The role of pneumovirus in Swollen Head Syndrome of Chickens: Review

Die Rolle des Pneumovirus beim Swollen Head Syndrome der Hühner: Übersicht

Herrn Prof. Dr. H. Woernle zum 70. Geburtstag gewidmet

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Manuskript eingegangen am 29. Juli 1992

Introduction

A pneumovirus is known to be the primary causative agent of turkey rhinotracheitis (TRT) and appears to be associated with swollen head syndrome (SHS), egg production losses and respiratory diseases complex in chickens.

Swollen head syndrome (SHS) has become a serious economic problem in many countries. The syndrome has been described and identified in all countries where tests for it have been carried out (Table 1).

The infection causes high economic losses by increasing mortality, increasing medication costs, drop in egg production, reduction of egg shell quality, and decreasing hatchability.

Aetiology

The initial aetiological hypotheses for the SHS were bacteria (E. coli), or coronavirus plus bacteria (MORELY and THOMSON, 1984). However, PICAULT et al. (1987a, b) isolated a TRT-like virus from SHS diseased chickens in France.

Since this virus was shown to be serologically related to TRT-virus, produced typical rhinotracheitis symptoms in SPF-turkey poult and classical coryza-like signs in SPF chickens and conventional guinea fowl after experimental infection, it was concluded that TRT virus is a common infectious agent affecting the upper respiratory tract of different poultry species. According to Buys et al. (1989), a significant difference between chicken and turkey isolates is that the latter cannot induce respiratory symptoms in either layer type or in meat type chickens, while chicken isolates could repeatedly produce respiratory signs in both type of birds as well as in turkeys. The isolated viruses from chickens with SHS signs appear to be identical with TRT-virus isolated from turkey. Buys et al. (1989) pointed out the possibility that chicken isolates might represent a subpopulation of the TRT virus that has adopted to chicken.

Size, morphological, physical, chemical and serological properties suggest that the virus belongs to the family Paramyxoviridae and genus Pneumovirus. The Pneumoviruses are RNA, pleomorphic, roughly spherical enveloped and about 70–600 nm in diameter. The envelope bears tightly arranged projections measuring 13 to 15 nm in length. The helical nucleocapsid is about 14 nm in diameter. It is sensitive to ether, chloroform and heat treatment at 56 °C and 60 °C. The virus is reported to be stable over a wide pH range between 3 and 9. All virus isolates are not able to haemagglutinate erythrocytes from different mammal and avian species (BUYS et al., 1989, COLLINS et al., 1986, COOK et al., 1987, GIRAUD et al., 1986, HAFEZ and WEILAND, 1990, WYETH et al., 1986). The TRT virus appears to be highly sensitive to different chemical disinfectants. Preparations of Lysovet-PA (Disinfectant based on Aldehyde, Phenol and Alcohol), VENNO-VET-1 (Disinfectant based on different organic acids) and H₂O₂ were able to inactivate the virus at concentrations of 0.5% within 15 minutes (HAFEZ and ARNS, 1991).

Virus multiplication in tracheal organ cultures from chicken and turkey embryos results in a ciliostatic effect after several passages (WILDING et al., 1986). The virus propagates in chicken embryos after yolk sac inoculation (ALEXANDER, 1991; Buys et al., 1989). Several blind passages (3–5) are necessary to induce embryo mortality. The virus can also be isolated in different cell lines, such as monkey kidney cell line — VERO (Buys et al., 1989, GIRAUD et al., 1986) or chicken embryo rough cell line — CER (HAFEZ and WEILAND, 1990). After several passages, a cytopathic effect with syncytium formation could be detected 5–6 days post infection. After initial isolation, the virus can be adapted to grow in chicken embryo fibroblasts, chicken kidney cells, BS-C-1 and QT-35-cell line (D’APRILE, 1989).

Transmission

The disease is spread by direct and indirect contact. Egg transmission has not been detected and, if it occurs, it can not be significant.

Disease signs

Clinical signs of the SHS could be observed in broilers, broiler breeders, layers, guinea fowl, pheasants and turkeys. The severity of clinical signs, duration of the disease and mortality are extremely variable and are influenced by many environmental factors such as poor management, inadequate ventilation, high stocking density, poor litter conditions, poor hygiene, high ammonia level, concurrent diseases and the type of secondary bacterial infection.

The virus appears to be capable to infect chickens and turkeys of any age. However, clinical signs in broilers have
been observed mostly between 4 and 6 weeks of age. The disease lasts from 2–3 weeks with a mortality rate of 1–5% under favourable conditions; if complicated with other adverse conditions or respiratory problems, the rate can be as high as 20–30%. The clinical signs identified are depression, decrease in food intake, nasal sneezing, coughing which progresses to conjunctivitis, followed by facial oedema starting around the eye extending over the head and descending to the submandibular tissue. The symptoms are mostly accompanied with varying degree of swelling of the infraorbital sinuses.

In Broiler breeders the disease primarily affects the birds at the peak of production or soon before entering production,- mostly between 24 and 52 weeks of age. Before the main symptoms are detected, a slight increase in mortality and decrease in feed intake may be observed. The first signs are apathy, mild respiratory rales, sneezing, conjunctivitis, uni- or bi-lateral facial swelling, which ascends over the head. These conditions are followed by cerebral disorientation, torticollis and opisthotonus. Many of the affected birds which show nervous signs are unable to move, show severe prostration, completely cease food and water intake and usually die as a result of starvation. The morbidity rate ranges between 8 and 10%, while mortality is variable and relatively low; about 1–3% in uncomplicated cases. The symptoms are generally accompanied with a drop in egg production reaching 1–10% for 2 to 3 weeks. Fertility and hatchability are unaffected in many cases. However, some increases in embryos mortality in the incubators (between 3 and 10%) with a low hatchability rate have been observed (O'BRIEN 1985, PELETIER, 1991).

Similar symptoms have been observed in layer flocks with morbidity up to 8% and a variable mortality rate from negligible to 2%. The symptoms are mostly accompanied with low egg production (between 2 to 4%) and in some cases poor shell quality (2%).

Table 1. Swollen Head Syndrome: Observations in different countries

<table>
<thead>
<tr>
<th>Country</th>
<th>Birds</th>
<th>Authors</th>
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<tbody>
<tr>
<td>South Africa</td>
<td>Broilers</td>
<td>MORELY and THOMSON (1984)</td>
</tr>
<tr>
<td>Spain</td>
<td>Broilers</td>
<td>DIAZ ESPADA et al. (1984)</td>
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<td>France</td>
<td>Hens</td>
<td>DROVIN et al. (1985)</td>
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<tr>
<td>UK</td>
<td>Broiler breeders</td>
<td>O'BRIEN (1985)</td>
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<td></td>
<td>Broiler breeders</td>
<td>WYETH et al. (1987)</td>
</tr>
<tr>
<td></td>
<td>Broilers</td>
<td>GOREN (1985)</td>
</tr>
<tr>
<td></td>
<td>Layers</td>
<td>GOREN (1985)</td>
</tr>
<tr>
<td>Canada</td>
<td>Broilers</td>
<td>ZELLEN (1988)</td>
</tr>
<tr>
<td>Israel</td>
<td>Broiler breeders</td>
<td>PERELMAN et al. (1988)</td>
</tr>
<tr>
<td>Germany</td>
<td>Broilers</td>
<td>HAFEZ (1988)</td>
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<tr>
<td>Morocco</td>
<td>Broilers</td>
<td>WYETH (1990)</td>
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<td>Broiler breeders</td>
<td>EL HOUDAPE et al. (1991)</td>
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<td>Mexico</td>
<td>Broiler breeders</td>
<td>DECANINI et al. (1991)</td>
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<td>Brazil</td>
<td>Broilers</td>
<td>ARNS and HAFEZ (1992)</td>
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<td></td>
<td>Broiler breeders</td>
<td>ARNS and HAFEZ (1992)</td>
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Pathology

Post mortem examinations have found lesions located in the head region and revealed a caseous haemorrhagic exudate in the subcutis. In addition, mild rhinitis, tracheitis and to lesser extent sinusitis has been observed. The involvement of airsacs is variable; but, as secondary bacterial infection supervenes, the airsacculitis becomes more severe and is usually accompanied with percarditis. In birds showing nervous manifestations, oitis of the middle ear with pus accumulation has been constantly observed. Inflammation of the ovary, oviduct and peritoneum has also been detected in affected layers.

According to SAN GABRIEL (1984), microscopical lesions in the brain tissue consist of marked gliosis, with hyperaemia, discrete perivascular cuffings and small haemorrhages. Degenerative changes were only noted in the Purkinje cells of the cerebellum. Furthermore, cellulitis, periostitis and purulent inflammation of the air spaces of the calvarial cancellous bone have been found consistently. Otitis externa and interna, meningitis and sinusitis have been observed less frequently (PATTISON et al., 1989).

Immune reaction

Previous investigations involving detection of TRT antibodies in the sera of different avian species have demonstrated that antibodies to TRT-virus were in sera from broilers, boiler breeders, layers and conventional guinea fowl which were either affected with SHS or from apparently healthy flocks (COOK et al., 1988, HAFEZ and LOHREN, 1990, PICAUT et al., 1987a, b, WYETH et al., 1987). Additional studies, however, have also demonstrated a lack of correlation between the serological results and clinical history. COOK et al. (1988) concluded that there is no association between the presence of TRT antibodies in chicken sera and any particular disease condition, and that TRT-virus can infect chickens without necessarily being responsible for clinical disease. On the other hand, PICAUT et al. (1987b) were able to increase the severity of clinical signs in experimentally infected chickens using additional infection with E. coli [078 K 80] and pointed out the importance of secondary infection in this syndrome. This may explain why some flocks have TRT antibodies without having shown any clinical disease signs. In addition, according to MORLEY and THOMSON (1984), failure to reproduce SHS by inoculating chickens in isolators is not entirely surprising, since heavy stocking density, high ammonia level and low ventilation rates influence the severity of the syndrome. These factors are absent in isolation facilities and in most of the well managed breeder flocks. In broiler breeders reinfecction occurs more than one time. Reinfecction is accompanied with a remarkable increase in antibody titer without clinical signs or drops in production (HAFEZ and WEMMER, unpublished data).

Examination of paired serum samples have revealed a significant increase in the number of positive sera with TRT antibodies in flocks with SHS signs, but not in flocks with respiratory manifestations. This indicates exposure to the TRT virus (HAFEZ and LOHREN, 1990; WYETH et al., 1987).

The titer of maternal antibodies in one day old chicks (meat type) is directly correlated to the antibody titer in parent flocks. The maternal antibodies persist on the average until the age of 15 to 20 days (HAFEZ and WEMMER, unpublished data).
Presently, no data are available on the persistence of humoral antibodies in chickens after infection. However, antibodies to the TRT virus could be detected 2–4 weeks after SHS infection using ELISA or serum neutralization tests. In addition, no information is available on the persistence of antibody level and its protection effect on birds after infection. Furthermore, little is known about the existence and persistence of local active immunity.

Diagnosis

Diagnosis of SHS on the basis of clinical features and pathological lesions is often difficult since they may be confused with other infectious conditions. Proof of infection therefore must be confirmed by laboratory methods.

Virus isolation and Virus detection

Recommended tissues for virus isolation are sinuses and nasal exudate, larynx, trachea and lungs from birds in the early stage of the disease. It is important to collect the samples as early as possible after infection. Isolation of the virus is less successful from birds showing severe signs, since secondary bacterial infections, especially E. coli, become dominant.

The virus can be successfully isolated from filtered homogenates of infected tissues after several passages in tracheal organ cultures from chicken embryos (Picault et al., 1987a), in embryonated chicken eggs after yolk sac inoculation, in VERO cell lines (Bays et al., 1989) and chicken embryo rough cell (CER) lines (Hafez, 1991 unpublished data). Immunofluorescence and immunoperoxidase tests can be applied to detect viral antigen in tissue sections or cell cultures.

Detection of antibodies

Different serological tests have been used with the aim of detecting antibodies for diagnostic purposes. They include a serum neutralization test, indirect immunofluorescence and the ELISA. However, the ELISA test is widely used since it has been developed in many laboratories and is available commercially as kits. This method has a low cost advantage and provides more rapid results in comparison to the neutralization test (Baxter-Jones et al., 1989; Chettle and Wyeth, 1988, Grant et al., 1987, Hafez and Löhren, 1990, Mc Dougall and Cook, 1986).

Differential diagnosis

Since many infectious disease conditions may produce similar clinical signs such as Newcastle disease, Pasteurella multocida, infectious bronchitis and infectious coryza, careful differential diagnosis must be conducted.

Prophylaxis and control

Treatment and management

Drug therapy directed to the secondary bacterial infections using different antibiotics revealed different results. However, therapy alone is of little value, unless it is accompanied with improvements in all aspects of management; especially the ventilation, stocking density, litter condition and general hygiene.

Vaccination

Two live TRT-vaccines from France and the United Kingdom are currently licensed. Both are prepared by attenuation of TRT-virus isolates obtained from turkeys.

In general, application of the live vaccines in turkeys, broiler breeders and layers appears to give good protection with low antibody responses. The level of antibodies is poorly correlated with protection. The vaccines are able to protect the birds against clinical signs and sharply reduce the economic losses.

Vaccination trials using inactivated vaccine alone or in combination with the live attenuated vaccine were conducted recently.

Chettle (1991) reported on the use of killed oil emulsion vaccine prepared from TRT-Virus in 2 broiler breeder flocks (At Central Veterinary Laboratory, Weybridge, Surrey, England). The vaccine was applied as a single dose, subcutaneously at twelve weeks of age. Sixteen weeks after vaccination, a marked drop in egg production accompanied by typical signs of SHS occurred in an unvaccinated control flock kept on the same farm in an other house, while none of the vaccinated chickens showed either clinical signs of SHS or a drop in production. In both flocks high increases of antibodies to TRT were observed. Through 47 weeks of age the vaccinated chickens gave an average of 102.4 settable eggs/bird, while the unvaccinated flock produced only 97.9 eggs/bird. The hatchability rate in vaccinated flocks was 88.8% and 86.9% in unvaccinated flocks.

A vaccination programme using live and inactivated vaccines prepared by Rhone Merieux was applied in France and more than two million breeders were immunized (Goater, 1991). The birds were vaccinated at 12 weeks of age by eye drop or spray using live vaccine, then boosted with an inactivated oil based vaccine at 16 (layer type) or 20 (meat type) weeks of age. Results showed that the vaccine had no adverse effect on birds although respiratory reaction was occasionally observed. Vaccinated flocks had higher egg production, lower mortality rates and less susceptibility to environmental changes in comparison to non vaccinated flocks housed at the same time in the same area.

The vaccination response based on serological conversion is mostly lower when there is a competition between TRT live vaccine and infectious bronchitis (IB) field viruses. According to Gaudry (1991), IB vaccine virus strain H 120 as well as Newcastle disease vaccine virus strain Hetchner B-1 are also able to inhibit the growth of TRT vaccine strain. Goater (1991) pointed out that the live vaccine must be applied alone, while inactivated vaccine can be associated with an oil based multivalent vaccine. The author concluded that better vaccine regime against IB strains, and maybe against E. coli, will be also necessary to provide a good vaccination against SHS, and that commercial layers kept in free ranges must be vaccinated in the same manner as breeders.

Currently, no vaccine has been of any benefit in SHS diseased broiler flocks, since some IB field or vaccine strains may in certain circumstances inhibit the multiplication of pneumovirus vaccine strain in the upper respiratory tract (Gaudry, 1991). El Houadfi et al. (1991) vaccinated broiler flocks at the first day of age with 1/3 dose of inactivated commercial TRT vaccine combined with Newcastle disease vaccine (OVO2, Rhône-Merieux) and a full dose of live attenuated TRT vaccine (AVIFFA-TRT, Rhône-Merieux) by eye drop. The applied vaccine programme against SHS did not result in protection of the flocks at 7 weeks of age.

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Conclusion

In conclusion, further work is required to determine the real role of pneumovirus infection in respiratory diseases complex in the poultry industry. Efficient vaccine methods and control programmes for different poultry production lines also need to be established, based on the findings of further work.

Summary

A pneumovirus is known to be the primary causative agent of turkey rhinotracheitis (TRT) and appears to be associated with swollen head syndrome (SHS), egg production losses and respiratory diseases complex in chickens. The infection in chicken causes high economic losses by increasing mortality, increasing medication costs, dropping in egg production, reduction of egg shell quality, and decreasing hatchability.

This paper reviews the literature related to SHS aetiology, mode of transmission, clinical signs, pathology, immune reaction, and diagnosis in chickens. Furthermore this review summarizes the literature available on vaccination in broiler and breeder flocks (meat and layer types).

Die Rolle des Pneumovirus beim Swollen Head Syndrome der Hühner: Übersicht

H. M. Hafez

Zusammenfassung

Ein Pneumovirus, das als Erreger der Rhinotracheitis der Puten (TRT) bekannt ist, scheint auch an dem Swollen Head Syndrome (SHS), Legeleistungsseinbußen und Atemwegsinfektionen bei Hühnern beteiligt zu sein. Von großer wirtschaftlicher Bedeutung ist die Infektion wegen erhöhter Tierverluste, erhöhtem Medikamentenbedarf, Legeleistungsabfall, vermindertem Schalenqualität und Brutfähigkeit.

Die vorliegende Arbeit gibt eine Literaturübersicht bezüglich der Ätiologie, Übertragung, Symptome, Pathologie, Immunologie und Diagnose des Swollen Head Syndrome. Darüberhinaus wird die verfügbare Literatur hinsichtlich der SHS-Impfung in Mehl- und Zuchtkohlen (Fleisch- und Legetyp) zusammengefasst.

Stichworte

Huhn, Pute, Pneumovirus, Swollen head syndrom, Serologie, Diagnose, Vakzination, TRT

References


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Die Aktivität der L-Gulonolactonoxidase in der Niere sowie der Gehalt an Ascorbinsäure in der Niere, in der Leber und im Plasma von Hühnern, Enten, Gänsern und Wachteln

The activity of the L-gulonolactone-oxidase in the kidney and the content of ascorbic acid in the kidney, in the liver and in the plasma of chickens, ducks, geese and quails

A. Alawad, E. Kolb und M. Wahren

Manuskript eingegangen am 31. August 1992

Einleitung


Bei der Bildung der Ascorbinsäure aus Glucuronat sind 3 Enzyme beteiligt, von denen die L-Gulonolactonoxidase eine Schlüsselrolle einnimmt; sie katalysiert folgende Reaktion (Grollmann und Lehninger, 1957).

\[ \text{L-Gulono-δ-lacton + NAD}^+ \rightarrow \text{L-Ascorbat} + \text{NADH + H}^+ \]

Untersuchungen am Überstand der Homogenate von Nieren von Hühnchen unterschiedlichen Alters zeigten, daß die