

Anal Stenosis in a Dairy Calf

Laura Rosa Corrêa^{ID}, Tainá Barbosa Cunha^{ID},
Thaís Mendes dos Santos^{ID}, Gabriely Araújo do Amaral^{ID} & Fernanda Carlini Cunha dos Santos^{ID}

ABSTRACT

Background: Congenital abnormalities, present at birth, affect both structure and function, posing remarkable challenges in livestock. Anal atresia is a congenital malformation categorized in 4 types and characterized based on whether the occlusion of the anal opening is complete or partial. Clinical signs usually manifest within the first days of life due to fecal retention. Diagnosis is based on clinical history and physical examination. Treatment involves surgical reconstruction, with outcomes depending on the type of atresia and the moment of intervention. This report describes the therapeutic management of a 7-month-old mixed-breed dairy calf diagnosed with type I anal atresia, specifically anal stenosis.

Case: A 7-month-old mixed-breed female calf (~80 kg) with weight loss and chronic defecation difficulty since birth presented with tenesmus, rectal distension, and a narrowed anal orifice measuring 0.5 cm in diameter. Upon presentation, the calf had a body condition score of 4, in a scale from 1 to 9, showing emaciation and notable underdevelopment relative to other age-matched calves. Throughout the clinical evaluation, the animal was diagnosed with type I congenital anal atresia (anal stenosis). The herd had no prior similar cases. Under field conditions, after sedation and local anesthesia, surgical correction was performed via anoplasty. Postoperative care involved wound cleaning, antibiotics, and anti-inflammatory treatment. Fifteen days postoperatively, the anal orifice had expanded to 1.3 cm in width and 2.5 cm in height, with marked improvement in appetite, defecation and increased fecal output. Delayed diagnosis adversely affected the calf's physical development; however, surgical intervention via anoplasty successfully restored fecal elimination. Given the potential for a hereditary component, the owner was advised not to include the calf in future breeding programs and to avoid repeating the parental cross that produced this individual.

Discussion: The case report demonstrates that even when surgical correction of type I anal atresia is delayed beyond the neonatal stage, favorable outcomes are achievable in animals without systemic compromise or additional malformations. The 7-month-old calf responded well to anoplasty, with restored fecal passage, improved condition, and no complications during follow-up. These results align with literature indicating that timely diagnosis and proper surgical technique are key to prognosis, though early intervention remains preferable. The case also reinforces the hereditary component suspected in some congenital anorectal malformations, making it advisable to exclude affected animals from breeding and avoid repeating the parental cross. Anoplasty remains the definitive treatment, being feasible under field conditions when performed with adequate technique to improve anal lumen size. Postoperative complications, though uncommon, can include fecal incontinence, infection, wound dehiscence, and restenosis, underscoring the need for regular follow-up and owner education. Congenital anal atresia is a rare, yet potentially life-threatening condition in calves, necessitating prompt surgical intervention to restore normal defecation and prevent systemic complications. Early diagnosis and timely treatment are essential to avoid growth retardation and enhance the quality of life in the long term. This case contributes to the growing body of evidence that surgical correction of anal atresia can successfully restore quality of life, even when performed later in life, although initial growth may be markedly impaired.

Keywords: anus, atresia ani, anal dysgenesis, fecal retention, congenital abnormality, gastrointestinal.

DOI: 10.22456/1679-9216.149524

Received: 19 December 2025

Accepted: 12 April 2026

Published: 16 May 2026

Instituto de Ciências Agrárias (ICA), Universidade Federal dos Vales Jequitinhonha e Mucuri (UFVJM), Campus Unaí, Unaí, MG, Brazil. CORRESPONDENCE: F.C.C. Santos [carlini.fernanda@hotmail.com]. ICA - Universidade Federal dos Vales Jequitinhonha e Mucuri (UFVJM). Av. Universitária n. 1000. CEP 38610-000 Unaí, MG, Brazil.

INTRODUCTION

Congenital abnormalities, which are present at birth, are defined as defects in structure and function [2] and are a major challenge under field conditions owing to their complex nature. Congenital malformations sometimes lead to prenatal mortality and reduce the value of defective neonates [3].

Congenital defects can occur in 0.5 to 3% of newborn cattle, and anomalies associated with the gastrointestinal and urogenital tracts are the most common [6].

Atresia ani a congenital alteration characterized by total or partial occlusion of the anal orifice, which may be accompanied by other congenital malformations [7]. Clinical signs are generally evident soon after birth and the diagnosis is supported by noting evacuation difficulties or the absence of defecation [18].

Four types of atresia ani have been reported, including Type I: anal stenosis; Type II: imperforate anus alone; Type III, imperforated anus with more cranial termination of the rectum as a blind pouch; Type IV: discontinuity of the proximal rectum with normal anal and terminal rectal development [1].

In domestic animals, surgical intervention is the only option for correction and different techniques have been practiced [9], according to the type of atresia ani.

In this context, this case report aimed to describe the therapeutic management in a 7-month-old dairy calf with type-I congenital anal atresia.

CASE

A 7-month-old mixed-breed female calf, weighing approximately 80 kg, was examined owing to chronic difficulty in defecation. According to the owner, the calf had shown signs of abnormal anal morphology, poor fecal output, and growth retardation since birth.

The animal came from a dairy herd of 150 animals, including 2 breeding bulls (<3 years), 75 adult cows, and 73 young females (>3 years). The herd was managed under continuous natural mating, with bulls cohabiting year-round. Endo- and ectoparasite control was conducted empirically, restricted to adult animals. Routine vaccination included coverage for rabies, foot-and-mouth disease, brucellosis, and clostridiosis.

The dam was an 8-year-old mixed-breed cow with multiple successful parturitions and no history of gestational or neonatal abnormalities. The sire was the Nelore breed. No similar congenital conditions had been reported in the herd.

Postpartum, the calf was allowed to nurse in the early morning hours and then housed with age-matched calves. The animal had *ad libitum* access to water and received a commercial concentrate feed.

Upon presentation, the calf had a body condition score of 4, in a scale from 1 to 9. Vital signs were within reference ranges: heart rate 60 bpm, respiratory rate 30 bpm, body temperature 38.6°C, capillary refill time 2 s, dehydration estimated at <5%; and pink mucous membranes. Abdominal auscultation revealed hypomotility.

The anal orifice measured approximately 0.5 cm in diameter, with pasty-to-dry fecal material adhered to the tail, perineum, and hind limbs (Figure 1). The calf exhibited tenesmus and passed only small quantities of feces with marked effort. Digital rectal palpation confirmed the distension of the rectal ampulla (Figure 2). No additional congenital anomalies were observed. The calf was notably underdeveloped relative to other age-matched calves (Figure 3).

Based on the clinical presentation and history, a diagnosis of type I congenital anal atresia (anal stenosis) was established. Surgical correction via anoplasty was selected.

For preoperative preparation, the calf underwent a 24-h fasting period for solids and 2 h for water. Sedation was achieved with xylazine¹ [Xylazin® - 0.025 mg/kg IM]. The animal was placed in right lateral recumbency. The perineal region was clipped, aseptically prepared, and infiltrated with lidocaine² for local anesthesia.

Surgery was performed with a midline incision over the anal orifice. The rectum was isolated, and the incision was extended dorsally and ventrally by approximately 2 cm. The rectal mucosa was sutured to the skin using 0 nylon in a simple interrupted pattern. The postoperative anal opening measured 3 cm in height and 2 cm in width (Figure 4).

Postoperative care included daily wound cleansing with clean water, single dose of sulfadoxime² [15 mg/kg] and trimethoprim³ [Trisulfim® - 3 mg/kg SC] and flunixin meglumine³ [1.1 mg/kg IM SID for 3 days].

At 15 days post-surgery, the surgical site showed proper healing, and sutures were removed. The anal orifice measured 1.3 cm in width and 2.5 cm in height (Figure 5). The calf showed improved defecation, with passage of an increased volume of feces.

The calf demonstrated improved appetite, reduced defecation effort, and increased fecal output through the anal orifice following surgery. At 24

months of age, 17 months post-surgery, examination of the anal region revealed incomplete anal closure. Despite this finding, the animal showed no apparent compromise in overall health status, as indicated by normal growth and a body condition score of 7 on a 1–9 scale (Figure 6). Given the potential for a hereditary component, the owner was advised not to include the calf in future breeding programs and to avoid repeating the parental cross that produced this individual.



Figure 1. Fecal accumulation and adherence in the perineal region of a 7-month-old calf diagnosed with type I anal atresia.



Figure 2. Distension of the rectal ampulla with marked fecal retention in a calf presenting with anal stenosis.



Figure 3. Emaciated 7-month-old mixed-breed heifer diagnosed with anal stenosis.



Figure 4. Dairy calf with type I congenital anal atresia immediately after surgical correction by anoplasty, showing a widened anal orifice measuring approximately 3 cm in height and 2 cm in width.



Figure 5. Surgical site in the same dairy calf, 15 days after anoplasty, demonstrating complete healing and a stable anal opening.

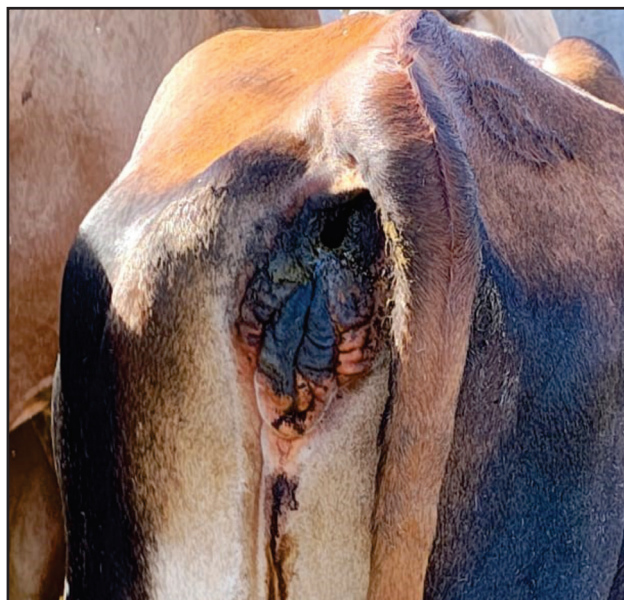


Figure 6. Heifer at 24 months of age with incomplete anal closure, 17 months after undergoing anoplasty for correction of type I congenital anal atresia.

DISCUSSION

Intestinal atresia has been documented across many animal species [20]. Anal atresia, which is a congenital anorectal malformation, is most frequently reported in ruminants and swine [5,12,21]. Atresia ani arises from a failure of recanalization of the anorectal membrane during embryogenesis and can manifest as either complete imperforation or a narrowed anal canal, consistent with type I. In the present case, a 7-month-old dairy calf exhibited classical clinical signs, including chronic tenesmus, perineal fecal soiling, and stunted growth, typical indicators of longstanding partial anorectal obstruction.

Congenital malformations may arise from genetic defects, environmental influences, or a combination of factors [17]. Many of these anomalies are primarily associated with autosomal recessive inheritance [4]. Hereditary factors have also been implicated in the pathogenesis of atresia ani [5,12]. Consequently, the breeding of affected animals is discouraged, and repeating the same parental cross should be avoided, as emphasized in this report.

Diagnosis was established through clinical history and rectal examination, consistent with literature reporting that early identification is often achievable by physical examination alone, although usually performed in newborns [16,23]. While imaging techniques such as ultrasonography can enhance diagnostic confi-

dence [24] and surgical planning, these were unavailable in this case because of owner financial constraints.

Anal atresia is classified into 4 types (I–IV) based on the degree of rectal and anal dysgenesis or agenesis [1]. Type I is characterized by a morphologically normal rectum and a stenotic anal opening. Despite signs of tenesmus, defecation may still occur, allowing affected animals to survive into puberty or even adulthood, although growth may be impaired. The condition can be associated with atresia recti, rectovaginal fistula, rectocystic fistula, vaginourethral agenesis, taillessness, hypospadias and cleft scrota [19]. The delayed surgical intervention of type I atresia ani at 7 months of age contrasts with most documented cases, where corrective procedures are typically performed during the neonatal period. Nevertheless, the calf the outcome of anoplasty was favorable, with immediate and sustained improvement in fecal passage. This outcome suggests that, even when performed at a later stage, surgical correction can be effective in animals with no systemic compromise or associated congenital anomalies. In alignment with the present case and existing literature, a successful surgical correction of type I anal atresia was reported in a 6-month-old male bovine, with no recurrence of clinical signs following the procedure [9].

Anoplasty remains the definitive treatment for anal atresia, as it enables the development of a functional neo-anus, thereby restoring fecal passage

and improving the animal's well-being [18]. The procedure can be effectively performed under field conditions and in the preset case, a vertical incision facilitated mucocutaneous anastomosis, resulting in a substantial increase in the anal opening from 0.5 cm to 3.0×2.0 cm postoperatively. Wound healing was uneventful, with no postoperative complications such as fecal incontinence or stricture formation observed over a six-month follow-up period.

When diagnosis is achieved in the early stages – particularly in the absence of systemic alterations – most reported cases of atresia ani show favorable outcomes following surgical correction. In a 3-year-old pony newborn, the surgical correction of type-II atresia ani was successful, with the anal orifice showing patency immediately after anoplasty and favorable prognosis [15]. A 3-day-old Sahiwal cow-calf presented relieved and marked improvement in defecation soon after surgery to correct atresia ani et recti along with pervious urachus, with uneventful recovery within 12 days post-operatively [18]. A day-old calf was diagnosed with atresia ani complicated by a rectovaginal fistula and, under epidural anesthesia, the anal opening was reconstructed and the fistula was closed, in this case, the prognosis was favorable [10]. A newborn male Anatolian water buffalo calf with type-II atresia ani (no anal opening) underwent anal repair with a plus-shaped incision of up to 2×2 cm in length, resulting in moderate stenosis after the procedure [22]. A 3-month-old female African buffalo with atresia ani and a rectovaginal fistula was diagnosed, after showing clinical signs of abdominal distension and bulging of the perineal skin. The female underwent a two-step surgery. The rectovaginal communication was closed and the calf was able to urinate and defecate normally. The animal grew to become a normal adult without any complications [14].

Postoperative complications following anoplasty for anal atresia in calves are relatively uncommon when the procedure is executed correctly and under aseptic conditions; however, they can still occur

and warrant careful monitoring. The most frequently reported complications include fecal incontinence, surgical site infection, wound dehiscence, and anal stricture formation. Fecal incontinence may result from congenital absence or underdevelopment of the external anal sphincter, or from iatrogenic nerve damage [13]. Anal stenosis or restenosis can develop owing to excessive scar tissue formation or insufficient dilation during the initial surgery, necessitating further intervention. In the present case, no postoperative complications were observed, suggesting that the technique was appropriate and healing proceeded as expected. Nonetheless, regular follow-up and owner education are essential to detect and address potential issues promptly.

In conclusion, congenital anal atresia is a rare but life-threatening condition in calves, requiring prompt surgical intervention to ensure normal defecation and prevent systemic complications.

Delayed treatment can significantly compromise the calf's growth and well-being owing to chronic pain and defecatory dysfunction. Despite the late intervention at 7 months of age, anoplasty was successfully performed, effectively enlarging the anal orifice and restoring fecal elimination. This outcome underscores the importance of early diagnosis and surgical correction to optimize prognosis and improve life quality in the long term in affected animals.

MANUFACTURERS

¹Syntec do Brasil Ltda. Santana de Parnaíba, SP, Brazil.

²Laboratório Novafarma. Anápolis, GO, Brazil.

³Ouro Fino Saúde Animal. Cravinhos, SP, Brazil.

Acknowledgments. The authors thank Universidade Federal dos Vales Jequitinhonha e Mucuri for support

Declaration of interest. The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

REFERENCES

- 1 **Aronson L. 2007.** Rectum and anus. In: D. Slatter (Ed). *Textbook of Small Animal Surgery*. 3rd edn. Philadelphia: WB Saunders, pp.682-707.
- 2 **Badawy A.M. 2011.** Some congenital malformations in ruminants and equines with special reference to the surgical treatment of recto–vaginal and cysto–rectal fistulae. *Benha Veterinary Medical Journal*. 1: 14-27.

- 3 Bademkiran S., Icen H. & Kurt D. 2009. Congenital rectovaginal fistula with atresia ani in a heifer: A case report. *YYU Veteriner Fakültesi Dergisi*. 20(1): 61-64. DOI: 10.5958/2277-3371.2015.00014.5
- 4 Bryan L., Schmutz S., Hodges S.D. & Syneder F.F. 1993. Bovine β mannosidosis: Pathologic and genetic findings in Salers calves. *Veterinary Pathology*. 30: 130-139. DOI: 10.1177/030098589303000205
- 5 Cassini P., Montironi A., Botti A., Hori T., Okhawa H., Stella A., Andersson L. & Giuffra E. 2005. Genetic analysis of anal atresia in pigs: Evidence for segregation at two main loci. *Mammalian Genome*. 16: 164-170. DOI: 10.1007/s00335-004-3024-6.
- 6 Dantas A.F.M., Riet Correa F., Medeiros R.M.T., Galiza G.J.N., Pimentel L., Anjos B.L. & Mota R.A. 2010. Malformações congênitas em ruminantes no semiárido do Nordeste brasileiro. *Pesquisa Veterinária Brasileira*. 30(10): 807-815. DOI: 10.1590/S0100-736X2010001000002.
- 7 Debasis J. & Mousumi J. 2010. Perosomus acaudatus (Anury) in a local non descript milch cow: A report. *North-East Veterinarian*. 10: 1-25.
- 8 Fernandes M.E.S.L., Caldas S.A., Rocha L.R., Chenard M.G., Freire K.R.F., Carvalho N.S., Nogueira V.A. & Helayel M.A. 2021. Atresia Ani (Imperforated Anus) in calves: Clinical, surgical and pathological aspects. *Acta Scientiae Veterinariae*. 49(Suppl 1): 681. DOI: 10.22456/1679-9216.112980.
- 9 Jubb K.V.F., Kennedy P.C. & Palmer N. 1993. *Pathology of Domestic Animals*. 4th edn. San Diego: Academic Press, p.747.
- 10 Kumar G., Arora N., Tiwari D.K., Sharma S. & Kaushik D. 2020. Surgical management of atresia ani complicated with rectovaginal fistula in a Sahiwal calf. *Journal of Entomology and Zoology Studies*. 8(3): 714-716.
- 11 Loynachan A.T., Jackson C.B. & Harrison L.R. 2006. Complete diphallia, imperforate ani (type II atresia ani), and an accessory scrotum in a 5 day old calf. *Journal of Veterinary Diagnostic Investigation*. 18(4): 408-412. DOI: 10.1177/104063870601800418.
- 12 Mauch T.J. & Albertine K.H. 2002. Urorectal septum malformation sequence: Insights into pathogenesis. *The Anatomical Record*. 268(4): 405-410. DOI: 10.1002/ar.10180.
- 13 Prassinis N.N., Papazoglou L.G., Adamama-Moraitou K.K., Galatos A.D. & Gouletsou P. 2003. Congenital anorectal abnormalities in six dogs. *The Veterinary Record*. 153(3): 81-85. DOI: 10.1136/vr.153.3.81.
- 14 Ryu J., Kang S.G., Yun J. & Yeo Y. 2018. Surgical repair of atresia ani with rectovaginal fistula in an African buffalo (*Syncerus caffer*). *Journal of Veterinary Clinics*. 35(3): 111-113. DOI: 10.17555/jvc.2018.06.35.3.111
- 15 Santos F.C.C., Feijo L., Pazinato F.M., Amaral L.A., Curcio B. & Nogueira C. 2013. Atresia anal em pônei – Relato de caso. *Revista Brasileira de Medicina Equina*. 49(49): 22-25.
- 16 Shakoar A., Muhammad S.A., Younus M. & Kashif M. 2012. Surgical repair of congenital recto vaginal fistula with atresia ani in a cow calf. *Pakistan Veterinary Journal*. 32(2): 298-300.
- 17 Shukla S.P., Nema S.P., Pandey A.K. & Garg U.K. 2007. Dystocia due to a bulldog calf in a she buffalo. *Buffalo Bulletin*. 26: 104-105.
- 18 Singh N.K., Kumar A. & Kumar P. 2018. Surgical correction of atresia ani et recti along with pervious urachus in Sahiwal cow calves. *Journal of Natural Science, Biology and Medicine*. 9: 288-290. DOI: 10.4103/jnsbm.JNSBM_226_17
- 19 Tyagi R.P.S. & Singh J. 1993. *Ruminant Surgery*. New Delhi: C.B.S. Publishers and Distributors Pvt Ltd., 484p.
- 20 van der Gaag I. & Tibboel D. 1980. Intestinal atresia and stenosis in animals: A report of 34 cases. *Veterinary Pathology*. 17(5): 565-574. DOI: 10.1177/030098588001700505
- 21 Vahar M., Hosseini S.M., Omidzahir S., Kenari E.O., Iraee M.A. & Ziabari A.R.H. 2015. Report of congenital colonoblaster fistula with atresia ani in a lamb and surgical treatment. *Asian Pacific Journal of Tropical Disease*. 5(1): S181-S183. DOI:10.1016/S2222-1808(15)60885-4
- 22 Varol K., Atalan G., Gunes V., Alpman U. & Yonez M.K. 2018. A case of atresia ani in an Anatolian water buffalo calf. *Erciyes Üniversitesi Veteriner Fakültesi Dergisi*. 15(3): 271-275.
- 23 Weaver A.D., Jean G.S. & Steiner A. 2005. In: *Bovine Surgery and Lameness*. 2nd edn. Oxford: Blackwell Publishing, 370p.
- 24 Yanaka R., Ferreira H.N., Assis M.M.Q., Oliveira G.K., Albuquerque V.B. & Sartori V.C. 2012. Defeitos congênitos múltiplos em bezerros nelores: Relato de caso. *Revista Arquivos Veterinária*. 28(3): 144-147.