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SEROBIOCHEMICAL CHANGES ASSOCIATED WITH HIGHLY PATHOGENIC AVIAN INFLUENZA IN LAYER CHICKENS IN JOS, PLATEAU STATE, NIGERIA

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Abstract

Aim: To assess some serobiochemical changes in layer chickens affected with H5N1 Highly Pathogenic Avian Influenza (HPAI) virus in Plateau State, Nigeria.

Methods: Five sera samples were collected from ten infected commercial farms (fifty from all the infected layer farms). Five sera samples from non-infected chickens (1 from each layer farm that was free from infection) were used as control. Serum Aspartate aminotransferase (AST), Serum Alanine aminotransferase (ALT), total protein, concentration and creatinine concentrations were determined using kits.

Results: There was a significant ($P<0.01$) increase in levels of Serum Aspartate aminotransferase (AST), Serum Alanine aminotransferase (ALT) and creatinine concentrations and significant decrease in urea, total albumin and protein concentrations in sera of all affected chickens which is suggestive of liver damage.

Conclusion: These changes confirm the evidence of liver and kidney damage, and could be responsible for the high morbidity and mortality usually associated with this disease.

Key Words: Highly Pathogenic Avian Influenza, Layer Chickens

INTRODUCTION

Influenza A viruses, members of the Orthomixoviridaefamily, infects a variety of animals, including wild domestic birds, including humans, pigs, horses and sea mammals. They are differentiated from type B and C influenza viruses on the basis of the major protein antigen, the nucleoprotein (NP) and the matrix (M1) proteins (Munster and Olsen (2009). Avian influenza (AI) viruses can be categorized into subtypes and pathotypes. The subtype distinction is based on serological typing of the two glycoproteins: the haemagglutinin (HA) and the neuraminidase (N). Sixteen antigenically different haemagglutinins (H1 to H16) and nine antigenically different neuraminidases (N1 to N9) are now recognized and all of them have been isolated in wild birds (Alexander, 2007; Swayne and Pantin-Jackwood, 2008). The pathotypes are based on the ability to produce disease and death in chickens (Gallus domesticus). Some of the H5 and H7 subtypes, carrying multiple basic amino acids adjacent to the haemagglutinin cleavage sites, are responsible for severe and acute disease with high mortality in poultry, namely Highly Pathogenic Avian Influenza (HPAI); while Low Pathogenic Avian Influenza (LPAI) viruses usually produce respiratory disease and decreased egg production in all types of poultry species.(Brown et al., 2006). Wild aquatic birds especially ducks, geese and swans as well as gulls shorebirds, auks, terns and are natural reservoirs of LPAI viruses (Alexander, 2007). The disease was reported as an infectious disease of chickens since 1878 (Bragstad, 2007). Nigeria reported its first Highly Pathogenic Avian Influenza (HPAI) outbreak by February, 2006, in Sambawa farms in Kaduna State, North Western Nigeria. Subsequently, the disease became widespread affecting several States in the country (OIE, 2008). In the earlier outbreak; morbidity and mortality ranged from 90-100% in affected farms (Najat, et al., (2006). The causative agent then was identified as HPAI, subtype H5N1. However, the current outbreak was first reported in Kano State, Nigeria, in the North Western part of the country. The clinical signs in the affected

chickens were inappetance sneezing, coughing, ruffled feathers, lacrimation, nasal discharge, edema surrounding the eyes, conjunctivitis, very high mortality within a very short time of onset of the disease, cyanosis of combs and wattles as well as breast and abdominal muscles, watery whitish diarrhea and sometimes nervous signs thus mimicking the clinical signs were noticed in the previous outbreak. Gross lesions were varied, but mostly showed severely hyperemic and congested trachea, petechial hemorrhages in abdominal fat, sciatic nerves and brain capillaries, friable and congested liver, nephritis, enteritis and ecchymotic hemorrhages at proventricular junction of the gizzard, and inner aspect of the kneel facia and bone (Kumbish 2008).

MATERIALS AND METHODS

Source of Samples: Some commercial layer farms in Jos, Plateau State where outbreaks of avian influenza occurred were investigated. The causative agent was characterized as HPAI/H5N1 virus.

Study location: The studied farms were located in Jos, Central geographical zone of Nigeria, designated as infected commercial layer farm 1, 2 3, 4 and 5 respectively.

Sampling: Five sera samples were collected from ten infected chicken farms (totaling fifty from all the infected layer farms). Five sera samples from non-infected chickens (1 from each layer farm which was free from infection) were used as control. Sample collection was done in a humane manner by a Veterinarian, using the wing vein. All infected and control birds were fed commercial ration. Source of water were all from boreholes and sometimes from well water or both.

Laboratory Analysis: Total protein, Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), albumin, urea and creatinine concentrations were determined using Randox kits according to manufacturer's instructions.

Statistical Analysis: The results were expressed as mean ± the standard deviation. Student's t-test was used to compare the data from infected chickens and controls.

RESULTS

There was a significant elevation ($P<0.01$) in AST, ALT in sera of the ten infected layers. Concentration of total protein and albumin were however, significantly decreased in all chickens in the infected layer farms. On the other hand, ten infected layer farms presented decreased urea concentrations (table 1) whereas the creatinine level was significantly increased in sera of four infected layer chicken 1, 2, 5, and 8.

Table 1a: The mean and standard deviation of serum biochemical parameters in avian influenza infected layers chickens

Parameters	Control layer	Infected Layer 1	Infected Layer 2	Infected Layer 3
SGOT (u/l)	67.9 ± 21.6	123 ± 16.5	113.8 ±29.5a	120 ±14.7
SGPT (u/l)	4.3 ± 0.8	23.3±13	24.1 ±1.3	22.3 ±2.4
TP gm/100ml	6.99 ± 0.3	6.4 ± 0.5	5.9 ± 0	46.2 ± 0.3
Albgm/100ml	3.1 ± 0.3	2.7 ± 0.2	2.3 ± 0.4	2.4 ± 0.2
Ur mg/100ml	24.1 ± 2	1 16.9 ± 1.3	25.0 ± 2.3	16.6 ± 1.5
Crt mg/100ml	1.3 ± 0.1	1.3 ± 0.8	1.4 ± 0.6	1.03 ± 0.9c

a: high significant difference ($P<0.01$). b: significant difference ($P<0.01$). c: not significantly different ($P>0.01$)

Table 1b: The mean and standard deviation of serum biochemical parameters in avian influenza infected layers chickens

Parameters	Control layer	Infected Layer 4	Infected Layer 5	Infected Layer 6
SGOT (u/l)	66.9 ± 21.5	122 ± 15.51	10.5 ±27.4a	126 ±15.6
SGPT (u/l)	4.1 ± 0.7	25.2±11	26.1 ±17	23.2 ±2.2
TP gm/100ml	7.99 ± 0.2	6.2 ± 07	6.0 ± 0.3	6.0 ± 0.2
Albgm/100ml	3.0 ± 0.2	2.9 ± 0.4	3.2 ± 0.6	2.2 ± 0.1
Ur mg/100ml	26.3 ± 3.1	18.0 ± 1.6	23.4 ± 1.9	17.4 ± 2.0
Crt mg/100ml	1.1 ± 0.01	1.1 ± 0.3	1.6 ± 0.4	1.01 ± 0.6c

Table 1c: The mean and standard deviation of serum biochemical parameters in avian influenza infected layers chickens

Parameters	Control layer	Infected Layer 7	Infected Layer 8	Infected Layer 9
SGOT (u/l)	65.9 ± 20.4	123± 14.21	10.4 ±26.4a	119 ±12.9
SGPT (u/l)	5.0 ± 1.0	21.9±16	25.0 ±1.6	24.1 ±2.7
TP gm/100ml	7.00 ± 0.1	6.1 ± 0.6	6.2 ± 0.6	6.4 ± 0.2
Albgm/100ml	3.1 ± 0.9	2.9 ± 0.1	3.4 ± 0.2	2.8 ± 0.4
Ur mg/100ml	21.9 ± 3.0	14.9 ± 1.2	27.1 ± 2.9	15.5 ± 1.3
Crt mg/100ml	1.2 ± 0.12	1.1 ± 0.4	1.5 ± 0.4	1.01 ± 0.7c

Table 1d: The mean and standard deviation of serum biochemical parameters in avian influenza infected layers chickens

Parameters	Control layer	Infected Layer 10
SGOT (u/l)	62.7 ± 19.9	124± 13.0
SGPT (u/l)	6.1 ± 2.0	23.7±19
TP gm/100ml	7.10 ± 0.2	6.5 ± 0.4
Albgm/100ml	3.4 ± 1.0	3.1 ± 0.5
Ur mg/100ml	22.4 ± 2.8	5.0 ± 1.1
Crt mg/100ml	1.1 ± 0.10	1.1 ± 0.1

Key: ur = Urea; crt = Creatinine

DISCUSSION

The results of the serum biochemical parameters obtained in this study strongly suggest biochemical evidence of liver and kidney damage of chickens infected with highly pathogenic strains of avian influenza virus. This might also partly be responsible for the very high mortality associated with this disease during the outbreak as a result of the rapid failure of these two important organs. These findings are in agreement with findings of Swayne and Halverson (2006). as well as that of previous studies conducted in chickens (Beato et al, 2009); Beato et al., (2009), that HPAI is associated with ecchymotic and/or pin-point haemorrhages, necrotic and edematous lesions in visceral organs especially the above mentioned organs. Elevation of serum AST and ALT suggested pathologic involvement of liver and this was supported by the macroscopic lesions observed in infected chickens. It is a known fact that liver is considered to be the major source of plasma proteins, damage to this organ will lead to decreased total protein. These changes were correlated with the changes in serum enzymes originating in the liver (Timm, et al., 2006; Capua, 2008). On the other hand, the reduction in albumin level was due to damage in the liver and kidneys by the highly pathogenic avian influenza virus. This was supported by the findings of Munster and Olsen (2009) who reported that the decreased serum albumin concentration had been correlated with liver necrosis and increased filtration and excretion by the kidneys. The decreased level of protein and albumin was also reported in chickens infected with velogenic strains of Newcastle Disease (Timm et al, 2006), and hypoalbuminemia has been reported in chickens in exudative process which occurs in vitamin E deficiency (Timm et al, 2006). In the same vein Beato et al., (2007) reported increased AST and uric acid in chickens with nephritis. Another possibility of decreased total protein is due to reduction in food intake as a result of resultant aneroxia which is one of the earlier mentioned clinical presentations of the disease or protein deficiency in the ration fed the birds. This study presents the first report of some of the Serobiochemical parameters related to liver and kidney functions during the course of avian influenza infection in Jos, Plateau State, Nigeria.

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