ATATURK
UNIVERSITY
PUBLICATIONS

Journal of Veterinary Case Reports

Official Journal of Atatürk University Faculty of Veterinary

Volume 5 • Issue 1 • June 2025

EISSN 2792-064X
https://dergipark.org.tr/tr/pub/jvetcrepy

Journal of Veterinary Case Reports

Journal of Veterinary Case Reports

AIMS AND SCOPE

Journal of Veterinary Case Reports aims to publish studies of the highest scientific and clinical value in all areas of Veterinary .

Journal of Veterinary Case Reports publishes rare case reports that will contribute to the literature on veterinary medicine. The main purpose of the journal is to disseminate the scientific knowledge produced in the field of veterinary obstetrics and gynecology, surgery, internal medicine, reproduction and artificial insemination, animal nutrition and nutritional diseases, biochemistry, pharmacology, microbiology, parasitology, pathology, virology, and zootechnics to a wide platform. In doing so, the journal aims to bring together researchers, educational practitioners and policy makers at a common intersection.

The target audience of the journal includes academicians, clinical researchers, medical/health professionals, students, and related professional and academic bodies and institutions.

Journal of Veterinary Case Reports is currently indexed **in EBSCO, Gale Cengage, and CABI.**

Disclaimer

The statements or opinions expressed in the manuscripts published in the journal reflect the views of the author(s) and not the views of the editors, editorial board, and/or publisher. The editors, editorial board, and publisher are not responsible for the content of the manuscripts and do not necessarily endorse the views expressed in them. It is the responsibility of the authors to ensure that their work is accurate and well-researched, and the views expressed in their manuscripts are their own. The editors, editorial board, and publisher simply provide a platform for the authors to share their work with the scientific community.

Open Access Statement

Journal of Veterinary Case Reports is an open access publication.

Starting on 2024, all content published in the journal is licensed under the Creative Commons Attribution-NonCommercial (CC BY-NC) 4.0 International License which allows third parties to use the content for non-commercial purposes as long as they give credit to the original work. This license allows for the content to be shared and adapted for non-commercial purposes, promoting the dissemination and use of the research published in the journal.

The content published before 2024 was licensed under a traditional copyright, but the archive is still available for free access.

All published content is available online, free of charge at https://dergipark.org.tr/en/pub/jvetcrep.

When using previously published content, including figures, tables, or any other material in both print and electronic formats, authors must obtain permission from the copyright holder. Legal, financial and criminal liabilities in this regard belong to the author(s).

AMAÇ VE KAPSAM

Journal of Veterinary Case Reports, veteriner hekimliğin tüm alanlarında bilimsel ve klinik değeri en yüksek çalışmaları yayınlamayı amaçlamaktadır.

Journal of Veterinary Case Reports, veteriner hekimliği literatürüne katkı sağlayacak nadir vaka raporlarını yayınlamaktadır. Derginin temel amacı veteriner doğum ve jinekoloji, cerrahi, dahiliye, üreme ve suni tohumlama, hayvan besleme ve beslenme hastalıkları, biyokimya, farmakoloji, mikrobiyoloji, parazitoloji, patoloji, viroloji ve zootekniyi geniş bir platforma taşımaktır. Bunu yaparken dergi, araştırmacıları, eğitim uygulayıcılarını ve politika yapıcıları ortak bir noktada buluşturmayı amaçlamaktadır.

Derginin hedef kitlesi akademisyenler, klinik araştırmacılar, tıp/sağlık profesyonelleri, öğrenciler ve ilgili profesyonel ve akademik kişilerden oluşmaktadır.

*Veteriner Vaka Raporları Dergisi şu anda **EBSCO, Gale Cengage ve CABI**'de indekslenmektedir..*

Sorumluluk Reddi

Dergide yayınlanan yazılarda ifade edilen ifadeler veya görüşler editörlerin, yayın kurulunun ve/veya yayıncının görüşlerini değil, yazar(lar)ın görüşlerini yansıtmaktadır. Editörler, yayın kurulu ve yayıncı yazıların içeriğinden sorumlu değildir ve bunlarda ifade edilen görüşleri mutlaka onaylamazlar. Çalışmalarının doğru ve iyi araştırılmış olmasını sağlamak yazarların sorumluluğundadır ve yazılarında ifade edilen görüşler kendilerine aittir. Editörler, yayın kurulu ve yayıncı, yazarlara çalışmalarını bilimsel toplulukla paylaşmaları için bir platform sağlar.

Açık Erişim Bildirimi

Journal of Veterinary Case Reports dergisi, açık erişimli bir yayındır. 2024 yılından itibaren dergide yayınlanan tüm içerik, orijinal esere atıfta bulunulduğu sürece üçüncü tarafların içeriği ticari olmayan amaçlarla kullanmasına olanak tanıyan Creative Commons Attribution-NonCommercial (CC BY-NC) 4.0 Uluslararası Lisansı kapsamında lisanslanmıştır. Bu lisans, içeriğin ticari olmayan amaçlarla paylaşılmasına ve uyarlanmasına izin vererek dergide yayınlanan araştırmanın yayılmasını ve kullanımını teşvik eder. 2024'ten önce yayınlanan içerik, geleneksel telif hakkı kapsamında lisanslanmıştı ancak arşiv hâlâ ücretsiz erişime açık. Yayınlanan tüm içeriğe çevrimiçi olarak ücretsiz olarak https://dergipark.org.tr/en/pub/jvetcrep adresinden ulaşılabilir. Şekiller, tablolar veya hem basılı hem de elektronik formattaki diğer materyaller de dahil olmak üzere daha önce yayınlanmış içeriği kullanırken, yazarların telif hakkı sahibinden izin alması gerekir. Bu konudaki hukuki, mali ve cezai sorumluluk yazar(lar)a aittir.

Contact (Editor in Chief) / İletişim (Baş Editör)

Akın KIRBAŞ

Atatürk University, Faculty of Veterinary Medicine, Erzurum, Türkiye.

Atatürk Üniversitesi, Veteriner Fakültesi, Erzurum, Türkiye.

✉ akirbas@atauni.edu.tr

✉ jvcr@atauni.edu.tr

https://dergipark.org.tr/en/pub/jvetcrep

☎ +90 442 231 71 64

Contact (Publisher) / İletişim (Yayıncı)

Atatürk University

Atatürk University, Erzurum, Türkiye

Atatürk Üniversitesi Rektörlüğü 25240 Erzurum, Türkiye

✉ ataunijournals@atauni.edu.tr

https://bilimseldergiler.atauni.edu.tr

☎ +90 442 231 15 16

Journal of Veterinary Case Reports

CONTENTS / İÇİNDEKİLER

ATATÜRK ÜNİVERSİTESİ YAYINLARI
ATATURK UNIVERSITY PUBLICATIONS

Case Report *Olgu Sunumu* DOI: 10.5281/zenodo.15758112

The First Case of *Hippobosca longipennis* (Diptera: Hippoboscidae) Detected in Dogs in Türkiye

Türkiye'de Köpeklerde İlk Kez Tespit Edilen *Hippobosca longipennis* (Diptera: Hippoboscidae) Vakası

İbrahim GÜLBAYRAK
Aynur GÜLANBER

İstanbul University-Cerrahpaşa, Faculty of Veterinary Medicine, Department of Parasitology, İstanbul, Türkiye

ABSTRACT

Hippobosca longipennis is an obligate blood-feeding ectoparasite of domestic and wild carnivores. This case report describes the presence of *Hippobosca longipennis* in seven stray dogs that were brought to a municipal animal care center in Istanbul for rehabilitation. During routine clinical examinations performed on the dogs, hippoboscid flies detected in their fur were carefully collected using forceps. The collected flies were preserved in glass bottles containing 70% ethyl alcohol and transported to the Laboratory of the Department of Parasitology, Istanbul University-Cerrahpaşa Faculty of Veterinary Medicine for species identification. The flies were morphologically examined under a stereomicroscope, and all specimens were identified as belonging to *Hippobosca longipennis*. This study presents the first report of *Hippobosca longipennis* in dogs in Türkiye and provides a significant contribution to the literature on the geographical distribution of this species.

Keywords: Dog louse fly, Ectoparasite, *Hippobosca longipennis*, Stray dogs

ÖZ

Hippobosca longipennis, evcil ve yabani etoburların zorunlu kanla beslenen bir ektoparazitidir. Bu vaka raporu, İstanbul'da belediyeye ait bir hayvan bakımevine, rehabilitasyon amacıyla getirilen yedi sokak köpeğinde, *Hippobosca longipennis* türünün varlığını bildirmektedir. Köpekler üzerinde gerçekleştirilen rutin klinik muayeneler sırasında, tüyleri arasında tespit edilen hippoboscid sinekler penset yardımıyla dikkatlice toplanmıştır. Toplanan sinekler, %70 etil alkol içeren cam şişelerde muhafaza edilerek tür teşhisi amacıyla İstanbul Üniversitesi-Cerrahpaşa Veteriner Fakültesi Parazitoloji Anabilim Dalı laboratuvarına getirilmiştir. Sineklerin morfolojik incelemeleri stereo mikroskop altında yapılmış ve tüm örneklerin *Hippobosca longipennis* türüne ait olduğu belirlenmiştir. Bu çalışma, Türkiye'de köpeklerde *Hippobosca longipennis*'in ilk raporunu sunmakta ve türün coğrafi dağılımına yönelik literatüre önemli bir katkı sağlamaktadır.

Anahtar Kelimeler: Köpek biti sineği, Ektoparazit, *Hippobosca longipennis,* Sokak köpekleri

Geliş Tarihi/Received 17.01.2025
Kabul Tarihi/Accepted 23.02.2025
Yayın Tarihi/Publication Date 30.06.2025

Sorumlu Yazar/Corresponding author:
İbrahim GÜLBAYRAK
E-mail: ibrahimgulbayrak4@gmail.com
Cite this article: Gülbayrak İ, Gülanber A. The First Case of *Hippobosca longipennis* (Diptera: Hippoboscidae) Detected in Dogs in Türkiye. *J Vet Case Rep*. 2025;5(1):1-4.

INTRODUCTION

Hippobosca longipennis, known as the dog louse fly, is an obligate blood-feeding ectoparasite of domestic and wild carnivores. This species parasitizes mammals such as those in the families Canidae (dogs, jackals, foxes), Felidae (lions, leopards, cheetahs, and domestic cats), Hyaenidae (hyenas), and Viverridae (civets, mongooses). Additionally, it has been reported on accidental hosts such as antelopes and birds, and it is occasionally known to bite humans.[1-3]

Hippobosca longipennis is a member of the family Hippoboscidae and order Diptera (suborder Cyclorrhapha). It is about 1 cm long and has a dorso-ventrally flattened body. Its body is covered with a flexible layer of chitin, which allows for expansion during blood-sucking. Morphological features include short antennae, large compound eyes and piercing-sucking mouthparts. The wings are long and well-developed and can fold along the body during rest. The vascularization of the wings is a distinctive feature, especially concentrated on the leading edge. Strongly built legs terminate in a pair of claws that allow it to cling tightly to the host's skin.[4]

Hippobosca longipennis has a highly specialized biology. Female flies give birth to one fully developed larva in each reproductive cycle. The larvae soon pass into the pupal stage, which can last between 19 and 142 days, depending on environmental conditions. Adult flies usually emerge from the pupal stage in the morning and search for a suitable host to suck blood. Mating takes place about 7 days after the adults settle on the host. Larvae complete development in the body of females in 3 to 8 days and are usually laid in cracks, under vegetation or organic debris. Female flies can give birth to 10-15 larvae in their lifetime and live for about 4-5 months.[5]

Hippobosca longipennis is adapted to hot, arid and semi-arid climates and has a wide geographical distribution in Africa, the Middle East and Asia. In Europe, its presence has been reported mostly from the Mediterranean basin countries and rarely from Central Europe.[1,3]

Hippobosca longipennis is one of the main vectors of the filarial nematode *Acanthocheilonema dracunculoides* and plays a critical role in the transmission of this parasite to dogs and other mammals.[6,7] It has also been found to have the capacity to mechanically transport the zoonotic *Cheyletiella yasguri*.[8] These characteristics suggest that the species may pose serious risks to both animal and human health.

CASE PRESENTATION

In September and October 2023, seven stray dogs, aged between 8 months and 10 years, 6 females, 1 male, all mixed breeds, collected from various districts of of Istanbul (Sultanbeyli [n=2], Maltepe [n=1], Pendik [n=1], Kartal [n=1], Başakşehir [n=1] and Sultangazi [n=1]) and brought to Istanbul Metropolitan Municipality Kisirkaya Animal Care Center for rehabilitation were subjected to routine clinical examinations. During the examinations, hippoboscid flies were observed on the ventral neck and anterior axillary regions of the dogs (Figure 1).

Figure 1. *Hippobosca longipennis* identified on one of the dogs examined in the present study

A total of 24 flies were carefully removed using forceps and placed in glass bottles containing 70% ethyl alcohol. The collected specimens were brought to the laboratory of Istanbul University-Cerrahpaşa Veterinary Faculty, Department of Parasitology for species identification. Morphological species identification of the collected flies was performed under a stereo microscope based on the identification key presented by Chalupsky[4],and all flies were identified as *Hippobosca longipennis* (18♀, 6♂) (figure 2).

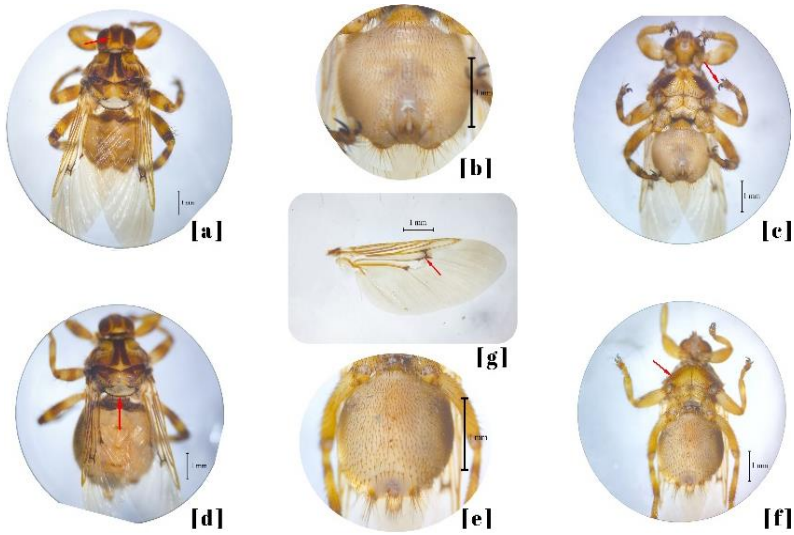

Figure 2. Morphological characteristics of *Hippobosca longipennis* (a) Dorsal view of the male, with a regular and sharp triangular apical lobe of the fronto-clypeus (red arrow)(b) Posterior end of the male (c) Ventral view of the male, showing the nail structure (red arrow) (d)

Dorsal view of the female, with an ivory-white scutellum (red arrow), (e) Posterior end of the female (f) Ventral view of the female, where the prosternum width is greater than its length (red arrow) (g) Wing structure, showing two cross veins (red arrow)

DISCUSSION

Hippobosca longipennis was first detected in Türkiye in 2020 in a red fox in Hatay province.[9] However, this species has not been encountered in previous studies on ectoparasite species diversity and prevalence in dogs in Türkiye. This study documents the presence of *Hippobosca longipennis* in dogs in Türkiye for the first time and provides an important contribution to the literature on the geographical distribution of the species.

Although the geographical distribution of *Hippobosca longipennis* appears to be mainly restricted to East Africa, North Africa, and the Middle East, the species has also been reported from Europe, Asia, and other regions. European records are generally from southern European countries with Mediterranean climates, particularly Italy, Greece, Cyprus, Bulgaria, and Spain. It has also been rarely reported in Central and Eastern European countries such as Hungary, Ukraine, Poland, Slovakia, and Romania.[1,3,10-12]

Hippobosca longipennis has been widely reported in Asia, mainly in India, Myanmar, Sri Lanka, Afghanistan and southern and central China. Its presence in South Korea and Japan has been reported to be limited and it is thought not to form permanent populations in these regions. However, it has also been recorded in arid and semi-arid areas in Central Asia, such as Afghanistan, Iran and Turkestan.[1]

International animal trade plays a critical role in the spread of *Hippobosca longipennis* to different regions. The species was transported to the United States with cheetahs imported from Africa to the San Diego Zoo, where it was recorded for the first time.[13] Similarly, *Hippobosca longipennis* was transported from Africa to the United Kingdom and Ireland with wild carnivores, and although it did not establish a permanent population in these regions, it demonstrates the importance of international transportation in moving exotic parasites to new geographies.[14,15]

Finally, *Hippobosca longipennis* is known to play an important role as a vector. This species acts as a mechanical vector of filarial nematodes, such as *Acanthocheilonema dracunculoides*, as well as a variety of pathogens of zoonotic importance.[6-8] The vectoring potential of this fly warrants further investigation of its effects on both animal and human health.

In conclusion, the geographical distribution of *Hippobosca longipennis* can be considered to continue to expand due to climate change and increased international animal transportation. The potential role of this species in the spread of zoonotic diseases is an important area of research for both veterinary and public health. More comprehensive studies in the future will provide more information on the dynamics of the species spread and vectoring potential.

Peer-review: Externally peer-reviewed.
Author Contributions: Concept-İG, AG; Design-İG, AG; Supervision-AG; Resources-İG; Data Collection and/or Processing-İG; Analysis and/or Interpretation-İG, AG; Literature Search-İG; Writing Manuscript-İG; Critical Review-AG.
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-İG, AG; Tasarım-İG, AG; Denetleme-AG; Kaynaklar-İG; Veri Toplanması ve/veya İşlemesi-İG; Analiz ve/ veya Yorum-İG, AG; Literatür Taraması-İG; Yazıyı Yazan-İG; Eleştirel İnceleme-AG.
Çıkar Çatışması: Yazarlar, çıkar çatışması olmadığını beyan etmiştir.
Finansal Destek: Yazarlar, bu çalışma için finansal destek almadığını beyan etmiştir.

REFERENCES

1. Bequaert JC. The Hippoboscidae or louse-flies (Diptera) of mammals and birds. Part I. Structure, physiology and natural history. *Entomologica Americana*. 1953;33:211-442.
2. Maa TC. A revised checklist and concise host index on Hippoboscidae (Diptera). *Pacific Insects Monograph*. 1969;20:261-299.
3. Mihalca AD, Păstrav IR, Sándor AD, Deak G, Gherman CM, Votypka J. First report of the dog louse fly *Hippobosca longipennis* in Romania. *Med Vet Entomol*. 2019;33(4):449-454.
4. Chalupsky J. Hippoboscidae. Krevsající mouchy a střeci. Fauna CSSR. Vol. 22. Prague, Czech Republic: Academia; 1980:447-478.
5. Hafez M, Hilali M. Biology of *Hippobosca longipennis* (Fabricius, 1805) in Egypt (Diptera: Hippoboscidae). *Vet Parasitol*. 1978;4:275-288.
6. Nelson GS. Dipetalonema dracunculoides (Gobbold, 1870), from the dog in Kenya: with a note on its development in the louse-fly, *Hippobosca longipennis*. *J Helminthol*. 1963;37(3):236-240.
7. Anderson RC. Nematode Parasites of Vertebrates: Their Development and Transmission. 2nd ed. Wallingford: CABI Publishing; 2000.
8. Rani PAM, Coleman GT, Irwin PJ, Traub RJ. *Hippobosca longipennis* - a potential intermediate host of a species of Acanthocheilonema in dogs in northern India. *Parasites Vectors*. 2011;4:143.

9. Zerek A, Erdem İ, Yaman M. Ectoparasites detected on a red fox (Vulpes vulpes Linnaeus, 1758) in Turkey and the first case of *Hippobosca longipennis* (Diptera: Hippoboscidae). *Turk J Vet Res*. 2020;4(2):99-101.
10. Millán J, Ruiz-Fons F, Márquez FJ, Viota M, López-Bao JV, Paz Martín-Mateo M. Ectoparasites of the endangered Iberian lynx Lynx pardinus and sympatric wild and domestic carnivores in Spain. *Med Vet Entomol*. 2007;21:248-254.
11. Straka V, Majzlan O. Dynamics of the abundance of flies (Diptera) in the National Nature Reserve Bábsky les near Nitra (South Slovakia). Rosalia (Nitra). 2010;21:167-184.
12. Sokół R, Gałecki R. Prevalence of keds on city dogs in central Poland. *Med Vet Entomol*. 2016;30(4):410-416.
13. Keh B, Hawthorne RM. The introduction and eradication of an exotic ectoparasitic fly, *Hippobosca longipennis* (Diptera: Hippoboscidae), in California. *J Zoo Wildl Med*. 1977;8:19-24.
14. O'Connor TP, Sleeman DP. Imported ectoparasites on African carnivores. *Irish Vet J*. 1987;41:239-241.
15. Chandler JA. Imported louse flies (Diptera: Hippoboscidae) in the UK. *J Zool*. 1998;246(1):115-119.

ATATÜRK ÜNİVERSİTESİ YAYINLARI
ATATURK UNIVERSITY PUBLICATIONS

Case Report *Olgu Sunumu* DOI: 10.5281/zenodo.15755375

A Case of Urachal Fistula, Omphaloarteritis, Abdominal and Inguinal Cryptorchidia in A Foal

Bir Tayda Urachal Fistül, Omfaloarterit, Abdominal ve Inguinal Kriptorşidia Olgusu

Umut ALPMAN[1]
Emre TÜFEKÇİ[2]
Nevzat Emre ASLAN[3]
Hanifi EROL[1]
Vehbi GÜNEŞ[2]

[1]Erciyes University, Faculty of Veterinary Medicine, Department of Surgery, Kayseri, Türkiye
[2]Erciyes University, Faculty of Veterinary Medicine, Department of Internal Medicine, Kayseri, TÜRKİYE
[3]Yozgat Bozok University, Faculty of Veterinary Medicine, Department of Surgery, Yozgat, Türkiye

ABSTRACT

A two-day-old male Friesian foal presenting with an inability to urinate was examined. Clinical evaluation revealed urine leakage from the umbilicus in a drip-like manner, absence of scrotal development, and bilateral cryptorchidism, along with thickening in the umbilical region indicated by palpation. Urachal fistula and omphaloarteritis were detected by palpation and transabdominal ultrasonography. As a result of the examination, it was decided that the foal would undergo comprehensive surgical intervention with conventional treatments. Operative intervention included resection of the infected umbilical cord and resection of the persistently open urachus. After the procedure, the resection of the right inguinal and left abdominal testicles was performed.

Keywords: Cryptorchidism, Fistula, Omphaloarteritis, Operation, Urachus

ÖZ

İdrar yapamama şikâyeti olan 2 günlük, erkek, Friesian tayın yapılan muayenesinde göbekten damla tarzında idrar geldiği ve skrotumun oluşmadığı, bilateral kriptorşidizm ve palpasyonda umblikal bölgede kalınlaşma olduğu belirlendi. Palpasyon ve transabdominal ultrasonografi ile urakus fistülü ve omphaloarteritis tespit edildi. Yapılan muayene sonucunda tayın konvansiyonel tedavilerle kapsamlı bir şekilde opere edilmesine karar verildi. Operatif müdahale ile enfekte göbek kordonu, persistan açık olan urakus, sağ inguinal ve sol abdominal testisin rezeksiyonları gerçekleştirildi.

Anahtar Kelimeler: Kriptorşidi, Fistül, Omphaloarteritis, Operasyon, Urakus

Geliş Tarihi/Received 19.12.2024
Kabul Tarihi/Accepted 03.03.2025
Yayın Tarihi/Publication Date 30.06.2025

Sorumlu Yazar/Corresponding author:
Umut ALPMAN
E-mail: umut_alpman@hotmail.com

Cite this article: Alpman U, Tüfekçi E, Aslan NE, Erol H, Güneş V. A Case of Urachal Fistula, Omphaloarteritis, Abdominal and Inguinal Cryptorchidia in A Foal. *J Vet Case Rep*. 2025;5(1):5-9.

INTRODUCTION

It is reported that congenital anomalies are rare in horses and the incidence of congenital malformations is between 3-3.5%. The most frequently reported malformations are musculoskeletal deformities, eye anomalies, heart defects and cryptorchidism.[1] Cryptorchidism is a congenital defect defined as the failure of the testicles to descend normally into the scrotum, either unilaterally or bilaterally. Descent of a testicle into the scrotum occurs in three sequential stages: abdominal translocation, transinguinal migration and inguinoscrotal migration. Due to deficiencies in these stages, the testicle may not descend into the scrotum and it may be found in the abdominal cavity, inguinal canal or outside of the abdominal wall.[2] This anomaly is seen in various species including horses, cattle, sheep, pigs, cats, dogs, rabbits and humans. It is reported that the prevalence in horses is 2-8%, and horses with bilateral cryptorchidism are sterile.[3] Cryptorchidism in horses is generally assumed to be related to the failure of the gubernaculum to expand the vaginal ring or the failure of the gubernaculum to regress, limiting the ability of the testicle to enter the scrotum. Cryptorchidism can be diagnosed by rectal palpation, physical examination, palpation of the scrotal and inguinal regions, and ultrasound.[4]

The structures that form the umbilical cord in horses are the umbilical vein, which extends cranially and regresses to become the ligament of the liver, and the umbilical arteries, which extend along both sides of the bladder and atrophy to become the ligament of the bladder. The urachus and surrounding soft tissues connect the bladder to the allantoic septum during intrauterine life and regress to form the median ligament of the bladder. All these structures can be affected by infectious and non-infectious diseases.[5] These diseases are the most common findings in newborn foals, especially younger than eight weeks.[6] Multiple congenital anomalies such as patent urachus, urachytosis, omphalo-arteritis/phlebitis and umbilical hernia are rare in foals. For umbilical cord care, a complete clinical examination of the foal should be performed during the first days of life, including observation and daily palpation of the umbilical region.[5] The pathogenesis of omphaloarteritis occurs when the umbilical artery retracts into the abdomen at birth, leading to infection of the clotted blood within the arteries. Upon palpation of the umbilical cord, one or both sides in the caudodorsal direction may exhibit thickening, pain, and an increase in local temperature.[7]

An urachal fistula may develop due to urethral obstruction after birth, a congenital urachal anomaly, incomplete closure caused by infection, or excessive shortening of the umbilical cord. Urinary irritation can prevent proper umbilical cord healing, increasing the risk of infection. In addition, necrosis occurs regionally in the umbilical cord, and the tissue becomes a fistula. Clinically, constant wetness, thickening of the umbilical cord, urine odor, and urine discharge at the end of the cord are observed in the urachal fistula.[8]

CASE PRESENTATION

The study material consisted of a 2-day-old, male, Friesian foal brought to Erciyes University Veterinary Faculty Training, Research and Practice Hospital and presenting with inability to urinate. In the anamnesis information, the foal was born 2 days ago, could not urinate and had difficulty standing up. The inspection of the foal revealed that urine was coming from the umbilicus in a drip-like manner, the scrotum was not formed, and bilateral cryptorchidism and palpation revealed thickening in the umbilical region. The foal's vital signs (temperature, pulsation and respiration) were within the normal ranges reported for horses. Routine hematology and biochemical tests were performed to identify and correct any electrolyte disturbances before anesthesia. After a general examination and confirmation that hematologic values were suitable for surgery, an immediate decision was made to proceed with surgical correction of the urachal fistula, omphaloarteritis, and cryptorchidism.

Anesthesia

Detomidine (Domesedan, Pfizer, Finland) was administered at a dose of 20 µg/kg intravenously (IV) for sedation. Ketamine (Ketasol 10%, Richter Pharma, Austria) was administered at a 2.2 mg/kg IV dose for anesthesia. General anesthesia was maintained with 2-3% sevoflurane (Sevorane Liquid 100%, AbbVie, Queenborough, England) throughout the operation.

Surgical Procedures

The anesthetized foal was laid in the ventro-dorsal (VD) position and the abdominal area was shaved (Figure 1). The prepared surgical site, following asepsis and antisepsis protocols, was isolated using a sterile surgical drape.

Figure 1. Image of the foal lying on the operating table during the operative intervention.

In examination, it was observed that urine was dripping from the umbilicus and there was no urethral urine output. After urethral catheterization, normal urine output was noted, indicating that the obstruction had been cleared. The surgical procedure was then initiated. After an elliptical incision in the umbilical region, the umbilical cord was dissected, ligated from the deepest part of the cord, and extirpated. In addition, the umbilical artery was extirpated by ligating it from its deepest point, and the persistently open urachus was resected as part of the surgical treatment for omphaloarteritis. The incision made for the urachus fistula was enlarged and the location of the testicles was determined. Right inguinal, and left abdominal cryptorchidism was detected. A castration procedure was performed. Images during the operative intervention are shown in Figure 2.

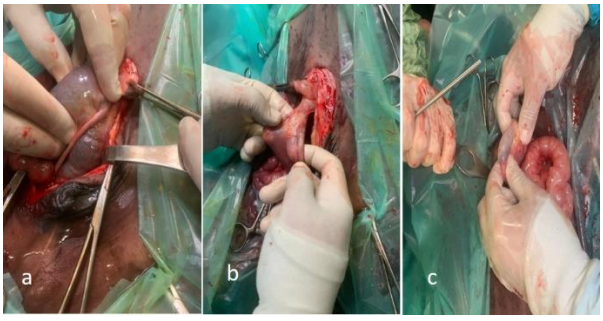

Figure 2. Image of the urinary bladder (a), umbilical cord (b) and abdominal testicle (c) during operative intervention.

Postoperatively, 10.000 IU/kg intramuscular (IM) penicillin (Pen-strep La, Provet, Türkiye) was recommended to be administered for 10 days, administration of 40 mg/kg IM metamizole sodium (Dolarjin, Topkim, Türkiye) for 5 days was recommended. The foal's postoperative image is presented in Figure 3.

Figure 3. Postoperative image of the foal

DISCUSSION

Cryptorchidism, which is defined as the failure of the testicles to descend into the scrotum, is considered as the most common congenital anomaly in horses. Unilateral retention is more common compared to bilateral cases. Abdominal retention was reported to be 2.5 times more common than inguinal retention in bilateral retention cases.[9] Hartman et al.[4] found that in 604 cases of cryptorchidism, the most common were left abdominal and right inguinal cryptorchidism. In the presented case, left abdominal and right inguinal cryptorchidism was detected. The anatomical location of the testicles in cryptorchid horses was similar to the study conducted by Hartmannn et al.[4]

Stratico et al.[10] performed cryptorchidectomy on 70 horses with cryptorchidism in their study. They compared laparoscopic and open orchidectomy operations. They stated that complications such as splenic perforation, spermatic cord bleeding and hyperthermia occur in laparoscopic surgery. They indicated difficulties in identifying the inguinal testicle during open cryptorchidectomy and that swelling was observed in the inguinal region postoperatively. In addition to the complications of the two operative interventions, they also reported that bilateral cryptorchidism was most common in the abdominal region.[4,10] This is especially important in surgical procedures related to the anatomical location of the abdominal testes and cryptorchidism in horses. In the presented case, bilateral cryptorchidism was detected as left abdominal and right inguinal. Open orchidectomy surgery was performed for treatment. Open surgery was preferred because the foal was 2 days old and had multiple anomalies such as cryptorchidism, urachus fistula and omphaloarteritis. In addition, it has been decided that an open multiple-surgery approach would facilitate deterioration in these cases. The testicle was easily detected by palpation in the abdominal and inguinal regions. After identifying the vaginal pouch, blunt dissection was used to separate the inguinal fascia and expose the external inguinal ring. Attention was paid to the critical pudendal veins passing through the region. No complications were observed during and at the end of the operation, but postoperative follow-up of the operation site to check possible swelling was recommended in the postoperative period.

Umbilical infections are frequently diagnosed in foals. It is emphasized that foals, which may be affected between one day and three months of age, require timely diagnosis and appropriate treatment. Failure to do so can result in bacterial spread to other organs, potentially leading to life-threatening complications. These complications include septic osteoarthritis, enteritis, pneumonia, patent urachus, peritonitis, uroperitoneum and intra-abdominal adhesion.[11] Perina et al.[5] evaluated 183 newborn foals in their study and reported that 40 had umbilical remnant diseases. They reported the distribution of disease

conditions seen in umbilical remnants as 60% patent urachus, 40% omphaloarteritis, 10% omphalophlebitis, 25% urachitis, 22.5% abscess, 7.5% periumbilical hematoma and 30% more than one condition. Operative intervention is often preferred in the treatment of the mentioned diseases. Postoperative complications include standard degree of incisional edema and surgical incision infection. Usually, subcutaneous infections resolve satisfactorily, but a severe incisional infection extending deeper than the subcutaneous tissues may result in a ventral abdominal wall hernia, which may only become apparent weeks to months after the initial surgery. The formation of abdominal adhesion is a risk associated with any surgical procedure involving entry into the abdominal cavity, and foals are particularly susceptible.[12] In this case, urachus fistula and omphaloarteritis were observed in the umbilical remnants of a 2-day-old newborn foal. The basic surgical approach was determined as the resection of an infected umbilical cord and the resection of a persistent open urachus. Routine hematology and biochemical tests were performed to identify and correct any electrolyte disturbances before anesthesia. Perioperative antibiotics were administered to minimize the risk of bacteremia associated with surgical manipulation, and attention was paid to hemostasis during resection of the umbilical remnants and the risk of contamination of the abdomen with urine during resection of the bladder apex.

In a foal presenting with urachus fistula, omphaloarteritis, and both abdominal and inguinal cryptorchidism, the outcome and prognosis depend primarily on the timeliness and effectiveness of medical and surgical interventions.[4,10] Umbilical infections may lead to complications such as a patent urachus; however, surgical treatment generally results in a favorable prognosis, provided there is no systemic involvement, such as septicemia. On the other hand, septic conditions may reduce survival rates in severe cases.[13,14] Our study supports the findings that a urachus fistula developed due to omphalite pathogenesis in this case. Urachal fistula has a high cure rate when treated promptly with stabilization and surgical intervention. However, septic or premature foals have a worse prognosis due to risks such as peritonitis and incision complications.[15] In the presented case, despite a successful surgical procedure, the foal died on the second postoperative day. Comprehensive medical management, including early diagnosis, aggressive antimicrobial therapy, and supportive care, is essential for improving outcomes in such complex cases.

According to our results, combining inguinal palpation, transabdominal ultrasonography, and transrectal palpation proved to be a reliable diagnostic approach for assessing surgical eligibility before planning the procedure.

Peer-review: Externally peer-reviewed.
Author Contributions: Concept-HE; Design-HE, VG; Supervision-HE; Resources-ET; Data Collection and/or Processing-ET; Analysis and/or Interpretation-UA; Literature Search-NEA; Writing Manuscript-UA, NEA; Critical Review-HE.
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-HE; Tasarım-HE, VG; Denetleme-HE; Kaynaklar-ET; Veri Toplanması ve/veya İşlemesi-ET; Analiz ve/ veya Yorum-UA; Literatür Taraması-NEA; Yazıyı Yazan-UA, NEA; Eleştirel İnceleme-HE.
Çıkar Çatışması: Yazarlar, çıkar çatışması olmadığını beyan etmiştir.
Finansal Destek: Yazarlar, bu çalışma için finansal destek almadığını beyan etmiştir.

REFERENCES

1. Binanti D, Zani DD, De Zani D, Turci T, Zavaglia G, Riccaboni P. Contracted foal syndrome associated with multiple malformations in two foals. *Anat Histol Embryol*. 2014;43:71-74.
2. Schurink A, Jong A, Nooij HR, Hellinga I, Ducro BJ. Genetic parameters of cryptorchidism and testis size in Friesian colts. *Livestock Sci*. 2016;190:136-140.
3. Eriksson S, Jaderkvist K, Dalin AM, Axelsson J, Lindgren G. Prevalence and genetic parameters for cryptorchidism in Swedish-born Icelandic horses. *Livestock Sci*. 2015;180:1-5.
4. Hartman R, Hawkins JF, Adams SB, Moore GE, Fessler JF. Cryptorchidectomy in equids: 604 cases (1977-2010). *J Am Vet Med Assoc*. 2015;246(7):777-784.
5. Perina F, Mariella J, Ellero N, Freccero F, Castagnetti C, Lanci A. Retrospective analysis of factors associated with umbilical diseases in foals. *J Equine Vet Sci*. 2024;135:105045.
6. Elce YA. Infections in the equine abdomen and pelvis: perirectal abscesses, umbilical infections, and peritonitis. *Vet Clin Equine Pract*. 2006;22(2):419-436.
7. Baird AN. Umbilical surgery in calves. *Vet Clin Food Anim*. 2016;467-477.
8. Cihan M, Aksoy Ö, Özaydın İ, Özba B, Baran V. Buzağılarda umblikal lezyonların değerlendirilmesi: 322 olgu (1996-2005). *Kafkas Univ Vet Fak Derg*. 2006;12:141-145.
9. Stickle RL, Fessler JF. Retrospective study of 350 cases of cryptorchidism. *J Am Vet Med Assoc*.

1978;172:343-346.
10. Stratico P, Varasano V, Guerri G, Celani G, Palozzo A, Petrizzi L. A retrospective study of cryptorchidectomy in horses: diagnosis, treatment, outcome and complications in 70 cases. *Animals*. 2020;10(12):2446.
11. Oreff GL, Tatz AJ, Dahan R, Segev G, Berlin D, Kelmer G. Surgical management and long-term outcome of umbilical infection in 65 foals (2010-2015). *Vet Surg*. 2017;1-9.
12. Obrochta B, Saitua A, Gracia-Calvo LA, et al. Short and long-term postoperative complications and outcomes after umbilical vein marsupialisation in 11 foals. *Equine Vet Edu*. 2024;00:1-8.
13. Wong D, McCoy AM, Wilkins PA. Urinary Tract Disorders. In: Wong DM, Wilkins PA, Eds. Equine Neonatal Medicine. New Jersey: Jhon and Wiley Sons; 2024;684-704.
14. Ford MG, Nelson BB, Ford TS, Souza CR, Easley JT, Hackett ES. Complications and comorbidities in foals undergoing surgical repair for uroperitoneum. *J Equine Vet Sci*. 2022;110:103852.
15. Steward SK, RubioMartinez LM. Complications of urinary surgery. In: RubioMartinez LM, Hendrickson DA, eds. Complications in Equine Surgery. New Jersey: Jhon and Wiley Sons; 2021;571-582.

ATATÜRK ÜNİVERSİTESİ YAYINLARI
ATATURK UNIVERSITY PUBLICATIONS

Case Report *Olgu Sunumu* DOI: 10.5281/zenodo.15755450

Innovative Nutraceutical Approach in the Treatment of Chronic Gingivostomatitis: Insights Gained from the Case

Kronik Gingivostomatitis Tedavisinde Yenilikçi Nutrasötik Yaklaşım: Vakadan Öğrenilenler

Songül ERDOĞAN
Tahir ÖZALP
Hasan ERDOĞAN
Kerem URAL
Aydın Adnan Menderes University, Faculty of Veterinary Medicine, Department of Internal Medicine, Aydın, Türkiye

ABSTRACT

A nine-year-old female domestic shorthair cat presented with lethargy, anorexia, recurrent severe oral lesions, and oral pain. The cat was diagnosed with feline chronic gingivostomatitis (FCGS) associated with feline Immunodeficiency Virus (FIV) infection, based on clinical signs and serological testing.. Management was followed with a nutraceutical supplement including primarily iodine and arginine. Over a 2.5-month period, significant clinical improvements were observed, including resolution of oral lesions, increased appetite, and improved activity levels. This case report highlighted the potential role of nutraceuticals as a treatment regime for managing FCGS in FIV-positive cats, particularly in cases resistant to traditional treatments.

Keywords: Arginine, Cat, Iodine, Stomatitis

ÖZ

Dokuz yaşında, dişi, evcil bir kedi, halsizlik, anoreksi, tekrarlayan şiddetli ağız lezyonları ve ağrı şikâyeti ile başvurdu. Kediye, Kedi İmmün Yetmezlik Virüsü (FIV) enfeksiyonu ile ilişkili Kedilerde Kronik Gingivostomatit (FCGS) tanısı serolojik analizler ve klinik bulgular temelinde konuldu. Hastada başlıca iyot ve arjinin içeren bir nutrasötik takviye başlandı. 2,5 aylık bir süre zarfında, ağız lezyonlarının çözülmesi, iştahın artması ve aktivite artışı ile önemli klinik iyileşme gözlemlendi. Bu olgu sunumu, özellikle geleneksel tedavilere dirençli vakalarda, FIV-pozitif kedilerde FCGS yönetimi için nutrasötiklerin potansiyel bir tedavi yöntemi olarak rolünü vurgulamaktadır.

Anahtar Kelimeler: Arjinin, Kedi, İyot, Stomatitis

Geliş Tarihi/Received 12.12.2024
Kabul Tarihi/Accepted 03.03.2025
Yayın Tarihi/Publication Date 30.06.2025

Sorumlu Yazar/Corresponding author:
Tahir ÖZALP
E-mail: tahirozlp@gmail.com

Cite this article: Erdoğan S, Özalp T, Erdoğan H, Ural K. Innovative Nutraceutical Approach in the Treatment of Chronic Gingivostomatitis: Insights Gained from the Case. *J Vet Case Rep*. 2025;5(1):10-14.

INTRODUCTION

Feline chronic gingivostomatitis (FCGS) is a complex and debilitating inflammatory mucosal disease that affects up to 26% of domestic cats. This condition, which causes severe oral pain, can become potentially life-threatening in at least 10% of cases.[1] The pathogenesis of FCGS remains unclear, and may involve various factors, including infectious agents such as feline immunodeficiency virus (FIV)[2]. Especially related viral infection are suspected to play a role in FCGS due to their association with increased prevalence in cats.[3] One proposed mechanism is that immune dysregulation disrupts the oral microbiome, thereby facilitating pathogenic colonization.[2]

The primary etiology of FCGS remains poorly understood, and a consistent and effective treatment approach has yet to be established. In recent years, various therapeutic strategies have been explored, typically categorized into medical and surgical methods.[4] Medical treatments often involve immunosuppressive therapies, with agents such as corticosteroids and cyclosporine being commonly utilized.[4] On the other hand, surgical interventions primarily focus on the extraction of premolar and molar teeth or, in some cases, all teeth to manage the condition effectively.[5] Additionally, the incorporation of L-arginine as part of therapeutic strategies has been considered due to its established immunomodulatory and wound-healing properties. L-arginine is a precursor to nitric oxide, a molecule crucial for immune response and tissue repair, and its metabolism is directly linked to the regulation of pro-inflammatory and anti-inflammatory pathways. This dual regulatory role is particularly relevant in chronic inflammatory conditions such as feline chronic gingivostomatitis, where immune dysregulation plays a significant role.[6] While the exact circulating levels of L-arginine in affected cats remain unmeasured, its supplementation aims to restore immune homeostasis and promote mucosal healing, thereby offering a complementary approach to traditional therapies.[7]

The aim of this case report is to evaluate the clinical effectiveness of a nutraceutical supplement with some essential amino acids and rich herbal composition in the management of FCGS in FIV-positive cat, with a focus on its impact on oral lesion healing, and overall quality of life.

CASE PRESENTATION

On May 19, 2024, a nine-year-old, 3.5 kg female domestic shorthair cat was admitted to the Veterinary Clinic of Aydın Adnan Menderes University. The cat presented with a history of weight loss, reduced water and food intake, and severe oral pain and inflammation as the primary complaints. The owner reported a history of advanced periodontal disease and a clinical suspicion of gingivostomatitis complex, as well as intermittent corticosteroid use over the past two years. The owner also stated that prednisolone treatment had significantly improved oral inflammation but did not result in complete resolution. To identify potential risks in the anamnesis, it was noted that the cat had indoor-outdoor access, lacked a vaccination history, but was regularly dewormed and had contact with other cats. On physical examination, the vital findings of cat were within normal limits; however, significant pain and resistance were noted during oral cavity examination. Generalized hyperemia in the alveolar mucosa, caudal oral mucosa, and palatoglossal folds, along with spontaneous bleeding were identified. Laboratory tests were conducted, including biochemical analysis and a complementary FIV Ab/FeLV Ag/FCoV Ab immune assay (Feline 3D, Bionote, South Korea). The cat tested positive for FIV but calicivirus elimination could not be performed. Initial laboratory results revealed globulin at 5.5 g/dL (reference range: 2.8–4.4 g/dL), an albumin/globulin ratio of 0.6 (reference range: >0.8), ALP at 137 U/L (reference range: 20–110 U/L), and serum amyloid A at 1667 U/L (reference range: 500–1500 U/L). To evaluate lesion severity and treatment response, scoring systems including the Caudal Stomatitis Intensity Score (CSIS),[8] Owner Disease Activity Index (ODAI),[9] and Disease Activity Index (DAI)[10] were applied.

Histopathological evaluation of lesions was not performed. Medications were prescribed to facilitate a follow-up oral cavity examination, but the owner did not return for the follow up monitoring. The patient returned 2.5 months later, with the owner reporting mild improvement in oral lesions with the prescribed medications. However, symptoms persisted, and the owner independently discontinued the medications, replacing them with corticosteroids for two weeks, which yielded no improvement. After that the owner only applied for a nutraceutical supplement provided by us (Petclinique Metabolique, Petclinique, Türkiye) for 1 month without consultation. This supplement contained primarily iodine, Chromium, L-arginine, L-taurine, and natural ingredients such as Gymnema sylvestre, turmeric, ginger, cayenne pepper, garlic, fenugreek, cinnamon, dandelion, and potassium iodide. The supplement had been prescribed at a dosage of half a tablet twice daily.

At the second consultation 2.5 months of the first initiation, the owner was enrolled to clinic with the mild recurrence of oral lesions following stress. The owner reported a gradual improvement in appetite, resumption of solid food intake, increased activity, and complete resolution of oral lesions during the administration of nutraceutical supplement and after completing the supplement, the clinical improvement remained stable for one month. However, mild oral lesions reappeared following stress exposure caused by a new environment and contact with another animal.

Upon examination, mild localized lesions were observed in the alveolar mucosa and upper palate. Clinical scoring revealed improvement compared to initial findings, even during this mild recurrence. After one additional week of treatment with the supplement, the lesions resolved completely, and the cat was deemed stable (Figure 1, Table 1).

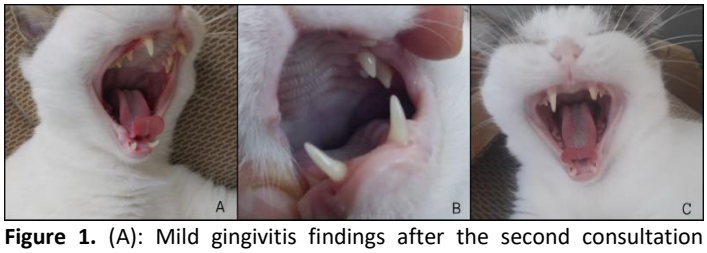

Figure 1. (A): Mild gingivitis findings after the second consultation (before) (B): Improvement of the lesion in the first week following supplement administration (after) (C): The lesion significantly improved one month after supplement, with the mucosa nearly resembling healthy oral tissue (after).

Table 1. Oral lesion scores pre- and post- treatment

Stomatitis Scores	Pre-Treatment	Post-treatment		
		After 1 week of the second consultation (After 2.5 months of the first initiation)		After 1 months of the second consultation
CSIS	2	0.5	0.25	0
ODAI	7	0	0	0
DAI	14	3	2	0

CSIS: The Caudal Stomatitis Intensity Score, ODAI: Owner Disease Activity Index, DAI: Disease Activity Index.

DISCUSSION

The management of FCGS remains a significant therapeutic challenge due to the lack of a universally effective drug to mitigate the chronic inflammation of oral mucosa and the proliferation of granulation tissue. In this study, we assessed the clinical effectiveness of a commercially available nutraceutical compound in an FIV-positive cat with chronic gingivostomatitis. Notable clinical improvements became evident within seven days of treatment initiation and continued over the subsequent two months, highlighting the potential utility of this approach in managing FCGS.

Comparable outcomes have been observed in a previously reported uncontrolled case study, where an adult cat in the terminal stage of FIV achieved complete recovery within eight weeks following the administration of a commercially available iodine solution. Remarkably, the cat remained free of clinical signs for at least five years post-treatment.[11] This recovery was attributed to iodine's broad-spectrum antimicrobial properties against opportunistic infections and its possible ability to reduce viral load. Retroviral infections such as FIV exhibit a viral load localized predominantly within the reticuloendothelial system, a feature also shared with HIV.[12] Notably, the lipophilic form of iodine has demonstrated significant antiviral and microbiocidal activity.[8] When administered orally, this form of iodine integrates into triglyceride-rich lipoproteins, enabling lymphatic transport to reticuloendothelial cells.[11] This mechanism supports its potential as an effective antimicrobial and antiviral agent, particularly in FIV cases.

L-arginine, a semi-essential amino acid included in the nutraceutical formulation used in this study, likely contributed to the observed clinical improvements. L-arginine is derived either from dietary intake or through the recycling of L-citrulline, itself synthesized from glutamate, glutamine, or proline. Within the urea cycle, L-arginine is metabolized by arginase-1 into L-ornithine and urea. Arginase-1 plays a pivotal role in cellular detoxification, proliferation, and collagen synthesis, linking L-arginine metabolism to inflammatory and wound-healing pathways.[9] The Arg-dependent pathways modulate the balance between pro-inflammatory and anti-inflammatory responses, which is critical during tissue regeneration. Dysregulation of these pathways has been implicated in impaired wound healing and the development of chronic wounds, conditions relevant to FIV-positive cats.[6] The therapeutic focus on L-arginine in this study was due to its well-documented roles in immune modulation, anti-inflammatory pathways, and tissue repair, which are critical in managing chronic inflammatory conditions such as FCGS. While the nutraceutical formulation included multiple bioactive compounds such as L-carnitine, the study primarily aimed to evaluate the clinical potential of L-arginine based on its established biological relevance. It is important to note that L-arginine supplementation was not intended to directly target retroviral infections; rather, it was employed to modulate immune responses and support tissue regeneration. However, the observed improvement

likely resulted from the synergistic effect of multiple components within the formulation rather than the isolated effect of L-arginine alone. The contribution of other components, including L-carnitine, to the observed improvement remains a subject for future investigation. This limitation is acknowledged, and further research should aim to isolate the individual effects of each component within such formulations.

The parallels between FIV and human HIV/AIDS underscore the relevance of these findings. FIV, much like HIV, is a retrovirus that induces immunosuppression through a progressive depletion of CD4+ T-helper lymphocytes, leading to systemic illness, chronic inflammation, and susceptibility to opportunistic infections.[13] Increased arginase-1 activity has been reported in patients with HIV, particularly those with low CD4+ T-cell counts, suggesting a mechanistic similarity between these retroviral diseases.[14,15] Previous studies have demonstrated that supplementation with L-arginine enhances immune responses in cats by promoting leukocyte proliferation and phagocytic activity, effects that may have contributed to the immunomodulation observed in this case.[15] Furthermore, L-arginine supplementation has been associated with increased CD4+ activity and improved immune homeostasis through pathways involving polyamine and collagen synthesis, as well as the maintenance of L-arginine–arginase–NOS balance.[6]

In this case, significant clinical improvement was observed over a 2.5-month period with the exclusive use of the nutraceutical compound, as evidenced by reduced clinical scores. However, the temporary recurrence of lesions suggests that environmental stressors may exacerbate underlying oral conditions in FIV-positive cats. This finding emphasizes the importance of long-term management strategies, including minimizing stress and providing supportive care, to sustain clinical remission.

The results of this study further underscore the association between chronic gingivostomatitis and systemic inflammatory burden in FIV-positive cats. The observed efficacy of the nutraceutical compound, particularly its herbal composition, highlights its potential as an alternative therapeutic approach for treatment-resistant conditions. The use of natural supplements may provide a promising solution for improving the quality of life in affected cats. However, further research, including controlled clinical trials, is warranted to confirm these findings and elucidate the underlying mechanisms of action. The clinical benefits observed in this case likely resulted from the synergistic effect of multiple bioactive compounds rather than the action of a single component, emphasizing the importance of evaluating combination therapies in further investigations. Future studies should focus on the synergistic effects of components such as iodine and L-arginine, which appear to play key roles in immunomodulation and tissue regeneration. By expanding our understanding of these mechanisms, we can optimize treatment strategies for FCGS and similar chronic inflammatory conditions.

Peer-review: Externally peer-reviewed.
Author Contributions: Concept - SE, TÖ, HE, KU; Design- SE, TÖ, HE, KU; Supervision- SE, TÖ, HE, KU; Resources- SE, TÖ, HE, KU; Data Collection and/or Processing- SE, TÖ, HE, KU; Analysis and/or Interpretation- SE, TÖ, HE, KU; Literature Search- SE, TÖ, HE, KU; Writing Manuscript- SE, TÖ, HE, KU; Critical Review- SE, TÖ, HE, KU.
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-SE, TÖ, HE, KU; Tasarım- SE, TÖ, HE, KU; Denetleme- SE, TÖ, HE, KU; Kaynaklar- SE, TÖ, HE, KU; Veri Toplanması ve/veya İşlemesi- SE, TÖ, HE, KU; Analiz ve/ veya Yorum- SE, TÖ, HE, KU; Literatür Taraması- SE, TÖ, HE, KU; Yazıyı Yazan- SE, TÖ, HE, KU; Eleştirel İnceleme- SE, TÖ, HE, KU.
Çıkar Çatışması: Yazarlar, çıkar çatışması olmadığını beyan etmiştir.
Finansal Destek: Yazarlar, bu çalışma için finansal destek almadığını beyan etmiştir.

REFERENCES

1. Soltero-Rivera M, Goldschmidt S, Arzi B. Feline chronic gingivostomatitis current concepts in clinical management. *J Feline Med Surg*. 2023;25(8):1098612X231186834.
2. Older CE, Gomes MDOS, Hoffmann AR, et al. Influence of the FIV status and chronic gingivitis on feline oral microbiota. *Pathogens*. 2020;9(5):383.
3. Belgard S, Truyen U, Thibault JC, Sauter-Louis C, Hartmann K. Relevance of feline calicivirus, feline immunodeficiency virus, feline leukemia virus, feline herpesvirus and Bartonella henselae in cats with chronic gingivostomatitis. *Berl Munch Tierarztl Wochenschr*. 2010;123(9-10):369-376.
4. Ahmad AM, Ali OJ, Marif HF, Ali BA. Therapeutic Management of Chronic Gingivostomatitis In Cats. A Comprehensive Review. *Basrah J Vet Res*. 2024;23(3):156-169.
5. Bellei E, Dalla F, Masetti L, Pisoni L, Joechler M. Surgical therapy in chronic feline gingivostomatitis (FCGS). *Vet Res Commun*. 2008;32:231-234.
6. Szondi DC, Wong JK, Vardy LA, Cruickshank SM. Arginase signalling as a key player in chronic wound pathophysiology and healing. *Front Mol Biosci*. 2021;8:773866.
7. Paßlack N, Kohn B, Zentek J. Effects of arginine and ornithine supplementation to a high-protein diet on

selected cellular immune variables in adult cats. *J Vet Intern Med*. 2020;34(2):852-856.

8. Druet I, Hennet P. Relationship between feline calicivirus load, oral lesions, and outcome in feline chronic gingivostomatitis (caudal stomatitis): retrospective study in 104 cats. *Front Vet Sci*. 2017;4:209.
9. Matsumoto H, Teshima T, Iizuka Y, et al. Evaluation of the efficacy of the subcutaneous low recombinant feline interferon-omega administration protocol for feline chronic gingivitis-stomatitis in feline calicivirus-positive cats. *Res Vet Sci*. 2018;121:53-58.
10. Johnston N. An updated approach to chronic feline gingivitis stomatitis syndrome. *Vet Prac*. 2012;44:34-38.
11. Mamo JC, Naissides M. Could iodine be effective in the treatment of human immunodeficiency virus and AIDS-associated opportunistic infections? *Int J Infect Dis*. 2005;9(5):292-293.
12. Bingen A, Nonnenmacher H, Bastien-Valle M, Martin JP. Tissues rich in macrophagic cells are the major sites of feline immunodeficiency virus uptake after intravenous inoculation into cats. *Microbes Infect*. 2002;4(8):795-803.
13. Vahlenkamp TW, Tompkins MB, Tompkins WA. Feline immunodeficiency virus infection phenotypically and functionally activates immunosuppressive CD4+ CD25+ T regulatory cells. *J Immunol*. 2004;172(8):4752-4761.
14. Cloke TE, Garvey L, Choi BS, et al. Increased level of arginase activity correlates with disease severity in HIV-seropositive patients. *J Infect Dis*. 2010;202(3):374-385.
15. Paßlack N, Kohn B, Zentek J. Effects of arginine and ornithine supplementation to a high-protein diet on selected cellular immune variables in adult cats. *J Vet Intern Med*. 2020;34(2):852-856.

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

Adem ŞAHAN
Erdem GÜLERSOY
Canberk BALIKÇI
İsmail GÜNAL
Esma KISMET
Harran University, Faculty of Veterinary Medicine, Department of Internal Medicine, Şanlıurfa, Türkiye

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

Sorumlu Yazar/Corresponding author:
Adem ŞAHAN
E-mail: ademsahan@harran.edu.tr

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).

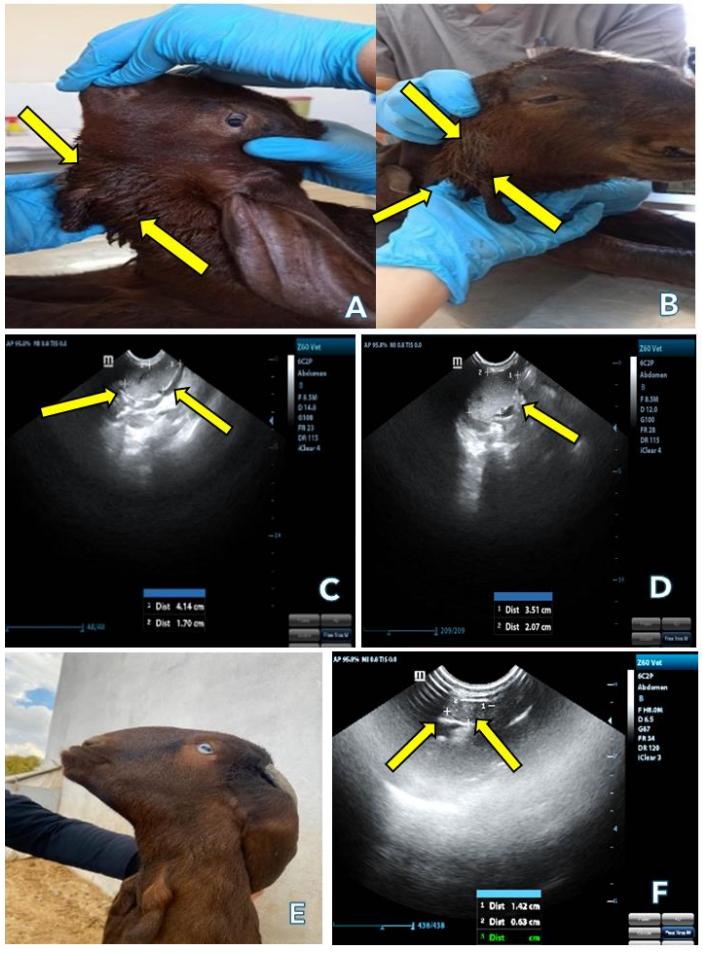

Figure 1. Indicated by yellow arrow, (A,B): clinical appearance before treatment, (C): appearance of the right thyroid gland, (D): appearance of the left thyroid gland, (E): post-treatment appearance of the kid, (F): post-treatment appearance of throid gland.

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.

REFERENCES

1. Kadum NB, Luaibi OK. Clinical study hypothyroidism in goats and treatment by iodine compounds. *J Entomol Zool Stud*. 2017;5(4):1956-1961.
2. De Vijlder JJ. Primary congenital hypothyroidism: defects in iodine pathways. *Eur J Endocrinol*. 2003;149:247-256.
3. Jarad A, Al Saad K. Goiter in cross breed goat kids at Basrah Province, Iraq. *Arch Razi Inst*. 2023;78:531.
4. Singh R, Beigh S. Diseases of thyroid in animals and their management. *Insights Vet Med*. 2013;9:233-239.
5. O'Dell BL, Sunde RA, eds. Handbook of Nutritionally Essential Mineral Elements. CRC Press; 1997.
6. Nagella N, Thounaojam R, Kumar TS, Balaji KS. A comprehensive review of iodine deficiency in goats. *Int J Vet Sci Anim Husb*. 2024;9(5):317-320.
7. Smith MC, Sherman DM. Iodine. In: Goat Medicine. 3rd ed. Hoboken, NJ: Wiley-Blackwell; 2023:80-84.
8. Nourani H, Sadr S. Case report of congenital goitre in a goat kid: Clinical and pathological findings. *Vet Med Sci*. 2023;9:2796-2799.
9. Singh JL, Sharma MC, Kumar M, Gupta GC, Kumar S. Immune status of goats in endemic goitre and its therapeutic management. *Small Rumin Res*. 2006;63:249-255.
10. Andrewartha KA, Caple IW, Davies WD, McDonald JW. Observations on serum thyroxine concentrations in lambs and ewes to assess iodine nutrition. *Aust Vet J*. 1980;56(1):18-21
11. Gürgöze S, Gökalp E. Şanlıurfa yöresi ankara tiftik

ve halep keçi ırklarına ait bazı biyokimyasal kan parametreleri ile malondialdehit düzeylerinin tespiti. *Harran Univ Vet Fak Derg*. 2018;7:19-23.
12. Wassner AJ, Brown RS. Congenital hypothyroidism: recent advances. *Curr Opin Endocrinol Diabetes Obes*. 2015;22:407-412.
13. Zimmermann MB. Iodine and the iodine deficiency disorders. In: Present Knowledge in Nutrition. 11th ed. Elsevier; 2020:429-441.
14. Pankowski F, Paśko S, Bonecka J, et al. Ultrasonographic and anatomical examination of normal thyroid and internal parathyroid glands in goats. *PLoS ONE*. 2020;15(5):e0233685
15. Ozmen O, Haligur M. Immunohistochemical observations on TSH secreting cells in pituitary glands of goat kids with congenital goitre. *J Vet Med A*. 2005;52:454-459.
16. Agrawal P, Philip R, Saran S, et al. Congenital hypothyroidism. *Indian J Endocrinol Metab*. 2015;19(2):221-227.
17. Wu Q, Rayman MP, Seviye H, et al. Low population selenium status is associated with increased prevalence of thyroid disease. *J Clin Endocrinol Metab*. 2015;100(11):4037–4047.
18. Paksoy N, İriadam M. Kilis keçilerinde serum selenyum düzeylerinin araştırılması. *Harran Univ Vet Fak Derg*. 2012;1:6-8.
19. Sabea AM, Al-Qaiym MA. The Impact of Selenium and Levothyroxine on the Immune System of Hypothyroid Rats. *J Fac Med Baghdad*. 2024;66:85-92.

ATATÜRK ÜNİVERSİTESİ YAYINLARI
ATATURK UNIVERSITY PUBLICATIONS

Case Report *Olgu Sunumu* DOI: 10.5281/zenodo.15755483

Case of Chondrodysplasia in a Holstein Calf

Holstein Irkı Bir Buzağıda Kondrodisplazi Olgusu

Kübra Asena TERİM KAPAKİN[1]
Esra MANAVOĞLU KİRMAN[1]
Serkan YILDIRIM[1]
Armağan ÇOLAK[2]

[1]Atatürk University, Faculty of Veterinary Medicine, Department of Pathology, Erzurum, Türkiye
[2]Atatürk University, Faculty of Veterinary Medicine, Department of Obstetrics and Gynecology, Erzurum, Türkiye

ABSTRACT

The aim of this study was to describe pathomorphologically a case of chondrodysplasia observed in a newborn Holstein calf. According to the anamnesis, the first remarkable finding in the calf, which was delivered in the Department of Obstetrics and Gynecology of our faculty, was that the joints were easily broken during birth. Multiple anomaly findings were observed in the calf that was born. Macroscopic examination revealed that the calf had a disproportionately short body and an enlarged skull. It was also observed that the forelimbs and hind limbs were short and arthrogryposis, scoliosis of the vertebrae and herniation in the umbilical region. When the chest cavity was opened, it was noted that the costae and sternum were not fully developed and the vertebrae were curved. It was noticed that the trachea was smaller and curved than normal and the bronchial structures of the lung were not formed. When the skull was opened, hydrocephalus was noted. The most striking finding was the softness and fragility of all the body bone and cartilage tissues. For histopathological examination, tissue samples were fixed in a 10% buffered formalin solution for 48 hours and decalcified. Following this, routine tissue processing procedures, 4-5 µm thick sections were taken with a microtome, stained with haematoxylin-eosin (H&E), and examined under a light microscope. Histopathological examination revealed degenerative and hypertrophic chondrocytes, as well as incomplete vascularization in the affected area.

Keywords: Calf, Chondrodysplasia, Histopathology

ÖZ

Bu çalışmada Holstein ırkı, yeni doğmuş bir buzağıda gözlenen kondrodisplazi olgusunun patomorfolojik olarak tanımlanması amaçlandı. Alınan anamnez bilgilerine göre, fakültemiz Doğum ve Jinekoloji Anabilim dalında doğumu gerçekleştirilen buzağıda ilk dikkat çekici bulgu doğum esnasında eklemlerinin kolayca kırılması durumuydu. Doğumu gerçekleştirilen buzağıda birden fazla anomali bulguları gözlendi. Yapılan makroskobik incelemede buzağının gövdesinin kısa, kafatasının ise oldukça büyük şekillendiği görüldü. Ayrıca ön ve arka bacakların kısa ve artrogripozisli olduğu, omurlarda skolyoz ve ayrıca umblikal bölgede fıtıklaşmanın şekillendiği gözlendi. Göğüs boşluğu açıldığında ise kostalar ve sternumun tam gelişmediği, vertebraların eğri olduğu dikkat çekti. Trakeanın normalden küçük ve eğri şekillendiği, akciğer bronş yapılarının şekillenmediği fark edildi. Kafatası açıldığında ise hidrosefalus dikkati çekti. En dikkat çekici bulgu ise vücudun tüm kemik ve kıkırdak dokusunun yumuşak ve kırılgan olmasıydı. Histopatolojik inceleme amacıyla doku örnekleri %10'luk tamponlu formalin solüsyonunda 48 saat süreyle fikse edilmiş ve dekalsifikasyon işlemi uygulanmıştır. Bunu takiben, rutin doku işleme prosedürleri gerçekleştirilmiş, mikrotom ile 4-5 µm kalınlığında kesitler alınmış, hematoksilen-eozin (H&E) ile boyanmış ve ışık mikroskobu altında incelenmiştir. Histopatolojik incelemelerde; dejeneratif ve hipertrofik kondrositlerin yanısıra bu bölgede damarlaşmanın da tam şekillenemediği gözlendi.

Anahtar Kelimeler: Buzağı, Kondrodisplazi, Histopatoloji

Geliş Tarihi/Received 03.03.2025
Kabul Tarihi/Accepted 27.05.2025
Yayın Tarihi/Publication Date 30.06.2025

Sorumlu Yazar/Corresponding author:
Kübra Asena TERİM KAPAKİN
E-mail: kubra.terim@atauni.edu.tr

Cite this article: Terim Kapakin KA, Manavoğlu Kirman E, Yıldırım S, Çolak A. Case of Chondrodysplasia in a Holstein Calf. *J Vet Case Rep*. 2025;5(1):20-23.

INTRODUCTION

Congenital anomalies are pathological conditions that can occur during embryonic or fetal development in all animal species including structural or functional defects in tissues, organs, or systems.[1] These malformations are common worldwide and can lead to fetal losses, neonatal deaths, or pregnancy termination. In cattle, in particular, such anomalies result in significant reproductive losses and cause economic losses in the livestock industry.[2]

Although the etiology of congenital malformations has not been fully elucidated, both hereditary and non-hereditary factors have been reported to play a role in the development of these anomalies.[3]

The etiological factors of non-hereditary congenital anomalies in animals include genetic predisposition,3 faulty breeding selection, breed characteristics, consanguinity,[4] parental age, stress factors, nutritional disorders,[5] vitamin deficiencies, exposure to teratogens, and endocrine disorders.[6]

In addition, factors such as fetal position in the uterus, exposure to carcinogenic substances, and pregnancy diagnosis by rectal palpation before day 42 or by rectal ultrasonography between days 28 and 90 of gestation may also contribute to the development of congenital malformations.[2] Mutant genes or chromosomal abnormalities have been shown to play a role in the occurrence of inherited malformations.[3] Chromosomal changes typically manifest as a broad spectrum of malformations rather than as isolated deformities.[1]

Chondrodysplasia, a genetic disorder, is defined as an extremely rare abnormality in cartilage growth or development, which can affect genes or chromosomes and result in varying degrees of disproportionate dwarfism.[2] It typically disrupts transverse or longitudinal cartilage growth, negatively impacting the entire skeletal system. Chondrodysplasia is characterized pathologically by the destruction of endochondral osteogenesis.[6] The probability of birth defects in calves has been reported to be between 0.2% and 0.3%, with 26.6% of these cases involving skeletal and muscular anomalies.[7]

In addition to cattle, chondrodysplasia has also been reported in dogs, pigs, rabbits, and sheep as well as in humans and mice. The aim of this study was to describe pathomorphologically a case of chondrodysplasia observed in a newborn Holstein calf.[5]

CASE PRESENTATION

The subject of this case report was a newborn Holstein calf that was delivered at Atatürk University, Faculty of Veterinary Medicine, Department of Obstetrics and Gynaecology, and died a few minutes after birth. Bone samples obtained from the systemic necropsy were fixed in 10% buffered formalin solution for 48 hours and decalcified in 36.8% formic acid and 6.8% sodium formate for histopathological examinations. They were then embedded in paraffin blocks following a routine tissue processing procedure. Sections of 4–5 µm thickness were taken using a microtome, stained with haematoxylin-eosin (H&E), and examined and imaged with an Olympus BX52 light microscope (Japan) equipped with a DP72 camera system.[8]

Macroscopic examination revealed that all bones of the calf were extremely soft and fragile at birth (Figure 1A). The body of the calf was short, and the skull was disproportionately large. The forelimbs and hind limbs were shortened, and arthrogryposis, scoliosis of the vertebrae, and herniation in the umbilical region were observed (Figure 1B). Upon opening the skull, hydrocephalus was detected (Figures 1C–D). Examination of the thoracic cavity revealed that the ribs and sternum were underdeveloped, and the vertebrae were curved (Figure 1E). Additionally, the trachea was smaller and more curved than normal, and the bronchial structures of the lungs were absent (Figure 1F).

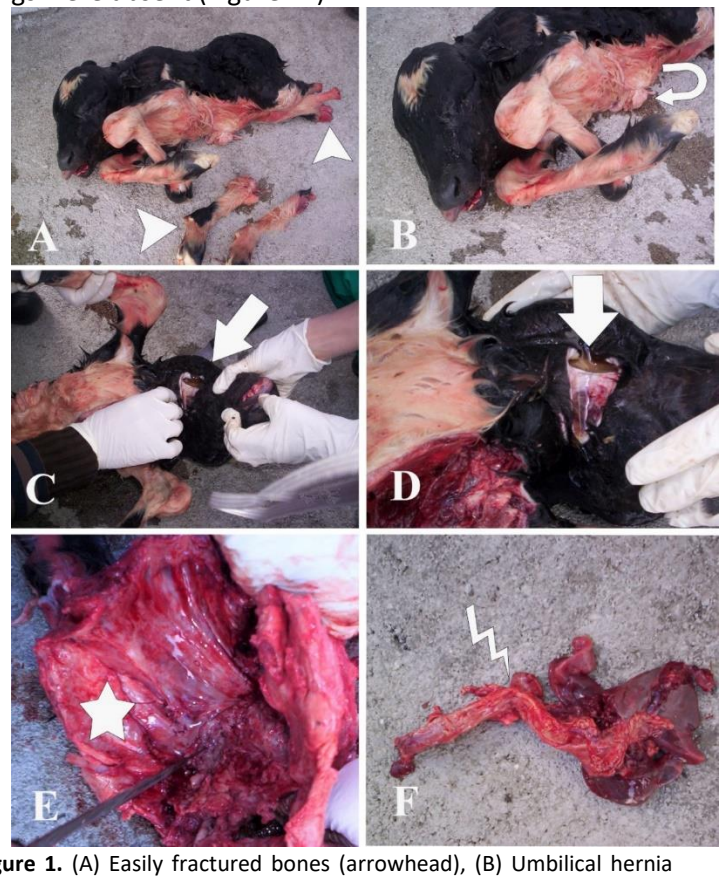

Figure 1. (A) Easily fractured bones (arrowhead), (B) Umbilical hernia (curved arrow), (C–D) Hydrocephalus (arrow), (E) Underdeveloped ribs and sternum (star), (F) Smaller than normal and curved trachea (lightning).

Microscopic examination revealed an increased number of immature chondrocytes, along with decreased or

incomplete vascularization and calcification (Figure 2A–B). Degenerative changes were observed in mature chondrocytes (Figure 2C–D).

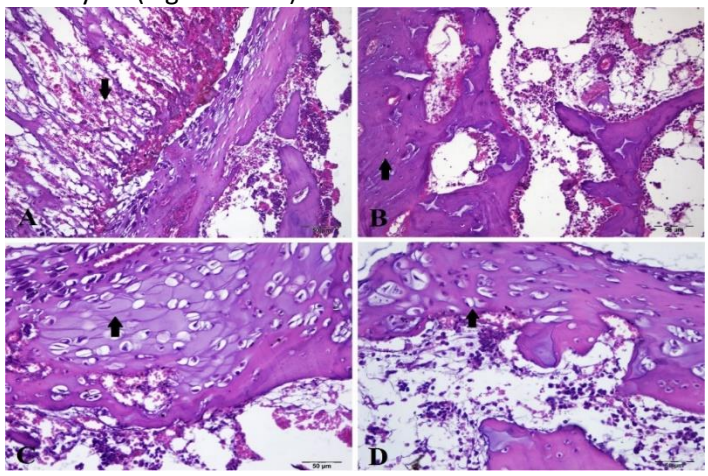

Figure 2. (A–B) Incomplete calcification (arrow), (C–D) Mature chondrocytes and degenerating chondrocytes (arrow), (H&E, 50 µm).

DISCUSSION

Although congenital anomalies in cattle are rare in cattle breeding, they represent a serious problem that can lead to significant economic losses. Various studies on the etiology of these anomalies have shown that multiple factors, including genetic predisposition,[3] environmental influences, and nutritional deficiencies,[5] may play a role.

Genetic factors have been identified as a significant contributor to the development of chondrodysplasia in cattle, and this condition has been shown to be fatal in affected individuals.[3] Chondrodysplasia is a skeletal malformation characterized by defects in endochondral osteogenesis. Macroscopic findings vary widely; however, the common feature in all cases is a disruption in transverse and longitudinal bone development.[6]

The term 'bovine prenatal fatal chondrodysplasia' refers to a genetically heterogeneous group of chondrodysplasias and is characterized by severe micromelic dwarfism.[9] Hereditary chondrodysplasia has been reported in humans, cattle, sheep, dogs, pigs, rabbits, and mice. According to the anamnesis, an aborted fetus with similar findings had previously been obtained from the same bull on this farm, leading to the conclusion that this condition may be hereditary.

It has been reported that in cases of chondrodysplasia, intrauterine death typically occurs between the 6th and 8th months of gestation; however, some affected calves may be born alive, with the majority of these cases classified as bulldog calves.[6] In the present case, chondrodysplasia was observed in a Holstein calf, which died a few minutes after birth. The results of our study aligned with those reported in the existing literature.

Generalized chondrodysplasia is primarily characterized by cartilage defects[10] and is associated with various skeletal and soft tissue abnormalities, including disproportionate dwarfism,[11] a large and domed skull,[10] short limbs,[11] a protruding tongue,[11] cleft palate,[11] tracheal malformations, underdeveloped lung lobes,[12] and ventral abdominal hernia.[13]

Various forms of chondrodysplasia have been identified in cattle, including snorter (brachycephalic), Dexter, and Telemark chondrodysplasias. Generalized chondrodysplasias have been observed in Dexter Holstein,[11,12] Jersey,[14] Highland,[15] Belted Galloway,[6] Nellore,[7] and Miniature Zebu,[16] cattle, and these conditions are collectively referred to as bulldog-type dwarfism.

The diagnosis of the bulldog fetus was based on its physical characteristics, including deviations and deformities from normal development, and was confirmed by previously reported cases in different cattle breeds.[12,15]

In our study, chondrodysplasia was detected in a Holstein calf, and the macroscopic findings were similar to those reported in cases of chondrodysplasia anomaly described by Jacinto et al.[11] These changes, which align with previously described findings in different cattle breeds, including Holstein, consist of structural anomalies thought to be associated with genetic mutations.[11,12]

Bulldog-type chondrodysplasia is characteristically defined by short limbs, a bulging abdomen, a flattened head, and micromelia. Such anomalies have been reported in various cattle breeds, including Jersey,[14] Highland,[15] Belted Galloway,[6] Nellore[7], and Miniature Zebu.[16] Similar macroscopic findings were observed in our case and were consistent with the literature.

In previously reported cases, histopathological examination of bones such as the humerus, radius, femur, and tibia revealed that intramembranous ossification occurred beneath the periosteum, whereas endochondral ossification was severely limited.[17] In addition to abnormal chondrocytes, a large number of incompletely matured chondroblasts were observed, and vascularization was found to be insufficient in these areas.[12] The histopathological findings observed in our case were consistent with those reported in the literature.

In conclusion, chondrodysplasia in calves leads to severe skeletal deformities, reduces the animal's quality of life, and causes economic losses. Early diagnosis is crucial to preventing the spread of this genetic disorder within herds. To achieve this, genetic isolation strategies should be implemented, and selective breeding programs should be established by removing individuals carrying the chondrodysplasia mutation from the herd.

Additionally, the widespread adoption of genetic

testing, raising awareness among breeders, and ensuring regular veterinary inspections of calves are critical measures to control the spread of the disease. These actions will not only improve animal welfare but also minimize potential negative impacts on the national economy. Therefore, this case was considered noteworthy and was reported to our department.

Peer-review: Externally peer-reviewed.
Author Contributions: Concept-KATK, EMK, SY, AÇ; Design-KATK, EMK; Supervision-KATK; Resources-KATK, SY; Data Collection and/or Processing-EMK; Analysis and/or Interpretation-SY, AÇ; Literature Search-EMK; Writing Manuscript-KATK, EMK; Critical Review-KATK, EMK.
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- KATK, EMK, SY, AÇ; Tasarım- KATK, EMK; Denetleme- KATK; Kaynaklar- KATK, SY; Veri Toplanması ve/veya İşlemesi-EMK; Analiz ve/ veya Yorum-SY, AÇ; Literatür Taraması-EMK; Yazıyı Yazan- KATK, EMK; Eleştirel İnceleme- KATK, EMK.
Çıkar Çatışması: Yazarlar, çıkar çatışması olmadığını beyan etmiştir.
Finansal Destek: Yazarlar, bu çalışma için finansal destek almadığını beyan etmiştir.

REFERENCES

1. Dart A. Congenital and inherited anomalies in animals [Internet]. MSD Veterinary Manual; 2020 [cited 2025 Mar 2]. Available from: https://www.msdvetmanual.com/generalized-conditions/congenital-and-inherited-anomalies-in-animals
2. Blowey RW, Weaver AD. Congenital disorders. In: Colour Atlas of Diseases and Disorders in cattle. 3 rd ed. Edinburgh, Mosby, 2011, s 1-11.
3. Gholap PN, Kale DS, Sirothia AR. Genetic diseases in cattle: A review. *Res J Anim Vet Fish Sci*. 2014;2(2):24-33.
4. Bosse M, Megens HJ, Derks MF, de Cara ÁM, Groenen MA. Deleterious alleles in the context of domestication, inbreeding, and selection. *Evol Appl.* 2019;12(1):6-17. https://doi.org/10.1111/eva.12691
5. Schwertz CI, Bianchi RM, Vielmo A, et al. Nutritional chondrodysplasia in cattle in Brazil. *Trop Anim Health Prod*. 2023;55(1):26. https://doi.org/10.1007/s11250-022-03438-7
6. Dittmer KE, Thompson KG. Approach to investigating congenital skeletal abnormalities in livestock. *Vet Pathol.* 2015;52(5):851-61. https://doi.org/10.1177/030098581557
7. Moura E, Pimpão CT. Veterinary dysmorphology. In: Perez-Marin CC, editor. A Bird's-Eye View of Veterinary Medicine. Rijeka: InTech; 2012.
8. Kapakin AKT, Kutsal O. Tavuklarda deneysel amiloid artropatinin ışık mikroskobik ve elektron mikroskobik incelenmesi. *Ankara Univ Vet Fak Derg.* 2008;55(1):37-42.
9. Kostyunina O, Yaryshkin A, Bykova O, et al. Identification of lethal recessive genetic variants in Holstein cattle. In: BIO Web of Conferences. Vol. 108. EDP Sciences; 2024. p. 01002.
10. D'Soares CS, Muramoto C, Silva AW, et al. Chondrodysplasia Bulldog type in cattle in the state of Bahia, Brazil. *Braz J Vet Pathol*. 2020;13(2):536-541. DOI: 10.24070/bjvp.1983-0246.v13i2p536-541
11. Jacinto JGP, Häfliger IM, Letko A, Drögemüller C, Agerholm JS. A large deletion in the COL2A1 gene expands the spectrum of pathogenic variants causing bulldog calf syndrome in cattle. *Acta Vet Scand*. 2020;62(1). https://doi.org/10.1186/s13028-020-00548-w
12. Agerholm JS, Menzi F, McEvoy FJ, Jagannathan V, Drögemüller C. Lethal chondrodysplasia in a family of Holstein cattle is associated with a de novo splice site variant of COL2A1. *BMC Vet Res*. 2016;12:1-9. DOI: 10.1186/s12917-016-0739-z
13. Wurster F, Bassuino DM, Juffo GD, et al. Chondrodysplasia Dexter type in aborted bovine fetuses. *Acta Sci Vet.* 2012;40(3):1060-1064.
14. Muhammad SA, Sajid M, Shahzad AH, et al. Delivery of bulldog type achondroplasia calf after management of dystocia in Sahiwal cattle. *Pak J Life Soc Sci*. 2021;19:43-5.
15. Cabrera LC, McNabb BR, Woods ES, Cartoceti NA. Hydrops associated with chondrodysplasia of the fetus in a miniature Scottish Highland cow. *J Am Vet Med Assoc.* 2016;248:552-6. https://doi.org/10.2460/javma.248.5.552
16. Struck K, Dierks C, Braun M, et al. A recessive lethal chondrodysplasia in a miniature Zebu family results from an insertion affecting the chondroitin sulfate domain of aggrecan. *BMC Genet*. 2018;19:91. https://doi.org/10.1186/s12863-018-0678-8
17. Reinartz S, Schütz E, Jagannathan V, Leeb T, Drögemüller C. Germline mutation within COL2A1 associated with lethal chondrodysplasia in a polled Holstein family. *BMC Genomics*. 2017;18:1-10.