WHAT IS YOUR DIAGNOSIS?

A two year male neutered crossbreed dog was presented to the R(D)SVS Internal Medicine Service for investigation and management of hypocalcaemia that had been identified 3 days previously by the referring veterinary surgeon. The dog had developed an acute onset of muscle spasms, and trembling, necessitating emergency management at the referring veterinary practice.

On presentation the dog was bright, alert and responsive, and only minor abnormalities were detected, namely constant panting, moderate hypersalivation and slightly increased body condition score (6/9).

The dog was admitted for further investigations and stabilisation of his blood calcium levels.

1. What are the main differential diagnoses of hypocalcaemia in this case?

2. How would you evaluate this case further?
1. Potential differential diagnoses for hypocalcaemia are listed below

   a. Parathyroid hormone disorders:
      i. Primary hypoparathyroidism
      ii. Hypomagnesaemia

   b. Parathyroid independent:
      i. Acute renal failure
      ii. Chronic renal failure (secondary renal hyperparathyroidism)
      iii. Secondary nutritional hypoparathyroidism
      iv. Acute pancreatitis
      v. GI malabsorption
      vi. Hypovitaminosis D
      vii. Medullary carcinoma of the thyroid gland

In female dogs eclampsia is an important differential for hypocalcaemia. Phosphate containing enemas and tumour lysis syndrome are also potential differential diagnoses in some cases of hypocalcaemia. In patients who have undergone thyroid surgery, iatrogenic damage of parathyroid gland is also an important differential diagnosis.

In this case, there was no history of access to toxins and no history of gastrointestinal signs. The sudden onset neuromuscular signs in the absence of other clinical signs made primary hypoparathyroidism the leading differential diagnosis.

2) Further evaluation of the case initially involved routine haematology and biochemistry.

Routine haematology was unremarkable, and his biochemistry revealed moderately decreased total calcium (2.04 mmol/L; ref range 2.3-3 mmol/L), and ionised calcium (1.01 mmol/L; ref range 1.15-1.5 mmol/L). Although inorganic phosphate was within normal limits, it was close to the upper limit of the range (1.97 mmol/L; ref range 0.9-2 mmol/L). Magnesium, which can also be low in cases of hypocalcaemia, was found to be normal. Both total and ionised calcium had been significantly lower before treatment with the referring veterinary practice.

Urinalysis was unremarkable and along with his biochemistry confirmed that renal disease was not the cause of his hypocalcaemia.

In order to examine whether the hypocalcaemia was caused by primary hypoparathyroidism, parathyroid hormone (PTH) was measured. The parathyroid hormone concentration was < 10 pg/ml (ref range 20-65 pg/ml) which is consistent with primary hypoparathyroidism in light of the concurrent hypocalcaemic state. In all other cases of hypocalcaemia PTH will increase as the body attempts to restore normal calcium homeostasis.
Emergency management was undertaken by the referring veterinary surgeon. Regardless of the aetiology initial emergency management is to give calcium gluconate 10% solution: 5-15mg/kg IV slowly over a 10-30 minute period to clinical effect. ECG monitoring is recommended and the infusion should be stopped if bradycardia, VPC’s or shortening of the Q-T interval are noted.

In this case, the dog was hospitalised and oral therapy was commenced with calcitriol, initially at a dose of 0.024 µg/kg daily and calcium carbonate 600mg q 6 hours. Intravenous calcium gluconate was continued to maintain appropriate calcium levels until oral supplementation took effect. During hospitalisation, his calcium blood levels were monitored regularly and once stabilised at the low end of the reference range, the intravenous infusion was tapered and stopped. Regular monitoring of serum calcium concentrations was undertaken to ensure that hypercalcaemia did not develop.

Discussion

Primary hypoparathyroidism is a rare condition, thought to be due to immune-mediated destruction of the parathyroid gland. The average age of onset of primary hyperparathyroidism has been reported to be 5 years with a slight female predisposition. As in this case, the clinical signs are usually related to abrupt and often intermittent neuromuscular dysfunction which is worsened by excitement or exercise. Typical clinical signs are face rubbing, nervous behaviour, twitching, tremors, stiff gait, behavioural changes and seizures. The availability of PTH assays in specialist veterinary endocrine labs enables this condition to be readily diagnosed. Dogs with primary hypoparathyroidism will require on-going therapy; generally calcium supplementation can be stopped, and patients maintained on vitamin D supplementation such as calcitriol which will promote calcium absorption from the intestine. However on-going monitoring of serum calcium concentrations is important and dose adjustments are likely to be required. The long term prognosis for primary hypoparathyroidism is good.


Bruyette and Feldman (1988) Primary hypoparathyroidism in the dog. Report of 15 cases and review of 13 previously reported cases. *Journal of Veterinary Internal Medicine*. 2, 7-14