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SHORT NOTE

FIBROUS OSTEODYSTROPHY WITH FACIAL HYPEROSTOSIS ASSOCIATED WITH RENAL SECONDARY HYPERPARATHYROIDISM IN DOGS

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Renal secondary hyperparathyroidism is a complication of chronic renal failure. It is clinically characterized by hyperostotic bone lesions such as facial swelling due to the deposition of unmineralized osteoid by hyperplastic osteoblasts and production of fibrous connective tissue exceed the rate of bone resorption and increased endogenous levels of parathyroid hormone (PTH) (Aiello *et al.,* 2015). Although secondary renal hyperparathyroidism and renal osteodystrophy are well known sequel of chronic renal failure (CRF) but clinically important renal osteodystrophy is rarely seen in dogs (Nagode *et al*., 1996, Rusenov *et al*., 2009; Chand *et al*., 2012). This paper presents two cases of fibrous osteodystrophy with facial hyperostosis associated with renal secondary hyperparathyroidism in dogs.

Fig. 1. A bitch (above) and a dog (below) with bilateral facial swelling due to renal failure

A 4-year old, Labrador bitch was presented (case No. 5199 dated 07/05/2016) at Small Animal Unit of GADVASU with a history of swollen face, weakness and inappetence for last 4 months. Clinical examination of bitch revealed pyrexia (39.6^0C), normal heart and respiration rate, moderate degree of dehydration, slightly pale mucus membrane and painless hard swelling of nasal and jaw bones (Fig. 1). Haematology revealed neutrophilia with toxic changes in neutrophils. Cytological examination of fine needle aspirate of swollen jaw did not show any neoplastic cells. Serum biochemistry showed increased levels of blood urea nitrogen (69.5 mg dL^{-1}), creatinine (3.27 mg dL^{-1}) and hyperphosphatemia (11.6 mg dL^{-1}). Rostroventral radiography of skull revealed generalized reduced bone density and soft tissue swelling of maxilla and nasal bones. Ultra-sonogrphic examination showed small and poor cortico-medullary differentiation of both kidneys.

Another Labrador dog (2.5-year old male, case No. 4995 dated 03/05/2016) was presented with primary complaint of bilateral facial swelling for last 2 months. Clinical examination showed normal temperature, normal heart rate, tachypnea and anemic mucus membrane and painless bilateral facial swelling (Fig. 1). Haematological studies revealed severe anemia (haemoglobin 4.9 gm%, packed cell volume 12.3%) and neutrophilia. Cytological examination of fine needle

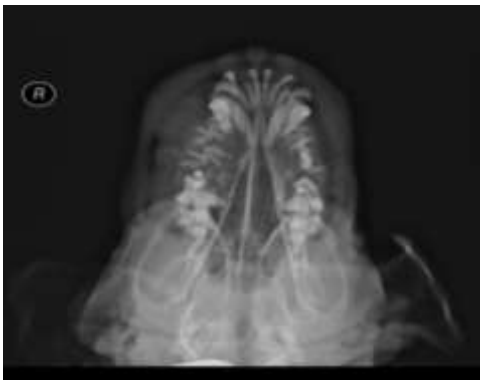

Fig. 2. Radiograph showing reduced bone density and soft tissue swelling of maxilla and mandible

aspirate of swollen jaw showed numerous inflammatory cells but did not show any neoplastic cells. Serum biochemistry showed increased levels of blood urea nitrogen (67 mg dL^{-1}), creatinine (5.7 mg dL^{-1}) and hyperphosphatemia (7.9 mg dL^{-1}). Rostroventral radiography of skull revealed generalized reduced bone density and soft tissue swelling of maxilla and nasal bones (Fig. 2).

On the basis of history, clinical signs, haemato-biochemical, ultrasonographgy and radiological findings both cases were diagnosed as renal secondary hyperparathyroidism (rubber jaw, renal rickets). Both the animals were treated with Ringer's lactate @ 500 mL, od, i.v. (Albert Davis), dextrose normal saline @ 500 mL, od, i.v. (Wokhardt), ampicillin @ 10 mg kg^{-1} b. wt. bid, im (Roscillin, Ranbaxy), ranitidine @ 2 mg kg^{-1}, bid, im (Aciloc, Cadila), multivitamin @ 2 mL im (Polybion, Merck), furosemide @ 2 mg kg^{-1}, bid, im, phosphate binder (Gelusil, Parke Davis) and phosphorus restricted diet. The treatment regimen was continued for a fortnight and during treatment the animals died within a month.

Parathyroid gland hyperplasia and hyperparathyroidism can occur secondary to CRF. The accepted explanation of the pathogenesis of this syndrome involves the kidneys' progressive inability to excrete phosphorus. Dogs with CRF have a progressive loss of nephrons and a decreased glomerular filtration rate (GFR) which leads to increase in serum concentrations of substances that are normally filtered from the blood by the kidneys, including phosphate. As the serum phosphate level remains high, the parathyroid glands are chronically stimulated to increase the serum calcium level, leading to parathyroid hyperplasia (Schenck *et al.*, 2006).

Anemia is common in chronic renal diseases and its main cause is inadequate production of erythropoietin by diseased kidneys to meet the demand for new red cells because of loss from hemolysis and hemorrhages (Nelson and Couto, 2014). Neutrophilia and lymphopenia reflect the stress of chronic disease. The skeletal lesions like bilateral facial swelling and poor mineralization of maxilla, mandible and nasal bones might be due to renal secondary hyperparathyroidism which resulted from impaired renal excretion of phosphate and consequently hyperphosphatemia (Rosol and Capen, 1996). Hyperphosphatemia and decreased calcium absorption from the gut lead to a chronically increased PTH level thereby causing mobilization of calcium stores which may lead to the conditions such as fibrous osteodystrophy, bone marrow suppression, soft tissue mineralization, urolithiasis, and neuropathy (Schenck *et al.*, 2006). Bones of the skull and mandible are most severely affected and marked proliferation of connective tissues associated with the maxilla causes distortion of face (Ettinger and Feldman, 2000). Chand *et al.* (2012) also reported two cases of osteodystrophy with facial hyperostosis due to chronic renal failure in dogs. They therapeutically managed these cases with conservative treatment consisting of fluid therapy, antiemetic, antacid, antibiotics, diuretics, multivitamins, phosphate binders and phosphorus restricted diet. Even with early diagnosis and treatment, CRF is a progressive and irreversible disease for which there is no cure, and the prognosis for animals with renal hyperparathyroidism is guarded to poor.

Ethical statement: The case study protocol was performed in compliance with institutional ethical guidelines. The owners of both dogs gave consent for clinical procedures.

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