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Rubber Jaw Syndrome in a Juvenile Labrador Retriever: A Rare Sequela of Babesia Gibsoni-induced Renal Secondary Hyperparathyroidism

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ABSTRACT

A seven-month-old male Labrador Retriever was presented to the Veterinary Clinical Complex, College of Veterinary Science, Assam Agricultural University, with complaints of progressive facial swelling, anorexia, intermittent vomiting and generalized weakness. Clinical examination revealed mandibular and maxillary enlargement, pliable jaw bones and loose teeth-suggestive of fibrous osteodystrophy. Hematological evaluation revealed macrocytic hypochromic anemia, while Giemsa-stained peripheral blood smears confirmed intraerythrocytic *Babesia gibsoni*. Serum biochemistry demonstrated marked elevations in blood urea nitrogen (BUN), creatinine, phosphorus and parathyroid hormone (PTH), alongside mild hepatic enzyme elevation and hypocalcemia, consistent with renal dysfunction and secondary hyperparathyroidism. Cranial radiographic imaging of the skull showed osteolytic changes in the facial bones, while abdominal ultrasonography revealed splenomegaly and renal cortical thickening with loss of cortico-medullary junction. Clinical findings such as mandibular pliability ("rubbery jaw"), hematobiochemical abnormalities (anemia, azotemia, hyperphosphatemia, hyperparathyroidism) and diagnostic imaging supported the diagnosis of fibrous osteodystrophy secondary to chronic kidney disease (CKD), complicated by canine babesiosis-induced renal insufficiency. This case highlights a rare presentation of metabolic bone disease associated with vector-borne hemoprotozoan infection in a juvenile dog. It underscores the need for integrated diagnostic approaches when systemic signs coincide with bone pathology, especially in endemic regions where babesiosis and renal disorders coexist.

Key words: Canine babesiosis, Chronic kidney disease (CKD), Fibrous osteodystrophy, Labrador retriever, Renal secondary hyperparathyroidism, Rubber jaw syndrome.

Case history and presentation

The present study was carried out at the Veterinary clinical complex (VCC), Assam Agricultural University (AAU), Khanapara, Guwahati, Assam, India, in the month of February 2025.

A seven-month-old male Labrador Retriever puppy, weighing approximately 10 kg, was presented to the Outpatient Department (OPD) of the Veterinary Clinical Complex, College of Veterinary Science, Assam Agricultural University, Assam, India. The primary complaints included gradual and progressive swelling of the facial region and difficulty in mastication persisting for the past month. The owner also reported partial anorexia, polydipsia and intermittent episodes of vomiting. The pup showed a preference for soft food and had a noticeable reduction in body weight over the course of illness. The clinical history indicated a chronic, insidious progression of signs, suggestive of an underlying systemic disorder involving both renal and skeletal systems.

Clinical examination and investigation

On physical examination, the puppy appeared to be in poor body condition and exhibited a depressed demeanor with a moderately reduced response to external stimuli. Rectal temperature and heart rate were within normal physiological limits; however, mild to moderate tachypnoea was noted. The conjunctival mucous membranes were pale and the capillary refill time (CRT) exceeded two seconds, suggestive of compromised peripheral perfusion. Cardiac and pulmonary auscultation revealed no abnormal sounds.

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Marked bilateral swelling of the mandible and maxilla was observed. The antebrachial bones were soft and pliable on palpation and the teeth exhibited increased mobility, imparting a characteristic “rubbery jaw” sensation-indicative of fibrous osteodystrophy.

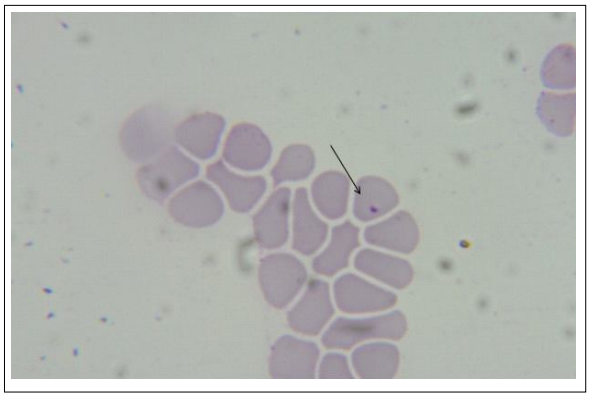
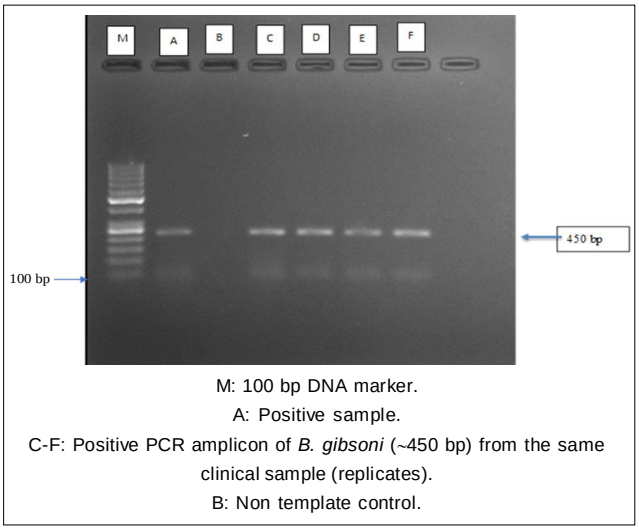


Fig 1: Demonstration of *Babesia gibsoni* in the erythrocyte by using giemsa stain viewed under light microscope (×1000).



M: 100 bp DNA marker.
A: Positive sample.
C-F: Positive PCR amplicon of *B. gibsoni* (~450 bp) from the same clinical sample (replicates).
B: Non template control.

Fig 2: Agarose gel electrophoresis showing a ~450 bp PCR amplicon of *Babesia gibsoni* on 1.5% agarose gel targeting 18S rRNA gene.

Hematological analysis revealed macrocytic hypochromic anemia (Table 1). Serum biochemistry showed a mild elevation in hepatic enzymes (ALT, AST) and a pronounced increase in renal parameters, including blood urea nitrogen (BUN) and creatinine. A significant rise in serum phosphorus levels was also detected (Table 2). Given these findings, parathyroid hormone (PTH) levels were evaluated and found to be markedly elevated. Microscopic examination of a Giemsa-stained peripheral blood smear confirmed the presence of intraerythrocytic *Babesia spp.* (Fig 1) and Molecular confirmation of *B.gibsoni* was carried out by PCR amplification of 18S rRNA gene of *B. gibsoni*, yielded a specific band of expected size, consistent with active canine babesiosis (Khan *et al*., 2025; Jena *et al*., 2021) (Fig 2). Urinalysis revealed presence of protein (++) in the urine, specific gravity of urine: 1.013, which is suggestive of isosthenuria and occasional granular casts. Absence of pyuria (WBC) and bacteriuria on microscopic examination.

Diagnostic imaging and final diagnosis

Cranial radiography revealed marked osteolytic changes in the nasal bones, along with generalized decreased radiopacity of the facial bones, particularly in the maxillary and mandibular regions (Fig 3). These findings were consistent with significant demineralization and bone resorption associated with metabolic bone disease.

Abdominal ultrasonography showed moderate splenomegaly, with the splenic parenchyma appearing diffusely hyperechoic-suggestive of chronic splenic involvement. Both kidneys were within normal size limits but exhibited irregular margins. The renal cortices were thickened and markedly hyperechoic, accompanied by a loss of cortico-medullary junction (CMJ) definition (Fig 4), indicating chronic renal pathology. Additionally, the parathyroid glands appeared enlarged, further supporting the diagnosis of secondary hyperparathyroidism.

Based on the integration of clinical signs (facial swelling, “rubbery jaw”), hematological abnormalities (macrocytic hypochromic anemia), biochemical findings

Table 1: Haematological parameters.

Parameters	Value	Reference range	Interpretation
RBC (million/mm^3)	1.60	4.95-7.87	Anaemia
PCV/HCT (%)	14.0	35-57	
Haemoglobin (g/dl)	3.2	11.9-18.9	
Thrombocytes (m/mm^3)	11.3	120-600	
MCH (pg)	20.0	19.5-24.5	
MCHC (cells/mm^3)	22.8	32-36.3	
MCV (fL)	87.9	66-77	
Neutrophils (%)	69.3	58-85	
Lymphocytes (%)	27.9	10.0-30.0	
Monocytes (%)	2.8	2.0-10.0	

*2016: Hematologic reference ranges, 11th edn. The merck veterinary manual.

(azotemia, hyperphosphatemia, elevated PTH), urinalysis and imaging evidence of bone demineralization and renal changes, the case was diagnosed as fibrous facial osteodystrophy secondary to chronic kidney disease (CKD)-induced secondary hyperparathyroidism, complicated by canine babesiosis due to *Babesia gibsoni* infection.

Parathyroid hormone (PTH) is a polypeptide secreted by the parathyroid glands that plays a pivotal role in maintaining calcium and phosphorus homeostasis. Its secretion is primarily regulated by serum calcium levels. When ionized calcium levels drop, PTH secretion is stimulated, leading to mobilization of calcium from bone, increased renal calcium reabsorption and enhanced activation of vitamin D, which in turn promotes intestinal calcium absorption (Lombardi *et al.,* 2011). In cases of secondary hyperparathyroidism-particularly renal in origin-this regulatory feedback becomes maladaptive. Chronic renal failure impairs the kidneys' ability to synthesize 1,25-dihydroxycholecalciferol (the active form of vitamin D), resulting in reduced intestinal calcium absorption and subsequent hypocalcaemia (Capen and Martin, 1977; Turkar *et al.,* 2018; Takahashi *et al.,* 2002). This persistent hypocalcaemia drives sustained PTH release, which, over time, leads to pathological bone resorption and replacement of mineralized bone with fibrous connective tissue- a condition known as fibrous osteodystrophy (Bovee, 1984; Rusenov *et al.,* 2009).

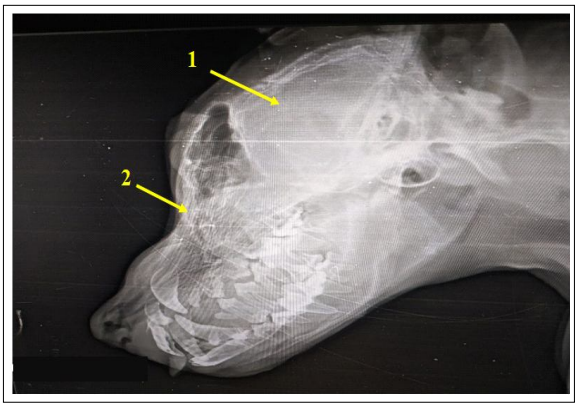


Fig 3: Radiographic examination of head, (1) Decreased radiographic density of skull bone (2) Striated appearance of maxillary bone.

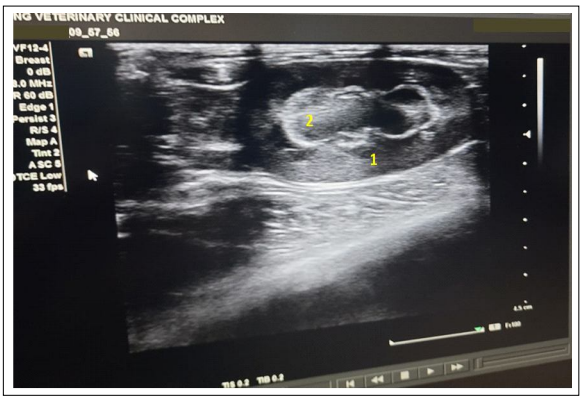


Fig 4: Ultrasonographic examination of Kidney, (1) Hyperechoic and thick cortex (2) Corticomedullary junction not clear.

In young dogs with secondary renal hyperparathyroidism, the jawbones and skull are often the most severely affected. This is due to their high rate of remodeling and susceptibility to resorptive processes. The hallmark clinical manifestation of this process is "rubber jaw"- a syndrome characterized by mandibular pliability, loose teeth and facial bone deformation (Svanberg *et al.,* 1973). In contrast, adult dogs more frequently present with generalized osteopenia. Hazewinkel (1989) observed that hyperostotic osteodystrophy of the jaw is more frequently encountered in juvenile canines, as seen in our 7-month-old Labrador puppy. Comparable cases have been previously reported by Norrdin (1975) in a 5-month-old Great Dane and by Rusenov *et al.* (2010) in a 4-month-old Pug, both exhibiting similar clinical and radiographic features, including facial swelling, tooth mobility and mandibular demineralization.

Chronic renal failure (CRF) is more commonly reported in older dogs, particularly those over seven years of age, with an estimated prevalence of 0.5-1% (Polzin, 2011; Ahmad *et al*., 2023). However, juvenile-onset CRF can occur

Table 2: Biochemical parameters.

Parameters	Value	Reference range	Interpretation
SGPT/ALT (U/L)	112	10-109	
SGOT/AST (U/L)	22.6	13-15	
Direct Bilirubin (mg/dL)	0.18	0-0.1	
Indirect Bilirubin (mg/dL)	0.12	0-0.3	
Total Bilirubin (mg/dL)	0.3	0-0.4	
BUN (mg/dL)	149.8	8-28	Azotemia
Creatinine (mg/dL)	8.3	0.5-1.7	
Phosphorus (mg/dL)	11	2.9-5.3	Hyperphosphatemia
Calcium (mg/dL)	4.5	9.1-11.7	Hypocalcaemia
PTH (pg/mL)	32.3	5-25	Hyperparathyroidism

*2016: Hematologic reference ranges, 11th edn. The merck veterinary manual.

in the presence of congenital renal dysplasia, immune-mediated nephritis, or nephrotoxic insults. In the present case, the puppy was positive for *Babesia gibsoni*, a haemoprotozoan parasite known to induce systemic illness and multi-organ complications, including renal involvement. Juvenile renal disease and associated metabolic disorders may also be arises from congenital, nutritional, or endocrines etiology. Recognizing these alternative etiologies is important to distinguish the direct and indirect effect of babesiosis on renal and skeletal function. Canine babesiosis is endemic in many parts of India and is caused by intraerythrocytic protozoa transmitted by *Rhipicephalus* spp. ticks. Infected dogs commonly present with fever, hemolytic anemia, thrombocytopenia and organ dysfunction (Kelly *et al*., 2015; Khan *et al*., 2025). Among complications, renal failure is one of the most prevalent and potentially fatal outcomes (Winiarczyk *et al.,* 2017). Mechanisms of renal injury in babesiosis may include immune-complex-mediated glomerulonephritis, hemoglobinuria-induced nephropathy due to hemolysis, renal hypoperfusion and direct tubular injury secondary to hypoxia and inflammation.

The early clinical signs of CKD include polyuria and polydipsia, which result from the inability of the damaged nephrons to concentrate urine, leading to compensatory water intake (Bartges, 2012). As renal function declines, systemic manifestations such as anorexia, vomiting, weight loss, muscle wasting and lethargy become evident (Foster, 2013). These clinical signs may be due to the accumulation of uremic toxins, development of metabolic acidosis and uremic gastritis (Quéau, 2012). The progressive cachexia observed in the present case is likely multifactorial-linked to anorexia, reduced nutrient absorption, insulin resistance and chronic inflammation (Oburai *et al.,* 2015).

The ultrasonographic findings in the present case-such as increased cortical echogenicity, thickened cortices and loss of cortico-medullary junction (CMJ)- are classic sonographic markers of chronic renal pathology and are consistent with descriptions in the literature (Oburai *et al.,* 2015; Waller *et al.,* 2019). These findings further substantiate the chronicity and severity of the renal insult.

Hematologically, the dog presented with macrocytic hypochromic anemia. In both babesiosis and CKD, anemia is a frequent finding. In babesiosis, hemolysis is direct due to parasitization of erythrocytes, while in CKD, anemia is multifactorial-stemming from reduced erythropoietin production, bone marrow suppression, shortened erythrocyte lifespan, gastrointestinal bleeding and poor nutritional status (Oburai *et al.,* 2015). Biochemically, elevations in BUN, creatinine and phosphorus levels reflect reduced glomerular filtration rate (GFR) and impaired excretion of nitrogenous and phosphate wastes (Ettinger and Feldman, 2010; Kaneko *et al.,* 2008). Concurrent hypocalcaemia and elevated serum PTH levels confirm the diagnosis of renal secondary hyperparathyroidism, in agreement with previous reports (Chew *et al.,* 1989; Weller *et al.,* 1985).

The constellation of findings in this case-including "rubber jaw," marked facial osteolysis, hyperphosphatemia, hypocalcaemia, elevated PTH and renal ultrasonographic abnormalities-conclusively point towards fibrous osteodystrophy secondary to chronic kidney disease, with an association noted between canine babesiosis and renal pathology. Although fibrous osteodystrophy secondary to renal hyperparathyroidism has previously been reported in Indian canines (Turkar *et al.,* 2017), the present case is the first to document this condition as a sequela to *Babesia gibsoni*-induced chronic kidney disease in a young Labrador Retriever, thereby highlighting a novel etiopathological association. This case underscores the complex interplay between infectious diseases and metabolic bone disorders, particularly in young animals where early diagnosis and intervention are critical.

CONCLUSION

This case report describes a rare clinical presentation of fibrous osteodystrophy ("Rubber Jaw Syndrome") secondary to renal hyperparathyroidism in association with *Babesia gibsoni* infection in a Juvenile Labrador Retriever. Combined assessment of clinical findings, haemato-biochemical parameters, hormonal evaluation and diagnostic imaging confirmed renal secondary hyperparathyroidism. Association of *Babesia gibsoni* infection might have acted as a precipitating factor for renal insufficiency and metabolic bone disorders. This report highlights the novel etiopathogenesis and emphasizes the importance of a multimodel diagnostic approach while managing skeletal deformities associated with systemic involvement.

STATEMENTS AND DECLARATIONS

Consent to publish

The authors took necessary consent from the owner of the animal for publication of the clinical details, images included in this manuscript.

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Author contribution

Utpal Barman conceptualized, supervised the case study and assisted in diagnosis and treatment. Arup das and Abhijit deka helped in diagnosis, treatment. Pallabi devi and Mousumi Hazorika contributed in laboratory investigation and data compilation. Bendangla Changkija and Shasanka Sandilya prepared and reviewed the manuscript. Anindita Sandilya and Ritam Hazarika assisted in literature review and clinical interpretation.

Conflict of interest

The authors declare no conflict of interest.

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