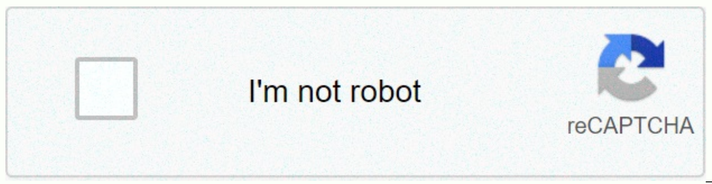

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Cmv infection treatment guidelines

The human stem cell ablation virus (CMV, HCMV, HHV-5) is a ubiloic beta herpes virus that infects only humans and human cells. As with all herpes viruses, CMV is a large, stranded dual DNA virus containing 162 capsomeres, a tegument between capsid and envelope, and an envelope containing fats and glycoproteins that are sensitive to fatty solvents and detergents (164). Once an individual is infected with CMV, the virus establishes latency and may reactivate in people with a normal immune system and is common during immune suppression. In individuals who specialize in natural inhibitor, the activation of CMV leads to local reactivation of urine, saliva and cervix in women and in men's semen. However, reactivation of CMV in the immune host often leads to viremia and can cause severe and life-threatening infections in people with cell-mediated immunodeficiency. The virus usually crosses the human placenta, which is the most common congenital infection in developed countries. Human-to-human CMV is transmitted from human to human; There's no animal tank. The virus has a global distribution and affects all ethnic, social and economic groups. CMV is the most common cause of congenital infection found in 0.4% to 2.5% of infants at birth. After the neonatal period, person-to-person transmission is commonly transmitted through salivary or sexual contact. Contact with urine can also lead to transmission. Mother-to-infant transmission can occur in the uterus, leading to a congenital infection, during childbirth during the passage of the infant through the infected cervix or through breast milk. From 20% to 40% of healthy women cast CMV in breast milk, resulting in injury to 30-70% of nursing infants. Getting babies on full-range CMV across this route rarely leads to complications, and the likelihood of CMV transferring to infants via human milk outweighs the benefits of breastfeeding. Thus, women should be encouraged to breastfeed infants without regard to their serological SV. The acquisition of CMV by preterm infants may lead to a symptomatic infection with CMV including fever, pneumonia, gastrointestinal diseases and hepatitis (201). In these cases when breast milk is desired, milk should be expressed and frozen and melted or heated to reduce the potential infection of milk. It should be noted, however, that freezing may not be 100% effective in eliminating CMV transmission through breast milk (111). CMV is commonly transmitted between young children and children in day-care centers and has reached 70% in some day-care settings. CMV can also be transmitted from an infected donor to the recipient through blood transfusions, blood products, bone marrow and solid organ transplantation. The risk of injury to day-care staff and pre-school places is increased. Transferring CMV to healthcare workers is uncommon. The Centers for Disease Control and Prevention (CDC) has made the following recommendations for individuals caring for infants and children: 1) Female employees should be educated about CMV and how it spreads, as well as health practices, such as hand washing, that reduce the risk of DEVELOPING CMV. 2) Non-pregnant women of reproductive age who have never been infected with CMV and who work with infants and children should not be routinely transferred to other work conditions to avoid CMV infection. 3) Pregnant women working with infants and children should be informed of the risk of CMV infection, the potential effects on the unborn child, and appropriate prevention strategies. 4) Routine laboratory tests of the antibody CMV (immune protein) are not currently recommended in female workers. However, workers who are pregnant or planning to become pregnant should be informed that cmv antibody testing can help them assess their risks. Whenever possible, CMV seronegative (without CMV antibodies) pregnant women should consider working in an environment with less exposure to young children. The Centers for Disease Control and Prevention (CDC) suggests steps that can be taken to reduce the risk of CMV transmission through exposure to saliva and urine that may contain CMV (. These include: often washing hands with soap and water for 15-20 seconds after changing diapers, feeding a small child, wiping a small child's nose or drooling and dealing with toys. Do not share the food, drinks or eating utensils used by young children; Avoid contact with saliva when kissing a child; CMV clinical manifestations cause a wide range of clinical diseases. It is the main cause of non-Epstein-Barr mononucleosis virus in prevented individuals, and is an important cause of the disease in immune groups including in addition to congenitally infected infants, people with malignant tumors, transplant recipients, people with AIDS and other acute diseases affecting cellular immunity. The main diseases associated with CMV include pneumonia, colitis, esophagitis, hepatitis and central nervous system (CNS) including encephalitis and multi-radial inflammation. Other common manifestations, including leukocytes, adrenal itis and mouth ulcers, were also described, especially in people with AIDS. Reactivation of CMV in people with AIDS in the pre-HAART period (high-activity ART) not only affects the development of CMV special diseases but on survival; CMV congenital INFECTION CMV is a major cause of birth Affect about 1% (.5 to 2%) of all children born in the United States and other developed countries. Of these infants are congenitally infected, approximately 5% symptoms at birth. Infants born with symptoms at birth are most likely to develop neurological problems including sensory deafness. However, between 5-17%

of infants with a symptomatic congenital infection will develop hearing loss (39). In other words, out of about 4 million births a year in the United States, 40,000 babies are born with congenital CMV, and 8,000 children will develop sensory hearing impairment or other consequences associated with their hostility. Hearing impairments associated with congenital CMV may not be stable, so infants with normal hearing in the neonatal period can be identified as suffering from hearing loss several years after birth. Infants born small in their gestational age with manifestations including petechiae, hepatic jaundice, microcephaly and hearing loss are poisoned to have a disease inserting a duplicator and they are at greater risk for having severe long-term neurological deficits. In such cases, thrombosis is also present in 75% at birth. Kororitis occurs in about 10-20% of infants with congenital symptomatic CMV. The presence of cardiac calcifications seen on CT scans are typical of congenital CMV and are often associated with acute neurological deficits. However, calcification may not always be around and does not require an infant to develop mental retardation or hearing impairment. The results of microcalification, cardiac calcifications, abnormal neurological testing or signs of sore infection increase the likelihood of long-term consequences of CNS. Although it was thought that the initial CMV infection of the pregnant mother was more likely to be associated with a serious congenital infection, recent studies suggest that the symptomatic infection occurs with a similar frequency in infants born to women regardless of CMV serostatus before they become pregnant. Thus, infants born with congenital CMV to previously seroactive women of the virus are at similar risk of serious complications including sensoineurall hearing loss such as those born to women who suffered an initial infection during pregnancy (15, 16, 135, 166, 229). Although the data are uncertain, it appears that women who give birth to infants with congenital CMV symptoms experience infection again during pregnancy. For this reason, pregnant women are encouraged whether or not to syllin for CMV to follow the CDC recommendations mentioned above. An internatal and acquired CMV infection in healthy infants can acquire CMV infection by exposure to the secretions of the genitals of infected mothers or after birth through taking CMV containing breast milk. Incubation period between Symptoms when present are 3 to 12 weeks. Most full-term infants are at risk of developing asymptomatic CMV. However, interstitial pneumonia, baroque cough, rashes, hepatic baby, and thrombosis may occur. Hepatitis when it occurs is usually mild and self-limited. Typically, alkaline phosphate is raised from the aspartame amyoco -asafraunferas (AST) and alanine aminetranferase (ALT). When preterm infants acquire CMV infection either through intra-natal exposure, breast milk or the transfer of cmv positive blood products, there is a greater risk of symptom infection including interstitial pneumonia, hepatitis, thrombosis, hepatopslenomegaly and regretful anemia. Whether CMV infection contributes to the risk of chronic lung disease associated with premature remains a topic of discussion. However, long-term sequels, including mental retardation and hearing impairment, are rarely associated with the acquisition of CMV compounds after birth. CMV infection in adults who are prepared for primary CMV infection symptoms of adolescence or adult infection usually results in a syndrome similar to the number of white blood cells. Wreghitt and his colleagues reviewed clinical presentations of 124 adults with primary CMV infection (228). Among patients with primary CMV infection, clinical and laboratory results included abnormal liver function tests at 69%, malaise at 67%, 46% sweating, 46% fever, 46% muscle pain, 36% muscle symptoms, respiratory symptoms 28%, lymphapathy 24%, arthralgias 17% and headaches 14%. All patients recovered without long-term complications. Of the patients reaget studied, 15% needed hospitalization. In a study of hospital patients diagnosed only with CMV disease, 99% of patients were febrile, 51% reported headaches and 36% were somegaly (2). The duration of the disease was 21 days. Many other clinical syndromes have been associated with CMV infection including Guilian-Barre syndrome, meningitis, myocarditis, colitis, and Minitryre's disease, among others. The causal role of CMV with these other diseases is unclear. Cmv retinitis was rarely identified by CMV in immunosuppressed and immunosuppressable individuals and was uncommon even in severely immune people before AIDS in adults (54, 63,153). The most common urination is observed in infants with congenital urinary removal syndrome (29). Prior to the availability of HAART, retinal CMV was a common complication identified in people with advanced AIDS (CD4+ +< 50 cells/µl) (82). CMV otitis was one of the original infections that identified AIDS. It can be diagnosed on an ophthalmology examination by a characteristic confluence, white yellow and white retinal retina Often with a granular appearance or a more fulminant appearance with retinal bleeding. Without treatment, as immunity suppresses, retinitis develops into retinal necrosis and permanent loss of vision especially when it involves the macula and optic nerve. CMV disease occurred in 21% to 44% of patients with AIDS before the introduction of HAART (64, 141). Of patients with CMV disease, approximately 85% had CMV retinitis, especially those with CD4 + lymphocytic counts less than 50 cells/µL (189). In contrast, the incidence of CMV in the HAART era was estimated at 0.36/100 people-year based on 29 accidents among 1,600 HIV-infected patients without CMV retin in the initial examination who were followed up for at least one year (8,134 people-years follow-up) (206). Cd4+ lymphocytes were less than 50 cells/µl in 26 of the 29 patients on a 3-month visit prior to diagnosis was the most important risk factor for CMV retinitis with a risk of 136 (95% C.I. 30 to 605) (206). A longitudinal study of 503 patients with dominant retinitis and the accident, corrected for follow-up length, revealed 9 times less mortality, 28 times less progression of retinitis, 4 times fewer retinal detachments, and 2 times less moderate or severe vision loss in patients who entered the study with retinosis CMV (91). Succession and complications do not stop completely even as the immune system improves. Most of the vision loss in CMV earinfection is attributed to the involvement of enteritis from the posterior pole of the eye among 379 patients from the HAART era (494 eyes) (208). Cataracts also caused vision loss in some patients but retinal detachment carried the worst diagnosis: 100% of eyes with retinal detachment had moderate vision loss and 42% had severe vision loss (208). The U.S. Department of Public Health recommended in 1999 that antiviral therapy be discontinued in patients with emitised CMV retin and D4+T lymphatic over 50-100 cells/µl for 3-6 months with LEVELS OF HIV RNA (211). In a group of 71 patients meeting these criteria, nearly half of the treatment was discontinued. During longitudinal follow-up, there was no statistical difference between the groups in mortality, the development of otitis, or loss of visual acuity (81). There are no similar guidelines for restarting secondary prevention in patients with otitis who have a deterioration in the immune system; Resume if CD4 + lymphocytes fall to less than 50-100 cells/microliter seems reasonable. Ophthalmology and home monitoring of vision changes are usually performed at least every 3 months for patients most at risk. Patients with CMV earinfection may have a buoy, flashes of light, blurred vision, or blind spots in addition to loss of vision. Moderate anterior ullitis, vitreous, and retinal vasculitis may occur even in the early stages of the disease. Immune recovery, estimated at 17.6% (95% C.I. 12.3% - 24.1%) of patients with CMV retinitis are at risk for intra-eye inflammatory complications of uveitis, macular edema, epiphany membrane and vision loss (103). Immune alveyinfection is more likely to have significant lesions than CMV. Does not respond to antiviral therapy as otitis is inactive. Eye complications related to the immune response to CMV ear infection may also occur in immune patients with active CMV otitis, especially older people and patients with relative immunity from medications or disease. These patients may develop complex atypical or chronic retinal infection due to network ischemia and vascular obstruction (37,171) diagnosis is usually delayed or patients are treated with ineffective antiviral therapy with acyclovir or valasiclovir (171), resulting in a higher risk of severe vision loss from vascular complications (37). Multiple and myeloid myelitis of various syndromes involving CMV in CNS, CMV polyculopathy is the most common and characteristic. Patients experience the beginning under the sphincter of weakness, loss of reactions, changing sensory loss, usually in the legs, in association with bladder and nerve dysfunction (Table 1). On a pathological point of view, there is a CMV infection of ventricular and thadeniroots roots of equine codeand often the spinal cord is adjacent, with acute inflammation and axial necrosis. A CSF examination usually shows pleural fibrosis, often with the predominance of polymorphic leukocytes and hypoglycorrhachia (32). CMV encephalitis there are two types of CMV encephalitis in patients with AIDS. The first is diffuse, multifocal, micronod CMV encephalitis that is difficult to distinguish from contracting dementia caused by HIV (3, 61). In a retrospective autopsy study, Holland et al. (84) reported that out of 220 autopsies performed on patients with AIDS, 14 showed CMV encephalitis, and 17 hiv dementia sat without CMV involvement in the central nervous system. Both disorders show cognitive and motor disorders such as confusion, forgetfulness, indifference and withdrawal, instability, impaired memory, and widespread hyperconfusion. CMV encephalitis was not associated with typical abnormalities or positive cultures of CMV in the spinal fluid. CMV PCR of CSF is almost always positive, a diagnostic test of choice when suspected OF CMV infection of the central nervous system (30, 73, 78, 184, 226). CMV dementia may be associated with hyposodiumemia in the blood and signs of Addison s. Type II encephalitis CMV is CMV ventritis. These patients often have characteristic ventricular inflammation that can be demonstrated by magnetic resonance imaging (MRI) with gadolinium enhancement of the temporal areas (156). Ventritis is associated with ependymal necrosis and under necrosis. Patients are usually at an advanced stage Of AIDS, with CD4 + lymphather counts less than 50 cells/µL. They have a sharp beginning of indifference, confusion, fellow cranial nerves, and nistagmus, usually advancing rapidly. Ventual enlargement is usually seen. CSF is usually abnormal, often with the predominance of neutrophils. As with other central nervous system complications associated with AIDS, the availability of effective artic therapy may make CMV encephalitis uncommon. Mono-euromitis is a common encephalitis, which is less common in CMV CNS disorders, caused by epineural necrosis vascular inflammation, characterized by the penetration of polymorphic leukocytes. Patients present with multiple focal or asymmetric sensory and motor deficits in the peripheral nerves or the main skull are in a position of severe immunosuppression. Larynx nerves may be particularly involved (181). This syndrome may coexist with CMV, multiradical inflammation or encephalitis. Electrophysiological studies may show axial neuropathy and CMV DNA is detected in CSF. Gastrointestinal disease including mouth, esophagus, stomach, small intestine, colon and rectum may be involved with CMV disease in immunosuppressive patients including transplant recipients and people with AIDS (69). As mentioned earlier, CMV disease in these locations requires a demonstration of the virus as well as local hemological evidence of specific lesions of CMV. CMV manifestations include painful erosions or ulcers in the mouth, epiglottis, or pharynx. Odynophagia is a common symptom. The esophagus may be involved in a solitary ulcer or diffuse esophagitis associated with upper gastrointestinal bleeding. Esophageal stenosis may occur after healing. Stomach ulcers may cause bleeding, stomach obstruction, or puncture. The involvement of the small intestine includes progressive diarrhea associated with ulcer necrosis that may be complicated by a hole. Myocardial infarction may mimic Crohn's disease clinically and may be associated with massive intestinal bleeding. Patients with colon disease may have diarrhea, hematochezia, cramps, and abdominal pain associated with constitutional symptoms such as fever, loss of appetite, and weight loss. Severe bleeding, especially from cecal ulcer, has been reported in transplant patients. The colon may appear diffuse ulcers, focal ulcers with skipping areas, or less pseudopolyps and pseudomembranes. The most common site of gastrointestinal involvement in AIDS patients is the colon, especially in the rectal area (46). Hematochezia is seen in a minority of AIDS patients (40, 46). The frequency of CMV disease in the gastrointestinal tract in transplant recipients varies from 2% to 15% in different reports (26). It seems to vary with the frequency of CMV disease in general, one of which is the type of implant (79). In the kidneys. In the liver recipients, the frequency varies from 2% to 6% (87, 100, 125, 151). In the heart, heart and lung recipients, it is 3% to 16% (100, 125, 128). The frequency is higher in the myelitis recipient, although the specific diagnosis is not always possible due to graft versus host disease (28). Central nervous system INFECTIONS CMV is associated with specific syndromes of the central nervous system (CNS) in patients with AIDS and other immune persons (126). As with CMV retinitis, these syndromes are most common in advanced AIDS when the number of CD4+ lymphocytes is less than 50 cells/µL and effective treatment is not given antiretrovirals. However, manifestations of the central nervous system including meningitis, encephalitis and transverse myelitis were observed in immune patients (158). Laboratory diagnosis depends on the diagnosis of CMV disease on the suspected disease, patient age and clinical background. For example, it can be assumed that an infant suspected of a congenital infection who has been detected by any rate within 2-3 weeks of his life is congenitally infected. Similarly, a person with AIDS and the number of CD4+ lymphocytes + less than 50 cells/µl with retinitis diagnosed by an experienced ophthalmologist, will always have CMV retinitis. By contrast, because almost all people with seroimmunity will reactivate CMV in urine or saliva, the detection of CMV at these sites does not indicate the disease. In many cases the final diagnosis requires the identification of CMV in the specific device in question and the exclusion of other potential pathogens. Serological sayings are most useful for identifying past infections; A positive examination of specific IGG CMV indicates previous infections. The conversion from the control state to the positive iam indicates the recent infection and indicates that a severe disease may be associated with CMV. However, when both CMV igm and IgG are positive, the initial infection of reactivation cannot be definitively determined unless the patient's previous CMV status is known. Although the detection of IgM CMV antibodies has been used to diagnose congenital CMV, up to 30% of infected infants will have negative IGM CMV; IgM fakes may also be positive as measured. Thus, it is recommended to identify infectious CMV of urine, detect antigen or detect specific CMV DNA to identify congenitally infected infants. Recently, sayings that measure the thirst of antibodies to CMV have become commercially available. These measures are based on the principle that within the first few months of infection, antibodies have a low thirst and over time increased the thirst of specific antibodies. These statements were particularly aimed at assessing pregnant women, where it is often important to distinguish between pregnant women of recurrent infection. However, the ability of these measures to distinguish between primary infections remains uncertain and such tests should be used with caution. Although culture remains an important tool for diagnosing CMV infections, in most cases culture has been replaced by more sensitive and rapid tests including antigen detection and DNA detection tests. High number of deaths in urine and saliva from congenitally infected infants or immune persons. In such cases, viruses can be isolated in culture within a few days. However, many cultures take from 3 to 6 weeks to prove a specific cytopathic viral effect. Cultures of urine, saliva and buffy coat can be enriched by centrifuge and when combined with the immunity of CMV using monoclonal antibodies specific to early or early proteins can often lead to the detection of CMV in culture within 24-72 hours (67, 146, 157). The detection of the antigen pp65 was used as a monoclonal antibody oriented towards the protein matrix CMV, pp65, to detect CMV directly in the peripheral blood polymorphonuclear leukocytes (74, 107, 146, 230). This examination is generally more sensitive to culture, is useful for rapid identification of acute CMV infection, and commercially available. The amount of antigen positive cells has been associated with the presence and risk of subsequent CMV infection in people with immunity, including transplant recipients and people with AIDS. The main disadvantages of antigen screening are that samples should be processed within a few hours of obtaining them, and fresh blood must be distributed to a segment that is likely to increase the risk of the transmission of an contagious agent to laboratory staff. DNA detection methods are available a number of different methods of detecting CMV DNA or RNA in clinical samples that either inflate the target or investigate allowing for high sensitivity and specificity. Among these PCR measures, words, quality or quantity, are the most common (4, 19, 20, 195, 200, 203, 227). PCR is extremely sensitive and may detect the presence of CMV in clinical samples when the risk of CMV disease is relatively low. PCR amounts for CMV have shown that the risk of CMV disease increases with increased cmv detection in blood samples (plasma or cells) if effective antiretroviral therapy is not available. In addition, in people with untreated AIDS with the amount of CMV DNA present in plasma increases, there is an inherent risk not only for the development of CMV disease but also mortality. In fact, in people with advanced and untreated AIDS (CD4 + lymphocytes chargeless less than 50 cells /µL), the presence and amount of CMV DNA is present in More predictive than survival of the amount of plasma HIV-1 RNA (197, 203). CMV detection by PCR in cerebrospinal fluid (CSF) is the non-invasive diagnostic procedure of his choice to diagnose CNS disease associated with CMV (30, 73, 174, 226). PATHOGENESIS DURING NATURAL INFECTION, CMV SEEMS TO ENTER THROUGH THE EPITHELIUM LINING OF THE RESPIRATORY TRACT, DIGESTIVE OR REPRODUCTIVE SYSTEM. However, the infection may also occur through other direct methods including through blood transfusion as it occurs with blood transfusion and blood products, and through the transplantation of sero-donor bone marrow and solid organs. During acute infection, CMV can be detected in copied and modular polymorphic leukocytes (35). Currently, the best evidence suggests that CMV establishes latency in myeloid ancestral cells and in endothelial cells of solid organs; The virus can also be detected in circulating monocytes of serological individuals. The genes associated with CMV latency are not clearly identified. Whether CMV establishes real latency or expresses a few latency texts is still under active investigation. How a cell can carry the virus genome but does actively replicate the switch from latency to active replication is also unknown. It is remarkable, however, that 50% to 100% of CMV serum transplant recipients reactivate CMV during immune suppression which is higher with greater immunosuppression and when seronegative recipients receive a transplant from a serum donor (79). After initial infection and reactivation in the immune host, CMV can be detected in the blood during acute infection. The infectious virus is present in circulating monocytes and polymorphinocyle leukocytes, and viral DNA is present within the plasma. The spread of CMV occurs during acute infection and when it cannot be controlled by the host immune system it may spread to almost all organs. Much evidence suggests that natural killer cells, cell-mediated anti-mixing responses and immune systems all play an important role in the fight against CMV infection. However, the fact that almost all major and life-threatening diseases occur in people with weak cellular immunity, including organ transplant recipients and AIDS patients, underscores the importance of cellular immunity in controlling acute CMV infection. A stark example of the success of the immune system's ability to control CMV is shown in people with advanced AIDS who before the availability of HAART have often developed a serious CMV disease. However, the same patients with severe CMV disease when treated with HAART and have increases in the number of CD4+ lymphocytes above 100-150 cells/µl are able to control their infection without the constant need for anti-CMV treatment (see below). allergies in the laboratory and in one vivo drug at present there Medications (ganciclovir, foscarnet and Cidofovir) approved for the treatment of CMV infection. Of these factors, Gancyclofer and foscarnet are used only routinely to treat the broad spectrum of CMV diseases and prevent diseases. Ganciclovir, foscarnet and cidofovir all prevent polymerase DNA CMV. Ganciclovir (9-1-1,3-dihydroxy-2-propoxy)methyl)-guanine, DHPG), analog nucleoside, inhibits CMV and also has activity against other herpes viruses including herpes simplex virus, varicella virus, Epstein-Barr virus, human herpes virus type 6 and human herpes virus type 8. It is the preferred treatment for CMV, but it is also effective in immune hosts for the prevention and treatment of herpes simplex infections and varicella zoster and in some studies in reducing the risk of lymphnode disease associated with EBV and in the treatment of oral hairy leukocytes. Ganciclovir is a phosphorus to its monophosphate form by phosphokinase encoded within the UL97 region of CMV and other phosphorous to its active triphosphate form by cellular kinases. The effective inhibitory concentration (IC50) in CMV gansilovwar ranges from 0.3 to 8 micrometers. Most insulationhas an IC50 of 1.5 µM or higher. The peak plasma concentration of ganciclovir after a dose of 5 mg/kg given the infusion of vein 1 h is 6.6 µg/ml (4 µm = 1 µg/ml). Trough levels were 1 µg/ml after 11 hours (192). The usual dose of ganciclovir for intravenous induction is 5 mg/kg twice daily for 10 to 21 days. The maintenance dose is 5 mg/kg per day. Foscarnet is a isotope of pyrophosphate which also inhibits CMV DNA polymerase. IC50S (µM) for CMV, HSV-1 and HSV-2, varicella zester virus and Epstein-Barr virus (EBV) are 50 to 800, 10 to 130, 48 to 90, and less than 500, respectively. Foscarnet penetrates the blood cerebrospinal fluid barrier (CSF) with a coefficient of 0.05 to 0.72. After one intravenous infusion of 90 mg/kg, CMAX in constant condition is an average of 623 µM (1 µM equals 0.3 mg/ml). Corresponding CSF levels are nearly two-thirds of plasma levels (75). Cidofovir (HPMPC, (S)-1-(3-hydroxy-2-phosphonylmthoxy) cytosin is an analog nucleotides that inhibit CMV DNA polymerase. In the laboratory, cidofovir has also been found to have activity against VZV, EBV, HSV-1 and 2, herpes virus 6, 7 and 8, papillomavirus, infectious rolysome virus, glandular virus and smallpox viruses (including smallpox). and ID50 is 0.5 to 2.8 µm for wild isolation. Cellular enzymes are responsible for the serial conversion to the form of diphosphate, an active intracellular antibody (78). The form of diphosphate has a long half-life within cells, exceeding 48 hours (22). Acyclovir is the most common treatment for herpes simplex virus infection and zika virus vifella with ZIKA virus with IC50s from 0.1 to 0.2 m for most HSV and 4 to 20 m strains of VZV vv and VZv. Thethemydin kinase is coded by acyclovir phosphorylates effectively into its monophosphate form which is then more phosphorus into its active triphosphate form by cellular kinases. CMV does not encode thymidine kinase and is sensitive to esclofvir in concentrations of approximately 100 micrometers (110, 141) (1 µg/ml acyclovir equivalent to 4.4 µM). The high ID50 of acyclovir to CMV prevents its use to treat CMV infections clinically. However, it has been used to prevent CMV disease in solid organs and bone marrow recipients of some success (but inferior to both ganciclovir and foscarnet for the prevention of CMV disease; see below). Drug blend research has examined a combination of antivirals against CMV in the laboratory. Most studies have found that the combination of ganciclovir and foscarnet is usually added and in some cases synergistic. Similar results have been found to a combination of cidofovir with either ganciclovir or foscarnet. A clinical treatment approach examining a combination of ganciclovir and foscarnet in some environments has shown a modest benefit of combination but is associated with added toxins. Antiviral drug treatment of ganciclovir's choice of preferred drug for the treatment of CMV is ganciclovir. Treatment for the immune host usually requires twice-daily induction therapy followed by maintenance therapy of ganciclovir once a day (Table 2). Venous ganciclovir is secreted almost exclusively by the kidneys while with standard oral formulation ~ 85% is eliminated in the stool. An adjustment of the dose of ganciclovir is required for patients in renal failure (Table 3). Ghancyclovir cerebrospinal fluid levels range from 24% to 70% of plasma levels with concentrations within Viriberry approximately 10% to 15% of plasma levels. Neutrophils are a common complication of the treatment of gancyclofer that occurs in 25% to 40% of those receiving treatment. Neutrophils usually respond to the granular colon stimulation factor (G-CSF); However, after bone marrow/stem cell transplantation, ganciclovir may slow down the engraftment that made foscarnet the drug of choice for some organ transplant services. In a direct comparison between ganciclovir and foscarnet in the treatment of CMV earinfection in people with AIDS, foscarnet was as effective as ganciclovir but was associated with greater toxic reactions (189, 190). Of interest, AIDS patients, in one study, treated with foscarnet in the pre-HAART era was lower than those receiving ganciclovir (probably because of the modest activity of foscarnet antiretrovirals; see below). Oral Ganciclovir Valganciclovir because ganciclovir has twice the oral bioavailability, Valinest valganciclovir is an oral formulation of choice. Valganciclovir is monovalyl ester prodrug of ganciclovir. When managing it Valin is rapidly decomposition to ganciclovir. The biological availability of valganciclovir to ganciclovir is approximately 60%. A dose of 900 mg of valganciclovir provides levels similar to those obtained with an intravenous dose of 5 mg/kg (Table 2). Valganciclovir can be used for induction therapy as well as maintenance treatment of CMV otitis has been found the equivalent of an intravenously administered ganciclovir (122). Alternative treatment Foscarnet Foscarnet sodium is an inorganic pyrophosphate that prevents the recurrence of CMV as well as other herpes viruses by selectively inhibiting the pyrophosphate binding site of the virus-specific DNA polymerase. Foscarnet also prevents the reverse version of HIV. Unlike most nucleosides, foscarnet does not require phosphorus by viral or cellular coded kinases for its activity. Cerebral cerebrospinal levels range from 55% to 75% of plasma concentrations. Foscarnet requires intravenous induction followed by maintenance therapy, and is associated with a number of potentially serious complications. Induction therapy with foscarnet is given as 90 mg/kg intravenously every 12 hours. Maintenance therapy is 90-120 mg/kg given intravenously daily (Table 2). In people with AIDS time for ear infection relapse and survival was improved with 120 mg/kg maintenance dose compared to 90 mg/kg dose (83, 94). Foscarnet controlled pump ing with ≤ 24 mg/ml (undiluted) is required when given by central line or < 12 mg/ml (diluted at 5% dextrose or saline) via the terminal. The 90-120 mg/kg dose should be given at least 2 hours by means of the infusion pump. Patients should stay well hydrated during treatment. The drug is secreted exclusively by the kidneys and patients receiving inadequate hydration may have developed cervical ulcers or penis. Phoscarnet levels of kidney failure must be adjusted (Table 4). Cidofovir Cidofovir is an analog nucleotide with activity against CMV as well as other viruses. It has a long half-life (t1/2 = 17 to 65 hours), allowing for a weekly or longer schedule. Secretes 70% to 85% in urine. Preclinical studies have shown that the main toxicity of dofofoffer is dose-based renal toxicity, which is characterized by degeneration and necrosis in nearby torsional renal tube cells (108). Probenecid, which is believed to compete with cidofovir absorption in nearby tube cells, protects against kidney poisoning in animals and is crucial when giving cidofovir systematically to humans. A weekly dose of 5 mg/kg of cidofofer given intravenously is the maximum allowable dose of the drug (152). For the treatment of CMV otitis the drug is given weekly for two weeks for induction and once every two weeks for maintenance (Table 2). Probenecid (2 g) should be given orally 3 hours. Before each intravenous dose. Great evidence suggests that Sedofoviver is the lowest Either ganciclovir or foscarnet to treat or prevent systemic diseases with CMV. Failure to remove CMV viremia is common during cidofovir treatment, and patients who have been successfully treated for CMV earinfection are at risk of other CMV diseases. Formulations of antiviral agents have been shown in controlled clinical trials, a combination of ganciclovir and foscarnet to be effective in people with AIDS with CMV earinfection who have failed monotherapy with any of the drugs but who have not developed resistance. This combination has also been given narrative in other CMV diseases including gastrointestinal diseases and polyradiculopathy in AIDS patients. Although this combination has been a modest success, the long time required for intravenous use and added toxins has limited its use (see below). Resistance to antiviral agents used in the treatment and prevention of CMV develops resistance to ganciclovir, foscarnet and cidofovir well described which is an almost inevitable result of acute CMV infection associated with prolonged immunosuppression. Great research has established genotypic and phenotypic resistance patterns ganciclovir, foscarnet and cidofovir (14, 27, 28, 48, 49, 50, 51, 56, 92, 182,198, 225). Low-level resistance to ganciclovir (ID50 between 8 and 30 micrometers) is associated with mutations in the UL97 area of CMV that encode phosphovicyinase involved in ganciclovir phosphorus (184). High-level gansiclofer resistance (> 30 micrometers) is usually associated with both UL97 mutations and mutation within UL54 (DNA polymerase). Although insulations with low-level resistance to ganciclovir are still sensitive to sydofwer, isolates with high level gancyclovir resistance are usually also resistant to cidofovir. Most DNA polymerase mutations that give resistance to ganciclovir and Cidofovir are still sensitive to foscarnet. However, in the author's experiment, patients with CMV strains with high resistance to ganciclovir within months develop fnetoscar resistance as well if they are still significantly immunosuppressive. There is no clinical evidence that resistant virus is less pathogenicity than sensitive CMV strains. When immune patients are treated with any of the three antivirals approved to treat CMV disease, CMV resistance may develop at a local location while the virus inside the blood circulation or urine remains sensitive. This is particularly true for people with AIDS and CMV earinfection where although there is a viral suppression within the blood, retin itis progresses. Genotypic analyses by Smith et al. showed that in such cases a resistant virus could be detected in a glass or watery humor removed from the eye in question (183). Infections especially otitis with treatment before the availability of HAART, cmv earinfection can be or contained but never healed. The development of retinal lesions can be extended from an average of 3 weeks to 180 days or more (120, 189) as long as maintenance treatment continues. There are five FDA-approved treatments for CMV otitis (venous cansiclovir, oral valganciclovir, foscarnet vein, Cidofovir vein, and an eye device containing ganciclovir) approved to induce and maintain the treatment of CMV ear infection. Two other medications have been approved either for maintenance treatment (ganciclovir oral) or rescue treatment (intiferi lephresin injection). Rescue treatment can also be done with injections within vireal of ganciclovir, foscarnet or cidofovir. In the case of cidofovir, caution should be exercised due to the risk of low eye pressure (loss) associated with injections. Oral ganciclovir reduces new CMV earinfection lesions and systemic CMV diseases in patients who are still immunosuppressive. Maintenance treatment with valganciclovir is likely to replace oral ganciclovir administration in such patients with CD4 + constantly low lymphatic counts. Ganciclovir vein before the license of the sexual intravenous clover has shown a great anecdotal experience of the effectiveness of ganciclovir against CMV otitis (7, 60, 61, 85, 86). However, the common experience was that the disease occurred after treatment was discontinued and that maintenance treatment was necessary for an indefinite period of time (132). Spector et al. (202) showed the effectiveness of ganciclovir intravenous therapy for CMV earinfection in a randomized controlled trial. During a 16-week follow-up period, otitis in 10 of the 13 study participants was offered randomized treatment compared to 20 of 22 random treatment for direct venous induction treatment ganciclovir (5 mg/kg twice daily for 14 days) followed by 5 mg/kg of maintenance. The average time for progress in the immediate treatment group was 49.5 days, compared to 13.5 days for delayed treatment. Although strict criteria for the development of otitis have been used in this study and ganciclovir was found to be effective, the benefit of treatment was relatively short-term indicating the need to restore immunity if people with AIDS were to fully recover from their CMV earinfection. Foscarnet Foscarnet was evaluated intravenously by Palestine et al. (145) in a randomized controlled trial in people with AIDS and CMV earinfection. The average time for progress was 3.2 weeks in the deferred treatment group compared with 13.3 weeks in patients who received immediate foscarnet induction (180 mg/kg/day divided into three doses) followed by maintenance therapy of 90 mg/kg per day. In a comparative experience of foscarnet vs. ganciclovir for the treatment of CMV earinfection in the pre-HAART era, no significant difference was observed for the two therapy; Progress was 56 days in the ganciclovir group compared with 59 days in the foscarnet group. However, the mortality rate in the Gansiclofer group was 77% higher in the Voscarnet group. The average survival times were 5 months in the ganciclovir group and 12.5 months in the foscarnet group. The best explanation for this difference was the modest anti-retroviral foscarnet activity that was supported by patients treated with foscarnet experiencing higher CD4+ lymphates charges in the 4 and 16 weeks after starting treatment. In this trial, conducted in conjunction with the Group of Clinical Trials of Adult AIDS (ACTG) and Studies of Eye Complications of AIDS (SOCA), neutrophils were more common in patients who received ganciclovir (34% vs. 14%). However, patients receiving foscarnet experienced a greater number of complications associated with pumping (58% vs. 24%), more urogenital symptoms in male patients (56% vs. 16%), and more abnormalities of renal and electrolyte poisoning (13% vs. 6%). Additionally, patients assigned to receive foscarnet were initially more likely to switch to gancyclofer treatment due to toxicity (46% vs. 11%). The incidence of seizures was the same in both groups (9 per cent to 12 per cent). The toxic effects were reversible, with no permanent disability or death (180). Thus, although foscarnet has been shown to have equal efficacy for ganciclovir for the treatment of CMV in people with AIDS and may improve survival (although this has not been confirmed in subsequent studies, it probably won't have any effect when CMV treatment is combined with HAART), increased toxicity associated with foscarnet and relative ease in the management of ganlovir led to gancicvir being the preferred drug in most centers for the treatment of CMV ear infection associated with AIDS. Cidofovir Cidofovir, the third approved drug for the treatment of CMV earinfection, was evaluated during the era of increased availability of various nucleosides and a neoclecide inhibitor, nevirapine. In a randomized controlled trial, Lalyzari and his colleagues (109) found that the development of otitis occurs in patients assigned to delayed treatment with an average of 22 days compared to 120 days in those assigned to immediate treatment. However, it was necessary to discontinue treatment in 24% of patients due to the development of 2+ protein or creatinine levels of 2 g/dislet or more. A second study comparing delayed therapy to an initial induction of 5 mg/kg once a week for two weeks followed by high dose maintenance (5 mg/kg once per week) or low dose maintenance (3 mg/kg once per week) found similar results for the Lisari study with no difference between the treated arms. It should be noted that in both studies, a probe at 5 mg/kg was administered with all doses to reduce the frequency and of renal poisoning. Although Cidoofvir has been found to be useful for the treatment of CMV otitis and has a non-recurring dostosis feature, the drug failure to clear vermia and the high risk of renal poisoning has generally led in Cidofovir being second-line treatment behind ganciclovir and foscarnet for the treatment of CMV otitis. Uveitis and secondary hepbutors to administer venous cidooffer can be vision-threatening and usually require dose interruption or alternative therapy (36). Combining Ganciclovir and Foscarnet in cases where a single agent has failed to combat CMV, a combination of ganciclovir and foscarnet has been found to be modestly successful. In a study of people with AIDS in pre-HAART who were consistently active or relapsed otitis CMV, patients were nested into three different therapeutic groups consisting of high dose foscarnet (120 mg/day maintenance), ganciclovir high dose (10 mg/kg/maintenance day) or foscarnet (90 mg/kg/maintenance day) plus ganciclovir (5 mg/kg/day maintenance) (189, 189, 189). The survival rates were equal in all three groups. The time for the development of otitis was 1.3 months for the foscarnet group, 2.0 months for the ganciclovir group and 4.3 months in the composite group. Interestingly, patients who remained on monotherapy but were switched treatment did not show any obvious benefit in time for the first progress compared to patients who were not switched. This data supports the concept that drug resistance may not be responsible for all INTS CMV earinfections. In the SOCA study mentioned above, the composite group also had the lowest rate of change in the retinal area involved in the loss of the CMV field and the optical field. The combination of superior therapy was not associated with more toxic effects than any monotherapy. However, combination therapy had the greatest negative impact on quality of life measures. This has been associated with more frequent treatment changes and longer infusion times than monotheistic treatments. In conclusion, the combination of ganciclovir and foscarnet showed superior suppression of CMV earinfection in people with advanced AIDS before the availability of HAART. The disadvantages of combination therapy including discomfort, higher cost and less patient acceptance have made the combination uncommon only in severely debilitating or life-threatening infections where monotherapy fails. With the recent availability of valganciclovir, it is possible that the combination of foscarnet and ganciclovir (using valganciclovir) will become the first line treatment more popular in some life-threatening CMV infections or high morbidity when the patient can take the drug orally. However, with the availability of HAART, the combination of ganciclovir therapy with effective antiretroviral therapy leads to successful treatment in most cases of otitis. Ganciclovir as maintenance treatment despite the low bioavailability of oral ganciclovir, large data support its use to treat maintenance of CMV ear infections in pre-HAART-era patients with advanced AIDS (49, 144, 196). The average number of days for the development of otitis in clinical trials has been found to be similar between oral formulation and intravenous form. However, the data also demonstrate the superiority of venous form with regard to the progression of otitis. Despite the benefit of intravenous maintenance, more than ganciclovir mouth is used to keep otitis stable due to increased comfort and less negative events. Diarrhea and niatbs are common adverse events with oral ganciclovir as well as intravenous form. Valganciclovir Valganciclovir is a prodrug ester monovalyl of ganciclovir that when administered orally it is hydrology quickly to ganciclovir. Once the drug has been decomposed the pharmacokins are identical to the mother compound. The absolute biological availability of ganciclovir of valganciclovir is approximately 60% (99, 122). A dose of 900 mg of valganciclovir achieves levels similar to 5 mg/kg giving a vein of ganciclovir. Recent data indicate that the treatment of CMV otitis with an enticing dose of valganciclovir 900 mg twice daily for three weeks followed by 900 mg daily maintenance equivalent to ganciclovir equivalent to ganciclovir 5 mg /kg intravenous induction followed by maintaining 5 mg /kg intravenously. The adverse events were similar in the two groups. Valganciclovir is a rapidly replacing venous ganciclovir in patients with CMV disease who are able to take oral medications. Moreover, valganciclovir is a ganciclovir oral replacement for maintenance treatment, as well as disease prevention/preventive treatment in most cases except when there are liver diseases and anxiety from changing the metabolism of valganciclovir. Intra-eye administration of intravenous therapy drugs with drugs currently approved against CMV otitis is complicated by the high frequency of side effects, the inconvenience of intravenous administration, and the high cost of daily intravenous drug administration. Experience with local treatment has accumulated since eye injections of ganciclovir were initially reported in 1987 (58, 76). Doses originally prescribed were 200 mcg in 0.1 ml of ganciclovir sodium and 1.2 mg at 0.05 ml to 2.4 mg in 0.1 ml of foscarnet. Gancyclofer concentrated sodium solutions can be made to deliver 2000 mcg in 0.1 or 0.05 ml with good safety and effectiveness (230). Induction therapy usually consists of injections twice a week for two to three weeks followed by weekly injections. Another approach to intra-eye management of ganciclovir was to develop a commercially available intravitreal device, which consists of propagation cells that are lined by permeability The membranes contains ganciclovir that are surgically implanted in glass. Using this approach, CMV earinfection can be successfully controlled (121, 134, 189). In the pre-HAART era, retinitis recurrence times were significantly prolonged by intracardiac implants compared to any other treatment method in patients with advanced AIDS. While the average time for progress with the venous formula was found to be about 70 days, the development of retinitis occurred after 220 days in two studies managing 1 µg/ml through intravitreal implants. It should be noted, however, that the risk of retinal itis in the unconcerned eye or other systemic CMV diseases was lower in advanced AIDS patients receiving ganciclovir in the vein than those with implantation. For this reason in patients who remain immune, systemic ganciclovir should be administered in conjunction with implantation to prevent the development of CMV disease at a new location. With HAART, patients responding to HIV treatment with an increase in cd4+ cells above 100 cells/µl may not need systemic treatment. The Cydophofer virus inside Vireal was given to patients with advanced AIDS and CMV ear infections (105). After one injection of 10 mg, the average time for the development of otitis in the pre-HAART era was about 55 days that was positively compared with the systemic use of ganistchelofer or phoscarnet. Multiple injections with cidofovir further prolong the time for the development of inflammatory bowel. Mild to moderate auretositis develops in approximately 35% of the eyes pollinated with cidofovir. In addition, low eye pressure (habutoni) is observed with intraocular cidofovir administration, and it is not currently recommended to manage intraocular limitation. In short, intra-eye administration of antivirals is effective in controlling CMV ear infections. The advantage is that a high concentration of the drug is delivered locally while avoiding the toxicity of intravenous drug therapy and disturbing the drug catheter intravenously. The disadvantages of local treatment are that treatment does not prevent the development of otitis in the other eye or CMV disease elsewhere in the body. For patients whose systemic antiviral therapy is insufficient to control the development of retinitis, treatment combined with an intra-eye device containing ganciclovir and either oral ganciclovir (120) or oral valganciclovir seems to be the best strategy to provide highly effective control of retinal infection while reducing the risk of systemic diseases or second eye involvement. Choosing the primary treatment for CMV is a multifactor decision that integrates the patient's immune condition and previous history of antiretroviral therapy (119). Patients who are naïve HAART or recently started on HAART have an excellent opportunity to respond to oral therapy alone without the need for treatments inside in fact. Patients with the development of otitis with systemic anti-CMV therapy should be quickly transferred to in-reality treatment. CMV retinitis in the HAART era with the availability of effective antiretroviral therapy, patient with AIDS and D4 + lymphatic count of less than 50 cells/µL has the opportunity to significantly reduce viral load and increase their CD4+ cells. Large data suggest that maintenance therapy for CMV retinitis can stop safely when patients with quiet retinitis and D4+ lymphocytes have risen above 100-150 cells/µL for at least 3 months (33, 210, 216, 218). It is essential that all patients who have CMV maintenance therapy stop their CD4 + number of lymphocytes routinely monitored as part of HIV/AIDS care and undergo regular eye examinations to make sure that otitis is still inactive. The syndrome associated with

immunoreshaping has been described in patients with CMV otitis, immunology uveitis, and has been prescribed (101, 136, 193). In such cases of immune activation, anecdotal reports of systemic and perforatal corticosteroid therapy have been helpful in controlling uveitis. CMV maintenance therapy should be re-treated with any recurrence of CMV earinfection without regard to the number of lymphocytes or when CD4+ lymphocytes decreased less than 100 cells /μL. Polyradiculo lumbiseral morbidity and myelitis although there are no controlled trials, clinical improvement was reported after induction therapy and maintenance with venous ganciclovir (32, 38, 104). In one study (32), 6 out of 16 patients with multiple neuropathy treated with ganciclovir responded with clinical improvement and stability. Factors associated with the non-response to treatment were the persistence of polymorphic leukocytes, low glucose levels in CSF, and severe parparesis before the start of treatment (38). Patients who developed polyradiculo therapy during anti-CMV therapy often did not respond. The lack of response is likely to be linked to irreparable damage to the nervous system or to the presence of a resistant virus (224). Treatment of patients with multiple neuropathy syndrome with foscarnet or a combination of ganciclovir and foscarnet has resulted in results similar to those treated with ganciclovir alone. In the author's experiment, unless the diagnosis is made within days of clinical symptoms, usually through the characteristic presentation in the patient with advanced AIDS confirmed by PCR positive for CMV DNA of cerebrospinal fluid, the stability of multiple neuropathy without significant improvement is the norm. CMV treatment of encephalitis for AIDS patients with CMV encephalitis with either ganciclovir and/or foscarnet has met with variable success. In some reports, treatment with ganciclovir led in radiology From ventritis and conversion of CMV cultures are positive but little improvement of objective clinical nerves (156). Salazar et al. (167) reported four confirmed cases of CMV ventricular inflammation with clinical and radiological responses to treatment. Three patients received gansilufer and two were received by phoscarnet. One patient showed remarkable neurological improvement and radiological accuracy by MRI after 4 weeks of ganciclovir treatment. Two other patients deteriorated on Gansiclofer and two on Voscarnet and died. CMV encephalitis may occur during patients with advanced AIDS receiving ganciclovir maintenance or foscarnet treatment for CMV ear infection, even when otitis is controlled (11). In such cases, failure of sufficient levels of medications to access the central nervous system or to choose a resistant virus within the central nervous system may contribute to the development of encephalitis. As with multiple neuropathy syndrome, early suspicion of the disease with the confirmation of PCR in CSF and the early institution of antiviral therapy has the highest likelihood of improving the patient's outcome. Treatment of CMV encephalitis in the patient's immune system is unclear of interest; But in the mode of encephalitis, if CMV DNA is detected inside the cerebrospinal fluid, treatment with gancicovir is recommended. Multiple euromitis may be followed by temporary relapse or improved polytheis treating ganciclovir or foscarnet (165). Pneumovirus pneumonia is one of the most common sites of CMV disease. CMV pneumonia may be caused by different mechanisms of causing diseases. The treatment of CMV pneumonia thus varies, depending on the type of patient. CMV pneumonia has been rarely prescribed in immunosuppressing patients, in combination with cmv white blood cells (121). Deaths in this case are negligible, and antiviral treatment is very effective (although it is usually not necessary). The most severe type of pneumonia with the highest mortality rate occurs in bone marrow recipients. Untreated, the mortality rate is between 70 and 85 per cent (129). Used alone, ganciclovir suppresses the virus but is not effective in reducing morbidity or mortality. Treatment combining ganciclovir with immunoglobulin is now widely used (55,161). Reed et al. (55) reported that deaths were reduced to 30% in 10 patients who received ganciclovir and intravenous immunoglobulin. None of the 11 patients treated with either ganciclovir or venous immunoglobulin survived alone. The patient's course may be complicated: some resolve after 14 days of treatment, others are still symptoms and require treatment for maintenance, and others may still have a relapse after remission requires retreatment. Neutrophils are often associated with recurrent treatment with ganciclovir (160, 161), but usually respond to treatment with G-CSF. In conclusion, CMV pneumonia in marrow recipients is extremely fatal if left untreated. Ganciclovir or immunoglobulin alone is an effective treatment. The mortality rate has been reduced by the combination of mortality but still significant. For this reason, preventive treatment/preventive treatment is better than treatment of CMV pneumonia in these populations. In solid organ transplantation, the course and treatment of CMV pneumonia is less well documented. Mortality for different types of transplanted organs varies, from 48% in an early series of kidney recipients (151) to 75% in heart and lung recipients (52). Recent data indicate that deaths associated with CMV pneumonia are generally lower in recipients of solid organs than in bone marrow recipients (97, 172) likely due to a level of immunosuppression among different groups of transplant recipients. Duncan et al. (53) reported that 48% of a group of 124 lung recipients were infected with CMV pneumonia. The group treated by Gansikolofer had a much higher survival rate for one year than the untreated group (86% vs. 38%). However, survivors had clinically significant chronic complications, compared to those who did not have CMV pulmonary infection. The need to add immunoglobulin not documented in recipients of solid organs remains controversial, although the effective combination has been reported in anecdotal reports (34, 66 and see below). Although cmv disease and post-mortem lung infections occur in people with AIDS, CMV pneumonia is uncommon. The lack of AIDS-related CMV pneumonia even before the availability of HAART most likely reflects differences in causing CMV pneumonia in AIDS patients compared to pneumonia observed after transplantation. It is the result of an overwhelming infection with CMV (77, 133). In people with AIDS, CMV is often present in association with other opportunistic pathogens especially Carinii Pneumocystis. Treatment of Pneumocystis alone often leads in clinical improvement without the need for CMV treatment (21, 95, 98). However, many reports in the pre-HAART era have documented cases of CMV pneumonia in people with AIDS (6, 47, 68, 204, 215) so that diagnosis should be considered in patients with advanced AIDS who do not respond to traditional antimicrobial therapy for carinii Pneumocystis and common bacterial pathogens (127). Gastrointestinal disease in an uncontrolled report of ganciclovir treatment of 41 AIDS patients with gastrointestinal involvement in the pre-HAART era, clinical improvement occurred in 30 and virologic response at 32 (26). In a randomized placebo-controlled trial of people with AIDS and CMV colitis, who were treated with (10 mg/kg per day for 14 days) experienced a significant reduction of CMV positive colon and urine cultures compared to placebo which was associated with a significant improvement in 63% of the treatment group compared with 33% for those randomized to placebo (43). The treatment group maintained body weight, but the placebo group lost weight. Diarrhea improves evenly in both groups. In additional uncontrolled reports, AIDS patients with CMV gastrointestinal diseases who failed ganciclovir treatment (which is known as the development of CMV disease) benefited from foscarnet. Dieterich et al. (44) foscarnet was administered to 19 of these patients and observed an improvement of histopathy in 67% and clinical improvement in 74%. In a random comparison of the two drugs, no difference was observed between ganciclovir and foscarnet in the treatment of gastrointestinal diseases (12). Survival in both therapeutic groups (in the pre-HAART period) was less than 40 weeks and maintenance therapy was not affected. Thus either ganciclovir or foscarnet was effective as a first-line treatment for gastrointestinal CMV disease. Maintenance treatment did not prevent the progression of the disease. The combination of Gancyclofer and phoscarnet has also been successfully used to treat enteric hepatitis CMV (45). Ganciclovir treatment in the case of reports led to improved symptoms of gastrointestinal CMV disease in up to 93% to 100% of solid organ transplant recipients (100, 125). Mayoral et al. (125) used Gancycloiver to treat 14 recipients of solid organ transplants with intestinal CMV disease (all but one laparoscopic); 13 improved, 4 requires additional treatment for recurrent diseases. Kaplan et al. (104) reported that gastrointestinal diseases are the most common infections in heart and lung transplant recipients. The incidence included 9.9% gastritis, 9; gastric ulceration, 4; enteritis, 3; esophagitis, 1; pyloric perforation, 1; and colon bleeding, 1. They were treated with 5 mg/kg gansicellofer twice daily for 2 to 8 weeks. Relapses occurred in 4 out of 9 patients who were followed for 18 months. Reed et al. (162) conducted a controlled trial of myeloid recipients with intestinal CMV; 14 patients treated with gancyclovir and 19 received placebo. The most common involvement was the esophagus. Ganciclovir was given a dose of 2.5 mg/kg every 8 h for 14 days. No patient had a solution to all symptoms after treatment with ganciclovir. A partial improvement was observed in both treated control groups and placebo. The only significant difference was the actual elimination of the virus from regular and local cultures in the treatment group; 73% of treatment and 79% of the placebo group showed some improvement. This study showed no obvious advantage to treatment, although higher doses of ganciclovir (i.e. 5 mg/kg twice daily) may showed an advantage. Hepatic hepatic hepatic hepatic hepatic hepatic hepatic hepatic hepatic hepatic hepatic hepatic hepatic hepatic disease (CMV). In liver transplant recipients however, CMV hepatitis occurs in 2-17% of patients, which is the most common specific organ manifestations of CMV infection (112, 173). Almost all patients with HEPATITIS CMV have Vermia. However, the cmv viral load or blood antigen levels may be variable (112). Final diagnosis requires the manifestation of CMV in liver tissue by immunological analysis or on-site hybridization. Viral inclusions are often absent in histological examination. Absceses and balloons from liver cells, while not specific to CMV are the most observed hetological results (112). In most patients, CMV hepatitis represents the participation of an isolated organ without evidence of the spread of the disease. Intravenous ganciclovir 5 mg /kg twice daily (modification of renal dysfunction) for 2-4 weeks is recommended treatment (154). In patients with documented viremia, ganciclovir should last for at least a week after the decision of Vermia. Valganciclovir is an alternative therapeutic option in stable patients able to take oral medications. Clinical and viral results with valganciclovir were in transplant recipients with invasive CMV disease of tissues, including hepatitis CMV similar to those found in patients who received venous ganciclovir (89). It is not recommended to routinely add CMVIG to antiviral treatment for hepatitis CMV. However, CMVIG can be considered as an adjunct in patients with acute gastrointestinal diseases or in those with globocolinic sodium deficiency (154). Recipients of liver transplants with hepatitis CMV were more likely to develop post-traumatic stress disorder (112). However, in patients who have been treated appropriately, long-term outcomes are not compromised. Cmv congenital trials of uncontrolled infection (137) and a randomized, controlled study carried out through a cooperative antiviral study group (CASG) for infants born with congenital CMV have suggested symptoms that ganciclovir administration 12 mg/kg/day divided intravenously in two doses for six weeks when administered during the first month of life may be associated with a decrease in hearing deficits (217).   Follow-up infants in the CASG study showed that infants receiving ganciclovir had less delay in growth at 6 and 12 months compared to untreated controls (142). A recent study carried out by CASG presented, so far, only in the form of a mere 6-week examination versus 6 months of valganciclovir in infants with congenital CMV symptoms. A dose of VGCV was given 16 mg/kg given twice daily to all participants in the study. The participants were then randomly to continue vgcv at the same dose or to the placebo. At 12 and 24 months, infants who received 6 months of VGCV had more Have improved or normal hearing from those who have received 6 weeks of treatment. In addition, in 24 months there was an improvement in language and communication accepted in the group receiving 6 months of treatment. Importantly, there was no difference in neutrophils, coagulation or kidney poisoning between the two therapeutic groups. The main adjuvant therapy treatments studied for the treatment and prevention of CMV disease are IVIG and CMVIG. As previously mentioned, IVIG's combination with ganciclovir appears in a number of studies to improve results modestly in the treatment of bone marrow transplant recipients and CMV pneumonia. Snydman and his colleagues have defended the use of CMVIG to prevent/relieve CMV disease in solid transplant recipients (185). CMVIG use with ganciclovir was evaluated in patients after heart and chest transplants (212). In the study conducted by Valentin et al. patients were given the following heart, lung, lung or lung transplant ganciclovir plus CMVIG and compared with identical historical controls. In 3 years, there was a significant decrease in CMV disease (76% vs. 39%), and the proportion of patients rejecting free (34% vs. 11%). Total stay (91% vs. 63%) For ganciclovir in addition to CMVIG group vs. ganciclovir identical controls, respectively. A smaller number of recipients of the heart and chest also experienced obliteration bronchitis, 86% in ganciclovir plus CMVIG compared to 59% for those receiving gancyclovir only. The main problem with these data is the use of historical controls as a comparative group and the possibility that other factors may have significantly affected the outcome of the disease. Thus, the effect of CMVIG on the heart, heart, lung, and lung transplant patients that started early after the transplant requires a randomized controlled trial before final recommendations can be made. The use of this system has generally not been successful for people living with HIV/AIDS. CMVIG has been found unsuccessful in the prevention of CMV disease. Additionally, CMVIG fails when given in conjunction with ganciclovir to treat CMV earinfection to prolong the time for the development of otitis in people with AIDS in advance before the availability of HAART. As previously mentioned, the use of CMVIG to prevent congenital infections is controversial and awaits controlled clinical trials before its use during pregnancy can be recommended. The end points for ganciclovir therapy control use of the Ganciclovir theme requires monitoring neutrophilcount because neutrophils are more important than severe adverse reactions that limit the use of ganciclovir. Nores or absolute neutrophils count ing below 1000/μL occurs in ~30% of patients receiving ganciclovir (62), but may occur in up to 70% when administered with zidovudine (80). Severe nutrient deficiency occurs (< 500/μL) in 20% of patients. Neutrophils are usually resolved with discontinuation of treatment and can be prevented by the administration associated with inequities or connective granulocytes as a colonic stimulating factor (G-CSF or GM-CSF). Blood clotting is less common but of potentialserious complications of ganciclovir, especially in patients with AIDS, further indicates its effect suppressing bone marrow. Other side effects of ganciclovir include rashes, nausea, fever, vomiting, anemia, diarrhea, yosnophilia, confusion, seizures, and psychotic reactions in a few patients (25). Foscarnet the main toxicity of phoschernet is renal insufficiency, which occurs to some extent in most patients treated. Approximately 33% of the 189 patients with AIDS and CMV ear infectionwho received venous treatment foscarnet developed a weakness manifested in the concentration of serum creatinine of 2 mg/dislet or higher. These spikes are usually, but not always, the opposite of stopping the drug. Because of the academic chelates foscarnet chelates, they have also been associated with serum electrolyte imbalance such as callastacle deficiency (15%), hypoglycemia (50%), and hypokalemia (16%). Foscarnet has been associated with seizures in AIDS patients. Hypoglycemia may play a role in unexplained seizures or arrhythmias. Anemia has been reported in 33% of patients receiving foscarnet. This has been controlled, and less than 1% required to stop the drug for this reason. Granulocytopenia has been reported in 17% of patients, but only 1% have been terminated from studies due to neutrophils. Penile ulceration may result from the exposure of the penis to foscarnet without a change in urine (5,93). Patients receiving foscarnet must remain well hydrated in order to prevent such complications. Although congenital infections with CMV remain the leading infectious cause of congenital malformations in the developed world, there is still no vaccine available for CMV protection. A great deal of research is currently under way in this area; however, the CMV vaccine is unlikely to be licensed and widely available over the next few years. The prevention of bone marrow transplant immune recipients although a number of early studies reported that the use of immunoglobulin reduced CMV disease in marrow transplant recipients, other trials failed to prove useful (17, 18, 106,131,143,220). In a study of 97 serol recipients, Boden et al. (18) examined four specific groups: (a) intravenous immunoglobulin CMV and cypress blood products, (b) seroblood disease products alone, (c) immunoglobulin CMV alone, and (d) no treatment. CMV infections in the four groups concerned were 5, 13, 54, and 40%, respectively. The main conclusion of this study was that of 57 patients with seronegative donors, those who received seronegative blood products had significantly Infection (1 in 32) of those who received standard blood products (8 out of 25; P< .007). Immunoglobulin alone had no effect. This study showed that CMV infection and disease can be prevented in seronegative recipients who have seronegative donors by providing CMV-free blood products. The remaining question is whether immunoglobulin can prevent infection and disease in serorecipients with serodonors. Boden et al. (17) in a controlled randomized study of 120 such patients showed that they could not. In a study of serological bone marrow recipients (207), among 308 serorecipients evaluated, 22% of control patients and 13% of immune recipients with interstitial pneumonia. The incidence of acute graft versus host disease (GVHD) has been reduced from 51% in controls to 30% in immunoglobulin recipients. Whether reducing pneumonia is a direct effect of immunoglobulin or a secondary effect of reducing GVHD by immunoglobulin is unclear. These findings were not confirmed in a subsequent study (163). In short, there is currently no convincing data to prove the usefulness of immunoglobulin or hyperimmune globulin to prevent or relieve CMV in bone marrow recipients and this treatment is not recommended in such environments. Antiviral factors patients who are CMV serological before bone marrow transplantation are more likely to develop CMV disease after transplantation than serongative recipients. Regardless of the donor serostatus, the frequency of infection in the first 100 days after transplantation is about 70% (129). This group of patients was primarily targeted for antiviral prevention testing and treatment. Myers et al. (130) I found that intravenous acyclovir (500 mg/m2 given every 8 h starting from 5 days before implantation of cmv patients and lasted until today 30 thereafter) reduced the incidence of CMV disease from 38% to 22% and the incidence of CMV infection from 75% to 59%. The mortality rate associated with organ transplantation has been reduced from 54% to 29%. The European Acyclovir Study Group (155) assessed intravenous acyclovir at 500 mg/m2 three times a day or oral acyclovir for the first 30 days after transplantation and then oral acyclovir or placebo until today 210. The incidence of CMV, frequency of vermia, and mortality were lower in the group that received intravenous acyclovir. However, the frequency of CMV disease was similar in both groups. Boeckh et al. (13) evaluated venous acyclovir from 5 days before autologous transplantation to 100 days in a retrospective study. There was no difference in CMV or pneumonia. Thus, although some studies have proven the benefit of acyclovir (including valacyclovir) for the prevention of CMV disease, its benefits are less than ganciclovir or foscarnet It is not recommended to prevent CMV disease. There are two approaches to using ganciclovir in CMV chapel recipients. The first is early treatment or preventive treatment where patients identified as suffering from historically active CMV infection are given by surveillance cultures or currently usually via a direct CMV antigen or PCR detection in ganciclovir blood. The second approach is to manage ganciclovir after engraftment for all serological patients. After the first approach, Schmidt et al. conducted (170) bronchial control cultures (BAL) on the average day of drift (day 22) and recorded those with positive CULTUREs CMV. Twenty patients were randomized to the placebo group and 20 to the treatment group, who received the Hemssilver from day 35 after transplantation to day 120. CMV pneumonia has been reduced from 70% to 25%. Of the 55 patients who were not positive with BAL (and therefore not eligible for the study), 22% were infected with CMV pneumonia. Goodrich et al. (71) patients chose for preventive by examining cmv positive cultures of blood, throat wash, or urine. Patients received either ganciclovir until today 100 after transplantation or placebo. Results were obtained on 102 patients. CMV disease was reduced from 43% (15 out of 35) in the placebo group to 3% (1 out of 37 patients) in the treatment group during the first 100 days after transplantation. After the second approach, Goodrich et al. (70) gave Gancyclofer to all 33 patients, from engraving to 100 days after transplantation; 45% of placebo recipients (14 out of 31) had CMV infection 100 days after transplantation compared to 1 (3%) The receiver Gansiclofer. Neutrophils occurred in 30% of ganciclovir recipients. The mortality rate between the two groups did not differ. Winston et al. (219) gave ganciclovir for 1 week before pumping the marrow and then starting after engraftment until the 100th day after transplantation. CMV infection has been reduced from 56% to 24%. The above studies show that ganciclovir is effective in reducing CMV disease if administered (a) routinely for all patients or (b) selective for those found to be positive by surveillance cultures. But if one avoids excess toxicity by restricting management to patients at greater risk, there is a risk of losing those who develop the disease without positive control cultures. At the moment there is no perfect solution to this problem. However, studies that use antigens or pcr quantity provide excellent but not ideal. Prevention of CMV in the transplantation of cmv recipients in transplant recipients using antiviral agents generally using one of two strategies: 1), preventive treatment where the antiviral agent is targeted towards patients with early viral recurrence or viria in an attempt to prevent the development of symptoms To CMV disease, 2), prevention where the drug is given to all patients for the period considered to be at risk for CMV, usually 90-100 days after transplantation (148, 176). The main advantages of the preventive approach include the fact that it avoids the need for repeated monitoring to detect early virus recurrence, which is reliably protective against CMV during the time the antiviral agent is used, and is likely to provide protection against indirect effects or secondary outcomes associated with CMV. The disadvantages of this approach is that most patients while accumulating cost and possibly the risk of toxicity associated with an antiviral agent, are unlikely to benefit from it. There is also a potential concern for the emergence of anti-virus resistance. In addition, accumulated data show that while CMV can be effectively prevented during the time antiviral prevention is used (the first 3 months after transplantation), a large proportion of patients, particularly in the receiving Serum Group (R-/D+), develop CMV when prevention is discontinued (1, 96, 114, 147). The late CMV disease, defined as CMV disease, which occurs 100 days after transplantation, has emerged as a major complication with the use of valganciclovir prevention in the present era (1, 96, 114, 147, 176). The advantages of a preventive treatment approach are that the antiviral agent is directed only towards patients at high risk of CMV disease, and treatment is used for a specific and shorter period. This strategy, including monitoring costs, is associated with lower overall costs. The existing data did not document the late CMV disease with valganciclovir used as a preventive treatment (41, 124,178). The proposed basis for this observation is that implicit non-contagious buttons in the preventive treatment approach may facilitate CMV immune responses that are critical to long-term protective immunity against the virus (178). Antiviral agents include prevention options for antiviral agents that can be used as acyclovir, valacyclovir, or gancyclovir derivatives. Aceclivir due to its poor activity in the laboratory against CMV and low oral bioavailability has not been shown to be particularly effective for the prevention of CMV disease. Comparative studies have shown lower rates of acecliver effectiveness compared to ganciclovir containing systems (222). Valacyclovir is a valil ester of acyclovir with improved oral bioavailability. In a placebo-controlled trial in kidney transplant recipients, Valasiclofer (2 grams orally four times a day) significantly reduced the incidence of CMV disease and acute allograft rejection (117). Although not as effective as ganciclovir, Valasiclofer represents a cost-effective alternative to ganciclovir in kidney transplant recipients. Vein Long-term use has been shown to be effective for CMV disease. In a randomized trial that mostly involved serological patients, prevention was associated with venous ganciclovir who was given to liver transplant recipients for 100 days with a CMV rate of 1% in all patients, and 10% in those at risk of developing primary CMV, i.e. serorecipients of serological allografts (222). However, this approach did not receive widespread acceptance due to the requirement of prolonged access to blood vessels in order to administer the drug intravenously.   Oral ganciclovir administration for 100 days after liver transplantation reduced the cmv disease rate to 5% among all patients, and to 15% among those at risk of developing the incidence of developing primary CMV (65). However, given the poor oral bioavailability of oral ganciclovir, one of the potential concerns in the mode of prolonged use of this agent is substandard suppression of the virus and the emergence of ganciclovir-resistant CMV. Patients at particular are those receiving severe immunosuppression and the R-/D+ group. In one institution that used prolonged prevention with oral ganciclovir, 10% of patients who underwent a solid organ transplant contracted CMV in the first year after transplantation (116). It is worth noting that 20% of patients with CMV disease had CMV resistance gansiflufer (116). In another report, 11 cases of gancicluvir-resistant CMV were documented in transplant recipients, the majority of which received long oral preventive treatment ganciclovir (90). Valganciclovir is ester fallin prodrug of ganciclovir. After ingestion, the vast majority of valganciclovir, before reaching the generalization of the system, is decomposed to ganciclovir (150). The oral bioavailability of valganciclovir is that the levels of ganciclovir are achievable similar to those reached with venous ganciclovir (150). In a randomized trial valganciclovir compared to 900 mg once a day with oral ganciclovir 1000 mg tid administered for 100 days as prevention in CMV recipient negative/donor positive organ transplantation recipients, ie a group experienced CMV disease during the treatment phase (147). However, the incidence of CMV in oral groups of valganciclovir and ganciclovir was 17.2% and 18.4%, respectively (147). Approaches to antiviral prevention options also include valganciclovir prevention (900 mg qd), ganciclovir oral (1 g ted), or venous sexualclovir (5 mg/kg/d). Penetration of CMV disease is rare while using any of these factors. However, CMV may occur late in the disease in 2.5-17% of patients receiving oral gancyclovir, and 5.5-18% of those receiving valganciclovir (1, 10, 96, 115, 147, 177). Valasiclofer (8 g/d) is an alternative in kidney transplant recipients, particularly in R+ However it should be noted that 57% of all EPISODES of CMV disease in patients receiving valasiclofer were also late in occurrence. Based on the high incidence of invasive tissue diseases with valganciclovir compared with oral ganciclovir in liver transplant recipients in the PV-16000 trial, the FDA has warned against the use of valganciclovir in liver transplant recipients. In clinical preparation, however, valganciclovir was used in liver transplant recipients (10, 96, 114, 178). Valganciclovir and venous ganciclovir are rational antiviral prevention options in lung transplant patients. Lung transplant recipients are at high risk of developing CMV ganciclovir resistance (113). The resistant virus has been documented in 1-4.4% of R+, and 9-27% of R-/D+ lung transplant recipients (113). Given that most of these cases have occurred after prolonged exposure to oral ganciclovir, this factor is a less desirable option, for CMV prevention in lung transplant beneficiaries, particularly in the R-/D + subgroup. The usual duration of antiviral prevention is 90-100 days after transplantation. It has been suggested that in a lung transplant 180 compared to 100 days may be a more ideal duration of prevention (231). The release from infection and disease of CMV was greater in patients receiving 180 days (90%) of patients receiving 180 days (90%) of patients receiving 180 days (90%) compared to those receiving 100-170 days (64%) Or < 100 days (59%) Prevention (231). The success of preventive treatment depends on the early and reliable detection of CMV viremia. Studies in organ transplant recipients have used either CMV or DNA-based CMV molecular measures to initiate preventive treatment. Table 5 provides a forecasting value of various diagnostic tests for early detection of CMV disease. CMV antigenemia is easy to do, has a fast turn around time and can be a quantity. Although CMV may precede detectable antigens, most studies in transplant recipients have validated the antigen as a reliable tool for initiating preventive treatment (178,213). Possible disadvantages of antigen testing include the need for immediate sample processing and leukocyte ≥ requirements of 200 cells/mm. Dna-based measurements avoid the need for immediate sample treatment. The detection of CMV-DNA by PCR in leukocytes may be overly sensitive and does not reliably distinguish the underlying CMV infection from the recurrence of cmv infection. The detection of CMV DNA by PCR of plasma, whole blood-clipper hybrid assay or CMV RNA (RNA) is considered to be a determinant of virus recurrence (72). The quantitative PCR system that predicts CMV disease and can be reliably used in a preventive strategy has not been defined. In one report, the number of pcr copies in the range of 2000-5000 copies/ml was considered to be the optimal for cutting CMV disease with a positive and negative predictive value of 50% and 96.6%, respectively (88). At present, preventive treatment for transplant recipients may be based on blood antigen test results or pcr quantity, depending on access to the test. The basic principle of preventive treatment approach and the intent of preventive treatment is to prevent the development of vermia symptoms that can be detected by a sensitive examination, for CMV disease. Thus, the CMV infection (Vermia) has already occurred before the onset of antiviral therapy. Ideally, therefore, the antiviral agent used must be able to completely suppress the replication virus. Acyclovir or its prodrug valacyclovir, due to their poor activity against CMV are largely unsuitable agents for preventive treatment. The use of venous ganciclovir, while effective as a preventive treatment, is impractical and uncomfortable due to the requirement of intravenous access. Oral ganciclovir administered preventively at ≥3 g/day doses has also been shown to be effective in reducing the risk of DEVELOPING CMV disease (149, 178, 209). In recipients of liver transplantation, preventive oral ganciclovir was associated with a significantly lower risk of CMV disease (RR 0.11, 95% CI, 0.01 -0.96, p = 0.04) compared to the control group that did not receive prevention (209). Gancyclofer oral for 6 weeks had begun to detect anti-CMV also effective in preventing CMV disease in liver transplant recipients in another report (178). However, oral gansifluvir may not be an ideal antiviral agent for preventive treatment. Due to poor bioavailability and low achievable serum levels, the ganciclovir mouth may not only be suboptimal as a preventive treatment in high viral load mode, but may be inclined to the appearance of ganciclovir resistance. The dynamics of CMV transcription in liver transplant recipients have shown that viral growth in the experienced HOST CMV will be largely inhibited by oral ganciclovir at a dose of 1 gram of tid orally (57). In cmv naivety host however, which must have an anti-CMV factor ≥ 93% in inhibiting viral recurrence, oral gansilufer may not be optimal (57). During preventive treatment with oral ganciclovir in liver transplant recipients the maximum viral load was achieved less, but did not differ significantly compared to placebo (average, 734 vs. 2603 copies /106 peripheral blood cells, respectively, p = .19) (159). Two of the 6 patients, both R-/D+, with a continuous viral recurrence on oral ganciclovir later developed CMV syndrome (159). Although the FDA has warned against the use of valganciclovir in liver transplant recipients, this agent has been shown to be effective as a preventive treatment in organ transplant recipients, including those undergoing liver transplantation. Of the 59 recipients of a liver transplant, including R-/D+ patients (17/59) valganciclovir received as a preventive treatment for INFECTION CMV (antigen) (178). On average follow-up 3.1 years, there were no episodes of CMV disease. Another report in solid organ transplant recipients compared the effectiveness of preventive treatment with valganciclovir used as a preventive treatment with venous ganciclovir (124). There were no bouts of CMV or CMV disease late in either team in a 12-month follow-up (124). Of the 301 at-risk serum transplant recipients who received preventive treatment with valganciclovir for CMV antigenemia, none had CMV when followed for an average of 14 months (42). The goal should be to reach an undetectable level of veremia by immunogeny or PCR, whichever test prompted the initiation of preventive treatment. The duration of preventive treatment usually ranged from 14-21 days. Antigen level with preventive valganciclovir decreased by 99.5% in 2 weeks compared to baseline (178). In another study, it was a good time to become a negative PCR with a preventive valganciclovir of 15.5 days, and the basic CMV viral load being 3.8 log10 gnomes /ml (124). Transplant recipients receiving antibodies are monoclonal or polyclonal antibodies at risk of CONTRACTING CMV infection. These patients should receive a powerful antiviral agent such as venous ganciclovir or valganciclovir started in conjunction with the antilymphocytic agent and lasted for ~2 weeks. This type of cmv prevention caused by a clinical event is what is called targeted or selective prevention. Prevention in HIV patients the most common and debilitating form of CMV disease is CMV retinitis, which may eventually lead to blindness despite effective antiviral therapy if antiretroviral therapy is unable to raise the number of CD4+ lymphocytes above 100-150 cells/μL. In pre-HAART, in a placebo-controlled study, Spector et al randomized (at a ratio of 2:1) 486 patients who received 1000 mg of oral gancyclofer three times a day and 239 controls (120, 199). The entry criteria were CD4+ 50/μL or less, 100/μL or less, and the date of the AIDS identification event. The incidence of CMV retin infection after 18 months of observation was 39% in the placebo group and 18% in the treated group. The total CMV disease decreased significantly through treatment (39% vs. 20%), including colitis reported by the investigator (13% vs. 4%). Although there is a trend towards benefiting from overall survival for patients treated with oral ganciclovir (relative risk = 0.8), the difference was not statistically significant (P = 0.14). Patients in the treatment group had more neutrophils, were more frequent on the granulocyte stimulating factor, and had more frequent elevations of serum creatinine. In a second study conducted by CPCRA, oral prevention ganciclovir in people with AIDS failed to prove useful in CMV disease (23). However, several important differences were found between the two studies, the most important of which was the monitoring of otitis in both studies. While the previous study by Spector and his colleagues examined baseline participants every two months with an extensive eye examination conducted by an experienced ophthalmologist, the second study did not perform dilated eye examinations at their clinical location unless the study participant complained of visual disorders. Although there was a trend in favor of ganciclovir observed in the CPCRA study, the difference was not statistically significant. Interestingly, however, a trend towards improved survival in the gancycloiver treatment group (relative risk = 0.84; P = 0.1) was also observed in this additional study. Spector et al. studied the effect of viral load as determined by CMV DNA PCR of plasma in 1654 study participants. The development of CMV disease was directly associated with the amount of CMV DNA present in the study entry. But more importantly, for participants in the randomized study to receive ganciclovir, those who moved from positive PCR plasma to negative developed significantly less than CMV disease than those who remained positive PCR (20% vs. 48%, respectively; relative risk 0.33, P< 0.001). It should be noted that survival in general was also expected through the presence and quantity of CMV DNA in plasma. Furthermore, participants in the randomized study to receive ganciclovir who moved from CMV positive DNA to negative significantly improved survival compared to those who remained pcr positive (24% vs. 47%, relative risk, 0.36, P< 0.001). In addition, a newer study conducted in the HAART era failed to show the benefit of valganciclovir when given as a preventive treatment due to the low rate of organ diseases in HIV-infected patients despite a decrease in the number of cells CD4+ and CMV viremia (223). In total, in the current recommendation of the American Department of Public Health/American Infectious Diseases Association guidelines for the prevention of opportunistic infection in people living with HIV do not recommend valganciclovir prevention in HIV-infected patients regardless of cd4+ number, CMV viremia or antiretroviral therapy. The guidelines recommend that the main way to prevent severe diseases is to recognize the early clinical symptoms of CMV with appropriate treatment. Infection control there are no special precautions necessary for patients in hospitals known to be infected with CMV activity. Patients do not need isolation or one room. It is important, however, that individuals adhere to strict hand washing after exposure to body secretions including saliva, urine and tears because infectious CMV can be present such fluids. In addition, seroimmune patients and non-infected preterm infants should be given CMV seronegative And blood products to prevent the transmission of infection from the donor to the recipient. Comprehensive precautions are recommended in dealing with blood, blood products and secretions in all patient care settings and sufficient to control CMV. There are still a number of important differences on the optimal approach to prevention and treatment of a wide range of CMV diseases observed in various sick populations. To a large extent, the different clinical conditions associated with CMV diseases have held a treatment approach. Generally, the underlying risk factor for serious CMV infection is immunosuppression, especially from cell-mediated immunity. However, it is clear that AIDS-related immunosuppression versus bone marrow transplantation versus solid organ transplantation varies greatly and appears to have a significant impact on the cause of CMV diseases in these environments. In CMV serological patients with organ transplant recipients either receiving serum transplantation or previously cmv positive, the use of preventive treatment versus comprehensive prevention remains controversial (179). Issues include effectiveness, logistics, duration, cost, relative risk of disease in a particular population, and the adequacy of the impact of treatment on decision-making. In the era of HAART, patients who have started effective antiretroviral therapy usually do not require preventive treatment. If preventive treatment is the chosen approach, the selection of the examination tool becomes important. Most studies have found that antigenemia or PCR tests are comparable to screening patients. Experience in a particular position is important in determining which of the many measures available is best for a particular center. In addition, the continuation of preventive/preventive treatment remains controversial. For people with AIDS, if preventive treatment begins it must continue until the patient is receiving HAART on his CD4 + lymphatic count above 100 cells/μL. When to stop the prevention of transplant recipients is more problematic. The cessation of prevention/preventive treatment after 100-120 days led to the late emergence of CMV. In implant settings the best continuation of preventive treatment will be determined by the clinical condition of the particular patient and the amount of immunosuppression required. In patients, who are still on high immunosuppression, the risk of CMV remains high and preventive treatment should probably continue. In contrast, patients who follow a transplant who are at minimal immunosuppression and who do not suffer from GVHD can likely have CMV preventive treatment discontinued. Monitoring patients to reactivate CMV and increase the risk of developing the disease is initially justified on a monthly basis when preventive treatment is discontinued. Prevention of congenital infections An important focus of CMV research. With no vaccine, a number of interventions have been evaluated to identify the congenitally infected fetus in the uterus and prevent infection-related sequels. From these strategies, the use of CMV hyperimmunoglobulin (HIG) has been widely studied and most controversial. To date, three studies suggest that HIG management may reduce the risk of developing congenital CMV (138, 139, 214). However, there is no standard dose nor duration of administration during pregnancy although most studies gave monthly doses of HIG until birth. While using HIG is expensive, proponents argue that lifetime care from CMV symptoms of congenitally infected infants far exceeds the cost. Unfortunately, there are no randomized controlled studies with long-term follow-up of infants exposed in utero to HIG. It is difficult to conduct this study because of the number of women to be examined during this study, the cost of this type of study, and the expense of long-term follow-up; however, such a study will need to be conducted in the United States before HIG is widely recommended for use during pregnancy.

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