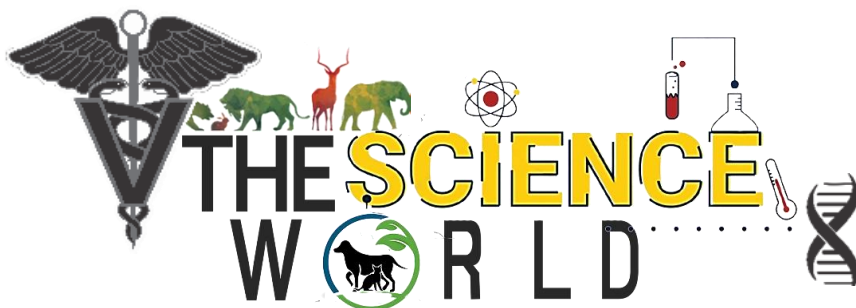




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Bran Disease (Fibrous Osteodystrophy): A Common Bone Disorder in Stall-Fed Goats

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Introduction:

Fibrous osteodystrophy (also called osteodystrophia fibrosa or osteitis fibrosa cystica) is a metabolic bone disorder caused by an imbalance of calcium and phosphorus, usually due to low dietary calcium, excessive phosphorus intake, or prolonged overactivity of the parathyroid glands. This imbalance stimulates continuous secretion of parathyroid hormone, which increases bone resorption to maintain blood calcium levels. As minerals are removed from the bones, they become weak and are gradually replaced by fibrous connective tissue, resulting in bone deformities, swelling, fractures, and reduced skeletal strength. Although the condition is most common in horses, it also occurs in goats, pigs, dogs, cats, and other animal species. It is observed that young goats maintained under stall-feeding systems and fed predominantly grain-based diets are particularly prone to developing nutritional fibrous osteodystrophy.

Etiology:

The main cause behind this disease is state of hyperparathyroidism in animal's body. This hyperparathyroidism could be due to primary parathyroid gland lesion or secondary renal or nutritional in origin. Nutritional secondary hyperparathyroidism, commonly resulting from diets containing excessive phosphorus—particularly from wheat bran (chunni)—is considered the main cause of fibrous osteodystrophy in goats. A decreased calcium-to-phosphorus ratio is recognized as a key indicator of this disorder.





Fig: Wheat bran used for feeding goats

Pathophysiology:

Fibrous osteodystrophy develops as a consequence of a prolonged imbalance in calcium and phosphorus metabolism. Diets containing excessive phosphorus or inadequate calcium result in a reduced calcium-to-phosphorus ratio, causing a relative or absolute deficiency of available calcium in the blood. In response to falling blood calcium concentrations, the parathyroid glands increase the secretion of parathyroid hormone (PTH), producing a condition known as secondary hyperparathyroidism.

Persistently elevated PTH stimulates osteoclast-mediated bone resorption to restore normal blood calcium levels. Continuous mobilization of calcium from the skeleton causes progressive loss of bone minerals, resulting in demineralization and weakening of the bones. Over time, the resorbed bone is replaced by fibrous connective tissue instead of normal mineralized bone, leading to skeletal deformities, enlargement of affected bones, reduced bone strength, and an increased risk of fractures. Hyperphosphatemia further aggravates the mineral imbalance and contributes to the progression of the disease.

Clinical Findings:

- Bilateral, symmetrical enlargement of the mandible and facial bones, without signs of inflammation.
- Inability to completely close the mouth due to jaw deformity.
- Persistent protrusion of the tongue from the oral cavity.
- On palpation, the mandibular bones may feel firm initially, while advanced cases may exhibit a soft, rubber-like consistency because of extensive bone loss.
- Loosening or displacement of teeth, resulting in difficulty grasping and chewing feed.



- Excessive salivation, often appearing as frothy drool around the mouth.
- Difficulty in mastication, leading to reduced feed intake, weight loss, and progressive emaciation.
- Prominent or bulging eyeballs caused by expansion of facial bones.
- Bow-legged stance, particularly in young kids, due to weakening of the limb bones.
- Abnormal gait, lameness, and reluctance to move.

Diagnosis:

Diagnosis of fibrous osteodystrophy is based on clinical signs, serum biochemical findings, and radiographic examination. Affected goats typically show decreased serum calcium, elevated serum phosphorus, and a reduced calcium-to-phosphorus ratio, while cephalometric radiographs reveal generalized radiolucency of the facial bones due to loss of bone mineralization.

Treatment and management:

Treatment of fibrous osteodystrophy focuses on correcting the calcium–phosphorus imbalance, controlling secondary hyperparathyroidism, and promoting bone mineralization. A therapeutic regimen may include intravenous calcium gluconate (or calcium preparations such as Calcium Sandoz®), oral calcium supplementation, phosphate binders (e.g., kaolin, aluminium hydroxide, sevelamer carbonate) to reduce phosphorus absorption, vitamin supplementation (A, D₃, and E or ADEH) to support calcium metabolism, and an anabolic steroid such as nandrolone to enhance bone formation and mineralization. Treatment should be continued for several weeks along with dietary correction by providing an appropriate calcium-to-phosphorus ratio. Feeding of bran and barley should be discontinued immediately.

Takeaway for goat farmers:

Osteodystrophy fibrosa in goats is mainly a secondary nutritional hyperphosphatemia due to excessive feeding of Phosphorus rich diet mostly wheat bran. Early detection of the condition, maintaining the dietary calcium-to-phosphorus ratio at approximately **2:1**, minimizing excess phosphorus intake through the use of phosphate binders, and avoiding



Fig: Facial swelling in young goat



feeds containing calcium-binding compounds are important measures for preventing fibrous osteodystrophy.

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