ATATÜRK ÜNİVERSİTESİ YAYINLARI
ATATURK UNIVERSITY PUBLICATIONS

Case Report *Olgu Sunumu* DOI: 10.5281/zenodo.15755465

Clinical Diagnosis and Management of a Congenital Goiter in a Kid

Bir Oğlakta Konjenital Guatrın Klinik Tanısı ve Tedavi Yönetimi

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ABSTRACT

Goiter in goats is characterized by the inflammatory and non-neoplastic hypertrophy of the thyroid gland due to iodine deficiency and is commonly seen in newborn and young animals. This case presentation involves a 40-day-old female kid of the Aleppo breed. Anamnesis revealed a complaint since birth of a painless, palpable, oval-shaped mass that had been gradually enlarging on both sides of the cranioventral neck region. In the ultrasound examination, the length of the left thyroid gland was measured at 4.14 cm, the right at 3.51 cm, and the width was measured at 1.7 cm on the left and 2.07 cm on the right. In the biochemical analysis, free triiodothyronine (FT3), free thyroxine (FT4), total T3, and total T4 levels were measured as low (1.18 pmol/L, 0.787 pmol/L, 0.47 µg/L and 0.46 µg/dL, respectively), while the levels of thyroid-stimulating hormone (TSH), triglycerides, and cholesterol were measured as high (0.15 mIU/mL, 70.8 mg/dL and 164.5 mg/dL, respectively). The treatment included levothyroxine sodium (0.2 mg/kg orally once daily for 100 days) and a single intramuscular dose of sodium selenite (1 mg). Additionally, it was suggested to supplement the soil with iodized salt or seaweed. After the treatment, FT3, FT4, triglyceride, and cholesterol levels increased while TSH reduced to the reference values measurement range. Congenital goiter, caused by iodine deficiency, was completely cured with the prescribed treatment protocol. Additionally, clinical examination, ultrasonography, and thyroid hormone analysis were found to be useful for diagnosing goiter in kids.

Keywords: Goiter, Goat, Hypothyroidism, Iodine, Kid

ÖZ

Keçilerin guatrı iyot eksikliğine bağlı olarak tiroid bezinin iltihabi ve neoplastik olmayan hipertrofisi ile karakterize, çoğunlukla yenidoğan ve genç hayvanlarda görülen bir hastalıktır. Bu olgu sunumunu 40 günlük Halep ırkı dişi bir oğlak oluşturdu. Anamnezde doğumdan itibaren kranioventral boyun bölgesinin her iki tarafında giderek büyüyen ağrısız, elle tutulabilen, oval şekilli bir şişlik olduğu belirlendi. Ultrasonografik muayenede sol taraftaki tiroit bezinin uzunluğunun 4.14 cm, sağ taraftakinin ise 3.51 cm, genişliğinin ise sol taraftaki 1.7 cm, sağ taraftaki ise 2.07 cm olarak ölçüldü. Biyokimyasal analizde ise serbest triiyodotironin (FT3), serbest tiroksin (FT4), total T3 (Triiyodotironin), total T4 (Tiroksin), düzeyleri düşük (sırasıyla 1.18 pmol/L, 0.787 pmol/L, 0.47 µg/L ve 0.46 µg/dL), tiroid uyarıcı hormon (TSH), trigliserit ve kolesterol düzeyler ise yüksek (sırasıyla, 0.15 mIU/mL, 70.8 mg/dL ve 164.5 mg/dL) olarak ölçüldü. Tedavi olarak günde 1 defa 100 gün boyunca 0.2 mg/kg oral yolla levotiroksin sodyum ve tek doz sodyum selenit 1 mg kas içi olarak uygulandı. Ek olarak, toprağın iyotlu tuz veya deniz yosunlarıyla zenginleştirilmesi önerildi. Tedavi sonunda biyokimyasal analiz sonuçlarında FT3, FT4, trigliserit ve kolesterol düzeyleri yükselerek, TSH ise azalarak referans düzeylere ulaştı. Konjenital guatr iyot eksikliği nedeniyle meydana gelen bir hastalık olup doğru tanı ve tedaviyle tamamen iyileşebilir. Sonuç olarak, iyot eksikliğinden kaynaklanan konjenital guatr, uygulanan tedavi protokolüyle tamamen iyileşti. Ayrıca, klinik muayene, ultrasonografi ve tiroid hormon analizinin oğlaklarda guatrın teşhisinde yararlı olduğu bulundu.

Anahtar Kelimeler: Guatr, Keçi, Hipotirodizm, İyot, Oğlak

Geliş Tarihi/Received 10.12.2024
Kabul Tarihi/Accepted 14.05.2025
Yayın Tarihi/Publication Date 30.06.2025

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Cite this article: Şahan A, Gülersoy E, Balıkçı C, Günal İ, Kısmet E. Clinical Diagnosis and Management of a Congenital Goiter in a Kid. *J Vet Case Rep*. 2025;5(1):15-19.

INTRODUCTION

Hypothyroidism is one of the most common thyroid disorders in both humans and animals. It results from congenital or acquired defects that lead to reduced thyroid hormone production.[1] Impairments in the synthesis, storage, or secretion of thyroid hormones can cause abnormal thyroid gland development or dysfunction, leading to congenital hypothyroidism.[2] Goiter is an iodine deficiency disorder characterized by inflammatory and non-neoplastic hypertrophy of the thyroid gland, mostly observed in neonates and juvenile animals.[3] The thyroid hormones, triiodothyronine (T3) and thyroxine (T4), have many functions in the body and generally regulate the growth, differentiation, metabolism of lipids, proteins, and carbohydrates.[4] Goiter, a symptom of hypothyroidism, manifests as the enlargement of the thyroid gland. This condition mainly causes iodine deficiency in the environment and the consumption of plants containing goitrogenic substances. Primary goiter occurs as a result of enlargement of the thyroid gland due to iodine deficiency in the diet. In animals, iodine deficiency is generally caused by environmental factors stemming from low iodine levels in soil, water, feed, and fodder. Especially in feed crops, insufficient iodine in the soil is the main cause of iodine deficiency in animals.[5-7] Congenital goiter is a fatal thyroid metabolic disorder characterized by low thyroid hormone levels, hypersecretion of thyroid-stimulating hormone (TSH) from the pituitary gland, and compensatory hyperplasia of the thyroid gland.[8] Hypertrophy of the thyroid gland is a physiological response aimed at increasing the uptake of iodine from circulation to support the synthesis of thyroid hormones.[6]

CASE PRESENTATION

This case presentation involves a 40-day-old female Aleppo breed kid, which was presented to the animal hospital with a complaint of a gradually increasing, visibly swollen neck region just behind the mandible since birth. According to the anamnestic data, the animals have been grazing on a cabbage field, which is known for its goitrogenic properties, for an extended period. No clinical abnormalities were observed upon physical examination of the mother goat. It has been reported that the same kid was born as a twin and that its twin died shortly after birth. A palpable, oval-shaped, slightly firm, well-defined, and painless structure was observed on palpation of the mass. Clinical (Figure 1A and B), ultrasonographic examination (Figure 1C and D), and biochemical evaluations (Table 1) were conducted. Hormone and biochemical analyses were performed using a Roche-Siemens Cobas 8000 biochemistry system. An ultrasound device (Mindray Z60, China) and probe (5-9 MHz convex probe) were used to examine the thyroid gland.

In B-mode, the image planes were scanned longitudinally across each lobe. Ultrasound images were assessed for the shape and borders of the gland, as well as the echogenicity and echo structure of the tissue. The results showed that the thyroid gland appeared larger on ultrasonography than healthy individuals. The left lobe measured 1.70 cm in width and 4.14 cm in length, while the right lobe measured 2.07 cm in width and 3.51 cm in length (Figure 1C and D). Based on the history of consuming goitrogenic food, along with physical and ultrasonographic examinations, possible congenital goiter was suspected. To confirm the diagnosis, blood samples (3–4 mL) were aseptically collected via jugular venipuncture for further laboratory analysis. Hormone analysis revealed that the serum concentrations of thyroid hormones were significantly lower than in healthy animals, with free T3 (FT3) at a concentration of 1.18 pmol/L, free T4 (FT4) at a concentration of 0.787 pmol/L, T3 at a concentration of 0.47 µg/L, and T4 at a concentration of 0.46 µg/dL (Table 1). Additionally, serum iodine levels were measured using the ICP-MS method, along with selenium level, to support the definitive diagnosis of congenital goiter. The serum iodine level was determined to be 20.50 ng/mL, while the serum selenium level was 43.76 µg/L. In the treatment protocol, levothyroxine sodium (Levotiron tablet®, Abdi İbrahim Company, Türkiye) was administered orally at 0.2 mg/kg/day for 100 days.[9]

Table 1. Biochemical values before and after treatment

Biochemical Parameters	Pre- treatment	Post-treatment	Reference Values
TSH (Thyroid-stimulating hormone) (mIU/mL)	**0.15↑**	0.02<	0.01–0.02[1]
FT3 (Free Triiodothyronine) (pmol/L)	**1.18 ↓**	6.71	3.67-5.11[10]
FT4 (Free Thyroxine) (pmol/L)	**0.787 ↓**	15.46	13.38-18.53[10]
Total T3 (Triiyodotironin) (µg/L)	**0.47 ↓**	*	2.82 ± 0.01[6]
Total T4 (Tiroksin) (µg/dL)	**0.46 ↓**	*	8.65 ± 1.86[6]
Triglyceride (mg/dL)	**70.8 ↑**	31	19.65 (7-65)[11]
Cholesterol (mg/dL)	**164.5 ↑**	58.8	40.88 (19-76)[11]

*No analyses carried out

Additionally, a single intramuscular dose of sodium selenite (Yeldif®, Dif, Türkiye) at 1 mg was given, and the removal of goitrogenic foods from the diet was recommended. Moreover, long-term supplementation of the soil with iodized salt or seaweed was recommended for both therapeutic and prophylactic purposes. Following the

treatment, a noticeable reduction in the thyroid gland was observed in the 4th week and returned to its normal size by the 10th week (Figure 1F).

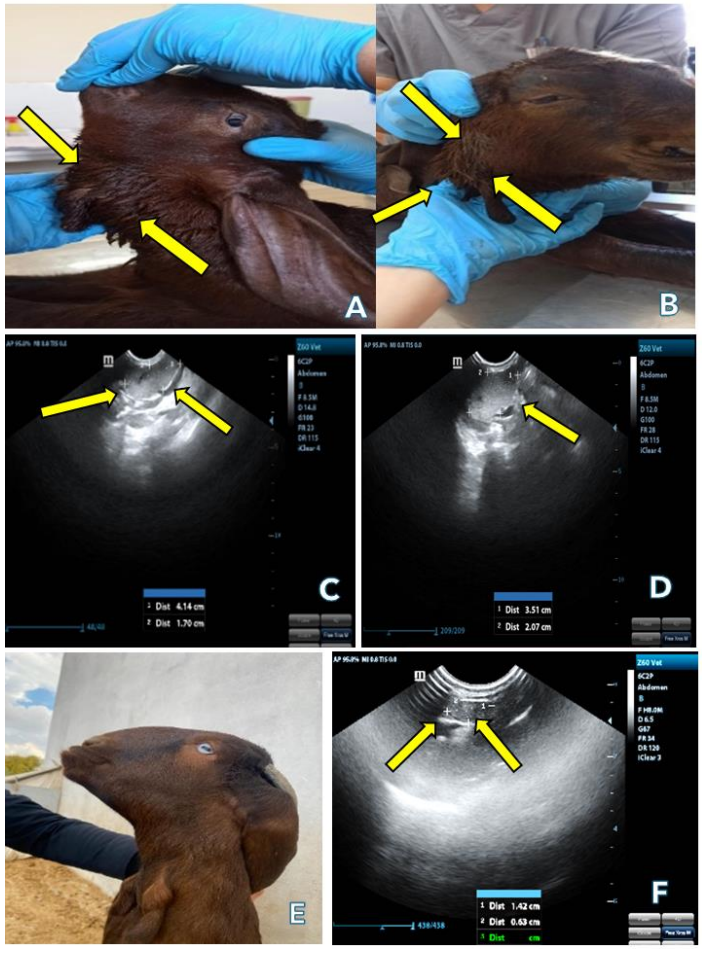

Figure 1. Indicated by yellow arrow, (A,B): clinical appearance before treatment, (C): appearance of the right thyroid gland, (D): appearance of the left thyroid gland, (E): post-treatment appearance of the kid, (F): post-treatment appearance of throid gland.

DISCUSSION

Thyroid hormone analysis, along with serum iodine measurement, are fundamental methods for confirming iodine deficiency or goiter in goats. The diagnosis is based on the evaluation of the animal's nutritional history, mineral supplementation status, and consumption of goitrogenic foods, along with compatible clinical signs such as stillbirths, abortions, and the birth of underweight or stillborn kids with congenital goiter. Clinically, the most apparent sign of goiter in goats is a noticeable enlargement of the thyroid lobes, at least twice their normal size.[6,7] In this case report, in addition to compatible anamnestic data and clinical findings, the diagnosis was confirmed through laboratory analyses, including low thyroid hormone levels and reduced serum iodine and selenium concentrations.

Thyroid gland problems are widely recognized in pet animals but are less known in livestock. Nutritional iodine shortages in farm animals, particularly in regions with iodine deficiency are more significant than those in the thyroid gland.[4] The enlargement of the thyroid glands in newborns, especially in those with high mortality rates, indicates iodine deficiency.[12] Goats are considered susceptible species for iodine deficiency because they are nomadic breeds that prefer to eat leaves, branches, vines, and bushes, and consume less soil than other grazing animals.[8] In ruminants, even when iodine intake is restricted, the thyroid glands store iodine efficiently and thyroid hormone levels are maintained for long periods, however the developing fetus lacks these thyroid hormone reserves and growth and development are restricted when iodine intake is restricted.[3] In general, clinical manifestations of iodine deficiency, such as goiter development, are more commonly observed in kids than in adult goats.[6] Thus, in this report, the probable cause of the clinically normal thyroid gland in the mother was thought to be adequate maternal iodine reserves.

When iodine deficiency occurs, the pituitary gland secretes an excessive amount of TSH. This situation causes the thyroid gland to increase the production of thyroid hormones to compensate for iodine deficiency. Growth and hyperplasia occur in the thyroid gland as a result of goiter.[13] Jarad et al.[3] reported a significant increase in TSH and cholesterol levels, along with a decrease in T3, T4, and FT4 hormone levels, in goitrous kids compared to the control group. In this report, TSH, triglyceride and cholesterol levels were high, but FT3, FT4, Total T3 and Total T4 levels were low (Table 1). It was interpreted that the thyroid gland was enlarged due to TSH activation.[7,13] In this report, TSH, cholesterol and triglyceride levels were higher before the treatment but cholesterol and triglyceride levels were lower compared to the reference values after treatment, which is similar to the previous results. Pankowski et al.[14] reported the average measurements of the thyroid lobes as follows: a length of 3.02 cm, a width of 1.05 cm, and a height of 0.63 cm. Ozmen and Haligur[15] stated that in their measurements of kids with congenital goiter, the length of the thyroid gland was ranged between 6.1-7.5 cm, and the width was ranged between 4.3-5 cm. In this report, the left lobe measured 1.7 cm in width and 4.14 cm in length, while the right lobe measured 2.07 cm in width and 3.51 cm in length. The ultrasonographic measurements indicated that the thyroid gland size exceeded the reference range (Figure1 C, D). It was highlighted that the underlying causes of hypertrophy in the thyroid gland are goitrogenic compounds and plants, iodine-deficient diets, excessive dietary iodine, and genetic enzyme defects in the biosynthesis of thyroid hormones.[7,16] Iodine deficiency in animals is often accompanied by

vitamin A and selenium deficiencies. If iodine deficiency occurs during pregnancy, problems such as dystocia, myxedema and prolonged pregnancy can occur in the fetus. This situation generally leads to death of the fetus and doe, resulting in significant economic losses.[8] Wu et al.[17] stated that iodine and selenium are the two main minerals required for thyroid hormone production. Paksoy et al.[18] conducted a study to determine the serum selenium levels of Kilis goats in the Şanlıurfa region and found that these levels were lower than those of other sheep and goats in the area. Sabea et al.[19] stated in their study on rats with hypothyroidism that the combination of selenium and levothyroxine is the most effective treatment for hypothyroidism. Considering this result, it should be noted that the potential cause of iodine deficiency may also be related to selenium deficiency. This conclusion was also supported by the serum selenium levels measured alongside serum iodine levels (43.76 µg/L and 20.50 ng/mL, respectively). In this case report, congenital goiter was diagnosed and confirmed based on thyroid-related hormone levels, as well as serum iodine and selenium levels. However, certain limitations should be acknowledged, including the inability to perform thyroid gland histopathology, the lack of follow-up measurements of serum iodine and selenium levels despite clinical improvement after treatment, and the absence of soil iodine/selenium level assessments. Given the limited number of reports on this subject, the findings of this case report may provide valuable insights for future studies on the diagnosis and treatment of congenital goiter.

This case report describes a disease caused by goitrogenic substances and iodine deficiency, which is both treatable and preventable. Prevention strategies include avoiding goitrogenic feeds during pregnancy and ensuring adequate iodine supplementation, either orally or via injection, to support thyroid function. Goats are particularly susceptible to iodine deficiency, especially due to their increased physiological demand in natural conditions. In conclusion, this report highlights the importance of thyroid-related hormone analysis, serum iodine and selenium measurements, and ultrasonographic examination in diagnosing and confirming the disorder. Additionally, levothyroxine treatment is effective, and the disease should be considered in regions prone to iodine and selenium deficiency.

Peer-review: Externally peer-reviewed.
Author Contributions: Concept- AŞ, EG; Design- AŞ, EG, CB; Supervision- AŞ, EG, CB, İG; Resources- AŞ, İG, EK; Data Collection and/or Processing- AŞ, İG, EK; Analysis and/or Interpretation- AŞ, EG; Literature Search- AŞ; Writing Manuscript- AŞ, EG; Critical Review- EG, CB.
Conflict of Interes: The authors have no conflicts of interest to declare.
Financial Disclosure: The authors declared that this study has received no financial support.

Hakem Değerlendirmesi: Dış bağımsız.
Yazar Katkıları: Fikir- AŞ, EG; Tasarım- AŞ, EG, CB; Denetleme- AŞ, EG, CB, İG; Kaynaklar- AŞ, İG, EK; Veri Toplanması ve/veya İşlemesi- AŞ, İG, EK; Analiz ve/ veya Yorum- AŞ, EG; Literatür Taraması- AŞ; Yazıyı Yazan- AŞ, EG; Eleştirel İnceleme- EG, CB.
Çıkar Çatışması: Yazarlar, çıkar çatışması olmadığını beyan etmiştir.
Finansal Destek: Yazarlar, bu çalışma için finansal destek almadığını beyan etmiştir.

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