An unusual case of nutritional Secondary hyperparathyroidism in a Mare- A case report

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ABSTRACT
A case of nutritional secondary hyperparathyroidism in a mare was diagnosed on the basis of history, clinical signs, radiography and reduced calcium excretion in urine.

Key words: Diagnosis, Mare, Nutritional secondary hyperparathyroidism.

Equine nutritional secondary hyperparathyroidism (NSHPT) results from feeding a ration with an improper calcium:phosphorus ratio, which causes hypersecretion of parathyroid hormone (PTH) and results in metabolic bone disease (fibrous osteodystrophy, bran disease, miller’s disease, big head, osteodystrophy fibrosa). This disease is characterized by intermittent shifting lameness, stiff, creaking joint and enlargement of facial bones (Rose and Hodgson, 1993). This disease is not reported in India. This communication presents an unusual case of NSHPT in a mare with normal facial bones and the diagnosis was made on the basis of anamnesis, clinical signs, radiography and measurement of excreted calcium in the urine.

Case history and observations: An 11 years old mare was brought to the Teaching Large Animal Clinic of the University (OPD NO. 5765, dated 15/11/16) with a history of shifting lameness for last two years and exclusively feeding on a grain rich diet, in particular with excessive amount of wheat and rice bran without mineral supplementation. Treatment with analgesics for lameness suggested by local veterinarian was unsuccessful.

Clinical examination of forelegs revealed swollen and painful knee joints (fig.1) and stiff gait. The facial bones were normal (fig 2). Haemogram depicted haemoglobin-7.8gm%, total leucocyte count-12.68x10^3/mL, packed cell volume-22%, differential lymphocyte count: neutrophils-78%, lymphocyte-20%, monocytes-0%, eosinophil-2% and basophil-0%. Blood smear examination for haemoprotozoa was negative. VITROS DT-II Chemistry system (Ortho-Clinical Diagnostics, Johnson and Johnson Company) was used for estimation of serum biochemical parameters. Serum biochemistry values included alkaline phosphatase- 170 IU/ L, serum calcium- 11.6 mg/dl and serum phosphorus- 2.9mg/dl. Medio-lateral radiograph of left carpal joint revealed irregular carpal bones with reduced bone density.

Criteria of diagnosis: The urine calcium concentration (4.09 mmol/L) was estimated by VITROS DT-II Chemistry system (Ortho-Clinical Diagnostics, Johnson and Johnson Company) and specific gravity of urine (1.030) was estimated by refractometer and adequate dietary calcium was calculated on the basis of following equation (Rose and Hodgson, 1993).

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\text{Calcium excretion (mol/mosmol)} = \frac{\text{Urine Ca concentration (mmol/L) x 0.04}}{\text{Specific gravity – 0.997}}
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Where, mmol/L= milimol per litre

Calcium excretion value greater than 15 mol/ mosmol indicates adequate dietary calcium (Rose and Hodgson, 1993). Whereas reduced calcium excretion value (4.96 mol/mosmol) indicated inadequate dietary calcium in this case.

Treatment and Discussion: Oral administration of calcium carbonate mixture @ 50 gm/ day, green fodder like Berseem and reduce quantity of grains in diet were advised for one month as treatment. After a month, marked reduction in shifting lameness was reported by owner and he was further advised to continue the same treatment for next one month.

In present case, the most important component of anamnesis was feeding of excessive grain including wheat bran with intermittent lameness. Grains with high phosphorus content and a higher phosphorus/calcium ratio often result in excessive absorption of phosphorus with respect to calcium and decreased absorption of calcium (Ronen et al. 1992; Reed et al. 2004). Excessive amounts of phosphorus in the intestinal tract reduce calcium absorption and may result in hyperphosphatemia, which stimulates parathyroid gland hormone (PTH). PTH increases the rate of bone resorption and inhibits reabsorption of phosphorus by the kidney until calcium and phosphorus levels are normal (Hester et al. 2002, Estepa et al. 2006). The normal serum calcium and

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phosphorus levels in this case may be due to the homeostatic mechanisms that regulate extracellular calcium concentration at the expense of the skeletal reserves. Early clinical signs usually involve intermittent shifting lameness and stiff gait. Loss of bony support of the articular cartilage of diarthrodial joints has been implicated as a cause of lameness related to the resultant joint pain (David et al., 1997). The classical sign of NSHPT is swollen facial bones (Radostits et al., 2000). But, in the present case the facial bones were found to be normal. Lower metabolism rate in bone in old animals might be the reason for normal facial bones. Radiography of affected joint showed decrease in bone mineralization which is believed to be decreased density of the laminae durae dentes, presumably because of their thin, discrete radiographic appearance and their rate of osseous turnover (David et al., 1997). Decreased excretion of calcium in urine is the most sensitive clinical pathology measurement in diagnosis of NSHPT (Rose and Hodgson, 1993). David et al. (1997) recommended feeding a ration that contains a calcium: phosphorus ratio of at least 4:1 and calcium carbonate as an effective management of NSHPT.

REFERENCES