Enterovirus is a genus of positive-sense single-stranded RNA viruses associated with several human and mammalian diseases. Enteroviruses are named by their transmission-route through the intestine. Serologic studies have distinguished 71 human enterovirus serotypes on the basis of antibody neutralization tests.

Serologic studies have distinguished 71 human enterovirus serotypes on the basis of antibody neutralization tests. Additional antigenic variants have been defined within several of the serotypes on the basis of reduced or nonreciprocal cross-neutralization between variant strains. On the basis of their pathogenesis in humans and animals, the enteroviruses were originally classified into four groups, polioviruses, Coxsackie A viruses (CA), Coxsackie B viruses (CB), and echoviruses, but it was quickly realized that there were significant overlaps in the biological properties of viruses in the different groups.

Enteroviruses isolated more recently are named with a system of consecutive numbers: EV68, EV69, EV70, EV71, etc.

Enteroviruses affect millions of people worldwide each year and are often found in the respiratory secretions (e.g., saliva, sputum, or nasal mucus) and stool of an infected person. Historically, poliomyelitis was the most significant disease caused by an enterovirus, namely poliovirus. There are 81 non-polio and 3 polio enteroviruses that can cause disease in humans.

Of the 81 non-polio types, there are 22 Coxsackie A viruses, 6 Coxsackie B viruses, 28 echoviruses, and 25 other enteroviruses.

Poliovirus, as well as coxsackie and echovirus, is spread through the fecal-oral route. Infection can result in a wide variety of symptoms, including those of: mild respiratory illness (the common cold), hand, foot and mouth disease, acute hemorrhagic conjunctivitis, aseptic meningitis, myocarditis, severe neonatal sepsis-like disease, acute flaccid paralysis, and the related acute flaccid myelitis.

Enteroviruses are members of the picornavirus family, a large and diverse group of small RNA viruses characterized by a single positive-strand genomic RNA. All enteroviruses contain a genome of approximately 7,500 bases and are known to have a high mutation rate due to low-fidelity replication and frequent recombination. After infection of the host cell, the genome is translated in a cap-independent manner into a single polyprotein, which is subsequently processed by virus-encoded proteases into the structural capsid proteins and the nonstructural proteins, which are mainly involved in the replication of the virus.

The enterovirus genus includes the following fifteen species:[7]

Enterovirus A (formerly Human enterovirus A)

Enterovirus B (formerly Human enterovirus B)

Enterovirus C (formerly Human enterovirus C)

Enterovirus D (formerly Human enterovirus D)

Enterovirus E (formerly Bovine enterovirus group A)

Enterovirus F (formerly Bovine enterovirus group B)

Enterovirus G (formerly Porcine enterovirus B)

continued

Enterovirus H (formerly Simian enterovirus A)

Enterovirus I

Enterovirus J

Enterovirus K

Enterovirus L

Rhinovirus A (formerly Human rhinovirus A)

Rhinovirus B (formerly Human rhinovirus B)

Rhinovirus C (formerly Human rhinovirus C)

These twelve species' serotype include:

Coxsackievirus

Enterovirus A: serotypes CV-A2, CV-A3, CV-A4, CV-A5, CV-A6, CV-A7, CV-A8, CV-A10, CV-A12, CV-A14, and CV-A16.

Enterovirus B: serotypes CV-B1, CV-B2, CV-B3, CV-B4, CV-B5, CV-B6, and CV-A9

Enterovirus C: serotypes CVA1, CVA11, CVA13, CVA17, CVA19, CVA20, CVA21, CVA22, CVA24, EV-C95, EV-C96, EV-C99, EV-C102, EV-C104, EV-C105, EV-C109, EV-C113, EV-C116, EV-C117, and EV-C118.

Echovirus

Enterovirus B: serotypes E-1 through E-7, E-9, E-11 through E-21, E-24 through E-27, and E-29 through E-33.

Enterovirus

Enterovirus A: serotypes EV-A71, EV-A76, EV-A89, EV-A90, EV-A91, EV-A92, EV-A114, EV-A119, EV-A120, EV-A121, SV19, SV43, SV46 and BA13.

Enterovirus B: serotypes EV-B69, EV-B73, EV-B74, EV-B75, EV-B77 through EV-B88, EV-B93, EV -B97, EV-B98, EV-B100, EV-B101, EV-B106, EV-B107, EV-B110 and SA5

Enterovirus C: serotypes EV-C95, EV-C96, EV-C99, EV-C102, EV-C104, EV-C105, EV-C109, EV-C116, EV-C117 and EV-C118

Enterovirus D: serotypes EV-D68, EV-D70, EV-D94, EV-D111 & EV-D120

Enterovirus H: serotype EV-H1

Enterovirus J: serotypes: SV-6, EV-J103, EV-J108, EV-J112, EV-J115, and EV-J121

Rhinovirus

Rhinovirus A: serotypes RV-A1, RV-A2, RV-A7 through RV-A13, RV-A15, RV-A16, RV-A18 through RV-A25, RV-A28 through RV-A34, RV-A36, RV-A38 through RV-A41, RV-A43, RV-A45, RV-A46, RV-A47, RV-A49, RV-A50, RV-A51, RV-A53, RV-A54 through RV-A68, RV-A71, RV-A73 through RV-A78, RV-A80, RV-A81, RV-A82, RV-A85, RV-A88, RV-A89, RV-A90, RV-A94, RV-A96, and RV-A100 through RV-A109

Rhinovirus :

Serotypes RV-B3 through RV-B6, RV-B14, RV-B17, RV-B26, RV-B27, RV-B35, RV-B37, RV-B42, RV-B48, RV-B52, RV-B69, RV-B70, RV-B72, RV-B79, RV-B83, RV-B84, RV-B86, RV-B91, RV-B92, RV-B93, RV-B97, and RV-B99 through RV-B106

Poliovirus: **Enterovirus C: serotypes PV-1, PV-2, and PV-3.[8]**

Main articles: Coxsackie A virus, Coxsackie B virus, and Echovirus.

Coxsackie viruses are a non-phylogenetic group.[9] Coxsackie A viruses are mainly associated with human hand, foot and mouth disease. Coxsackie B viruses can cause signs and symptoms, similar to a cold, but these viruses also can lead to more serious diseases, including myocarditis (inflammation of the heart); pericarditis (inflammation of the sac surrounding the heart); meningitis (inflammation of the membranes that line the brain and spinal cord); and pancreatitis (inflammation of the pancreas).

Echoviruses are a cause of many of the nonspecific viral infections. It is mainly found in the intestine, and can cause nervous disorders. needed] The usual symptoms of Coxsackie and echovirus are fever, mild rash, and mild upper respiratory tract illness.

Non-cytolytic (non-cytopathic) enterovirus

Enteroviruses are usually only capable of producing acute infections that are rapidly cleared by the adaptive immune response. However genome mutations, which enterovirus B serotypes may acquire in the host during the acute phase, may transform these viruses into the non-cytolytic form (also known as non-cytopathic or defective enterovirus). This is a mutated quasispecies of enterovirus, which can cause persistent infection in human cardiac tissues especially in some patients with myocarditis or dilated cardiomyopathy. In persistent infections viral RNA is present only on very low levels and is not believed to contribute to any ongoing myocardial disease being a fading remnant of a recent acute infection[11] although some scientists think otherwise.

Enterovirus 68

EV-D68 first was identified in California in 1962. Compared with other enteroviruses, it has been rarely reported in the U.S. in the past 40 years. Most people who get infected are infants, children and teens. EV-D68 usually causes mild to severe respiratory illness; however, the full spectrum of EV-D68 illness is not well-defined. Most start with common cold symptoms of runny nose and cough. Some, but not all, may also have fever. For more severe cases, difficulty breathing, wheezing or problems catching your breath may occur. As of October 4, 2014, there has been one death in New Jersey directly linked to EV-D68,as well as one death in Rhode Island[citation needed] attributed to a combination of EV-D68 and sepsis caused by an infection of staphylococcus aureus.

Main article: Enterovirus 71

Enterovirus 71 (EV-71) is notable as one of the major causative agents for hand, foot and mouth disease (HFMD), and is sometimes associated with severe central nervous system diseases. [17] EV71 was first isolated and characterized from cases of neurological disease in California in 1969. To date, little is known about the molecular mechanisms of host response to EV71 infection, but increases in the level of mRNAs encoding chemokines, proteins involved in protein degradation, complement proteins, and proapoptotis proteins have been implicated.

Poliovirus

Main article: Poliovirus

There are three serotypes of poliovirus, PV1, PV2, and PV3; each with a slightly different capsid protein. Capsid proteins define cellular receptor specificity and virus antigenicity. PV1 is the most common form encountered in nature; however, all three forms are extremely infectious.[21] Poliovirus can affect the spinal cord and cause poliomyelitis.

Polioviruses were formerly classified as a species belonging to the genus Enterovirus in the family Picornaviridae. The Poliovirus species has been eliminated from the genus Enterovirus. The following serotypes, Human poliovirus 1, Human poliovirus 2, and Human poliovirus 3, were assigned to the species Human enterovirus C, in the genus Enterovirus in the family Picornaviridae. The type species of the genus Enterovirus was changed from Poliovirus to Human enterovirus C. This has been ratified in April 2008.[22] The 39th Executive Committee (EC39) of the International Committee on Taxonomy of Viruses (ICTV) met in Canada during June 2007 with new taxonomic proposals.[23]

Two of the proposals with three changes were:

Code 2005.261V.04: To remove the following species Poliovirus from the existing genus Enterovirus in the family Picornaviridae.

Code 2005.262V.04: To assign the viruses; PV-1, PV-2, PV-3 to the existing species Human enterovirus C in the genus Enterovirus in the family Picornaviridae.[24]

Code 2005.263V.04: To change the type species Poliovirus from the existing genus Enterovirus in the family Picornaviridae to the type species Human enterovirus C.[25]

Proposals approved at the (EC39) meeting of 2007, were sent to members of ICTV via email for ratification and have become official taxonomy. There have been a total of 215 taxonomic proposals, which have been approved and ratified since the 8th ICTV Report of 2005.[26]

The ratification process was performed by email. The proposals were sent electronically via email on March 18, 2008 to ICTV members with a request to vote on whether to ratify the taxonomic proposals, with a 1-month deadline. The following are two of the taxonomic proposals with three changes that were ratified by ICTV members in April 2008:

Picornaviruses

2005.261V.04: To remove the following species from the existing genus Enterovirus in the family Picornaviridae: Poliovirus. (Note: Poliovirus hereby loses its status as a virus species.)

2005.262V.04: To assign the following viruses to the species Human enterovirus C in the existing genus Enterovirus in the family Picornaviridae: Human poliovirus 1, Human poliovirus 2, Human poliovirus 3. (This is not strictly necessary as a taxonomic proposal because it concerns entities below the species level, but it is left in to clarify this reorganization of the Picornaviridae.)

2005.263V.04: To change the type species of the genus Enterovirus in the family Picornaviridae, from Poliovirus to Human enterovirus C.

Diseases caused by enterovirus infection

Enteroviruses cause a wide range of symptoms, and while their long list of signs and symptoms should put them on the differential diagnosis list of many illnesses, they often go unnoticed. Enteroviruses can cause anything from rashes in small children, to summer colds, to encephalitis, to blurred vision, to pericarditis. Enteroviral infections have a great range in presentation and seriousness. Non polio enteroviruses cause 10–15 million infections and tens of thousands of hospitalizations in the US each year.[27] Enteroviruses can be identified through cell culture or PCR assay, collected from fecal or respiratory specimens.[28] Below are common enterovirus related diseases, including poliomyelitis.

Poliomyelitis primarily via the fecal-oral route

Polio-like syndrome found in children who tested positive for enterovirus 68.

Nonspecific febrile illness is the most common presentation of enterovirus infection. Other than fever, symptoms include muscle pain, sore throat, gastrointestinal distress/abdominal discomfort, and headache. In newborns the picture may be that of sepsis, however, and can be severe and life-threatening.

Enteroviruses are by far the most common causes of aseptic meningitis in children. In the United States, enteroviruses are responsible for 30,000 to 50,000 meningitis hospitalizations per year as a result of 10–15 million infections.

Bornholm disease or epidemic pleurodynia is characterized by severe paroxysmal pain in the chest and abdomen, along with fever, and sometimes nausea, headache, and emesis.

Pericarditis and/or myocarditis are typically caused by enteroviruses; symptoms consist of fever with dyspnea and chest pain. Arrhythmias, heart failure, and myocardial infarction have also been reported.

Acute hemorrhagic conjunctivitis can be caused by enteroviruses.

Herpangina is caused by Coxsackie A virus, and causes a vesicular rash in the oral cavity and on the pharynx, along with high fever, sore throat, malaise, and often dysphagia, loss of appetite, back pain, and headache. It is also self-limiting, with symptoms typically ending in 3–4 days.

Hand, foot and mouth disease is a childhood illness most commonly caused by infection by Coxsackie A virus or EV71.

Encephalitis is rare manifestation of enterovirus infection; when it occurs, the most frequent enterovirus found to be causing it is echovirus 9.

Myocarditis is characterized by inflammation of the myocardium (cardiac muscle cells). Over the last couple of decades, numerous culprits have been identified as playing a role in myocarditis pathogenesis in addition to the enterovirus, which at first was the most commonly implicated virus in this pathology.[33] One of the most common enteroviruses found to be responsible for causing Myocarditis is the Coxsackie B3 virus.

A 2007 study suggested that acute respiratory or gastrointestinal infections associated with enterovirus may be a factor in chronic fatigue syndrome.

Diabetes mellitus type 1 valid theory that type 1 is a virus-triggered autoimmune response

Dengue fever is a mosquito-borne tropical disease caused by the dengue virus.

Symptoms typically begin three to fourteen days after infection. This may include a high fever, headache, vomiting, muscle and joint pains, and a characteristic skin rash. Dengue is spread by several species of female mosquitoes of the Aedes Aegypti. The virus has five types; infection with one type usually gives lifelong immunity to that type, but only short-term immunity to the others. Subsequent infection with a different type increases the risk of severe complications. A number of tests are available to confirm the diagnosis including detecting antibodies to the virus or its RNA. Acute symptoms are present for 3—5 days then a few days without then starts again for another 3—5 days. Because this is a virus, recovery might take up to 6 weeks. The virus never leaves the host, causes encephalomyelitis, encephalitis. Strains are DENV1, DENV2, DENV3, DENV4. once the host experiences one stain they never get that particular strain again but the patient can develop the other strains. The patient can also develop Myalgic Encephalomyelitis as it is Post Viral and considered an acquired brain injury for those who don't recover.

The Aedes Aegypti Mosquito carries the following (chikungunya virus, yellow fever, dengue fever, etc).

Sepsis & Post Sepsis

Sepsis and Post-Sepsis, present life-threatening issues arising from a dysregulated response to an infection. Many survivors experience a range of physical and psychological symptoms collectively known as post-sepsis syndrome. The effects of post-sepsis syndrome vary but can be devastating and life altering. There has been recent neurological studies showing that Viruses can be carried within the Sepsis bacterial cell. Sepsis can cause brain injury similar to the issues a Stroke Victim experiences.

Viral caused Encephalitis, Encephalomyelitis, Encephalopathy

Encephalitis refers to an acute, usually diffuse, inflammatory process affecting the brain. While meningitis is primarily an infection of the meninges, a combined meningoencephalitis may also occur. An infection by a virus is the most common and important cause of encephalitis, although other organisms may sometimes cause an encephalitis. An encephalitic illness caused by alteration of normal immune function in the context of a previous viral infection or following vaccination is also well recognised (acute disseminated encephalomyelitis, ADEM). An infectious encephalitis may also be difficult to distinguish from an encephalopathy that may be associated with numerous metabolic causes. Among the factors which have helped to focus attention on viral encephalitis over the last few years have been:

****#the development of effective antiviral agents for this condition, most notably acyclovir for herpes simplex virus encephalitis (HSE) which is caused by herpes simplex virus (HSV)-1 or HSV-2.**
****the advent of human immunodeficiency virus (HIV) infection of the central nervous system (CNS) with its wide range of associated acute viral infections.**
****the recent recognition of emerging viral infections of the CNS such as West Nile encephalitis and Nipah virus encephalitis.**

https://jnnp.bmj.com/content/75/suppl_1/i10

Involvement of the brain is one of the most serious consequences of a viral infection. Many virus families have the ability to invade and replicate in brain tissue, but fortunately serious brain infections are rare. Clinically, neurological diseases caused by viruses can be divided into acute and chronic syndromes. The pathology may be due either to multiplication of virus in the cells of the brain or, due to the (misdirected) immune response of the host - post-infectious encephalo-myelitis.

Viruses which infect the brain may reach the central nervous system either by the blood stream or by spread along peripheral nerves. Asymptomatic infection of the brain is common.
Where a virus infects the brain directly, it can usually be isolated either from brain tissue or from the cerebrospinal fluid. This is not the case with the post infectious syndromes.

Acute neurological syndromes: There are four main syndromes:

1. **Aseptic meningitis**
2. **Acute flaccid paralysis**
3. **Encephalitis**
4. **Post infectious encephalomyelitis**

Aseptic Meningitis:

This is the commonest viral syndrome. The condition is self-limiting and has a good prognosis. Infection is confined to the meninges. The clinical features include fever, headache, neck stiffness, photophobia and vomiting. CSF findings include a pleocytosis consisting of both polymorphs and lymphocytes, but usually with a lymphocyte predominance, normal glucose and no bacterial growth (hence the term aseptic).
Viruses are by far the commonest cause of meningitis. Infections may occur at any age, but are particularly common in children and young adults.
Common viral agents include: enteroviruses and mumps virus (and less commonly HSV-2 and varicella-zoster virus).
Sometimes, the underlying brain tissue may also be involved, giving rise to meningo-encephalitis. The prognosis depends on the extent of damage done to brain parenchyma.

Encephalitis (grey matter disease)

Viral replication occurs in the brain tissue itself, causing destructive lesions in the grey matter. The main symptoms include: fever, drowsiness, confusion, depressed level of consciousness, convulsions and focal neurological signs. Morbidity and mortality is very high. Viruses that cause this condition include herpes simplex, rabies and some of the arboviruses.

The arboviruses are a miscellaneous group of enveloped, ssRNA viruses that infect animals. They are transmitted from one vertebrate host to another via blood sucking arthropods. The main reservoirs are wild birds and small mammals. Man may be infected if bitten by the insect vector.

In South Africa, there are no enzootic arboviruses that specifically cause encephalitis. Rarely, however, encephalitis may occur as part of the clinical course of infection with viruses such as, West Nile virus, Rift Valley fever virus and Sinbis virus. These viruses are enzootic in livestock herds in certain parts of the country and farm workers or vets may occasionally be infected.

Rabies:

Rabies virus is an enveloped (bullet shaped) ssRNA virus. It primarily infects warm blooded vertebrates. It is enzootic in most parts of the world. Virus is shed in the saliva of infected animals and humans are occasionally infected if bitten by an infected animal. The behaviour of the infected animal is altered and it is more likely to bite humans or other animals that it comes into contact with (thus ensuring the viruses survival). The most common sources of human infection are dogs and bats.

Pathogenesis:

Virus is introduced into the tissues through a bite. It enters peripheral nerves and travels up the axon to the brain where it replicates. It causes a fatal encephalitis.

Incubation period:

It varies from 9-90 days, depending on the severity and site of the bite. Incubation period is determined by how long the virus takes to reach the brain. (Bites on the foot take longer than bites on the face.)

The disease can be prevented in an exposed person by administration of post exposure prophylaxis in the form of rabies vaccine and rabies immunoglobulin.

Acute flaccid Paralysis: **This syndrome is due to direct infection of motor neurones (grey matter) in the spinal cord by a virus. Patients present with fever and flaccid paralysis of a group of muscles. Signs of meningitis such as headache and neck stiffness are frequent accompanying features. The most common aetiological agents include the Polioviruses 1, 2 and 3, but with the reduction in prevalence of wild type polio due to successful global vaccination, other (non polio) enteroviruses are responsible for most cases. (see information on enteroviruses and poliomyelitis)**

Post infectious encephalitis (white matter disease): **This uncommon complication may develop in the convalescent phase, following a number of common viral infections, including: measles, mumps, rubella and primary varicella-zoster virus infection. In addition it may develop following exposure to certain vaccines, such as: vaccinia virus and the older neurotissue rabies vaccines. Widespread demyelinating lesions develop involving the white matter in the brain and spinal cordcuffing of adjacent blood vessels. The causative agent cannot be isolated from brain tissue or CSF. The aetiology is somewhat obscure, but it is thought to be a T cell-mediated auto -immune phenomenon, triggered by exposure to foreign antigens which are closely related to host proteins normally present in brain tissue (molecular mimicry).. Characteristic histological features include: lymphocytic infiltration and perivascular**

Gillain Barre Syndrome: **This syndrome is characterized by poly-neuritis which develops a few days to weeks after the acute phase of a certain bacterial or viral infections. The disease is due to demyelination of peripheral nerves. Patients present with an ascending paralysis, associated with paraesthesia. Like post infectious encephalomyelitis, it is believed to be an immunological phenomenon. Patients usually recover spontaneously over a few weeks or months as affected nerves are re-myelinated.**

Reference for this page is found here: http://www.virology.uct.ac.za/vir/teaching/mbchb/acute-neurological-syndromes

Sepsis

Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection. Sepsis can be caused by a broad range of pathogens; however, bacterial infections represent the majority of sepsis cases. Up to 42% of sepsis presentations are culture negative, suggesting a non-bacterial cause.

Despite this, diagnosis of viral sepsis remains very rare. Almost any virus can cause sepsis in vulnerable patients (e.g., neonates, infants, and other immunosuppressed groups). The prevalence of viral sepsis is not known, nor is there enough information to make an accurate estimate.

The initial standard of care for all cases of sepsis, even those that are subsequently proven to be culture negative, is the immediate use of broad-spectrum antibiotics. In the absence of definite diagnostic criteria for viral sepsis, or at least to exclude bacterial sepsis, this inevitably leads to unnecessary antimicrobial use, with associated consequences for antimicrobial resistance, effects on the host microbiome and excess healthcare costs.

It is important to understand non-bacterial causes of sepsis so that inappropriate treatment can be minimized, and appropriate treatments can be developed to improve outcomes. In this review, we summarize what is known about viral sepsis, its most common causes, and how the immune responses to severe viral infections can contribute to sepsis. We also discuss strategies to improve our understanding of viral sepsis, and ways we can integrate this new information into effective treatment.

Although bacterial or fungal infections are commonly attributed as the cause of sepsis, sepsis is infrequently attributed to viral infections. In some cases, viral sepsis is regarded as virus-induced direct tissue or cell damage (e.g., influenza virus-induced pulmonary epithelial damage) instead of systemic dysregulation caused by virus. However, the above-mentioned consensus definitions of sepsis, either for adult or paediatric populations are not pathogen-specific, so the same definitions should also apply to viral infection.

Therefore, in this review article, viral sepsis is defined as life-threatening organ dysfunction due to a dysregulated host response to viral infection in both adult and paediatric populations. Viral infection can be diagnosed by associated clinical presentations plus positive results of culture, antigen detection, molecular detection (e.g., polymerase chain reaction, PCR), serology, histopathology or immunohistochemistry. Viral sepsis should always be considered in septic patients lacking evidence of bacterial, parasitic or fungal infection, and laboratory tests for viruses should be arranged accordingly. In the following sections, we will review the current evidence available about the epidemiology, aetiology, immune pathogenesis, and potential treatments of viral sepsis.

However, the identified viruses could be the single causative agent of sepsis (e.g., dengue), a contributor to secondary bacterial sepsis (e.g., influenza and staphylococcal sepsis), coinfection of unknown significance (e.g., rhinovirus), prolonged or persistent shedding of a previous infection (e.g., adenovirus), an “innocent” latent infection (e.g., Epstein-Barr virus) or a false positive result. This also needs to be taken in the clinical context.

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