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1. Introduction

The Ebola and Marburg viruses are distinct filoviruses that share common clinical presentations and clinical management protocols. However, the Marburg virus is not as well-known as its relative, the Ebola virus. The largest Ebola epidemic in history occurred in West Africa from 2013 to 2015 in which 28,616 persons were reported to be infected. Following a small outbreak of 54 probable and confirmed cases in the Équateur Province of the Democratic Republic of the Congo (DRC) from May to June 2018, a second and larger epidemic has occurred in the Kivu and North Ituri Provinces since August 2018 [1]. This epidemic has infected 2592 persons as of July 2019 and is the second largest Ebola epidemic in history. In contrast to the Ebola virus, Marburg virus disease (MVD) occurs much less frequently. MVD is caused by two marburgviruses that are clinically indistinguishable—the Marburg virus and Ravn virus. There is scant information available concerning MVD in pregnancy, but it appears clear that, similar to Ebola virus, MVD infection is associated with an extremely high maternal and fetal mortality rate. This chapter will examine what is known about Marburg and Ravn virus infections in pregnant women, their clinical outcomes, and the pathogenesis of MVD in experimental animal models of infection. These data will be compared with the more comprehensive information available regarding Ebola virus disease in pregnancy including its effects on pregnant women and the fetus.

Keywords: Ebola virus, Marburg virus, Ravn virus, pregnancy, maternal death, fetal death, filovirus, maternal infection, hemorrhagic fever, maternal mortality, maternal morbidity, fetal mortality, West African Ebola epidemic, epidemiology
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The marburgviruses are single-stranded RNA viruses belonging to the family Filoviridae, which also includes the Ebolavirus genus. The genus Marburgvirus comprises a single species, Marburg marburgvirus, that includes two variants - the Marburg virus (MARV) and the Ravn virus (RAVV) [3, 4]. Similar to other members of the family Filoviridae, marburgvirions have a filamentous configuration that appears by electron microscopy to resemble a shepherd’s crook, or in the shape of a “U” or a “6”; coiled, toroid, or branched forms can be seen (Figure 1). The marburgviruses cause a severe viral illness in humans termed Marburg virus disease, or MVD (formerly termed Marburg hemorrhagic fever). Illness due to the marburgviruses is clinically indistinguishable from Ebola virus disease (EVD). Although Marburg virus disease and Ebola virus disease have historically been labeled as hemorrhagic fevers, hemorrhage is found in less than 50% of patients [5]. According to some authors, they may be considered as gastrointestinal diseases that develop severe systemic organ involvement including hemorrhages [6]. After an incubation period that varies between 4 and 10 days, infected individuals abruptly develop flu-like symptoms characterized by fever, chills, malaise, and myalgia. This is followed by signs and symptoms indicating systemic involvement, including prostration and gastrointestinal symptoms (anorexia, nausea, vomiting, abdominal pain, and diarrhea), respiratory complaints (chest pain, shortness of breath, and cough), vascular findings (conjunctival injection, postural hypotension, and edema), and neurological symptoms (headache, confusion, and coma). The typical hemorrhagic manifestations of Marburg virus disease include petechiae and ecchymoses, and, less commonly, frank hematemesis, hematochezia, melena, or hemoptysis. The mortality rate is high, with most deaths occurring within the first week of illness. The diagnosis of MVD is difficult, as symptoms are nonspecific and similar to those of EVD. Laboratory confirmation is necessary for early recognition and prompt treatment. The treatment of MVD is supportive, with emphasis on maintaining fluid and electrolyte balance and preventing secondary infections. The use of antiviral agents is controversial, but some case reports have suggested a potential for benefit. The prevention of MVD involves practicing basic infection control measures, such as hand hygiene and the use of personal protective equipment, to prevent the spread of the virus. There is no known prophylactic vaccine for MVD, but efforts are underway to develop one. The control of MVD outbreaks requires rapid recognition, isolation of infected individuals, and rigorous infection control measures to prevent further transmission. The marburgviruses are a significant public health threat, and ongoing research is needed to better understand their biology, pathogenesis, and transmission.
The marburgviruses are transmitted from person-to-person through direct and unprotected contact with blood, body fluids, and tissues of infected persons. Risk factors for acquiring secondary MVD include close contact with severely ill patients or their body fluids in the acute phase of the disease, either at home or in a hospital, thus placing caregivers at risk for acquiring the infection. In addition, unsafe burial practices are common routes of infection. These are the identical mechanisms for transmission for Ebola virus. MVD has not been reported to be transmitted via the aerosol route. Women who are pregnant and infected with either marburgviruses or Ebola virus can be especially infectious—the placenta has a high viral load, and maternal blood, vaginal secretions, amniotic fluid, urine, sweat, saliva, feces, vomit, and breast milk are all potential sources of virus [11]. Products of conception as occur in miscarriages can also be infectious, as can fetal tissues.

Based upon the history of primary MVD infection occurring in association with exposure to bat-infested caves and mines and additional environmental and occupational risk factors for acquiring the infection, it appears unlikely that Marburg virus infection of a pregnant woman would occur as the index case of an outbreak. Thus, unlike the situation of some viral infections (notably hepatitis E) where infections in pregnant women can represent the index cases of a community-wide outbreak [12], MVD occurring in pregnant women would likely represent secondary infections within the community, prompting an epidemiological investigation to identify the index case(s). During the West Africa Ebola epidemic, pregnant women often became infected through the traditional female roles of caregiver to the sick as well as through preparation of the dead and via unsafe burials [13]. The high rate of transmissibility of filoviral infections was demonstrated in one Liberian village named Joe Blow Town. There, all of the mothers in the town became infected and died after acquiring EVD after caring for a woman who was infected and, following her death, preparing her body and then bathing in the water that had been used to wash her corpse [14].

Following acute infection, both marburgviruses and Ebola virus can persist in a variety of bodily fluids. Ebola virus and Marburg virus have both been found by culture from ocular aqueous humor 2 and 3 months after disease onset, respectively. Ebola virus RNA has been identified in breast milk for up to 21 days after the onset of the disease and in vaginal secretions up to 33 days after its onset. In one report, a 9-month-old infant is believed to have acquired Ebola virus infection through breastfeeding from a mother who did not report having a febrile illness—persistent Ebola virus RNA was identified in both the mother’s breast milk and in the father’s seminal fluid [15]. In men, Ebola virus has been identified in the semen of survivors for many months after acute infection, with some having persistence of Ebola RNA for up to 18 months [16]. It is likely that the marburgviruses would also demonstrate persistence in seminal fluid of male survivors. Sexual transmission of marburgviruses was reported in 1968 after the initial outbreak of Marburg virus disease [17]. In a study in crab-eating macaques, it was found by Coffin et al. [18] that experimentally infected males had persistent MARV infection of the seminiferous tubules, an immunologically privileged site. Affecting primarily the Sertoli cells, this viral persistence resulted in severe testicular damage including spermatogenic cell depletion, inflammation, and breakdown of the blood-testis barrier [18].

4. Outbreaks of Marburg virus disease

Marburg virus disease was initially discovered in 1967 when 31 persons unexplainably became ill in the cities of Marburg and Frankfurt am Main in Germany and Belgrade in the former Yugoslavia. The illness was traced to exposure to tissues or cell cultures obtained from a group of imported African green monkeys (grivets).
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In February of 1975, the first outbreak of MVD to occur in Africa was recognized in a young Australian man who acquired the infection while traveling in Rhodesia (now Zimbabwe). He died in a Johannesburg hospital on the 7th day of infection. Two secondary cases developed—a traveling companion and a nurse—and both survived [21].

In 1980 a French electrical engineer working in Nzoia, Kenya, in a sugar factory acquired MVD and died shortly after admission to Nairobi Hospital. His attending physician also contracted MVD but survived [22]. Although it remains unknown how he acquired his infection, he worked at the base of Mount Elgon, where Kitum Cave is located. Kitum cave, 165 meters long and up to 60 meters wide with walls rich in salt, is inhabited by thousands of Egyptian fruit bats as well as by other bat species.

A 15-year-old boy from Denmark developed MVD infection during a visit to Kenya in 1987. He had visited a cave—Kitum Cave—on Mount Elgon and subsequently traveled to Mombasa where he was noted to be ill. He died following transfer to Nairobi Hospital [23]. The causative agent was later found to be a new strain of MVD—the Ravn virus—and thus this was first report of this marburgvirus agent and its causes with human disease.

Two laboratory-acquired infections occurred with MARV occurred in the former Soviet Union in 1988 and 1990. Scant information is available regarding these events, although in one case it is known that the individual became infected after an accidental self-inoculation of MARV with a syringe while working with guinea pigs and which resulted in his death [24, 25].

A major epidemic of MVD occurred in 1998 among gold miners from the Goroumbwa mine in the DRC. This was the largest epidemic to have occurred up to that time and continued sporadically in the cities of Durba and Watsa up to 2000. A total of 154 cases occurred (48 confirmed and 106 suspected), with 52% in young male miners. The large majority (94%) of infected miners worked underground, and cessation of the outbreak coincided with the flooding of the mine [26]. Retrospective virologic and epidemiologic analysis revealed evidence for multiple introductions of MARV and RA VV viruses into the population as there were at least nine genetically distinct lineages of virus circulating during the outbreak [26]. This epidemic had a case fatality rate of 83% and importantly was the first MVD outbreak to report infection of pregnant women and their infants (see below). It also significantly affected children and early teens—for the 145 patients for whom demographic data were available, 18 of them (12%) were under the age of 15 years including 15 infants [26].

The largest outbreak of MVD to develop in Africa began in October 2004 in Angola [27, 28]. Centered in the northeastern Uige Province, this epidemic was not identified as being due to MVD until March 2005 following the transmission of the disease to healthcare workers, which alerted the community to the possibility of Marburg or Ebola virus disease. The outbreak persisted until July 2005 [29], and eventually there were 252 persons infected, of whom 227 died—a case fatality rate of approximately 90% [29, 30]. Case finding and follow-up during this outbreak was hampered by several factors. Individuals and patients often resisted medical assistance and epidemiologic studies because of circulating rumors that the foreign teams were responsible for bringing or spreading the virus. Clandestine practices including families hiding ill members, avoiding going to the hospital, immediately burying the deceased, utilizing native healers, and bringing patients to hospitals just before death suggest that the true severity of the outbreak and data on morbidity and lethality might never be known. In addition, patient records were maintained only upon admission and not during hospitalization [31]. Approximately 75% of the reported cases had occurred in children aged 5 years or younger [32]. There were no available data on the number of pregnant women, if any, who…
became infected during this large outbreak. However, a report from Jeffs et al. and Médecins Sans Frontières (MSF) [33] working at the Uige Provincial Hospital, the initial epicenter of the outbreak, confirmed that screening for MVD was performed in the maternity ward of the hospital. Assessment of pregnant women was particularly challenging, and many of these women were febrile and fulfilled the suspected case definition for MVD, especially as bleeding during pregnancy is common. The authors [33] stated:

“It was often difficult to rule out MHF without testing, but, because many women required constant obstetric assistance, it would have been difficult to admit them all to the formal Marburg ward for assessment. Therefore, a well-equipped isolation area was set up in the maternity ward, including a delivery area and a ward area. Maternity staff were trained in infection control, and separate teams were assigned to the isolation area and the normal maternity ward. Any patient testing positive for MHF was admitted to the Marburg ward. ”

Thus, it would appear that there were possible cases of pregnant women with infection during the Uige outbreak, but that cannot be confirmed.

Between 2007 and 2008, there were two outbreaks of MVD in Southwest Uganda—one among miners working in Kitaka Mine in the Kamwenge District [34] and the other in two tourists, one Dutch and the other American, that had separately visited Python Cave in Queen Elizabeth National Park [35, 36]. Both Python Cave and the Kitaka mine are inhabited by Egyptian fruit bats (Rousettus aegyptiacus) [37].

An outbreak of MVD was declared in October 2012 in the western Uganda districts of Kabale, Ibanda, and Kamwenge [38] that resulted in 20 confirmed or probable cases and 9 deaths. This outbreak was also linked to mining activity in the Ibanda District.

In September 2014 a 30-year-old male healthcare worker (radiographer) developed symptoms of a viral hemorrhagic fever. Following 1 week of illness, he was admitted to a district health facility in the Mpigi District and later transferred to a hospital in Kampala, Uganda. He expired 2 weeks after the onset of illness, and it was later confirmed that he was infected with MARV. The source of his infection was not identified, and there were no other infected persons identified [37, 39].

In October 2017 an outbreak of MVD occurred in the Kween District of Uganda, near the border with Kenya [40, 41]. The initial three infected persons all belonged to the same family and died. The (probable) initially infected person was a 35-year-old herdsman who frequently hunted near the area of Kaptum, which is known for its bat-infested caves. A healthcare worker also became infected.

5. Marburg virus disease in pregnant women, fetuses, and infants

There is very little information available on the effects of MVD on pregnant women, their fetuses, and infants, including clinical obstetrical and neonatal outcomes following MVD infection and the persistence of virus post-infection. Similar to some of the initial outbreaks of Ebola virus disease, the pregnancy status of women suspected or confirmed as having MVD was not generally recorded during outbreaks and may not have even been evaluated at the time of their illness [1]. Based upon the reported cases of filovirus infections occurring in pregnancy, there is no evidence that women who are pregnant are more susceptible to becoming infected with either marburgviruses or the Ebola virus [6]. However, it does appear that once they acquire a filovirus infection, pregnant women are more likely...
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have a fatal outcome than are nonpregnant individuals [1, 6]. Pregnant women with EVD and MVD are at high risk of spontaneous abortion and stillbirth. EVD is associated with pregnancy-related hemorrhage, and although it has not been reported, it can probably also complicate MVD infection. Evidence from a number of reports confirms that hematogenous spread of Filovirus infection through the placenta is the most common source of fetal infection, as high viral titers have been detected in placental tissue not only for Ebola but also for other hemorrhagic fever viruses [11].

The initial report of MVD occurring in pregnant women and fetuses was derived from the outbreak occurring in the gold mining village of Dursa and district capital of Watsa in the DR Congo in 1998–1999 [6, 26]. During this outbreak, at least three pregnant women with MVD were reported, all of whom died. The infection was also lethal for their infants—one woman had a miscarriage, and another delivered an infant who died 7 h after birth. Thus, the only information available on the clinical effects of MVD occurring in pregnant women indicates a 100% case fatality rate among infected mothers and their infants. This is higher than the mortality rates for pregnant women in the initial 1976 outbreak of Ebola virus in Yambuku, Zaire, in which 9 of 82 Ebola-infected pregnant women survived—a case fatality rate of 89%. During that EVD outbreak, ten live infants were born to mothers who subsequently died of the infection. All of these children also died within 19 days [42].

The 100% maternal and infant case fatality rate that has been reported for MVD is most similar to the Ebola outbreak of 1995 in Kikwit, Zaire, in which 1 of 15 EVD-infected women survived (case fatality rate of 95.5%). All of the pregnant women during the Kikwit EVD outbreak presented with severe hemorrhage. In addition to the maternal deaths that occurred during the Kikwit outbreak, ten women (66%) had spontaneous abortions, and one woman delivered a premature stillborn infant. Four of the pregnant women died during the third trimester of pregnancy. The single maternal survivor among this group had a curettage because of an incomplete abortion after 8 months of amenorrhea [42, 43].

The first (and only) report of the potential effect of MVD on the post-infection reproductive health of female survivors of the disease was reported from the initial outbreak of MA VN in Marburg, Frankfurt, and Belgrade in 1967 [20]. There were a total 32 persons who became infected at the three geographic locations, of whom 12 were female. Two of the 12 died, and 4 of the survivors had secondary infections that resulted in milder disease symptoms, compared with cases of primary infection. Three women who had been infected and survived became pregnant 1–2 years later. In all three cases, the pregnancy outcomes were normal. The placentas were tested for Marburg virus and were found to be negative. Umbilical cord blood was tested for antibodies to MARV and was positive for IgG but negative for IgM. When the infants were tested for MARV antibodies 1 year after birth, they were negative [20].

Because clinical disease caused by the marburgviruses and Ebola virus is clinically indistinguishable, it is reasonable to postulate that they have a similar, if not close to or even an identical, pathophysiology when affecting pregnant women and their fetuses. The first report of EVD occurring in pregnant women originated in the first reported outbreak of this disease in Zaire (now DRC) in 1976 [44]. This outbreak in the rural town of Yambuku infected a total of 316 persons, causing 280 deaths over a period of 11 weeks. There were 73 deaths among the 82 pregnant women infected with Ebola virus, a case fatality rate (CFR) of 89% [44]. Analysis of all EVD outbreaks prior to the West Africa Ebola epidemic reveals that there were 112 cases of pregnant women reported who had acquired the infection—an aggregate maternal mortality rate of 86% [11].
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One of the significant findings pertaining to pregnancy arising from the West Africa Ebola epidemic was the potential long-term persistence of filovirus in the tissues of women following clinical recovery from acute infection. The investigation of a family cluster of Ebola virus disease infections that occurred in Liberia provided evidence for long-term persistence of virus in some infected women [50]. Following the infection of a 15-year-old boy in Liberia with EVD in November 2015 and his subsequent death, the evaluation of other family members revealed that his 8-year-old brother had Ebola RNA in his blood, a 5-year-old brother had no evidence of infection, and a 2-month-old brother born in September 2015 had IgG antibodies to Ebola virus that were attributed to maternal transfer. The father had Ebola virus RNA in his blood and an antibody profile that was positive for Ebola-specific IgG and IgM that was consistent with previous EVD infection. The mother/wife had provided care for her adult brother in July 2014—he had died of presumptive EVD after he cared for EVD patients as a nurse’s aide. Shortly after her brother died, she developed clinical illness that was compatible with EVD, but did not seek care, and had a miscarriage in August 2014. She was found to have a high titer to IgG and low titer to IgM anti-Ebola antibodies. In addition, with the results of genomic analysis, these findings indicated that the most plausible explanation for this family cluster of Ebola virus infection was that the mother/wife had survived an episode of EVD in 2014 following her acquiring it from providing care for her infected brother. She then developed persistent Ebola infection, transmitting the virus to her three family members 1 year later [16, 50].

6. Non-human primate models of Marburg virus disease

Pathological examination of the placenta and fetus and in those cases of maternal death, autopsy of the mother, have proved very helpful in understanding of the mechanisms of maternal-fetal transmission of emerging infectious diseases. This has been most recently demonstrated with the role of placental pathology in helping to understand vertical transmission of the newly emergent TORCH virus infection caused by Zika virus [51–54]. However, in the case of filovirus infections such as EVD and MVD, the recommendations from international organizations against pathology examination of placentas, autopsies, and fetal tissues to minimize risk of infection to healthcare workers have diminished our knowledge of the effects on pregnant women, fetuses, and neonates [11].

Experimental studies of laboratory animal infection with infectious agents can be a major source of information on the mechanisms of maternal-fetal transmission of disease, as well as the role of the placenta in vertical infections. There have been many experimental studies of infection with marburgviruses using a variety of non-human primates (NHPs)—these have included cynomolgus macaques (Macaca fascicularis), rhesus macaques (Macaca mulatta), African green monkeys (Cercopithecus aethiops), and squirrel monkeys (Saimiri sp.) [31]. Unfortunately, they have not addressed pregnancy or vertical viral transmission. There is, in addition, a paucity of information available on the effects of experimental MVD infection in non-human primates on the pathological effects on the female genital organs in nonpregnant animals, despite the performance of many autopsies. However, some recent data are available specifically on the pathology of MVD of the female genitalia in NHPs. Four female rhesus macaques were experimentally infected via the intramuscular route with a target dose of 1000 plaque-forming units of Marburg virus/H.sapiens-tc/AGO/2005/Ang-1379v (BioSample identifier SAMN05916381), passage Vero E6p4 [55]. Microscopic
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