A CASE OF NUTRITIONAL SECONDARY HYPERPARATHYROIDISM IN A DOG

Nutritional secondary hyper parathyroidism is a metabolic disorder in which bone production is normal but osteopenia results from excessive bone resorption. It is caused by diets providing excess phosphate and/ or insufficient calcium. This paper reports a case of nutritional secondary hyper parathyroidism in a Great Dane male dog.

A three month old male Great Dane dog was presented to the University Veterinary Hospital, Mannuthy with a history of swelling of the right forelimb since two weeks. The metacarpal bones of the right forelimb were bent and deviating laterally (Fig. 1). The carpal joints of both forelimbs were enlarged. Enlargement of the costochondral junctions of the ribs was also evident. The bones of the hind limbs did not reveal any abnormalities. The clinical data were within the normal range. The conjunctival mucous membrane was congested. Other physiological functions such as appetite, defaecation and urination were normal. The vaccination and deworming history was regular. The dietary history of the animal included milk and eggs in the morning, rice and beef in the afternoon, followed by the same again at night.

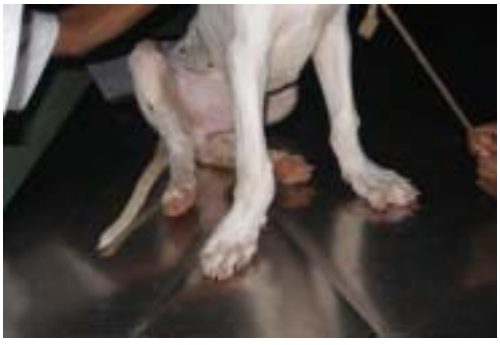

Fig. 1. Bending and lateral deviation of metacarpal bones

The haematobiochemical examination revealed normocytic, normochromic anaemia with hyperphosphatemia (7.9 mg/ dL) and normocalcemia (9.9 mg/ dL). The serum alkaline phosphatase level was 145 IU/ L. The radiographic images showed a thinning of the cortex of the radius and ulna distally along with a thickening of the epiphyseal region of the bones (Fig. 2). On the basis of the history, clinical observations, haematology, biochemistry and radiography, the condition was diagnosed as nutritional secondary hyperparathyroidism.

The dog was treated with 10% calcium gluconate (Calcium Sandoz) injection intravenously @ 10 ml once a day for three consecutive days followed by tab shelcal 250 mg once daily as orally with substitution of chicken meat which contains less phosphorus when compared to beef. The dog was brought for a review after twenty one days at which time it showed almost complete recovery (Fig. 3).

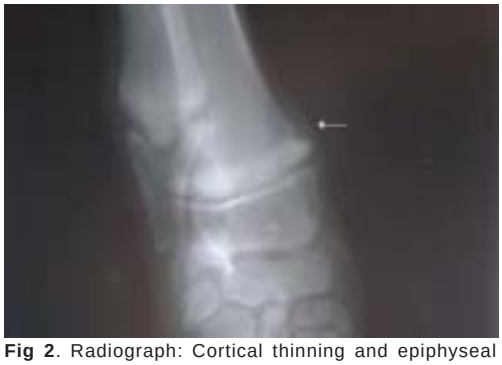

Fig 2. Radiograph: Cortical thinning and epiphyseal thickening of radius and ulna (white arrow)

Fig. 3. Reduction in deformity

Nutritional secondary hyperparathyroidism is caused by diets providing excess phosphate and/ or insufficient calcium. Affected animals have usually been fed mainly meat which contains more phosphorus and less calcium. The Ca: P ratio of meat is 1: 16 to 1: 35 in contrast to the recommended ratio of 1.2: 1 for dogs. This imbalance induces hypocalcaemia, which increases secretion of parathyroid hormone. Increased parathyroid activity tends to normalise blood calcium and inorganic phosphate concentrations by promoting mineral resorption from bone and a continued ingestion of the defective diet sustains the hyperparathyroid state and causes progressive skeletal demineralisation and consequent clinical signs. The clinical signs observed in the present study were similar to those observed by Svoboda *et al*. (1994).

Radiographic features concurred with the findings of Lourens (1980). An area of relative radiopacity occurred in the metaphysis adjacent to growth plates, representing the area of primary mineralisation and could be best appreciated in the distal radius and ulna. Blood biochemical tests were of little value in confirming this condition. Normocalcemia might be due to compensatory mechanisms as suggested by Johnson and Watson (2002). Even though the animal responded well to the treatment adopted, permanent skeletal deformity could be observed in the present case.

Summary

A case of nutritional secondary hyperparathyroidism in a three month old Great Dane male dog and its successful treatment with intravenous calcium injection is placed on record.

References

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