Communications

A severe nervous disease in fancy pigeons caused by paramyxovirus-1 in Saudi Arabia

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AL AFALEQ (A.I.), ABU ELZEIN (E.M.E.), GAMEEL (A.A.), AWAAD (M.), HASSANEIN (M.M.). Description d'une pathologie neurologique grave provoquée par paramyxovirus-1 chez le pigeon d'agrément en Arabie Saoudite. *Revue Élev. Méd. vét. Pays trop.*, 1993, **46** (4) : 545-547

Une pathologie neurologique grave a frappé les pigeons d'agrément pour la première fois en Arabie Saoudite en février-mars 1992. Le taux de morbidité était de 60 % et le taux de mortalité de 40 %. On a isolé un paramyxovirus-1 aviaire chez les pigeons malades. La maladie a pu être reproduite expérimentalement chez les pigeons, chez qui le virus a été réisolé et identifié.

Mots clés : Pigeon - Paramyxovirus aviaire - Trouble du système nerveux- - Infection expérimentale - Épidémiologie - Arabie Saoudite.

Introduction

Pigeons (*Columba livia*) in Saudi Arabia are mainly kept as fancy birds. For this purpose, various breeds are provided from abroad, and reared under good conditions.

Inspite of reports on poultry viral diseases in Saudi Arabia (1), pigeons have always been free from such infections. However, SHALABY *et al.*, 1985 (10) reported the presence of herpes virus infection in pigeons in the Eastern Province.

Between February and March 1992, a severe disease outbreak occurred in pigeons at Dirab (Locus 24°25'N ; 46°36'E), the central region of Saudi Arabia. The total number was thousand birds. The morbidity rate was 60 % while the case fatality rate was 40 %. The clinical signs resembled the neurotropic form of Newcastle disease (ND) in chickens. The birds were listless, unable to fly and had raffled feather. They showed incoordination, anorexia and torticolis. Greenish diarrhoea was also seen. The course of the disease took five to ten days during which the birds either died or gradually recovered with torticolis as sequelae (photo 1).

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Materials and Methods

Gross-and-histopathology

Pigeons showing symptoms were sacrificed and postmortem examination carried out. Brains, livers, spleens, respiratory organs and whole blood in EDTA were collected for virus isolation assays.

Samples from the brain, lungs, livers, kidney, intestine, spleen and skeletal muscle, were also collected in 10 % formol-saline. Paraffin sections were prepared and stained with haematoxylin and eosin (HE) and examined for histopathological changes.

Virus isolation assays

Tissue samples for virological investigations were put into 50 % (W/V) suspension in phosphate buffered saline (PBS), pH 7.4 , and centrifuged at 750 rpm for ten minutes. The supernatant was collected to which antibiotics were added and used to inoculate 9-day old specific-pathogen free (SPF) embryonating chicken eggs via the allantoic cavity (7). Inoculated eggs were incubated at 37 °C and candled daily. Eggs dying within the first 24 h were discarded. Subsequently, dying eggs were collected and kept for 3 h at 4 °C before the allantoic fluid was harvested.

Haemagglutination test (HA)

The haemagglutination test (HA) was performed on the original material and on the allantoic fluids from inoculated eggs using microtitre plates according to HANSON (8).

Photo 1 : A sick pigeon shown backward bending of the head and neck.

Haemagglutination inhibition test (HI)

The beta-method of the haemagglutination test (HI) was performed (8) employing two conventional Newcastle antisera (classical avian paramyxovirus serotype 1).

Agar gel immunodiffusion test (AGID)

Fifty percent homogenates from the brain and other visceral organs, collected from ailing pigeons, and allantoic fluid from passage one were reacted against the ND antiserum in agar gel immunodiffusion tests (AGID) employing a known ND virus as a positive control antigen (8). The brain from a healthy pigeon was used as a negative control antigen. Non-immune pigeon serum was also used as a control.

Thermostability

Thermostability test on the virus was carried out at 56°C for various times as described by HANSON (8).

Agglutination of mammalian RBCs

The ability of the virus to agglutinate mammalian erythrocytes involved examination of sheep, goat, cattle and horse and also pigeon red blood cells (RBC), as described by HANSON (8).

Experimental infection of pigeons

Fifteen indigenous seronegative pigeons were used. Five were kept as uninoculated controls in a separate room. Five were inoculated each intramuscularly with 10^8 50 % egg infectious dose (EID_{50}) of the virus isolated from the naturally-infected pigeons. The remaining 5 pigeons were inoculated intravenously with a similar dose of the virus . Each group was kept separately in a cage and provided with feed and water *ad lib.* The birds were kept under daily observations. Blood for serum was collected every two days post inoculation to test for seroconversion. To detect excreated virus, cloacal swabs were taken every two days post inoculation.

Post-mortem examination was performed on dead and sacrificed pigeons, and brains, blood and tissue samples from visceral organs were collected for virus reisolation.

Results

Virus isolation and identification

The embryonating eggs, inoculated with the original material died whithin 3-5 days post inoculation. Most HA activity was detected in allantoic fluids from eggs inoculated with the brain material (1/1024), followed by the liver (1/512), blood (1/128), spleen (1/32) and respiratory organs (1/8), respectively. The same picture was seen in the original material with the brain giving the highest HA end-point titres followed by the liver, spleen and respiratory organs respectively .

The HA activity of the isolated virus was inhibited by the ND sera (avian paramyxovirus 1).

The AGID

A complete precipitation line of identity was produced between the known ND antiserum and the virus contained in the brain and liver homogenates and the allantoic fluid of passage one respectively. This line completely merged with the line produced between the known ND virus and the ND antiserum. No lines were seen between the brain from healthy pigeons and the positive serum, and no reaction was seen between the negative serum and the brain or livers from sick birds.

Thermostability

The virus HA activity was completely lost following heating for 30 min at 56°C.

Agglutination of mammalian RBCs

The virus agglutinated best pigeon and chicken RBCs. Low HA activity was seen with sheep and goat RBCs, but no HA activity was seen with horse or bovine RBCs.

Gross-and-histopathological findings

The P.M. picture showed congestion of the brain and fatty changes in the liver. The kidneys were oedematous. Splenomegaly was evident. There was massive myocardial necrosis and the lungs looked congested, and there was serous air-saculitis. The intestinal serosa and mucosa showed some haemorrhagic spots.

The histopathological changes were seen in the brain, lungs, liver, kidneys, intestines and skeletal muscles. Changes in the brain tissue were characterized by congestion and capillary proliferation associated with focal areas of haemorrhages. Perivascular mononuclear cell cuffs were seen in the cerebral cortex with diffuse (occasionally focal) gliosis. Neuronal degeneration was also seen. Similar degenerative changes were seen in Purkinji cells in the cerebellum associated with vaculations in the white matter. Mild to severe changes were seen in the liver characterized by hepatocyte swelling, degeneration, sinosoidal dilatation and infiltration of mononuclear cells. In the heart, blood vessels were

congested. Mild myocardial degeneration associated with slight to moderate proliferation of interstitial cells was seen. Changes in the kidneys were mainly congestion and haemorrhages accompanied by tubular degeneration and infiltration of the interstitium with mononuclear cells.

Reproduction of the disease in experimental pigeons

Two pigeons of the I/V group started showing symptoms by day three. Both pigeons died on day seven. The remaining three showed symptoms between day four and six and all died by day eight. The five pigeons inoculated I/M showed symptoms between day three and four and all died by day eight. The symptoms were nervous signs and loose droppings. Virus was isolated from all inoculated pigeons from day three. Low level HI titres were detected in 30 % of the inoculated pigeons.

Discussion

The clinical signs, gross P.M. lesions and histopathological picture of the examined pigeons together with the virus isolation and identification and reproduction of the disease in pigeons, were highly suggestive of the avian paramyxovirus-1 infection (3, 4).

Paramyxovirus-1 infections have caused great losses in pigeons in Continental Europe, Great Britain (2) and the Sudan (4) and was reported in Egypt during the last few years (9).

The present PPMV-1 infection in Saudi Arabia showed a high degree of host specificity to pigeons. No other avian species were reported to be infected during the natural outbreak of the disease in pigeons. This was also the case elsewhere (4, 5). Such a unique host specificity for a paramyxovirus-1 was rather unusual (3). However, experimental infection of chickens through the natural routes with virulent PPMV-1 failed to produce overt clinical signs (3, 6).

Conclusion

It is rather difficult to speculate on the threat of this virus for domestic poultry. However, it might get adapted to chickens through natural passage. This should be borne in mind when handling pigeon outbreaks due to this virus in Saudi Arabia. Further classification studies on the virus are underway.

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A severe nervous disease struck fancy pigeons for the first time in Saudi Arabia during February-March. 1992. The morbidity rate was 60 % while the case fatality rate was 40 %. An avian paramyxovirus-1 was isolated from affected pigeons. The disease was reproduced experimentally in pigeons and the virus was reisolated and identified.

Key words : Pigeon - Avian paramyxovirus - Nervous system disease - Experimental infection - Epidemiology - Saudi Arabia.