

Although very little is known of the specific conditions necessary for the development of overt disease, in ruminants the encephalitic form of listeriosis can result when the organism enters through minor lesions in the oral cavity, nasal cavity, or conjunctiva and migrates along the peripheral nerves to the central nervous system.^{15,17} Necrosis then occurs in the brain stem, often beginning in the trigeminal ganglion.¹⁵ When mice were injected with *Listeria monocytogenes* in the sciatic nerve, the bacterium actively migrated along the nerve axon, resulting in flaccid paralysis within 7-14 days postinoculation.¹⁶ The broilers involved in these outbreaks had been debeaked and had received a subcutaneous vaccination in the neck area several weeks before the onset of the first symptoms, suggesting possible routes of entry for the organism. The only 2 reported outbreaks of listeriosis in broilers in California have involved breeder replacement birds, whereas infection in meat-type chickens, which are far more common in this region but are not debeaked or vaccinated subcutaneously at 7-10 days of age, has not been reported.

Sources and manufacturers

- a. Salisbury Laboratories, Charles City, IA.
- b. BBL Microbiological Systems, Cockeysville, MD.
- c. Oxoid, USA, Columbia, MD.

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Campylobacter jejuni isolated from ratites

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Ratites (ostrich, emu, and rhea) are increasingly popular as alternative livestock in the United States. These birds are valuable, and public interest in them is increasing. Many diagnostic laboratories, therefore, may become involved with ratite disease diagnosis. This report describes clinical, histologic, and bacteriologic findings in a rhea from which *Campylobacter jejuni* was isolated and the subsequent isolation of the bacterium from yolk sacs of ostrich chicks.

A 3-month-old rhea chick was submitted August 30, 1990,

to the Texas Veterinary Medical Diagnostic Laboratory, Amarillo, Texas. Seven rheas in a flock of 14 died over a 1-week period. The birds appeared emaciated before death. Rheas of all ages were housed in an outdoor enclosure adjacent to several ostrich pens.

Necropsy of the rhea chick revealed multifocal, light tan 1-2-mm-diameter foci (some with darkened centers) at 1 pole of the left lobule of the liver. The musculature was pale and mildly emaciated. Other significant gross lesions were not observed.

Heart, lung, ventriculus, proventriculus, small intestine, colon, liver, kidney, skeletal muscle, and multiple sections of brain were fixed in 10% buffered formalin and examined

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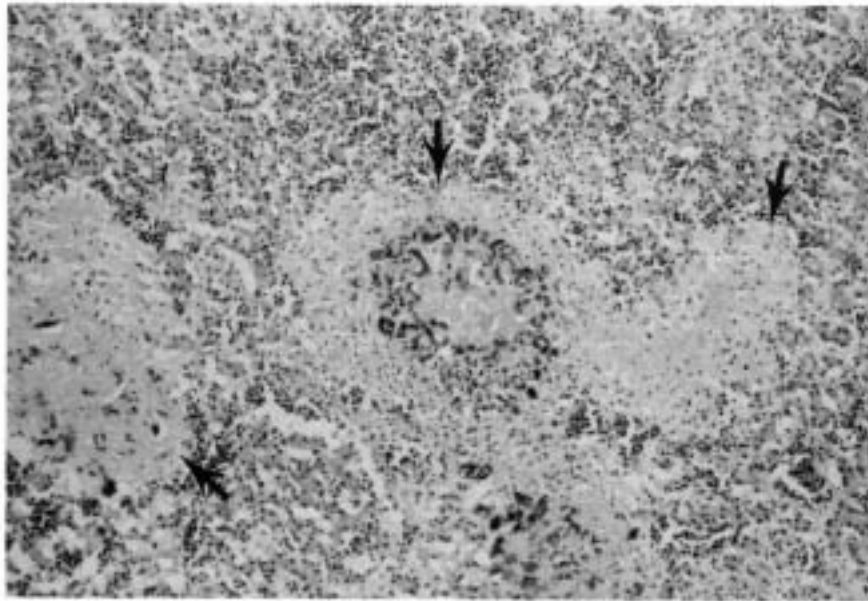


Figure 1. Foci of hepatic necrosis and granuloma formation in a rhea chick. 20x, HE stain.

microscopically. Histologic findings in the liver were multifocal necrotizing granulomatous hepatitis (Fig. 1). Multiple fibrinonecrotic hepatic foci were surrounded by a mantle of histiocytes with multinucleated giant cells and occasional granulocytes (Fig. 2). Groups of small spiral-shaped silver stain-positive (Warthin-Starry) bile stain (Halls)-negative bacteria were present in the tissue adjacent to granulomas and between hepatocytes near bile canaliculi (Fig. 3). Other tissues were not remarkable.

Routine aerobic bacteriologic culture of the liver was negative. Anaerobic incubation on 5% bovine blood agar at 37 C for 48 hours yielded a pure culture of large numbers of tiny gray bacterial colonies. The bacteria were gram-negative curved or seagull-shaped rods with morphology typical of

Campylobacter sp. The bacterial isolate was oxidase and catalase positive, grew in the presence of 1% glycine, and did not grow in medium containing 3.5% NaCl. It produced hydrogen sulfide on lead acetate strips incubated over a cysteine-containing medium, did not produce hydrogen sulfide (H_2S) in TSI agar, did not grow in a minimal medium,⁷ was sensitive to nalidixic acid (30 μ g), was resistant to cephalothin (30 μ g), and did not hydrolyze hippurate by the rapid method of Hwang and Ederer.⁴ More abundant growth was obtained by incubation in a microaerophilic environment using gas-generating envelopes.^a The isolate grew well at 42 C but not at 25 C. The isolate resembled *C. jejuni*, based on biochemical test results.

It is difficult to differentiate *C. coli* from *C. jejuni* in the

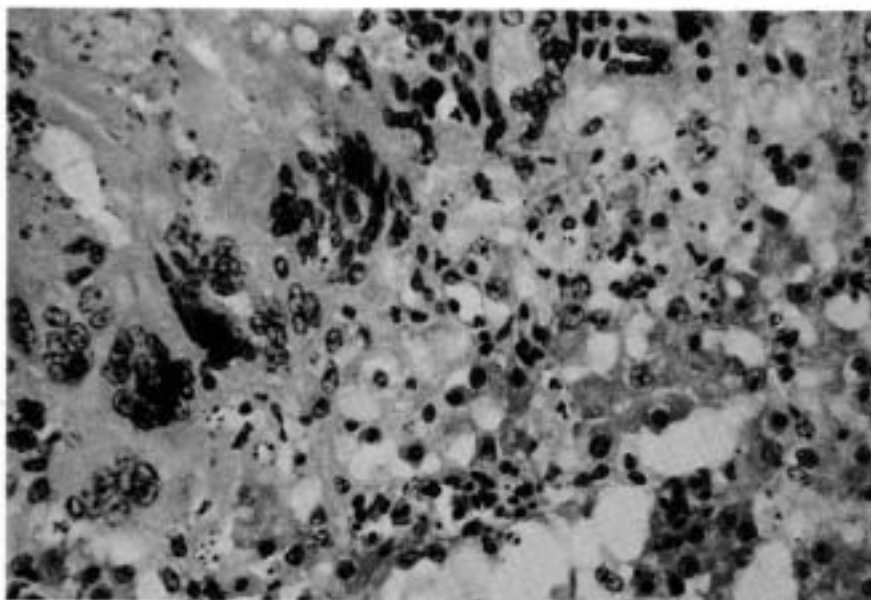


Figure 2. Margin of granuloma with multinucleated giant cells in liver of a rhea chick. HE stain.

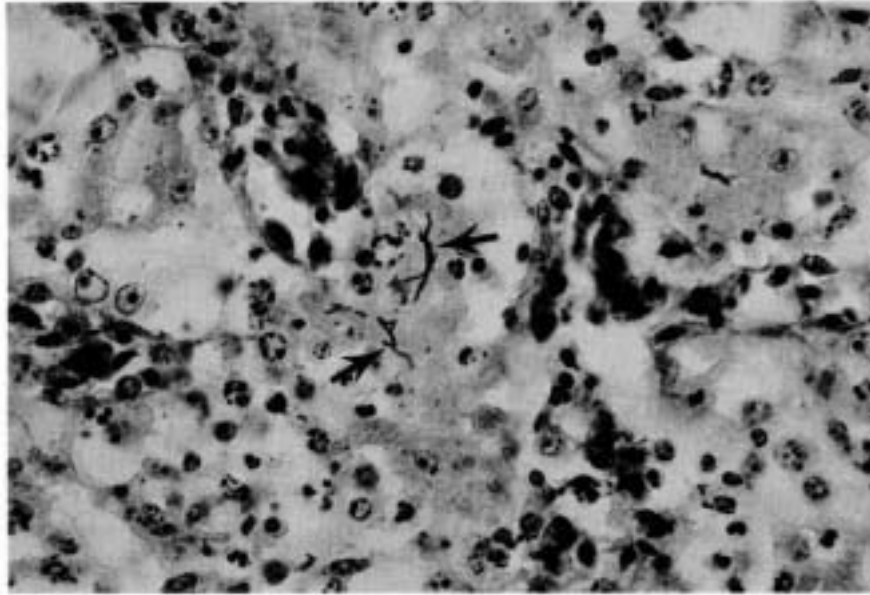


Figure 3. Spiral-shaped bacteria between hepatocytes (arrow heads) in a rhea chick. Warthin-Starry stain.

clinical laboratory. Species differentiation is currently based on only a few biochemical tests: hippurate hydrolysis, growth in a minimal medium, and production of H₂S at the junction of the slant and butt of a TSI slant.^{2,7} This isolate did not hydrolyze hippurate, would not grow in minimal medium, and did not produce H₂S on TSI slants, therefore it was classified as *C. jejuni*. There are reports of hippurate-negative strains of *C. jejuni*, and some have been isolated from poultry.^{3,10}

Three weeks after the initial isolation of *C. jejuni* from the rhea, ostrich chicks on the same premises began dying. An approximately 10-14-day-old ostrich chick was presented for necropsy. Examination revealed an abnormal (8 cm in diameter) intra-abdominal yolk sac. The wall of the sac was yellow with a red mottled surface pattern. Mesenteric membranes were adhered to the surface of the yolk sac. The sac contained a yellow inspissated, pasty, fetid material. *Campylobacter jejuni* was isolated in pure culture from the yolk sac. The next week, 2 more ostrich yolk sac swabs were submitted from the same farm for bacterial culture, and *C. jejuni* was isolated in pure culture.

The hepatitis observed microscopically in the rhea chick is similar to avian vibronic hepatitis first described in the 1950s as a contagious bacterial disease of chickens.^{1,5,8} This chronic disease of chickens has a low mortality and high morbidity with striking liver lesions, described as small yellow necrotic foci disseminated throughout the liver. Textbooks list the etiologic agent of avian vibronic hepatitis as *C. jejuni*.^{6,9} The incidence of this disease has been very low since the late 1970s.⁶

Since the initial isolations of *C. jejuni*, hippurate-positive strains of *C. jejuni* have been isolated from the liver and yolk sac of 8 additional ratite cases. The isolation of *C. jejuni* in the absence of other etiologic agents warrants additional study to determine if a causal relationship exists.

Sources and manufacturers

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