The first arenavirus to be isolated was lymphocytic choriomeningitis virus (LCM). It was discovered in 1933 during an epidemic of St Louis encephalitis in the USA. The virus can infect mice. Neonatally infected mice become chronic carriers and excrete the virus for a long time in their urine. The course of the infection is determined by age, immunological resistance, the virus strain and the genetic makeup of the rodent. Both *Mus musculus* and *Mus domesticus* (the common house mouse) can be infected. Other rodents, such as hamsters, which are sometimes kept as pets, can also become infected and can be responsible for transmission. Lymphocytic choriomeningitis virus can also be transmitted via organ transplantation.
In humans it is mainly known for causing an “aseptic” meningitis, with or without fever about 10 days before the meningeal signs appear, though infection is more often without symptoms or a mild febrile illness. LCMV infection in immune compromised patients tends to be severe. Sometimes there is severe damage to the central nervous system. Transient hydrocephalus has been described. Chorioretinitis and congenital hydrocephalus may occur in foetal infection. The cerebrospinal fluid exhibits lymphocytic pleocytosis, an elevated protein content and in 25% of patients there is also reduced sugar. Rarely transverse myelitis, ascending myelitis or bulbar paralysis occur. Some cases of residual deafness have been described after LCM infection. At present, a significant fraction of cases of neonatal mental retardation and blindness remain unexplained. Congenital LCMV infection is an understudied potential cause of a portion of these cases.

There is no specific treatment. There is no vaccine. In general, mortality is less than 1%.