Tikrit University
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Viral Enteric Infections
1-Turkey Coronavirus Enteritis

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1-Turkey Coronavirus Enteritis

Summary

Turkey coronavirus (TCV) is the cause of enteritis in young turkeys and decreased egg production in turkey breeder hens. Turkey coronavirus (TCV) is the cause of an acute highly contagious enteric disease of turkeys characterized by depression, anorexia, diarrhea, and decreased weight gain . Bluecomb disease, transmissible enteritis, and coronaviral enteritis are synonyms of TCV enteritis of turkeys.

Turkey coronavirus is classified as a member of the Coronaviridae. The Coronaviridae comprise a large family of RNA-containing viruses that infect a wide variety of avian and mammalian species. The Coronaviridae is in the order Nidovirales, an order composed of viruses having linear, nonsegmented, positive-sense, single-stranded RNA genomes with similar genomic organization and nested sets of subgenomic mRNAs. The coronavirus genome consists of an RNA molecule that is approximately 28 kilobases in size. Coronaviruses possesses four major structural proteins referred to as spike (surface) glycoprotein (90–180 kilodaltons [kDa]), integral membrane protein (20–35kDa), small envelope protein (12.5 kDa), and nucleocapsid protein (50–60kDa). Some coronaviruses also contain a fifth major structural protein, the hemagglutinin-esterase (120–140kDa).

Coronaviruses have been subdivided into four genera based on serological and nucleotide sequence analyses. Infectious bronchitis virus (IBV) and TCV are members of the Gammacoronavirus genus Turkey coronavirus has been shown to be closely related to IBV based on antigenic and nucleotide sequence analyses. Studies have indicated a high degree of sequence identity between integral membrane protein, nucleocapsid protein and polymerase (ORF1b) genes of TCV and IBV, but only limited sequence identity between TCV and IBV spike glycoproteins.

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

Replication

Turkey coronavirus replicates primarily in enterocytes in the jejunum and ileum , and epithelium of the bursa of Fabricius. Viral antigens in intestinal enterocytes were found predominately in enterocytes lining the upper one-half to two-thirds of intestinal villi. In the bursa of Fabricius, viral antigens are found in both follicular and interfollicular epithelium of the bursa of Fabricius ; viral antigens are not found in bursal lymphoid follicles. In inoculated embryos, virus replication occurs exclusively in intestinal epithelial cells and bursa of Fabricius; virus replication has not been detected in allantoic, yolk, or amniotic membranes. Thin-section electron microscopy of intestines from TCV-infected embryos and poults has shown that TCV replication occurs in the cytoplasm. Turkey coronavirus acquires its envelope by a process of budding through membranes of the endoplasmic reticulum and Golgi apparatus; virus particles accumulate in cisternae of the endoplasmic reticulum

Susceptibility to Chemical and Physical Agents

Turkey coronavirus was demonstrated to be stable at pH3 at 22°Cfor 30min, and resistant to 50°C for 1 hour, even in the presence of 1M magnesium sulfate. Chloroform treatment at 4°C for 10 minutes readily inactivated the virus. Turkey coronavirus remained viable when stored in intestinal tissues at −20°C or lower for over five years. The virus was shown to survive in buildings and ranges for extended periods of time even after turkeys were removed from these premises. Storage of the virus for 10 days at 21.6 °C resulted in inactivation, but the virus survived for up to 20 days when stored at 4°C. Saponified cresol and formaldehyde were shown to be effective disinfectants for eliminating TCV from contaminated buildings.

Pathobiology and Epizootiology

1- ncidence and Distribution

Turkey coronavirus has been identified in turkeys in the United States, Canada, Brazil, Italy, the United Kingdom, France, Poland, Turkey, and Australia (17, 18, 20, 54, 59, 63, 81). The virus has been identified in most turkey producing regions of the United States.

2- Transmission, Carriers, Vectors

Horizontally

Incubation Period:

The incubation period may vary from 15 days, but typically is 2–3 days.

Clinical Signs:

In field cases, clinical signs occur suddenly, usually with high morbidity. Birds exhibit depression, anorexia, decreased water consumption, watery diarrhea, dehydration, hypothermia, and weight loss. Droppings typically are green to brown, watery, frothy, and may contain mucus and urates. Flocks infected with TCV experience increased mortality, growth depression, and poor feed conversion compared with uninfected flocks. Mortality is variable in affected flocks; high mortality may occur depending on the age of the birds, concurrent infection, and management practices. Experimental studies using egg-adapted strains of TCV indicate that TCV infection results only in mild disease and moderate growth depression; mortality generally is negligible. Turkey coronavirus infection of turkey breeder hens during production is a potential cause of decreased egg production. In experimental studies, TCV infection of turkey breeder hens resulted in transient drops in egg production; however, combined infection with TCV and turkey astrovirus resulted in prolonged and more severe drops in egg production compared with hens infected with TCV alone.

Gross (P.M.) lesions:

Gross lesions are seen primarily in intestines and bursa of Fabricius. Duodenum and jejunum generally are pale and flaccid; ceca are distended and filled with watery contents. Emaciation, dehydration, and atrophy of the bursa of Fabricius may be observed.

Histologic changes:

Microscopic lesions are observed in intestines and bursa of Fabricius of TCV-infected turkeys. In intestines, microscopic lesions in experimentally infected turkeys consist of decreased villous length, increased crypt depth, and decreased intestinal diameter. The columnar epithelium of intestinal villi changes to a cuboidal epithelium and these cells exhibit a loss of microvilli. There is a decrease in number of goblet cells, separation of enterocytes from lamina propria, and infiltration of lamina propria with heterophils and lymphocytes. Epithelial repair is evident beginning at 5 days PI, and complete by 21 days PI. By 5 days PI, columnar epithelium with microvilli begins to replace cuboidal cells, and goblet cells begin to reappear. In the bursa of Fabricius, changes in epithelial cells are evident by 2 days PI and consist of epithelial necrosis and hyperplasia. The normal pseudostratified columnar epithelium of the bursa of Fabricius is replaced by a stratified squamous epithelium. Intense heterophilic inflammation is observed

within and subjacent to the epithelium. Moderate lymphoid atrophy of bursal follicles is observed Ultrastructural changes in intestines of TCV-infected turkeys are confined to epithelial cells . Ultrastructural changes include loss of microvilli, disruption of the terminal web region, degeneration of mitochondria, dilation of cisternae in the endoplasmic reticulum, increases in intracellular lipid, excessive sloughing of cells at villous tips, and shortening of villi. Coronavirus particles (80– 140nm in diameter) are observed within cisternae of the endoplasmic reticulum

Pathogenesis of the Infectious Process

Turkey coronavirus replicates preferentially in enterocytes lining the apical portions of intestinal villi and in epithelium of the bursa of Fabricius. The site of intestinal TCV infection suggests that the virus may cause diarrhea in a manner similar to other enteric coronaviruses. Malabsorption, maldigestion and diarrhea likely result from TCV-induced destruction of villous epithelium; however, the virus may exert its effects in a more subtle manner through alterations in the physiology of these cells. Turkey coronavirus also may exert its effects by altering the normal intestinal flora. Severe disease characterized by high mortality was a common feature of early descriptions of TCV infection (bluecomb disease) and early experimental studies using inocula composed of crude fecal/intestinal homogenates. More recent experimental studies using embryo- propagated TCV indicate that mortality due to TCV infection usually is negligible, at least under laboratory conditions. Management practices, crowding, and secondary infections may exacerbate the effects of TCV infection and result in increased losses. Antibiotics have been shown to reduce mortality in TCV-infected flocks, most likely because they control secondary bacterial infections. Experimental studies with TCV and an enteropathogenic strain of Escherichia coli provide evidence suggesting an interaction between TCV and bacteria in the development of severe clinical disease. In these experiments, young turkeys inoculated with only TCV developed moderate growth depression without significant mortality, and turkeys inoculated with only enteropathogenic E. coli did not develop clinically apparent disease. However, turkeys dually inoculated with TCV and enteropathogenic E. coli developed severe growth depression and high mortality.

Immunity

Active

Turkeys that recover from TCV infection are resistant to subsequent challenge. Turkeys that survived experimental TCV infection at 4 days of age showed no clinical signs when challenged at 11 and 22 weeks of age. Field observations indicate that flocks that recover from TCV infection are resistant to subsequent infection. The nature of protective immunity in recovered birds is not fully understood. Specific secretory IgA, humoral, and T-cell mediated immunity have been demonstrated in recovered turkeys. Specific secretory IgA was shown to persist in intestinal secretions and bile of recovered turkeys for at least six months. Concentrations of specific secretory IgA antibodies in feces of TCV-infected turkeys peaked at 3–4 weeks PI and disappeared at approximately 6 weeks PI.

Passive

Poults passively immunized against TCV by subcutaneous inoculation of serum from immune birds were not protected from challenge. Poults from immune and nonimmune breeder hens were equally susceptible to TCV challenge.

Diagnosis:

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

3- RT-PCR.

Differential Diagnosis

Enteric disease caused by TCV must be distinguished from other enteric diseases of turkeys, particularly those caused by other viruses, bacteria, and protozoa.

Treatment

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

Vaccination

No licensed vaccine is available.