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Erysipelothrix Rhusiopathiae Septicemia in a Laughing Kookaburra (*Dacelo Novaeguineae*)

T. Opriessnig, R. K. Vance and P. G. Halbur

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medial necrosis as well as disruption of the elastic network and collagen fibers.[3] Other reported causes of dissecting hematomas with cystic medial necrosis of arterial trunks in humans include defects in type III collagen as seen in Ehlers–Danlos type IV syndrome[3,8] and hypertension,[7,13] but neither of these have yet been reported in dogs.

It cannot be concluded based on these 2 cases of canine spontaneous aortic dissecting hematoma whether the condition has a common underlying genetic basis with that described in Marfan syndrome, but several aspects of the cases including gross and histologic lesions indicative of elastin dysplasia are similar. Although the number of cases described in this report is low, they could represent a hereditary condition predisposing to arterial cystic medial necrosis in dogs with Border Collie parentage.

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T. Opriessnig, R. K. Vance, P. G. Halbur[1]

Abstract. *Erysipelothrix rhusiopathiae* (*E. rhusiopathiae*) septicemia was demonstrated in a captive Laughing kookaburra (*Dacelo novaeguineae*). The bird died after a 2-week period of weakness and weight loss. At necropsy, the bird was emaciated and had reddened and wet lungs. Microscopic lesions were limited to hepatic and pulmonary congestion with focal thrombosis. *Erysipelothrix rhusiopathiae* was isolated by routine bacterial culture from several organs. Further characterization of the isolate by pulsed-field gel electrophoresis indicated that the isolate has a new genotype pattern 3A(III), which is 91.7% homologous to an *E. rhusiopathiae* that was isolated from a pig in 2001 and 88% homologous to an isolate recovered in 2000 from a turkey with septicemia. This is the first report of *E. rhusiopathiae*–induced septicemia in a kookaburra.

Key words: *Erysipelothrix rhusiopathiae*; kookaburra; septicemia.

The gram-positive bacterium *Erysipelothrix rhusiopathiae* (*E. rhusiopathiae*) is known to infect swine, sheep, fish, reptiles, birds, and humans.[17] *Erysipelothrix rhusiopathiae* infection is increasingly being reported in fish-eating mammals such as whales and dolphins.[9,16,15]

In animals, the disease caused by *E. rhusiopathiae* is called erysipelas, whereas the infection of humans with *E. rhusiopathiae* is called erysipeloid.[17] Twenty-eight serotypes have been described, and in pigs, which are the primary host for E. rhusiopathiae, the most prevalent serotypes are 1a and 1b. Serotypes 1a and 1b are associated with cases of acute septicemia with or without evidence of widespread hyaline thrombosis or ischemic necrosis of perivascular tissues and skin lesions. Serotype 2 is associated with the chronic form

From the Department of Veterinary Diagnostic and Production Animal Medicine, Iowa State University, Ames, IA 50011 (Opriessnig, Halbur), and the Hartland Animal Hospital, Horse Cave, KY 42749 (Vance).

[1]Corresponding Author: P. G. Halbur, Department of Veterinary Diagnostic and Production Animal Medicine, College of Veterinary Medicine, Iowa State University, Ames, IA 50011.

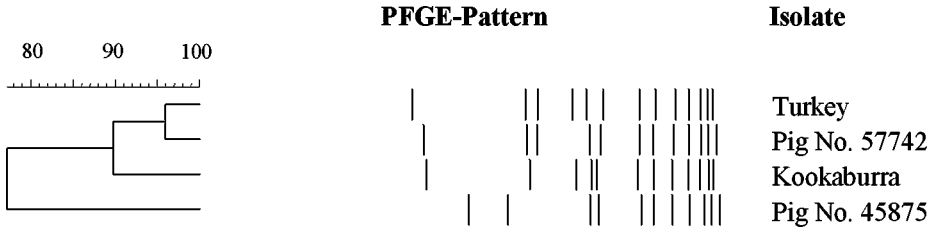

Figure 1. Genetic relationship between pulsed-field gel electrophoresis (PFGE) patterns of the 4 selected *Erysipelothrix rhusiopathiae* field isolates obtained after restriction enzyme digestion with *SmaI*. The classification and divergence of isolates was calculated by the unweighted pair group method averages from the PFGE results.

of erysipelas characterized by lameness and endocarditis.[17] In birds, *E. rhusipathiae* infection is most important in turkeys, which seem to be especially susceptible to septicemia.[2,8] Sporadic cases of erysipelas have also been described in other avian species such as caged laying chickens,[11] ducks,[5] chukars (*Alectoris chukar*),[3] quails,[13] farmed emus,[6] ring-necked pheasants,[7] a free-ranging Hawaiian crow (*Corvus hawaiiensis*),[18] and in a little blue penguin (*Eudyptula minor*).[1]

A Laughing kookaburra (*Dacelo novaeguineae*) of unknown age was presented to the Veterinary Diagnostic Laboratory at Iowa State University, with a history of death after a 2-week period of weakness and weight loss. The kookaburra is one of the largest members of the Australian kingfisher group and is native to eastern Australia. In its natural habitat, kookaburras feed mainly on insects, reptiles, frogs, freshwater crayfish, earthworm, small birds, and rodents. The diet of the bird described in this case consisted of frozen rodents, mealworms, and occasional insects and earthworms. The bird had been kept in captivity and was housed alone in a 5 × 5 × 8–feet cage with a sandy soil bottom. The animal had no contact with turkeys or pigs; however, feral avian animals including peacocks were in the area.

A routinely performed fecal float analysis was negative for parasites. At necropsy, the pectoral muscle was moderately atrophied; the lungs were red, wet, and heavy; and the cloacal orifice was covered with dried feces. Sections of all organs were immersed in 10% neutral-buffered formalin, routinely processed for histopathology, stained with hematoxylin and eosin (HE), and evaluated microscopically. In addition, routine bacterial cultures using aerobic and anaerobic incubation on 5% sheep blood agar plates,[a] Tergitol-7 agar plates with tripheyltetrazolium chloride,[b] and brilliant green agar plates with novobiocin[b] were performed on sections of lung, liver, heart, and cloacal swabs. Mycotic cultures (Sabouraud dextrose agar[b] and mycobiotic agar[b]) were performed on lungs at 25°C and at 35°C. Microscopically, there was severe pulmonary congestion with focal thrombosis and congestion of the liver. No significant changes were detected in brain, gastrointestinal tissues, kidneys, and lymphoid tissues. Browns and Hopps Gram stain[14] on formalin-fixed, paraffin-embedded lung tissue demonstrated gram-positive rods within congested blood vessels. Moderate numbers of *E. rhusiopathiae* were isolated from the lung and from the liver. The *E. rhusiopathiae* was further identified on the basis of hydrogen sulfide production in Kligler iron agar.[b] Mycotic cultures were negative. Polymerase chain reaction assay for West Nile virus[10] on brain tissue homogenate was negative.

Table 1. Pulsed-field electrophoresis patterns (genotype) and serotypes of 4 erysipelas field isolates.

Isolate no.	Date isolated	Genotype	Serotype	Species recovered from
44997	October 11, 2000	3A (IV)	1b	turkey
45875	August 14, 2001	1A (I)	1a	pig
57742	October 19, 2001	3A (I)	1b	pig
16440	June 25, 2004	3A (III)	NT*	kookaburra

* Not tested because of unavailability of antiserum.

For further characterization of the *E. rhusiopathiae* isolate, pulsed-field gel electrophoresis (PFGE) was performed on cultures as described previously.[12] The PFGE has been used previously to characterize Midwest regional and US erysipelas isolates. For the purpose of comparison, 2 porcine *E. rhusiopathiae* isolates as well as a turkey *E. rhusiopathiae* isolate were run on the same gel with the kookaburra isolate. The turkey isolate had been recovered during an acute outbreak of septicemia in a turkey flock in 2000 and was at that time determined to be serotype 1b (Table 1). The 2 swine isolates (No. 45875 and 57742) were both recovered in the Midwest United States during 2001, were both genetically characterized by PFGE and serotyped (Table 1), and are representative of the most prevalent porcine field isolates in the United States.[12] Because of lack of antiserum for serotyping, it was not possible to serotype the kookaburra isolate. The PFGE patterns were analyzed visually and compared with BioNumerics software.[c] Dendograms used the unweighted pair group method using arithmetic averages, dice coefficient, and 0.9% optimization with 2.0% band position tolerance, as described previously.[12] The results of the PFGE indicated that both the turkey and the kookaburra isolates had a PFGE pattern not previously observed and were designated as 3A(IV) (Turkey) and 3A(III) (kookaburra). Direct comparison of the PFGE patterns of the 2 pigs, the turkey, and the kookaburra isolates revealed that the 2 serotype 1b isolates (pig and turkey) were similar to the kookaburra isolate, whereas the PFGE pattern of the serotype 1a pig isolate did not match the pattern (Fig. 1). Compared with the porcine serotype 1b isolate, the turkey isolate had one additional band. The kookaburra isolate had an additional band and lacked another band that was present in both the porcine and the turkey isolates (Fig. 1). Data analysis of the homogeneity of the PFGE patterns among the isolates revealed that the

kookaburra isolate was most related to pig No. 57742 isolate with 91.7% identity, was 88% homologous to the turkey isolate, and was 78.3% homologous to pig No. 45875 isolate. It is not known whether the kookaburra isolate is also biologically different from other *E. rhusiopathiae* isolates. The antibiogram of the kookaburra isolate was similar to what has been previously reported for *E. rhusiopathiae* isolates[12] (e.g., the isolate was susceptible to ampicillin, ceftiofur, enrofloxacin, erythromycin, gentamicin, penicillin, and trimethoprim and resistant to sulfadimethoxine).

The clinical history of the bird was suggestive of chronic disease. However, no infectious or noninfectious causes to explain the prolonged clinical presentation were detected, and acute erysipelas septicemia was diagnosed in the Laughing kookaburra. Routes of transmission for *E. rhusiopathiae* are believed to be direct horizontal transmission by asymptomatic carriers such as pigs, turkeys, or contaminated fish food as well as indirect horizontal transmission by the means of fomites such as contaminated soil or mechanical vectors such as arthropods.[4] In the case reported in this study, the source of infection remains undetermined. This is, to the authors' knowledge, the first observation of erysipelas in this avian species.

Sources and manufacturers

a. Remel Inc., Lenexa, KS.
b. Difco Laboratories, Detroit, MI.
c. Applied Maths, Kortrijk, Belgium.

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