East Coast Fever

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East Coast Fever (ECF) is a fatal protozoal disease of cattle and Indian water buffalo characterized by fever, enlarged lymph nodes, respiratory distress and very high mortality. The disease is caused by subspecies of the protozoan parasite *Theileria parva* which are transmitted principally by ticks of the genus *Rhipicephalus.*

Synonyms

African theilerosis, East African coast fever, Rhodesian tick fever, Rhodesian redwater, Buffalo Disease, January disease.

History and Distribution

Robert Koch in 1898 described the small piroplasms, now known to represent a stage of *Theileria parva,* but believed them to be a developmental stage of *Babesia bigemina.* The disease East Coast Fever was recognized in Rhodesia in 1901 and in South Africa in 1902, where Sir Arnold Theiler recognized the small piroplasms and other developmental forms of the parasite as the causative agents of the disease. In 1902, Lousbury showed that the "brown ear tick", *Rhipicephalus appendiculatus* was a vector of the parasite.

East Coast Fever is endemic in Kenya, Uganda, Tanzania, Mozambique, Malawi, Zaire and Zimbabwe, and in most of these East and Southern African countries it is the most important killer disease in cattle. Mortality rates as high as 90% have been reported in some areas, and an annual loss of 50% of the calf crop in endemic areas is not uncommon.

Hosts

Cattle *(Bos taurus)* and the Indian Water Buffalo are highly susceptible. The Cape buffalo and Zebu cattle (*Box indicus)* are relatively resistent. Twenty-four species of Africa bovidae have been shown to harbor theileria-like parasites.

Etiologic Agents and their Vectors

The three suspecies of *Theileria parva* which are recognized as causative agents of ECF are *T. parva parva, T. parva bovis* and *T. parva lawrenci. Theileria parva parva* is the most virulent of these parasites for cattle, *T. parva bovis* is intermediate, and *T. parva lawrenci* is the least virulent, but all three cause ECF in cattle and water buffalo. The close relationship between these parasites has been demonstrated by cross-protection tests and the indirect flourescent antibody procedure. In cattle, *T. parva parva* infection results in large numbers of the intraerythrocytic piroplasms and large numbers of schizonts in the mononuclear leucocytes. *Theileria parva bovis* occurs in intermediate numbers, and in *T. parva lawrenci* infection, both the piroplasms and the schizonts are few in number. *Theileria parva lawrenci* is a benign parasite of the Cape buffalo, and it has been propsed that the two more virulent subspecies represent degrees of cattle adaptation of the parasit.

The principal vectors of *T. parva* subspecies are ticks of the genus *Rhipicephaus, r. appendiculatus* (the brown ear tick) being the most common vector. Other rhipicephalid-transmitted theilerias recognized in East and Southern Africa remain to be characterized. Included among these is *T. taurotragus,* an eland parasite.

Theileria mutans, now believed to be limited to the African continent is transmitted by ticks of the genus *Amblyomma.* Strains of this agent have been shown to cause a disease resembling ECF in cattle in Kenya. *Theileria velifera,* a parasite of buffalo transmitted by *Amblyomma* ticks is not known to be a pathogen in cattle.

The taxonomic niche of the North American theilerias, formerly referred to as *T. mutans,* remains uncertain.

Theileria parva sporozooites enter the bovine body via the saliva of the infected tick. The first detectable developmental stages of the parasite are the macroschizonts (Koch's bodies) which appear in mononuclear leucocytes, first in the lymph nodes and later in the peripheral blood. These are followed by the microschizonts which rupture their host cells releasing micro-merozoites which enter the erythrocytes to appear as a small rod-shaped piroplasms.

Rhicephalus appendiculatus is a 3-host tick which favours the eyes and ears. Larvae feed on infected animals and transmit the parasite as nymphs, or the nymphs pick up the infection and transmit as adults. Adult ticks can live without feed for 18 months and remain infective. Nymphs may remain infective for up to 6 months. No transovarial transmission of the parasite occurs. *Rhipicephalus everstii,* a 2-host tick, other rhipicephalid ticks, and ticks of the genus *Hyalomma* play minor roles as vectors of ECF.

Clinical signs

The incubation period is 9-24 days under natural conditions. The first clinical sign is a febrile reaction with

temperatures ranging from 105-108°F. This is followed by listlessness, anorexia, cessation of lactation and rumination, salivation, lacrimation, and nasal discharge. Marked swelling of lymph nodes is usually seen the parotid, prescapular, and prefemoral nodes being especially prominent. In the terminal stages of the disease respiratory distress, coughing, and frothing at the nose is seen. Diarrhea which may be tar-like and mucoid is often seen. Nervous symptoms may be seen in the cerebral form of the disease which is known as "turning disease" or "cerebral theilerosis". Mortality of over 90% may occur, and the morbidity is often 100%. Death occurs in 5-15 days after onset of symptoms.

Pathologic changes

Pathogenesis - The sporozoites injected by the tick are carried via the lymphatics to the lymph nodes and spleen. Massive multiplication and parasitizing of lymphoid cells is followed by invasion of the erythrocytes and their destruction by small piroplasms.

Portmortem Lesions - Subcutaneous edema, which may be blood-tinged, is a prominent lesion. petechial hemorrhages are seen in the kidney pelvis, under the tongue, and in the muscles and heart fat. Brownish discoloration of fat occurs, but icterus is seldom seen. When icterus is present, mixed infection with *Babesia* or *Anaplasma* should be suspected. Lung edema is usually pronounced and the septae are very distinct. The trachea and bronchii are usually filled with froth. Enlargement of lymph nodes is seen. They may be moist in section and are often haemorrhagic. Hydropericardium, hydrothorax, and ascites are frequently present. Focal accumulation of lymphoid cells in the kidney cortex is a common lesion, and similar lesions may occur in the liver. Hemorrhage and ulceration of the abomasum are common, and may occassionally occur in the intestines. Peyer's patches are usually swollen. The spleen may be normal or enlarged. In the cerebral form of the disease, hemorrage, embolism, and perivascular lymphoid hyperplasia may be seen.

Immunity

Immunity is lifelong in enzootic areas. In the absence of reinfection, the immunity wanes. The mechanism of immunity is not understood. Recent reports indicate that immunologically — different strains may exist. Highly pathogenic strains of *T. mutans* may cause ECF-like disease simulating breakdown of immunity.

Diagnosis

Early diagnosis is based on the clinical signs, lesions and the presence of a vector of ECF. Diagnosis is confirmed by microscopic examination of Giemsa-strained blood, spleen, and lymph node smears. Koch's Bodies occur in the mononuclear leucocytes or occassionally lying free. They appear before the small piroplasms, and are present in a very high percentage of cells in *T. parva parva* infection. In "turning disease" Koch's bodies are seen in brain smears. *Theileria mutans* and *T. annulata* are differentiated from *T. parva* by cross-immunity tests. The piroplasms and Koch's bodies are more frequent in ECF than in other theilerial infections. The fluorescent antibody technique is now a useful diagnostic aid for ECF. ECF can closely resemble bovine leukosis and malignant catarrhal fever.

Treatment

Theileria parva is sensitive to tetracycline antibiotics which may reduce the severity of the disease. The antimalarial menoctone drugs are potentially useful for the treatment of ECF. Therapeutic use of drugs is not widely practised in endemic areas.

Control

The disease is eradicated by eliminating cattle from infected farms and keeping them free of susceptible animals for 2 years. Infected ticks are believed by some to lose the infection when feeding on non-susceptible animals. Tick control by spraying and dipping at appropriate intervals with DDT, BHC, arsenic, or Toxaphene at 3-day intervals markedly reduces the incidence of ECF in endemic areas.

Immunization

The first method was artificial infection of cattle with spleen or lymph node suspensions. This resulted in mortality of up to 25%. Later, Neitz infected cattle with ticks and then controled the infection with chlortetracyclines. This is an effective but expensive procedure. A major advance in ECF research was the cell culture propogation of *T. parva* by Hulliger (1965) and Malmiquist et al. (1970). Cattle have been experimentally immunized with cell culture propogated macroshizonts, but this method has not been widely adopted. Cunningham et al. (1970) experimentally immunized cattle with schizonts obtained from ticks artificially fed *T. parva.*

Another method of immunization which has been experimentally used is infection with tick suspension followed by treatment with tetracycline antibiotic.

Conclusion

Theilerias occur in the United States of America where their vector is *Boophilus annulatus.* This illustrates that theilerias can survive in N. America. *Rhipicephalus* and *Hyalomma* ticks are very hardy and could easily establish in the United States of America. The following potential ECF vectors have been encountered in United States zoos and game compounds in the past decade: *Rhipicephalus everstii, R. pulchellus,* and *Hyalomma* species. Three species of

Amblyomma are present in the USA. Are they potential vectors of pathogenic *T. mutans? Amblyomma variegatum* is now present in Puerto Rico and is a proven vector of pathogenic theilerias. Numerous wild African ungulates are introduced into the USA each year. They could carry in theilerias since 24 species have been shown to be carriers of theilerias-like organisms.

All of these facts make it obvious that the possibility exists that *T. parva* could become established in the United States of America if it were introduced.

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Malignant Catarrhal Fever

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Malignant catarrhal fever (MCF), also known as bovine malignant catarrh or snotsiekte, is an acute, usually fatal, generalized in disease of cattle and several species of wild ruminants. The disease is characterized by fever, catarrhal rhinitis and conjunctivitis, enlarged lymph nodes, and mortality of nearly 100 percent. The causative agent of at least one form of MCF is a herpesvirus.

The disease is worldwide in its distribution, but it is sporadic in its occurence.

Etiology

MCF should not be considered as an etiologic entity, but rather as a clinico-pathologic syndrome appearing in several forms, possibly caused by different agents.

In the so-called "African" of "wildebeest-related" form of the disease the causative agent has been shown to be a cell-assiciated herpesvirus which can be propogated in cell cultures of bovine origin, and which has been serially passaged in rabbits. This virus is carried by clinically normal wildebeest *(Connochaetes-taurinus)* or black wildebeest *(Connochaetes gnu)* which transmit the disease when they come into contact with cattle. A similar virus has been isolated in Africa from hartebeest *(Alcelaphus buselaphus cokei)* an antelope species related to the wildebeest. The "wildebeest-associated" form of MCF has only been described in Africa.

A second form of MCF is the so-called "sheep-associated" form of the disease, so named because it occurs in cattle which have been in contact with sheep. The etiologic agent of this form of the dieease, which is worldwide in its distribution, remains obscure, but there is strong evidence suggesting that a virus may be the causative agent.

Disease resembling MCF has also been described in cattle not in contact with sheep or wildebeest, and a similar clinico-pathologic syndrome has been described in several species of deer, in bison and in kudu. The etiologic agents of these syndromes remain unknown.

Hosts

MCF in its several forms is important as a disease of cattle. MCF-like disease has also been described in white-tailed deer, axis deer, Pere David's deer, mule deer, sika deer, kudu, bantang, gaur, bison and waterbuffalo. Wildebeest and hartebeest are known to be carriers of MCF virus without exhibiting disease, and topi and oryx have been shown in Kenya to harbour MCF virus antibodies. Sheep are suspected, but not proven carriers of MCF virus.