In the Literature

**Diagnosing Mumps: Don’t Be So Sure**


During an outbreak of mumps affecting university students in Nova Scotia, Canada, in 2007, only 298 (14.3%) of 2082 persons in whom mumps was clinically suspected and who were tested by polymerase chain reaction (PCR) proved to be infected with this paramyxovirus. Hatchette and colleagues examined buccal specimens from a 148-patient sample chosen from among those with negative results for mumps to look for evidence of infection with other viral pathogens. Patient evaluation by the public health department found that only 85 of the 148 met clinical criteria for the presence of parotitis. Buccal specimens were tested using the Lumine xTag Respiratory Virus Panel, which utilizes a multiplex PCR methodology, and by quantitative PCR for Epstein-Barr virus (EBV) and cytomegalovirus. Of the 63 patients without parotitis, 1 each had acute EBV, parainfluenzae-3, human metapneumovirus, and rhinovirus infection. In addition, 12 were believed to have “EBV reactivation” as the result of the presence of viral shedding in the presence of anti–Epstein-Barr nuclear antigen immunoglobulin (Ig) G. Fifty-four patients had bilateral parotitis, and 31 had unilateral parotitis; 3 had acute EBV infection, and 1 each had adenovirus, parainfluenzae-3, and influenza virus A infection. Seven were believed to have “EBV reactivation.”

Thus, the frequency of detection of acute viral infection in patients with suspected mumps who failed to have evidence of infection with this virus is low, and there was no significant difference in frequency of pathogen detection or of etiologies between those with and without parotitis.

Thus, even in the setting of an outbreak, a large number of patients with parotitis, whether unilateral or bilateral, did not have mumps. Furthermore, the etiology in these mumps virus–negative cases remained unknown, despite extensive testing. This lack of diagnostic sensitivity is especially true when one considers that “EBV reactivation” may well have had nothing to do with the clinical syndrome.

These results are not dissimilar to those found in a study of Finnish children with “mumps-like illness,” only 17 (2%) of 848 of whom had serological evidence of acute mumps virus infection [1]. Another viral etiology was serologically detected in 84 (14%) of 601 patients, including some viruses not detected by Hatchette and colleagues, such as enterovirus, parvovirus, and human herpesvirus-6. Parainfluenzae viruses 1 and 2, influenza virus B, and bocavirus may also cause parotitis.

In response to continuing outbreaks of mumps in vaccinated individuals together with the recognition that serological testing in such subjects is insensitive because of their frequent lack of an IgM or anamnestic IgG response together with limited viral shedding, the Centers for Disease Control and Prevention has instituted a program of enhanced laboratory surveillance in patients with parotitis.

**Reference**


**Marseillevirus: A DNA Scavenger**


The nucleocytoplasmic large DNA viruses (NCLDVs), so named because they have a stage of replication that takes place within the cytoplasm during which the nascent virus is physically separated from the replication and expression activities of the host genome, are a monophyletic group that infect eukaryotes [1]. Among the viral families included in the NCLDV are the Mimiviridae and a newly recognized family represented by the Marseillevirus. Like the Mimivirus and the Mamavirus, Marseillevirus was detected by cultivation of the free-living amoeba, *Acanthamoeba polyphaga*, with cooling tower water samples and its subsequent detection within the eukaryotic cell. The Marseillevirus genome, which consists of 368,454 bp, is the fifth-largest viral genome known (Mimivirus and Mamavirus are first and second, respectively). Its genome contains 457 predicted genes. These include, for example, 15 predicted protein kinases, presumably of eukaryotic host cell origin, suggesting involvement of the virus in host cell signaling and genes acquired from other NCLDV. Ten genes encode predicted endonucleases, including a family acquired by horizontal transfer by bacteriophage. Thus, Marseillevirus contains genes originating in other NCLDV, genes of apparent bacterial and bacteriophage origin, and genes of eukaryotic origin, including some recently acquired from *Acanthamoeba*. To be more precise, the Marseillevirus contains 51 genes of NCLDV origin, 49 of probable bacterial or bacteriophage origin, and 85 apparently originating in eukaryotes. The potential for amoebae to serve as “mixing vessels” for DNA from multiple forms of life can be seen by the investigators demonstration that *Acanthamoeba castellani* can be simultaneously infected with Marseillevirus and two bacteria, a *Legionella* and a *Para-chlamydia*. As the authors state, this makes the amoeba “a veritable factory for gene mixing between the eukaryotic host, its various viruses, and bacterial parasites and symbionts. The amoebal genetic melting pot seems to produce organisms with complex, chimeric genomes such as the giant viruses.”

Such parasitism at the level of microorganisms and the promiscuity of their...