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Familial Congenital Hyperplastic and Colloidal Goitre in a Beetle Goat

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ABSTRACT

n investigation was conducted at the Veterinary Clinical Complex, College of Veterinary Science, COVS, GADVASU 🔼 Rampura Phul, Punjab, India during May, 2023 to study the pathomorphological alterations of Hyperplastic and Colloidal Goiter in a beetal. Two new born kids were considered that included one dead and other with history of no suckling, lassitude, not able to rise its head and swelling in cranioventral neck region. Post mortem examination of dead kid showed markedly swollen thyroid gland and samples collected from them were subjected to routine H&E examination. Histopathological examination revealed extensive hyperplasia of the thyroid follicles along with follicles filled with varying amount of colloid suggesting hyperplastic and colloidal goitre. Areas of Hyperplastic follicles showed cuboidal to tall columnar follicular epithelial cells with highly vacoular cytoplasm whereas follicles with colloidal deposition showed flattened epithelium. Hemato-biochemical examination of samples collected from live kid showed moderate anaemia with decreased leucocyte count. There was macrocytic hypochromic anaemia. T, was significantly lower and there was significant rise in TSH levels when compared to reference values. The kid born co-twin with the dead kid survived after treatment and showed significant improvement in clinical condition after 5 days of treatment. Based on the gross and histopathological examination the cause of death was confirmed to be congenital hyperplastic and colloid goitre which is usually a non-inflammatory and non-neoplastic enlargement of the thyroid caused due to iodine deficiency is most common in new-born animals in iodine-deficient areas.

KEYWORDS: Colloidal, goat, goitre, hyperplastic, thyroid gland

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Data Availability Statement: Legal restrictions are imposed on the public sharing of raw data. However, authors have full right to transfer or share the data in raw form upon request subject to either meeting the conditions of the original consents and the original research study. Further, access of data needs to meet whether the user complies with the ethical and legal obligations as data controllers to allow for secondary use of the data outside of the original study.

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1. INTRODUCTION

Noitre is non neoplastic and non-inflammatory **J**enlargement of thyroid gland. 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). Thyroid gland is very essential that controls the metabolism (Barrea et al., 2021). Kids have similar picture of goitre as that of lambs but degree of severity is more (Constable et al., 2017). Goitre in utero is caused due to either primary or due to secondary iodine deficiency (Maxi, 2007; Nourani and Sadr, 2023. The major factors responsible are primary goitre-iodine deficient diets, secondary goitre -goitrogenic compounds that interfere with thyroxinogenesis (brassica plants, soybean byproducts and water with high content of calcium and nitrates), in rare cases excess of dietary iodine and genetically determined inherited enzyme defects responsible for biosynthesis of thyroidal hormones (Constable et al., 2017; Jamshidi, 2022).

Iodine has a principal role in the production and secretion of triiodothyronine (T_3) and thyroxine (T_4) (Smith and Sherman, 2009; Campbell et al., 2012). T_3 and T_4 are very important for maintaining control over metabolism. Shortages or excesses of T_3 or T_4 have very pronounced effects on the affected animal which may result in fatality. Goitre is frequently visible in kids and lambs after birth. Due to poor thermoregulation, reduced secretion of surfactant, reduced cardiac activity, and arrhythmia, these conditions lowers the survival probability of the lambs and kids (Davoodi et al., 2022; Pathak et al., 2024). Animals with iodine deficit may also have deficiencies in vitamin A and selenium (Pearce et al., 2013).

Congenital goitre is frequently reported in new born animals especially kids (Hasan et al., 2013; Blood, 2000 and Singh and Beigh, 2013). Most of the times congenital goitre are associated with late-term abortions, stillbirths or early postnatal death and the major gross lesion observed in these cases will a bilateral enlargement of the thyroid glands. Severe cases may also exhibit alopecia and myxoedema (Cheema et al., 2010; Ankita et al., 2023). Prolonged gestation, dystocia, myxoedema and a rise in the size of the fetus are signs of this disorder. (Bhardwaj et al., 2022). The pathogenesis involved in the development of congenital goitre include diets of dam being deficient in iodine, goitrogenic compounds interfering with thyroid synthesis, excess dietary iodine and in some cases genetic defects with enzymes involved in the biosynthesis of thyroid hormones. It is important to remember that certain plants, like rape, cabbage, turnips, cabbage, kale and soyabeans, have the potential to cause goiter, particularly when ingested in sufficient quantities and regarded as goitrogens (Palanivel

and Sharma, 2020; Muzzaffar et al., 2022). Furthermore, supplying food containing goitrogenic substances or an inadequate iodine ration to pregnant animals can interfere with thyroxinogenesis, which in turn increases the incidence of goiter in goat kids (Botta et al., 2017; Mihai et al., 2017). When animals are provided a diet high in calcium also leads to cause the iodine deficiency because it reduces the intestinal absorption of iodine (Joshi et al., 2017). It has been demonstrated that organo-chloride chemicals, including lithium and dichlorodiphenyltrichloroethane and related group compounds, can also cause goiter in animals (Medrano-Macías et al., 2016). These factors result in inadequate synthesis and decreased levels of thyroxine (T₄) and triiodothyronine (T_2) in the blood circulation (Capen, 1995; Singh and Kaushal, 2024). The aim of the present case report was to study the hematological, biochemical, gross and histopathological alterations in Hyperplastic and Colloidal Goiter.

2. MATERIALS AND METHODS

2.1. Anamnesis

A two-day old kid was presented to Veterinary Clinical Complex, College of Veterinary Science, GADVASU, Rampura Phul, Punjab, India in the month of May, 2023 with history of no suckling, lassitude, not able to rise its head and swelling in cranioventral neck region. On anamnesis it was found that the dam gave birth to two kids in last kidding and both died with similar signs. In present parity two kids were born and among them one died that was presented for post-mortem along with live one for diagnosis and treatment of the condition. Live as well dead kids had bilateral swelling on ventral aspect of neck.

2.2. Hematological and biochemical analysis

Blood samples were collected in Ethylenediaminetetraacetic acid (EDTA) vacutainers from the live kid and subjected to haematological analysis using haematological autoanalyzer (ADVIA 2120 Hematology System, Siemens Healthcare Diagnostics Inc. Deerfield, IL, USA and Orphee Mythic 18-VET). Similarly, some blood was collected in red vacutainer to separate serum for biochemical analysis using biochemical autoanalyzer ((M/s Fujifilm India Pvt. Ltd. a fully owned Indian subsidiary of Fujifilm Corporation, Japan, Model: NX 600 V (Veterinary)).

2.3. Histopathological examination

Detailed post-mortem examination was conducted on the dead carcass as per standard procedure and tissue samples were collected in 10% Neutral buffered formalin from the swollen thyroid gland. Tissue samples were processed by routine standard protocol and 4 μ m thick sections were prepared and stained by Haematoxylin and Eosin method.

3. RESULTS AND DISCUSSION

n Gross examination, both kids showed significant swelling in subcutaneous tissues of the neck regions (Figure 1). Of the two kids one of them died and was presented for post-mortem examination. On opening the carcass, thyroid glands on both the sides revealed marked enlargement with the left lobe measuring 6.2×6.9 cm² and right lobe 7.1×5.9 cm² in size which were more or less uniformly enlarged on both the sides and about 4 to 5 times its normal size. The enlarged glands were oval in shape, solid in consistency and showed grossly visible lobulations on its surface which were suggestive of goiter (Figure 2). Both the glands showed marked congestion. Similar findings were also observed by Singh and Kushal, 2024. Further, the haematological examination showed increased MCV and decreased MCHC values suggesting moderate macrocytic hypochromic anaemia with decreased leucocyte count. SGOT was within normal range (Constable et al., 2017) (Table 1). T_3 and T_4 values were found to be lower whereas TSH was found to be significantly high when compared to reference values (Paulikova et al., 2011). Similar significant rise of TSH and decreased T₄ levels were mentioned by



Figure 1: A two-day old dead kid with swelling in cranioventral neck region



Figure 2: Marked enlargement of both the thyroid glands

Table 1: Hematological, biochemical and thyroid hormone estimation

Parameter	Result	Reference values
Hemoglobin	$6.1~\mathrm{g~dl^{\text{-1}}}$	8–12 g dl ⁻¹
TLC	1800 μl ⁻¹	$400013000~\mu l^{1}$
Neutrophil	1224 μl ⁻¹	1000–7200 μl^{1}
Lymphocyte	504 μl ⁻¹	2000–9000 μl^{1}
Monocyte	72 μl ⁻¹	0–550 μl ⁻¹
TEC	6.42 (106 μl ⁻¹)	8–18 (106 μl ⁻¹)
PCV	20.6%	22-38%
MCV	32.08.2fl	16-25fl
MCH	9.5 pg	5.2–8 pg
MCHC	$11.2~\mathrm{g~dl^{\text{-1}}}$	$30\text{-}36 \mathrm{~g~dl^{-1}}$
Platelets	$517000~\mu l^{-1}$	$300000600000~\mu l^{1}$
SGOT	127 U l ⁻¹	60–280 U 1 ⁻¹
T_3	2.44 ng ml ⁻¹	2.82±1.01 ng ml ⁻¹
T_4	$0.3~\mu g~dl^{1}$	$8.65 \pm 1.86~\mu g~dl^{1}$
TSH	126.11 μIU ml ⁻¹	0.01–0.10 μIU ml ⁻¹

Constable et al. (2017). Low levels of plasma T₃ and T₄ causes the hypothalamus to secrete neurohypophyseal peptide THR (thyroid-releasing hormone), which in turn causes the pituitary gland to release TSH (Andrewartha et al., 1980). Thyroid hormones directly affect bone marrow progenitor cells, which in turn promotes erythropoiesis. Apart from its direct impact, it also has an indirect influence via controlling the absorption of iron, vitamin B12, and erythropoietin (Patel, 2017). Thyroid disfunction causes various morphological types of anemia, including macrocytic, normocytic normochromic, and microcytic hypochromic anemia that are commonly linked with goitre (Mehmet et al., 2012). Therefore, due to less T_3 and T_4 levels of the present case might be one of the reasons of Macrocytic hypochromic anemia that directly affects the absorption of iron, vitamin B12 and erythropoietin.

Histopathological analysis of the samples collected from the enlarged thyroid gland were processed by routine histological procedure (Bancroft and Gamble, 2007). Tissues sections of thyroid glands from both the sides showed extensive hyperplasia of the thyroid follicles and interstitial thickening. The follicles were found to highly variable in its size predominantly having a spherical shape (Figure 3). The follicles in some areas were filled with varying amounts of homogenous eosinophilic content indicating colloid and some fields showed hyperplastic follicles with thickening of interstitial tissues with no colloid in it as also been reported by Cheema et al., 2010. Colloid filled follicles were composed of a single layer of flattened

follicular epithelial cells (Figure 4). Some follicles having less amount of colloid showed cuboidal form of follicular cells. Hyperplastic follicles showed cuboidal to tall columnar follicular epithelial cells with highly vacoular cytoplasm as also reported earlier (Kumar et al., 2014; Jarad and Al-Saad, 2023). Increased interstitial tissues with highly engorged and vascular blood vessels could be observed in the areas where hyperplastic changes were prominent. Decrease levels of T3 and T4 causes hypothalamus to release thyrotropinreleasing hormone (TRH) which further release thyroid stimulation hormone by pituitary gland and prolonged stimulation by TSH causes hyperplasia and hypertrophy of follicular cells and cause hyperplastic goitre (Pathak et al., 2024). Second form of goitre i.e., colloid goitre is the involuntary phase of diffuse hyperplasia of thyroid gland (Ozmen et al., 2005). In the present study, histopathological features of both types of goitre viz., hyperplastic and colloid

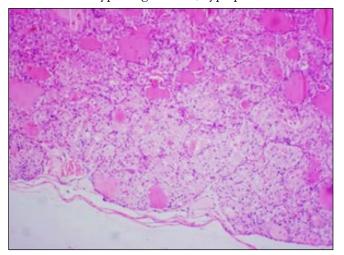


Figure 3: Thyroid gland: extensive hyperplasia of the thyroid follicles with colloid filled in few follicles. H&E-10X

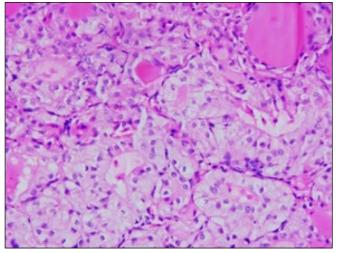


Figure 4: Thyroid gland: hyperplastic follicles with cuboidal to tall columnar follicular epithelial cells and highly vacoular cytoplasm. H&E-40X

goitre were found similar to previous studies (Reddy et al., 2016; Bhardwaj, 2018; Singh et al., 2002). The negative consequences of iodine shortage during pregnancy include retarded fetal development and weak or dead neonates with goitre that might be the reason of goitre in both the kids (Bhardwaj et al., 2022).

Treatment of the live kid showing subcutaneous swelling at neck region was started with Levothroxine sodium at the dose rate of 0.1 mg day⁻¹ orally which is the generally followed treatment for such cases (Omantry, 2000; Ozmen et al., 2005). Topically lugol's iodine was applied on the enlarged glandular part after shaving the area. Inj. concentrated iodine along with vitamin E (Inj I fer H, Carevet Pharma) was injected 0.5 ml by intramuscular route. Iodine supplementation is suggested for the treatment of goitre cases (Constable et al., 2017; Reddy et al., 2016). The kid showed significant improvement in clinical condition after 5 days of treatment. Suckling was normal along with improvement in activity of the kid. Further, the present case might be represented of familial in nature since dam had history of kidding with kids affected with goiter in previous parities also.

4. CONCLUSION

On the basis of gross, hematological, biochemical and histopathological examination of the present case, it was concluded that the kids were suffering from goitre. Based on the previous history it might be said that the goitre exhibited could be familial nature since dam has the history of kidding with kids affected with goitre in previous parties also.

5. ACKNOWLEDGEMENT

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