

Transmissible gastroenteritis: a fatal swine enteric coronavirus disease

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1.1 Introduction

Transmissible gastroenteritis (TGE), an infectious and contagious enteric viral disease of pigs is caused by TGE virus (TGEV) of the genus *Alphacoronavirus*, family *Coronaviridae* (OIE, 2018). The disease is characterised by vomiting, profuse watery diarrhoea, dehydration and high mortality that may reach upto 100% in the piglets under two weeks of age (Doyle and Hutchings, 1946). The aetiological agent i.e. the TGEV is mostly circular in shape with a diameter ranging from 65 to 90 nm and has surface projections (Pensaert *et al.*, 1970). TGEV is an enveloped virus containing single-stranded, the positive-sense RNA genome of about 28.5 kb. TGEV genome is capped at the 5'-proximal end and has a polyadenylated tail at the 3'-end. The virion RNA is infectious and serves as both the genome and viral messenger RNA. The TGEV genome is comprised of nine open reading frames (ORFs) including five ORFs (ORF1a, ORF1b, ORF3a, ORF3b, and ORF7) which code for non-structural proteins and four ORFs that code for structural proteins *viz.* spike (S), envelope (E), membrane (M) and nucleoprotein (N) (Wang *et al.*, 2010).

TGEV is most often transmitted by the faecal-oral route. It is excreted in large quantum in faeces of clinically diseased pigs. Thus, faeces act as the most common source of TGEV transmission directly through carrier pigs or indirectly through mechanical means (OIE, 2018). Pig is the only known natural host for the disease.

TGE is worldwide in occurrence and has been reported from almost all continents except in Australia and Antarctica. Till now, the occurrence of TGE has not been reported from India. However, due to its prevalence in swine population of neighbouring China and in other Southeast Asian countries, there is a risk of its emergence into India.



1.2 Disease prevalence

TGE was first reported from the United States in the year 1946 (Doyle and Hutchings, 1946) and later it has spread to almost all the continents and reported from North America (Canada and Mexico), Central America (Cuba), South America (Brazil and Argentina), Europe (Germany, France, Belgium, The Netherlands, Spain, England, Belarus and Slovakia), Russia, Asia (Japan and China) and Africa (South Africa and Uganda) (Hu *et al.*, 2015; Pineyro *et al.*, 2018). Due to the emergence of porcine respiratory coronavirus (PRCV), a deletion mutant of TGEV, the occurrence of TGE has become sporadic (Saif and Sestak, 2006).

In the recent past, sporadic occurrence of TGE has been reported from China (Yuan *et al.*, 2021). However, disease outbreaks still occur in immunologically naive herds. An epidemic TGE in China revealed the emergence of new TGEV strain that closely resembled with the US strain, which indicates the mutating ability of TGEV (Kanner-Acerbo and Lowe, 2016).

1.3 Current status in India

Clinical occurrence of TGE has not been reported from India. However, a surveillance study showed the serological evidence of TGEV in Assam and Uttar Pradesh states (Barman *et al.*, 2003). Moreover, during a limited surveillance study to monitor swine enteric viruses in Indian swine population, we have also observed the antibodies against TGEV (unpublished data). It indicates possibilities of exposure of Indian pig population with TGEV at certain point of time. Moreover, due to occurrence of TGE in swine population of China and other Asian countries, there is always a risk of its emergence into India.

1.4 Impact on animal production and trade

Economic loss to the swine industry due to the occurrence of TGE is attributed directly because of the mortality of pigs and production losses. Further economic losses occur because of the cost of vaccination and biosecurity. A substantial loss of 260 to 330 US dollars per breeding sow during 12 months after TGEV infection was predicted in an economic analysis study from Australia (Mullan *et al.*, 1994).

TGE is in the list of diseases notifiable to the WOA (OIE). Due to its occurrence in neighbouring countries, the border areas of North-Eastern states are always at high risk for the spread of this disease into India. Being the important disease in swine rearing countries, it is essential to prevent the entry of TGE across the border.



1.5 Prevention and control

Acute TGE can be diagnosed based on the clinical characteristics of the disease. However, the clinical picture of endemic TGE is not so characteristic to make a diagnosis. TGE must be differentiated from diseases with similar clinical manifestations, such as PED, rotavirus diarrhoea, coccidiosis, colibacillosis, cryptosporidiosis, vomiting and wasting disease, *etc* (Saif and Sestak, 2006). Therefore, confirmatory diagnosis of TGE requires laboratory testing by one or more of the following specific techniques *viz.* immunodiagnostic assays (for antigen and antibodies), specific detection of viral nucleic acid, virus isolation in cell culture and electron microscopy (Saif and Sestak, 2006).

Serological screening of animals is valuable when increasing antibody titre is observed. In a naive herd, even a single seropositive case of TGE has diagnostic value. ELISA and virus neutralisation test (VNT) are most often used for serological diagnosis of TGE. However, antibodies against PRCV can cross-neutralize TGEV and give false-positive results. Therefore, VNT is not recommended for differentiation of TGEV and PRCV antibodies. A monoclonal antibody (MAb)-based competitive ELISA using TGEV spike protein can be applied for differentiation of TGEV and PRCV antibodies, and it is the most acceptable method to qualify animals for trade (OIE, 2018).

Molecular diagnostic tests such as reverse transcriptase-polymerase chain reaction (RT-PCR) and real-time RT-PCR (qRT-PCR) can be used for detection and differentiation of TGEV and PRCV.

Although virus isolation is the most definitive method of TGE diagnosis, it is difficult and less commonly attempted.

Like most other viral diseases, treatment of TGE is not effective (Kanner-Acerbo and Lowe, 2016); however, symptomatic treatment can reduce the severity of clinical symptoms. Increasing herd immunity and application of strict biosecurity measures is the most effective strategy to prevent and control TGE in a herd.

Herd immunity to TGE can be achieved either by exposure of all the animals to the TGEV by feeding them infected intestinal tritirates, or by offering them feed contaminated with the faeces of clinically affected pigs, or by vaccination (Kanner-Acerbo and Lowe, 2016). There are several inactivated and live-attenuated vaccines licensed for immunization against TGE; however, most of the TGE vaccines are not completely protective (OIE, 2018). The main reason for their low efficacy is that these vaccines are unable to stimulate a high level of secretory IgA in milk, unlike the natural infection of TGEV (Saif and Sestak, 2006).

Herd management implementing strict biosecurity measures proves very effective to check the spread of the virus from one farm to another (Kanner-Acerbo and Lowe, 2016).



1.6 Challenges

Development of suitable and effective vaccine producing protective mucosal immunity against TGE is still challenging. Vaccine strategies for TGE should be focused to stimulate the secretory IgA in sow's milk, similar to natural infection. Improving disease outcome by saving the host cells during the clinical course of the disease can be another alternative approach to restore the function of damaged alimentary tract during an outbreak (Kanner-Acerbo and Lowe, 2016). As TGE poses a serious threat to food security and animal health, a worldwide effort to eradicate this disease should be the ultimate goal.

1.7 Conclusion

Transmissible gastroenteritis is a contagious, fatal enteric viral disease in newborn piglets. The disease is caused by transmissible gastroenteritis virus, an *Alphacoronavirus*. It is worldwide in occurrence. Although, TGE is not prevalent in India, however, due to its occurrence in China and other Asian countries, there is always a risk of its emergence into India. Acute TGE can be diagnosed based on the clinical characteristics of the disease. However, confirmatory diagnosis of TGE requires laboratory testing. Development of effective and useful vaccine producing protective mucosal immunity against TGE is still challenging. Biosecurity measures are effective to check the disease spread.

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