.4 mmol/l) and creatinine 203 umol/l (ref 40-132 umol/l). There was marked hyperphosphataemia of 3.27 mmol/l (ref 0.9-2.0 mmol/l), and total hypocalcaemia of 1.57 mmol/l (ref 2.3-3 mmol/l). Ionised calcium was markedly reduced at 0.66 mol/l (ref 1.12-1.4 mmol/l). Parathyroid hormone (PTH) was < 10 pg/ml (ref 20-65 pg/ml) consistent with primary hypoparathyroidism. Urinalysis including sediment examination revealed USG 1.015, pH 6, proteinuria, erythrocytes, leukocytes, and large numbers of rod-shaped bacteria, consistent with urinary tract infection. Urine culture yielded a heavy growth of *E.coli*, sensitive to a number of antibiotics. Ultrasonography of the urinary tract revealed hyperechoic renal cortices with poor corticomedullary definition, suggestive of bilateral renal dysplasia.

Initial treatment consisted of Hartmann’s intravenous fluid therapy supplemented with calcium gluconate to address hypocalcaemia. Oral calcitriol and calcium carbonate were started, allowing parenteral calcium gluconate to be discontinued two days later. Antibiotic therapy for the urinary tract infection consisted of clavulanate potentiated amoxicillin. Aluminium hydroxide was added to the food to limit phosphate absorption, and the dog was switched to a renal diet. Follow up urine cultures were negative, and urine protein: creatinine ratio was unremarkable (0.44, ref < 0.5). At follow up several months later the azotaemia has progressed with urea 33.0 mmol/l and creatinine 379 mmol/l. On-going management consists of a renal diet, aluminium hydroxide, calcium carbonate and calcitriol.

Sub-normal PTH in the presence of ionised hypocalcaemia is diagnostic for primary hypoparathyroidism. The history and clinical findings suggest a concurrent juvenile nephropathy in this case. Familial renal disease has been described in 6 Chow Chows. In addition to azotaemia and non-regenerative anaemia, 4 of these dogs were hypocalcaemic (mean 1.64 mmol/l) and hyperphosphataemic (mean 6.14 mmol/l). Calcium and phosphorus...
derangements may be multifactorial in this case. Renal biopsy would be required for further classification.

References:
