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Author(s): Laurie Hess, Tracy Bartick, Heidi Hoefler

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Case Report

Clostridium tertium Infection in a Moluccan Cockatoo (*Cacatua moluccensis*) with Megacolon

Laurie Hess, DVM, Tracy Bartick, DVM, and Heidi Hoefler, DVM

Abstract: A 4-year-old Moluccan cockatoo (*Cacatua moluccensis*) was examined because of lethargy and a necrotic wound on the lateral base of the tail. Over the previous 2 years, the bird had chronic diarrhea and was treated with long-term antibiotics. A contrast radiographic study demonstrated slow gastrointestinal transit time and a large filling defect in the caudal abdomen. *Clostridium tertium*, a bacterium rarely found in psittacine birds, was isolated on bacterial culture of a fecal sample. Despite wound debridement and parenteral antibiotic treatment, the bird died. At necropsy, the colon was severely dilated. Histopathologic examination revealed diffuse, severe lymphoplasmacytic inflammation of the colon and massive ulceration and bacterial colonization of the pygostyle.

Key words: colon, megacolon, *Clostridium tertium*, psittacine birds, cockatoo, septicemia, avian

Introduction

Clostridia are part of the autochthonous enteric flora in raptors and also can be found in birds with well-developed ceca (Galliformes and Anseriformes). However, in birds that lack ceca, including Psittaciformes, clostridia are not usually present in the intestines.^{1–4} After colonizing in the gastrointestinal tract, clostridia can produce potent bacterial exotoxins that cause gastrointestinal signs.¹ Clostridial enteritis is well documented in domestic poultry, waterfowl, and gamebirds but is less frequently reported in psittacine birds.^{1–7} Diseases of the colon are uncommon in birds, and none have been reported in association with clostridial infection. Undocumented colonic lesions have been seen infrequently in psittacine birds with *Clostridium perfringens* infection (B. Ritchie, DVM, PhD, personal communication, 1997).

One rarely reported species of *Clostridium* is *Clostridium tertium*, a non-toxin-producing, predominantly anaerobic, partially aerotolerant bacterium.^{8–13} *Clostridium tertium* has been isolated from the intestinal contents of broiler chicks,¹⁴ but it has not been cultured from the cloaca of clinically normal psittacine birds.¹⁵ In humans, *C. tertium* causes

septicemia and abscess formation, particularly in neutropenic cancer patients receiving chemotherapy.^{8,10–13,16–19}

In this report we describe a Moluccan cockatoo (*Cacatua moluccensis*) with megacolon and chronic diarrhea. *Clostridium tertium* was isolated on bacterial culture of a fecal sample.

Case Report

A 4-year-old Moluccan cockatoo was examined because of a 1-day history of lethargy and an ulcerated wound at the base of the tail. There was no known history of trauma or self-mutilation. Over the previous 2 years, the bird had been treated intermittently for chronic diarrhea with tylosin (dosage unknown) (Tylan, Elanco, Indianapolis, IN, USA).

On physical examination, a 3 × 3-cm, hemorrhagic, necrotic wound extended from the bird's left flank to its left lateral pygostyle. The left flank was swollen and palpably firm. The bird appeared weak, pale, and slightly thin. It was tachypneic at rest and became dyspneic with handling. The bird had profuse, watery, green, fetid diarrhea.

A blood sample was collected for a complete blood count (CBC), plasma biochemical analysis, plasma protein electrophoresis, chlamydial elementary body agglutination testing, and measurement of

From the Departments of Medicine (Hess, Hoefler) and Pathology (Bartick), The Bobst Hospital of The Animal Medical Center, 510 E. 62nd Street, New York, NY 10021, USA.

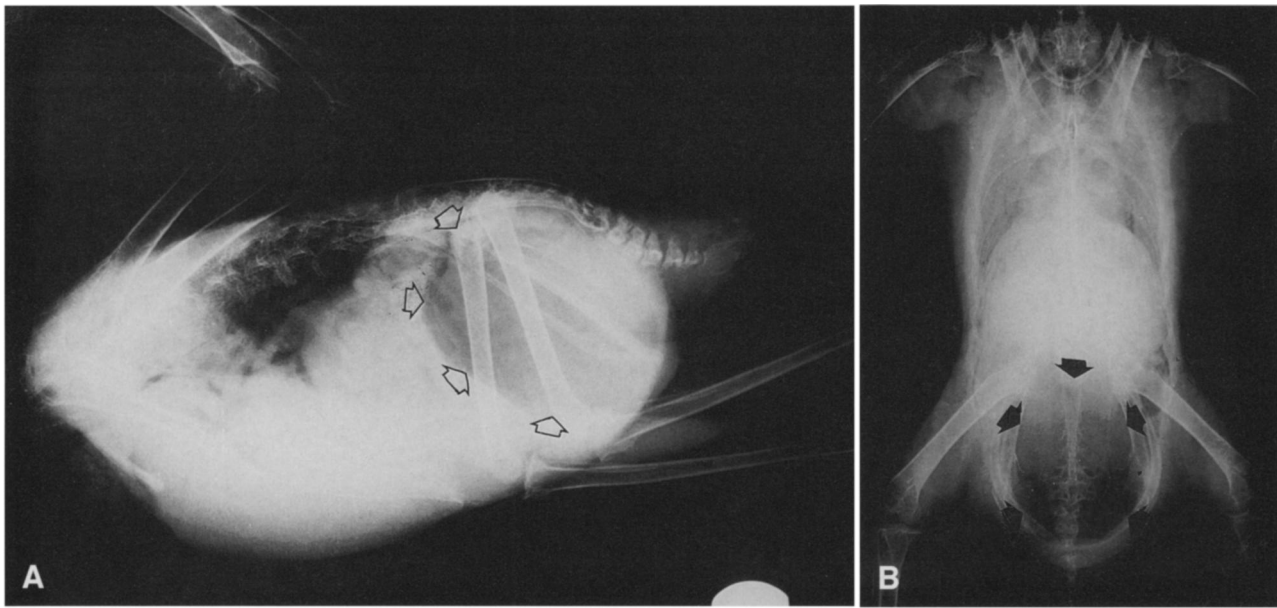


Figure 1. (A) Right lateral survey radiograph of a Moluccan cockatoo with chronic diarrhea. A large, gas-filled structure (arrows) is visible in the caudal abdomen. Contrast studies revealed this structure to be a dilated colon. (B) Ventrodorsal survey radiograph of bird described in Figure 1A. Arrows point to the dilated colon.

the packed cell volume (PCV) and blood lead concentration. A fecal sample was submitted for Gram's, trichrome, and acid-fast staining and anaerobic and aerobic bacterial culture and sensitivity testing. Survey radiographs of the body were taken, and a barium gastrointestinal study was performed.

Supportive treatment was begun with lactated Ringer's solution (15 ml subcutaneously [SC] q12h) and iron dextran (0.1 ml intramuscularly [IM] once). Enrofloxacin (Baytril, Bayer, Shawnee Mission, KS, USA) (15 mg/kg IM q12h), calcium ethylenediaminetetraacetic acid (EDTA) (Calcium disodium versenate, 3M Pharmaceuticals, Northridge, CA, USA) (30 mg/kg SC q12h), and metronidazole (Flagyl, Rhone-Poulenc Rorer, Jose Ma Rico, Mexico) (10 mg/kg per os [PO] q12h) were also administered. The tail wound was cleaned, debrided, and bandaged with Biodres (DVM Pharmaceuticals, Miami, Fla, USA).

The CBC results revealed that the bird had profound, nonregenerative anemia (PCV 10%, reference range 38–48%²⁰) and monocytosis (42%, reference range 0–1%²⁰). Results of the plasma protein electrophoresis revealed slight hypoalbuminemia (1.27 g/dl, reference range 1.8–3.1 g/dl²⁰) and mild polyclonal gammopathy (0.90 g/dl, reference range 0.21–0.65 g/dl²⁰). Results of all other blood tests were within reference ranges. Scattered, safety pin-shaped clostridial bacteria were seen on the Gram's stain of the feces. Anaerobic fecal culture yielded *C. tertium*, but a sensitivity test was not done. On

survey radiographs, a dilated proventriculus and a large, circular to oval, gas-filled, caudal abdominal structure were visible (Figs. 1A, B). Results of the contrast study revealed a filling defect in the caudal abdomen and an extremely slow gastrointestinal transit time, with barium remaining in the intestines 20 hours after it was administered.

Treatment with fluids, calcium EDTA, and antibiotics was continued for 4 days. On daily rechecks, the PCV increased only to 12–13%. The cockatoo was more alert, but the diarrhea continued. The owner elected to continue therapy at home with ciprofloxacin (Ciprofloxacin hydrochloride, Bayer, West Haven, CT, USA) (15 mg/kg PO q12h) and daily bandage changes.

Three days after discharge, the bird became profoundly weak and recumbent and was returned to the hospital. On examination, it was stuporous, dehydrated, bradypneic, laterally recumbent, and seizing. Malodorous, watery, green diarrhea was present. A blood sample was collected to measure the PCV and blood glucose concentration. A catheter was placed in the right medial metatarsal vein, and lactated Ringer's solution with 5% dextrose was administered (16 ml intravenously [IV] q8h). Fluoroquinolone-associated seizure activity was suspected; thus, ciprofloxacin treatment was stopped. The bird was treated with piperacillin (Pipracil, Lederle, Carolina, Puerto Rico) (100 mg/kg IV q8h), and chloramphenicol (Chloromycetin sodium succinate, Parke-Davis, Morris Plains, NJ, USA) (50 mg/kg

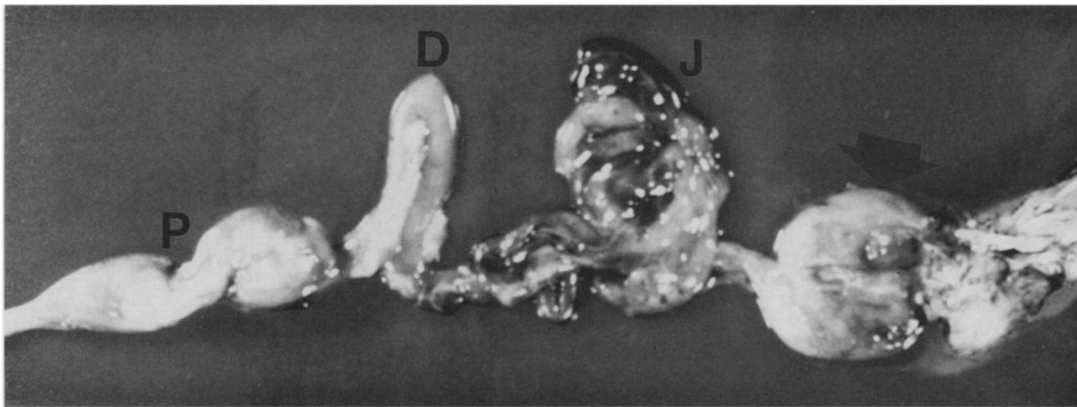


Figure 2. Alimentary tract from a Moluccan cockatoo with chronic diarrhea. The dilated colon is indicated by an arrow. P = proventriculus/ventriculus; D = duodenum/pancreas; J = jejunum.

IM q12h). Diazepam (0.5 mg/kg IV once) was administered to control seizure activity.

The bird remained anemic, with a PCV of 14%. Blood glucose concentration was within the reference range. Despite treatment, the bird remained semicomatose and laterally recumbent. The bird died approximately 6 hours after it was readmitted to the hospital.

Results of necropsy revealed a massively dilated, thin-walled, fluid-filled colon, approximately 4 cm

in diameter (Fig. 2). An irregular, 3 × 3-cm wound with necrotic edges was present at the left lateral pygostyle. Several yellow foci of necrosis, 0.5 cm in diameter, were observed in the pectoral muscles.

Histopathologic examination of the dilated colon revealed diffuse, severe lymphoplasmacytic inflammation (Fig. 3). Ulcerative, necrotizing dermatitis of the pygostyle was present, with granulation tissue and bacterial colonization. Other findings included necrosis of the granular cell layer of the cerebellum,

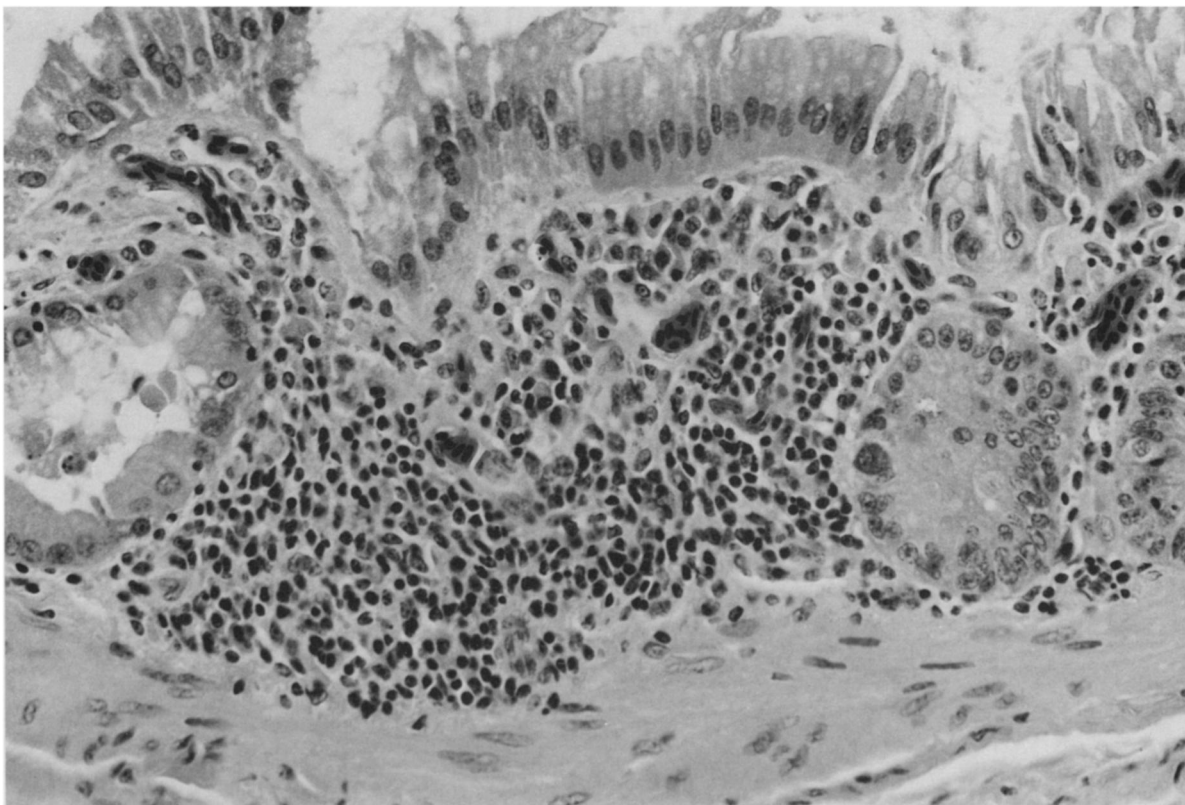


Figure 3. Photomicrograph of the colon from the Moluccan cockatoo described in Figure 2. The lamina propria is expanded by a dense population of lymphocytes and plasma cells. H & E stain. ×200.

extensive cardiac myocyte degeneration, heterophilic splenitis, and pectoral muscle myositis. Sepsis secondary to the severe pygostyle infection was postulated as the cause of death.

Discussion

Clostridia are gram-positive, spore-forming bacterial rods.^{8,9,21} Infectious spores are highly resistant and can persist for years in the environment.⁸ Clostridia can colonize the gastrointestinal tract and cause severe tissue damage by producing bacterial exotoxins.¹ Factors that promote intestinal colonization of clostridia include reduced gastrointestinal motility from chronic antibiotic use, insufficient dietary fiber, and viral infection, most notably by reovirus.¹

Clinical signs of clostridial infection vary depending on the infecting species of clostridia and the affected avian species. Necrotic enteritis, caused by *C. perfringens*, can cause anorexia, depression, diarrhea, and death in chickens, turkeys, and, less commonly, quail.^{1,22} This disease has been associated with dietary changes and damage to intestinal mucosa, especially by coccidia.²² Necrotic mucosa and pseudomembrane formation, the characteristic lesions of necrotic enteritis, are usually confined to the small intestine and cecum.^{1,22}

Ulcerative enteritis, or quail disease, is an acute intestinal infection caused by *Clostridium colinum*. It occurs in quail, grouse, turkeys, chickens, partridges, pheasants, pigeons, robins, and, rarely, psittacine birds.^{1,5,6,22-27} Affected birds may exhibit diarrhea, weakness, weight loss, and sudden death. Characteristic lesions include focal, yellow, mucosal ulcerations with hemorrhagic borders in the small intestine and cecum and necrosis of the liver and spleen.^{1,5,6,22-27}

Gangrenous dermatitis, or malignant edema, is caused by colonization of damaged skin by *Clostridium septicum*, *Clostridium novyi*, or *C. perfringens*. Characteristic lesions include feather loss, edema, subcutaneous emphysema, and painful, blue-black discoloration of skin and muscle. Affected birds usually become toxemic and die acutely.^{7,28,29}

Botulism, or limberneck, occurs in waterfowl after ingesting food contaminated with toxins produced by *Clostridium botulinum*. The toxins from this organism, which rarely directly colonizes the gastrointestinal tract, ascend through enteric nerves to the spinal cord. There they block production of acetylcholine, leading to generalized flaccid paralysis and death from respiratory paralysis. Often, no gross lesions are seen at necropsy.^{1,30,31}

Tetanus, caused by infection with *Clostridium tetani*, has been sporadically documented in birds.¹ However, the validity of these reports is now questionable because birds have since been found to be highly resistant to *C. tetani* infection.¹

Clostridium tertium is rarely isolated in birds and has only been reported in poultry.¹⁴ In the cockatoo we describe, *C. tertium* was cultured from a fecal sample. *Clostridium* sp. are not normal enteric flora in psittacine birds because these birds lack ceca.^{1,3,4} However, for 2 years this cockatoo was treated repeatedly with tylosin at an unknown dosage. Chronic antibiotic use has been implicated as a cause of clostridial-associated diarrhea in humans.^{8,13,18,32} The repeated treatment with tylosin possibly selected for *C. tertium* in the cockatoo's gastrointestinal tract, which then led to chronic diarrhea.

The development of *C. tertium* septicemia in humans during antibiotic treatment emphasizes the importance of antibiotic sensitivity testing in patients infected with *C. tertium*. This organism has shown resistance to many commonly used antibiotics, including the beta-lactams and clindamycin.^{11,12,16} Unfortunately, sensitivity testing was not done in this case.

A possible association has been made by B. Ritchie, DVM, PhD (personal communication, 1997) between clostridial infection in birds and close contact with dogs. The cockatoo we describe was kept in contact with dogs. The canine gastrointestinal tract, which normally contains *Clostridium* sp.,³² may serve as a source for fecal-oral transmission of clostridia to closely associated birds.

Clostridium tertium infection often develops in immunocompromised, neutropenic humans.^{12,13,16,19} Although this cockatoo was not heteropenic, underlying viral infection is impossible to eliminate as a cause of immunocompromise because viral testing was not done. This bird did have profound, nonregenerative anemia and severe monocytosis. The anemia may have been caused by chronic clostridial infection, whereas the monocytosis may have resulted from inflammation and necrosis of the pygostyle.

Interestingly, the gross and histologic alimentary lesions were confined to the colon, with the small intestine appearing normal. Colonic disease secondary to clostridial infection has been described in rabbits, cats, and dogs, as well as in humans infected with *C. tertium* and *Clostridium difficile*.^{8,16,21,33-35} Necrotizing enterocolitis has been reported in ostriches (*Struthio carmelus*) infected by *C. difficile*.³⁶ However, pathologic lesions of the colon are not well documented in birds, and no references to megacolon could be found. According to

R. Schmidt, DVM, PhD (personal communication, 1997), megacolon has been seen in birds that were obstructed secondary to colonic neoplasia and in those with proventricular dilatation and colonic ganglioneuritis. However, the colonic ganglia in the cockatoo appeared normal. Birds affected with avian viral serositis also have had megacolon (B. Ritchie, DVM, PhD, personal communication, 1997).

When megacolon developed in the course of this bird's disease is unclear. The bird may have had megacolon and subsequently developed diarrhea from colonic stasis and clostridial overgrowth. However, megacolon could have developed from tenesmus caused by the chronic, clostridial-induced diarrhea. Lymphoplasmacytic inflammation, but no ulceration, was present in this bird's severely dilated colon. Although necrotic and ulcerative enteritis caused by clostridia are characterized by small intestinal and cecal ulcers, neither condition has been previously associated with colonic damage.^{1,22,23,25,26}

Possibly, the cockatoo mutilated its pygostyle because of discomfort from the dilated colon. The ulcerated, necrotic wound on the pygostyle was not cultured. However, the rod-shaped bacteria that colonized the wound were morphologically different from the safety pin-shaped, spore-forming clostridia isolated from the gastrointestinal tract.^{6,8} Bacterial invasion of the ulcerated pygostyle, with necrosis and inflammation of the spleen, cerebellum, and heart, suggests that the cockatoo was septic. Sepsis may have led to seizures and subsequent death. The possible selection of antibiotic-resistant clostridia and sepsis secondary to wound contamination emphasize the importance of routine diagnostic tests, including Gram's stains and bacterial culture and sensitivity testing of appropriate samples.

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