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Short Communication

Lymphocytic Choriomeningitis

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Abstract

Lymphocytic choriomeningitis virus (LCMV) is an arena virus which causes nervous disorders in human beings as well as in mice and hamsters. Mice and hamsters are principal reservoir host of the virus and cover a large geographic area. They infect large number of human population especially during late autumn and early winter due to increase human-mouse contact. It is characterized by aseptic meningitis, encephalitis or meningoencephalitis and infected individual have abnormally high levels of lymphocytes during infection. The pathogenecity is determined by whether the virus is acquired postnatally by children or adults or prenatally. The clinical manifestations in postnatal infection are aseptic meningitis with good prognosis. However prenatally, LCMV can lead to infection in the fetal nervous system and retina, where it causes substantial injury with permanent dysfunctioning and grave prognosis.

Keywords: Lymphocytic Choriomeningitis Virus; Aseptic Meningitis; Encephalitis; Meningoencephalitis

Etiology

Lymphocytic Choriomeningitis Virus (LCMV) is a pleomorphic arena virus containing a single-stranded RNA genome. *"Arena"* is a latin word which means "sandy". The Cross-sectional study of the virus shows grainy particles which are basically host cell ribosomes. Virus particles are spherical in shape and have an average diameter of 110-120 nanometers. Wild and laboratory mice are the natural hosts of lymphocytic choriomeningitis virus. Snakes are also natural host of the virus. Hamsters, mice and guinea pigs are susceptible to LCMV infection, however only mice and hamster are known to spread the virus. This virus has a significant zoonotic potential. Poor hygiene and recent reports of recipient human with asymptomatic donors are detrimental factors for fatal infection. It is associated with meningoencephalitis. Meningitis usually occurs during autumn season and starting of winters. During this time of month, the mice population harbors homes to escape cold, thereby increasing the house- mouse contact. The congenital LCMV infection cause chorioretinitis, hydrocephalus, microcephaly or macrocephaly, and mental retardation in human. Acquired LCMV infection can cause hydrocephalus, encephalitis, myelitis and a Guillain–Barré-type syndrome associated with nerves.

Transmission

Wild mice are natural reservoir of infection. They develop asymptomatic infection and are carrier of the disease. Virus particles are excreted in saliva, nasal secretion and urine. The virus is transmitted horizontally as well as vertically. It is shed at a high number in mouse excreta and urine but not spread by any of the arthropod vectors. Another host of this virus is Syrian hamster (*Mesocricetus auratus*) which can haven persistent infection and act as carrier of infection. Recently, LCMV infections have been reported in humans who have received transplanted organs from a dead individual who had previously contracted this viral infection from a pet hamster. Horizontal transmission in humans is very rare but the virus can cross placental barrier and may infect fetus [3].

Pathogenesis

The pathogenic mechanisms of spread of LCMV is different in postnatal and prenatal infection. In prenatal infection, virus infects brain parenchyma and leads to congenital LCMV infection and in post-natal infection primary predilection site is meninges and choroid plexus. The postnatal infection is immune mediated as viral antigen in these tissues acts as the target of acute mononuclear cell infiltration driven by cytotoxic T lymphocytes (CD8+ cells). The lymphocytes infiltration occurs in large numbers within the meninges and cerebrospinal fluid and leads to the symptoms of meningitis and this is the characteristic sign of acquired LCMV. Lymphocytes neutralize the virus from the brain and CSF (cerebrospinal fluid) due to which population of lymphocytes subsquently declines [1]. Congenital LCMV infection can pass the placental barrier can result in miscarriage or teratology. Pregnant women should avoid contact with rodent's especially pet hamsters. Infection of the fetus usually occurs during early first trimester of pregnancy and ovum can also take up the infection prior to implantation. Almost all the cells in fetus can become infected to the virus with no adverse effects. Infection also involves the thymus, resulting in immunological tolerance and depletion of viral responsive cytotoxic t cells. The viral immune tolerance allows systemic and persistent infections in the body. On sexual maturity, Virus transmitted to next generation. Tolerance is not absolute. Development of LCMV-specific antibody is accompained with high viral antigen. These Ag - Ab complex deposits in various tissues including arterial walls, choroid plexus of brain and glomeruli in kidneys. Chronic infiltration of lymphocytes in various tissues and immune complex mediated glomerulonephritis occurs in late disease. LCMV also causes chorioretinitis, ependymitis, ependymal calcifications, polymicrogyria, microcephaly, and hydrocephalus [2].

Clinical signs

In an experimental study, LCMV infection was induced and four basic patterns of clinical disease were recognized.

- **Cerebral form:** Subacute illness can be associated with following signs: Head tremors, clonic and tonic convulsions, ruffled fur, hunched posture, stance giat, photophobia, and neurological deficits. Animals usually die within 24hrs of exposure in acute cases or recover in several days in acquired LCMV infections.
- **Visceral form:** Animals inoculated with viscerotropic strain manifest this form of disease. It is characterized by severe conjunctivitis, ascitis, yellowish discolouration of eyes and death. Recovery can take many weeks if mice survives.
- **Runt form:** Neonatally infected suckling mice manifest this form and it can cause transient illness or death. Clinical signs are however very nonspecific with slow recovery and mice may remain runted if they survive.
- **Late-onset:** It can take place in previously asymptomatic mice that develops glomerulonephritis. It arises due to neonatal or prenatal infection and occurs in persistently LCMV infected mice when they are nine to twelve months old. Clinical signs are quite nonspecific which includes hunched posture and ruffled fur. In some cases cachexia and ascites may also occurs.

Microscopic lesions

In severe infection, nonsuppurative inflammation is found in affected tissues. Liver lesions include hepatocytic necrosis accompanied with nodular infiltrates of lymphoid cells and Kupffer's cells, Lymphoid organs develop fibrinoid necrosis.

Diagnosis

LCMV infection can be diagnosed serologically by using IFA or ELISA. Deployment of contact sentinel mice is a very useful model for detecting this viral infection through seroconversion. Haematological samples can also be collected from infected live animals, so as to inoculate cultured cells or mice. Intracerebral inoculation of lymphocytic choriomeningitis positive tissue exhibits nervous symptoms in mice within ten days of infection, whereas infants remain asymptomatic. Histopathological investigation of brain collected from affected adults may show nonsuppurative inflammation along with perivascular cuffing. Virus can be cultivated in several continuous cell lines like N-18 cells, L cells and BHK-21 cells. However, all the procedures involving live virus should be carried out under strict containment conditions to avoid spread of virus in laboratory.

Conclusion

Lymphocytic choriomeningitis virus (LCMV) has significant zoonotic potential. It is transmitted from mice and hamster through

direct contact especially during autumn season and early winter. The prenatal form of infection is more dangerous than post-natal form. The strong neurotropism of virus can cause chorioretinitis, hydrocephalus, microcephaly or macrocephaly, and mental retardation in human. It also crosses placental barriers and can cause abortions and fetal malformations. Pregnant women are often advised to avoid pet hamsters. The LCM virus has gained much attention with increased cases of transmission through organ donor with previous history of LCMV infection.

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