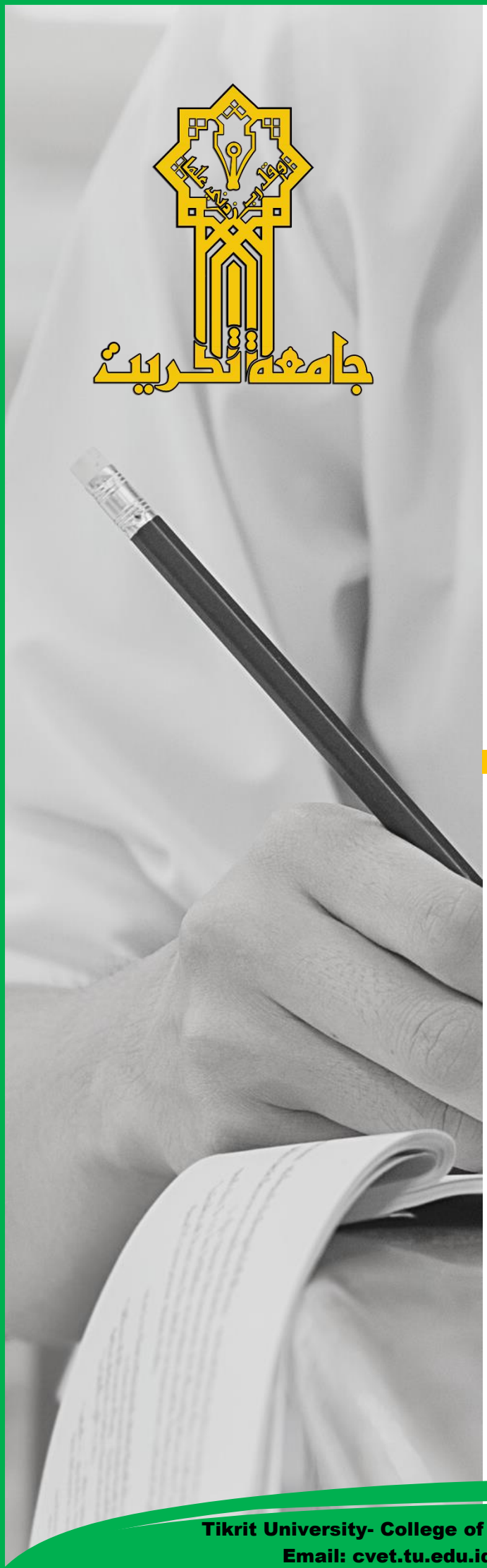

Tikrit University
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Viral Enteric Infections 2- Rotavirus Infections

Subject name: Poultry Diseases

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Viral Enteric Infections
2-Rotavirus Infections

Summary

Rotavirus infection in avian species is shows diarrhea and general flock depression, and recognized as enteric syndrome in poultry. The virus belongs to Reoviridae family, which is characterized by virions that contain 10–12 linear double-stranded RNA (dsRNA) segments. The rotavirus genome consists of 11 segments of dsRNA with conserved 5' and 3' ends, and each genome segment codes for a single protein with the exception of segment 11, which codes for two. The genome is enclosed by a triple-layered capsid (virion) with a characteristic wheel-like appearance (rota=wheel) when viewed via electron microscopy.

Mature prototypical mammalian rotavirus virions are nonenveloped icosahedrons and have an overall diameter of approximately 1000Å (100nm), including the VP4 cell attachment protein "spikes", while the diameter of the outer shell of the rotavirus virion excluding the protruding spikes is approximately 750Å. The smooth appearance of the outer protein shell distinguishes the rotaviruses from other members of the Reoviridae when viewed via electron microscopy. avian rotaviruses have genome segments ranging from approximately 3.3kb to 700bp, with a total genome size of approximately 19kb.

Susceptibility to Chemical and Physical Agents

There are no changes in viral titers after a 30 minute treatment with chloroform; treatment at pH3.0 reduced the titer of both isolates approximately 100-fold by 8 hours of treatment. Titers were reduced by about 100-fold after treatment at 56°C for 30 minutes, but neither virus was inactivated following 8 hours at 56°C. The virus was inactivated after incubation at 82.2°C for 140 seconds, but was initially resistant to heating in a thermal cycler gradient from 25°C to 50°C. The virus also inactivated after a 6-hour, 82.2°C incubation. Heating to 50 °C reduced the titer 10 times and heating to 50°C in the presence of 1MMgCl2 further reduced titers 1000 times. The infectivity was resistant to 20% ether, 10% chloroform, and 0.1% sodium deoxycholate. phenolic- based disinfectant effectively reduced the titer of rotavirus, even in the presence of a significant amount of organic matter, while glutaraldehyde- and peroxygen-based disinfectants only showed effective reductions in titer in the absence of organic material.

Public Health Significance: There are no reports of ARV as being zoonotic agents.

Replication

Rotavirus infected cells display characteristic electron- dense **cytoplasmic inclusion** bodies called **viroplasms**, which are viral factories in which genome replication and initial packaging of the viral genome segments occur. Viroplasm formation is directed by the affinity of certain rotavirus structural proteins for newly translated positive-sense RNA **[(+)RNA].** The replication of the dsRNA genome takes place fully **within** the newly formed viral cores; the dsRNA genome is thus **never exposed** to the **interior of the cell**. The association of the rotavirus transcriptase complex with the intact viral core also allows the transcription of each genome segment to occur **without complete uncoating of the dsRNA genome**. A group D rotavirus produced electron-dense viroplasms and mature viral particles in pheasant chick duodenal enterocytes by seven days postinfection (DPI), while viroplasms and virions in **rough ER** were observed by 8DPI in chicken villous **epithelial cells** infected with an antigenically novel rotavirus. Similarly, PO-13 rotavirus produced virions in the rough ER of MA-104 cells.

Transmission

Horizontally and probable vertically.

Incubation Period:

The incubation period 2–3 days.

Clinical Signs:

1- crude intestinal homogenates

2- No mortality occurred in experimentally infected turkeys or chickens.

3- Mild or no clinical signs were observed following experimental infection of chickens.

4- Chicks had mild diarrhea or passed increased quantities of cecal droppings.

5- Drop in egg production 4–9 DPI.

6- Subclinical infections to outbreaks of severe diarrhea with associated dehydration, poor weight gain, and increased mortality.

7- Poults, clinical signs include very mild diarrhea in the first week of life. 8-Mortality between 4 and 7%.

Gross (P.M.) lesions:

1- Abnormal amounts of fluid and gas in the intestinal tract and ceca.

2- Pallor of the intestinal tract accompanied by loss of tonicity may be evident.

3- Dehydration, stunting of growth, pasted vents, inflamed vents, anemia due to vent pecking, litter in the gizzard, and inflammation of the feet.

4- Hemorrhages were observed in the cecal walls.

5- Discrete, multifocal, superficial, brownish-red erosions were found in the duodenum and jejunum of turkeys experimentally infected at 84 and 112 days of age.

Histologic changes:

1- The virus replication in the cytoplasm of mature villous absorptive epithelial cells in the small intestine.

2- Infected cells were most numerous in the distal third of villi.

3- Small numbers of infected cells were detected in colon epithelium, cecal tonsils, and lamina propria of some villi.

4- No IF was observed in proventriculus, gizzard, spleen, liver, or kidney. 5-Rotavirus grew best in the duodenum, the jejunum and ileum.

6-Basal vacuolation of enterocytes, separation of enterocytes from the lamina propria with subsequent desquamation, villous atrophy accompanied by widening of the lamina propria, scalloping of the villus surface, fusion of villi, and leukocytic infiltration of the lamina propria.

7-In general, mean villous lengths were decreased and crypt depths were increased following experimental infection; morphometric changes were more pronounced in the duodenum and jejunum than in the ileum.

8- There was infiltration of polymorphonuclear and mononuclear cells into the lamina propria of the cecum and colon in some birds.

9- Roughened villous surfaces, irregularly shaped and sized villi, and loss of microvilli in enterocytes located at the tips of villi.

10- Rotavirus was detected in the distal portion of the villi in the jejunum of experimentally infected poults, and separation of enterocytes from the lamina propria was evident at 4 DPI.

11- Minimal leukocytic infiltration of the lamina propria, with minimal loss of microvilli on cells at villus tips was observed.

12- Moderate villous atrophy, mainly in the ileum

Pathogenesis of the Infectious Process

With both avian and mammalian rotaviruses, the target cells are mature columnar absorptive cells that are located in the villous epithelium. The cell attachment protein, indicate that when initiating infection avian rotaviruses utilize sialic acid-containing molecules as receptors on the surface of cells. There is histological evidence that rotavirus infection of enterocytes in the turkey jejunum leads to destruction of the distal portion of the villi. Structural damage to the villi ("scalloping") is also observed in the turkey duodenum during rotavirus infection. The frothy fluids found in the ceca of infected birds may result from impaired absorption of carbohydrates that leads to their fermentation by cecal bacteria, producing metabolites that draw water into the ceca by osmosis. However, malabsorption may not be the only explanation for rotavirus-induced diarrhea. NSP4 proteins of mammalian rotaviruses are enterotoxins, causing diarrhea in suckling mice. Avian rotavirus NSP4 glycoproteins have similar biological activity, and there are conserved structural regions in the NSP4 enterotoxin domain between avian and mammalian rotaviruses.

Immunity

Active

Chickens and turkeys inoculated orally with rotaviruses showed serum antibody responses as early as 4–6 DPI measured by indirect IF. In general, older birds developed higher antibody titers and responded more quickly than younger birds. Using immunoglobulin class-specific enzyme-linked immunosorbent assays (ELISAs) to follow antibody responses in chickens experimentally infected with a group A rotavirus, rotavirus-specific IgM, IgG, and IgA were detected in serum, while the intestinal antibody response consisted almost entirely of IgA. Natural killer cell-like activity has been demonstrated in chick intraepithelial leukocytes against rotavirus-infected target cells, and this may be an impor□tant in vivo immune response

Passive

Maternal antibodies to rotavirus are passively transferred to the avian embryo through the egg yolk and are detectable until about 3–4 weeks of age. Progeny of hyperimmunized turkey hens were more resistant to experimental infection with rotavirus at 2 or 5 days of age, but not at 12 days of age. Circulating maternally- derived IgG may protect the intestinal mucosa against rotavirus infection in 1–7 day old poults . During the first week of life, maternally-derived anti-rotavirus IgG titers in intestinal washings of poults derived from hyperimmunized (vaccinated) hens were 200–500-fold less than titers in serum. The presence of maternal antibody in the serum in two other studies had no apparent effect on susceptibility of chickens and turkeys to experimental group A rotavirus infection. Maternally derived IgG could not be detected in intestinal washings of progeny derived from naturally infected hens. Similarly, an increase in serum neutralizing antibodies was observed in pheasant hens vaccinated with an inactivated group A pheasant rotavirus vaccine. These results and those cited previously for the progeny of vaccinated turkeys suggest: (1) that maternally derived antibodies in the progeny of unvaccinated turkeys and pheasants are unlikely to provide significant protection against a field challenge with rotavirus; and (2) that much higher titers of antibody would need to be produced by vaccination to completely protect young birds even for the first week of life.

Diagnosis:

1-Clinical signs and gross and histologic lesions. 2-virus isolation

3- RT-PCR.

4-ELISA (group A)

Differential Diagnosis

Diarrhea

Treatment

1-Antibiotics: 2-Anti-inflammatory:3-Supplements:

Vaccination

No licensed vaccine is available.