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Mandibular fracture in a Lhasa Apso with renal secondary hyperparathyroidism

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SUMMARY

A three-year-old Lhasa Apso was presented with a manibular fracture following a fight with another dog. The physical examination revealed generalised excessive mobility of the entire dentition. Radiographs revealed generalised mandibular demineralisation. Routine laboratory analyses revealed severe anaemia and azotaemia, compatible with chronic renal disease. The findings were considered consistent with renal secondary hyperparathyroidism resulting in bone demineralisation and minimal-trauma fracture.

Keywords: dog, jaw, hyperparathyroidism, renal disease, osteopenia

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Introduction

A three-year-old, female Lhasa Apso was presented on an emergency basis for facial trauma following a fight with another dog, which lead to a dropped jaw, ptyalism and pain on mandibular manipulation.

Physical examination findings

The dog was quiet and alert on physical examination. No external lesions were found on the head. The jaw was partially dropped and displaced to the left. Manipulation of the mandible appeared painful and the dog was anaesthetised for further examination. Oral examination revealed moderate plaque formation, mild periodontitis with pocket formation not exceeding 3mm, and marked loosening of the teeth of both the mandible and maxilla.

Radiographs

Intra-oral radiographs were performed in order to evaluate the tooth roots and alveolar bone. These revealed a marked generalised decrease in bone density with loss of trabecular structure and absence of the lamina dura (Figs. 1 and 2). The teeth, which appeared to be freely floating in the alveolar bone, were of normal radiodensity. No evidence of receding of the alveolar bone was observed. A fracture line was apparent on the ventral border of the body of the left mandible (Fig. 3). This was barely visible because of the diminished thickness of the bone cortex and reduced bony contrast due to bony demineralisation (Fig. 4).

Laboratory examinations

Routine blood analyses revealed severe anaemia and severe azotaemia (Table 1)

	Results	Reference interval
Haematocrit (%)	14	37 – 55
Red blood cells (x10^{12}/l)	2.02	5.5 – 8.5
Leukocytes (x10^{9}/l)	6.2	6 – 17
Haemoglobin (mmol/l)	3.1	12 – 18
Urea (mmol/l)	51.1	0 – 8.5
Creatinine (µmol/l)	476	0 – 130

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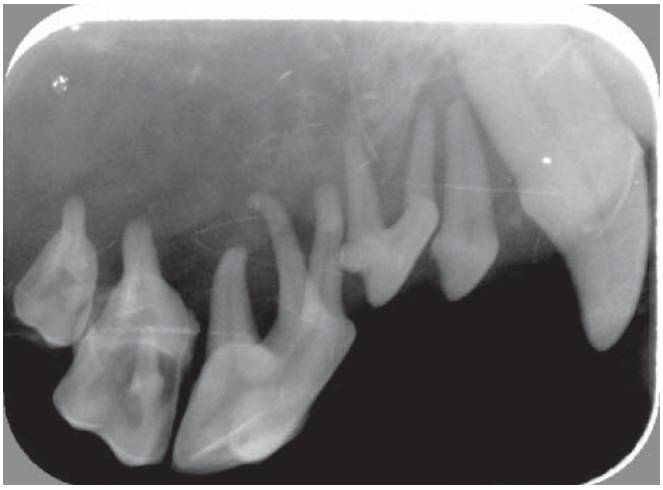

Figure 1: Intra-oral radiograph of the right maxillary arcade. The lamina dura has completely vanished around the roots of the premolar and molar teeth. The maxilla has lost its trabecular structure and bone density is severely diminished although the density of the teeth is unaffected.

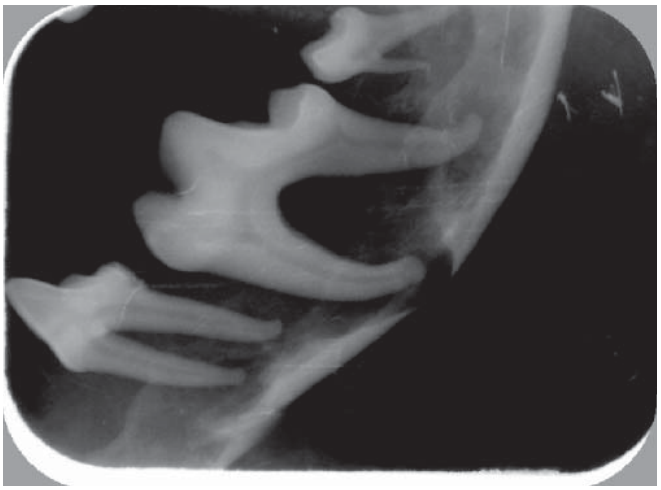

Figure 2: Intra-oral radiograph of the right manibula at the level of the fourth premolar and first two molar teeth: periapical radiolucency and loss of vertical bone structure along the tooth roots are evident. A faint old fracture line can be seen along the ventral border of the mesial cortical bone near the fourth premolar.

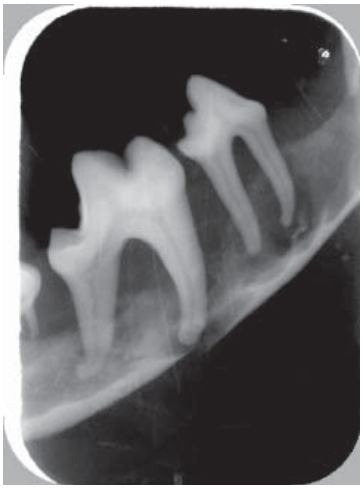

Figure 3: Intra-oral radiograph of the left mandible at the level of the fourth premolar and first two molar teeth: periapical lesions, similar to those observed in the preceding radiograph, are evident. A fracture line is visible along the mesial root of the first molar tooth.

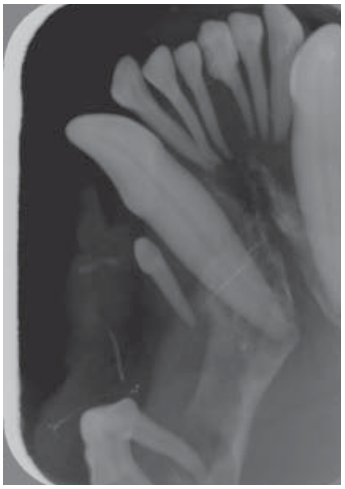

Figure 4: Intra-oral radiograph of the mandible at the level of the canine and incisive teeth: osteopenia is particularly marked around the incisive teeth. The lamina dura is still visible around the apical third of the roots of the canine teeth.

Diagnosis

Findings were considered consistent with renal secondary hyperparathyroidism.

Discussion

Hyperparathyroidism is an endocrine disorder, characterised by an excess in circulating parathyroid hormone (PTH). This results in increased resorption of calcium from bone due to stimulation of osteoclastic activity. Primary hyperparathyroidism is due to a primary dysfunction of the parathyroid glands; secondary hyperparathyroidism can be nutritional, due to an excessively low calcium-to-phosphorus ratio in the diet or renal, due to chronic renal insufficiency (CRI). [Garcia-Rodriguez M.B. *et al*] To confirm the diagnosis, laboratory evaluation in this case should have been completed with serum calcium and phosphorus levels, PTH levels, and a urinalysis. [Polzin D.J. *et al*]

Renal secondary hyperparathyroidism is caused by a deficient metabolism of vitamin D in CRI. This secondary hypovitaminosis D leads to hypocalcaemia, which results in a compensatory increase in parathyroid production of PTH. Decreased glomerular filtration and impaired renal excretion of phosphorus in CRI exacerbate the hypocalcaemia. [Fukagawa M. *et al*]

Evidence of bone demineralisation is generally seen first in the mandible, then the maxilla and other bones of the head, followed by the remaining bones of the axial and appendicular skeleton. Both maxiallary bones are generally severely demineralised before signs of osteopenia are apparent in the rest of the skeleton. [Ziólkowska H. *et al*] This can lead to a pliable jaw, [Wiggs R.B., Lobprise H.B.] referred to as rubber jaw. Boney demineralisation of the jaw is evident on radiographs and characterised by marked thinning of the cortices. Osteopenia of the jaw may be so severe that a very marked increase in radiographic contrast between bone and tooth is apparent, since teeth are the only hard tissues not affected by demineralisation. One of the most manifest changes is the complete disintegration of the lamina dura. Lastly, the trabecular structure of the bone tissue vanishes, leaving a homogenous radiographic image. [White S.C., Pharoah M.J.].

The teeth are normally maintained in place by Sharpey's fibres, which constitute the fibrous part of the periodontal ligament. These fibres form the external layer of the dental roots, anchored in the cementum on one side and the alveolar bone, on the other. Histological studies demonstrate that these fibres remain anchored in the cementum but the insertion into alveolar bone is practically lost in hyperparathyroidism. [Harvey C.E., Emily P.E.]. This explains the loosening of teeth observed in this disorder.

Chronic renal disease is common in aging animals but is infrequent to rare in young animals. A bilateral renal fibrosis, referred to as progressive juvenile nephropathy, has been described in young dogs of various breeds. [Camichael D.T. *et al*]. In addition, renal dysplasia is recognised in some breeds, including the Golden Retriever and Shih Tzu. [Ohara K. *et al.*) No biopsy or post-mortem examination was performed in the dog in the present case report and a suspicion of a progressive juvenile nephropathy remains speculative.

The owners of the dog in this report did not suspect disease in their pet prior to presentation, and only retrospectively were aware of a progressive increase in water intake and urination. Surprisingly, the dog was fed exclusively on a dry food, which it managed to eat despite the weakness of the jaw. Based on the laboratory findings, osteopenia and jaw fracture, a poor prognosis was given, and the owner elected for euthanasia.

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