

ISSN 1682-8356
ansinet.org/ijps



INTERNATIONAL JOURNAL OF
POULTRY SCIENCE

ANSI*net*

308 Lasani Town, Sargodha Road, Faisalabad - Pakistan
Mob: +92 300 3008585, Fax: +92 41 8815544
E-mail: editorijps@gmail.com

Outbreak and Persistence of Mareks Disease in Batches of Birds Reared in a Poultry Farm Located in Nsukka, South East Nigeria

E.C. Okwor and D.C. Eze

Department of Veterinary Pathology and Microbiology, University of Nigeria, Nsukka, Nigeria

Abstract: Marek's Disease (MD) is an important viral disease that has been described in Nigeria. Though poultry farmers may not be familiar with the disease when compared to other common diseases like Newcastle Disease (ND) and Infections Bursal Disease (IBD) sporadic outbreaks of this disease occurs in poultry farms resulting in significant economic losses. This work investigated an outbreak and persistence of MD in batches of birds reared in a poultry farm in Nsukka, South East Nigeria between 2001 and 2009. The farm maintains many pens situated close to each other and introduces birds of different ages thereby housing different batches of birds throughout the year. The first outbreak occurred in a batch of 300 brown pullets at the age of 12 weeks. Infection occurred in all the subsequent batches of pullets introduced into the farm within the study period. Batches of broilers introduced into the farm and kept up to six weeks and above had the infection. Those that were sold before six weeks of age as brooded birds did not show signs of MD. Cockerels that were kept and reared to maturity showed signs of the disease while those that were reared and sold as brooded birds at four-five weeks did not show signs of the disease. In most cases, infection among the flock (morbidity) varied ranging from 5-15% with few acute cases up to 25%. We therefore suggested an 'all-in-all-out' system in case of outbreaks of the disease with reasonable period or interval between each stock.

Key words: Mareks disease, herpes virus, contamination to the environment

INTRODUCTION

Marek's Disease (MD) is the most common of the lymphoproliferative diseases of chickens characterized by mononuclear infiltrations of peripheral nerves, gonads, iris, various visceral organs, muscles and skin (Calnek and Witter, 1997; Witter *et al.*, 2005). It is caused by a cell associated herpes virus belonging to the family Herpesviridae (Witter *et al.*, 1969). Witter (2001) observed that MD is a ubiquitous infection of poultry throughout the world and outbreaks in farms resulting in significant losses are very common (Payne, 1985; Buscaglia *et al.*, 2004). Infection is mostly by inhalation of infectious dander and poultry dust (Abdul-Careem *et al.*, 2009). Epithelial cells in the keratinized layer of feather follicles replicate full infectious virus (Calnek *et al.*, 1970). These cells serve as a source of contamination to the environment. The feather follicle cells are the most important source of infection and are responsible for the infectivity of dander, poultry house dust and litter and the vaccine virus genome load in feather tips have been correlated with protection (Baigent *et al.*, 2007). The infectivity of these materials can last for at least one year at room temperature (Witter and Schat, 2003). In Nigeria, incidence of MD has been reported in the Northern States (Nawathe *et al.*, 1978) and in many parts of South western States (Fatumbi

and Adene, 1986; Oni and Owoade, 2009; Olabode, 2009). This has resulted in huge economic losses. Low environmental hygiene, little or no biosecurity measures in many farms with local chicken population moving about on a free range has helped in the maintenance of the disease in infected farms and also in the spread of the disease from farm to farm. Environmental stress and lack of coordinated vaccination regimens in hatcheries at day old also contributes substantially to the incidence of MD in many farms in Nigeria. In developed countries, serious vaccination campaigns are carried out and this has helped to reduced the incidence and economic losses associated with the diseases (Purchase, 1985). This paper reports the incidence of MD in the South eastern part of Nigeria, an eight-year period.

MATERIALS AND METHODS

An outbreak of MD was reported in a farm located in Nsukka, South East Nigeria in March, 2001. On presentation, the flock history was taken. The farm was visited and the flock was examined. Clinical signs were observed and recorded. Post Mortem (PM) examinations were regularly conducted on dead birds. MD was diagnosed based on the clinical observations, post mortem lesion and Agar Gel Precipitation Test (AGPT). AGPT was carried out according to the method described by Sharing (1989) using MD viral antigen

Table 1: Age at onset, clinical manifestations and gross lesion in pullet, cockerels and broilers affected with MD

	Types of bird		
	Pullets	Broilers	Cockerels
Age of onset of disease (weeks)	10-12	4-6	10-12
Clinical disease	Mostly classical MD	Mostly acute MD	Mostly classical MD but less severe as compared to pullets
Gross lesions	Mostly classical lesions present	Predominantly enlarged liver and spleen	Mostly classical lesions present

store in liquid nitrogen which was requested and collected through a hatchery in Nigeria. The nature of the disease was monitored in all the batches of birds reared in the farm within the study period of 2001-2009. The observations were recorded.

RESULTS

Outbreaks in pullets: The first outbreak occurred in March, 2001 in a batch of 300 brown shaver pullets brought at day old. These birds were reared on deep litter and were vaccinated against Newcastle Disease (ND), Infections Bursal Disease (IBD) and Fowlpox (FP). Outbreak occurred in all other subsequent batches (25 batches) of pullets numbering between 500 - 1500 that were reared in the farm between this period and the end of 2009.

Age at onset: Onset of disease was mostly between 10 and 12 weeks of age in all the batches examined.

Clinical disease: The clinical disease ranged from classical MD in most of the batches to acute MD in few of the batches. The clinical signs observed were reduction in feed and water consumption during the acute clinical period of the disease, greenish diarrhea, gray eyes and weight loss; morbidity and mortality were low except in acute cases where it was up to 25%. There was severe atrophy of the breast and thigh muscles in classical cases. Morbidity and mortality reduced as the bird got older with occasional loss of weight and death when the birds had advanced into lay.

Gross lesions: The major lesions were enlargement and tumours of the liver, enlargement of the spleen was seen in most cases examined. The kidneys were enlarged in many cases. Very few classical cases showed enlarged sciatic nerves as well as enlargement and tumors of the feather follicles. The carcasses examined were dehydrated.

Outbreak in broilers: Thirty two batches of 200-500 broilers were reared in the farm within the study period. Nine sets of these broilers were reared and sold as matured birds while others were reared and sold as brooded birds between 3-4 weeks of age. There was no outbreak of disease noticed in the broilers that were sold as brooded birds. Outbreaks occurred in some of the batches that were reared to maturity.

Age of onset: Disease was noticed in broilers at 4-6 weeks of age.

Clinical disease: All the cases seen in the broilers were acute MD with mortality up to 25%. The clinical signs were reduction in feed and water consumption and soiled vents. Many of the affected birds died.

Gross lesion: The common gross lesion seen in the broilers were enlargement of the liver and spleen. There was soiled vents and dehydration.

Outbreak in cockerels: Twenty two batches of 500-1500 white cockerel were reared in the farm within the study period. Out of this number of batches, only two batches were raised to maturity. The rest of the batches were reared and sold as brooded birds between 4-5 weeks of age. No clinical disease was observed in the birds reared and sold as brooded birds. Disease was observed in the two batches that were raised to maturity.

Age at onset: The disease in the 2 batches of the cockerels was observed between 10-12 weeks of age.

Clinical disease: This was as described for pullets. However, the severity was lower in the cockerel especially as they approached maturity.

Gross lesions: The gross lesions were also as was described for the pullets. Table 1 summarizes the results stated above.

DISCUSSION

This investigation examined the outbreak, nature of disease and persistence of MD in a farm for an 8 year period. The results showed a persistent infection over this period and most of the batches of birds that were brought in after the first outbreak were affected. This persistence and spread of infection in the farm was in agreement with the observations of Calnek and Witter (1997) and Pagne (1999) as per infection and persistence of MD in infected farms. Witter *et al.* (1971) noted that infection can persist indefinitely in infected farm. Virus is associated with feather and infectious dander from chickens and these are the most important source of infection that is responsible for the infectivity of dander, poultry house and litter. Virus in feather follicles and dander can remain infectious for several months or

years depending on the ambient temperature (Calnek, 1980). Many apparently healthy birds are carriers of the infection and secrete the virus in desquamated feather follicles; epithelial cells dander, oral, nasal and tracheal secretions for transmission to susceptible birds (Kenzy and Cho, 1969). The farm under study brought in batches of birds at regular interval. Moreover, the sanitary and biosecurity measures that were in place were not of standard and this may have contributed in perpetuating the infection.

The age at which the clinical signs were observed was earlier in the broilers than in the pullets and cockerels. It is possible that these birds may have picked up these infections early in life but showed clinical signs later and the incubation period was shorter in the broilers than in the other two. It is difficult to determine the incubation period of the disease under field conditions (Calnek and Witter, 1997). Under experimental conditions, the incubation period is rather well established with birds inoculated at day old excreting the virus beginning at 2 weeks with maximal shedding occurring between the 3rd and 5th week (Witter, 1972; Calnek, 1980).

Factors that can affect the onset of infection and the incidence of disease include, virus strain, dosage at infection, maternal antibody status, route of infection, age at infection, genetic strain or breed of the bird and the sex of the host (Witter *et al.*, 1973; Calnek and Witter, 1997). The fast growth rate of broilers may explain why the incubation period was shorter than that observed with pullets and cockerels. Therefore, development of lesions and tumor may occur at a faster rate in broilers than in pullets and cockerels. This may also explain why the cases in broilers were mostly acute. It could also be that pullets and cockerels are more resistant than broiler. Purchase (1985) noted that clinical signs in pullets may not appear until 16-20 weeks and sometimes may be delayed up to 24-30 weeks. Most lesions in this study were seen in the pullets and cockerels because they had the classical form of the disease which is a more chronic form. This allowed for the development of the lesions. Acute MD may kill birds before most of the gross lesions become prominent. As observed, the signs and lesions of MD were more severe in pullets than in the cockerel. This observation was in agreement with that made by Pagne (1999) who observed that males tend to be more resistant to MD than females. There were different levels of severity even within the same breed of birds and this may have to do with the genetic constitution of the birds (Calnek and Witter, 1997). The immune status of the different batches of birds when they were introduced into the farm coupled with environmental stress at the particular period of the year can also contribute substantially in the development of the disease. Freeman *et al.* (1984) noted environmental stress as an important factor affecting the incidence and disease production. The birds were

brought in at different periods of the year with different weather or climatic condition. The different periods of the year may have contributed in the clinical manifestations of the disease in the different batches.

Conclusion: We therefore report the incidence or outbreak and persistence of MD in a farm in Nsukka South East Nigeria which is the tropical region. The disease persisted in the farm throughout the period of study. We recommend an all-in-all out system with long periods before stocking as a way of controlling outbreaks MD in infected farms. Continuous stocking without adequate measures to control dust may perpetual the infection. The farms should be thoroughly disinfected before stocking. Attempts should also be made to procure resistance breeds; moreover, the birds should be vaccinated against MD in the hatchery.

REFERENCES

- Abdul-Careem, M.F., K. Haq, S. Shanmuganathan, L.R. Read, K.A. Schat, M. Heidari and S. Sharif, 2009. Induction of innate host responses in the lungs of chickens following infection with a very virulent strain of Marek's disease virus. *Virology*, 393: 250-257.
- Baigent, S.J., L.P. Smith, R.J. Currie and V.K. Nair, 2007. Correlation of Marek's disease herpesvirus vaccine virus genome load in feather tips with protection, using an experimental challenge system. *Avian Pathology*, 36: 467-474.
- Buscaglia, C., P. Nervi and M. Risso, 2004. Characterization of four very virulent Argentinian strains of Marek's disease virus and the influence of one of those isolates on synergism between Marek's disease vaccine viruses. *Avian Pathology*, 33: 190-195.
- Calnek, B.W., H.K. Adlindger and D.E. Kahn, 1970. Feather follicle epithelium: A source of enveloped and infectious cell-free herpesvirus from Marek's disease. *Avian Diseases*, 14: 219-233.
- Calnek, B.W. and R.L. Witter, 1997. Marek's disease. In: B.W. Calnek, H.J. Barues, C.W. Beard, L.R. McDougald, Y.M. Saif. *Diseases of Poultry*. 10th Edn., pp: 369-413.
- Calnek, B.W., 1980. Marek's disease virus and lymphoma. In: F. Rapp (Ed) *Oncogenic Herpesviruses*. CRC Press, Boca Raton, FL, pp: 103-143.
- Fatunmbi, O.O. and D.F. Adene, 1986. A ten year prevalence study of Marek's disease and avian leukoses at Ibadan, Nigeria. *Acta Vet. Brno.*, 55: 49-53.
- Freeman, B.M., A.C.C. Manning and R.S. Phillips, 1984. Failure to induce stress reactions following vaccination against Marek's disease or Newcastle disease. *Res. Vet. Sci.*, 36: 247-250.

- Kenzy, S.G. and B.R. Cho, 1969. Transmission of classical Marek's disease by affected and carrier birds. *Avian Dis.*, 13: 211-214.
- Nawathe, D.R., C.K. Ojeh and O. Onunkwo, 1978. Incidence of Marek's disease in northern states of Nigeria. *Vet. Rec.*, 102: 128.
- Olabode, H.O.K., 2009. Prevalence of avian leukosis and Marek's disease in Ilorin, Kwara State, Nigeria. *Nig. Vet. J.*, 30: 64-68.
- Oni, O.O. and A.A. Owoade, 2009. Seroprevalence of Marek's disease virus antibody in some poultry flocks in southwestern Nigeria. *Anim. Prod. Res. Adv.*, 5 (1) (In press).
- Pagne, L.N., 1999. Marek's disease. In: F.T.W. Jordan and M. Pattison (Eds) *Poultry Diseases*. 4th Edn., N.S. Saunders, London, pp: 112-122.
- Payne, L.N., 1985. Historical review. In L.N. Payne (Ed) *Marek's disease*. Martinus Nijhoff, Boston, M.A., pp: 1-15.
- Purchase, H.G., 1985. Clinical disease and its economic impact. In: L.N. Payne (Ed) *Marek's disease* Martinus Nijhoff, Boston, MA., pp: 17-24.
- Sharing, J.M., 1989. Marek's disease in: H.G. (1989). *Marek's disease* In: H.G. Purchase, L.H. Arp, C.H. Domermuth, J.E. Pearson (Eds) *A laboratory manual for the Isolation and Identification of Avian Pathogens*. 3rd Edn. A.M. Association of Avian Pathologists. New Boston P.A., pp: 89-94.
- Witter, R.L. and K.A. Schat, 2003. Marek's disease virus. In: Y.M. Saif, H.J. Barnes, J.R. Glisson, A.M. Fadly, L.R. McDougald and D.E. Swayne (Eds.) *Disease of Poultry*, 11th Edn., Iowa State University Press, Ames, Iowa, USA., pp: 407-464.
- Witter, R.L., J.J. Solomon, L.R. Champion and K. Nazerian, 1971. Long term studies of Marek's disease infection in individual chickens. *Avian Dis.*, 15: 364-365.
- Witter, R.L., B.W. Calnek, C. Buscaglia, I.M. Gimeno and K.A. Schat, 2005. Classification of Marek's disease viruses according to pathotype-philosophy and methodology. *Avian Pathol.*, 34: 75-90.
- Witter, R.L., G.H. Burgoyne and J.J. Solomon, 1969. Evidence for a herpesvirus as an etiologic agent of Marek's disease. *Avian Dis.*, 13: 171-184.
- Witter, R.L., 1972. Epidemiology of Marek's disease-A Review. In: P.M. Biggs G. deTke and L.N. Payne (Eds) *Oncogenesis and Herpes viruses*. 1 ARC Lyon France, pp: 111-122.
- Witter, R.L., 2001. Marek's disease vaccines-past, present and future [chicken versus virus-a battle of the centuries]. *Current Progress on Marek's Disease Research*. In K.A. Schat, R.M. Morgan, M.S. Parcells and J.L. Spencer (Eds), American Association of Avian Pathologists, Kennett Square, PA, USA., pp: 1-9.
- Witter, R.L., J.M. Sharma, J.J. Solomon and L.R. Champion, 1973. An age-related resistance of chickens to Marek's disease: Some preliminary observations. *Avian Pathol.*, 2: 43-54.