SEROPREVALENCE AND PATHOLOGY OF EGG DROP SYNDROME (EDS-76) IN COMMERCIAL CHICKEN LAYERS

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ABSTRACT

From September 1994 to April 1996, the aetiology of sudden decrease in egg production in commercial layer flocks was investigated. Clinical manifestations were recorded and the egg-shell quality was evaluated in 80 affected flocks. Serological screening was conducted twice at fortnightly intervals to record antibody titres against egg drop syndrome (EDS). Out of 80 flocks with lowered production, 34 (42.5 %) flocks were serologically positive for EDS. No specific gross lesions were observed but histopathological alterations were seen in the oviducts. Based on these findings, the farmers were advised to vaccinate their flocks against EDS between the age of 14-16 weeks using killed vaccines.

INTRODUCTION

During the previous few years in Pakistan, poultry industry has developed very rapidly. However, further expansion is confronted by the prevalence of various infectious and non infectious diseases (Siddique and Javed, 1989). Among these now-a-days egg drop syndrome (EDS) is posing a serious threat to layer industry. EDS was first reported in chickens during 1976 by Dutch workers (VanEck *et al.*, 1976). The changes seen in eggs were similar to those associated with infectious bronchitis and Newcastle disease. Later on, the causal agent of EDS in chickens was identified as haemagglutinating adenovirus (McFerran *et al.*, 1978; McFerran, 1991). The affected flocks show a sudden drop in egg production along with defective egg-shell quality. Chalky thin and soft shells or shell less eggs are observed. In case of brown layers, eggs may lack normal pigmentation.

During mid 1993, a number of commercial and breeder chicken flocks were reported to have a sharp decline in egg production. Many cases of EDS were suspected but not yet confirmed serologically in commercial layer flocks. However, the problem has been confirmed in broiler breeder flocks around Rawalpindi (Naeem, 1994).

The objective of the present study was to investigate the prevalence of EDS and to record its clinical findings, egg shell quality and gross as well as histopathological lesions.

MATERIALS AND METHODS

Flock History

From September 1994 to April 1996, commercial layer flocks around Faisalabad, Samundri, Gojra, Kamalia, Chichawatni and Arifwala were surveyed and the flocks with sudden drop in egg production with unknown aetiology were selected for detailed investigations. The flock history was recorded on a predesigned proforma including location of the farm, flock size, vaccination schedule, type of breed, nature of feed, clinical manifestations, various treatments and their response etc.

Sample collection

From each suspected flock, 20-40 eggs were collected for egg shell quality. Blood samples were collected from 15-20 birds from each flock at fortnightly intrvals and serum was separated for serological studies.

Serological studies

Haemagglutination inhibition (HI) test against EDS-76 virus (4 HA units) was done by the Beta procedure as described by Giambrone *et al.* (1976) using EDS antigen (Solvay International, USA).

Gross and histopathological studies

Ten to 15 birds from each suspected flock were sacrificed and gross pathological lesions in various

organs were recorded. Reproductive organs were collected after detailed necropsy. Various portions of the reproductive tract were preserved in 10 per cent buffered formalin. These were processed in ascending grades of alcohol, cleared in xylene and embedded in paraffin wax. Sections of 4-6 μ thick were cut and stained with haematoxylin and eosin stain to study the histopathological alterations (Humason, 1972).

RESULTS

Prevalence

Among 80 flocks with lowered egg production, 34 (42.5%) flocks were serologically positive for egg drop syndrome (EDS). Seroprevalence varied in different areas from 35 to 51 per cent among the suspected flocks. However, there was no significant difference in birds of various breeds and being maintained on different feeds. Regarding the seasonal prevalence, it was found higher during winter months as compared to other seasons of the year. Age of the birds did not have remarkable effect and the syndrome was recorded in the laying flocks varying from 20 to 60 weeks of age.

Clinical manifestations

All the flocks seropositive for EDS flocks showed drop in egg production ranging from 10 to 62 per cent (Table 1). The most striking symptom was laying of soft, thin-shelled or shell-less eggs. In most of the cases, such eggs were laid during night times and during early stages, many of the farmers were not aware of laying of such eggs because these were eaten by the birds. In such cases, however, egg shells were seen in the litter. In brown layers (3 flocks) there was loss of pigmentation. In some instances, size of the eggs was also reduced. Defective egg shell quality in the form of granular rough appearance was seen in many of the positive flocks and this particular appearance lasted even for many weeks after recovery. Drop in egg production was noticed for variable time periods from two to even upto eight weeks. Increase in production was slow and it took two to four weeks in different affected flocks. However, preinfection production levels were not attained even after recovery.

Low feed consumption (60-80 gm/bird) was seen in 32 per cent of the seropositive flocks. Greenish-white diarrhoea was also noticed in these flocks with lowered egg production. In few of the flocks, the birds appeared depressed, and normal chirping sounds were lacking.

Serological studies

EDS haemagglutination inhibition (HI) geometric mean titre (GMT) and a significant increase at fortnightly intervals were considered as indication of seropositivity. In 34 flocks (42.5 %), GMT was 52 or higher and it increased to 184 or higher after two weeks. Remaining 46 (57.5 %) flocks were found seronegative for EDS. HI titres were also recorded against Newcastle disease and infectious bronchitis. However, in 40 of these flocks, these titres did not show a significant increase after two weeks. In 16 flocks, haemagglutinating inhibiting antibodies were also demonstrated against *Mycoplasma gallisepticum*.

Gross and histopathological studies

In EDS seropositive birds, no specific gross lesions were seen except the presence of shell-less or defective shelled eggs. However, in some of the birds, presence of gelatinous fluid was recorded in the oviduct. Histopathologically there was pronounced oedema of the uterine submucosa, atrophy of tubular glands and infiltration of entire mucosa by mononuclear cells. The uterine epithelium was devoid of cilia and in some cases intranuclear inclusions were also seen in superficial epithelial cells.

DISCUSSION

Sudden drop in egg production may be seen in a number of infectious and non infectious conditions. Among the infectious causes, egg drop syndrome (EDS), Newcastle disease (ND), infectious bronchitis (IB), infectious laryngotracheitis (ILT), mycoplasmosis and infectious coryza are most important. Feed deficiencies, chemical and biological toxins may be the salient noninfectious causes of drop in egg production (Calnek *et al.*, 1991). During 1993-94, the problem of sudden drop in egg production along with defective egg shell was seen in some flocks and it was suspected for egg drop syndrome (EDS). In the present study, among the 80 flocks with lowered egg production, 34 (42.5 %) were seropositive for EDS. Naeem (1994) reported EDS in 12 breeder flocks around Rawalpindi but probably no report in commercial layers in Pakistan.

Loss of pigmentation may also be recorded with low antibody titres against Newcastle disease. However, in many of these problems, there is also respiratory involvement with typical gross and histopathological lesions. Even then, serological investigation is the only way to confirm EDS. Keeping in view, the chances of mixed infections, tests for some of these conditions were also included in this study. Serological studies revealed a remarkable increase in the EDS antibody titres in the affected flocks after two weeks.

EDS virus has been previously isolated from chickens in Australia, Belgium, India, Italy, Japan, Netherland, Irelands, Singapore, South Africa and Taiwan (McFerran, 1991). The syndrome has not been earlier documented in this country in commercial layer

Table 1: Egg production, egg shell quality and feed consumption in EDS seropositive and negative flocks

Parameters	Seropositive flocks		Seronegative flocks	
	Average	Range	Average	Range
Drop in egg production (%)	25.5	10-62	11.0	7-27
Misshapen eggs (%)	2.4	1-4.8	0.6	0-1.2
Soft shelled eggs (%)	4.0	2-7	1.5	0-2.4
Shell less eggs (%)	10.4	5-20	0.8	0-10
Non pigmented eggs (%)	12.0	8-19	-	-
Reduction in feed consumption(gm/bird)	9.0	0-22	12.5	7-27

* In case of brown layers.

flocks. The present investigations confirmed the presence of EDS avian adenovirus in and around Faisalabad.

No gross pathological lesions have been described in naturally occuring outbreaks except inactive ovaries and atrophied oviducts. The histopathological alterations revealed that the lesions were confined to the oviduct. VanEck *et al.* (1978) also reported similar histopathological alterations. Intranuclear inclusion bodies were present in the superficial epithelial cells. The formation of intranuclear inclusion bodies is a characteristic feature of adenoviruses (Smyth *et al.*, 1988).

Based on these findings, the farmers of the area were advised to vaccinate their flocks with EDS-killed vaccine between the age of 14-16 weeks. The vaccination will help in controlling the spread of this disease to other poultry raising areas.

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