Chlamydiosis in a Captive Group of Houbara Bustards (Chlamydotis undulata)

Arnaud Greth, Bruno Andral, Hermann Gerbermann, Marc Vassart, Helga Gerlach, and Frederic Launay

**SUMMARY.** A chlamydiosis outbreak occurred in a Houbara bustard (Chlamydotis undulata) captive breeding group in Saudi Arabia, inducing peracute deaths, highly variable clinical signs, and pathological and histological lesions. Typical inclusion bodies in stained impression smears of spleen and prevalence (80%) of antibodies against Chlamydia, detected by a competitive enzyme immunoassay test, provided the bases for the diagnosis. This is the first report on a chlamydiosis outbreak in birds of the family Otididae.

**CASE REPORT**

**Case history and necropsy.** Three different groups of Houbara bustards kept at the NWRC were involved. Group A consisted of 55 bustards that arrived from Pakistan at the end of 1989 and were kept in quarantine. Group B was represented by 180 adult breeders kept in a nearby breeding unit. Group C consisted of the 47 chicks obtained during the 1990 breeding season.

The Houbara bustard belongs to the family Otididae, whose diseases and pathology are poorly documented. Antibodies against Chlamydia have been demonstrated by complement fixation technique in one species of the family, the Great bustard (Otis tetrax) (2). This report describes the first case of an outbreak of chlamydiosis in birds of the family Otididae.
Two birds from Group A were affected by a paralysis of both legs 15 days after their arrival at the NWRC. Despite treatment, they did not recover. One died after 3.5 months, mainly because of its poor condition. Postmortem examination showed no lesions to explain the paralysis. However, the spleen was enlarged and the liver was of an unusually dark color. Spleen impression smears stained with Köster-Stamp stain showed many intracytoplasmic inclusions resembling *C. psittaci* morphologically.

Fourteen of 17 birds of Group A that were present. The spleen was hypertrophied and congested. Staining of spleen impression smears showed numerous inclusions.

One 10-day-old chick from Group C died in the breeding unit. The chick had refused to eat and had been force-fed. Postmortem examination revealed congested lungs. Staining of spleen impression smears showed inclusions similar to those seen with chlamydiosis.

Another bird in quarantine died of a peracute disease in June 1990, starting an outbreak in a batch of 20 birds issued from Group A. Postmortem examination revealed tracheitis, jejunitis, colitis, and congestion of most of the organs. Eight bustards among the 20 showed prostration, signs of tracheitis, and a nasal discharge. All of the birds were treated. Four birds, showing a peracute course of the disease, were found dead over the next 10 days. The morbidity rate reached 45% and the mortality rate 25%. At postmortem examination, one of the four displayed airsacculitis and peritonitis (with numerous white spots covering the air sacs and the peritoneum), as well as enteritis and pericarditis. The three other carcasses did not have many lesions, except colitis and congestion of the lungs in one individual. *C. psittaci* was suspected again on the basis of staining of spleen impression smears.

No attempt was made to culture *Chlamydia* in any case.

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### Table 1. Houbara bustards with antibodies against *Chlamydia* by enzyme-linked immunosorbent assay (ELISA) in Groups A, B, and C at different periods.

<table>
<thead>
<tr>
<th>Sampling time</th>
<th>Group A</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Group B</th>
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<th>Group C</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Negative</td>
<td>Weak positive</td>
<td>Positive</td>
<td>Strong positive</td>
<td>Negative</td>
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<td>Strong positive</td>
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<td>Weak positive</td>
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<tr>
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<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>2/27</td>
<td>1/27</td>
<td>24/27</td>
<td>0/27</td>
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<td>ND</td>
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<tr>
<td>1/34 (2.9)</td>
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<td>2/34</td>
<td>0/34</td>
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<td>2/34</td>
<td>2/34</td>
<td>2/34</td>
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<td>1/90 (4.5)</td>
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<td>19/44</td>
<td>23/44</td>
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<td>60/180</td>
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<td>3/180</td>
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<td>4/49 (8.2)</td>
<td>10/49</td>
<td>35/49</td>
<td>0/49</td>
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<td>2/34</td>
<td>31/34</td>
<td>1/34</td>
<td>(5.9)</td>
<td>(91.2)</td>
</tr>
</tbody>
</table>

*ELISA values: >80% = negative, ≤80%–>60% = low antibody titer (weak positive), ≤60%–>30% = medium antibody titer (positive), ≤30% = high antibody titer (strong positive). Ratios show number of serum samples at given level of antibody/total tested (%).*
Histopathologic findings. Because of their similarity, the lesions found upon histological examination can be summarized as follows: The heart showed a high degree of edema and a myodegeneratio cordis (degeneration of the muscle fibers by degenerative, non-inflammatory disease processes occurring with dystrophic or chronic disease processes, and causing cardiac insufficiencies). The liver showed hyperemia, edema, small hemorrhages, and bile congestion. The spleen revealed a depletion of lymphocytes, mainly in the white pulp, and necrosis of some reticular cells. In the kidney, the signs of circulatory disturbance were prominent (hyperemia, edema including the walls of the blood vessels, hemorrhages, and thrombi). Necrosis of the tubular epithelium and glomerulonephritis were also seen. The lungs were hyperemic and edematous, with small hemorrhages. The trachea of one bird showed a hemorrhagic tracheitis with loss of cilia and proliferation of mucus-producing cells.

It was also noted that fibrinous or fibrous peritonitis, particularly affecting the serosa of the intestinal trace as well as parts of the intestinal wall, occurred fairly frequently.

Serological studies. C. psittaci infection was confirmed by serological findings, using the commercially available competitive enzyme immunoassay Chlamydia-psittaci-AK-EIA (Röhm Pharma GmbH, Darmstadt, Germany). A semiquantitative interpretation was made, according to the results of Janeczek (6) and Gerbermann (3). Most of the bustards had antibodies against C. psittaci (Table 1). Of 34 individuals examined in Group A obtained in mid-December 1989, 33 had low antibody titers against C. psittaci. Of the 17 birds kept in the pen, nine were tested and had a positive reaction. Workers in contact with the birds also were tested with the same serological technique; of 11 persons, 10 were positive and one was strongly positive.

REFERENCES
5. Greth, A., H. Gerlach, B. Andral, and M. Vassart. Pathology of the Houbara bustard in captive breeding colony in 1987, 1988, and 1989 mainly owing to digestive lesions. Of primary importance in this respect was a sporadic, acute, fatal enteritis-peritonitis syndrome. Catarrhal enteritis and a dry fibrinous peritonitis were frequently seen together with splenomegaly, marbled and congested liver, pancreatitis, and pneumonia (5). Chlamydiosis was not considered during this outbreak in 1989, but it was proved by serological results that C. psittaci was already present at the NWRC. From the clinical signs and pathological lesions described here, it is likely that C. psittaci contributed to the enteritis-peritonitis syndrome. In a previous study, it was also shown that the birds were infected by a variety of other viral and bacterial agents (5). Thus, it is assumed that the cases described here were caused by a multifactor etiology in which C. psittaci is one of the more important agents. Andral et al. (1) described an outbreak of rhinotracheitis in turkeys with the involvement of C. psittaci and viral agents (hemorrhagic enteritis adenovirus and paramyxovirus II).

Considering the serological results (kinetics), the clinical signs, and the necropsy findings, it appears that the birds of Group A, recently introduced to the NWRC, did not have previous contact with C. psittaci, whereas the latent infection had perhaps existed for years in the breeding unit and may have given a pre-


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