31. MARBURG AND EBOLA VIRUSES, G. LLOYD pdf

1: JDR Vol.6 p () | Fuji Technology Press: academic journal publisher

Marburg viruses are the agents of Marburg haemorrhagic fever. It was initially detected in after outbreaks in Marburg, Frankfurt and Belgrade. The Ebola and Marburg viruses are the two members of the Filoviridae family, which cause outbreaks with high fatality rates.

Outbreak of Marburg hemorrhagic fever among miners in Kamwenge and Ibanda districts, Uganda, Journal of Infectious Diseases. Seasonal pulses of Marburg virus circulation in juvenile Rousettus aegyptiacus bats coincide with periods of increased risk of human iInfection. Development of vaccines for Marburg hemorrhagic fever. Expert Review of Vaccines. Marburg hemorrhagic fever associated with multiple genetic lineages of virus. New England Journal of Medicine. Treatment of Marburg and Ebola hemorrhagic fevers: A strategy for testing new drugs and vaccines under outbreak conditions. Forty-five years of Marburg virus research. Centers for Disease Control and Prevention. Imported case of Marburg hemorrhagic fever â€" Colorado, Morbidity and Mortality Weekly Report. Haemorrhagic fevers of Africa: Journal of the South African Veterinary Association. Prospects for immunisation against Marburg and Ebola viruses. Reviews in Medical Virology. Pathogenesis of Marburg hemorrhagic fever in cynomolgus macaques. Characterization of a new Marburg virus isolated from a fatal case in Kenya. Basic clinical and laboratory features of filoviral hemorrhagic fever. Ebola and Marburg Hemorrhagic Fevers: A hitherto unknown infectious disease contracted from monkeys. Clinical aspects of Marburg hemorrhagic fever. Future Virology ;6 9: Manual of Clinical Microbiology. Interferon-beta therapy prolongs survival in rhesus macaque models of Ebola and Marburg hemorrhagic fever. Studies of reservoir hosts for Marburg virus. A probable case of Ebola virus haemorrhagic fever in Kenya. East African Medical Journal. Isolation of genetically diverse Marburg viruses from Egyptian fruit bats. Marburgvirus genomics and association with a large hemorrhagic fever outbreak in Angola. Advanced antisense therapies for postexposure protection against lethal filovirus infections.

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2: Resources | Marburg Hemorrhagic Fever (Marburg HF) | CDC

Abstract. Clinical specimens from patients infected with Lassa, Ebola, or Marburg virus may present a serious biohazard to laboratory workers. We have examined the effects of heat, alteration of pH, and gamma radiation on these viruses in human blood and on the electrolytes, enzymes, and coagulation factors measured in laboratory tests that are important in the care of an infected patient.

False color scanning electron microscope image of a single filamentous Ebola virus particle Phylogenetic tree comparing ebolaviruses and marburgviruses. Numbers indicate percent confidence of branches. The outer viral envelope of the virion is derived by budding from domains of host cell membrane into which the GP spikes have been inserted during their biosynthesis. Viral proteins VP40 and VP24 are located between the envelope and the nucleocapsid see following , in the matrix space. This viral genome codes for seven structural proteins and one non-structural protein. Sections of the NP, VP35 and the L genes from filoviruses have been identified as endogenous in the genomes of several groups of small mammals. Using third-generation sequencing technology, investigators were able to sequence samples as quickly as 48 hours. The first is a cholesterol transporter protein, the host-encoded Niemannâ€"Pick C1 NPC1 , which appears to be essential for entry of Ebola virions into the host cell and for its ultimate replication. Silencing its effect with siRNA prevented infection of Vero cells. Together, these studies suggest NPC1 and TIM-1 may be potential therapeutic targets for an Ebola anti-viral drug and as a basis for a rapid field diagnostic assay. These then self-assemble into viral macromolecular structures in the host cell. The virus begins its attack by attaching to host receptors through the glycoprotein GP surface peplomer and is endocytosed into macropinosomes in the host cell. These two molecules assemble, first into heterodimers, and then into trimers to give the surface peplomers. Secreted glycoprotein sGP precursor is cleaved to sGP and delta peptide, both of which are released from the cell. As viral protein levels rise, a switch occurs from translation to replication. Intermediary hosts have been reported to be "various species of fruit bats Evidence of infection in bats has been detected through molecular and serologic means. However, ebolaviruses have not been isolated in bats. Pigs in the Philippines have been reported to be infected with Reston virus , so other interim or amplifying hosts may exist. Ebola virus disease Ebola virus is one of the four ebolaviruses known to cause disease in humans. It has the highest case-fatality rate of these ebolaviruses, averaging 83 percent since the first outbreaks in , although fatality rates up to 90 percent have been recorded in one outbreak â€" There have also been more outbreaks of Ebola virus than of any other ebolavirus. The first outbreak occurred on 26 August in Yambuku. The symptoms resembled malaria , and subsequent patients received quinine. Transmission has been attributed to reuse of unsterilized needles and close personal contact, body fluids and places where the person has touched. During the Ebola outbreak in Zaire , Ngoy Mushola travelled from Bumba to Yambuku , where he recorded the first clinical description of the disease in his daily log: Since the first recorded clinical description of the disease during in Zaire, the recent Ebola outbreak that started in March , in addition, reached epidemic proportions and has killed more than people as of January This outbreak was centered in West Africa, an area that had not previously been affected by the disease. The toll was particularly grave in three countries: Guinea, Liberia, and Sierra Leone. A few cases were also reported in countries outside of West Africa, all related to international travelers who were exposed in the most affected regions and later showed symptoms of Ebola fever after reaching their destinations. Hence the variability in the severity of illness was suspected to correlate with genetic differences in the victims. This has been difficult to study in animal models that respond to the virus with hemorrhagic fever in a similar manner as humans, because typical mouse models do not so respond, and the required large numbers of appropriate test subjects are not easily available. In late October , a publication reported a study of the response to a mouse-adapted strain of Zaire ebolavirus presented by a genetically diverse population of mice that was bred to have a range of responses to the virus that includes fatality from hemorrhagic fever. The name "Ebola virus" is derived from the Ebola River â€"a river that was at first thought to be in close proximity to the area in Democratic Republic of Congo , previously called Zaire , where the Zaire Ebola virus outbreak occurredâ€"and the taxonomic suffix virus. Consequently, in , a group of

researchers recommended that the name "Ebola virus" be adopted for a subclassification within the species Zaire ebolavirus, with the corresponding abbreviation EBOV. In , the ICTV explicitly rejected a proposal A Terrifying True Story: A best-selling book by Richard Preston about Ebola virus and related viruses including an account of the outbreak of an Ebolavirus in primates housed in a quarantine facility in Reston, Virginia, USA.

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3: Marburg virus - Wikipedia

Bowen ET, Lloyd G, Harris WJ, Platt GS, Baskerville A, Vella EE. Viral haemorrhagic fever in southern Sudan and northern Zaire. Preliminary studies on the aetiological agent.

Watery diarrhoea occurs most frequently. In case of recovery symptoms improve over a period of several weeks. Reconvalescence requires up to several months. In more severe cases hemorrhagic disorders occur. Most remarkable are hemorrhages from the gastro-intestinal tract connected with poor prognosis. Other symptoms are mental disturbances, hyperaesthesia, and myelitis. Considerable bradycardia may be one of the symptoms in the beginning except in fatal cases during agony, where tachycardia is prevailing. Thus, the mortality is comparable with the mortality from Lassa fever. Leukopenia followed by leukocytosis is an almost typical feature, usable for diagnosis. Differential blood count demonstrates shift to the left of the granulocytes, as well as pseudo-Pelger cells, and atypical lymphocytes with activated nuclei, plasma cells or lymphoblasts 7. Considerable thrombocytopenia may cause bleeding tendency. In some cases laboratory parameters may suggest dissiminated intravascular coagulation with subsequent kidney failure 8. ECG changes are comparable with myocarditis or other damage of the myocardium. An interesting fact is that the association with active virus material can be observed for a long time. In one case, who recovered, virus particles could be detected in sperma. In this case there was evidence that the patient infected his wife by sexual intercourse. A secondary case showed that even two months after the disease was observed, virus particles could be isolated on the eye-chamber fluid 2 PATHOLOGY Postmortem sections showed that almost in all organs of corpses with Marburg virus focal necrosis could be detected by routine histology. Damage of the parenchyma of the kidneys suggested tubular deficiency. Hemorrhagic diathesis and plasma cellular infiltration could be observed in various tissues. Cerebral damage as described in panencephalitis with glial nodules may be present. Furthermore, morphology of peripheral blood smears and other hematological parameters including platelet count may assist the diagnosis. Direct examination of blood or organ biopsies can be done by electron microscopy. Cytopathic effect in Vero cell culture or the detection of virus from guineapigs inoculated with blood from a diseased patient will give evidence of the diagnosis. This includes balance of fluids and electrolytes, as well as treatment of hemorrhagic disorder which, of course, must be defined by laboratory parameters exactly. It is questionable whether convalescent plasma will prevent severe disease or, in any way, improve the status of the patients. The use of interferon seems to be experimental at the time being. However, in case of a suspected case of Marburg virus disease, preventive measures have to be taken in order to protect attending personnel from infection. It seems that routine isolation procedures may be sufficient. However, the occurrence of secondary cases in the Marburg virus infection in Johannesburg in teaches us that secondary infection may occur despite these measures. More strict isolation facilities have been used for reverse isolation of patients with susceptibility to infection, e. Plastic isolation systems have been used Therefore, also in isolation of cases with hemorrhagic fever the use of plastic isolation systems with negative pressure and completely tied plastic bag should be safer, and will avoid any quarantine procedures for attending hospital staff". Materials from the patients have to be processed with specific care! Therefore, any laboratory investigations including the definite diagnosis has to be done under strict regulations of safety. Consequently, only few institutions all over the world can deal with such cases under optimal conditions. Despite the fact that the first outbreak was caused by contact with monkeys, the natural reservoir is still unknown. Transmission may occur by contacts with infected patients. Animal experiments showed that even Aedes aegypti could be a transmitter. Important to know that association with the virus or virus particle may last for several months. Treatment is supportive only, because specific treatment is not known. Special attention has to be given to strict isolation procedures as necessary in patient care and for diagnostic laboratory investigations. Kurstak, New York, Academic Press. The Johannesburg-Marburg index case is of course the only Marburg case known that has been acquired in nature. The patient complained of some kind of sting or bite that he acquired one week prior to onset of illness. This was a very painful lesion. We did a rather thorough epidemiological investigation, and we in fact were able to identify the tree on the roadside under

which the patient sat when he was stung or bitten by an unidentified agent. We proceeded to tear the roadside bank apart literally. The only sign of life we found from the entomological point of view was hundreds of spiders. We collected all the spiders we could find and took these to the laboratory. About half of these have now been investigated at the CDC in Atlanta as well as in our Laboratory in Johannesburg and have yielded no results. I think that these are the only comments I want to make on this particular case. Does the fact that we heard nothing about changes in the blood vessels mean that there is no vasculitis present? Do you have any clue from findings in the gastro-intestinal tract why there is diarrhoea and intestinal bleeding? Around the vessels there were, as far as I remember, some cellular infiltrates, no pure vasculitis as such. The same holds true for the GI tract.

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4: Filoviridae | Viral Hemorrhagic Fevers (VHFs) | CDC

Abstract. Ebola and Marburg viruses, members of the filovirus family, cause severe hemorrhagic fever in human and nonhuman primates and are classified as biosafety level 4 agents.

The IgM capture assay detected anti-EBO subtype Reston antibodies in the sera of 5 of 5 experimentally infected animals at the time they succumbed to lethal infections. IgM antibodies were also detected in the serum of a human who was infected with EBO subtype Reston during a postmortem examination of an infected monkey. Although these data are limited by the number of sera available for verification, the IgM assay seems to have great promise as a diagnostic tool. The ecology and epidemiology of Ebola EBO virus subtypes have been the topic of considerable study since the occurrence of high-mortality EBO epidemics [1â€”3]. Such studies have been hampered by the lack of effective serologic tools with which to survey for individuals, populations, or other vertebrate hosts with specific serologic evidence of past infection. It has shown good sensitivity for antibodies during early convalescence and has been used extensively in serologic surveys for EBO virus antibody prevalence [6â€”14]; however, it has been criticized for a perceived lack of specificity in populations with no apparent probability of infection with the African filoviruses [15] and because of an inability to confirm its results with those of other immunologic techniques [16]. Other serologic tests for EBO viral antibody have been described: ELISA and RIA are procedures that have been used commonly for the screening of antibodies to other viral diseases; however, they have not been commonly applied in filovirus research, and their practical value remains largely unevaluated. The RIPA and the Western blot measure antibody responses to individual viral proteins in slightly different ways and offer the advantage of showing the molecular specificity of the response; however, both techniques are most commonly used as confirmatory or research tools to dissect the immune responseâ€”the tests are too cumbersome to be used as primary serologic tools in studies of an epidemiologic scale. The duration of antibodies measured by serologic tests used for epidemiologic studies is a central question in their use: How rapidly do antibodies become detectable and how long do they remain measurable? The rapid decline of IFAT antibodies in persons infected with EBO virus has been reported [2]; however, the use of the various tests including the IFAT on sera documented to be from genuine cases is limited, thus handicapping for epidemiologic purposes the interpretation of results from existing serologic tests for filoviruses. We also present data that support the sensitivity and longevity of these tests in a limited number of experimentally infected primates and humans who survived EBO infections. Materials and Methods Viral Antigens Viral antigens for the IgG test were made by detergent basic buffer extraction of infected tissue culture cells. In brief, MA cells in roller bottles were infected with EBO virus strains and allowed to proceed until advanced cytopathic effect was observed. Cells were washed once in 0. The cell pellet was resuspended with a vortex in borate saline, pH 9. Uninfected MA cells were similarly prepared and used as a control or comparison antigen. The virus pellet was resuspended in 0. Two of the animals were Macaca mulatta that had been infected with a virus seed inadvertently contaminated with simian hemorrhagic fever virus SHFV ; however, both animals were also clearly infected with EBO-R. Human sera were from an animal handler who had become infected during the â€” epizootic in a quarantine facility [20 , 21] or from persons who survived either laboratory [22] or field infections [23] with EBO-Z. After a final wash, the coverslips were mounted with glycerol-PBS Antigens and antibodies were adsorbed to wells after dilution in 0. Wash buffer was comprised of PBS with 0. Dilutions of antigens used to coat polyvinyl chloride microtiter plates were determined by checkerboard titration with reference sera. An uninfected MA cell culture antigen was also coated to plates and used to determine the specific binding of antibody to viral antigens. Antigen was removed from the wells by washing 3 times with PBS-T. Sera were diluted 1: ODs at nm were recorded on a microplate spectrophotometer, and the OD of the uninfected antigen-coated well was subtracted from its corresponding viral antigenâ€”coated well to yield the adjusted OD at nm. A panel of 5 to 6 normal sera are run each time the assay was used. This represents the cutoff value for the assay and is usually about an OD at nm of 0. Captured viral antigen was detected with a polyclonal hyperimmune, polyvalent anti-EBO mouse ascitic fluid prepared by immunization with the 3 EBO

virus strains. A negative CV7 cell antigen was used to adjust for the specific reaction of the reagents with captured viral antigen, and the adjusted OD for each dilution at nm was calculated as above. Optimal dilutions of all reagents were determined by checkerboard titration with early convalescent serum from 1 of the experimentally infected monkeys. Detectable levels of IgG antibody were reliably detectable at about days 10â€” Once the antibodies became detectable, the adjusted OD at the 1: As shown in figure 2 , the antibody levels to the homologous EBO-R strain appeared to be consistently higher during early convalescence. However, with time, the antibody levels became approximately equivalent for antigens of all 3 strains of EBO virus. There was no cross-reaction of the sera of these EBO virusâ€”infected monkeys with a similarly prepared antigen of Marburg virus. We also tested the sera of a number of humans who had been infected with strains of EBO virus isolated from the epidemic in the Democratic Republic of the Congo DRC. In both instances, the adjusted OD and titers were similar to those seen in the late convalescent sera of the experimental monkeys table 1. Also shown in table 1 are ELISA data that were collected for other individuals in association with the epidemic in DRC, but specific time intervals for the collections were not known. We assessed the specificity of the IgG assay by testing sera of primates imported under quarantine requirements and which had no evidence of active filoviral infection, as determined by IFAT of paired blood samples. We interpret these results as being a good indication of the specificity no false positives of the ELISA IgG test, not as a lack of sensitivity false negatives:

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5: Ebola Virus Haemorrhagic Fever

The filoviruses Ebola and Marburg are zoonotic agents that are classified as both biosafety level 4 and category A list pathogens. These viruses are pathogenic in humans and cause isolated infections or epidemics of viral hemorrhagic fever, mainly in Central Africa.

So far, three genera of this virus family have been identified: Cuevavirus, Marburgvirus and Ebolavirus. Six species of Ebolavirus have been identified: Reston virus is known to cause disease in nonhuman primates and pigs, but not in people. Bombali virus was recently identified in bats, and it is unknown at this time if it causes disease in either animals or people. Structurally, filovirus virions complete viral particles may appear in several shapes, a biological features called pleomorphism. Viral filaments may measure up to 14, nanometers in length, have a uniform diameter of 80 nanometers, and are enveloped in a lipid fatty membrane. Each virion contains one molecule of single-stranded, negative-sense RNA. Filovirus history The first Filovirus was recognized in when a number of laboratory workers in Germany and Yugoslavia, who were handling tissues from green monkeys, developed hemorrhagic fever. A total of 31 cases and 7 deaths were associated with these outbreaks. The virus was named after Marburg, Germany, the site of one of the outbreaks. In addition to the 31 reported cases, an additional primary case was retrospectively serologically diagnosed. After this initial outbreak, the virus disappeared. It did not reemerge until , when a traveler, most likely exposed in Zimbabwe, became ill in Johannesburg, South Africa. The virus was transmitted there to his traveling companion and a nurse. A few sporadic cases and 2 large epidemics Democratic Republic of Congo in and Angola in of Marburg hemorrhagic fever Margurg HF have been identified since that time. For information on known Marburg HF cases and outbreaks, please refer to the chronological list. Ebolavirus was first identified in when two outbreaks of Ebola hemorrhagic fever Ebola HF occurred in northern Zaire now the Democratic Republic of Congo and southern Sudan. The outbreaks involved what eventually proved to be two different species of Ebola virus; both were named after the nations in which they were discovered. Since , Ebolavirus have appeared sporadically in Africa, with small to midsize outbreaks confirmed between and For information on known Ebola HF cases and outbreaks, please refer to the chronological list. Animal hosts It appears that Filoviruses are zoonotic, that is, transmitted to humans from ongoing life cycles in animals other than humans. Despite numerous attempts to locate the natural reservoir or reservoirs of Ebolavirus and Marburgvirus species, their origins were undetermined until recently when Marburgvirus and Ebolavirus were detected in fruit bats in Africa. Marburgvirus has been isolated in several occasions from Rousettus bats in Uganda. Spreading Filovirus infections In an outbreak or isolated case among humans, just how the virus is transmitted from the natural reservoir to a human is unknown. Once a human is infected, however, person-to-person transmission is the means by which further infections occur. Specifically, transmission involves close personal contact between an infected individual or their body fluids, and another person. During recorded outbreaks of hemorrhagic fever caused by a Filovirus infection, persons who cared for fed, washed, medicated or worked very closely with infected individuals were especially at risk of becoming infected themselves. Nosocomial hospital transmission through contact with infected body fluids â€” via reuse of unsterilized syringes, needles, or other medical equipment contaminated with these fluids â€” has also been an important factor in the spread of disease. When close contact between uninfected and infected persons is minimized, the number of new Filovirus infections in humans usually declines. Although in the laboratory the viruses display some capability of infection through small-particle aerosols, airborne spread among humans has not been clearly demonstrated. During outbreaks, isolation of patients and use of protective clothing and disinfection procedures together called viral hemorrhagic fever isolation precautions or barrier nursing has been sufficient to interrupt further transmission of Marburgvirus or Ebolavirus, and thus to control and end the outbreak. Because there is no known effective treatment for the hemorrhagic fevers caused by Filoviruses, transmission prevention through application of viral hemorrhagic fever isolation precautions is currently the centerpiece of Filovirus control. The manual can help healthcare facilities recognize cases and prevent further hospital-based disease transmission using locally available materials and few financial resources. VHFs caused by Filoviruses.

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6: Marburg and Ebola viruses - Oxford Medicine

The virus pellet was resuspended in M PBS, pH , of about a th of the original volume from which it had been spun, gamma-irradiated with 5 Ã— 10 6 rad (50, Gy), and safety tested for residual virus infectivity.

Five species within the genus Ebolavirus have been identified: Marburg marburgvirus is the single species in its genus. Fruit bats Rousettus aegypticacus are the natural reservoirs for Marburg virus. Outbreaks occur when a person becomes infected after exposure to the reservoir species or a secondarily infected nonhuman primate or antelope species and then transmits the virus to other people in the community. In the community, Ebola virus and Marburg virus are generally transmitted by direct physical contact between unprotected skin or mucous membranes and blood or other infected body fluids of patients in the acute phase of Ebola virus disease EVD or Marburg virus disease MVD or from patients who have died from EVD or MVD. Aerosol transmission of Ebola virus in humans has not been documented. Ebola virus RNA has been detected in breast milk up to 21 days after the onset of the disease and in vaginal secretions up to 33 days after onset. Ebola virus and Marburg virus have been cultured from ocular aqueous humor at 2 and 3 months after disease onset, respectively. Evidence suggests that Ebola and Marbug viruses can be sexually transmitted from a male survivor to his female partner months after onset of disease. In pregnant women with EVD, there can be in utero transmission of Ebola virus to the fetus. EPIDEMIOLOGY People at greatest risk of EVD or MVD include family members, health care workers or others who come into direct contact with infected patients or corpses without protective equipment, people who have come into contact or close proximity to bats visiting bat caves , and those who have handled infected primates or carcasses. Additionally, sexual partners of recent male EVD or MVD survivors may be at risk if they have had contact with virus-infected semen. Typically, previous Ebola outbreaks had been limited in scope and geographic extent. However, in March of , an outbreak of Ebola virus was detected in a rural area of Guinea near the border with Liberia and Sierra Leone. By June of , cases were reported in all three countries and across many districts. The outbreak was the largest and most complex Ebola epidemic ever reported. Countries with confirmed human cases of Marburg hemorrhagic fever include Uganda, Kenya, Democratic Republic of the Congo, Angola, and possibly Zimbabwe. Four cases of Marburg hemorrhagic fever have occurred in travelers visiting caves harboring bats, including Kitum cave in Kenya and Python cave in Maramagambo Forest, Uganda. Miners in the Democratic Republic of the Congo and Uganda have also acquired Marburg virus infection from working in underground mines harboring bats. Reston virus is believed to be endemic in the Philippines but has not been shown to cause human disease. Signs and symptoms of EVD and MVD can vary but, in general, patients present with an abrupt-onset fever, weakness, myalgias, arthralgias, and headache. This is often followed by gastrointestinal symptoms including anorexia, abdominal discomfort, nausea, vomiting, and diarrhea. Conjunctival injection, rash, and hiccups have also been reported. Intravascular volume depletion is common and may be associated with profound electrolyte depletion, hypo-profusion, and shock. Coagulopathy is a late manifestation and can present with a petechial rash, ecchymoses, and sometimes overt bleeding epistaxis, melena, bloody diarrhea. Hypoxia, another late manifestation, was noted in half of EVD patients median oxygen saturation Laboratory abnormalities include elevations in liver enzymes, initial drop in leukocyte count, and thrombocytopenia. Because the incubation period may be as long as 21 days, patients may not develop illness until returning from travel; therefore, a thorough travel and exposure history is critical. Pregnant women with EVD appear to be at high risk of spontaneous abortions, stillbirth, and pregnancy-related hemorrhage. Limited evidence suggests that the prognosis for neonates born to mothers with EVD is poor, and most die within 19 days of birth. Personal protective equipment is indicated for any patients where EVD or MVD is suspected and includes droplet and contact precautions. Postmortem skin biopsies fixed in formalin and blood collected within a few hours after death by cardiac puncture can be used for diagnosis. Diagnostic testing of blood and other body fluid specimens calls for special handling procedures. The mainstay of treatment is early aggressive supportive care directed at maintaining effective intravascular volume and correcting electrolyte imbalances. Several additional experimental immune therapy

treatments and antivirals are currently under investigation. EVD patients may also have concomitant malaria infection. As such, empiric use of antimalarial therapy should be considered when rapid diagnostic testing is not immediately available. In general, NSAIDs such as ibuprofen and diclofenac are not recommended due to their platelet activity. Experimental Ebola vaccines are under development, including a recombinant vesicular stomatitis virusâ€"based vaccine and a chimpanzee adenovirusâ€"based vaccine. However, these investigational products are in the early stages of product development and are not yet available. Clinical presentation of patients with Ebola virus disease in Conakry, Guinea. N Engl J Med. A monovalent chimpanzee adenovirus Ebola vaccine boosted with MVA. Caring for critically ill patients with Ebola virus disease. Perspectives from West Africa. Transmission of Ebola viruses: A Recombinant vesicular stomatitis virus Ebola vaccineâ€"preliminary report. Clinical management of Ebola virus disease in the United States and Europe. Clinical care for survivors of Ebola virus disease.

7: Ebola virus and Marburg virus - Symptoms and causes - Mayo Clinic

Ebola virus and Marburg virus are related viruses that cause hemorrhagic fevers â€" illnesses marked by severe bleeding (hemorrhage), organ failure and, in many cases, death. Both viruses are native to Africa, where sporadic outbreaks have occurred for decades.

8: Ebola virus - Wikipedia

The development of Marburg virus and the Sudanese and Zaire strains of Ebola virus in Vero cells as visualized by electron microscopy is described. Despite differences in timing, all three strains appear to pass through identical stages of development.

9: Ebola virus transmission. - Europe PMC Article - Europe PMC

A year-old hospital technician died of the Marburg virus this weekend in Uganda, health officials there announced www.enganchecubano.comg, like Ebola, is a hemorrhagic fever. It's rare but severe.

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