Haryana Veterinarian (June, 2026) **65(1),** 137-140 ISSN 0033-4359 (P)

Clinical Article

SUCCESSFUL THERAPEUTIC MANAGEMENT OF CONGENITAL BILATERAL GOITRE IN A FEMALE CALF

TARINEE RANJAN PRADHAN[1],VINITA RAJPUROHIT[1], SIRAJ ANSARI[1]* and HİMANSHU BEHERA[2]
[1]Division of Medicine, ICAR-Indian Veterinary Research Institute, Izatnagar, Bareilly, Uttar Pradesh, India
[2]Department of Animal Reproduction, Gynaecology and Obstetrics, College of Veterinary Science and Animal Husbandry, OUAT, Bhubaneswar, India

Received: 03.06.2025; Accepted: 25.02.2026

SUMMARY

Congenital goitre is a metabolic disorder in neonates, primarily caused by iodine deficiency during pregnancy, leading to thyroid hormone insufficiency. This case reports an 18-day-old female calf presenting with progressive neck swelling, inspiratory dyspnoea and signs of hypothyroidism such as weakness, unthriftiness and poor suckling reflex. The dam grazed on a mixed pasture without mineral supplementation was likely iodine deficient. Clinical examination revealed bilateral thyroid gland enlargement. Diagnostic tests including serum free T4 levels, radiography and ultrasonography confirmed congenital goitre and thyroid gland enlargement which was compressing the trachea. Haematological analysis revealed no major abnormalities. The calf was treated with Levothyroxine sodium 0.01- 0.02 mg/kg body weight orally once daily for first month of treatment and supportive therapy with prednisolone acetate@ 0.5-1 mg/kg intramuscularly for 5 days every 24 hrs and iodine supplementation through iodized salt and tincture iodine for the calf and dam. Significant clinical improvement was observed within 30 days with reduced thyroid gland size, normalization of serum free T4 levels and restored suckling reflex. This case highlights the importance of iodine in prenatal nutrition, early diagnosis and treatment with thyroid hormone replacement therapy can effectively manage congenital goitre preventing long-term morbidity in affected animals.

Keywords: Calf, Congenital goitre, Iodine deficiency, Levothyroxine

How to cite: Pradhan, T.R., Rajpurohit, V., Ansari, S. and Behera, H. (2026). Successful therapeutic management of congenital bilateral goitre in a female calf. *Haryana Veterinarian.* **65(1)**: 137-140.

Thyroid hormones are general metabolic hormones that neonates require to develop their immunological competence and perform other homeostatic functions. The thyroid hormones triiodothyronine (T3) and thyroxine (T4) control calorigenesis, basal metabolic rate (BMR) and the metabolism of fats and carbohydrates (Capen and Martin, 2003). Additionally, they impact the animal's immunological, gastrointestinal, reproductive, cardiovascular, neuromuscular, haematological and endocrine systems and they may serve as markers of nutritional and metabolic health in animal (Riviere and Papich, 2017). The major causes responsible for thyroid gland enlargement include iodine deficient diets-primary goitre; goitrogenic substances that disrupt thyroxinogenesis-such as brassica plants, soybean byproducts, and water with high calcium and nitrate content - are the main causes of thyroid gland enlargement. Cruciferous vegetables, such as cabbage, kale, cauliflower, broccoli, turnips and rapeseed, contain glucosinolates, whose metabolites compete with iodine for thyroidal uptake (Zimmermann, 2020; Radostits *et al.*, 2017). In rare instances, secondary goitre may result from an excess of dietary iodine and genetically determined hereditary enzyme abnormalities that affect the synthesis of thyroidal hormones (Agrawal *et al.*, 2015). Congenital goitre is a fatal thyroid metabolic disease marked by low thyroid hormone levels, excessive secretion of thyroid-stimulating hormone (TSH) from pituitary gland and compensatory thyroid gland enlargement (Nourani and Sadr, 2023). It is frequently seen in newborn animals to dams on iodine deficient diet, in-utero deficiency of iodine, ingestion of goitrogenic plants and hereditary (Ankita *et al.*, 2023). Iodine deficient diet is reported to be the most common cause of congenital diffuse hyperplastic goitre in calves (Homerosky *et al.*, 2019). Thyroid gland enlargement results in tracheal compression, dyspnoea and cardiovascular abnormalities leading to severe economic losses than in adults (Singh and Beigh, 2013). The inotropic and chronotropic effects of the thyroxine could be responsible for the major cardiovascular abnormalities (Naveen *et al.*, 2024). Calves are commonly weak necessitating assistance to stand and nurse for several days after birthor are stillborn (Seimiya *et al.*, 1991). Retardation of foetal development and dead or weak neonates with goitre are adverse effects of iodine deficiency during pregnancy (Zimmermann, 2020). The condition is described as 'neonatal animal hypothyroidism' with colloidal goitre, alopecia, unthriftiness, thyroid thrill and a high rate of mortality, associated with decreased serum T3, T4 levels and increased TSH activity (Hassan *et al.*, 2013; Singh *et al.*, 2003). Ramakrishna *et al.* (1992) reported that goitre as an emerging mineral disorder in animal grazed on iodine deficient endemic areas. The clinical survey in 13 districts of Uttar Pradesh and Uttarakhand revealed 58.23%, prevalence of goitre in goats. The prevalence was highest in Bareilly (69.04%)

*Corresponding author: sirajansari1998@yahoo.com

Fig. 1. Broad based stance and dyspnoeic calf

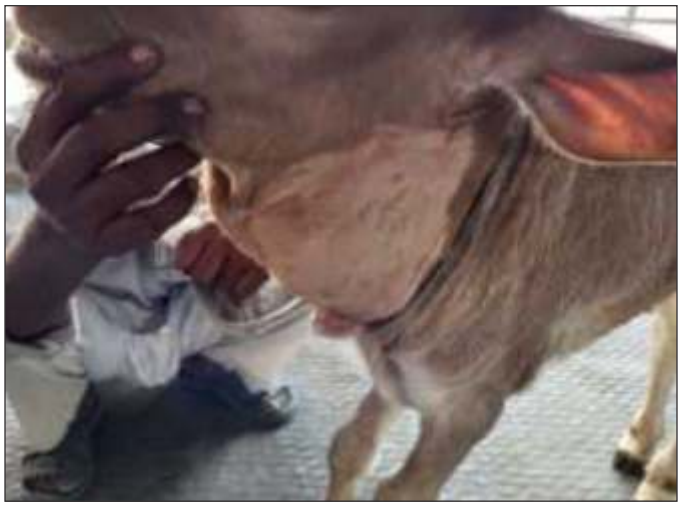

Fig. 2. Enlarged thyroid glands on presentation

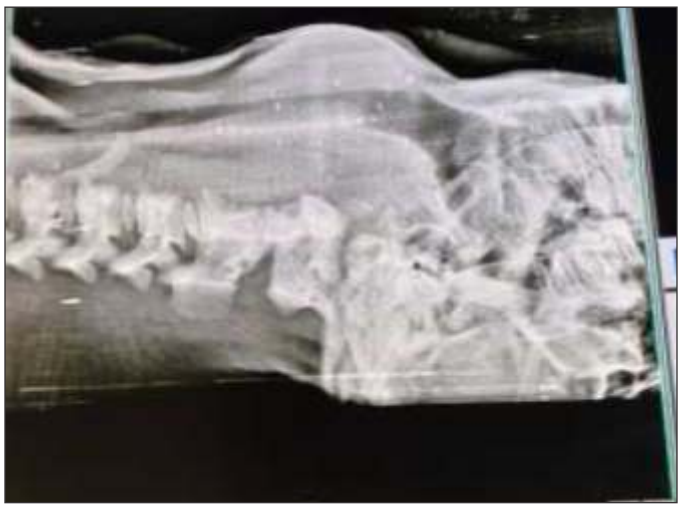

Fig. 3. Radiography of cervical region with swelling ventral to trachea

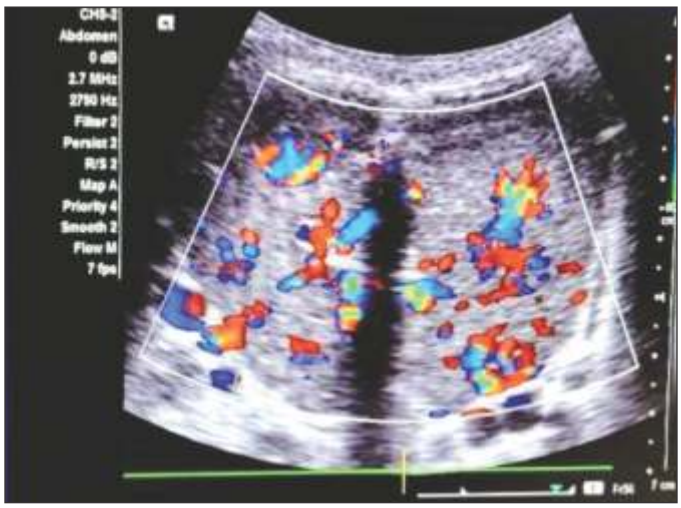

Fig. 4. Profuse central and peripheral circulation in thyroid gland

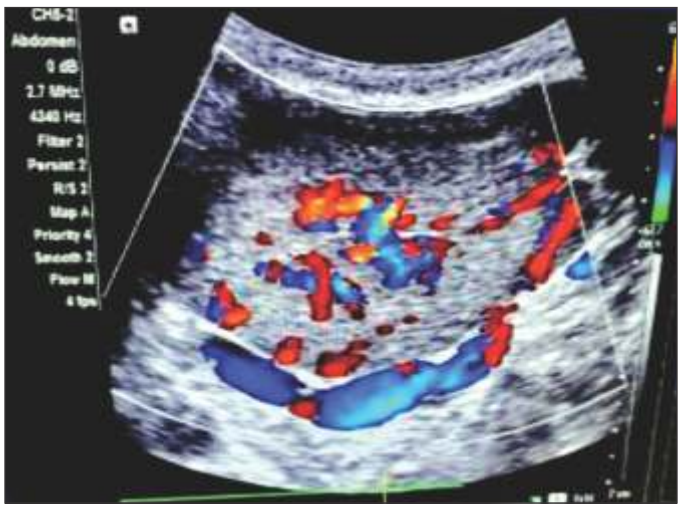

Fig. 5. Complete recovery after thyroxine medication

Fig. 6. USG revealed bilobed thyroid gland with isthmus

can be designated as goitre centre (Kumar *et al.*, 2011). The aim of the present report is to describe therapeutic management of congenital goitre in animals on endemic iodine deficient area.

A non-descript female calf weighing 32 kg of aged 18 days was brought to the Referral Veterinary Polyclinic, ICAR-Indian Veterinary Research Institute, Bareilly with progressive enlargement of throat swelling from birth,

Parameter	Day 0	Day 30	Reference Range
Haemoglobin (g/dl)	12.6	10.8	11.3 ± 1.02
PCV (%)	41.5	38.7	35 ± 3
RBC count (millions/mm^3)	8.5	8.3	8.86 ± 0.68
Total WBC count (10^3 cells/mm^3)	9.3	8.8	4.9-12
Neutrophil (%)	36	34	18–63
Lymphocyte (%)	55	57	16–56
Eosinophil (%)	4	5	0-9
Monocytes (%)	3	4	0-8
Basophils (%)	2	2	0-1
Platelet count (lakhs/mm^3)	4.7	4.6	2-6.5
Free T_4 (ng/dl)	0.09	3.1	3.3

"Table" (Haematology and serum free T4 level)
Reference Ranges are from Constable, P.D., Hinchcliff, K.,W., Done, S.H. and Grünberg, W. Veterinary Medicine: A Textbook of The Diseases of Cattle, Horses, Sheep, Pigs and Goats, Eleventh Edition, 2017.

inspiratory dyspnoea since last 5days with abdominal respiration and shallow breathing with discomfort and extension of neck, sunken eyeball and dull haircoat. Dam of affected calf appeared clinically normal and in adequate body condition. A full hair coat and expected calving date indicated the calf was likely term birth but failed to respond to the suckle reflex test. Calf seemed to have difficulty in raising the feet and the limbs were spread out so as to form a broad base of support (Fig. 1).

A feeding history was obtained from the farmer regarding Dam which grazed on mixed pasture and no free choice of loose mineral was offered. On clinical examination rectal temperature was found 103.2° F, heart rate was 50 beats per minute, respiratory rate was 66 per minute, and mucous membrane was pinkish. Animal had no history of deworming and no abnormality was detected in Defecation and Urination. There was bilateral swelling in antero-ventral region of neck below trachea which fluctuates on palpation and no pain or pits on pressure was evident. The size of thyroid gland measured as 7.6 cm × 5.3 cm × 2.5 cm (Length × Width × Height) (Fig. 2).

For investigation blood sample is taken aseptically from jugular vein and collected in EDTA vial and Clot activator vial for haematological analysis and serum free T4 test. There was no significant changes in haematology of calf. Serum free T4 level was found to be reduced from reference range to a level of 0.09 ng/dl. A lateral cervical radiograph was taken and mass of 6-7 cm that was compressing the lumen of trachea was found (Fig. 3). Ultrasonographic examination of the affected area revealed homogenous echotexture in ventral neck region ventral to carotid artery and jugular vein. There was profuse peripheral and central circulation. Both lobes were evident with presence of a stalk/isthmus between both lobes (Figs. 4 and 5).

Based on the history, clinical signs, laboratory diagnosis, the current case was diagnosed as Congenital Goitre. Calf was treated with levothyroxine sodium tablets at a dose of 0.01-0.02 mg/kg body weightorally for first month of treatment and prednisolone acetate at a dose of 0.5-1 mg/kg intramuscularly for 5 days every 24 hrs as anti-inflammatory to reduce respiratory distress. The owner was also instructed to paint cow's teat with tincture iodine and also paint throat of affected calf regularly. Iodised salt was provided orally to calf. Treatment continued for one month and marked improvement was noticed with visible decrease in size of glands and positive suckle reflex with normal appetite (Fig. 6).

Serum free T4 level was measured after one month and found to be 3.1 ng/dl which is within normal reference range. We advised owner to add potassium iodate to the feed if the dams are fed cabbage when they are pregnant. Levothyroxine sodium (Commonly used in human medicine) was found effective for treating goitre in kid with high T3 and low T4 levels (Ozmen *et al.*, 2005). Significant clinical response was observed after 15 days in tentatively diagnosed calf with goitre when treated with thyroxine with complete recovery in 20-25 days. (Kashyap *et al.*, 2015). The major pathogenic mechanisms responsible for development of thyroid hyperplasia include iodine deficient diets (Paulíková *et al.*, 2002). An iodine-deficient diet resulting in decreased thyroxine and subsequent secretion of excess thyroid-stimulating hormone (TSH) from the pituitary is the most common cause of congenital diffuse hyperplastic goitre in calves (Homerosky *et al.*, 2019; Radostits *et al.*, 2017). Dietary iodine deficiency is suspected in the current case due to the lack of mineral supplementation throughout mid to late gestation. Mountainous regions, low-height regions far from the sea, and areas with low soil iodine levels can result in iodine deficiency (Nyström *et al.*, 2016).

REFERENCES

Ankita, Kumar, R., Jamwal, S., Verma, A., Choudhary, S., Patil, R.D. and Asrani, R.K. (2023). Pathology of congenital goiter in a goat kid. *Haryana Vet.* **62(SI-2)**: 174-175.

Agrawal, P., Philip, R., Saran, S., Gutch, M. and Razi, M.S. (2015). Congenital hypothyroidism. *Indian J. Endocrinol. Metabolism.* **19(2)**: 221.

Capen, C.C. and Martin, S.L. (2003). The Thyroid Gland. McDonald's Veterinary Endocrinology and Reproduction. (5th Edn.), Iowa State Press. Blackwell Publishing Company. p. 53.

Hassan, N., Randhawa, C. and Hussian, S. (2013). Treatment of congenital bilateral goitre in a kid-a case report. *Indian J. Sci. Technol.* **1(3)**: 19-20.

Homerosky, E.R., Johnsen, M., Steinmann, M., Matejka, C. and Jelinski, M.J. (2019). An outbreak of congenital goitre and chondrodystrophy among calves born to spring-calving beef cows. *Canadian Vet. J.* **60(9)**: 981.

Kashyap, D.K., Giri, D.K. and Dewangan, G. (2015). Therapeutic management of goitre in calves. *Intas. Polivet.* **16(1)**: 88-90.

Kumar, S., Shukla, S.K., Singh, J.L. and Kumar, M. (2011). Clinico-epidemiological feature of endemic goitre in goats maintained in sub-himalayantarai of U.P. and Uttarakhand. *Indian J. Vet. Med.* **31(2)**: 69-74.

Naveen, N., Lakshmi Rani, N., Basava, Reddy, K. and Devi, Prasad, V. (2024). Clinico-epidemiological studies on hypothyroidism in dogs. *Haryana Vet.* **63(2)**: 292-295.

Nourani, H. and Sadr, S. (2023). Case report of congenital goitre in a goat kid: Clinical and pathological findings. *Vet. Med. Sci.* **9(6)**: 2796-2799.

Nyström, H.F., Brantsæter, A.L., Erlund, I., Gunnarsdottir, I. and Hulthén L. (2016). Iodine status in the Nordic countries - past and present. *Food Nut. Res.* **60(1)**: 31969.

Ozmen, O.Z., Sahinduran, S.I. and Sezer, K.E. (2005). Clinical and pathological observations and treatment of congenital goitre in kids. *Bull. Vet. Inst. Pulawy.* **49(2)**: 237-41.

Paulíková, I., Kováč, G., Bíreš, J., Paulík, Š. and Seidel H. (2002). Iodine toxicity in ruminants. *CAAS Agril. J.* **47(12)**: 343-50.

Radostits, O.M., Gay, C.C., Hinchcliff, K.W. and Constable, P.D. (2017). Veterinary Medicine. (11th Edn.), Philadelphia, Pennsylvania: Elsevier. pp. 1722-1725.

Riviere, J.E. and Papich, M.G. (2017). Veterinary Pharmacology and Therapeutics. (10th Edn.), Lowa, USA: Wiley-Blackwell Publication. p. 751.

Seimiya, Y., Ohshima, K., Itoh, H., Ogasawara, N. and Matsukida, Y. (1991). Epidemiological and pathological studies on congenital diffuse hyperplastic goitre in calves. *J. Vet. Med. Sci*. **53(6)**: 989-994.

Singh, J.L., Sharma, M.C., Kumar, M., Varshney, V.P. and Ahmad, A.H. (2003). Clinico-biochemical profile and therapeutic management of congenital goitre in kids in endemic area. *Indian J. Vet. Med.* **23(2)**: 83-87.

Singh, R. and Beigh, S.A. (2013). Diseases of thyroid in animals and their management. *Insights Vet. Med. Intech.* **9**: 233-9.

Zimmermann, M.B. (2020). Chapter 25-Iodine and the iodine deficiency disorders, in: B.P. Marriott, *et al.* (Eds.), Present Knowledge in Nutrition. (11th Edn.), Academic Press. pp. 429-441.

THE HARYANA VETERINARIAN

Editors/Editorial Board Members are highly thankful to all the distinguished referees who helped us in the evaluation of articles. We request them to continue to extend their co-operation and be prompt in future to give their valuable comments on the articles for timely publication of the journal.