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AVIAN PARAMYXOVIRUS TYPE 1 INFECTION IN HOUBARA BUSTARDS (*CHLAMYDOTIS UNDULATA MACQUEENII*): CLINICAL AND PATHOLOGIC FINDINGS

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Abstract: Clinical and pathologic findings of avian paramyxovirus type 1 (PMV-1) in 19 houbara bustards (*Chlamydotis undulata macqueenii*) imported from Pakistan into the United Arab Emirates and one captive-bred bird are reported. Clinical signs included circling, walking backward, ataxia, opisthotonos, torticollis, recumbency, head tilt, head shaking, head tremor, tucking of head under keel, nasal discharge, conjunctivitis, and diarrhea. The length of time imported birds exhibited clinical signs varied from 4 days to 18 mo after importation. Hemagglutinating antibodies against PMV-1 were detected in the sera of all 17 birds from which blood samples were collected, and PMV-1 was isolated from pooled brain, spleen, and lung tissues from two birds with acute clinical signs. There were no distinctive gross lesions at necropsy, and histologic findings were consistent with but not pathognomonic for PMV-1. All houbara bustards managed in a captive breeding and restoration program established by the National Avian Research Center have been vaccinated against PMV-1 since October 1992, and no case of PMV-1 has been reported in this collection since that time.

Key words: Houbara bustard, *Chlamydotis undulata macqueenii*, Newcastle disease, paramyxovirus type 1, serology, virology.

INTRODUCTION

The houbara bustard (*Chlamydotis undulata macqueenii*) belongs to the family Otididae and is found in harsh, arid habitats as three subspecies across North Africa and parts of Asia.[14] Houbara bustards have declined in number throughout most of their range as a result of overgrazing, intensive farming, and pesticide use in their breeding areas coupled with overhunting, disturbance, and trapping in the countries in which they overwinter and through which they migrate.[8,14] The International Union for the Conservation of Nature classifies the houbara as "vulnerable," which describes populations that are seriously depleted or decreasing as a result of overexploitation or other factors and are at risk of becoming "endangered" if causal factors continue unchecked.[13] Attempts at captive breeding and restoration have been undertaken, and in the Middle East, the houbara bustard has been the focus of several conservation programs in Bahrain,[26] the Kingdom of Saudi Arabia,[23] and the United Arab Emirates (UAE).[20,22] Captive populations of houbara bustards are susceptible to a wide variety of diseases including avian paramyxovirus type 1 (PMV-1) infection,[4,29] which causes Newcastle disease (ND).[1] The literature on diseases of houbara bustards is scarce, and to our knowledge, there is no published report describing the presentation of PMV-1 infection in this species. This work describes the clinical, virologic, bacteriologic, fungal, serologic, and pathologic findings in houbara bustards that were serologically or virologically positive to PMV-1.

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CASE REPORT

Clinical history

Three groups of birds were examined and are described separately. Group 1 birds had exhibited clinical signs for 6–18 mo, group 2 birds had exhibited clinical signs for 1–2 mo, and group 3 birds had exhibited clinical signs for 4 days before examination. None of the birds had been vaccinated against avian PMV-1.

Group 1: Eight houbara bustards were examined in the quarantine facility of Al Ain Zoo, UAE, in September 1992. All eight birds had developed central nervous system (CNS) signs 6–18 mo before they were examined. One bird was captive bred at Al Ain Zoo; it developed clinical signs while housed in a display aviary and was moved to the quarantine facility. The remaining seven birds were wild-caught adults and had been imported from Pa-

Figure 1. Houbara bustard with neurologic PMV-1 showing torticollis.

kistan at different times over the previous 2 yrs. These birds developed CNS signs while housed in the quarantine facility. Detailed clinical histories on these birds were not available. In late September 1992, blood was collected and all eight birds were euthanatized and necropsied.

Group 2: A flock of 36 wild-caught, adult, male and female houbara bustards was imported from Pakistan to a private farm in the UAE in the first week of March 1993. One month after their arrival, three of the birds developed CNS signs and died. No examination was conducted on these birds. During a preliminary visit to the unit in April 1993, six of the remaining 33 birds were showing CNS signs. Clotted blood samples were collected from the four birds with the most severe CNS signs and were submitted for avian PMV-1 serology. Unfortunately, the birds were not marked, and it was not possible to individually identify these birds on subsequent visits to the unit. Two days after the first visit, two houbara bustards with CNS signs were clinically examined, blood was collected, and the birds were euthanatized and necropsied. Five weeks later, four more houbara bustards with CNS signs were clinically examined, euthanatized, and necropsied. Three frozen houbara bustards that had died after developing CNS signs 2 wk prior were also necropsied.

Group 3: A flock of 200 wild-caught houbara bustards was imported from Pakistan to a private farm in the UAE in February 1995. Four days after their arrival, many of the birds developed CNS signs, and the farm manager later reported that approximately 50% of the birds died over the following month. On the same day that CNS signs were observed, two dead birds and one live bird were submitted to the Central Veterinary Research Laboratory (CVRL), Dubai, UAE, for necropsy. It was not possible to gather any further information on this flock.

Clinical findings

Six female and two male houbara bustards were examined in Group 1. The CNS signs included circling, walking backward, ataxia, torticollis (Fig. 1), incoordination, tucking of head under keel, head tilt, and opisthotonos. Other clinical findings included cestode proglottids in the feces and traumatic lesions on the wing tips and above the keel. All birds except one were in good body condition with weights in the normal range;[25] the mean body weight for five females was 1,029 g and for the two males was 1,715 g. The bird with the most marked CNS signs (a female) had the poorest body condition and weighed 609 g.

In Group 2, 12 of 36 (33%) birds developed CNS signs. Four females and two males were clinically examined, and CNS signs of these six birds included ataxia, circling, head tremor, head shaking, torticollis, and exhibited opisthotonos. Other clinical findings included inappetance, mucoid nasal and conjunctival discharge, green diarrhea, clear mucoid fluid dribbled from beak, recumbency, wasted pectoral muscles, traumatic lesions on wing tips,

and feather lice. The birds were in poor body condition; the mean body weight for the four females was 695 g and for the two males was 1,030 g.

The CNS signs exhibited by the birds in Group 3 included circling, head tilt, and torticollis.

Necropsy findings

The birds were euthanatized with an intracardiac injection of pentobarbitone sodium (Euthatal, Rhone Merieux, Harlow, Essex, United Kingdom). Necropsy examinations were performed according to a standard technique,[18] and tissues were preserved in 10% neutral buffered formalin.

Necropsy findings for the birds in Groups 1 and 2 were variable, and most did not correlate with the clinical signs. One bird in Group 1 had a shrunken, yellow left cerebral cortex. In Group 2, seven birds had wasted pectoral muscles and five birds had 1–3-mm-diameter cream and green nodules on their abdominal air sacs. Additional necropsy findings for Groups 1 and 2 birds included endoparasites in the gastrointestinal tract; trauma to the wing tips and keel; small foci of hemorrhage in the medial and lateral thigh muscles; nares blocked by crusted exudate; small, translucent nodules on the gut mesentery; proventricular mucosal inflammation and nodules on the mucosal ileum associated with endoparasites; an infected keel ulceration; and a pale liver with a caseous nodule on the surface. The parasitic findings have been discussed elsewhere.[15]

The necropsy findings for the three birds in Group 3 included one case of a fractured clavicle. All birds had otherwise good body condition.

Histopathology and immunohistochemistry findings

Only formalin-fixed brain tissues were collected from the eight birds in Group 1. The samples were submitted to Ravenscourt Laboratories (London, United Kingdom) for histopathology. Of the Group 1 birds, the bird with the shrunken cerebral cortex had focal, chronic lymphohistiocytic meningitis, but no organism was seen on gram-stained sections. One brain had focal meningeal thickening with lymphocytic infiltration.

Formalin-fixed tissues from seven Group 2 birds were submitted to Cambridge University (Cambridge, United Kingdom) for histopathology and immunohistochemistry. Microscopically, all spleens had reactive changes and mild hemosiderin accumulations. All kidneys had mild lymphoid or lymphoplasmacytic aggregates in the cortex, and two had mild hemosiderin accumulation in the tubular epithelia. One bird had more diffuse lymphoplasmacytic interstitial nephritis. Six livers had mild-to-marked vacuolar hepatopathy, and all showed mild hemosiderin accumulation and small lymphoid accumulations within the parenchyma. All lungs had peribronchial accumulations of refractile crystalline material (presumably inhaled dust) and mononuclear cells. Other findings in the lungs included congestion in four birds and edema with occasional inflammatory changes in two birds. Brain sections, including cerebellum, cerebrum, midbrain, and hindstem, appeared normal. The spinal cords were not examined.

Immunohistochemistry was performed on paraffin-embedded sections of brain, liver, kidney, lung, and trachea from seven Group 2 birds. Formalin-fixed chick brain tissue known to be either PMV-1 infected or PMV free served as positive and negative control tissue for immunohistochemistry. Sections were dewaxed, and endogenous peroxidase activity was blocked with 0.06% hydrogen peroxide in methanol. Digestion in proteinase K (10 μl/ml) was carried out for 30 min at 37°C. Tissue was incubated in normal horse serum (Sigma Chemical Ltd., Poole, Dorset, United Kingdom) at 1:10 dilution, before mouse monoclonal antibody to PMV antigen was applied at 1:1,000 dilution. Biotinylated horse anti-mouse antibody (Vector Laboratories, Bretton, Peterborough, United Kingdom) was applied at 1:200 dilution, followed by Vectastain Elite (Vector Laboratories). Chromogenic substrate was 0.06% diaminobenzidine (Sigma Chemical Ltd.). Viral antigen was detected in the positive control brain but was absent from all tissues in Group 2 birds.

Formalin-fixed brain, liver, and spleen samples from Group 3 birds were submitted to the CVRL. One bird had a few gliotic foci and mildly hyperplastic endothelial cells in the brain. No other histopathologic abnormality was detected.

Virology findings

Virology was conducted at the CVRL. Virus isolation was undertaken on pooled samples of brain, spleen, and lung, as described by Wernery and others.[30] All samples were passaged three times through quail embryo fibroblasts. At the onset of cytopathic effects appearing in the quail embryo cells 3–6 days after inoculation, the cultures were subjected to one freeze/thaw cycle and sonicated; the cell debris was then removed by centrifugation, and the supernatant was tested for hemagglutinating activity. Hemagglutinating viruses were isolated from pooled tissue samples from two birds in Group 3. Preliminary screening in hemagglutination inhibition (HI) tests using polyclonal chicken PMV-1 antiserum showed the two viruses were inhibited with antibody titers of 1:16 and 1:32. Both

Table 1. Results of indirect hemagglutination inhibition tests for antibodies against paramyxovirus type 1 in houbara bustards with central nervous system signs for 6–18 mo (Group 1).

Bird ID	Titer (29 Sep 1992)
1	>1:128
2	1:32
3	1:64
4	1:128
5	>1:128
6	1:128
7	1:128
8	1:64

isolated viruses have been submitted to Central Veterinary Laboratory (New Haw, Weybridge, Surrey, United Kingdom) for further identification.

Bacteriology and mycology findings

Bacteriology and mycology were conducted at the CVRL. Samples of feces and duodenal contents and a swab of the shrunken cerebral cortex of one bird were cultured for *Salmonella* spp. using standard microbiology techniques.[28] No *Salmonellae* was isolated from any of the samples. Culture of the lesion in the brain from a bird in Group 1 did not yield any organism.

Air sac lesions collected at necropsy were cultured for fungi using standard microbiology techniques.[21] *Aspergillus* spp. were isolated from all air sac lesions of Group 2 birds.

Serology findings

Serum samples were submitted to the CVRL, where serology was performed using a HI test (NDV HI) to detect PMV-1 antibodies.[30] Serology results of eight birds in Group 1 are presented in Table 1. All six Group 2 birds from which serum samples were collected had high PMV-1 HI titers; five were >1:128 and one was 1:64. Both of the two birds sampled 2 days after the preliminary visit had titers >1:128. No serum sample was collected from Group 3 birds.

DISCUSSION

Houbara bustards are known to be transported from Pakistan to the UAE by dhow (local boats) and planes in crates that are often in close proximity to crates containing chickens and pigeons. Conditions during transport are unhygienic; houbara bustards are transported in three-tiered crates with welded mesh floors where the birds in the upper levels defecate on the birds in the lower levels. The birds are often unable to stand upright in the cramped, crowded crates; food and water provision is inadequate and frequently absent. Consequently, birds typically arrive in the UAE in poor body condition. These factors are likely to be extremely stressful for wild birds that have been recently captured.

Avian PMV-1 is transmitted primarily by respiratory aerosols, fecal contamination of food and water, direct contact with infected birds, and fomites.[1,11] The unhygienic methods used to transport houbara bustards and the direct contact with domestic avian species facilitate virus transmission. In pigeons, the incubation period for PMV-1 varies from a few days to several weeks, and new clinical cases can continue to appear 5–8 wk after the onset of the outbreak.[9]

Paramyxovirus type 1 infection is a variable disease in exotic birds, and susceptibility varies between avian orders.[7,9] The disease produced following infection varies with the virus pathotype; other factors include the species, age, immune status, general health of the bird and environmental conditions.[1] Clinical findings in other affected exotic birds include ruffled plumage, ataxia, torticollis, opisthotonos, tremors, leg paralysis, conjunctivitis, dyspnea, green diarrhea, limp neck, enteritis, airsacculitis, extreme respiratory distress, lateral recumbency, loss of visual acuity, and polydipsia.[9,19,24,27,29,31] Central nervous system signs were the most commonly observed clinical signs in this study in both chronic (Groups 1 and 2) and acute (Group 3) cases examined.

Pigeons may return to health after a convalescence of 6 mo.[9] Some of the houbara bustards described in this paper had survived for up to 18 mo after developing CNS signs, but the CNS signs observed did not regress before euthanasia. The body conditions of the Group 1 birds with chronic CNS signs and the Group 3 birds with acute signs were generally good. In comparison, the body conditions of the Group 2 birds were very poor, presumably resulting from stress, inappetance, endoparasites, and aspergillosis.

In this study, there were no distinctive gross findings other than parasitism and aspergillosis. The macroscopic lesions of PMV-1 are variable and depend on many factors.[5,6] No pathognomonic lesions for PMV-1 are described, and there may be no gross lesions.[6] Necrosis and lymphoplasmacytic infiltration in the liver, kidney, and pancreas have been reported in PMV-1 infection, with pulmonary congestion and lymphoplasmacytic inflammation.[5] Central nervous system lesions include nonsuppurative meningoencephalitis, mild edema, lympho-

cytic perivascular cuffing, endothelial cell hypertrophy, vasculitis, neuronal degeneration, and focal gliosis.[6,12] Definitive diagnosis requires virus isolation, demonstration of the viral antigen, or rising specific antibody titers.[2]

The absence of detectable viral antigen in sections of brain from birds with chronic CNS signs and positive HI titers is problematic. Viral antigen can be detected in neurons, glia, and endothelial cells when CNS signs are absent early in the course of disease but may be undetectable by the time CNS signs have developed.[31] Immunohistochemical detection of PMV-1 antigen may give false negative results because of degradation of viral antigen after formalin fixation or tissue processing.[17]

Virus was isolated from only two birds in the acute stages of the disease. No virus was isolated from any bird in Groups 1 or 2, probably because the samples were submitted too long after infection. Alexander (pers. comm.) was unable to isolate avian PMV-1 from bowel contents and brain samples of pigeons presenting with nervous signs and high PMV-1 HI titers. Possible reasons for the difficulty in isolating virus are that virus was present in low titers and was neutralized by contaminating antibodies when the birds were sampled or that virus was not present because infection had occurred previously and the clinical signs resulted from damage that had occurred at the time of infection (Alexander, pers. comm.). The combination of clinical signs, serologic results, and virus isolation confirmed the diagnosis of PMV-1.

A titer of 1:8 is usually considered evidence of exposure to PMV-1.[1] In unvaccinated birds, positive titers coupled with clinical signs are regarded as strong diagnostic evidence of PMV-1 infection.[1] All serum samples collected from Groups 1 and 2 houbara bustards with CNS signs were seropositive for avian PMV-1. Serum samples collected from the surviving members of Group 1 were also serologically positive, with antibody titers greater than 1:8 (Bailey, unpubl. data).

Morbidity and mortality of houbara bustards infected with PMV-1 appear to be highly variable. Thirty-three percent of the birds in Group 2 developed clinical signs, 16% died naturally, and 16% were euthanatized. Approximately 50% of the birds in Group 3 died. Review of Al Ain Zoo veterinary records revealed that in 1986, all 30 recently imported, wild, adult houbara bustards died of suspected PMV-1 infection; in January 1991, four of six recently imported houbara bustards died after developing suspected PMV-1; and in January 1992, two of 14 recently imported houbara bustards were euthanatized with suspected PMV-1. Five other species of bustard are managed at Al Ain Zoo: rufous-crested bustard (*Eupodotis ruficrista*), Heuglin's bustard (*Neotis heuglini*), kori bustard (*Ardeotis kori*), black bustard (*Eupodotis afra*) and white-bellied bustard (*Eupodotis senegalensis*). Paramyxovirus type 1 infection has been suspected in only a single white-bellied bustard and not in other bustard species (Bailey, unpubl. data). We have observed PMV-1 epizootics in palm doves (*Streptopelia senegalensis*), collared doves (*Streptopelia decaocto*), and feral pigeons (*Columba livia*) in the UAE, and it is possible that these birds are a source of infection for birds housed in outdoor aviaries. In order to reduce the risk of infection, all bustards managed by the National Avian Research Center (NARC) in the UAE are vaccinated annually with a s.c. injection of 1.0 ml/kg body weight of inactivated ND vaccine (Newcavac Nobilis, Intervet, Cambridge, United Kingdom). Since a vaccination program was introduced in October 1992, no case of PMV-1 infection has been reported in this collection.

Avian PMV-1 has been described in birds in the Middle East previously.[10,16,30] This disease has important implications for the husbandry and conservation of houbara bustards. The introduction and spread of ND by imported houbara bustards represents a real risk to captive breeding programs such as NARC's as well as to the UAE's domestic poultry industry. Ashton and Cooper[3] reviewed measures to minimize the risk of spreading diseases such as ND and welfare problems associated with the import and export of birds into and from the United Kingdom. A national quarantine center within the UAE, where imported domestic and non-domestic birds could be quarantined and receive medical attention, is urgently needed to facilitate the birds' welfare and to minimize the introduction of pathogens into the UAE.

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