

IMMUNOGENICITY OF NIAB ANGARA VACCINE IN BROILERS

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ABSTRACT

The present study was conducted to evaluate the efficacy of NIAB ANGARA vaccine under field conditions. For this purpose 2000 broiler chicks were vaccinated against Newcastle disease (ND) and Infectious Bursal disease (IBD). NIAB ANGARA vaccine was injected to 1000 broilers on 30th day of age. When the flock showed 5 to 10 mortalities per day, HPS was suspected in post mortem reports. Remaining 1000 broilers were kept as control. Seventy to eighty birds died in treated group within 2-3 days after vaccination then mortalities reduced to 1 and 2 birds per day. Flock became normal after 7th day of vaccination, whereas mortalities increased slowly and remained high in control group. Blood samples were collected from birds of treated and control groups on 37th and 44th day of age. Antibody titer against HPS vaccine was determined by Indirect Haemagglutination test. Results showed that NIAB ANGARA vaccine triggered the production of antibodies against HPS virus. The treated flock recovered from the disease within a week.

Key words: Hydropericardium syndrome, angara disease, immunogenicity, vaccine

INTRODUCTION

Hydropericardium syndrome (HPS) was first observed in 1987 at Angara goth, a broiler chicks raising area near Karachi, Pakistan so, the syndrome was named as *Angara disease* (Jaffery, 1988). Later, the disease was noticed at two other broiler farms situated near Jurah pull in Lahore, Punjab. A closely resembling syndrome has been reported in Canada (Mc-Cuaige *et al.*, 1992), England (Jones, 1976), Germany (Bergmann *et al.*, 1979), Iraq (Abdul Aziz and Al-Attar, 1991), North and South America (Cown, 1992), and Chile (Toro *et al.*, 1999).

The disease has rapid onset without noticeable signs. However, some birds are seen as depressed and off-feed. The affected birds show difficulties in movement, loose greenish to yellowish droppings and chalky-pasted vents (Khwaja *et al.*, 1988; Tariq, 1988). The course of disease is usually 10–15 days with 100% morbidity and 30–90% mortality (Tariq, 1988). The causative agent of the syndrome has been identified as a filterable virus, which belongs to Avi Adeno virus- 4 (Rabbani *et al.*, 1998).

To overcome huge economic losses to poultry industry, different vaccines against HPS are in use giving variable results. However, outbreaks are common in vaccinated flocks. Lack of required quantity of virus and storage of vaccine under improper conditions seem to be the possible reasons of vaccine failure. It is the need of hour to produce more effective and safer vaccine.

The present study was designed to evaluate the efficacy of a water based, formalized NIAB ANGARA vaccine in broilers during a natural outbreak of HPS.

MATERIALS AND METHODS

Experimental chicks

Two thousand broiler chicks (1-day old) kept at a broiler farm at Chak No. 37 near Satiana, Faisalabad, were reared under field conditions. These chicks were vaccinated according to a usual schedule in broilers i.e., Newcastle disease on day 7 and Infectious Bursal disease on day 14 (Anjum, 1997). As HPS was prevalent in the area, so at 26th day of age flock got infected and as was confirmed by post mortem reports. Birds were divided into two lots of 1000 birds each. One lot was vaccinated with NIAB ANGARA vaccine in double dose at 30th day of age and other was kept as control (Afzal and Ahmad, 2000).

Collection of serum samples

Fifty blood samples were collected randomly from each group in disposable syringes at day 37 and 44 of age. From each bird 1-3 ml blood was drawn in a syringe and held at 25⁰C for 4-6 hours. The serum was separated and collected in sterile screw capped Pyrex tubes. These tubes were labeled and stored at -20⁰C.

Parameters studied

Mortality in both groups was recorded (pre and post vaccine) due to natural infection of HPS virus. Serum antibody titer was determined against HPS vaccine by Indirect Haemagglutination Assay (IHA) (Rehman *et al.* 1989).

Data thus collected was statistically analyzed by applying unpaired t-test (Steel and Torrie, 1982).

RESULTS AND DISCUSSION

NIAB ANGARA vaccine initiated the antibody production against the HPS virus during outbreak of the disease, as vaccination was done in treatment group on 30th day of age after natural infection of the virus. The flock was vaccinated against Newcastle disease & Infectious Bursal disease and no vaccine of HPS was done at proper time so the flock was susceptible to HPS. An outbreak of HPS was recorded in that area on adjacent farms. Postmortem examination revealed the accumulation of serous fluid in pericardial sac, oedema of abdominal cavity and enlarged flabby heart. These signs indicated outbreak of HPS (Rabbani *et al.*, 1998).

Mortality started on 26th day of age, 5–10 birds died daily. In treated group mortality increased up to 20–30 birds daily for four days after vaccination, this high mortality rate is due to stress on birds during vaccination. Then it reduced gradually and flock became normal 4-5 days after vaccination (Table 1).

Table 1. Daily mortalities in treated and control groups

	Age (days)	26	27	28	29	30										Total
Pre Vaccination	Treatment	5	7	8	10	20										50 ^{NS}
	Control	5	8	7	10	11										41 ^{NS}
																4.1%
	Age (days)	31	32	33	34	35	36	37	38	39	40	41	42	43	44	Total
Post Vaccination	Treatment	25	30	25	15	5	1	0	0	1	0	1	0	0	0	103*
	Control	13	12	15	18	24	30	35	38	40	45	50	48	52	55	475*
																47.5%

NS = Non Significant; * = Significant Difference (P<0.05)

Table 2: Antibody titer of broilers against HPS Virus after 7th & 14th day of vaccination in treated and control groups

Age of birds (days)		Distribution of birds on the basis of IHA antibody titer (well no)										GMT (Log ₂)
		1	2	3	4	5	6	7	8	9	10	
Treated	37				11	14	14	11				5.5
	44						16	18	16			7.0
Control	37	25	25									1.5
	44		25	25								2.5

In control group, mortality increased gradually from 5–10 to 50–55 birds daily and this number increased continuously day by day up to 44th day of age (Table 1). Serum samples collected from treatment group at 37th day of age showed that Geometric Mean Titer (GMT) of antibody against HPS Virus was log₂ 5.5, which indicates that antibody production has started in vaccinated birds. Whereas serum samples collected at 44th day of age showed that GMT of antibody against HPS Virus was log₂ 7.0, which indicates that serum antibody titer is sufficient at 10 to 14 days post vaccination (Table 2). This sharp increase of titers in treated group was due to use of formalized vaccine which produce instant immunity but for short time as compare to oil based vaccine which produce immunity slowly but for long period of time (Arfan, 2002).

Serum samples from control group at 37th day of age showed that GMT of antibody against HPS virus was log₂ 1.5, which indicates that antibody titer was very low. Whereas serum samples collected at 44th day of age showed that GMT of antibody against HPS Virus was log₂ 2.5, which indicates that antibody titer in unvaccinated birds was non-protective even after 2 weeks. Moreover antibody titers on both sampling days in control group were incomparable with treated one (Table 2).

It may be concluded that NIAB ANGARA vaccine is effective against HPS of poultry, as it triggers antibody production in infected birds and reduces the loss significantly. This vaccine can be used in healthy as well as HPS infected flock at early stage of disease.

REFERENCES

- Abdul-Aziz, T. A. and M. A. Al-Attar (1991). New syndrome in Iraqi chicks. *Vet. Rec.*, 129: 272.
- Afzal M. and I. Ahmad (2000). Efficacy of an inactivated vaccine against hydropericardium syndrome in broilers. *Trop. Anim. Health Prod.*, 32: 99-111.
- Arfan, A. (2002). *Comparative immunogenicity of different Hydropericardium syndrome (HPS) vaccines in Broilers*. M. Sc. (Hons.) thesis, University of Veterinary and Animal Sciences, Lahore, Pakistan.
- Bergmann, V., K. Muller-Molenar and H. Birnbaum (1979). Occurrence of hydropericardium ascities syndrome (oedema disease) in broiler flocks: *Monatshefte-fur-veterinarmedizin*, 34: 626- 628.
- Cown, B. S. (1992). Inclusion body hepatitis anaemia and hydropericardium syndrome: aetiology and control. *World Poult. Sci. J.*, 48: 247- 254.
- Jones, R. C. (1976). Reoviruses from chicken with hydropericardium. *Vet. Rec.*, 99: 458.
- Jaffery, M. S. (1988) *A treatise on Angara disease in chicken*. Pakistan Vet. Med. Assoc. Karachi, pp: 1-33.
- Khwaja, D. A., S. Ahmad, A. M. Rauf, M. Zulfiqar, S. M. I. Mahmood and M. Hussain (1988). Isolation of an adenovirus from hydropericardium syndrome in broiler chicks. *Pakistan J. Vet. Res.*, 1: 51- 52.
- Mc-Cuaige, L. W., H. C. Carlson and I. Motzok (1992). Observations on hypervitaminosis A and hydropericardium in chicks. *Poult. Sci.*, 51: 1206-1210.
- Rabbani, M., M. A. Muneer, K. Naeem and H. A. Hashmi (1998). Purification of an avian adenovirus PARC-1 isolate causing Angara disease (HPS) in chickens of Pakistan. *Proc. Intl. Seminar on "Microbial Diseases of Livestock and Poultry"*, Lahore, Pakistan.
- Rehman, S. U., M. Ashfaq, A. D. Anjum and T. A. Sindhu (1989). Indirect Haemagglutination test for detecting Angara disease (hydropericardium) agent antibody. In: *Poultry Souvenir, 1st Intl. Conf. and Trade Show on Poultry Production and Development Society*, Karachi, pp: 73-74.
- Steel, R.G.D. and J.H. Torrie (1982). *Principles and Procedures of Statistics*. 2nd Ed., McGraw Hill book Co. Inc, New York. pp: 137-171.
- Tariq, M. A. (1988). Preliminary studies on hydropericardium syndrome in broiler chicks and its control measures in Pakistan. *Proc. 2nd Natl. Seminar on Hydropericardium Syndrome*, Rawalpindi, Pakistan, pp: 49-57.
- Toro, H., C. Prusas, R. Raue, L. Cerda, C. Geisse, C. Gonzalez and M. Hess (1999). Characterization of fowl adenoviruses from outbreaks of inclusion body hepatitis/hydropericardium syndrome in Chile. *Avian Dis.*, 43: 262-270.

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