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THE EBOLA VIRUS

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Introduction:

Ebola virus (EBOV) belongs to the family filoviridae and has been known to cause a systemic illness with a high mortality. Its usual clinical features include sudden onset of headaches, muscle aches, sore throat, rash and bleeding manifestations. Transmission from person to person can occur both in and outside the hospital environment which may result in intermittent outbreaks of infections.

Aetiology:

The EBOV is a close relative of another virus, the Marbag virus. Both are members of the family Filoviridae. The Marbag virus has only one subtype while the Ebola virus has five, namely: the Zaire, the Sudan, the Reston, the Tai Forest (Cote d'Ivoire) and Bundibugyo subtypes [1 – 3]. The Reston and Tai Forest subtypes are not as pathogenic to humans as the other three subtypes. Both the Marbag and Ebola viruses can be isolated in a variety of cultures including the monkey kidney cells. Both viruses are stable viruses that are capable of surviving for long periods of time at room temperature. They can be destroyed by heat and lipid solvents [1 – 4].

Epidemiology:

In 1976, out of 550 patients presenting with severe haemorrhagic fever in both the Sudan and Zaire, 470 died. In both locations Ebola virus was isolated [1, 4]. The epidemics spread in both these locations from person to person close contact as well as from injections with reused needles. It was important to note that in both locations epidemics ended when strict quarantine procedures were applied [1, 4].

It was interesting to note that in 1989, numerous deaths occurred from haemorrhagic fevers among quarantine primates in Reston Virginia, USA [1, 4]. In the Philippines and Indonesia in

the same year Ebola virus (Reston strain) was isolated from the cynomolgus monkey which were kept in the quarantine facility. Four employees were infected but none died [1, 4].

In 1995, an epidemic of haemorrhagic fever occurred in Kikwit in Zaire and out of 250 clinically identified cases, 80% died [1, 4].
During the outbreak, Ebola virus was isolated from sweat glands of symptomatic patients. This suggested that contact with perspirations of patients with the virus could have facilitated the spread of the virus. It was noted in this setting that strict quarantine measures controlled the epidemic. The reservoir for this virus however is still elusive despite very extensive research. Even though the virus is known to be zoonotic, attempts to identify its natural reservoir has not been successful [1, 4].

Pathology:
Just like its relative the Marbag virus, Ebola virus is "pantropic" which means it is capable of replicating in almost all the organs of the body [1].

Clinical Features:
The incubation period of the EBOV is 3 to 9 days [1, 5, 6, 7]. The initial symptoms appear to be headache in the frontal and temporal areas, malaise, muscle aches in the lumbar area, nausea and vomiting. Fever usually ranges from 39.4 to 40 degrees Celcius. About half of the patients usually complain of conjunctivitis. In the first 3 days diarrhoea can be severe. There may be some lethargy and a change in mentation may be noted [1, 7].

Involvement of the palate, tonsil and cervical lymph nodes may be seen in the first week of the illness. On the first to the fifth day one may see a non-pruritic maculopapular rash in the face and neck which gradually spreads to the periphery. Four to five days later one may see desquamation of the rash especially on the palms and soles. From day 5 to 7 bleeding may be seen in the gastrointestinal tract, the kidneys, vagina and conjunctivae [1, 7].

During the first week the temperature remains high at about 40 degrees celcius. It starts to decrease but increases again by the 12th to fourteenth day. The other clinical signs which appear in the second week are splenomegaly and hepatomegaly, facial oedema, scrotal or labial reddening [1, 7].

Complications of the disease include orchitis which may include testicular atrophy, myocarditis with irregular pulse and electrocardiographic abnormalities and pancreatitis.

Those who die usually do so on the eighth to sixteenth day of the illness. Recovery is usually very slow during which hair loss, abdominal pain, poor appetite and prolonged psychotic disturbances may be seen. Some late sequels like transverse myelitis and uveitis have also been noted [1, 7].

Laboratory findings:

Leukopenia has been noted in the first day. Counts as low as 1000/microltre have been noted. Later atypical lymphocytes and neutrophyls may appear. Thrombocytopenia can be severe and may be seen early. Fatal cases may include disseminated intravascular coagulation. Hypoproteinaemia, proteinuria and renal failure may also be seen. A lumbar puncture is usually normal or reveals minimal pleocytosis. The erythrocyte count is usually low [1, 7].

Diagnosis:

The characteristic epidemiologic features of the virus usually help in the diagnosis. Specific diagnosis of course requires isolation of the virus. In fatal cases of filoviral infection there is a high titre of the virus but low evidence of any host immune response. Gamma irradiation is the most common way to inactivate the virus. Specialized laboratories may be able to conduct polymerase chain reaction of viral antigens [1].

Treatment:

There is no definitive treatment for the virus other than supportive care [1, 5 – 7].

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