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CLINICO-PATHOLOGICAL AND HUSBANDRY FEATURES ASSOCIATED WITH THE MAIDEN DIAGNOSIS OF AVIAN INFLUENZA IN NIGERIA

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SUMMARY

Referring to skewed trend in the appearance of avian influenza in Africa, this presentation describes the enabling clues in the maiden diagnosis of the epidemic in Nigeria. The investigations revealed high and sudden mortality in all poultry species and ages in the farm and up to 100% within two weeks. The clinical signs involved respiratory and digestive systems in all ages but also the nervous system in young stock. In the chickens, there was extensive cyanosis or haemorrhages and other haemo-vascular lesions in the combs, wattles, the shanks and trachea. There was congestion and necrosis of the liver and spleen; intestinal ecchymoses, oophoritis, swollen and congested ceacal tonsils. Although, the stereotype diagnoses for such severe outbreak, mortality and lesions should include Newcastle disease (ND) and fowl cholera, the laboratory findings of protective ND HI antibody titres (mean 4.4 $\log_2$) and absence of *Pasteurella multocida*, served to exclude such other fulminant diseases from the diagnosis. The field diagnosis of avian influenza was based on the strong husbandry, epidemiological, clinical and pathological features of highly pathogenic avian influenza (HPAI).The diagnosis was subsequently confirmed at reference laboratories as H5N1 and HPAI. It illustrates the complications from defective biosecuriy practices in poultry health.

KEYWORDS: Clinical, pathological, diagnosis, maiden, avian influenza, Nigeria

INTRODUCTION

The trends in the current Avian influenza (AI) epidemic started with reports of outbreaks in Southeast Asian poultry towards the end of 2003 (WHO, 2005a). It continued to spread from Asia westward to the Middle East and reached eastern Europe with outbreaks in Croatia and Romania in 2005 (WHO, 2005b). At a point when the first outbreak was reported in Turkey, the proximity to Africa kindled a grave possibility that the AI epidemic might soon move across the Mediterranean into Africa. Although, it was difficult to know the potential first port of call in Africa, it was logical to speculate on any of the North African countries. It was therefore a real surprise that the first African country to report the ingression of AI was Nigeria, in West Africa.

This article presents the vital clinical and pathological clues in the signalment of AI as well as the epidemiologic and husbandry features which accompanied the first field diagnosis of AI in Nigeria and Africa; as a facilitator to diagnosis in similar circumstances, worldwide.

CASE HISTORY

On Saturday, 14th January, 2006, carcasses from a farm located in the outskirts of Kaduna in North of Nigeria, were presented to Ahmadu Bello University Veterinary Teaching Hospital with the history of sudden and high mortality in the farm flocks. The losses which started earlier on in the week had claimed about 6,000 birds from a total farm stock of nearly 58,800 birds. The farmer reportedly observed difficulty in breathing, sneezing, greenish-yellow diarrhoea and bleeding from nostrils in the affected birds.

With this alarming mortality and case history, it became necessary to conduct a careful and detailed diagnostic investigation of the flocks, to establish the epidemiological, clinical and pathological features as well as the housing and management factors involved in the case and farm.

Flocks and epidemiological data

The farm's poultry stock included layers, broilers and breeder chickens mainly, but also some turkeys, geese and ostriches. All these four species were housed on the same site, intensively for the chickens and turkeys but on extensive range for the geese and ostriches. The details of stock numbers, ages, and housing are shown in Table I, which reflects the multiple species, ages and cumulative mortality on the farm. Thus the chickens consisted of pullets, broilers, layers and breeders while the three other species were mainly adults of unspecified ages.

The stocks were initially acquired and placed on site in a multiplicity of stages, like; day old pullets and broilers, point of lay (POL) hens and as point of lay breeders housed separately but in contiguous pens. Some recent batches of POL hens and pullets placed 10, 5 and 3 weeks earlier respectively were among the flocks in the front line of the outbreak, which was noticed 2 weeks after the placement of the last group.

Farm records were defective or totally lacking on precise flock size, ages, production, medication, vaccination and biosecurity. Most farm data were therefore based on approximates from inquires. As at the time of visit on the 6th day of outbreak, morbidity had extended to all species and all flocks with a maximum of 100% mortality in turkeys; over 70.5% in chickens, 50% in geese and 4.6% (the site minimum) in the ostriches.

Clinical and pathological findings

A visit to the farm revealed that systemic clinical signs occurred extensively in the chicken flocks and initially affected the 17-35-week-old birds in cages and subsequently in all ages, which ranged from 2 weeks to 50 weeks. The mean rectal temperature from selected chickens was 43 ± 1.0°C. Thus, apart from general non-specific symptoms like weakness, somnolence and sternal or lateral recumbency in the chickens; there were cases of brownish, yellowish-white or greenish diarrhoea; dyspnoea, moist rales, sneezing and coughing. In the younger stocks aged between 2 to 9 weeks, stretching of the neck, convulsion and torticollis (Fig. 1) were observed.

Many of the affected mature chickens also

presented oedema of the head and facial region with extensive cyanosis or hemorrhagic patches of the combs and wattles (Fig. 2). The shanks and toes were congested and dark red in some cases.

Among the other species of poultry on the farm, the disease was rather fulminant in the turkeys. The signs included recumbency and swollen paranasal sinuses. All 40 turkeys on the farm died within one week. Recumbency, unsteady gait, backward movement, torticollis, yellowish diarrhoea and serous nasal discharges were observed in the ostriches. Apart from these clinical changes, the carcasses were generally in good bodily conditions.

Gross pathology

Although the chicken carcasses were in good bodily frames, most of the external appendages and the viscera bore gross lesions. The combs and wattles were either generally cyanosed and/or haemorrhagic in some. The shanks and toes were congested and darkened. The respiratory system presented with catarrhal tracheitis in all carcasses, cloudy and diphtheritic airsacculitis in some while the lungs in the chicken carcasses examined in the first two days were grossly normal. The liver, spleen and kidneys in each carcass were enlarged, congested or diffusely necrotic. In one carcass, the liver was fatty and friable. This carcass was also egg-bound and with perigastric petechiae and fat. Ecchymotic patches up to 2 cm in diameter were noticed on the serosa of the intestines. There was catarrhal enteritis, especially in the duodenum; while the caecal tonsils were thickened and haemorrhagic (Fig. 3).

At the later stages in the outbreak, carcasses presented with milder effects on the combs and wattles. There were haemorrahages on proventricular mucosa and margin (Fig. 4), oophoritis and egg yolk peritonitis.

ND HI titres

The haemagglutination inhibition (HI) titres for ND antibodies in the various age groups of chicken ranged from 2 $\log_2$ to 8 $\log_2$, with a mean of 4.4 $\log_2$ and a modal titre of 5 $\log_2$. (Table II). These values are indicative of adequate ND immunity in most of the chickens in the flocks.

DIAGNOSIS AND DISCUSSION

A postmortem (PM) and field diagnosis of AI was established in this case on presentation, based on very strong epidemiologic, clinical and postmortem findings. Thus, the sudden onset and high mortality in varied species and ages, typical lesions especially on the combs, wattles and shanks together with the haemo-vascular lesions in most visceral organs provided the strong PM diagnostic clues for AI in this case. Although Newcastle disease, in its most fulminating (VVND) form could share some of these diagnostic features in fully susceptible and in all probability unvaccinated flock, it would more usually do so to a lesser extent and definitely not in the range of species and ages involved in this case. Indeed, the ND antibody HI titres from the affected chicken flocks reflected no or negligible ND virus activity in the flocks at the time. The ND antibody mean titre of 4.4 ± 1.6 $\log_2$ is above the protective baseline for ND of 3.0 $\log_2$ recommended by the FAO (Allan *et al.*, 1978) as in Table II. Similarly, the absence of *P. multocida* in the bacteriology, helped to exclude these two other major causes of sudden and high mortality in the differential diagnosis.

Finally, these findings when placed in the background of the globally active flu epidemic, facilitated and lent a strong ranking to the first field diagnosis of AI in this case. Previous records of AI in Nigeria were either inconclusive in nature or based on limited serology which did not touch on the pathotypes and the clinical disease (Adeniyi *et al.*, 1993; Owoade *et al.*, 2002).

The poor husbandry standards on this farm were reflected in the examples of simultaneous or contiguous stocking of four different poultry species; multi-age site and the absence of useful farm records. It is obvious that poor farm management and low biosecurity standards as represented in unrestricted movement of people and materials as well as the absence of foot dips must have contributed as epidemiologic propellants to the outbreak. It is therefore logical to expect that other secondary

infectious agents, stress and even nutritional problems like the fatty liver in one of the carcasses examined in the initial presentation of the case, must have been partly responsible for the very high mortality of 70.5% and 100% in chickens and turkeys respectively on the farm.

It is noteworthy that, most of the carcasses examined at the early per-acute/acute phases of the disease did not present pulmonary lesions. This was interpreted as a feature of such a primarily upper respiratory disease like AI. Similarly, the nervous involvement in the young stock could be suggestive of differential age susuceptibilty. The findings from this outbreak, especially the very high morbidity, rapid and very high mortality are those to be associated with highly pathogenic avian influenza (HPAI). The subsequent virological works in the National Veterinary Research Institute of Nigeria (NVRI) and at the FAO reference laboratory in Italy, eventually confirmed the isolation of H5N1 virus and the HPAI status of the outbreak, three weeks later, in March, 2006. Thus, the maiden diagnosis of avian influenza, which could have otherwise proved elusive, was indeed correctly and strongly diagnosed clinically on the 14th of January in Nigeria and subsequently confirmed by laboratory findings.

This article is therefore a useful contribution and practical re-emphasis of the field diagnostic criteria for AI and contributory husbandry factors in AI outbreak.

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TABLE I: Epidemiological data of the first outbreak of AI in a commercial farm in northern Nigeria

Chickens	2 weeks (pullets)	Deep litter	3,621	(Unknown)	NWN
	9 weeks (broilers)	Deep litter	3.000	(40)	SWN
	Growers (broiler breeders)	Deep litter	4,000	(95)	SWN
	17-35 weeks (layers)	Battery cage	30,000	(70)	NWN
	45-50 weeks (layers)	Battery cage	18,000	(85)	SWN
Turkeys	Adults*	Deep litter	40	(100)	Unknown
Geese	Adults*	Range	4	(50)	Unknown
Ostriches	Chicks*	Range	9	(33)	Unknown
	Juveniles*	Range	3	(100)	Unknown
	Adults*	Range	118	(0)	Unknown
Total			58,795	(>70.3)	

* = Specific age unknown; SWN=South West Nigeria, NWN=North West Nigeria

TABLE II: Haemagglutination inhibition antibody titres of Newcastle Disea

Titre (in $\log_2$)	2	3	4	5	6	7	8	
Age (in weeks) and type of bird				Number of birds with titre				
2 pullets	1		2		1		1	
9 broilers			1	3				
Growers breeders		2	1					
17-35 layers	2	3		2				
45-50 layers			2	4	1		1	
Total	3	5	6	9	2	0	2	
Mean titre							4.4	1.6

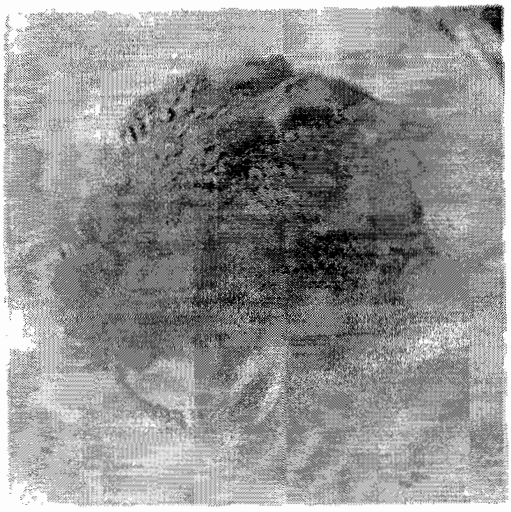

Plate 1: Torticollis in a nine week old chicken

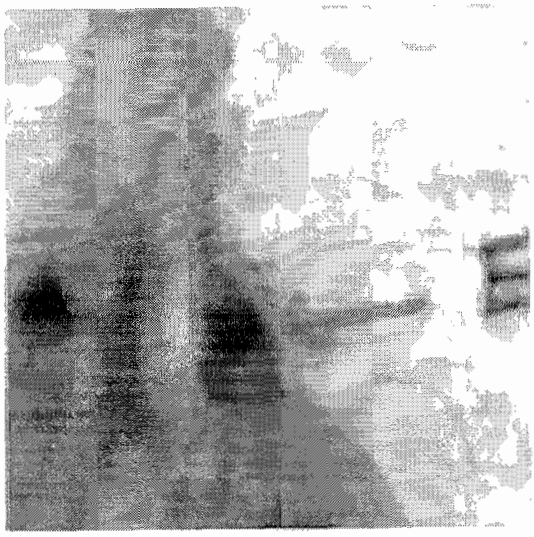

Plate 3: Haemorrhagic (E) caecal tonsils

Plate 2: Cyanosis of the comb and wattles

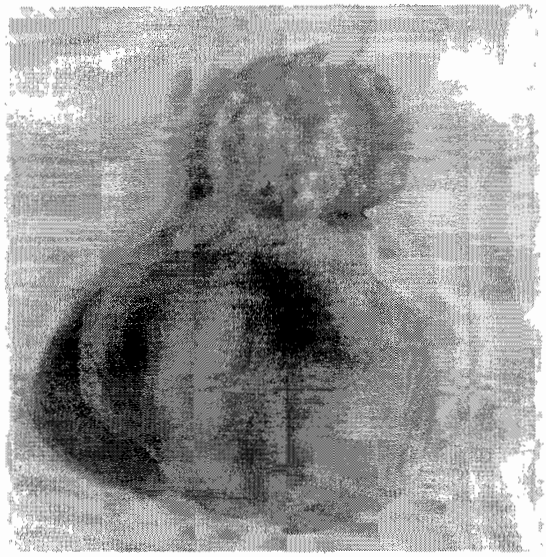

Plate 4: Haemorrhages in the proventicular/gizzard junction

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