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Enterovirus D68

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Enterovirus D68

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The Virus

Enterovirus D68 (EVD68) “belongs to the family Picornaviridae, genus Enterovirus, and species Human enterovirus D: (Kaida et al., 2011, para.2). It is a small nonenveloped RNA virus (Xiang et al., 2012). EVD68 “shares characteristics with rhinoviruses, such as infection of the respiratory tract and acid lability” (Lauinger et al., 2012).

Outbreaks

EVD68 was first isolated in 1962 in California, where it was associated with bronchiolitis and pneumonia in children (Linsuwanon et al., 2012). EVD68 was rarely identified from 1962-2007. Since 2008, several outbreaks of EVD68 have been identified. The Philippines had an outbreak October 2008- March 2009, Japan had an outbreak July-October 2010, and the Netherlands experienced an outbreak August-November 2010. The United States has had three prior outbreaks: one in Georgia in September 2009, one in Pennsylvania also in September 2009, and one in Arizona August-September 2010 (Imamura et al., 2011). From mid-August to the present time, the USA has had 1116 confirmed cases in 47 states as well as the District of Columbia (“Enterovirus D68 in the USA”). Most of the confirmed cases have been among children and EVD68 “has been detected in specimens from 11 patients who died” (“Enterovirus D68 in the USA”).

Testing

Testing is not done on a routine basis. Nasopharyngeal or oropharyngeal swabs are collected for testing. In Ohio, testing must be approved by the Ohio Department of Health and specimens are sent to the CDC for testing (*Enterovirus D68*, 2014). The CDC has developed a new test with just a few day turnaround for results (Enterovirus D68 in the USA).

Pathophysiology

Several pathogenic viral mechanisms have been identified. They include virus implantation at the portal of entry, local replication, viral spread to target organs, and shedding of the virus into the environment (Baron, Fons, & Albrecht, 1996). Viral affinity is determined by cell-receptors, “cell transcription factors that recognize viral promoters”, cell’s ability to support replication of the virus, as well as local temperature and pH (Baron et al., 1996, para. 3). The primary site of infection of airway viruses is the epithelial cell of the airway (Jacoby, 2004). Airway epithelial cell infection “leads to functional changes that may initiate and orchestrate the early inflammatory and immune responses” (Jacoby, 2004, para. 2). The epithelial cells then “release a wide range of inflammatory cytokines: (Jacoby, 2004, para.2). Marked changes are noted in sensory nerve function, which may contribute to cough and bronchoconstriction. Tachykinins in the lungs are increased and neutral endopeptidase activity is decreased leading to increased sensitivity to cough. Vagally-mediated bronchoconstriction “is due to increased acetylcholine release as a result of inhibitory M2 muscarinic receptors on the cholinergic nerve fibers” (Jacoby, 2004, para. 5). “Endogenously-produced interferons can also increase vagally-mediated bronchoconstriction” (Jacoby, 2004, para. 8). Eosinophils localize to airway nerves and also contribute to cough (Jacoby, 2004).

Signs/Symptoms

Typical signs and symptoms include fever, runny nose, cough, sneezing, and body aches; while more severe symptoms include wheezing and dyspnea (“EVD68”, 2014). Clinical diagnosis consists of asthma exacerbations, pneumonia, and upper respiratory infections (Rahamat-Langendoen et al., 2011). EVD68 has also been implicated in some neurologic symptoms: limb weakness or paralysis, “cranial nerve dysfunction and abnormalities” in gray matter of the spine (Wilson & Botelho, 2014, para. 12). Asthmatics seem to be more severely affected by EVD68.

Transmission

EVD68 is spread by coming in contact with an infected person or their secretions, especially respiratory secretions (“What Families Need to Know”, 2014). The virus can also live for some time on inanimate surfaces that have been contaminated with secretions (“Separating Fact from Fiction”, 2014).

Treatment

There is no specific treatment for EVD68. Since the infection is caused by a virus, antibiotics would be ineffective. There are no antiviral medications currently available to treat EVD68 (Herold, 2014). Treatment may include antipyretics, cough medicines, bronchodilators, oxygen, and mechanical ventilation if required.

Preventing Spread of EVD68

Prevention is key to decreasing the spread of EVD68. Preventative techniques include good hand-washing, not sharing cups and eating utensils, covering coughs and sneezes, regular disinfection of frequently touched objects, and staying home when sick. Avoid touching eyes, nose, and mouth.



Educating Parents

Because of all the media attention, many parents are terrified of this virus. Teach parents the importance of preventing the spread of all viruses, including EVD68, by the aforementioned strategies. Encourage them to train their children. Parents of asthmatics need to keep on top of their child’s asthma action plan. Make sure their children are taking maintenance medications as prescribed and that they always have their rescue inhaler available. Instruct parents to seek immediate treatment if their child is having trouble breathing or drinking, appears distressed, or exhibits other worrisome symptoms. Remember, parents know their children better than we do.



Picture from “Enterovirus D68 in the USA” on CDC- activity of EVD68 week of 10/26/2014-11/01/2014

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