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YOLK INOCULATION IN GUMBORO INFECTED CHICKEN.

By

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SUMMARY

Hundred and twenty chicks of 42 day old were divided into 8 groups, 3 of them were Gumboro infected and treated with dilutions 1/10, 1/100 And 1/1000 of yolk from immune dams as compared to 3 other parallel groups treated with yolk from non-immune dams. Infected nontreated group and non-infected nontreated group were kept as controls for the experiment.

Clinical signs, mortaity seroconversion and bursal body weight ratio were measured . The different parameters were presented and effect of yolk inoculation from immune dams on the course of Gumboro infection was discussed.

INTRODUCTION

Infectious busal disease virus (IBD) is a pathogen of major economic importance fo poultry industry in Egypt. It is considered as a virulent field capable of causing high mortality in young chicks.

Since the first outbreak of IBD in USA (Cosgrove, 1962), the disease had been reported in almost all parts of the world as reviewed by Faragher (1971) and Okoye and Dip Phill (1984) including Egypt (El-Sergany et al., 1974; Ayoub and Malek, 1976). IBD caused a major disease problem in the last three years characterized by severe clinical signs with higher levels of mortality, and was recorded in England , Denamark, Holland, Australia, Irland and Middle East area including. Jordan, Syria and Israel (Box, 1989) and Van der Marel, 1989 and Zahid, 1990).

In Egypt, a very virulent IBD strain had been isolated (Khafagy et al., 1991) which caused high mortality up to 70%. Field trials had been made to minimize mortality by yolk inoculation and gave promising results. The aim of this work is to varify the role of yolk inoculation to Gumboro infected birds and the possibility of using it during Gumboro outbreak putting some light on the bursal body weight ratio, mortality rate and seroconversion in inoculated and non-inoculated birds.

MATERIAL AND METHODS

* Chicken:

On hundred and twenty one day Lohman chicks were purchased from a commercial hatchary. The chicks were housed in isolated pens in separate groups.

* Virus:

The locally isolated very virulent Infectious Bursal Disease (VVIBD) strain (Khafagy et al.,

1990) was used.

*** Yolk material:**

Two sources of eggs were chosen , the first was from immune dams with high antibody titre (5.50 by AGPT and 5.20 by NT). the second from non-immune ones.

*** Experimental design:**

At 42 day old (Khafagy et al., 1990) the chicks were divided into eight (8) groups of 15 birds each. Those birds were examined for maternal antibodies of IBD and found free in our tests on 42 days (test age). Chicks of group 2 to 8 were infected intraocculary with 100 ml per bird of 1:10 dilution in PBS (10^5 EID_{50}/ml VVIBD, Khafagy et al., 1990). While birds of group one were left as noninfected control. Experimental groups were observed for signs, deaths and post mortem findings.

As soon as symptoms appeared at 3 days post infection, the experimental birds were treated by yolk material intramuscularly (0.5 ml in 1% skimmed milk in different dilutions in PBS) . Groups 3,4 and 5 were treated with yolk of immune birds while groups 6,7 and 8 were treated with yolk of non-immune source in dilutions of 1/10, 1/100, and 1/1000, respectively. Group 2 was infected not treated deaths and post mortem findings were recorded at 0,5,12 and and 19 days post infection.

Bursae were collected from dead or sacrificed birds, weighed and bursal indices were calculated according to Dohms et al., (1988). Blood samples were collected in the same intervals for Agar Gel Precipitation test (AGPT) and Serum Neutralization test (SNT).

- Neuralization test:

Serum samples were examined by microtitre V test using chicken embryo kidney cells starte with dilution 1:16 against 100 TCID of ce culture adapted lukert strain of IBD virus a described by Nagi et al., (1980).

Micro-gel precipitation test:

It was carried out in the agarose and 8% Nac medium (Woernle, 1966). Preceipitating line were recorded after 24 and 48 hours.

RESULTS

Clinical signs of the disease started on the 3rd day with high mortality with peak on 5th up to 6-7 day. Course of the disease ranged between 10 to 12 day. Clinical signs included whitish-yellowish greenish and sometimes bloody watery diarrhoea. Post mortem findings showed haemorrhages at proventriculus-gizzard junction. Kidneys were enlarged, ureters sometimes filled with ureates, liver congested. The bursa of Fabricius was enlarged up to twice the normal size, filled with creamy and sometimes bloody exudate.

Mortality rate decreased in groups by yolk from immune dams especially group 2 that treated by 1/10 dilution which shows a lowest mortality rate as in table (1). Anyibody titre estimated by both AGPT and SNT showed increasing antibody titre in groups treated by yolk from immune dams. Group 3, which was treated by 1/10 dilution of yolk from immune dams showed the highest antibody titre especially by SNT. These titres increased in the second dollected samples on 5 th day post infection reaching peak on 12 th and 19th days. In some groups titers started to ecline on 19 days post infection as shown in table (2).

Table (1): Mortality rate of different groups of Gumboro infected yolk inoculated and non-yolk inoculated .

Group No.	Treatment	No.of deaths total No	Mortality rate
G_1	Non-infected non treated 0	0 / 15	Zero
G_2	Infected non-treated	11 / 15	73.3
G_3	Infected treated immune 1/10	4 / 15	26.7
G_4	Infected treated immune 1/100	5 / 15	33.3
G_5	Infected treated immune 1/1000	8 / 15	53.3
G_6	Infected treated nonimmune 1/10	9 / 15	60.0
G_7	Infected treated nonimmune 1/100	10 / 15	66.7
G_8	Infected treated nonimmune 1/1000	10 / 15	66.7

Table (2): Serum antibody level measured by both AGPT and SNT in different groups at different intervals .

Group No.	Treatment	AGPT				SNT			
		Days post infection				Days post infection			
		0	5	12	19	0	5	12	19
G_1	Control non-infected non treated	0	0	0	0	1	2	2.1	2
G_2	Infected non-treated	0	3.0	5.0	4.77	1	3	4	2
G_3	Infected treated immune 1/10	0	3.2	6	3.29	1	6	5.25	5.25
G_4	Infected treated immune 1/100	0	2.1	3.2	5.7	1	4.5	4.77	4.66
G_5	Infected treated immune 1/1000	0	2.0	2.66	5.5	1	4.8	5	5
G_6	Infected treated nonimmune 1/10	0	2.1	4.52	2.27	1	2.33	4.28	3.7
G_7	Infected treated nonimmune 1/100	0	2.33	3.0	2.0	1	1.5	2.2	2
G_8	Infected treated nonimmune 1/1000	0	0.25	1.5	1.0	1	1	2	1

N.B: Figures represented arithmatic means of force (log 2) .

The bursal index increased reaching the peak on day 5 post infection then decline on day 12 and 19 post infection, this occur in all groups. In group 3, the bursal index is the lowest index in compared with other groups and control one as shown in Table (3)

Table (3): Bursal index of samples collected at 0, 5, 12 and 19 days post infection from the different treated and non treated birds .

Group No.	Treatment	Bursal index			
		Days post infection			
		0	5	12	19
G_1	Control non-infected non treated	3.3	3.2	3.7	1.1
G_2	Infected non-treated	3.3	5.9	4.8	2.4
G_3	Infected treated immune 1/10	3.3	3.6	3.2	1.4
G_4	Infected treated immune 1/100	3.3	4.4	3.3	1.2
G_5	Infected treated immune 1/1000	3.3	4.7	2.9	1.1
G_6	Infected treated nonimmune 1/10	3.3	4.2	2.5	1.5
G_7	Infected treated nonimmune 1/100	3.3	4.6	2.2	1.5
G_8	Infected treated nonimmune 1/1000	3.3	5	3.2	2.1

Bursal index: Bursal weight/body weight X 10^3 (Dohms et al; 1988)

DISCUSSION

It has been concluded laterly that vaccination of chicken against IBD become not compative against the infection as recorded field reports on the recent outbreaks of very virulent infectious bursal disease (VVIBDV) in vaccinated chicken flocks which had been observed in England and susbequently other countries (Box, 1989; Luticken and Van der Marel, 1989 and Zahid, 1990). It has been also reported in Egyptian chicken flocks with high morbidity, usually close to 100% and high mortality up to 70% (Khafagy et al., 1990).

A high level of maternal antibodies may either protect against death, clinical IBD, bursal lesions and immunsuppressive effects of early IBDV infection (Rosenberger et al., 1975) or neutralize the vaccine virus (Rinaldi et al., 1947). Maternal antibodies passed from hen progeny very considerably for two reasons. First, titre levels in breeder hens differ from bird to another . Secondly, chicks can receive as little as 50 percent or as much as 70% of the hen's titre. Mixing chicks from different breeder flocks will create a mixture of maternal antibody levels which will make vaccination timing even more connfusing (Giambrone, 1983 and Salsbury, 1986).

Because of the previously mentioned facts and the increasing liability of Gumboro field outbreaks,

there was a need for a tool to interfere during breaking the disease. In the current study different groups of experimentally infected birds with Gumboro disease virus were inoculated with yolk from immune and other groups from nonimmune dams on onset of disease signs.

In our experiment, the group inoculated with yolk material from immune dams showed the best result lowering mortality rate and increasing antibody titre in chickens especially with birds inoculated with dilution 1/10 which is considered due to the increased level of antibody titers of the inoculated yolk material as indicated in our results.

One measure of protection was bursa body weight ratio, the larger the ratios, the less damage caused by IBD challenge (Dohams et al., 1988), wgich was clearly evidenced in our results, bursal index (Table 3) decreased sharply within two weeks except in groups treated by yolk from immune dams which slightly decreased specially group 3 with 1/10 dilution of yolk material from immune dams.

Out of all different parameters measured to evauate the effect of egg yolk inoculation on Gumboro infected birds, we could have a discriptive evidence of the anti-Gumboro antibodies in yolk material of immune dams and its effect on the course of the disease, changes of bursa body weight ratio, clinical signs and post mortem lesions. Those results are supportive for field trials using yolk material therapeutically for control of Gumboro disease outbreaks. Further studies are in progress.

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