CASE REPORT

An Infrequent Case of Renal Secondary Hyperparathyroidism in a Juvenile Dog

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Chronic Kidney Disease (CKD) and its associated complications are commonly leading to high mortality in dogs. The elevated phosphorus in CKD is one of the major serum biochemical alterations observed in dogs. Hyperphosphataemia in CKD leads to hyper-parathyroidism, which is called as renal secondary hyperparathyroidism (RSHPT). Dogs with RSHPT in association with CKD are unexplored and mostly under diagnosed, which may be the reason for metabolic complication in management of CKD (Cortadellas *et al.,* 2010). Hyperphosphataemia and hyperparathyroidism are important factors that might influence to reduce the lifespan of dogs with CKD (Polzin, 2011). To the best of author's knowledge, the renal osteodystrophy secondary to CKD is rare in dogs and very few reports on renal secondary hyperparathyroidism in dogs are available. The present study describes clinical case of renal secondary hyperparathyroidism in young dog.

Case History and Observations

An eight month old male Labrador retriever dog weighing 15.2 kg was presented to Small Animal Medical Referral Clinic, Department of Veterinary Clinical Medicine, Veterinary College and Research Institute, Namakkal, Tamil Nadu with the history of facial swelling for the past three months. The history evidenced prolonged urination, anorexia, general weakness, weight loss, nasal discharge, nausea and difficulty in breathing.

Clinical examination showed pale conjunctival mucus membrane, decreased skin elasticity, emaciation, reluctant to move with preference to lie down. There was marked, symmetric non-painful swelling of the maxilla with dental displacement and mild bilateral nasal serous discharge. No softening of the jaw was appreciated on palpation. The feces were watery, dark coloured with unpleasant odour. Haematology revealed decrease in the level of Hb, PCV, RBC and increase in WBC along with neutrophilia. Serum biochemistry showed elevated BUN, creatinine, phosphorus, potassium, alkaline phosphatase and decrease in ionized calcium (Table 1). Serum sample was further subjected to canine parathyroid hormone estimation (competitive ELISA, AssayGenie). It showed most significant elevation of PTH 802 pg/mL. Urinalysis revealed isothenuria (1.010) with 1+ protein on dipstick analysis (DIRUI, Netherland urine strips). Urine protein creatinine ratio (UPCR) was 7.5. The dog was slightly hypertensive with systolic pressure of 160 mm Hg. Clinical signs, lesions and laboratory results supported the diagnosis of renal secondary hyperparathyroidism.

The lateral radiographic image of the maxilla, revealed a noticeable decrease in bone radiopacity throughout, which is suggestive of the existence of fibrous osteodystrophy (Fig. 1). Shrunken kidneys, indistinct corticomedullary junction and severe loss of cortex with hyperechoic capsule were the findings in ultrasonography. Small hyperechoic foci within the renal parenchyma indicated mineralization and fibrosis (Fig. 2). Computed Tomography of the head

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was performed using CT scanner (Toshiba, Alexion) under general anaesthesia by positioning in sternal recumbency to rule out neoplasm. CT of the head revealed poor mineralization of the facial and calvarial bones, especially the maxillary and frontal bone as compared to healthy skull (Fig. 3, 3a). The maxillary and palatine bones were replaced by proliferative, heterogeneous tissue of mixed soft tissue and granular mineral attenuation. The dog was hospitalized and treated with intravenous crystalloid fluid therapy and blood transfusion. The dog collapsed subsequent day of presentation. Autopsy was performed and samples from kidney were collected for histopathology.

Table 1: Haemato-biochemical changes of young dog with RSHPT

Parameters	Value	Reference Value (Ettinger *et al.,* 2017)
Haemoglobin (g/dL)	3.7	12-19
Packed cell volume (%)	13	37-57
Red blood count ($\times10^6$ /µL)	1.8	5.0-9.0
White blood count ($\times10^3$/µL)	18.69	5.0-15.0
Platelet count (10^5 /µL)	5.05	1.6-5.1
Neutrophils (%)	84	60-75
Lymphocytes (%)	11	17-21
Monocytes (%)	05	2-10
Total protein (g/dL)	7.6	5.4 -7.1
Albumin (g/dL)	3.8	2.3 -3.3
ALT (U/L)	71	10-109
SAP (U/L)	76	13-66
Total bilirubin (mg/dL)	0.8	<1.2
Direct bilirubin (mg/dL)	0.3	<0.3
BUN (mg/dL)	169	8-28
Creatinine (mg/dL)	9.8	0.5-1.8
Calcium (mg/dL)	10.3	9-11.7
Phosphorus (mg/dL)	26.3	2.6-5.3
Glucose (mg/dL)	129	65-118
Potassium (mEq/L)	5.6	3.9-5.1
Sodium (mEq/L)	122	141-142
Chloride (mEq/L)	99	105-115
PTH (pg/mL)	802	10-65
iCa (mmol/L)	0.68	1.25-1.48

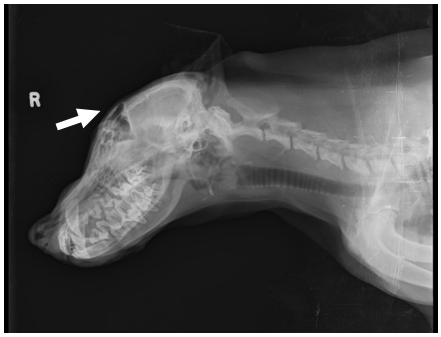

Fig. 1: The lateral radiographic image of the maxilla revealed widespread reduction in bone radiopacity, indicating the presence of fibrous osteodystrophy.

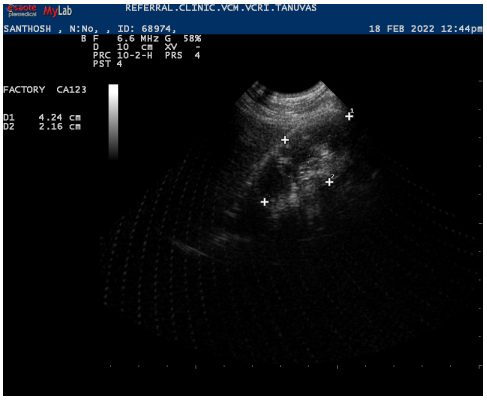

Fig. 2: Ultrasonography of kidney – shrunken kidney with indictinct corticomedullary junction, loss of cortex with hyperechoic capsule & mineralization of renal parenchyma

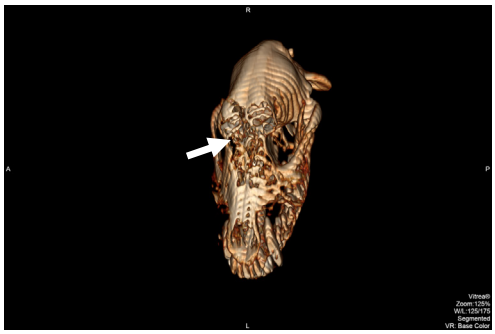

Fig. 3: Computed Tomography (CT) image of RSHPT dog

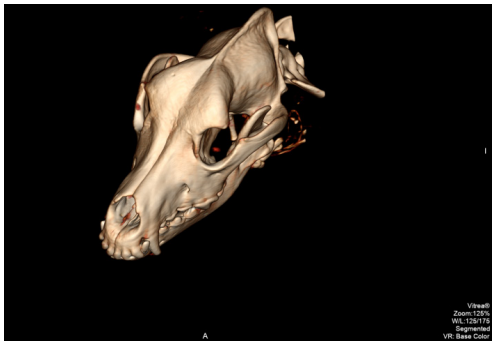

Fig. 3a: CT image of one year old apparently healthy Labrador dog

The gross examination of kidneys revealed significant abnormalities in both kidneys, including shrinkage, disfigurement, and strict adhesion of the capsule to the underlying parenchyma (Fig. 4). The cortex of the kidneys displayed an irregular and uneven surface with raised granularity. On cutting the kidneys, the cortex appeared thin and extensively damaged in some regions. The cortical surface exhibited raised white streaks and nodular areas. Additionally, the renal pelvis showed signs of destruction. Histopathology revealed proliferation of fibrous tissue in interstitium leading to compression of glomeruli and loss of glomerular tuft. There were severe degeneration and desquamation of renal tubular epithelium, mineralization of necrotic tubules and peritubular fibrosis (Fig. 5 & 6).

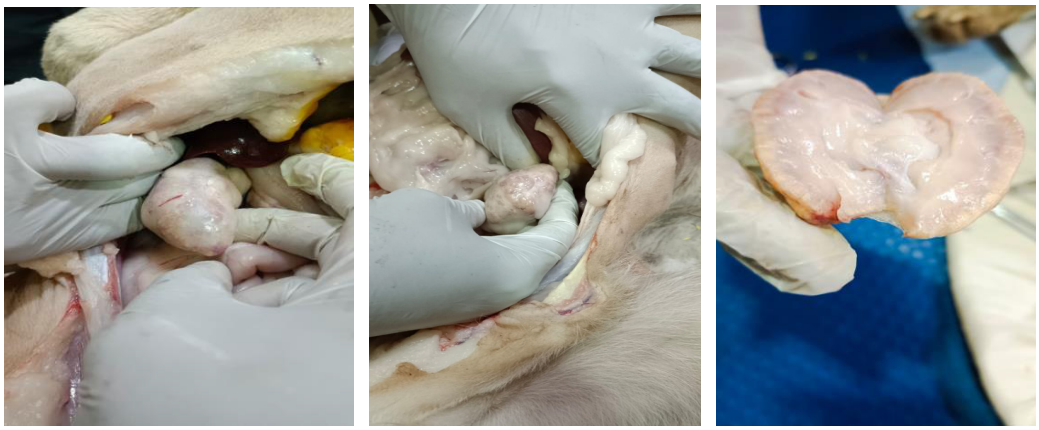

Fig. 4: Gross **i**mage from left to right: Right kidney, Left kidney and Cut section.

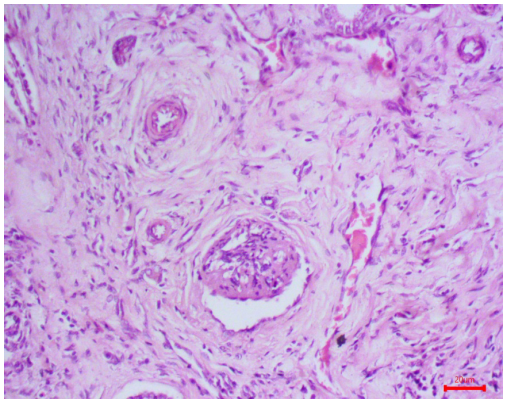

Fig. 5: Histopathology of the kidney of RSHPT dog (H&E staining, ×20). Proliferation of fibrous tissue in interstitium leads to compression of glomeruli and loss of glomerular tuft.

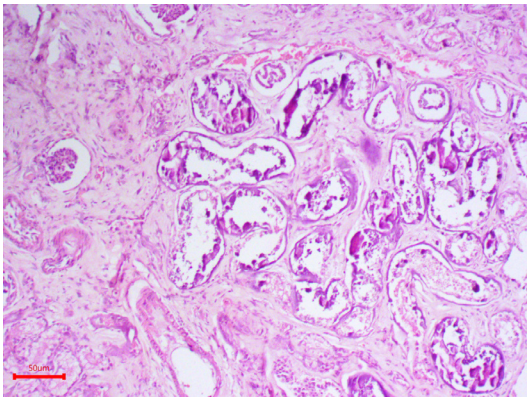

Fig. 6: Histopathology of the kidney of RSHPT dog (H&E staining, ×50). Severe degeneration and desquamation of renal tubular epithelium, mineralization of necrotic tubules and peritubular fibrosis.

Discussion

Renal secondary hyperparathyroidism and renal osteodystrophy were well known sequela of chronic renal failure. Renal osteodystrophy was not commonly observed in dogs and cats and clinical signs associated with this condition were relatively rare. In dogs, young and growing animals are more likely to be affected, likely because their bones were more sensitive to the effects of PTH (Nagode *et al.*, 1996). The present study correlated with authors that the age of the dog discussed was eight months.

Dogs suffering from CKD may exhibit 10 times higher PTH concentration than the upper limit which is associated with renal secondary hyperparathyroidism-RSHPT (Gerber *et al.*, 2003). In this case, PTH concentration was almost 12 folds higher. Dogs with CKD an elevated serum phosphorus concentration above 32.04 mg/dL is indicative of RSHPT (Foster, 2016). In this particular case, the serum phosphorus level of 26.3 mg/dL was almost closer to reference limit. Measurement of the serum iCa concentration was helpful for differentiation between primary hyperparathyroidism and renal secondary hyperparathyroidism. The serum iCa level was usually high with primary hyperparathyroidism and normal to low with RSHPT (Schenck *et al.*, 2006; Bartges, 2012). In the present case, the low iCa level of 0.68 mg/dL was indicative of renal secondary hyperparathyroidism.

The significant loss of bone density observed in the skull CT scans, particularly in the maxillary and mandibular bones, suggests the presence of a metabolic bone disorder such as secondary hyperparathyroidism, which can be caused by renal or nutritional issues. De Fornel-Thibaud *et al.*, (2007) reported that CT had the potential to be useful for evaluating skull osteopenia due to two advantages: eliminating structure superimposition and accurately measuring bone mineral density using enhanced contrast between bone structures. In their study, CT images revealed significant bone tissue loss in the maxillary and frontal regions, indicating widespread bone resorption in those areas.

Even though early diagnosis and treatment for RSHPT was initiated, Chronic Renal Failure was a progressive and irreversible disease for which there was no cure, and the prognosis for animals with renal hyperparathyroidism was guarded to poor (Stillion and Ritt, 2009). In the present study, the dog collapsed on the third day and subjected to detailed

postmortem. The gross and histopathological findings of kidneys observed in case under study are consistent with previous observations made by Headley *et al.* (2008).

These case findings resembled those described in a previous case reports (Rusenov, 2010; Vanbrugghe *et al.*, 2011; Barczak *et al.,* 2020), demonstrating significant bilateral swelling of the maxilla and inadequate mineralization of the facial bones. In conclusion, young dogs presented with facial swelling must be screened for CKD associated renal secondary hyperparathyroidism.

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