CASE REPORT
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RENAL OSTEODYSTROPHY IN A GERMAN SHEPHERD DOG – A CASE REPORT

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ABSTRACT

A female German Shepherd dog aged 10 years was presented to the Referral Veterinary Polyclinic, Indian Veterinary Research Institute, Bareilly, with the chief complaint of facial swelling for the last 7 days. Physical examination revealed a painless, soft swelling of the maxilla and mandible. Radiographic examination showed fibrous osteodystrophy. Serum biochemistry revealed hypocalcemia, hyperphosphatemia and azotemia. Based on the clinical, laboratory and imaging studies, the case was diagnosed as renal osteodystrophy.

Keywords: Osteodystrophy, Kidney failure, Hyperphosphatemia, Hypocalcemia

INTRODUCTION

Renal osteodystrophy results in a series of skeletal abnormalities, including osteoporosis, osteopenia, osteoporosis and osteitis fibrosa. Phosphorus retention favours bone disorders in dogs with chronic kidney failure. Osteodystrophy is clinically characterized by painless swelling of facial bones. The relationship between parathyroid hormone (PTH), 1, 25 dihydroxycholecalciferol, calcium and phosphorous and fibroblast growth factor (FGF-23) in chronic kidney failure is complex (Arnaud, 1973). Dogs with chronic kidney disease (CKD) stage 1 as per International Renal Interest Society (IRIS) classification, reported to have developed renal secondary hyperparathyroidism (Cortadellas *et al*., 2010). Persistent elevation of PTH noticed in patient with CKD, can lead to numerous bone disorders with mineral imbalance which are collectively called as renal dystrophy (Foster, 2016). PTH mediated bone resorption has been well documented in pet animals wherein, osseous tissue is being replaced by fibrous tissue (Barber and Elliott, 1998). Maxilla and mandible were the commonly affected bones in canine renal osteodystrophy (Sarkiala *et al*., 1994). This article reports a case of renal osteodystrophy or rubber jaw syndrome in a dog.

CASE HISTORY AND OBSERVATION

Anamnesis

A 10 year old German Shepherd female dog weighing about 20 kg was presented with the history of bilateral facial swelling for the last 7 days (Fig. 1), weight loss, general weakness and frequent urination. The dog was symptomatically treated with corticosteroid, antihistaminic and diuretic but there was no improvement in the condition.

Clinical observations

Animal was found to be dull and depressed. Clinical examination revealed a rectal temperature of 102°F, a pale mucous membrane, respiratory distress, and a heart rate of 80 beats per minute. Physical examination revealed a painless, soft swelling involving maxilla and mandible. X-ray of the lateral view of skull (Fig. 2) confirmed the presence of dystrophic changes in facial bones.

Fig. 1. Facial swelling

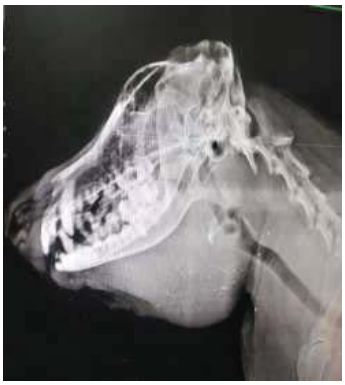

Fig. 2. Soft tissue swelling in X-ray

Table 1. Result of blood/serum analysis

Parameters	Observed value	Reference range*
Hemoglobin (g/dl)	4	11.9 – 18.9
Total Erythrocyte Count (10^6/cmm)	2.68	4.95 – 7.87
Total Leukocyte Count (10^3 /cmm)	12.25	5.0 – 14.1
Calcium (mg/dl)	4	9.1 - 11.7
Phosphorous (mg/dl)	10	2.9 – 5.3
Creatinine (mg/dl)	5.9	0.5 – 1.7
Blood urea nitrogen (mg/dl)	38	8 - 28

(* Reference values form The Merck Veterinary Manual, 10th Ed.,)

Clinical Pathology

Blood and serum samples were collected for estimation of complete blood count, kidney profile and mineral estimation, respectively (Table 1). Based on the history, clinical observation and laboratory investigation, the case was confirmed as renal osteodystrophy and animal was treated accordingly.

TREATMENT AND DISCUSSION

As the illness was identified as osteodystrophia consequent to chronic renal failure, hemodialysis and emergency blood transfusion were advised. Meanwhile, it was treated intravenously with 200 ml DNS, 200 ml RL and 20 ml of 10 % calcium gluconate in normal saline. To address the anaemic state, the first dose of injection Eporise 2000 IU (recombinant erythropoietin) @ 75 IU/ kg body weight and Inj. Imferon 2ml IM were administered. The animal was further supported with oral haematinics (aRBC pet) and sucralfate. Soon after the initiation of treatment there was marked improvement in the condition of the dog. Animal started taking feed and there was a reduction in the swelling of the face. Unfortunately, the animal died on the third day of treatment.

Chronic renal failure is an irreversible disease state characterized by polyuria, polydypsia, abnormal production of parathyroid and erythropoietin hormones, bouts of vomiting, anemia with hypertensive crisis. Initial stabilization of animal is very much essential before going for dialysis. Even with timely medical intervention, one can only prolong the survival of the patient rather than a permanent cure because of the multisystemic involvement in chronic kidney failure. Parenteral fluids administration will resolve the electrolyte abnormality. Calcium gluconate administration keeps the PTH in basal level and thereby prevents further calcium mobilization from bones. Phosphate binder such as aluminium hydroxide may be prescribed. Aluminum hydroxide is considered to be first line of phosphate binder @ 80-100 mg/kg/d (Foster, 2016). Compromised kidney could not produce sufficient amount of erythropoietin. Hence, it requires administration of recombinant erythropoietin. As hyperphosphatemia plays a pivotal role behind the renal osteodystrophy, minimizing the serum phosphorus level by dietary modification and medical intervention may prevent subsequent consequences

SUMMARY

A 10-year old, female German shepherd dog with painless, soft swelling of the maxilla and the mandible, and azotemia was diagnosed with renal osteodystrophy. Phosphorus retention is the central

concept in the pathophysiology of renal osteodystrophy. Much emphasis has to be given for mitigating the phosphorus level in CRF patients.

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ADENOMA OF THE NICTITANS MEMBRANE GLAND IN A DOG: – A CASE REPORT

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ABSTRACT

A Ten-year-old male non-descript dog weighing 14.5 kg was presented to the Teaching Veterinary Clinical Complex (TVCC), Pookode, with the history of a mass protruding at the medial canthus of left eye with epiphora for the past few months. Physical examination revealed a bright pink mass with the surface ulcerated. Due to its chronic nature and ulcerative appearance, surgical resection was resorted to. Histopathology revealed variably sized tubular glands and inflammation and confirmed the condition as adenoma of the nictitans membrane gland. Animal recovered uneventfully.

Keywords: Adenoma, Canine, histopathology, Nictitans membrane gland

INTRODUCTION

Adenoma of the nictitans membrane gland (NMG) is infrequently reported. Neoplasia of the gland of the third eyelid is uncommon in dogs (Wilcock *et al.,* 2002; Dubielzig *et al.,* 2010) and histopathological diagnostic criteria for these tumours have not been established adequately. The third eyelid –nictitans or nictitating membrane lies in the ventro-medial orbit of dogs, cats, and horses, where it physically protects the cornea and acts like a wind shield wiper to distribute pre-corneal tear film. Also, the third eyelid comprises of a T-shaped piece of cartilaginous skeleton, conjunctiva covering the bulbar and palpebral surfaces, numerous superficial lymphoid follicles under the bulbar surface and a large sero-mucoid gland surrounding the base of the cartilage responsible for 25% to 40% of tear production. Frequent abnormalities of the third eyelid are protrusion, eversion of the nictitans cartilage, prolapse of the nictitans gland ("cherry eye"), foreign bodies behind the nictitans, enlargement of the gland of the third eyelid due to neoplasia or cyst formation, and hyperplasia of the bulbar lymphoid tissue (follicular conjunctivitis) (Dees *et al.,* 2016).