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STUDIES ON EGG DROP SYNDROME IN IRAN

Aghakhan,S.M., Khodashenas,M.

ABSTRACT. **Seven outbreaks of EDS 76 in broiler breeders have been studied. In all cases, except one,the egg drops occurred after peak laying and ranged from 25% to 38% . The falls were accompanied by a loss of brown colour and production of thin-shelled , soft-shelled , shell-less and also eggs with rough shells and chalky deposits.**

The presumptive diagnosis of the condition was confirmed by haemagglutination-inhibition (HI) tests using an antigen prepared in chick embryo liver (CEL) cell culture. Differences in clinical and epidemiological features with some other conditins are indicated and the high probability of existance of EDS also in commercial layers is pointed out. In view of the present situation the urgent need for vaccination of all flocks at risk is stressed.

Key words: LAYER CHICKENS/ANIMAL DISEASES/EGG DROP SYNDROME

INTRODUCTION

Egg drop syndrome 76 (EDS 76) in chickens is a viral disease characterized by a fall in egg production or a failure to achieve production target , accompanied by

thin-shelled, soft-shelled and shell-less eggs and also a loss of colour in brown egg laying stocks (Mc Ferran et al.1978., Mc Ferran 1984). The affected birds appear otherwise healthy.

The causal agent is an adenovirus different from conventional fowl adenoviruses in that it agglutinates chicken red blood cells. Moreover, it appears that the agent lacks the avian adenovirus group antigen . It is prevalent in duck and goose populations and has presumably been introduced into chickens through contaminated vaccines. Shortly after the first description of the disease in Western Europe (Van Eck et al 1976, Baxendale 1977 , Mc Ferran et al 1977), suspicious cases occurred in broiler parent stocks in Iran, which were not committed to laboratory examinations at that time. Due to importation of parent stocks from those countries, however, vaccination against the disease was practiced for a few years but was later abandoned.

This article summarizes the studies on seven outbreakes of EDS during a two and a half years period , starting from November 1987, in broiler breeders in Iran.

MATERIALS AND METHODS

Cell culture - Chick embryo liver cell (CEL) monolayers were prepared from 14-day-old SPF embryos (Valo eggs , Lohmann, Cuxhaven , Germany).Tissues were dispersed using 0.05% trypsin and cells grown in Hanks' solution + yeastolate and lactalbumin hydrolysate (LYH), supplemented with 10% foetal calf serum + antibiotics. Cultures were maintained in the same medium except that foetal calf

serum was substituted with 2% calf serum.

Virus and virus pool - EDS virus strain 127 originally described by Mc Ferran et al (1977) was used in these studies.

The virus was grown in CEL cells and cultures harvested following three cycles of freezing and thawing . After centrifugation at low speed the supernatant was distributed in small aliquots, and stored at -50°C . The virus pool had a haemagglutinating (HA) titre of 1/512.

Antiserum - 8-week-old SPF chickens were beld and then infected orally with approximately 10^5 TCID50 of the virus . The birds were bled 4 weeks later and collected serum kept at -20°C.

Serological test - The haemagglutination-inhibition test was performed in U- bottomed microplates,using doubling dilution of serum, in 0.025ml. volumes, 0.8% chicken red blood cells and 4 HA units of the virus. Titres were expressed as reciprocals of the highest dilution of serum which caused complete inhibition of H.A.Positive and negative control sera were included in the tests.

Breeding farms - The farms A,B,C,D,E,F and G were multi-housed and located in widely separated geographical areas but A and B were properties of the same organisation.The birds in all farms were vaccinated against Marek's disease, Newcastle disease, infectious bronchitis,infectious bursal disease, infectious laryngotracheitis , fowl pox and avian encephalomyelitis but not against EDS, except for 10 out of 20 houses in farm E,which comprised another age group. The flocks were free of Mycoplasma gallisepticum infection but were Mycoplasma synoviae positive.

Specimens - Blood samples were collected from flocks

exhibiting a fall in egg production. Whenever available, samples were taken prior to, at the time of , and about three weeks after the onest of the signs in a house. Serum was separated and stored at -20°C until required . On occasions live birds were also received for post-mortem examinations.

RESULTS

Clinical symptoms and post-mortem lesions - No outward signs were observed except for an occasional delay in feed intake and diarrhoea in some birds. Mortality rates remained at normal levels in all outbreaks.

Similarly, no gross abnormalities were seen in any of the internal organs of the live birds submitted for post-mortem examinations.

Fall in egg production - In most cases egg drops occurred after peak production. The first sign was a loss of bloom and brown colour of the shell . This was followed in 1-2 days by production of thin-shelled , soft-shelled and shell-less eggs.

Rough shells and eggs with chalky deposits were also frequently seen but misshapen, ridged and small eggs were not a characteristic feature. No effect on quality of albumen was observed when comparisons were made between affected and normal eggs.

Egg drops varied between the farms and also the houses within a farm . It ranged from 25% to 38% at the lowest point in individual houses.

Shell-less eggs were usually eaten by birds and therefore included in the statistics as a fall in

production. Shell abnormalities subsides and production rates returned to normal for the age in five to nine weeks.

Farm E comprised two age groups housed in two rows of ten houses each, about 50m. apart , and only one age group was vaccinated against EDS. These remained unaffected throughout their economic life. The problem in farm B took a different from in most houses. There was no true fall in production, merely a failure to peak. The incidence of abnormal eggs was much lower than those in the other farms.

Serological findings - Most serum samples collected about three weeks after the onest of the disease produced positive results, often with relatively high titres(table 1). Parid sera could be obtained from a number of houses in farms E,F and G. Samples collected within 2-3 days of the first changes in the egg shells proved to be largely negative . Positive samples showed relatively low titres i.e. 4-5 log 2 or less . All sera obtained prior to the onest of the disease produced negative results.

Table 1: The results of haemagglutination inhibition tests.

Sera collected:	Heamagglutination-inhibition tests	
	No.positive sera	Total No.of sera tested
Prior to the onest of disease	0	238
At the onest of disease	18	114
3 weeks after the onest of disease	495	522

DISCUSSION

The transmission of the infection was rather slow and several weeks elapsed before adjacent houses showed a production drop. This was particularly true where reasonable hygienic measures were observed . This would probably imply that the virus is not yet fully adapted to its new host. However, once the virus gained entrance into a hose, the further spread between the birds in that house seemed to take place fairly rapidly.

The origin of infection in each case could not be determined definitely but except in farm B . all other drops occurred sometime after peak production. Farm B , showing a different pattern of fall in production compared to the other farms, which took the form of just a failure to peak, had most probably become infected prior to the point of lay, resulting in an immune response in some birds.

Some features of egg shell changes, the course of the disease and paticularly transmission of the infection were at variance with those in infectious bronchitis and Newcastle disease, but definite results were obtained from serological findings which left no doubt as to the involvment of EDS. It is most likely that the condition also exists in commercial laying flocks and that , at present, EDS is one of the major causes of falls in egg production in the country.

The results of these studies strongly emphasize the urgent need for regular vaccination of parent stocks and commercial layers against the disease at least for some years to come.

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