TO THE POINT CLASS LECTURES

SYSTEMIC MEDICINE - I (MED 404) FINAL- 1

Top Stories

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Listeriosis

causative agent of listeriosis is *Listeria monocytogenes* (intracellular parasite).

Susceptible:

Mostly sheep but also in other ruminants. It has 16 serotypes on the basis of flageller or somatic Ag. Virulent strain multiply in macrophages and monocytes and produce Lesteriolysin O (major virulent factor) and can grow on wide range of pH i.e. from 4.5 to 9.6. It blocks the enzymatic activity of lysozyme.

Organism is susceptible to almost all common disinfectants. It is wide spread in nature.

Primarily the disease of ruminants, there may be development of encephalitis, pregnant animal may abort, spinal myelitus, septicemic disease, ophthelmitis, gastroenteritis and rare chances of development of mastitis.

Sources f Infection:

Organism is common in environment and infection is not limited jut to the animals, but mammals, birds, insects, fish. Organism can be isolated from animal fecal material, soil water trough, animal feed, walls and floor of farm, animal itself can be source of Listeria.

Most of its complication is due to presence of this organism in silage. It is commonly present in silage but do not multiply in properly fermented silage whose pH is below 4.5.

Transmission:

Lambs can ingest contaminated feed, or it may be congenital disease is due to naval infection. Stress factors also play important role in disease onset. Encephalitis form of disease results from the infection of the nerves after some injury to the buccal mucosa from feed or infection of the tooth cavities and browsing.

Spinal myelitus occurs due to involvement of spinal nerves; there are certain risk factors that help in multiplication of microorganism. Weak the host resistance mechanism. Poor nutrition of the animal Sudden change of weather.

Spinal mellitus: weakness of hind limb, paralysis of forelimb, constriction of pupil, iris swelling, and corneal ulceration of mostly found dead. If survive, green colour feaces. Animal is weak lethargy.

Differential Diagnosis:

Encephalitis, middle ear disease, polioencephalomaletia, rabies, nervous ketosis on cattle, gid , pregnancy toxemia in sheep if enteritis problem then differentiate from salmonellosis..

Zoonosis:

Listeriosos is a feed born disease in humans e.g. milk products and raw milk contaminated with Listeria through feacal material or valerians aim treating dystokia may get dermatitis. Conjunctivitis may occur in humans.

Pathogenesis:

Ingestion of microorganism… penetration in musosa of intestine…. septicemia occur, ultimately there is high rise in temperature. Inapparent infection with prolong faecal excretion of microorganism to a subclinical bacteremia which clears with the development of immunity, bacteremia is subclinical and microorganism is excreted in milk. Splenic Listeriosis with or without menegitis occurs in new born ruminants and pregnant sheep and goats. Listeria monocytogenes is a facultative intracellular pathogen that can infect cells including intestinal cells by direct endocytosis. It can survive in macrophages and monocytes. Bacterial superoxide dismutase protects against bactericidal activity of phagocytes and listereolysin O damages the lysosomal membrane allowing the microorganism to grow in

cytoplasm. In pregnant animals, invasion of placenta and fetus occur within 24 hours of onset of bacteremia, edema and necrosis of placenta leads to abortion, 5-10 days post infection or newly born young ones they may suffer from fetal septicemia. Encephalitis occurs due to acute inflammation of brain stem and is unilateral. Portal of entry is ascending infection is on the nerves (cranial and trigeminal nerves). Spinal myelitus mastitis. Sever gastroenteritis may result.

Necropsy finding:

Microabscesses in intestine

Clinical pathology:

CFT, agglutination test and ELISA.

Clinical signs:

Flock depressed, fever (104-107), in coordination, head deviation (tilting), walking in circles, unilateral facial hypelgesia (pain sensitivity decreased), facial paralysis, dropping of ears, lips paralysis, third eye lid paralysis (ptoris), panopthalmitis (pus in anterior chamber of one or both eyes), prehention and mastication become slow, drooling of saliva, food hanging from mouth.

Duration of disease in cattle is 2-3 weeds while in sheep and goat is 2-4 days. If Listerial abortion in last trimester, retention of placenta, fever 107 ^{0}F, abortion assert soon after commencement of silage feeding, retained placenta, necrosis of placenta, blood stained vaginal discharge for several days.

Septicemic Listeriosis::

Depression, weakness, fever, diarrhea, death within 12 hours. In case of mastitis single or both quarters are involved, somatic cell count increased but milk remains normal.

Treatment:

Chlorotetracyclin 10 mg per Kg body weight.

Penicillin 44000 IU/Kg b. wt I/M for 7 days or may continue upto 14 days.

Supporting therapy:

Fluid therapy such as Ringer lactate solution (500-1000 ml IV) for 3 days, in sever diarrhea. Penamin-G and Amino-fuel-600 should be given for 3-5 days.

Vaccine:

Formalized killed vaccine (alum precipitated vaccine) 3ml twice in a years (in March & September).

Bacillary Haemoglobinuria

Its causative organism is Chlostridium haemolyticum it provides hemolytic toxin (lysis of blood). These are spore forming, G +ve, anaerobic bacteria. In infected animal, organism is found in liver of healthy animal. Whenever animal ingest organism it is lodged in liver. Association of this disease is with poorly drained pasture or more irrigated pasture because of presence of liver fluke or snails and in summer and in autumn.

Pathogenesis:

Organism is ingested through contaminated feed. It enters into animal, lodges in liver,. Liver fluke invasion: infarct areas due to necrosis of liver tissue or portal vein thrombus or due to Fusebacterium necrobacillosis or due to cysticercus. These infarct areas are site for proliferation of Clhlostridium. They produce hemolytic toxin and go into blood and cause systemic toxemia and also cause vascular damage.

Clinical signs:

There is fever (106 ^{o}F) but in terminal stages sub normal temperature, off feed, anorexia, week and anemic animal, jaundice, abortion. Blood in urine (haemoglobinuria); dark red colour urine.

Treatment:

First specific/immediate treatment: antitoxic serum, penicillin, tetracycline (6-10 mg/Kg), 20,000-40,000 IU/Kg in large animals. By this treatment haemoglobinuria disappear within 12 hours.

In supportive treatment: blood transfusion (10ml/kg body weight), fluid therapy (Dextrose 5%), haemopiotic drugs (iron or copper, B-complex), liver tonic (hepamer, Levagen).

Control:

Vaccination: 4-6 week before expected season, pasture management. Do regular deworming of the animals (2-4 times in a year). Control Snail prevalence and provide balanced diet to the animals. There are more liver fluke infestations in Chiniot, Jhang, Shorkot and Muzzafargarh, due to which animals in these area are more prone to bacillary hemoglobinuria infection.

Differential Diagnosis:

Hematuria (intact blood in urine). This condition develops in kidney infection or in trauma of urinogenital tract. In this case RBCs will settle down at bottom.

Lysed blood mostly in parturient hemoglobinuria, babisiasis, theleriasis. But in parturient haemoglobinuria (mostly in winter): no toxemia, deficiency of phosphorus, oxidants are more in amount in feed, fever does not occur, non responsive to antibiotics.

In babesiasis and theleriasis parasite present is seen in blood smear, staining with Giemsa stain. If red water haematuria due to phosphorus deficiency then urine colour will be chocolate coloured. In haemoglobinuria temperature remains increased. If phosphorus deficiency then wall of RBC will become fragile as phosphorus is required for the formation of cell wall. When it breaks down the RBCs will be lysed and come to urine.

Theleria and babesia: dark yellow colour. When parasites are present in RBC then animal suffer fever and when RBCs burst, fever falls down. So intermittent fever and they are not treated with antibiotics. Animal will not respond to antibiotics.

Vaccine: Formalized killed vaccine – twice in a year

Anthrax

Anthrax is originated from Greek word means coal. Other names are spleenic fever, woolsorter's disease. Locally it called as golle or sut.

It is an acute, contagious and septicemic disease. Highly fatal and affecting a wide range of mammalian species including human beings. Before the availability of an effective vaccine, anthrax was one of the most important causes of death in livestock throughout the world. It became available in late 1930. The western countries have controlled but fatal in underdeveloped countries. The results of national epidemiological survey of important diseases of livestock in Pakistan has indicated that anthrax is one of the leading causes of death among sheep, goat cattle in hilly and desert areas. Anthrax occurs in all vertebrates but it is more common in cattle and sheep and less frequent in horses and goats. Humans occupy an intermediate position. Dogs and cats are relatively resistant.

Anthrax is caused by a bacterium known as *Bacillus anthracis*. The organism is G +ve, non motile, aerobic, facultative anaerobe and spore forming. There are two forms of this organism: vegetative and spore forming. Vegetative form occurs inside the body of affected animals and is responsible for producing clinical signs and pathological lesions. The spore formation occurs outside the body of host and is the result of exposure of vegetative form to oxygen.

When disease is septicemic (usually in herbivores); blood secretion, excretion (urine and feaces) and tissues of affected animals are filled with vegetative form of B. anthracis. If carcass is not opened, the vegetative form of organism will die within few hours.

Transfer:

- Mostly animals are infected while grazing in areas that have previously experienced anthrax.
- The spores are also transmitted through the consumption of contaminated water, hay, and fodder.
- Eating of bone meal and blood meal of infected animals also cause transmission.
- Eating of dry fodder or spiky grass produces lesions in gastrointestinal mucosa, and the chances of infection are increased.
- Flies are also a source of transmission.

Zoonosis:

In developing countries it is a major cause of human illness. It is a serious cause of mortality in humans who eat infected meat and develop alimentary form of disease. Cutaneous form occurs in veterinarian following postmortem examination of carcass infected with anthrax and lesions are mostly restricted to fore arms and neck.

Pathogenesis:

Upon ingestion of spores, infection occurs through intact mucous membrane, through defects in epithelium around erupting teeth or through scratches form tough and fibrous food materials. Organism is resistant to phagocytosis due to Poly D glutamic acid in capsule. It proliferates in regional draining lymph nodes passing through lymphatic vessels into blood stream, causing septicemia. Bacillus anthracis produces lethal lesions that cause edema and tissue damage. Death occurs due to shock, acute renal failure and anorexia.

Clinical Findings:

Its incubation period is 1-2 weeks, some says 7 weeks. Most common sign of disease is sudden death. In cattle and sheep there are three forms of disease:

Peracute: It is most common at the beginning of out break. Animals are found dead without signs. Course of disease is only two hours. Signs may be fever, dyspnia, congestion of mucosa and muscle tremor and animal dies after convulsion. After death there is discharge of blood from natural orifices (mouth, nostrils, anus, vulva etc.).

Acute: Course of disease is 48 hours. There is severe depression, increased body temperature upto 107 ^{o}F, rapid and deep respiration, and congested mucosal lining.

Pathogenic signs are congestion of mucous membrane, hemorrhage from natural orifices, increased heart rate, animal off feed, ruminal stasis, abortion in pregnant cows, blood stained or deep yellow milk, diarrhea, dysentery, and local edema of tongue

Chronic: Chronic infection is characterized by localized, subcutaneous, edematous swelling that can be quite extensive. Areas most frequently involved are ventral neck, thorax, and shoulders.

Diagnosis:

It is based on the history of the occurrence of disease in an area, then clinical signs, necropsy findings.

Post mortem is not allowed. But if by mistake the carcass is opened, septicemic lesions are seen. Blood is dark, thickened, and hemolysed and fails to clot readily. Dark clotted blood in spleen. Spleen is enlarged, soft and hemorrhagic. The apparent petechial hemorrhages may be visible throughout the organs. Intestinal mucosa is dark red and edematous with areas of necrosis. The carcass undergoes rapid purification. Small hemorrhages are detectable in mucosa of serous membranes and subcutaneous tissues. There is accumulation of blood stained fluid in serous cavities and gelatinous fluid in loose connective tissue.

To prepare blood smear, blood is obtained from ear by giving incision. Blood film should be dried and fixed by heat or immersion for one minute in absolute methanol and stained with polychrome methylene blue. Then it is washed after thirty seconds into hypochlorite solution. After drying the slide, it is examined under microscope for reddish purple capsular material and deep blue Bacilli. This reaction is termed as M- Fadyean reaction.

Treatment:

Because it is rapid in onset and with large mortality rate upto 90 %, this is insufficient to initiate treatment before death. If anthrax is suspected, segregation of animal should be done. Early supportive and antimicrobial therapy is useful and Bacillus anthracis is highly susceptible to a wide range of antibiotics including benzylpenicillin @ 200,000-400,000 IU / Kg, tetracycline, and ciprofloxacin.

First dose of antibiotic should be administered I/V and then I/M for 5 days. Serum is also available with dose rate of 50-100 ml s/c 2-4 times per day. Prognosis is not favourable and no time to treat the animal.

Differential Diagnosis:

In cattle and buffalo differentiate it from acute fatal blot, per acute babesiasis, gross tetany, black quarter, acute poisoning, and enteritis. Anthrax should be considered in differential diagnosis when an animal dies after having observed apparently good health during the preceding 24 hours.

Control Strategy for Anthrax:

Control measure aim is to break the cycle of infection. The important thing is to correct the disposal of carcasses. When a cow or buffalo dies inside a shed, paddle or barn, its carcass should be received for burial incineration.

Plug the natural orifices properly before disposing carcass. Burial should be away from water supply and pasture.

The pit should be 180 cm deep. The top layer after burying should be covered with unslacked lime. Decontaminate the area, bedding, unconsumed feed, and room. Dip the equipments in 4 % formaldehyde solution for 12 hours.

Vaccination:

VRI has developed anthrax spore vaccine. It is a suspension of live, attenuated spores of non-capsulated Bacillus anthracis in glycerin saline. It imparts solid immunity for one year. Packing contains 300 ml. Its shelf life is one year. Its dosage in cattle, buffalo, and horse is 1 ml s/c; in sheep and goat it is 0.5 ml.

Scrapie

Disease of sheep and goat; characterized by pruritis. It has long incubation period. Causative agent is viroid, the protein particle which effect ventrolaterla side of the spinal cord. This particle causes degeneration of neurons. This particle multiplies in host and look as it is virus but it is not virus. Similarity to virus is that it can survive boiling for 30 min, survive antivirals, freezing and thawing. Jerk can not kill. There is no effect of either and 20 % formalin. Viroid is an agent which has no antigenecity. So there is no antibody production. No serotype and can not be cultured and no immunity against scrapie.

Occurrence:

Most common in Europe and less in Australia, Canada, Newzeland, USA and India.

Susceptible:

Foreign breeds of sheep and goat. 20-30 % morbidity and 100 % mortality.

Transmission:

Direct from and animal to another and can transmit from dam to offsprings. It is genetically inherited.

Pathogenesis:

This particle multiplies in lymphatic tissues and then transfer to the spinal cord and brain through sympathetic nervous system. This particle causes vacuolation in medulla, ventrolateral and dorsal spinal cord and also in brain. Lesions are bilateral. It also causes the degeneration of optic and ophthalmic nerves. There is imbalance of cerebellum.

Clinical Findings:

2-12 months and in some cases average within 6 months. Effected animal show changes in behavior; tremor, pruiritis, and locomotory disorder. In early stages nervous signs for weeks. It may be confused with BSE. Signs occur at different intervals for several weeks**.** These episodes show collapse, and sudden change of behaviour. Sheep charge at walls, doors or closed gates. Rubbing and biting at skin. Most of time nervous signs may not be observed. There is pruritis on rum, thighs and base of the tail where animal bites usually. Upper portion of head and dorsum of the neck may also involve. But ribs behind the elbow are less commonly involved. You will see the bilateral symptoms (both on left and right). In advance cases there is severe pruritis, muscular tremors and abnormality of gait and emaciation. Persistent rubbing can result in loss of wool on the area. Hematoma of ear and swelling of face may occur due to rubbing. Whenever application of heat or cold or pressure then animal will show nibbling (pleasant feelings), light or deep pressure, and pricking needle may cause nibbling and scrapie reaction (elevate the head and nibbling movement of tongue and lips). This reaction shows that animal feels pleasant. Impairment of locomotion, hind limb abnormality appears first and there is incomplete flexion of hock, shortening of steps jump. Weakness and lack of balance and hyper excitability present. Nodding or jerking of head. Other clinical signs: no proper swallowing, pretension, vomiting, loss of vision due to optic nerve damage. Anorexia not present upto 4-5 weeks but after that anorexia.

Goat:

Pruiritis, hyperexcitability, same as in sheep but less loss of body conditions. Clinical course period is 2-24 weeks.

Diagnosis:

There is vacuolation in optic nerve, medulla, spinal cord. Vacuolation is more visible at microscopic level.

Differential diagnosis:

Looping ill: it is short term and affect young and old but scrapie affects aged animals.
Pseudo rabies: very rare
Photosenstization.
Pregnancy toxemia: have ketourea.

Treatment:

No specific treatment so animal should be slaughtered.
For pruritis: give sedatives and anti-inflammatory drugs.

Control:

Adopt strict quarantine measures. No vaccination.
Do culling of infected/incurable animals.

Johne's Disease

Synonym: Paratuberculosis, Intestinal T.B.

It is chronic infection of intestine, mostly present in cattle, sheep, and goat. Disease named after Henry Johne.

Transmission:
By nursing of calf by infected dam. Organism shed directly in colostrums and milk and through contaminated feed. It also have oral feacal route. Also transmitted from dam to fetus also present in genitalia.
Most important is infection occurs under thirty days of age and incubation period is very long so clinical signs appear at 3-5 years of age.
Organism is also transmitted by trade.
Pathogenesis:
Organism → ingestion → localized in small intestine → phagocytosized by microphages → do not able to change in phagolysosome, so organism remain viable in microphages (infected macrophages) → immune system fight against macrophages as a result injuries to epithelium of intestine due to which malbsorption, hypoproteinemia and diarrhea occur. This disease is also called the tuberculosis of intestine.
Clinical Findings:
Stage one is silent stage in which no clinical signs and occur in younger animals, carrier but healthy.
In subclinical stage animal becomes carrier and potential source of infection. It is at adult stage.
In clinical stage chronic diarrhea with presence of air bubbles and no odour, feed intake is normal, all other parameters normal but more thirst.
In advanced clinical stage emaciation, dehydration and ultimately death.
Treatment:
No treatment is recommended because organism is intracellular. But in this disease streptomycin (50 mg/kg b. wt.) or isoziazid (for T.B) (20 mg/Kg b. wt. for 100 days. Refampicin (used for T.B.). Erythromycin can also used in higher doses
Losses:
Low milk production, low feed efficiency, more chances of infertility and mastitis, premature culling
Diagnosis:
Blood test, ELISA, faecal examination.
Johnin test: There is a tuberclin like test. 0.2 ml Johnin is injected Intradermally at the neck. If swelling occurs after 48-72 hours, test is positive. Johnin is purified protein derivative (PPD).
Zoonosis:
Chronic inflammatory disease 7.5-38 %, Corhen disease. Mycobacterium avium is isolated from the patient of Corhen Disease. It may be due to consumption of unpasteurized milk.
Control:

i) Test and slaughter policy should be adapted.
ii) Do vaccination
iii) Genetic selection

Lecture Delivered by Dr. Nadeem Asi

Babesiosis

Babesiosis is caused by intraerythrocytic protozoan parasites of the genus *Babesia* . The disease, which is transmitted by ticks, affects a wide range of domestic and wild animals and occasionally humans. While the major economic impact of babesiosis is on the cattle

industry, infections in other domestic animals, including horses, sheep, goats, pigs, and dogs, assume varying degrees of importance throughout the world.
Two important species in cattle— *B bigemina* and *B bovis* —are widespread in tropical and subtropical areas and are the focus of this discussion.

Transmission and Epidemiology

The main vectors of *Babesia bigemina* and *B bovis* are 1-host *Boophilus* spp ticks, in which transmission occurs transovarially. While the parasites can be readily transmitted experimentally by blood inoculation, mechanical transmission by insects or during surgical procedures has no practical significance.

In *Boophilus* spp ticks, the blood stages of the parasite are ingested during engorgement and undergo life cycles in the replete female, eggs, and subsequent parasitic stages. Transmission to the host occurs when larvae (in the case of *B bovis*) or nymphs and adults (in the case of *B bigemina*) feed. The percentage of larvae infected can vary from 0-50% or higher, depending mainly on the level of parasitemia of the host at the time the female ticks engorge. Under field conditions, the rate of tick transmission is generally higher for *B bigemina* than for *Bbovis* .

In endemic areas, 2 features are important in determining the risk of clinical disease: 1) calves have a degree of immunity (related both to colostral-derived antibodies and to age) that persists for ~6 mo, and 2) animals that recover from *Babesia* infections are generally immune for life. Thus, at high levels of tick transmission, all newborn calves will become infected with *Babesia* by 6 mo of age, show few if any clinical signs, and subsequently be immune.

Clinical Findings and Pathogenesis

The acute disease generally runs a course of ~1 wk. The first sign is fever (frequently 105.8°F [41°C] or higher), which persists throughout, and is accompanied later by inappetence, increased respiratory rate, muscle tremors, anemia, jaundice, and weight loss; hemoglobinemia and hemoglobinuria occur in the final stages. CNS involvement due to adhesion of parasitized erythrocytes in brain capillaries can occcur with *B bovis* infections. Either constipation or diarrhea may be present. Late-term pregnant cows may abort, and bulls may undergo temporary infertility due to transient fever.
With virulent strains of *B bovis* , a hypotensive shock syndrome, combined with generalized nonspecific inflammation, coagulation disturbances, and erythrocytic stasis in capillaries, contribute to the pathogenesis. With most strains of *B bigemina* , the pathogenic effects relate more directly to erythrocyte destruction. Hypercoagulability (DIC)
Animals that recover from the acute disease remain infected for a number of years with *B bovis* and for a few months in the case of *B bigemina* . No clinical signs are apparent during this carrier state.
The susceptibility of cattle breeds to *Babesia* infections varies;for example, *Bos indicus* cattle tend to be more resistant to *B bovis* and *B bigemina* infection than are European breeds.

Lesions:

Lesions include an enlarged and friable spleen; a swollen liver with an enlarged gallbladder containing thick granular bile; congested, dark-colored kidneys; and generalized anemia and jaundice. The urine is often, but not invariably, red. Other organs, including the brain and heart, may show congestion or petechiae.

Diagnosis

It can be based on:
Consideration of host susceptibility: - Babesiosis like other protozoan (e.g. anaplasmosis) diseases is far more prevalent in purebred exotic (e.g. Holstein-Friesian, Jersey etc.) and crossbred cattle than native cattle (e.g. Sahiwal, Dhanni) and buffaloes. The affected cattle are usually older than 9 months.

History: - history of occurrence of disease in the area or ona particular farm.
Clinical signs: - high fever, jaundice, anaemia, weakness, haemoglobinuria, pneumonia.
Epidemiological characteristics of the disease: - occurrence of disease in summer, and fall months, presence of ticks, occurrence of disease in cattle older than 9 months.
Microscopic examination of thick an dthin blood smears (preferably prepared from the ear tip or tail) stained with Giemsa's stain usually reveals the presence of piroplasm in the red blood cells.
In case of acute *B.bovis* infection, only about 1% of erythrocytes in the circulation contain the parasites. Whereas in case of *B.bigemina* infection,10-30% of erythrocytes may contain parasites.

Differential Diagnosis

1. **Parturient hemoglobinuria**: a disease of females that are lactating or in advancedpregnancy. Contrary to babesiosis, it is generally nonfebrile and affects usually buffaloes, whereas babesiosis usually affects cattle (both male and female). Prevalence of parturient hemoglobinuria is higher in winter and spring months, whereas prevalence of babesiosis is higher in summer and autumn months.
2. **Anaplasmosis**: Less acute, relapses are more common, and hemoglobinuria is extremely rare in this case.
3. **Leptospirosis**: a febrile disease characterized by fever, hemoglobinuria, a diminished milk yield with soft and pliable udder (cold mastitis), the milk I yellow and thickened an may be stained pink or red. Abortions are very frequent. It responds very well to antibiotics.
4. **Hematuria**: fever is absent, intact red blood cells in the urine will deposit as a sediment if the urine is centrifuged.

Treatment and Control

A variety of drugs have been used to treat babesiosis in the past, but only diminazene aceturate and imidocarb dipropionate are still in common use. These drugs are not available in all endemic countries, or their use may be restricted. Diminazene (Inj. Pronil, Selmore Pharma, Pakistan) is given IM at 3-5 mg/kg. For treatment, imidocarb (Inj. Imizol 12% w/v, marketed by ICI, Pakistan Ltd.) is given SC at 1-3 mg/kg. At a dosage of 3.0 mg/kg, imidocarb provides protection from babesiosis for ~4 wk and will also eliminate *B bovis* and *Bbigemina* from carrier animals. Long-acting tetracycline (20 mg/kg) may reduce the severity of babesiosis if treatment begins before or soon after infection.
Supportive treatment is advisable, particularly in valuable animals, and may include the use of anti-inflammatory drugs, antioxidants, and corticosteroids. Blood transfusions may be life-saving in very anemic animals.
Vaccination using live, attenuated strains of the parasite has been used successfully in a number of countries. Several recombinant antigens have been shown experimentally to induce immunity, but commercial vaccines are not available.

Zoonotic Risk

A small number of cases of human babesiosis have been reported, but the species involved often has not been identified with certainty. *Babesia divergens* , *B canis* , *B microti* , and an unnamed species (WA-1), have been incriminated. Cases reported in splenectomized or otherwise immunocompromised individuals are often fatal.

----------Up to Date: 19 Dec 2010