Abstract

Researches on avian bursal disease

Considering the huge extent of the avian technology at global level, more complex technologies were developed, therefore new morbid entities appeared, those included in immunodeficiency syndromes. Even so that virus responsible for bursal disease isn't related, taxonomically, with mammal's immunodeficiency viruses, his effect is immunosuppressive because of his effect on primary lymphoid system, represented by Fabricius Bursa and thymus.

Because of the extent of its effects, evolution, economical loss this disease entered in the center of attention, joining other morbid entities involved in avian pathology, as : Newcastle disease, avian influenza, infectious anemia, Marek disease.

By immunosuppressant status that is created by this virus, a negative effect is generated on specific antibodies development (in case of vaccine delivery). Specific resistance for portage germs is decreased, very frequent in chicks, where immunosuppressive states are favored by food quality fluctuations.

The extent of the economical losses made the International office for epizooties to include this viral disease on list "B", being the object of annual information bulletins by "World Animal Health".

Till 1986 avian bursal disease had a shallow evolution but since this year was reported the first "hot focal" caused by a virulent strain.

Because the very virulent virus wasn't controlled in an efficient way using common prophylactic methods available at that time, all factors implicated in avian industry were alerted.

Later the "hot" strains extended their coverage excepting Asia and Oceania. In ours days, in this areas evolve a high pathogen strain, probably favored by an influenza virus strain present in this area.

Because of the coverage extent of this disease, in 1993 the Regional commission for Animal protection and Health commission for Asia and Pacific started a trial, who leaded to the fact that the hot strains are present in all regions where avian industry is intensive. This is the reason why this disease is one of the most debated pathogens, is uncontrolled.

Bursal disease until appearance till now had two distinct evolution periods.

a. Until 1987, the episodes had a subclinical evolution the economical losses were produced by virus immunosuppressive effect, which favors secondary infections or infestations. Usually the infections appear when the maternal antibodies levels drop, nearby the age of 2-3 weeks. In those broods the secondary infections incidence rises, up to age of 4-5 weeks. The economical losses are translated in production drop, forage low conversion, and animals confiscated from slaughterhouse. The estimated percentage is from 0, 5% to 5%.

b. After 1987, with very pathogen strains, hot ones, with a higher financial impact on flash and egg production. The economical loss is higher, mainly because mortality, up to value of 30%, especially in broods with a high bird density.

Because of the continuous appearance of the disease in spite of the prophylactic measures taken, we started some epidemiological, clinic and serologic investigations and prevention trials for infectious bursal disease.

The paper organized in XI chapters and is structured in two parts, one who gathers the main bibliographical data from scientific literature, the second part represents own researches in this field of interest.

Every chapter from the second part is organized in table of contents, material and method, results and partial conclusions. In chapter eleven are structured the final conclusions and the main aspects of the research.

In paper are shown 35 tables, 66 graphics and the bibliographical index gathers 183 titles.

First part of the thesis represents a synthesis of the scientific literature on this topic considering etiology, epidemiological status, global distribution, and latest researches on virus impact over animal organism.

First chapter gathers available data on history, distribution and economical importance.

Second chapter presents etiology and epidemiology in avian bursal disease. Were overseen virus taxonomy, growth conditions, pathogenicity, receptivity, infection sources, transmitting ways and pathogenesis.

Third chapter discusses clinical evolution and lesion changes considering disease evolution status.

Fourth chapter presents the main diagnostic methods, surveillance methods and defensive strategies. Are overlooked isolating methodology, specific antibody detection, nucleic acids evidencing, and immunotherapy.

The research premises are represented by identifying particularities between disease evolution in broods, from intensive breeding system, immunized or not.

The own researches were presented in six chapters and pursue epidemiology and clinical evolution in an intensive breeding unit for broiler chicks (chapter five). Also was investigated the virus evolution in extensive breeding process (chapter six). Immune effectors were monitored from the point of view of the changes induced by bursal disease virus (chapter seven). Anatomopathological findings were discussed in chapter eight and immune response in broilers was designated to chapter nine. In chapter ten were treated issues as surveillance and prevention strategies.

The final chapter gathers the conclusions of this study.

Epidemiological, clinical and diagnostic investigations were done during the following time interval 2005-2006, in an intensive breeding unit. From ours researches outcome the fact that occurrence and disease evolution were favored by various factors as age, breeding system and health status.

Evolution was variable, with a sporadic appearance and only in few cases evolved as two successive series in the same farm. Infectious bursal disease prevalence during that period was 30% and 29%. The specific mortality for this pathogen was 2,42% to an flock of 859215 chicks (year 2005) and 2,45% to an brood of 1563550 chicks in 2006. The mortality limits were variable, between 0, 78-8, 09% and the extent depended by the number of groups affected inside the flock.

Disease entry occurred at 16-24 days age with an evolution of 5-9 days in 2005. In 2006 the disease occurrence was at 14-33 days with an evolution of 4-9 days.

No matter age and evolution duration the mortality curve had a specific dynamic, a continuous climb for the first two days, a plateau evolution next 2-5 days followed by an abrupt drop till normal technological parameters.

If we make a comparison between the flocks with infectious bursal disease and those healthy we can see identical levels for mortality prior disease occurrence. After disease consumption the mortality levels in broods affected is almost double, and at higher levels in the last weeks of life.

The total mortality in the broods affected by disease was 10, 27% (7, 19%-12, 18%), at the same time the mortality in the chicks not affected by disease was 7, 29%. The difference between these values testifies the extent of losses caused by this specific pathology.

The clinical exam in a 250 individuals flock from hall 1 and 100 chicks from hall 3, revealed the following aspects:

- Apathy, ataxia, to 20% chicks from hall 3 and 50% chicks from hall 1
- Weather like diarrhea, whitish feces who agglutinate the feathers from cloacal region, observed in 100% of the chicks from hall 1
- Sudden death to a 15% chicks in hall 3 and 25% in hall 1

Embryos inoculation by allantoidal or chorioallantoic way with 0.2 ml of affected bursal tissue triturate leaded, with the 2-4 passage, to embryonic death in 75-90% of cases between day 3 and day 7, with the highest peak in the 4^{th} day and 5^{th} day.

Considering the immunodepressant effects of this virus the serum levels of lysosyme were measured in various stages of disease comparing with witness group of healthy birds. We monitored also the gammaglobulin total levels in various stages of Gumboro disease.

The maximum serum levels of lysosyme were encountered in diseased chicks, with levels between 7 and 64 μg/ml (medium value 35,43 μg/ml) levels which indicate a total mobilization of body defensive mechanisms.

To chicks passed through disease manifested serum lysosyme levels with a medium level of 22, 86 μg/ml, levels which expose a slow return to normal.

Gammaglobulin level in affected chicks was very high considering the immunodeficiency, fact that do not correspond to B lymphocytes depletion. The chicks during convalescence show a slow recovery to normal levels of the antibodies.

The researches in the extensive sector were done on a flock of 578 chicks, from eggs with a controlled provenience (birds not immunized for infectious bursal disease).