FETAL CONGENITAL GOITER IN GOAT: A CASE REPORT

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ABSTRACT

A Case report of a pregnant doe aged 14 months old enabled to deliver fetus was presented in the Referral Veterinary Polyclinic, ICAR-Indian Veterinary Research Institute, Bareilly. Doe fails to deliver the foetus due to a large increment in the size of the foetus, resulting in dystocia. The doe was treated with PGF_2 alpha, estrodiol and velathamate which lead to the opening of the cervix. The presentation of foetus was brought in to normal in the uterus and a dead fetus was successfully removed. The dead fetus had a large swelling in the cranio-ventral neck region with the skin devoid of hair, pale-white and thickened with myxedema indicating congenital goitre. The most prevalent cause of congenital goitre in kids is low iodine intake or dietary iodine deficiency during the third trimester of pregnancy, which results in the birth of a dead or stillborn foetus. Iodine deficient diets, goitrogenic substances that interfere with thyroxinogenesis and excess dietary iodine are the main pathogenic pathways responsible for thyroid hyperplasia. This communication reports a case of dystocia due to congenital goiter in a goat. The defective fetus was delivered successfully by changing its presentation inside the uterus.

Keywords: Goitre, hypothyroidism, thyroid hyperplasia, Goitre Kids, dystocia

INTRODUCTION

The goiter is non-neoplastic and non–inflammatory enlargement of the thyroid. Goiter is one of the rare causes of head and neck masses in newborns, which is observed in one case per 40 000 births (Kobayashi et al., 2017). It can affect any domestic mammal, bird, or other vertebrate, although it's most commonly seen in goat youngsters in endemic locations (Hasan *et al*., 2013). Congenital goiter has been reported mostly in newborn animals to dams with insufficient iodine consumption or failure to obtain dietary iodine, and is of greater economic importance than in adults due to substantial economic losses (Blood, 2000 and Singh and Beigh, 2013). Iodine deficient diets-primary goiter; goitrogenic chemicals that interfere with thyroxinogenesis are the main causes of thyroid gland hypertrophy (brassica plants, soybean byproducts and water with high content of calcium and nitrates) - Secondary goiter, which can result from an excess of dietary iodine or genetically determined hereditary enzyme abnormalities in the manufacture of thyroid hormone (Radostits *et al.,* 2007).

Congenital goiter can occur due to continuous feeding of diets deficient in iodine like Subabul (Leucaenaleucocephala) (Sastry and Singh, 2008). Premature kidding also occurs due to goitrogenic plant (Honparkhe *et al*., 2017). The main pathogenic mechanisms liable for the event of thyroid hyperplasia include iodine deficient diets, goitrogenic compounds that interfere with thyroxinogenesis, excess dietary iodine, and genetically determined defects with the enzymes liable for the biosynthesis of thyroidal hormones. These mechanisms end in inadequate thyroxine synthesis and decreased blood concentrations of thyroxine (T4) and triiodothyronine (T3) (Capen, 1995). This is often detected by the fetal hypothalamus and pituitary, stimulating a rise in thyroid stimulating hormone (TSH) production, which ends up in hypertrophy and hyperplasia of follicular cells of the thyroid. The goiter is caused by thyroid enlargement because it tries to supply the thyroid hormones needed by the animal. In utero, goiter is caused by either primary or secondary iodine deficiency (Maxi, 2007 and Kotwal *et al*., 2007).

Most cases of congenital hypothyroidism are related to multiple late-term abortions, stillbirths, or early postnatal death. Animals born by dams being on iodine deficient diets are more likely to develop severe thyroid hyperplasia and have clinical evidence of hypothyroidism. In most cases, the sole gross lesion evident in aborted or neonatal animals may be a bilateral enlargement of the thyroid glands. Congenital goiter with alopecia and myxedema was diagnosed on the basis of gross appearance (Cheema *et al.,* 2010; Navdeep *et al*., 2019). Primary goiter is caused by deficient dietary iodine intake whereas secondary goiter is caused by interference with dietary uptake of water with high content of calcium, nitrates, goitrogenic plants (Brassica sp. and a few clovers). Dystocia due to fetal goiter in doe has also been reported by (Cheema *et al*., 2010; Kumar *et al.,* 2014). This is the case report of a pregnant doe giving birth to a stillborn fetus with congenital goiter which was presented in the Referral

Veterinary Polyclinic, ICAR-Indian Veterinary Research Institute, Bareilly

CASE HISTORY AND OBSERVATION

A crossbred doe aged 14 months was brought to Referral Veterinary Polyclinic, ICAR-Indian Veterinary Research Institute, Bareilly, U.P with the complaint that after completing the full term there were no signs of kidding. Per vaginal examination revealed that the cervix was not dilated; so, the animal was treated for incomplete cervical dilation. The doe failed to deliver even after the treatment with PGF_2 alpha and Estradiol but per vaginal examination revealed that the cervix was dilated and the fetus was without any reflex, was in anterior longitudinal presentation with severe lateral deviation of the head.

On gross examination, the skin of the dead kid was pale, thick with myxedema and without hair (Fig. 3). Congenital goiter with alopecia and myxedema was diagnosed on the basis of the gross picture.

After pervaginal examination the doe was treated for incomplete cervical dilation with PGF_2 alpha@250 mg, estrodiol @2-3mg, dexamethasone @ 2-5 mg and valethamate @ 10-20 mg/kg body weight intramuscularly. After 24 hours, vaginal examination revealed dilatation of the cervix with the fetus in the anterior longitudinal presentation without any reflex. However, the doe was unable to deliver by itself. Adequate lubrication of the birth canal with liquid paraffin and gentle traction (Figure1) resulted in the successful delivery of the dead fetus (Figure2). The goat was discharged with the routine prescription of antibiotics and supportive therapy.

CASE DISCUSSION

Congenital goitre is a non-inflammatory and non-neoplastic enlargement of the thyroid gland in the foetus and is regarded as a common anomaly in goats (Ani *et al*., 1998). The condition is accompanied by an increase in the size of the foetus, myxedema, prolonged gestation and dystocia (McDonald and Pineda, 1989). Low iodine intake or failure to get dietary iodine is the common cause of congenital goiter in kids (Paulikova *et al.,* 2002). The major pathogenic mechanisms responsible for thyroid hyperplasia include iodine deficient diets, feeding of goitrogenic compounds that interfere with thyroxinogenesis, more than required dietary iodine. These mechanisms result in inadequate thyroxine synthesis and decreased blood concentrations of (T4) and (T3). The hypothalamus and pituitary gland, stimulate an increase in (TSH) production, which results in hypertrophy and hyperplasia of follicular cells of the thyroid (Capen, 1995). Congenital hypothyroidism is associated with multiple late-term abortions, stillbirths, or early postnatal deaths (Jones *et al.,* 1997). The kid born with Congenital goitre is generally presented with certain gross lesions like swelling on the ventral region of the neck, partial or complete alopecia of the body coat. The soft and thickened fetal skin when cut revealed myxedema. The removal of skin from the swelling within the neck revealed two massive lobes of the thyroid. These may be firm, solid and dark brown to red in colour.

Tissues from different areas of the thyroid, lungs, liver, kidneys and heart can be processed by the routine histological procedure. Tissue sections are cut at 4 µm thickness and stained by routine hematoxylin and eosin method for histopathology (Bancroft and Gamble, 2007). Thyroid tissue consists of well-developed follicles of various sizes and shapes and is lined by one layer of cuboidal epithelium. Marked variation can be noted within the contents of follicles. Most of the follicles crammed with dense and dark-red colloid (thyroglobulin) and lined by flat cuboidal cells, but many follicles have a light-weight pale or dirty brownish colloid. When the follicles are depleted of thyroglobulin, the liner epithelial cells will be elongated and move towards the centre of the follicles and give a collapsed appearance. Within the lungs, the pleura and interlobular septae may also be thickened with a light-pink proteinaceous material. Similar materials are also present within the alveoli and in blood vessels. In most cases, the only gross lesion evident in aborted or neonatal animals is the bilateral enlargement of the thyroid glands. Because of the variation in the size and appearance of normal thyroid glands, histopathology must be used to confirm goitre in most cases (Capen, 1995). Ultrasonography of the thyroid gland has been frequently reported in human literature but has not been done in animals. Congenital hypothyroidism can be diagnosed during pregnancy or at birth_(Mastrolia *et al*.,2015). Possible treatment for kids, with a deficiency of T4, is Levothyroxine sodium, which is commonly used in men, has also been studied in kids (Ommaty, 2000; Ozmen *et al.,* 2005). Lambs with goitre have been successfully treated with 20 mg potassium iodide per os, once which is the cheapest and safest source of iodine which also give good results (Constable *et al*., 2017). supplementation of Lugol's iodine in drinking water during the last month of gestation can also help the farmer to avoid iodine deficiency and such animals had normal kidding (Reddy *et al*., 2016). Ozmen *et al.,* (2005) observed no congenital case of goitre when owners were advised to add potassium iodide (KI) to the feed if the dams are fed cabbage during pregnancy. Congenital goitre in a kid can successfully be treated by the use of oral administration of KI if diagnosed early.

CONCLUSION:

In this case enlarged thyroid glands deviated the head of the foetus, resulting in dystocia. The rotation of the foetus inside the uterus to change its presentation, however, beneficial in the delivery of the kid. The congenital goitre is revealed by an enlarged thyroid with myoxedma, which is caused by dietary inadequacies or the feeding of goitrogenic chemicals in the third trimester of pregnancy.

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Figure1: Removal of the fetus by gentle traction

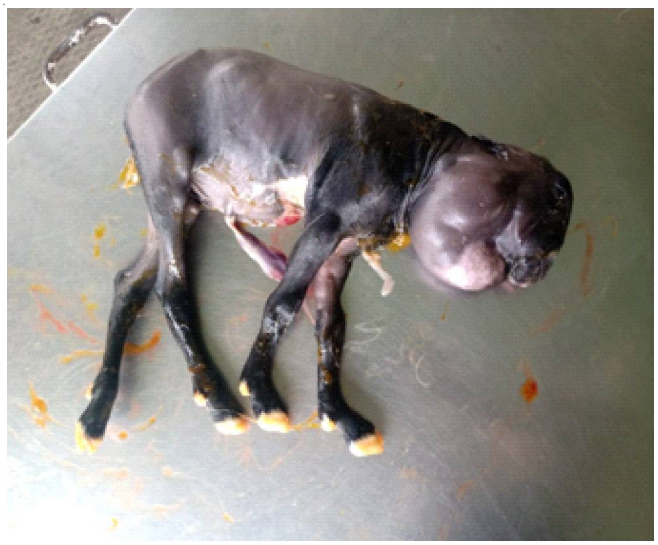

Figure 2: A dead kid

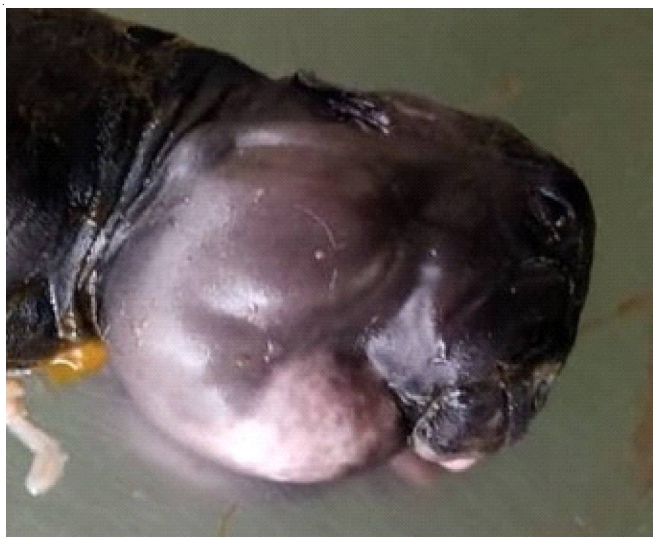

Figure 3: Dead kid with alopecia and myxedema