

Typhlocolitis by *Edwardsiella tarda* in a Cow

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ABSTRACT

Background: *Edwardsiella tarda* has been report as etiology of gastroenteritis in both human and veterinary medicine, usually associated with exposure to aquatic environments in immunocompromised individuals. The present report describes a case of typhlocolitis in a cow in the west region of Santa Catarina state, Brazil.

Case: After clinically evaluation of animal and proceeding euthanasia and realized the necropsy. Tissue samples were collected, and routinely processed for histological examination. Histopathological lesions were described as mild, moderate and severe. Tissue and swabs samples of small and large intestine were sent to standard microbiological culture processing. At necropsy, cattle presented severe dehydration and emaciation. Eye and vaginal mucosa were severely congested. The opening of the abdominal cavity revealed a great amount of greenish fluid and a large amount of fecal material, associated with diffuse severe peritonitis, evidenced by hyperemia and severe deposition of fibrin in the visceral and parietal peritoneal surface. In the serosa of the cecum, there were two points of rupture, observed in the proximal colon. The mucosa of cecum and colon were severely edematous, hyperemic, and presented diffusely distributed pinpoint round hemorrhages, as well as fibrinonecrotic material adhered to the surface. Histologically, in the mucosa of cecum and colon, moderate to severe diffuse inflammatory infiltrate of neutrophils, lymphocytes and plasma cells associated with multifocal severe necrosis were observed. Moderate diffuse fibrin deposition was evidenced in the submucosa and muscular, as well as multifocal moderate necrosis in the muscular layer. In the serous, severe diffuse inflammatory infiltrate of neutrophils associated with fibrin deposition and innumerable coccoid bacterial colonies were observed. The samples subjected to bacterial isolation showed growth of *Edwardsiella tarda*. All samples were negative for *Salmonella* spp. and *Yersinia* spp.

Discussion: The final diagnosis was established through the association of clinical history, clinical signs, gross and histopathological lesions, as well as, bacterial isolation of the etiological agent, *Edwardsiella tarda*. In this case, it is conjectured that the reservoirs which cattle had access represented the source of infection. The fact that the animal was in the immediate postpartum period may have predisposed to the development of clinical disease due to immunosuppression. In domestic animals, *Edwardsiella tarda* has been reported in swine, and as a cause of septicemia in calves. Clinically, intestinal manifestations observed in edwardsiellosis in cattle are indistinguishable from several other conditions that cause diarrhea, such as infectious, nutritional or parasitic diseases. The main differential diagnoses are salmonellosis and yersiniosis due to the similarities regarding to gross and histopathological lesions in these cases compared to cases of edwardsiellosis. Salmonellosis is characterized by grey to yellowish, fetid diarrhea in which blood and mucus are oftentimes observed. At necropsy, catarrhal, hemorrhagic or fibrinous enteritis may be evidenced. The lesions initially are seen in the ileum. However, in the chronic stages of infection, foci of necrosis and ulceration may be noted mainly in the cecum and colon. Histologically, a fibrin layer associated with necrosis and mucosal ulceration can be observed in the small intestine and initial portion of large intestine. Inflammatory infiltrate composed predominantly by neutrophils, as well as fibrin thrombi in capillaries and venules are also observed. Lesions observed are similar that described in ulcerative colitis by *E. tarda* in human patients. In conclusion, *Edwardsiella tarda* can lead to a fatal typhlocolitis in cattle, being an important differential diagnosis in cases of acute diarrhea.

Keywords: edwardsiellosis, gastroenteritis, cattle.

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INTRODUCTION

Edwardsiella tarda is a gram negative, facultative anaerobe, motile bacterium member of the *Enterobacteriaceae* family [3]. These microorganisms have been associated with many intestinal and extra-intestinal diseases in humans. *Edwardsiella tarda* has been isolated from freshwater and saltwater fish, reptiles, birds and mammals [1]. Exposure to environmental risk factors, such as aquatic environments and wildlife [5], and dietary habits, such as eating raw fish in tropical and subtropical countries may favor the infection [9].

Taking into account its zoonotic potential [6], and its importance in both human and veterinary medicine, a case of typhlocolitis by *Edwardsiella tarda* in the west region of Santa Catarina state is described. The present report describes a case of typhlocolitis in a cow in the west region of Santa Catarina state, Brazil.

A 4-year-old recently calved Holstein cow, with history of untreatable acute diarrhea was submitted for necropsy to the Veterinary Pathology Laboratory of the Instituto Federal Catarinense (IFC) - Campus Concórdia. Epidemiological and clinical information were obtained from the farmer and the field veterinarian.

The farm was located in the municipality of Peritiba, Santa Catarina state, Brazil, and the herd was composed of 42 Holstein cattle, of which 23 were lactating dairy cows. During the day the animals were kept in paddocks with pastures of oats (*Avena sativa*) and ryegrass (*Lolium multiflorum*) and supplemented with corn silage and concentrated feed twice a day. The water supplied to the animals came from two reservoirs to which animals had access at night.

CASE

After clinically evaluation of animal and proceeding euthanasia due the poor prognosis, a veterinary pathologist conducted a standard necropsy. Tissue samples were collected, fixed in 10% buffered formalin, paraffin embedded, routinely processed for histological examination, blocks were sectioned (4 µm thick) and stained with hematoxylin and eosin. Histopathological lesions were described as mild, moderate and severe. Tissue and swabs samples of small and large intestine were sent to standard microbiological culture processing, using agar McConkey¹, Deoxycholate citrate agar¹ and Yersinia Selective Agar¹.

The animal presented acute profuse diarrhea unresponsive to antibiotic therapy for three days. Treatment was performed with Florfenicol, 20 mg/kg, intramuscularly, once a day as well as electrolyte support. In this period, none of other animals in the farm showed similar clinical signs; however, the field veterinarian reported the occurrence of a similar case in another cow from the same herd, although necropsy was not conducted.

At necropsy, cattle presented severe dehydration and emaciation. Eye and vaginal mucosa were severely congested [dark red] (Figure 1). The opening of the abdominal cavity revealed a great amount of greenish fluid (8 L) and a large amount of fecal material (Figure 2), associated with diffuse severe peritonitis, evidenced by hyperemia and severe deposition of fibrin in the visceral and parietal peritoneal surface (Figure 3). In the serosa of the cecum, there were two points of rupture, measuring 2 cm and 4 cm long (Figure 4a). Also, two ruptures, measuring 6 cm



Figure 1. Typhlocolitis caused by *Edwardsiella tarda* in cattle. Ocular mucosa severely congested.



Figure 2. Typhlocolitis caused by *Edwardsiella tarda* in cattle. Large amount of greenish liquid draining (arrow) out of the abdominal cavity.



Figure 3. Typhlocolitis caused by *Edwardsiella tarda* in cattle. Serous membrane of organs of the abdominal cavity presenting hyperemia associated with deposition of large amounts of fibrin (arrow).

long each were observed in the proximal colon. The mucosa of cecum and colon were severely edematous, hyperemic, and presented diffusely distributed pinpoint round hemorrhages [petechiae] (Figure 4b), as well as fibrinonecrotic material adhered to the surface. In the abomasum, many ulcers ranging from 1 cm to 4 cm in diameter were also noted.

Histologically, in the mucosa of cecum and colon, moderate to severe diffuse inflammatory infiltrate of neutrophils, lymphocytes and plasma cells associated with multifocal severe necrosis were observed (Figure 5). Moderate diffuse fibrin deposition was evidenced in the submucosa and muscular, as well as multifocal moderate necrosis in the muscular layer. In the serosa, severe diffuse inflammatory infiltrate of neutrophils associated with fibrin deposition and innumerable coccoid bacterial colonies were observed. The samples subjected to bacterial isolation showed growth of *Edwardsiella tarda*. All samples were negative for *Salmonella* spp. and *Yersinia* spp.

DISCUSSION

The final diagnosis was established through the association of clinical history, clinical signs, gross and histopathological lesions, as well as, bacterial isolation of the etiological agent, *Edwardsiella tarda*. The bacterium is capable of surviving outside the host's organism [11] usually associated with freshwater environments [6]. The optimum growth of these bacteria is at 25°C, being favored by high temperatures and abundant organic matter. Therefore, it is important to take into account the origin of drinking water offered to the herd. In this case, it is conjectured that the reservoirs which cattle had access represented the

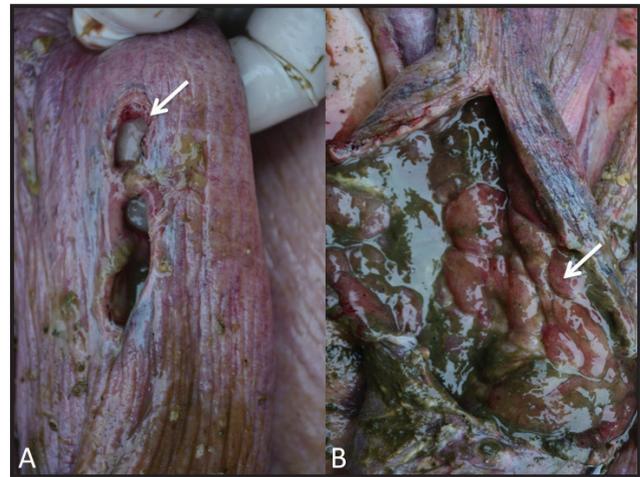


Figure 4. Typhlocolitis caused by *Edwardsiella tarda* in cattle. A- Cecum: Two points of rupture (arrow) in the serosa allowing the passage of fecal material into the abdominal cavity. B- Colon: The mucosa is severely edematous, hyperemic and presenting diffusely distributed petechiae (arrow).

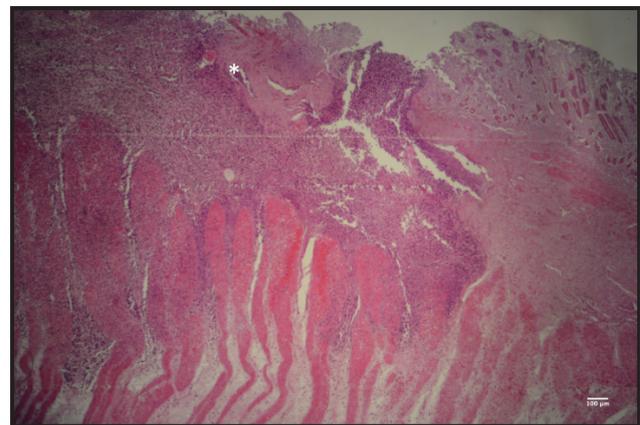


Figure 5. Typhlocolitis caused by *Edwardsiella tarda* in cattle. Cecum. In the mucosa, submucosa and muscular; severe focally extensive necrosis (*) associated with cellular debris, severe inflammatory infiltrate of neutrophils and bacterial colonies are observed [HE, obj.10x].

source of infection. The fact that the animal was in the immediate postpartum period may have predisposed to the development of clinical disease due to immunosuppression [6,10,16].

Human beings can be considered accidental hosts of this microorganism [6]. In humans *Edwardsiella tarda* has been isolated in cases of gastroenteritis [6,2], myonecrosis, wounds, bacteremia, abscesses, cholecystitis [15,19], urosepsis [16] and endocarditis [7]. Elderly and children are the most susceptible groups to develop the disease. *Edwardsiella tarda* infections can be localized or systemic, and can induce intestinal, mainly gastroenteritis [6,16,19] and extraintestinal disease, which are frequent [9,15,19].

The knowledge about the pathogenicity of *Edwardsiella tarda* in domestic animals is very limited.

However due to its wide geographic distribution and host range, the bacteria can cause major economic losses to fish farming [13]. Besides infecting many species of fish, birds, reptiles, amphibians and aquatic invertebrates, these species may act as asymptomatic carriers, which contributes to the spread of the microorganism [11]. In domestic animals, *Edwardsiella tarda* has been reported in swine [12], and as a cause of septicemia in calves [8].

Clinically, intestinal manifestations observed in edwardsiellosis in cattle are indistinguishable from several other conditions that cause diarrhea, such as infectious, nutritional or parasitic diseases. As a result, clinical diagnosis is difficult and unlikely, and in order to reach the final diagnosis, histopathological examination, as well as bacterial isolation is indispensable. The main differential diagnoses are salmonellosis and yersiniosis due to the similarities regarding to gross and histopathological lesions in these cases compared to cases of edwardsiellosis.

Salmonellosis is characterized by grey to yellowish, fetid diarrhea in which blood and mucus are oftentimes observed. At necropsy, catarrhal, hemorrhagic or fibrinous enteritis may be evidenced [9,18]. The lesions initially are seen in the ileum [18]. However, in the chronic stages of infection, foci of necrosis and ulceration may be noted mainly in the cecum and colon [4]. Histologically, a fibrin layer associated with necrosis and mucosal ulceration can be observed in the small intestine and initial portion of large intestine. Inflammatory infiltrate composed predominantly by neutrophils, as well as fibrin thrombi in capillaries and venules are also observed [18].

Enteritis caused by *Yersinia* spp. can be mild or severe [4] and are presented as profuse, fetid diarrhea, which may contain blood or fibrin [14]. In its acute presentation, yersiniosis is characterized by a

fibrinous or fibrinohemorrhagic enteritis [14,18], markedly affecting the small intestine [14]. Hemorrhagic foci are oftentimes observed in the intestinal serosa. In subacute or chronic manifestations, the lesions are milder, such as congestion, edema, focal hemorrhages, bowel wall pleating, as well as and mild ulceration of the large intestine [18]. Microscopically, gram-negative cocobacilli colonies are evidenced in the mucosa and submucosa [18], associated with severe inflammatory infiltrate, predominantly neutrophilic [4,14,18]. Additionally, microabscesses and granulomas, surrounded by giant cells, are distributed in the intestinal lamina propria and crypts [4,18].

The intense mixed inflammatory infiltrate observed in the case reported is similar to that described in a case of ulcerative colitis by *Edwardsiella tarda* in human patients [2]. Although, the presence of microabscesses is described in edwardsiellosis in humans [2,17], they were not observed in the present case.

In conclusion, *Edwardsiella tarda* can lead to a fatal typhlocolitis in cattle, being an important differential diagnosis of salmonellosis and yersiniosis in cases of acute diarrhea. The report of enteric disease in the bovine species caused by *Edwardsiella tarda* is important, since humans are susceptible to becoming infected through consumption of contaminated animal products, as well as, the disease produced is oftentimes fatal.

MANUFACTURER

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REFERENCES

- 1 Arya A.V., Rostom A., Dong W.F. & Flynn A.N. 2011. Crohn's Disease Exacerbation Induced by *Edwardsiella tarda* Gastroenteritis. *Gastroenterology*. 5(3): 623-627.
- 2 Engel J.J. & Martin T.L. 2006. *Edwardsiella tarda* as a cause of postdysenteric ulcerative colitis. *International Journal Colarectal Disease*. 21(2): 184-185.
- 3 Ewing W.H., McWhorter A.C., Escobar M.R. & Lubin A.H. 1965. *Edwardsiella*, a new genus of enterobacteriaceae based on a new species, *E. tarda*. *International Journal of Systematic and Evolutionary Microbiology*. 15(1): 33-38.
- 4 Gelberg H.B. 2013. Sistema Alimentar, Peritônio, Omento Mesentérico e Cavidade Peritonial. In: McGavin M.D. & Zachary F.M. (Eds). *Bases da Patologia em Veterinária*. 2.ed. Rio de Janeiro: Elsevier, pp.378-382.

- 5 Janda J.M. & Abbott S.L. 1993. Infections Associated with the Genus *Edwardsiella*: the role of *Edwardsiella tarda* in human disease. *Clinical Infectious Diseases*. 17(4): 742-748.
- 6 Leung K.Y., Siame B.A., Tenkink B.J., Noort R.J. & Mok Y.K. 2012. *Edwardsiella tarda* – Virulence mechanisms of an emerging gastroenteritis pathogen. *Microbes and Infection*. 14(1): 26-34.
- 7 Litton K.M. & Rogers B.A. 2016. *Edwardsiella tarda* Endocarditis Confirmed by Indium-111 White Blood Cell Scan: An Unusual Pathogen and Diagnostic Modality. *Case Reports in Infectious Diseases*. 2016: 1-3.
- 8 Magalhães H., Freitas M.A., Santos J.A. & Costa C.H.C. 1984. Septicemia por *Edwardsiella tarda*, em bezerro. *Pesquisa Agropecuária Brasileira*. 19(3): 367-370.
- 9 Hirai Y., Ashata-Tago S., Ainoda Y., Fujita T. & Kikuchi K. 2015. *Edwardsiella tarda* bacteremia. A rare but fatal water – and foodborne infection: Review of the literature and clinical cases from a single centre. *The Canadian Journal of Infectious Diseases & Medical Microbiology*. 26(6): 313-318.
- 10 Mikamo H., Ninomiya M., Sawamura H. & Tamaya T. 2003. Puerperal intrauterine infection caused by *Edwardsiella tarda*. *Journal of Infection and Chemotherapy*. 9(4): 341-343.
- 11 Mohanti B.R. & Sahoo P.K. 2007. Edwardsiellosis in fish: a brief review. *Journal of biosciences*. 32(7): 1331-1344.
- 12 Owens D.R., Nelson S.L. & Addinon J.B. 1974. Isolation of *Edwardsiella tarda* from Swine. *Applied Microbiology*. 27(4): 703-705.
- 13 Park S.B., Aokil T. & Jung T.S. 2012. Pathogenesis of and strategies for preventing *Edwardsiella tarda* infection in fish. *Veterinary Research*. 43(1): 67.
- 14 Riet-Correa F., Schild A.L., Méndez M.d.C. & Lemos R.A.A. 2007. *Doenças de Ruminantes e Equídeos*. 3.ed. Santa Maria: Pallotti, 998p.
- 15 Slaven E.M., Lopez F.A., Hart S.M. & Sanders C.V. 2001. Myonecrosis Caused by *Edwardsiella tarda*: A Case Report and Case Series of Extraintestinal *E. tarda* Infections. *Clinical Infectious Diseases*. 32(10): 1430-1433.
- 16 Tamada T., Koganemaru H., Mastsumoto K. & Hitomi S. 2009. Urosepsis caused by *Edwardsiella tarda* *Journal of infection and chemotherapy*. 15(3): 191-194.
- 17 Thune R.L., Stanley L.A. & Cooper R.K. 1993. Pathogenesis of gram-negative bacterial infections in warm water fish. *Annual Review of Fish Diseases*. 3: 37-68.
- 18 Uzal F.A., Plattiner B.L. & Hostetter J.M. 2015. Alimentary system. In: Maxie M.G. (Ed.), *Jubb, Kennedy, and Palmer's Pathology of Domestic Animals*. v.2. 6th edn. St. Louis: Elsevier, pp.167-177.
- 19 Wang I.K., Kuo H.L., Chen Y.M., Lin C.L., Chang H.Y., Chuang F.R. & Lee M.H. 2005. Extraintestinal manifestations of *Edwardsiella tarda* infection. *International Journal of Clinical Practice*. 59(8): 917-921.