

BJAHE – BRAZILIAN JOURNAL OF ANIMAL, HEALTH AN ENVIRONMENT

http://bjahe.com.br

Case report

Sabulous cystitis due to idiopathic bladder paralysis syndrome in a gelding

[Cistite sabulosa devido à síndrome de paralisia idiopática da bexiga em um equino castrado]

Joandes Henrique Fonteque¹, Anderson Fernando de Souza^{1*}, Milena Carol Sbrussi Granella¹,

Fabrício Desconsi Mozzaquatro¹, Mere Erika Saito¹, Nádia Cristine Weinert¹, Julieta Volpato¹

¹Departamento de Medicina Veterinária, Centro de Ciências Agroveterinárias da Universidade do Estado de Santa Catarina, Lages, SC, Brazil.

ABSTRACT

Correspondence author: anderson.sji@hotmail.com

Received: March 23, 2024 Accepted: May 15, 2024

Copyright:

This is an open access article distributed under the terms of the Creative Commons Attribution License which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Idiopathic bladder paralysis in horses can cause accumulation of macroscopic concretions in the ventral aspect of the organ, named sabulous cystitis. A 16-yearold crossbred gelding, weighing 290 kg, that had been experiencing urinary incontinence for approximately two months was reported. Physical examination revealed urinary incontinence, edema, and irritation and crust formations in the preputial region, with vital parameters within normal limits. Transrectal palpation showed a distended bladder easily emptied through manual pressure, with the urine presenting a large amount of sediment with a sandy appearance. Urinalysis revealed an abundance of crystals of calcium carbonate, amorphous phosphate, and calcium phosphate, and the transrectal ultrasound examination showed thickening of the urinary bladder mucosa and content with a large amount of sediment, diffuse hyperechogenic formations, and distended pelvic urethra with fluid in its entire length. The treatment included the administration of bethanechol chloride, glycerin in the prepuce region and limbs to avoid skin lesions, and correction in the feeding. Idiopathic bladder paralysis syndrome is an infrequent case in the clinical routine of horses that covered the urinary tract disorder, but it did not compromise the animal's survival.

Keywords: horse, neuropathy, urinary bladder, urinary incontinence, urolith

RESUMO

A paralisia idiopática da bexiga em equinos pode causar acúmulo de sedimentos macroscópicos no aspecto ventral do órgão, denominada cistite sabulosa. Relata-se o caso de um equino castrado de 16 anos de idade, 290 kg, que apresentava incontinência urinária há aproximadamente dois meses. O exame físico revelou incontinência urinária, edema, irritação e formações de crosta na região do prepúcio, com parâmetros vitais dentro da normalidade. À palpação transretal identificou-se a bexiga distendida e facilmente esvaziada por pressão manual, com a urina apresentando uma grande quantidade de sedimentos com aparência arenosa (sabulosa). O exame de urina revelou abundância de cristais de carbonato de cálcio, fosfato amorfo e fosfato de cálcio, e ultrassonografia transretal mostrou espessamento da mucosa da bexiga e conteúdo com grande quantidade de sedimentos, formações hiperecogênicas difusas e uretra pélvica distendida com líquido em toda a sua extensão. O tratamento incluiu a administração de cloreto de betanecol, glicerina tópica na região do prepúcio e membros para evitar lesões cutâneas e correção na alimentação. A síndrome da paralisia idiopática da bexiga é infrequente na rotina clínica de equinos que apresentam distúrbio do trato urinário, mas não compromete a sobrevivência do animal

Palavras-chave: bexiga urinária, equino, incontinência urinária, neuropatia, urólito

INTRODUCTION

Urinary tract disorders occur less frequently in the clinical routine of equine practitioners, occasionally as a primary event usually associated with mechanical obstructions or dysfunctions that prevent the normal flow of urine (Duesterdieck-Zellmer 2007). The most easily perceived clinical manifestation is drop urination or urinary incontinence. It may be due to neurogenic causes involving dysfunctions associated with equine herpesvirus type 1 (EHV-1) myeloencephalopathy, neuritis of the cauda equina, sacral trauma, cervical compressive myelopathy, equine protozoal myeloencephalitis, or myelopathy (Mair 2022). Non-neurological causes include developmental abnormalities (ectopic ureters), estrogen-sensitive sphincter, cystolithiasis, pregnancy, parturition, sorghum poisoning, and idiopathic bladder paralysis syndrome (Holt 1996; Mair 2022).

Urinary incontinence associated with idiopathic bladder paralysis syndrome determines the incomplete emptying of the organ, secondarily causing the formation of macroscopic concretions with a sandy appearance, a condition known as sabulous urolithiasis (Schott II 2006). The permanence and accumulation of crystalloid content inside the urinary bladder cause potential inflammation and consequent cystitis, in addition to predisposing to disorders of the detrusor muscle (Rendle 2008). Some authors proposed that the term sabulous cystitis would be more appropriate than sabulous urolithiasis because there is no formation of uroliths. The deposition of calculus in the urinary bladder is predisposed because the urinary pH is alkaline and forms large amounts of calcium carbonate (CaCO₃), which, associated with dietary factors, bacterial infections, abnormal mineral excretion, vesical atony, and urinary incontinence, results in the formation of macroscopic sediments mainly in the ventral portion of the organ (Keen 2006; Duesterdieck-Zellmer 2007).

Adult geldings over 10 years old are more susceptible to the development of clinical signs compared to mares (Holt 1996; Duesterdieck-Zellmer 2007). This context is attributed to the longer and less distensible urethra, which favors the deposition of calculus in males (Duesterdieck-Zellmer 2007). The etiopathogenesis of sabulous cystitis is uncertain but it is thought to represent the final stage of a series of conditions that result in incomplete emptying of the urinary bladder (Keen 2006). Considering the infrequent clinical presentation of urinary tract alterations in horses, this case report aimed to describe a case of sabulous cystitis as a condition that should be included in the differential diagnosis in a 16-year-old crossbred

CASE REPORT

A 16-year-old gelding weighing 290 kg was referred to the Veterinary Teaching Hospital of the Agroveterinary Sciences Center of the Santa Catarina State University, Lages, Brazil. The animal presented a history of urinary incontinence for two months. Physical examination revealed continuous urination in drops (urinary incontinence), edema, irritation, and crust formations in the preputial region with the presence of purulent secretion in moderate amounts, distal skin lesions in hindlimbs due to continuous contact with urine, and vital parameters within normal limits (Figure 1). The neurological examination (including evaluation of mental state and behavior, cranial nerves, muscle tone and spinal reflexes, as well as evaluation of movement, posture and postural reactions (Andrade et al. 2021)) showed no alterations. The animal was used for work (carting). It received commercial feed with 12% crude protein, twice a day (1% body mass in total), native pasture, and water ad libitum.

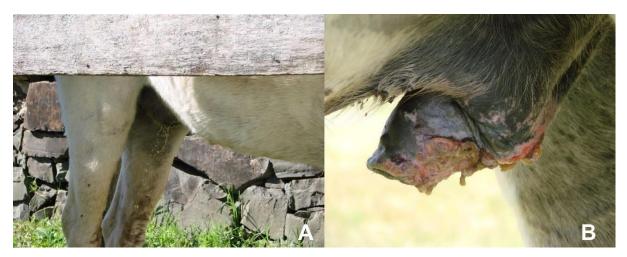


Figure 1. A 16-year-old gelding with clinical suspicion of sabulous cystitis and showing urinary incontinence (A). Skin lesions characterized by edema, irritation, and crust formations in the preputial region with the presence of purulent secretion in a moderate amount (B).

Transrectal palpation presented a distended bladder that was easily emptied by manual pressure, with the urine having a large amount of substance with a sandy appearance (Figure 2). Blood count and serum biochemistry showed no changes. Urinalysis (collection by natural urination) revealed in the physical examination: volume of 60 ml, brownish-yellow color, sui generis odor, intensely turbid aspect, a density of 1036; chemical test: proteinuria (+), negative

glucose, negative acetone, negative bilirubin, normal urobilinogen, occult blood (+++), pH 8.5; sediment examination: rare renal cells, rare bladder cells, rare leukocytes, 1–2 erythrocytes per field, absence of hyaline, granular, and waxy casts, absence of mucus and spermatozoa, rare bacteria, an abundance of calcium carbonate crystals (+++), amorphous phosphate crystals (+++), and calcium phosphate (+++) (Figure 3). Transrectal ultrasound examination revealed thickened urinary bladder mucosa and content with large amounts of sediment and diffuse hyperechoic formations. The pelvic urethra was distended with fluid throughout its length. The clinical suspicion was sabulous cystitis due to idiopathic bladder paralysis syndrome.



Figure 2. Brownish-colored urine showing a large amount of sediment with a sandy appearance (sand accumulation) from a 16-year-old gelding with clinical suspected sabulous cystitis.

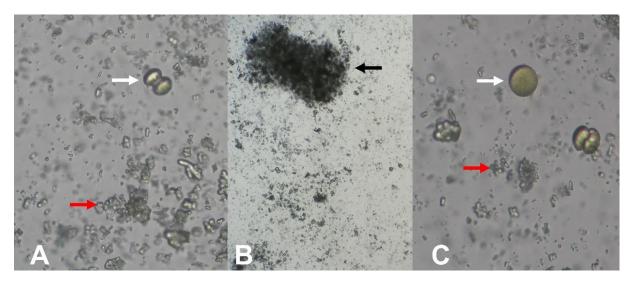


Figure 3. A, B and C, microscopic analysis of urine, presence of amorphous phosphate crystals (black arrow), calcium carbonate (white arrows) and calcium phosphate (red arrows) from a 16-year-old male gelding with suspected sabulous cystitis. 40x magnification, no staining was used.

It was recommended to use topical glycerin to avoid the formation of crusts and

possible skin lesions in the regions that came into direct contact with the urine, mainly the prepuce, the medial side of the hindlimbs, and distally to the hock. Bethanechol chloride (0.025 mg/kg, PO, TID) was administered, allowing the exposure of the penis with urination mimicry, and dripping after approximately 30 minutes. Daily follow-up and observation of the animal were indicated to monitor possible consequences of the disease. Dietary modifications were recommended and included monitoring the calcium and protein content of the commercial feed offered to the animal and offering alfalfa-based feed with caution. In addition, it was also recommended to stimulate water consumption to increase urine production, by including common salt in the diet.

DISCUSSION

The present report could not identify the potential cause of bladder paralysis, thus being characterized as idiopathic. However, etiologies of urinary bladder paralysis include secondary dysfunction of the detrusor muscle arising from nerve damage resulting in cauda equina syndrome, lower motor neuron damage or equine protozoal myeloencephalitis, equine herpesvirus type I infection, sorghum poisoning, neoplasms, or related to iatrogenic causes, predisposing to urinary incontinence (Reichelt; Lischer 2013; Scarratt et al. 1999). Due to financial limitations, additional laboratory diagnostic tests to explore causes of infectious origin could not be carried out.

Although mineral supersaturation is considered the main risk factor for the formation of uroliths in species in general, it is surprising that urolithiasis does not occur excessively in horses (Edwards; Archer 2011; Schott II et al. 2018). The considerable amount of mucus secreted by the glands in the renal pelvis and ureters contributes to the cloudy appearance of the urine, a factor that may be associated with the inhibition of the aggregation of calcium carbonate (CaCO₃) crystals and formation of uroliths (Schott II et al. 2018). The types of crystals identified in this case were within the expected range for the species (Diaz-Espineira et al. 1995, 1997), indicating only that there was an accumulation of this material, reinforcing the fact that this finding is a consequence of bladder paralysis. In another study, that described in a short case series, where crystalluria due to calcium carbonate was present in 8/13 cases of sabulous cystitis (Zakia et al. 2021).

Flaccid paralysis of the urethral sphincter and detrusor muscle promotes urinary bladder filling and overflow incontinence (Mair 2022). Incomplete emptying with stasis of the

urinary flow leads to contact between the crystalloid material and the uroepithelium, favoring precipitation. The diagnosis can be aided using transrectal ultrasonography, which configures a solid, hyperechoic, well-defined structure with acoustic shadowing. Another diagnostic option is transurethral endoscopy (Reichelt; Lischer 2013; Zakia et al. 2021), which was not available in this case.

Therapeutic possibilities for end-stage neurogenic bladder dysfunction are similar regardless of its cause. The aim is to promote bladder emptying while waiting for spontaneous recovery or at least improvement as the reflected neurological activity develops. There is no pharmacokinetic data for bethanechol chloride in horses, but doses between 0.025 to 0.075 mg/kg three times daily subcutaneously or 0.25 to 0.75 mg/kg orally two to four times daily have been recommended. Bethanechol chloride is a parasympathomimetic drug, which acts predominantly on the smooth muscle of the bladder and the gastrointestinal tract at low doses. However, it can induce cardiorespiratory side effects when administered at very high doses intramuscularly or intravenously. The occurrence of detrusor myopathy is secondary to the chronic condition of sediment permanence inside the organ and may prevent the drug from having beneficial effects (Rendle et al. 2008).

Support therapies, such as flunixin meglumine (1.1 mg/kg, IV, SID) and phenazopyridine hydrochloride (4mg/kg, PO, TID), and antibiotics have been suggested when dysuria and cystitis are identified. Repeated bladder washings were considered an effective conduct (Zakia et al. 2021), when possible, to be performed, as they require professional skills, equipment, and specific materials. Although the pathological changes that result in bladder paralysis are generally irreversible, the results obtained with five horses by Rendle et al. (2008) showed that the affected animals do not need to be euthanized and it is possible to return to athletic function if owners are committed to continued treatment.

Treatment with antibiotics and non-steroidal anti-inflammatory drugs was not performed in the present report, as the patient showed no changes in vital parameters, blood count or serum biochemistry. Diet correction and intense dedication of the owner were suggested as a means of control considering the evolution history of two months. A diet with reduced calcium concentration has been recommended to limit sediment accumulation, which is also promising as a method to decrease the urine pH and moderate the development of crystals, configuring diets that aim at the cation-anion balance (Schott II 2006). In this case, this was achieved by recommending a reduction in calcium and protein-rich foods and encouraging water intake.

The primary cause of the functional paralysis of the bladder can sometimes not be completely established, directly implicating the elaboration of effective clinical conduct, culminating in treatments that only palliatively minimize the clinical signs.

CONCLUSION

The diagnosis of idiopathic bladder paralysis resulting in a sabulous cystitis can be applied to cases of urinary incontinence in horses associated with accumulation of sabulous sediment in the bladder, when a diagnosis of the underlying cause cannot be defined. This condition is uncommon in the clinical routine and should be included in the differential diagnosis of urinary tract disorders in horses.

Conflict of interest The authors have no conflict of interest to declare.

Ethical Approval No ethical approval was required for this report.

Authors' contributions

All authors contributed equally for the conception and writing of the manuscript. All authors critically revised the manuscript and approved of the final version.

REFERENCES

Andrade LC, Cintra CR, Lima JTB, Palhares MS, Maranhão RPA, Teixeira RBC. Exame neurológico equino. Cad Tec Vet Zootec. 2021; 99:20–48.

Diaz-Espineira M, Escolar E, Bellanato J, Medina JA. Crystalline composition of equine urinary sabulous deposits. Scanning Microsc. 1995;9(4):1071–1079.

Diaz-Espiñeira M, Escolar E, Bellanato J, De La Fuente MA. Infrared and atomic spectrometry analysis of the mineral composition of a series of equine sabulous material samples and urinary calculi. Res Vet Scienc. 1997;63(1):93-95.

Duesterdieck-Zellmer KF. Equine urolithiasis. Vet Clin North Am Equine Pract. 2007;23(3):613–629.

Edwards B, Archer D. Diagnosis and treatment of urolithiasis in horses. In Pract. 2011;33(1):2–10.

Holt PE. Urinary incontinence in mature horses. Equine Vet Educ. 1996;8(1):8–12.

Keen JA, Pirie RS. Urinary incontinence associated with sabulous urolithiasis: A series of 4 cases. Equine Vet Educ. 2006;18(1):11–16.

Mair T. Urinary incontinence and urinary tract infections. Vet Clin North Am Equine Pract. 2022;38(1):73–94. Reichelt U, Lischer C. Complications associated with transurethral endoscopic- assisted electrohydraulic lithotripsy for treatment of a bladder calculus in a gelding. Equine Vet Educ. 2013;25(2):55–59. Rendle DI, Durham AE, Hughes KJ, Lloyd D, Summerhays GE. Long-term management of sabulous cystitis in five horses. Vet Rec. 2008;162(24):783–788.

Scarratt WK, Buechner-Maxwell VA, Karzenski S, Wallace MA, Robertson, JL. Urinary incontinence and incoordination in three horses associated with equine protozoal myeloencephalitis. J Equine Vet Sci. 1999;19(10):642-645.

Schott 2nd HC. Urinary incontinence and sabulous urolithiasis: chicken or egg? Equine Vet Educ. 2006;18(1):17-19.

Schott 2nd HC, Waldridge B, Bayly WM. Disorders of the urinary system. In: Reed SM, Bayly MW, Sellon DC. Equine Internal Medicine. 4. ed. St. Louis: Elsevier; 2018. p. 888-990.

Zakia LS, Gomez DE, Kenney DG, Arroyo LG. Sabulous cystitis in the horse: 13 cases (2013-2020). Can Vet J. 2021;62(7):743-750.