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Current information of Menangle virus, a novel zoonotic paramyxovirus: A review

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Abstract

Menangle virus (MenPV) is an enfold paramyxovirus belongs to the genus Rubulavirus. The two known strains of this virus are bat MenPV (bMenPV) and pig MenPV (pMenPV). MenPV isolated in August 1997 subsequent to an outbreak of reproductive disease in a piggery. It is the second formerly uncategorized member of the family *Paramyxoviridae* to be identified in Australia since 1994. MenPV is a zoonotic paramyxovirus causing disease in pigs and humans and was first isolated in 1997 from stillborn piglets at a commercial piggery in New South Wales (NSW), Australia. Neutralizing antibodies to MenPV were detected in several pteropid bat species in Australia and the fruit bats were suspicious to be the source of the virus liable for the outbreak in pigs. In this review, we discuss about the epidemiology, pathogenesis, diagnosis and treatment of MenPV.

Key words: Menangle virus, zoonotic, infection, stillborn piglets, paramyxovirus

1 Introduction

Menangle virus (MenPV), a newly analyze paramyxovirus of pteropid bats and it was isolated in 1997 from still born piglets in the study of a serious outbreak of reproductive disease at a huge commercial piggery in New South Wales, Australia. Natural host of MenV were fruit bats of the genus *Pteropus poliocephalus* (Philbey et al., 1998; Barr et al., 2012). Between 1997 and 1999, there were large outbreaks of disease in pigs in Australia and Malaysia due to infection with viruses that have been shown to be new members of the Paramyxoviridae family. The virus isolated in Australia is now known as Menangle virus (Recently added to the family Paramyxoviridae, subfamily Paramyxovirinae). MenPV isolated from the urine samples of black flying fox, *Pteropus alecto*. MenPV is the etiological carrier of a single epidemic of reproductive disease, which eventuated in a reduction in both the increase in the part of mummified and stillborn piglets as well as the farrowing rate which means the production of litter of pig and the number of live piglet births per litter, irregular abortions. Particularly, there were no clinical signs in perinatal and growing or adult pigs of any age (Kirkland et al., 2001; Love et al., 2001; Philbey et al., 1998; Philbey et al., 2007). Macroscopic pathology in affected stillborn piglets was differentiate by serious degeneration of the brain (especially the cerebral hemispheres, cerebellum and brain stem), and spinal cord as well as brachygnathia, arthrogryposis, and kyphosis (Zvirbliene et al., 2010; Love et al., 2001). An enormous amount of straw-coloured fluid, consistently fibrinous, was exist in the body cavities of almost 40 % of stillborn piglets, and considered pulmonary hypoplasia was further occasionally observed (Philbey et al., 1998; Philbey et al., 2007).

2 Etiology

It is a newly known virus belongs to the genus Rubulavirus in the family Paramyxoviridae (Bowden et.at., 2001; Bowden & Boyle 2005). Virions are pleomorphic with both elongated and spherical forms that vary in size within 150-350 nm and there is a single layer of surface spikes which around 17 nm in length. Ruptured particles expose long herring bone-shaped nucleocapsids that have a

diameter of relatively 19 nm in diameter (Philbey, A. W et al., 1998). Further closely related Tioman virus, rubulavirus, has been newly identified in flying fox populations in Malaysia; however it has not been associated with any disease (Chua et al., 2001).

3 Epidemiology

This virus does not seem to be as extremely contagious as several porcine pathogens as it took some time to spread by the affected pig population, although all pigs were supposed to be naive. The pervasiveness of high antibody titres to MenPV in finishing age and adult pigs exceeded 95%. Previously the virus had spread through the breeding population, signs of reproductive disease and also foetal infection were no longer evident but the virus remained endemic in the population as a result continuous cycle of infection in young growing age pigs because they lost protection given by maternal antibodies (Kirkland et al., 2001). It has not been possible to show evidence for tenacious infections. The route of infection is unknown; it seems that the virus can be spread by the oral-faecal route (Bowden & Boyle 2005).

4 Clinical Signs

In the pig population by which there was infection with MenV, disease was perceive only where there were breeding females and the reproductive disease was distinguish by reduced litter size, reduced conception rates and the delivery of a huge number of mummified and stillborn fetuses involving some with severe skeletal and craniofacial defects (Kirkland et al., 2006). Congenital defects comprises hydranencephal, arthrogryposis , hypoplasia of the lungs and spinal cord as well as degeneration of the cerebellum, spinal cord, and brain stem (Zvirbliene et al., 2010; Philbey et al., 2007) and there was no evidence of disease in pigs of any age after birth. MenPV is zoonotic which causing a serious febrile illness in farm workers following close contact with infected pigs (Chant, et al., 1998) and also the affected people experienced headaches and significant weight loss.

5 Occurrence and Distribution

This virus caused disease outbreak near Sydney in 1997 (Philbey et al., 1998). Despite, additionally to the large breeding farm, it was also indicate that an active virus transmission on two contract growing farms which received weaned pigs from the breeding farm. There was no other confirmation that MenPV infection found in Australia through following serological surveys of pigs in NSW and also other States, aside from two positive sera that had been assembled at different times at a small farm in northern coastal NSW. This farm had capable a reproductive problem and was close by a flying fox colony. But MenPV had been on this farm, it is entirely possible that the infection had become self-limiting in a small population (Bowden et al., 2012).

6 Pathology

MenPV infected many fetuses that were mummified at birth. Though, there were several stillborn and also aborted fetuses, which exhibit a variety of gross pathological changes involving arthrogryposis, craniofacial and spinal deformities, pulmonary hypoplasia and degeneration of the brain and spinal cord. Wound in the central nervous system (CNS) comprises marked reduction in size of the cerebral hemispheres, cerebellum, spinal cord and brain stem as well as hydranencephaly. The most persistent change was reduced size or absence of the cerebellum. Histologically, changes were most constant observed in the CNS where there was extensive degeneration, infiltration of macrophages, necrosis, and gliosis (Philbey et al., 1998).

7 Diagnosis

Menangle virus is a recently known pathogenic agent for pigs; most porcine populations would be expected to be fully susceptible. The birth of litters, in which there is a marked reduction of normal live piglets and a number of stillborn piglets showing teratologic defects suggest possible MenPV infection. The quickest method of excluding MenPV infection is to test sows for the existence of particular antibody either by virus neutralization or ELISA, using entire virus as antigen. Fetal specimens must be collected for virus isolation, serology, and also pathology. Virus may be isolated from a number of organs of still born piglets, particularly brain, lung, and myocardium. A vast range of cell cultures carry replication of MenPV, except baby hamster kidney cells (BHK21) it has been used for the isolation of the virus from field specimens. Three to five passages are required before cytopathologic changes are noticed. The above changes consist of vacuolation of cells, focal cell lysis and formation of syncytia. As the virus does not hemagglutinate, recognition depends on electron microscopy and its neutralization with distinct antiserum. Neutralizing antibodies can be detected in body cavity fluids of some still born piglet (Kirkland 2017).

8 Differential diagnosis

The birth of litters with mummified fetuses of differing size and stillborn piglets is expressive of an in-utero viral infection. Actually the most ordinary cause of this syndrome in pigs is porcine parvovirus, but a several variety of other viral infections, including , porcine reproductive, encephalomyocarditis virus, Aujeszky's disease, classical swine fever, Japanese encephalitis and respiratory syndrome (Lelystad virus infection), and blue eye (La Piedad Michoacan) paramyxovirus can cause this syndrome (Stephan et al., 1988). A characteristic that differentiate MenPV infection from all but Japanese encephalitis infection is the existence of congenital malformations in the piglets. It should be borne in mind, but, that these malformations are clear in only around one third of affected litters. Many of these other viral infections may also cause disease in both piglets and adults (Philbey et al., 1998)

9 Control

Fruit bats (megachiroptera) are examined the primary source of infection for the pig population and are not found in North America but are present in Africa. It is not known like other bats (microchiroptera) can become infected by MenPV, though it is important to inhibit direct and indirect contact between pigs and bats to restrict introduction of this virus to the pig population. Fruit bats do not enter pig farm buildings, but they defecate and urinate during flight over and around buildings, and sometimes inadvertently drop their young during flight. All outside areas must be covered to prevent possible infection and contamination. Flowering trees as well as fruiting trees should not be grown in the instant vicinity of pig farm buildings due to these trees may attract fruit bat activity. In an onset of reproductive disease, the infection likely will have already spread in the whole population of a pig farm by the time the first affected litters are farrowed. There would be insufficient numbers of susceptible animals available to maintain a cycle of infection in small piggeries because there is dissimilar parvovirus, no carrier state, and environmental survival is poor. In large piggeries, infection can become endemic, with the infection being continued in groups of pigs as they lose their maternally obtain protection. In that situation, it is major to maximize the opportunity for infection of all preferred replacement breeding stock before mating (Kirkland, et al., 2002)

10 Eradication

Eradication of MenPV from a common infected pig population may be attained by removing to another site all the age groups in which infection is active (eg, pigs between 10–16 weeks of age) (Love et al., 2001). Renew with unexposed pigs or pigs known to be immune to the virus through a cleaned environment and previously vacated will break the cycle of endemic infection in the herd.

11 Public health

In comparison to Nipah virus, Menangle virus does not appear to be highly infectious for human beings; but, care must be taken when working with possibly infected pigs or suspect reproductive specimens. Serologic studies have examine that only 2 of more than 30 human beings immediately experienced a severe febrile illness related with a macular rash followed by prolonged debility (Chan et al., 1998). There was no confirmation of infection in a large number of other people, in addition to abattoir workers, veterinarians, and laboratory workers, who had less direct and also less protracted contact with potentially infective material and this low infectivity for human beings has been clarified to mean that transmission may necessary the contamination of cuts and abrasions with infectious body fluids or possibly splashing of material onto the conjunctivae or tissues.

12 Future prospects

In the past years, a range of new viral diseases have developed affecting domestic animals and human beings, with several species of fruit bats as the accepted source. Such events recommend a serious change in the relationship between US domestic species and fruit bats. This change can simply be a either reflection of the worldwide destruction of forcing the wild and domestic species into much more intimate associations, forest habitat. In Australia, the fruit bat population will persist a potential source of Menangle virus infection for pigs and likely other species, just as Nipah virus poses a continuing threat in Malaysia. The possibility that these viruses pose to pigs in other countries and also to other animal species has not been resolved but should not be neglect.

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