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.

References

1. Barnett, S. F., and Brocklesby, D. W.: *Theileria lawrenci* in Kenya. Bull Epiz Dis Afr, 7, (1959): 345-347. - 2. Barnett, S. F., and Brocklesby, D. W.: Recent Investigations in Theileridae of Cattle and Buffalo in Africa. A mild form of East Coast Fever *(Theileria parva)* with persistence of infection. Brit Vet J, 122, (1966): 361-370, 395. - 3. Branagan, D.: The maintenance of *Theileria parva* Infections by Means of the Lxodid Tick, *Rhipicephalus appendiculatus*. Trop Anim Hlth Prod, 1, (1969): 119-130. - 4. Brocklesby, D. W., and Bailey, K. P.: Oxytetracycline Hydrochloride in East Coast Fever *(Theileria parva* Infection). Brit Vet J, 118, (1962): 81-85. - 5. Brocklesby, D. W., and Bailey, K. P.: The Immunization of Cattle against East Coast Fever (Theileria parva Infection) Using Tetracyclines: A review of the Literature and a Reappraisal of the Method. Bull Epiz Dis Afr, 13, (1965): 161-168. - 6. Burridge, M. J.: Application of the Indirect Fluorescent Antibody Test in Experimental East Coast Fever *(Theileria parva* Infection of Cattle). Res Vet Sci, 12, (1971): 338-341. - 7. Cunningham, M. P., and others: Immunization Against East Coast Fever, the Relationship between Infective Dose and the Severity of the Disease in Cattle. J Parasitol, 58, (1970): 61 (966). - 8. Hulliger, L.: Cultivation of Three Species of *Theileria* in Lymphoid Cells *in vitro*. J Protozool, 12, (1965): 649-655. - 9. Malmquist, W. A., and Brown, C. G. D.: Cell Culture of *Theileria parva*. J Parasitol, 56, (1970): 66-67 (975). - 10. Malmquist, W. A., Nyindo, M. B. A., and Brown, C. G. D.: East Coast Fever: Cultivation *in vitro* of Bovine Spleen Cell Lines Infected and Transformed by *Theileria parva*. Trop Anim Hlth Prod, 2, (1970): 139-145. - 11. Neitz, W. O.: Theileriosis, Gonderioses and Cytauxzoonoses: A Review. Onderstepoort J. Vet Res, 27, (1975): 275-430. - 12. Neitz, W. O.: The Immunity of East Coast Fever. J. S. Afr Vet Med Assoc, 35, (1964): 5-6. - 13. Oteng, A. K.: (East Coast Fever) In: Infect Anim Dis of the Near East. Beirut, Lebanon, Near East Anim Health Inst Handb No 4, 1970. - 14. Wilde, J. K. H.: East Coast Fever. In *Adv Vet Sci.* Edited by C. A. Bradley and C. Cornelius. Academic Press, New York, NY (1967): 207-259.

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.

Transmission

The exact mode of transmission of the various forms of MCF have not been elucidated. In the African (herpesvirus) form of the disease contact transmission between cattle does not seem to occur, but contact transmission from wildebeest to cattle or from wildebeest to wildebeest commonly occurs. Transplacental transmission has been demonstrated in wildebeest and cattle. Transmission from wildebeest to cattle seems to reach its peak during the wildebeest calving season.

In the other forms of MCF of unknown etiology, the modes of transmission remain obscure.

Clinical signs

The incubation period following experimental infection of cattle with injected blood ranges from 12 days to 6 weeks. Several clinical syndromes may then occur. The peracute form of the disease (relatively rare) is characterized by fever of up to 108°F followed by sudden death. The far more common "head and eye" form of the disease is characterized by fever, dullness, mucopurulent nasal and ocular discharges, photophobia, oral congestion with occasional erosions, conjunctivitis and peripheral or diffuse corneal opacity with occasional blindness, coughing, enlargement of lymph nodes, and watery to bloody diarrhea. Skin lesions are occasionally seen. Most cattle with this form of the disease die in one to seven days after the appearance of clinical signs. In rare instances the disease is manifested only by intestinal signs, and nervous signs such as incoordination may be seen.

The few cattle which survive MCF (about 5 percent) may remain carriers of the virus, but are not known to transmit it.

Lesions

In addition to the external lesions described above, macroscopic lesions are found at necropsy in several internal organs. Congestion and erosion of the nasal passages, oral cavity and occasionally the larynx, trachea and bronchii are seen. Hyperemia, edema and erosions of the esophagus, forestomachs, abomasum, and occasionally the small and large intestines, are also encountered. Lymph nodes are usually markedly edematous, often hyperemic, and occasionally hemorrhagic and necrotic. Cystitis may be seen, and small white foci occur in kidney cortices often accompanied by hamorrlagic infarcts.

The most consistent histological lesions are severe necrotizing vasculitis, especially of the small arterioles, and pronounced infiltration of various tissues and organs with mononuclear leucocytes. Necrosis of reticuloendothelial and lymphoid elements of the lymph nodes are usually prominent.

Diagnosis and Differential Diagnosis

Since MCF is probably several different etiologic entities rather than a single disease, there is no single diagnostic test. Disease characterized by the clinical and pathological features described above is now called MCF. As the etiology of the various components of this syndrome become elucidated it should be possible to develop definitive diagnostic tests, none of which currently exist.

On the basis of clinical and pathological findings the MCF syndrome in cattle can easily be confused with bovine viral diarrhea (mucosal disease), infectious bovine rhinotracheitis, bluetongue, and even some forms of metallic intoxication.

Immunity and Immunzation

Since few cattle survive MCF, little is known about immunity to the disease. Attempts to immunize cattle against the African form of the disease with killed virus vaccines have consistently failed. A hartebeest-derived strain to the MCF herpesvirus has been attenuated by serial passage in cell cultures, and has been used to induce immunity to MCF under experimental conditions.

Control

In Africa the disease is controlled by preventing contact with wildebeest. In some areas of the world separation of cattle from sheep is believed to prevent infection, but the disease still occurs in areas free of both wildebeest and sheep, suggesting the presence of some other carrier of the disease, or perhaps cattle to cattle transmission.

Conclusion

MCF is not an etiologically defined disease entity, but rather a collection of similar clinico-pathological syndromes most of which are of unknown etiology. The herpesvirus-induced wildebeest-associated African form of the disease is the entity which should legitimately be called MCF since it is the only form of the syndrome of known etiology. If other forms of the disease are shown to be caused by different agents, these forms of the disease should be given distinctive new names to help clarify the current confusion in terminology.

Finally it should be stressed that the African form of MCF is currently considered to be foreign to the U.S.A. However, in view of the fact that wildebeest are known to be inapparent carriers of the MCF virus, the possibility of introducing this agent into the U.S.A. remains a distinct possibility.

References

1. Boever, W. J. and Kurka, B. Malignant Catarrhal Fever in Greater Kudus. JAVMA. 165:817. 1974. - 2. Clark, K. A. and Adams, L. Garry. Viral Particles Associated with Malignant Catarrhal Fever in Deer. Am. J. Vet. Res. 37:827. 1976. - 3. Karstad, Lars H., Malignant Catarrhal Fever. Infectious Diseases of Wild Animals. Davis, J. N., Karstad, L. H. and

Trainer, D. O. (Eds.) Iowa State University Press. pp. 131-133. 1970. - 4. Pierson, R. E., Storz, J., McChesney, A. E. and Thake, D. Experimental Transmission of Malignant Catarrhal Fever. Am. J. Vet. Res. 35:523. 1974. - 5. Pierson, R. E., Thake, D., McChesney, A. E. and Storz, J. An Epizootic of Malignant Catarrhal Fever in Feedlot Cattle. JAVMA. 163:349. 1973. - 6. Plowright, W., Malignant Catarrhal Fever in East Africa. I. Behavior of the Virus in Free-Living Populations of Blue Wildebeest *(Gorgon taurinus, taurinus,* Burchell). Res. Vet. Sci. 6:56. 1965. - 7. Plowright, W., Malignant Catarrhal Fever in East Africa. II. Observations on Wildebeest calves at the laboratory and contact transmission of the Infection to cattle. Res. Vet. Sci. 6:69. 1965. - 8. Plowright, W., Malignant Catarrhal Fever in Africa. III. Neutralising Antibody in Free-Living Wildebeest. Res. Vet. Sci. 8:129. 1967. - 9. Plowright, W., Malignant Catarrhal Fever. JAVMA. 152:795. 1968. - 10. Plowright, W., Herniman, K. A. J., Jesset, D. M., Kalunda, M. and Rampton, C. S. Immunization of Cattle Against the Herpesvirus of Malignant Catarrhal Fever: Failure of Inactivated Culture Vaccines with Adjuvant. Res. Vet. Sci. 19:159. 1975. - 11. Plowright, W., Kalunda, M., Jesset, D. M. and Herniman, K. A. J. congenital Infection of Cattle with Herpesvirus Causing Malignant Catarrhal Fever. Res. Vet. Sci. 13:37. 1972. - 12. Plowright, W., Macadam, R. F. and Armstrong, J. A. Growth and Characterization of the Virus Bovine Malignant Catarrhal Fever in East Africa. J. Gen Microbiol. 39:253. 1965. - 13. Ramyar, H. and Hessami, M. A Preliminary Report on the Isolation of Bovine Malignant Catarrhal Fever Virus in Iran. Vet. Rec. 91:630. 1972. - 14. Reid, H. W., and Plowright, W. and Rowe, L. W. Neutralising Antibody to Herpesviruses Derived from Wildebeest and Hartebeest in Wild Animals in East Africa. Res. Vet. Sci. 18:269. 1975. - 15. Reid, H. W. and Rowe, L. The Attentuation of a Herpes Virus (Malignant Catarrhal Fever Virus) Isolated from Hartebeest *(Alcelaphus buselaphus cokei* Gunther). Res. Vet. Sci. 15:144. 1973. - 16. Sanford, S. E., Little, P. B. and Rapely, W. A. The Gross and Histopathologic Lesions of Malignant Catarrhal Fever in Three Captive Sika Deer *(Cervus nippon)* in Southern Ontario. Wildl. Dis. 13:29. 1977. - 17. Storz, J., Okuna, N., McChesney, A. E. and Pierson, R. E. Virologic Studies on Cattle with Naturally Occurring and Experimentally Induced Malignant Catarrhal Fever. Am. J. Vet. Res. 37:875. 1976. - 18. Wyand, D. S., Hemboldt, C. F. and Neilson, S. W. Malignant Catarrhal Fever in White-Tailed Deer. JAVMA. 159:605. 1971.

Rinderpest

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Definition

Rinderpest is usually described as an acute, febrile, virus disease of ruminants, especially cattle and water buffalo, characterized by inflammation, hemorrhage, necrosis and erosion of the digestive tract accompanied by a wasting, frequently bloody diarrhea and a mortality approaching 90%. This is an excellent definition when fully virulent strains of the virus infect completely susceptible cattle populations. However in countries where rinderpest is enzootic and particularly where vaccination has been carried out for many years, a much different picture evolves. In these countries it is quite rare to see the fulminating form of the disease. Instead rinderpest takes on a smouldering, less acute characteristic with milder lesions and much lower mortality. Naturally attenuated strains of the virus are sometimes responsible for the milder forms of the disease. These milder forms of rinderpest complicate field diagnosis. It should also be kept in mind that all ruminant game animals are probably susceptible to rinderpest as are Asiatic domestic pigs and African wart hogs. European type domestic pigs are also susceptible but the disease frequently is subclinical. The American peccary and the white tailed deer were shown to be susceptible at the Plum Island Animal Disease Center.

History and Current Status

Rinderpest, also known as cattle plague, probably has caused more losses in cattle and water buffalo than any other single disease in the history of livestock production. It has been compared to the vast bubonic plague epidemics that decimated human populations until the 19th century.[19] Starvation and suffering of large human populations followed the enormous cattle losses in affected countries. Deaths of large numbers of cattle and water buffalo not only caused marked decreases in meat and milk but also resulted in a decrease in work oxen which were, and still are in many developing countries, the main source of farm power.

Accounts of cattle plague go back as far as the seige of Troy in 1184 B.C. and the disease has often been associated with the ravages of war and the movements of armies since that time. Charlemagne is said to have introduced cattle plague into France when he returned with his armies in 810 A.D. During that same year the scourge appeared in Britian. From the 9th century to the times of Napoleon, epizootics of rinderpest swept over Europe about every 50 years, each time severely reducing the cattle populations in the countries affected. The latest outbreak of rinderpest in Britain occurred in 1865, when infection was imported from Russia across the Baltic Sea. The last panzootic of rinderpest in Western Europe occurred in 1870, after the Franco-Prussian War.

However in 1913, during the Balkan War, the infection was introduced into several Balkan countries from Turkey and during World War I parts of Russia were infected. After the war, the disease spread to Poland where it persisted for several years.

The first Veterinary College was established in 1762 by Bourgelat at Lyon, France, as a result of the serious losses from the rinderpest epizootic that terminated in 1750.[6]

The only recent outbreak of rinderpest in Western Europe occurred in Belgium in 1920, when infected cattle from India