OUTBREAK OF HIGHLY PATHOGENIC AVIAN INFLUENZA IN LOCAL CHICKENS IN NIGERIA

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ABSTRACT
Highly Pathogenic Avian Influenza (HPAI) is a highly contagious viral disease affecting the digestive, nervous, respiratory and/or reproductive systems of all domestic and wild birds. The outbreaks in local/backyard chickens were reported in 5 States (Jigawa, Kano, Nassarawa, Katsina, and Taraba). In this paper, we describe the clinical and pathological findings in local chickens during the outbreak of HPAI caused by H5N1 subtype in Nigeria and compared them with those reported in other parts of the world.

The disease in local chickens in Nigeria was found to show little or no clinical signs and gross lesions. This was possibly due to the fact that the birds died per acutely before signs or lesions could develop.

KEY WORDS:
Highly Pathogenic Avian Influenza, local chickens, Nigeria, clinical signs and lesions.

INTRODUCTION

Highly Pathogenic Avian Influenza is a viral disease affecting the digestive, nervous and respiratory systems of all domestic and wild birds that is characterized by respiratory, reproductive, digestive and/or nervous signs with high morbidity and mortality with an incubation period of few hours to few days. The disease affects all ages, but is more serious in the young.

HPAI was first reported in Italy 1878, South Africa 1961, USA 1971, Australia 1975, England 1979, Ireland 1983, Mexico 1994 and Pakistan 1994. In recent years HPAI has become topical in Asia including Peoples Republic of China 1996, Hong Kong 1997, 2001, 2002 and 2003, Cambodia, Indonesia, Japan, Malaysia, Republic of Korea, Laos, Taiwan, Thailand, Vietnam, Turkey and Romania 2005 (6). HPAI viruses are members of the family Orthomyxoviridae and genus Influenza A. The influenza viruses that constitute this family are classified into types A, B or C based on differences between their nucleoprotein and matrix protein antigens.

HPAI viruses belong to type A genus. Influenza viruses are further categorized into subtypes according to the antigens of the haemagglutinin (H) and neuraminidase (N) projections on their surfaces. There are 15 haemagglutinin subtypes and 9 neuraminidase subtypes of influenza A viruses. AI virus is infective for almost all commercial, domestic and wild avian species, Chickens and turkeys are highly susceptible to infection and clinical disease, and ducks and geese although

susceptible to infection with all AI virus strains, suffer clinical disease from only highly virulent strains. Pigs and humans are equally susceptible to infection by the HPAI virus while pigs serve as a potential mixing vehicle for re-assortment (7).

Reports of infection and mortality in domestic and wild cats have occurred in Thailand and more recently in Egypt. It may be pertinent to note that domestic ducks can be infected without showing clinical signs and may serve as a source of infection for domestic poultry. However, in Nigeria the reverse is the case, as duck were found to more susceptible and thus die more quickly resulting in the near absence of clinical signs, gross and microscopic lesions (6), thus complying with the reports from USA, where a high rate/percentage infection was reported in Mallard ducks (1, 2).

It should be noted that of recent mallard ducks have gained so much popularity in Nigerian villages, most likely because of their egg laying attributes, while geese are revered as ornamental/pet birds among the elites. Therefore the possibility that they may have a role in the source and transmission of AIV in Nigeria is very high. Spread of the disease into the country could be also through importation or smuggling of infected poultry and poultry products as well as ornamental and pet birds (4, 6).

Five months after the first outbreak was reported in Nigeria, HPAI involving local chickens were confirmed in 5 states of the Federation, namely, Jigawa, Kano, Katsina, Nasarawa, and Taraba states, as at June 30th 2006.

In this study we described the clinical signs, gross and microscopic lesions in affected local/indigenous chickens during the outbreak of HPAI caused by H5N1 subtype and submitted to the National Veterinary Research Institute, (NVRI) Vom during the 2006 HPAI outbreaks in Nigeria. In addition we assess the relatedness of the signs/ lesions observed in the Nigerian outbreaks with those reported in other parts of the world.

MATERIALS AND METHODS

History of the outbreak

On 27 January 2006, a suspected outbreak was reported in Janguza farm with a GPS of latitude 12° 0`N, longitude 8° 3`E in Kano metropolis. The infection was ravaging local chickens in both Kano and Jigawa States. Jigawa State has a GPS of latitude 12° 37` N and longitude 9° 24` E. Kano and Jigawa are in the North-western part of Nigeria and are located on same axis as Jaji, where the first outbreak in the country occurred. A total of 84,000 and 369,760 local birds were either culled and/or died as a result of the outbreak of avian influenza in Kano and Jigawa states respectively representing 2.38% and 8.42% of total poultry affected respectively (5).

More outbreaks were later reported from Katsina, Nassarawa and Taraba States of Nigeria. By 13th February, 2006, mortality of 500 was recorded in Katsina with a GPS of latitude 12° 59` N and longitude 7° 35` E; while an unspecified number was either culled or died. Nassarawa state (GPS latitude 8° 29` N, longitude 8° 30`E) reported the outbreak on the 17th February 2006, in Kokona LGA in a locality with about 301 local birds and 284 died. In Taraba state, the outbreak occurred in two locations (Wukari and Ibi LGAs with GPS latitude 7° 53` N, 8° 11`N and longitude 9° 46` E, 9° 44` E respectively). In one of the cases, the outbreak caused a loss of 251 birds within a period of 3 days out of an original population of 356. However, the infection spread to other parts of the

LGA as well as the neighbouring Ibi LGA with a cumulative mortality of over 264,000 local birds. As at July 2006, 5 States had reported positive outbreaks of the HPAI caused by H5N1 subtype as diagnosed by the (NVRI), Vom and confirmed by the reference laboratory in Padova, Italy.

Submission of samples from suspected flocks

During these outbreaks, suspected cases showing severe mortality with or without clinical signs of cyanosis and swelling of heads and eyelids were submitted to the NVRI, Vom for post mortem examination and collection of samples for virus isolation and other laboratory analysis. The carcasses were examined immediately on arrival.

Post Mortem Examination (PME)

The PME comprised macroscopic examination, especially of the head of the carcass. After removal of the skin, the abdominal cavity was opened and the trachea, heart and liver were evaluated. Subsequently, samples of the lung, trachea, spleen, liver and intestine were taken for virologic investigation. The alimentary tract was then removed and all organs in the abdominal cavity were evaluated. Tissues with gross pathological changes were then placed in 10% buffered formalin for histopathological examination.

Virus culture

Virus culture was carried out as described by (OIE, 2005). Nine days old chicken (9 day-old) embryonated eggs obtained from specific antibody negative (SAN) flocks were used for viral isolation. 0.2 ml of the supernatant (of 20% w/v tissue homogenate) was inoculated into five viable embryonated chickens eggs via the Allantoic cavity. The eggs were incubated at 37°c in a humidified incubator. They were candled daily and those with dead embryos were chilled at 4oc for 12-18 hrs, after which a qualitative analysis was carried out on the dead embryos to check for haemagglutination (HA) activity using 10% washed chicken red blood cells from SAN flocks as indicator. Allantoic fluid from eggs that showed positive for HA activity were harvested aseptically and tested for sterility by plating on blood agar and incubated at 37°c for 24 hrs (2, 6).

AGID Test

AGID test was carried out as described in OIE manual 2005. The supernatant was inactivated using 0.1% formaldehyde and used as the test antigen with standard avian influenza antigen and antisera obtained from national Veterinary Service Laboratory (NVSL) Iowa, USA. The agar gel plates were incubated at 37°c in a humidified incubator and examined after 24 hrs. Positive specimen showed Precipitating lines of identity to avian influenza type A group specific Ribonucleic Protein (RNP) antigen in the AGID test (6).

Haemagglutination Inhibition test (HI)

Alpha haemagglutination Inhibition test was carried with serially diluted positive Allantoic fluid using standard procedures (OIE, 2005). Newcastle disease monospecific antisera showed no inhibition of the test antigen.

RESULTS

Clinical signs and lesions

The outbreaks involved about 785,571 local chickens in five states from the North Central and North Western parts of Nigeria. The clinical signs observed in this case were swelling of the head, unsteady gait and massive mortality affecting all ages of birds, this was inconsistent and least characteristic with what has been reported in literature such as depression and cyanotic combs/wattles and affected only adult birds, yellowish green diarrhoea,

drooling seromucous secretions from the beaks and nostrils, torticolis, hyperaemia of shanks and swelling of the foot pad as was the cases involving commercial layers in Nigeria (4). There were severe congestion and haemorrhages of nasal sinuses while the tracheae had marked congestion of serosal and mucosal surfaces. The lungs were congested and often haemorrhagic.

Microscopic lesions observed include vascular disturbances leading to exudates and/or haemorrhages of lungs and wattles. In a few cases pulmonary vessels were engorged with red blood cells and heterophils with evidence of pulmonary vessel damage and haemorrhage. The gross and microscopic lesions were found to be different from what have been reported elsewhere. In which there were little or no clinical signs or lesions, possibly due to the birds dying too quickly before signs or lesions develop.

In this study we described the clinical signs, gross and microscopic lesions in local chickens submitted to the National Veterinary Research Institute, (NVRI) Vom and confirmed to be positive for AI during the 2006 outbreak in Nigeria. In addition we assess the relatedness of the signs/ lesions observed in the Nigeria outbreak with those reported in other parts of the world.

Discussion

The presence of swollen eyelids and cervical/neck regions; cyanosis of combs, wattles and featherless areas of the head were infrequently encountered as well as sudden and increasing high mortalities. However, in the outbreaks involving local chickens in China and India respiratory rales, sneezing, coughing, serous discharge from nostrils and beaks as well as paralysis, diarrhoea, depression were reported (3).

This might be attributed to the husbandr systems, complete or near absence of an form of Veterinary health care in thes types of chickens in Nigeria, thu exposing them to higher risk in the ever of exposure to the HPAI virus. Th mortality trend in the local chickens wa more severe in the Taraba Stat outbreak, which involved local/backyar poultry and the entire flock made up c approximately 264,000 birds were wipe out in about 2 weeks.

The gross lesions described in literatu are made up of a variety of congestiv haemorrhagic, transudative an necrobiotic changes in both laying her and local chickens in China and Indi (8). The internal organs (spleen, live kidney, pancreas and intestine) wer enlarged, haemorrhagic and blotched b necrotic foci. Furthermore, pancreatiti tracheitis, pneumonia and haemorrhag caecal tonsils were observed, as well a the petechial haemorrhages on th epicardium, abdominal fats an occasionally in the muscles (breast an thigh).

The gross lesions that were consistentl seen in positive cases of HPAI involvin local chickens in this study wer congestion/haemorrhages of sinuse presence of tracheitis, congestion oedema of the lungs. The gross lesions local flocks in Nigeria were inconsiste with what was reported in local chicker in China and India (1; 4; and 8).

It is possible that absence of some these gross and microscopic lesions our local chickens in the case of HPA may be because the birds die ver quickly before clinical signs/ lesion could develop (5). In conclusion, ou study indicates that the clinical sign gross and microscopic lesions in infecte local chickens are not completely simil to the descriptions in literature.

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