

ANNEXURE - XD

COMMONLY FIND DISEASES IN LIVESTOCK

Specialization: **Animal Health**

Subject :**Disease of livestock**

Disease: **Babesiosis**

Species: **Buffalo/Cattle**

Source of the material: *The Merck Veterinary Manual, Eighth Edition, CD-ROM*

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Country of origin: India

Author's name

Institution:

Organization:

Content:

Symptoms: 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 loss of weight with hemoglobinemia and hemoglobinuria in the final stages. CNS involvement due to slugging of parasitized erythrocytes in brain capillaries occurs frequently with *B. bovis* infection. Either constipation or diarrhea may be present. Pregnant buffalo often abort. 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. 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 signs are apparent during this carrier state. 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 petechial hemorrhages.

Diagnosis

(i) Barnyard diagnosis

(ii) **Laboratory diagnosis** While clinical signs and lesions often clearly point toward a diagnosis of babesiosis, this should always be confirmed by examination of Giemsa-stained blood or organ smears. From the live animal, thick and thin blood smears should be prepared, preferably from capillaries in the ear or tail tip. Jugular blood in EDTA should also be forwarded to the laboratory for hematological examination. Smears of heart muscle, kidney, liver, lung, and brain and from a blood vessel in an extremity (eg, lower leg) should be taken at necropsy. A number of serologic tests are available for the detection of carrier animals. The most commonly used are the indirect fluorescent antibody test and ELISA. A procedure that may occasionally be justified to confirm infection in suspected carrier animals is the inoculation of blood (~500 mL) into a fully susceptible animal, preferably a splenectomized calf, and subsequent monitoring of the recipient for infection. DNA probes capable of detecting extremely low parasitemias, as occur in carrier animals, are being developed but are not in general use. Clinically, babesiosis can be confused with other conditions that cause fever, anemia, hemolysis, jaundice, or red urine. Therefore, confirmation of a diagnosis by

smear examination is essential. The species of *Babesia* involved can generally be determined morphologically. *Babesia bovis* is small, with the parasites in paired form at an obtuse angle to each other and measuring ~1-1.5 x 0.5-1.0 µm. *Babesia bigemina* is larger (3-3.5 x 1-1.5 µm), with paired parasites at an acute angle to each other.

Treatment

(i) At farmer level

(ii) At village level: There are a number of effective babesiacides, including quinuronium sulfate, diminazene aceturate, amicarbalide, phenamidine isethionate, and imidocarb. However, not all are still available, and the use of some is restricted in certain countries. Diminazene aceturate and imidocarb dipropionate are two of the most widely used. Diminazene aceturate is given IM at 3-5 mg/kg. For treatment, imidocarb dipropionate is given SC at 1.2 mg/kg. At a dosage of 3.0 mg/kg, imidocarb dipropionate provides protection from babesiosis for ~4 wk and will also eliminate *B bovis* and *B bigemina* from carrier animals. Long-acting tetracycline (20 mg/kg) reduces the severity of babesiosis if treatment begins before, or soon after infection; therefore, the drug may have application in certain circumstances such as to reduce adverse effects after vaccination with live vaccines. Supportive treatment is sometimes desirable, particularly in valuable animals. Blood transfusions may be life-saving in very anemic animals. Anti-inflammatory drugs, such as phenylbutazone, help relieve the inflammatory processes that occur, particularly with *B bovis* infections.

Vaccination using live, attenuated strains of the parasite has been used successfully

While the transmission cycle can be broken by controlling the tick vector, this approach (short of complete eradication) is rarely feasible in the long-term and can lead to instability in endemic areas.

(iii) District level

4. Precaution
5. International status
6. Advisory -Seasonal, etc

Specialization: **Animal health**

Subject :**Disease of livestock**

Disease: Brucellosis, Bang's disease, Malta fever (man), Contagious abortion, Infectious abortion, Undulant fever (man)

Species: **Buffalo/Sheep/Goat/ Man**

Source of the material: *The Merck Veterinary Manual, Eighth Edition, CD-ROM*
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Brucellosis is primarily a reproductive disease characterized by abortion, retained placenta and impaired fertility in the principal animal host. *Brucella abortus* is to a certain extent distinguishable from other brucellae by biochemical reactions and by serological means. The serological differences are related to the amounts of A and M antigens that a *Brucella* strain possesses. Transmission of *B. abortus* is very likely to occur via the oral route because buffalo tend to lick aborted fetuses and the genital discharge of an aborting animal (Cunningham, 1977). Exposure to *Brucella* organisms is also likely to occur in utero (Fensterbank, 1978) or when calves born to healthy dams are fed on colostrum or milk from infected dams (Bercovich et al., 1990). It has been established that brucellosis in bulls does not always result in infertility, although semen quality may be affected. Bulls that remain fertile and functionally active will shed *Brucella* organisms with the semen during the acute phase of the disease. Shedding, however, may cease or become intermittent (McCaughy et al., 1973). In contrast to artificial insemination, bulls used in natural service may fail to spread the infection, as the infected semen is not deposited in the uterus (Ray, 1979). Contamination of a buffaloshed or pasture takes place when infected buffalo abort or have a full-term parturition. Although it is generally accepted that *B. abortus* is not excreted for any considerable time before abortion occurs, excretion in the vaginal discharge of infected animal may occur as early as 39 days after exposure (Philippon et al., 1970). A massive excretion of *Brucellae* starts after abortion and may continue for 15 days. Once the foetal membranes are expelled the uterine discharge diminishes and the number of *Brucella* organisms excreted decreases rapidly (Nicoletti, 1980). Although the infectious material from the genital tract usually clears after 2-3 months, some infected cattle become carriers of *Brucella* and excrete it intermittently for many years (Philippon et al., 1970; Herr et al., 1990). *Brucellae* can survive 30 days in urine, 75 days in aborted fetuses and more than 200 days in uterine exudate. In bedding contaminated with infected faecal material *Brucella* will be destroyed at 56 °C-61 °C within 4.5 hours (King, 1957). However, there are conflicting reports as to its survival in liquid manure. According to one study *B. abortus* can survive at least 8 months at 12 °C (Plommet, 1972) whereas another study indicates that *Brucellae* could not be recovered from slurry after 3 months (Rankin and Taylor, 1969). Yet another study indicates that the survival of *Brucella* is subject to seasonal influences. It has been found that *Brucella* can survive in faeces, slurry, or liquid manure 85-103 days in the winter, 120-210 days in spring, 30-180 days in summer, and 50-120 days in autumn (Kerimov, 1983). Although *B. abortus* is relatively resistant and may survive for a considerable time, the environment is not considered to be an important source of infection (Wray, 1975).

Brucellae are facultative intracellular bacteria that can survive within host cells causing a chronic infectious disease that may persist throughout the life of an animal. Enright (1990) extensively reviewed the pathogenesis and pathology of Brucellae infection in domestic animals. It seems that the initiation of Brucella infection depends on exposure dose, virulence of the organism and natural resistance of the animal to Brucella . Resistance to infection is based on the hosts ability to prevent the establishment of a mucosal infection by the destruction of the invading organism. Invading Brucella usually localize in the lymph nodes, draining the invasion site, resulting in hyperplasia of lymphoid and reticuloendothelial tissue, and the infiltration of inflammatory cells. Survival of the first-line of defense by the bacteria, results in a local infection and the escape of Brucellae from the lymph nodes into the blood. During the bacteraemic phase (which may last 2-8 weeks) the bones, joints, eye and brain can be infected, but the bacteria are most frequently isolated from supramammary lymph nodes, mammary lymph nodes, milk, iliac lymph nodes, the spleen and uterus. The tropism of Brucella to the male or female reproductive tract was thought to be by erythritol, which stimulates the growth of the organism, but Brucella has also been found in the reproductive tract of animals with no detectable levels of erythritol. In the acute stage of infection, abortion occurs at four or five months into pregnancy and cattle usually abort only once. Abortion and retention of the placenta, late abortions or birth of infected full-time calves is common in herds with endemic brucellosis. Excretion of Brucella after parturition may persist for months or years and may re-occur after any consecutive normal parturition. Infected cattle excrete Brucellae in the colostrum or milk although it cannot always be detected (Manthei and Deyoe, 1970; Ray, 1979). In bulls the predilection sites for infection are the reproductive organs and the associated lymph nodes. During the acute phase of infection the semen contains large numbers of Brucella but as the infection becomes more chronic the number of brucellae excreted decreases and excretion may cease altogether. However, it also may continue to be excreted for years or just become intermittent. Usually, orchitis, epididymitis and infection of the accessory sex glands also occur (Jubb and Kennedy, 1963). Abortion and expulsion of the foetus was thought to be the results of a placentitis caused by Brucellae. Proliferation of Brucella in the uterus induces necrosis and destruction of the foetal and maternal placental membranes resulting in death and then expulsion of the foetus. The pathologic changes in the caruncles and cotyledons prevent normal separation and expulsion of the placenta (Jubb and Kennedy, 1963). Although placentitis impairs the normal function of the placenta Brucella endotoxins may also play a role in inducing abortion (Anderson et al., 1986). Brucella abortus may induce production of high concentration of cortisol that decrease progesterone production and increase estrogen production. Decreases in progesterone level and increases in estrogen levels induce a premature parturition (Enright et al., 1984).

1. Symptoms:

Symptoms Table

Symptom	Life Stages Affected	Type
General Signs		
Swelling mass penis, prepuce, testes, scrotum	Cattle & Buffaloes: Bull: breeding male over a year old	Sign
Reproductive Signs		
Purulent discharge, vulvar, vaginal	Cattle & Buffaloes: Cow: female after birth of first calf	Sign
Foul smelling discharge, vulvar, vaginal	Cattle & Buffaloes: Cow: female after birth of first calf	Sign
Abortion or weak newborns, stillbirth	Cattle & Buffaloes: Heifer: young female up to the birth of first calf Cattle & Buffaloes: Cow: female after birth of first calf	Sign
Retained placenta, fetal membranes	Cattle & Buffaloes: Heifer: young female up to the birth of first calf Cattle & Buffaloes: Cow: female after birth of first calf	Sign
Abnormal size testes/scrotum	Cattle & Buffaloes: Bull: breeding male over a year old	Sign

2. Diagnosis:

(i) Barnyard diagnosis

(ii) **Laboratory diagnosis:** Diagnosis is based on bacteriology or serology. *Brucella abortus* can be recovered from the placenta but more conveniently in pure culture from the stomach and lungs of an aborted fetus. Most cows cease shedding organisms from the genital tract when uterine involution is complete. Foci of infection remain in some parts of the reticuloendothelial system, especially supramammary lymph nodes, and in the udder. *Brucella abortus* can frequently be isolated from secretions of the nonlactating udder. Serum agglutination tests have been the standard diagnostic method. Agglutination tests may also detect antibodies in milk, whey, semen, and plasma. An ELISA has been developed to detect antibodies in milk and serum. When the standard plate or tube serum agglutination test is used, complete agglutination at dilutions of 1:100 or

more in serum samples of nonvaccinated animals, and of 1:200 for animals vaccinated between 4 and 12 mo of age are considered positive, and the animals are classified as reactors. Other tests that may be used are complement fixation, rivanol precipitation, and acidified antigen procedures.

Screening Tests: 1) *Brucella* milk ring test (BRT): In the official control and eradication programs on an area basis, the BRT has been effective in locating infected dairy herds, but there is a large percentage of false positive tests. The brucellosis status of dairy herds in any area can be monitored by implementing the BRT at 3- to 4-mo intervals. Pooled milk samples from individual herds are collected at the farm or milk

processing plant. Cows in herds with a positive BRT are individually blood tested, and reactors are slaughtered.

2) **Market cattle testing:** Nondairy and dairy herds in an area may also be screened for brucellosis by testing serum samples collected from cattle destined for slaughter or replacements through intermediate and terminal markets, or at abattoirs. Reactors are traced to the herd of origin, and that entire herd is tested. The cost of identifying reactors by this method is minimal compared with the cost of testing all cattle in all herds. Screening tests, including the brucellosis card test and plate test, may be used in the markets and laboratories to identify presumptively infected animals, thus reducing the number of more expensive diagnostic tests. Brucellosis-free areas can be achieved and maintained, effectively and economically, by using the BRT on dairy herds and market cattle testing. Supplemental tests may be used in cattle in which the brucellosis status is unclear on sensitive screening tests. Use of a battery of these tests improves the probability of detecting infected cattle that have remained in some herds as possible reservoirs of infection. Supplemental tests are also used to clarify the results of plate or card tests, especially in serum samples from vaccinated cattle. These tests, which include complement fixation and rivanol precipitation, are designed to detect primarily the antibodies specifically associated with *Brucella* infection. Another supplemental diagnostic procedure is testing milk samples from individual udder quarters by serial dilution BRT; this is often an excellent method for detecting chronic infection in udders of cows that may have equivocal serum reactions.

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(i) **Farmer level**

(ii) **At village level**

(iii) **District level:** Prolonged treatment of infected domestic animals with a high dosage of antibiotics is not used due to the appearance of antibiotics in the human food chain and it interferes with the production of milk products. Moreover, as *Brucellae* are facultative intracellular bacteria, relapses after treatment usually occur. Therefore, efforts are directed at prevention or eradication of brucellosis. Legislation is needed to effectively control and eradicate brucellosis. There are various combinations of diagnostic tests that can be used to detect buffalo infected with *B. abortus* in tagged animals. Untagged animals can repeatedly (every 5-6 weeks) be tested with the SDTH test, as the test does not sensitize cattle for subsequent SDTH tests (Bercovich et al., 1992). Suspect herds must be tested at regular intervals until all animals test negative. Animals that test positive should be removed from the herd. In areas with endemic brucellosis only vaccination will control brucellosis. Vaccination reduces the number of infected animals and eventually permits disease control. *Brucella* vaccines in use are the live *B. abortus* Strain-19 vaccine and to a lesser extent the whole cell killed adjuvant *B. abortus* 45/20 vaccine. In the last decade a new *B. abortus* vaccine RB51 has been introduced. Vaccination with $40-120 \times 10^9$ CFU (classical dose) of living *Brucella abortus* Strain-19 gives a fair to good protection but it also has some disadvantages (Plommet, 1991). It may cause abortion in pregnant cattle and/or induce an antibody response that confuses the serological diagnosis of brucellosis for 12-36 months. It is excreted in the milk and may induce brucellosis in humans. To diminish these undesirable effects of vaccination with S-19, two vaccination procedures have been suggested. In one procedure calves are vaccinated once with $3-10 \times 10^9$ CFU (reduced dose) at an age of 4-8 months and for the second time with $3-10 \times 10^9$ CFU as adults. The second procedure suggests a conjunctival vaccination of calves with two drops of vaccine containing $4-10 \times 10^9$ CFU at an age of 4-10 months and a second conjunctival vaccination with the same dose six months later. *Brucella abortus* strain RB51 used for vaccination was selected by growth of *B. abortus* strain 2308 in the presence of rifampicin. The protective effect of this vaccine in cattle is similar to that of S-19. Compared with S-19 *B. abortus* RB51 vaccine causes less abortion (Cheville et al., 1996) and does not induce production of agglutinating antibodies of the IgM type, although specific IgG is produced (Stevens and Olsen, 1996). Depending on the doses used it may cause placentitis that leads to pre-term expulsion of the foetus. The vaccine has been approved for use in the USA to allow additional data on field use under controlled conditions (Information Circular, WHO Mediterranean Zoonoses Control Center).

The use of *Brucella abortus* 45/20 vaccine is less common than S-19 because in comparison to S-19 it does not give lasting immunity. The vaccine does not induce

detectable agglutinating antibodies and is not harmful but it gives a marked skin reaction on the injection site. Two initial vaccinations at specific intervals and an annual booster are needed for good protection (Plommet, 1991).

4. Precaution
5. International status
6. Advisory -Seasonal, etc

Specialization: **Animal health**

Subject: **Disease of livestock**

Disease: Foot and mouth disease

Species: **Buffalo/Cattle/Sheep/goats/pigs**

Source of the material: *The Merck Veterinary Manual, Eighth Edition, CD-ROM*

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Foot and mouth disease (FMD) is a highly infectious viral disease of cattle, buffalo, and. It is characterized by fever and vesicles in the mouth and on the muzzle, teats, and feet. In a susceptible population, morbidity approaches 100%. The disease is rarely fatal except in young animals. FMD is caused by an aphthovirus of the family Picornaviridae. There are 7 immunologically distinct serotypes: A, O, C, Asia 1, and SAT (Southern African Territories) 1, 2, and 3. Within each serotype, there are a large number of strains that exhibit a spectrum of antigenic characteristics; therefore, more than one vaccine strain for each serotype, particularly O and A, is required to cover the antigenic diversity. Strains are characterized by their genomic relationships and their antigenic similarities with established vaccine strains. (Previous classification into subtypes became untenable as the number of subtypes rapidly increased.) The virus is quickly inactivated outside the pH range of 6.0-9.0 and by desiccation and temperatures >56°C, although residual virus may survive a considerable time when associated with animal protein (for instance, a proportion of FMD virus in infected milk will survive pasteurization at 72°C for 15 sec). FMD virus is resistant to lipid solvents such as ether and chloroform. Because of the sensitivity of the virus to acid and alkaline pH, sodium hydroxide, sodium carbonate, and citric or acetic acid are effective disinfectants. Transmission of FMD is generally by contact between susceptible and infected animals. Infected animals have a large amount of aerosolized virus in their exhaled air, which can infect other animals via the respiratory or oral routes. All excretions and secretions from the infected animal contain virus, and virus may be present in milk and semen for up to 4 days before clinical signs appear. Aerosolized FMD virus can spread a considerable distance as a plume, depending on weather conditions, particularly when the relative humidity is >60% and when the topography of the surface over which it is dispersing does not cause turbulence. FMD has been transmitted to calves via infected milk, and milk tankers carrying infected milk have been implicated in the spread of disease between farms. Fodder can become contaminated after contact with infected animals and iatrogenic spread of FMD has been reported.

1. **Symptoms:**

The incubation period for FMD is 2-14 days, depending on the infecting dose, susceptibility of the host, and strain of virus—in pigs, it may be as short as 18 hr with some strains of FMD virus. The clinical signs are more severe in cattle and intensively reared pigs than in sheep and goats, and FMD has frequently been ignored or misdiagnosed in small ruminants. In cattle and pigs, after the incubation period, anorexia and fever of up to 106°F (41°C) may develop. Cattle salivate and stamp their feet as vesicles develop on the tongue, dental pad, gums, lips, and on the coronary band and interdigital cleft of the feet. Vesicles may also appear on the teats and udder, particularly of lactating cows and sows, and on areas of skin subject to pressure and

trauma, such as the legs of pigs. Young calves, lambs, kids, and piglets may die before showing any vesicles because of virus-induced damage to the developing cells of the myocardium. Milk yield drops dramatically in milking animals, and all animals show a loss in condition and growth rate that may persist after recovery. Sheep and goats may develop only a few vesicles on the coronary band and in the mouth. Vesicles in

2. Diagnosis

- (i) Barnyard diagnosis: the mouth, even when severe, usually heal within 7 days, although recovery of the tongue papillae takes longer. Lesions on the mammary gland and feet frequently develop secondary infections, resulting in mastitis, under running of the sole and chronic lameness.



- (ii) Laboratory diagnosis: Samples of vesicular epithelium or vesicular fluid should be sent in phosphate-buffered saline (pH 7.4) to the national laboratory responsible for the diagnosis of FMD. Samples must be kept as close as possible to pH 7.4 to prevent destruction of the FMD virus and antigen. They should be securely packed in double leak-proof containers that comply with national and, when appropriate, international regulations for the shipment of pathologic and hazardous material. Samples are prepared as a 10% suspension, inoculated onto susceptible tissue culture, and directly typed by ELISA. Isolated FMD virus is characterized by antigenic comparison with existing FMD vaccine strains, and the nucleotide sequence of a segment of the 1D gene is determined for comparison with other strains of the same serotype to identify a possible origin of the outbreak. ELISA are available to show serologic evidence of vaccination against FMD or recovery from infection: either the liquid phase blocking ELISA, or the more recently introduced solid phase competition ELISA, which is equally sensitive but more specific. Tests for antibodies to the nonstructural proteins (NSP) of FMD virus can be used to distinguish an animal that has been infected from one that has been vaccinated, as only infected animals will have supported live replicating FMD virus, which express the NSP as part of their replication cycle. Virus in FMD vaccine is dead, and consequently there is no expression of NSP; therefore, no antibodies are formed in the host to these proteins. However, there may be sufficient NSP contamination in some vaccines: to cause an antibody response, particularly to the 3D protein, in some animals that have received multiple vaccinations. Conversely, vaccinated animals that have had contact with live virus and become carriers of live FMD virus may fail to produce antibody to NSP, as the immunity provided by the vaccination suppresses viral replication. Rapid diagnostic kits are becoming available for on-farm diagnosis, but they will require stringent validation. PCR is also becoming more frequently used for rapid diagnosis; although difficult to fully validate, this test is likely to be more widely used in the future.

Treatment

(i) At farmer level

(ii) At village level: This has the occurrence of FMD in countries previously free of the disease can have a major effect on local and international trading arrangements. Many countries free of FMD have a policy of slaughter of all affected and in-contact susceptible animals and strict restrictions on movement of animals and vehicles around infected premises. After slaughter, the carcasses are either burned or buried on or close to the premises, and the buildings are thoroughly washed and disinfected with mild acid or alkali and by fumigation. Tracing is done to identify the source of the outbreak and premises to which FMD virus could have already been transmitted by infected animals or animal products, by contaminated vehicles or people, or aerosol. In areas or countries free of FMD in which this is not possible, control is by movement restriction, quarantine of affected premises, and vaccination around (and possibly within) the affected premises. Disadvantage that many carrier animals may remain after the outbreak, and quarantine may not be sufficiently long to prevent their subsequent movement. In countries in which FMD is endemic, protection, particularly of high-yielding buffalo is by a combination of vaccination and prevention of FMD virus entering the dairy premises. This can be difficult if prevalence of FMD in the unvaccinated population is high and climatic conditions are suitable for aerosol transmission. FMD vaccine is a killed preparation and, at best, affords good protection against challenge for 4-6 mo. However, the antigenic diversity of virus strains within each of the serotypes is an additional complication, so it is necessary to ensure that vaccines contain strains antigenically similar to the potential outbreak strains. Otherwise, the duration of immunity provided by vaccines containing dissimilar strains may be very short.

(iii) District level

4. Precaution:
5. International status:
6. Advisory -Seasonal, etc

Specialization: **Animal health**

Subject :**Disease of livestock**

Disease: Hemorrhagic septisemia

Species: **Buffalo/Cattle/sheep/goat**

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Hemorrhagic septisemia (HS) is an acute pasteurellosis, caused by particular serotypes of *Pasteurella multocida* and manifest by an acute and highly fatal septicemia principally in Buffalo. HS is a major disease of Buffalo and water buffalo in Asia, Africa, and some countries of southern Europe and the Middle East. Although it may occur at any time of year, the worst epidemics occur during the rainy season. It is most common in the river valleys and deltas of southeast Asia among buffaloes used in rice cultivation. The HS serotypes of *P multocida* have not been recovered from human infections. However, because many serotypes of *P multocida* have the potential to infect man, appropriate precautions should be taken. : Epidemic HS is caused by one of two serotypes of *P multocida*, designated B: 2 and E: 2. Serotype E: 2 has been recovered only in Africa; B:2 causes the disease elsewhere and also has been recovered from cases in Egypt and the Sudan. Serotypes closely related antigenically to serotype B:2 have been implicated in limited outbreaks of a disease indistinguishable from HS in deer and elk. *Pasteurella multocida* is an extracellular parasite, and immunity is primarily humoral. Infection occurs by direct or indirect contact. The source of infective bacteria is thought to be the nasopharynx of bovine or buffalo carriers. As many as 5% of Buffalo may be carriers in endemic regions. It is hypothesized that animals become susceptible as a result of various stresses, eg, the inanition seen in Buffalo at the beginning of the rainy season. Natural infection occurs by ingestion or inhalation. The initial site of proliferation is thought to be the tonsillar region. In susceptible animals, a septicemia develops rapidly, and death due to endotoxemia ensues within 8-24 hr after the first signs are seen. Exotoxins have not been demonstrated. The mortality rate is high when the agent is introduced to virgin or nonendemic regions. Losses vary widely in endemic areas. The heaviest losses occur during the monsoon rains, and it is thought that the organisms, which can survive for hours and probably days in the moist soil and water, are transmitted widely at this time.

1. **Symptoms:** Most cases are acute or peracute, resulting in death within 8-24 hr after onset. Because the course is so short, clinical signs may easily be overlooked. Animals first evince dullness, then reluctance to move, fever, salivation, and serous nasal discharge. Edematous swelling is frequently seen, beginning in the throat region and spreading to the parotid region, neck, and brisket. Mucous membranes are congested. There is respiratory distress, and usually the animal goes down and dies within hours. Occasional cases linger for several days. Recovery is rare. There appears to be no chronic form.

2. **Diagnosis**

(i) **Barnyard diagnosis:** The most obvious changes in affected animals are the edema, widely distributed hemorrhages, and general hyperemia. In most cases, there is an edematous swelling of the head, neck, and brisket region. Incision of the swellings reveals a clear or straw-colored serous fluid. The edema is also found in the

musculature, and the subserous petechial hemorrhages, which are found throughout the animal, are particularly characteristic. Blood-tinged fluid is often found in the pericardial sac and in the thoracic and abdominal cavities. Petechial hemorrhages are particularly prominent in the pharyngeal and cervical lymph nodes. Gastroenteritis is seen only occasionally and, unlike pneumonic pasteurellosis, pneumonia usually is not extensive.

(ii) **Laboratory diagnosis:** Some characteristic epidemiologic and clinical features aid in the recognition of HS. Of particular significance is a history of earlier outbreaks and a recent failure to vaccinate. Sporadic cases are more difficult to diagnose clinically. The season of the year, rapid course, and high herd incidence, with fever and edematous swellings indicate typical HS. Characteristic necropsy lesions support the clinical diagnosis. Although typical outbreaks are not difficult to recognize clinically, particularly in endemic regions, acute salmonellosis, anthrax, pneumonic pasteurellosis, and rinderpest should be considered. A presumptive diagnosis is based on the isolation of *P multocida* from the blood and vital organs of an animal with typical signs. Definitive diagnosis depends on identifying the serotype as B:2 (or closely related serotypes) or E:2. Other serotypes cause various infections in Buffalo and buffalo but not typical HS. The passive mouse protection test using specific B:2 and E:2 immune rabbit sera is used in Asia and Africa to identify these serotypes. More precise tests, such as indirect hemagglutination, coagglutination, and counterimmunoelectrophoresis and immunodiffusion tests, are available in some laboratories. If there is postmortem decomposition, the causative agent may be overgrown and obscured by extraneous bacteria. In such cases, the subcutaneous inoculation of mice or rabbits with small amounts of blood and tissue suspensions facilitates the recovery of the pasteurellae in pure or nearly pure culture. Serologic tests are of no value in diagnosis. However, the indirect hemagglutination procedure and passive mouse protection test are of value in determining the immune status of animals.

Treatment

(i) **At farmer level**

(ii) **At village level:** Various sulfonamides, tetracyclines, penicillin, and chloramphenicol (where its use is permitted) are effective if administered early. Because of the rapid course of the disease and the frequent difficulty of access to animals, antimicrobial therapy often is not practicable. Although multiple antibiotic resistance has been reported for some strains of *P multocida*, it has not been described for the HS serotypes. The principal means of prevention is by vaccination. Three kinds of vaccine are widely used: plain bacterin, alum-type precipitated bacterin, and oil-adjuvant bacterin. The most effective bacterin is the oil-adjuvant—one dose provides protection for 9-12 mo; it should be administered annually. The alum-precipitated-type bacterin is given at 6-mo intervals. Maternal antibody interferes with vaccine efficacy in calves. The oil-adjuvant vaccine has not been popular because of difficulty in syringing and occasional adverse tissue reactions. A live vaccine prepared from a B:3,4 serotype of deer origin is being used with reported success in southeast Asia.

(iii) **District level**

4. Precaution
5. International status
6. Advisory -Seasonal, etc

Specialization: **Animal health**

Subject :**Disease of livestock**

Disease: Mastitis

Species: **Buffalo/ Cattle**

Source of the material: *The Merck Veterinary Manual, Eighth Edition, CD-ROM*

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Content:

Staphylococcus aureus causes both acute and chronic mastitis that responds poorly to treatment. It is easily transmitted at milking time and colonizes the teat canal but, contrary to prior opinion, does not colonize the skin. In herds in which staphylococcal mastitis is a problem, >50% of the buffalo may have chronic, subclinical infections. *Staphylococcus aureus* may cause peracute mastitis; peracute gangrenous mastitis (in which the skin of the quarter and teat becomes cold and bluish and eventually sloughs); as well as acute, subacute, and chronic subclinical mastitis. Infections lasting more than a few months often are refractory to treatment because of the development of a tissue barrier between the antibiotic and the organism.

Streptococcal Mastitis: The mammary gland is required for perpetuation of *Streptococcus agalactiae* in nature. All other streptococci, whether saprophytes or potential pathogens, enter the mammary gland by chance and do not depend on it for survival. Therefore, *S. agalactiae* mastitis is a specific infectious disease that can be eradicated from dairy herds. The organism enters the gland through the teat opening and resides in the milk and on the surface of the milk channels. It does not penetrate the tissue. Initially, it multiplies rapidly, causes an outpouring of neutrophils into the ducts, and damages the ductal and acinar epithelium, which leads to ductal obstruction with cells and cellular debris. Fibrosis of interalveolar tissue and involution of acini in affected lobules quickly follow and lead to a loss of secretory function. Because *S. agalactiae* spreads from Buffalo to Buffalo during milking, shedder Buffalos should be milked last. Some believe that calves fed on milk containing *S. agalactiae* may transmit it to the immature glands of pen mates if they are permitted to suckle each other. For this reason, milk-fed calves are housed separately. *Streptococcus agalactiae* is most likely to contribute substantially to unacceptable bacterial counts in milk.

Coliform Mastitis: The most common coliforms are *Escherichia coli*, *Enterobacter aerogenes*, and *Klebsiella* spp. In quarters with low cell counts, coliforms multiply rapidly. The inflammatory reaction that follows destroys the coliform population, thereby releasing endotoxin. The resulting toxemia produces the local and systemic signs of acute or peracute mastitis (including gangrene in occasional cases), and death may occur. Rectal temperature in acute or peracute mastitis is 103-108°F (39-42°C). Milk secretion ceases (even though usually only one gland is infected), and anorexia, depression, dehydration, and rapid weight loss are prominent. The secretion of the clinically affected quarter(s) is usually brownish and watery. Diarrhea also may occur. A unique feature is that, on recovery, the udder tissue may return to normal so that, in a subsequent lactation, no fibrosis is found and the gland can produce to full potential. Buffalos producing milk with low WBC counts (<100,000 cells/mL) are more subject to episodes of acute coliform mastitis, and older Buffalos may be even more so because of increased patency of the streak canal.

Pseudomonas aeruginosa Mastitis: Generally, a persistent infection occurs, which may be characterized by intermittent acute or subacute exacerbations. The organism is found in soil-water environments common to dairy farms. Herd-wide infections have been reported after extensive exposure to contaminated wash water, teat cup liners, or intramammary treatments administered by milkers. Failure to use aseptic techniques for udder therapy or use of contaminated milking equipment may lead to establishment of *P. aeruginosa* infections within the mammary glands. Severe peracute mastitis with toxemia and high mortality may follow immediately in some Buffalos, while subclinical infections may occur in others. The organism has persisted in a gland for as long as five lactations, but spontaneous recovery may occur. Culling is recommended for Buffalos infected with *Pseudomonas*.

Actinomyces pyogenes Mastitis: *Actinomyces pyogenes* is common in suppurative processes of buffalo, and it produces a characteristic mastitis in heifers and dry Buffalos. Occasionally, it is seen in mastitis in the lactating udder after teat injury, and it may be a secondary invader. The inflammation is typified by the formation of profuse, foul-smelling, purulent exudate. Mastitis due to *A. pyogenes* is common among dry Buffalos and heifers that are pastured during the summer months on fields and that have access to ponds or wet areas. The vector for animal-to-animal spread is the fly *Hydrotaea irritans*. Control of infections is by limiting the ability to stand udder-deep in water and by controlling flies. Preventive treatment of heifers and dry Buffalos in susceptible areas with long-acting penicillin preparations has been effective in reducing infections. Therapy is rarely successful, and the infected quarter is usually lost to production. Infected Buffalos may be systemically ill, and Buffalos with abscesses usually should be slaughtered.

Unusual Forms of Mastitis: *Mycoplasma* spp can cause a severe form of mastitis that may spread rapidly through a herd with serious consequences. *Mycoplasma bovis* is the most common cause. Other significant species include *M. californicum*, *M. canadense*, and *M. bovis genitalium*. Typically, onset is rapid, and the source of infection is believed to be endogenous after outbreaks of respiratory disease in heifers or Buffalos. The disease is often seen in herds undergoing expansion in which animals, from outside sources, have been added. Some or all quarters become involved. Loss of production is often dramatic, and the secretion is soon replaced by a serous or purulent exudate. Initially, a characteristic fine granular or flaky sediment may be seen in the material removed from infected glands. Despite the severe local effects on udder tissue, Buffalos usually do not manifest signs of systemic involvement. The infection may persist through the dry period. Because there is no satisfactory treatment, affected Buffalos should be segregated at least for that lactation or for their lifetimes, or slaughtered. Sanitary measures should be strictly enforced, especially at milking or during treatment. *Nocardia asteroides* causes a destructive mastitis characterized by acute onset, high temperature, anorexia, rapid wasting, and marked swelling of the udder. Response in the udder is typical of a granulomatous inflammation and leads to extensive fibrosis and formation of palpable nodules.

Mastitis due to various yeasts has appeared in dairy herds, especially after the use of penicillin in association with prolonged repetitive use of antibiotic infusions in individual Buffalos. Yeasts grow well in the presence of penicillin and some other antibiotics; they may be introduced during udder infusions of antibiotics, multiply, and cause mastitis. Signs may be severe with a fever, followed either by spontaneous recovery in ~2 wk or by a chronic destructive mastitis. Other yeast infections cause minimal inflammation and are self-limiting. If mastitis due to yeast is suspected, antibiotic therapy should be stopped immediately.

1. **Symptoms**

2. **Diagnosis**

(i) **Barnyard diagnosis:** in case of peracute mastitis the skin of the quarter and teat becomes cold and bluish and eventually sloughs); as well as acute, subacute, and chronic subclinical mastitis. Loss of production is often dramatic, and the secretion is soon replaced by a serous or purulent exudates.

(ii) **Laboratory diagnosis**

Treatment

(i) **At farmer level**

(ii) **At village level:** Treatment of buffalo with subclinical infections during lactation is not as successful as dry-*Buffalo* treatment; hence, these buffalo should be treated at drying off with an approved dry-*buffalo* infusion product (eg, penicillin-streptomycin preparations, cephalosporin, novobiocin, or benzathine cloxacillin). Peracute and acute staphylococcal mastitis may be treated systemically with an appropriate antibiotic (eg, erythromycin, oxytetracycline). For intramammary therapy, cloxacillin is recommended, but sensitivity tests may reveal that other infusions, such as erythromycin or penicillin, may be effective. Coagulase-negative staphylococci cause subclinical and clinical mastitis. These environmental bacteria also inhabit bovine and human skin. They are believed to be spread from *Buffalo* to *Buffalo* at milking or by contamination of the teat canal after milking. They are commonly found in the udder of springing heifers that have not yet had their first calf. They may be responsible for increased herd somatic cell counts (300,000-400,000) and decreased milk production. Control of these infections depends on a clean environment, use of a pre-dip, proper milking machine function, use of a postmilking teat dip, and dry-*Buffalo* therapy.

Mastitis caused by *S agalactiae* responds well to penicillin, but some of the other streptococci appear to be more resistant. The antibiotic is infused into the infected gland through the teat canal after thorough disinfection of the teat orifice. Cephalosporin or sodium cloxacillin also may be used. Benzathine cloxacillin, penicillin-novobiocin, cephalosporin, or long-acting penicillin preparations may be used in dry-*Buffalo* treatment. Post milking teat dipping will reduce new infections by 50%, and total dry-*Buffalo* therapy will cure >90% of *S agalactiae* infections. *Streptococcus uberis*, *S dysgalactiae*, and other environmental streptococci pose threats of mastitis infections on most farms. These infections arise from environmental exposure of the teat after milking and from contamination of teat skin between milkings. Most environmental streptococcal infections last 14-30 days. About 50% become clinical and may respond to intramammary treatment with lactam antibiotics. Subclinical mastitis caused by environmental streptococci may result in high somatic cell counts on individual *Buffalos* for short periods (30 days). These infections may also result in higher than desired bacterial counts in the bulk tank milk. Prevention of environmental streptococcal infections is based on keeping stalls clean, keeping udders clean and udder hair short (by clipping or singeing), using less water during udder preparation, and sanitizing the teat before milking with an approved pre-dip.

In peracute coliform mastitis, systemic treatment with sulfadimethoxine or antibiotics (such as penicillin, oxytetracycline, ampicillin, or others) is indicated. The affected quarter is infused after the evening milking and repeatedly stripped out during the day to remove bacteria and toxins. Oxytocin may be used to remove more secretion before treatment. Single or repeated injections of flunixin meglumine, antihistamine, or IV administration of a corticosteroid with isotonic, balanced electrolyte solutions or hypersaline plus water orally may be of use as supportive therapy in severe cases. Calcium borogluconate or isotonic bicarbonate solutions may be needed if levels are

low. In acute coliform mastitis, intramammary antibiotic infusions with or without systemic antibiotics initially (depending on the severity) are usually sufficient if the organism is sensitive to the antibiotic in use. Ampicillin, cephalosporins, and amoxicillin have been used with variable results. Frequent stripping is also recommended. Fortunately, most coliform udder infections are eliminated by the Buffalo, often before treatment can be instituted; therefore, cultures of clinical cases may often be negative. Coliform infections are usually of short duration (2-4 wk) and may be subclinical; however, ~90% result in some clinical signs. Herd histories suggest that infection of the udder may be associated with failure to ensure asepsis in intramammary treatment of the common forms of mastitis. Slaughter is recommended for infected Buffalos. *Serratia mastitis* may arise from contamination of milk hoses, teat dips, water supply, or other equipment used in the milking process. The organism is resistant to disinfectants. Buffalos with this form of mastitis should be culled. Yeast or other mastitis infections can be reduced if the tip of the plastic infusion tube is only partially (rather than completely) inserted through the teat canal during intramammary therapy. A chronic, indurative mastitis similar to that caused by the tubercle bacillus has been reported to be caused by acid-fast *Mycobacterium* spp derived from the soil, such as *M fortuitum*, *M smegmatis*, *M vaccae*, and *M phlei*, when such organisms are introduced into the gland along with antibiotics (especially penicillin) in oil or ointment vehicles. The oil apparently enhances the invasiveness of these organisms, and such therapy is contraindicated. These organisms otherwise tend to be saprophytic and to disappear from infected quarters, at least by the next lactation. In the meantime, mastitis is usually moderate. Distinct outbreaks do occur and several have been reported, especially with *M fortuitum* and *M smegmatis*.

(iii) **District level**

4. Precaution
5. International status
6. Advisory -Seasonal, etc

Specialization: **Animal health**

Subject :**Disease of livestock**

Disease: Milk fever

Species: **Buffalo/cattle**

Source of the material: *The Merck Veterinary Manual, Eighth Edition, CD-ROM*
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Author's name

Institution:

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Content: Parturient paresis is an afebrile disease of mature dairy Buffalos that occurs most commonly at or soon after parturition and is manifest by changes in mentation, generalized paresis, and circulatory collapse. At or near the time of parturition, the onset of lactation results in the sudden loss of calcium through milk. Serum calcium levels decline from a normal of 10-12 mg/dL to 2-7 mg/dL. Commonly, serum magnesium is increased, serum phosphorus is decreased, and Buffalos are hyperglycemic. The disease may occur in Buffalos of any age but is most common in high-producing dairy Buffalos >5 yr old.

1. **Symptoms:** Initial signs include excitability, hypersensitivity and restlessness. Tachycardia and mild hyperthermia are commonly associated with tetany in the early stages. Subsequently gradual, worsening muscular weakness begins that progresses into sternal then lateral recumbency. Gastrointestinal atony predisposes to constipation and mild bloating. As calcium levels decrease other signs may include weak pulses, poor pupillary light response, flaccid paralysis, severe bloating, and coma. A weak, trembling cow is first seen. Localized spasmodic muscle contractions may cause a mild increase in body temperature. The heart rate is often elevated. The heart rate remains elevated but the temperature declines with progression. In advanced stages the most common symptom is a "downer" cow that is usually unable to rise when stimulated. An affected cow will often have its head turned into its flank. If left untreated the cow will lie down on her side and stretch out, thus predisposing her to bloat. The cow will become progressively more depressed until she is unresponsive and comatose, with dilated, unresponsive pupils.

2. **Diagnosis**

(i) **Barneyard diagnosis:** Parturient paresis usually occurs within 72 hr of parturition. The disease can contribute to dystocia, uterine prolapse, and retained fetal membranes. There are three discernible stages of parturient paresis. During stage one, Buffalos are able to stand but show signs of hypersensitivity and excitability. Buffalos may be slightly ataxic, have fine tremors over the flank and loins, and display ear twitching and head bobbing. Buffalos may appear restless, shuffling their rear feet and bellowing. If calcium therapy is not instituted, Buffalos will progress to stage two. In stage two, Buffalos are unable to stand but can maintain sternal recumbency. In stage three, Depression, anorexia, dry muzzle, abnormal body temperature, and cold extremities are seen. Auscultation reveals tachycardia and decreased intensity of heart sounds. Smooth muscle paralysis leads to GI stasis, which can be manifest as bloat, failure to defecate, and loss of anal sphincter tone. Ability to urinate can also be lost. Buffalos often tuck their heads into their flanks or, if the head is extended, an S-shaped curve to the neck may be noted. Buffalos lose consciousness progressively to the point of coma. They are unable to maintain sternal recumbency, have complete muscle flaccidity, are unresponsive to stimuli, and can suffer severe bloat. As cardiac output worsens, heart rate can approach 120 beats/min, and pulse may be undetectable. Buffalos in stage

three may survive only a few hours. Differential diagnoses include toxic mastitis, toxic metritis, other systemic toxic conditions, traumatic injury (eg, stifle injury, coxofemoral luxation, fractured pelvis, spinal compression, etc), obturator paralysis, or compartmental crush syndrome. Some of these diseases, in addition to aspiration pneumonia, may also occur concurrently with parturient paresis or as complications.

(ii) **Laboratory diagnosis**

Treatment

(i) **At farmer level**

(ii) **At village level:** Treatment is directed toward restoring the serum calcium level to normal as soon as possible to avoid muscular and nervous damage and recumbency. Recommended treatment is IV injection of a calcium gluconate salt, although SC and IP routes are also used. A general rule for dosing is 1 g calcium/45 kg (100 lb) body wt. Most solutions are available in single-dose, 500 mL bottles that contain 8-11 g calcium. In large, heavily lactating Buffalos, a second bottle given SC may be helpful because it is thought to provide a prolonged release of calcium into the circulation. Calcium provided SC alone may not be adequately absorbed due to poor peripheral perfusion, and SC treatment should not be the sole route of therapy. No matter what route is used, strict asepsis should be practiced to lessen the chance of infection at the injection site. Solutions containing formaldehyde or >25 g dextrose/500 mL are irritating if given SC. Many solutions contain phosphorus and magnesium in addition to calcium. Although the administration of phosphorus and magnesium is not usually necessary in uncomplicated parturient paresis, detrimental effects of their use have not been reported. Magnesium may protect against myocardial irritation caused by the administration of calcium. Calcium is cardiotoxic; therefore, calcium-containing solutions should be administered slowly (10-20 min) while cardiac auscultation is performed. If severe dysrhythmias or bradycardia develop, administration should be stopped until the heartbeat has returned to normal. Endotoxic animals are especially prone to dysrhythmias caused by IV calcium therapy. Administration of oral calcium avoids the risks of cardiotoxic side effects and may be useful in mild cases of parturient paresis. Calcium propionate in propylene glycol gel is effective and avoids the potential for metabolic acidosis caused by calcium chloride. Oral administration of 50 g of soluble calcium results in ~4 g calcium being absorbed into the circulation. Hypocalcemic Buffalos typically respond to therapy immediately. Tremors are seen as neuromuscular function returns. Improved cardiac output results in stronger heart sounds and decreased heart rate. Return of smooth muscle function results in eructation, defecation, and urination once the Buffalo rises. About 75% of Buffalos stand within 2 hr of treatment. Buffalos not responding by 4-8 hr should be reevaluated, and treatment repeated if necessary. Of Buffalos that respond initially, 25-30% relapse within 24-48 hr and require additional therapy. Incomplete milking has been advised to reduce the incidence of relapse. In the past, udder inflation has been used to reduce the secretion of milk and loss of calcium; however, the risk of introducing bacteria into the mammary gland is high.

(iii) **District level**

4. **Precaution :** Feeding diets low in calcium and normal to high in phosphorus during late pregnancy helps prevent parturient paresis. However, such rations are difficult to devise and, if continued for long periods in heavy-milking Buffalos, may result in dangerous depletion of skeletal mineral reserves. Delayed or incomplete milking after calving, while maintaining pressure within the udder and decreasing milk production may aggravate latent mammary infections and increase incidence of mastitis. Prophylactic treatment of susceptible Buffalos at calving helps reduce

parturient paresis. Buffalos are administered either calcium SC the day of calving, or oral calcium gel at calving and 12 hr later.

The prevention of parturient paresis has been revolutionized by using the dietary cation-anion difference. This method is more effective and more practical than lowering calcium in the prepartum diet. Excess anions are provided by adjusting the diet or adding anionic salts, or a combination of the two. Anionic excess apparently enhances calcium resorption from bone and absorption from the intestinal tract. A drawback to feeding anionic salts is poor palatability.

Administration of vitamin D₃ and its metabolites is effective in preventing parturient paresis. Large doses of vitamin D (20-30 million u, daily), given in the feed for 5-7 days before parturition, reduces the incidence, but if administration is stopped more than 4 days before calving, the Buffalo is more susceptible. Dosing for periods longer than those recommended should be avoided because of the danger of toxicity. A single injection (IV or SC) of 10 million IU of crystalline vitamin D given 8 days before calving is an effective preventive. The dose is repeated if the Buffalo does not calve on the due date. Newer compounds used (where available and approved) in lieu of vitamin D and less likely to cause hypervitaminosis include 25-hydroxycholecalciferol; 1, 25-dihydroxycholecalciferol; and 1-hydroxycholecalciferol. After calving, a diet high in calcium is required. Administering large doses of calcium in gel form (PO) is commonly practiced. Doses of 150 g of calcium gel are given 1 day before, the day of, and 1 day after calving. Use of synthetic bovine parathyroid hormone (PTH) may prove to be superior to vitamin D metabolites. Vitamin D metabolites enhance GI calcium absorption, whereas PTH enhances GI calcium absorption

5. International status
6. Advisory -Seasonal, etc

Canine Rheumatoid arthritis: Emerging drug discovery targets And therapeutic candidates

Introduction

Canine rheumatoid arthritis is an erosive polyarthritis. It is a noninfectious, inflammatory, immune-mediated disease. Rheumatoid arthritis in dogs is not very common, and it has no sex predilection. It occurs mainly in small and toy breed dogs. Rheumatoid arthritis has been reported to occur in dogs from 8 months to 8 years of age, with the most common occurrence being 2 to 6 years of age. Rheumatoid arthritis is a chronic problem that can result in joint deformity.

Etiology

The specific cause of rheumatoid arthritis is unknown. It has been speculated that canine distemper virus and the body's immune response to this virus may play a role in the development of canine rheumatoid arthritis. Other possibilities are that type II collagen serves as an autoantigen⁴ and that some type of altered host immunoglobulin (IgG) is the inciting antigen that stimulates the immune response. Autoantibodies subsequently are formed and directed against the altered host immunoglobulin. These autoantibodies are called rheumatoid factors. The autoantibodies form complexes with the altered IgG molecules, and these immune complexes are deposited in the synovium of the joints. Inflammatory mediators are then activated, leading to a severe, erosive polyarthritis known as rheumatoid arthritis.

Clinical Signs Associated with Rheumatoid Arthritis

Animals with rheumatoid arthritis often present with discomfort or pain in their joints. This can be seen as a shifting leg lameness or difficulty rising, walking up steps, and impaired ambulation. The joints that are most commonly affected are the carpal and tarsal joints. Affected joints may display signs of inflammation such as excessive warmth and/or swelling on palpation. Anorexia and malaise often are observed by the owner. The dog also may display a persistent fever. Splenomegaly and muscle wasting also have been reported with rheumatoid arthritis.

Clinicopathologic Findings

Hemogram - A dog with rheumatoid arthritis can have a normal hemogram or may have leukocytosis, neutrophilia, and/or hyperfibrinogenemia. These changes reflect a generalized inflammatory process but do not lead to a specific diagnosis of rheumatoid arthritis.

Synovial Fluid Analysis - Diagnostic indicators of rheumatoid arthritis usually are not present in the hemogram of diseased dogs. Synovial fluid analysis is more diagnostic of this condition. Arthrocentesis of multiple joints should be performed on a suspect animal. The complete synovial fluid analysis is composed of five major categories including physical appearance of the fluid, a mucin clot test, determination of protein concentration, performance of a nucleated cell count, and cytologic evaluation of the fluid.² Serology also may be helpful if rheumatoid arthritis is suspected.

Physical Appearance

Normal synovial fluid should appear clear, colorless to pale yellow or straw colored, and lack turbidity. Viscosity also is evaluated with appearance. Synovial fluid is very viscous because it contains a high amount of hyaluronic acid. Normal viscosity is suggested when a strand of synovial fluid reaches 2 cm or greater before breaking. Viscosity also may be evaluated by examining a cytologic preparation of synovial fluid in which normal viscosity may cause cells to be arranged in rows. Rheumatoid arthritis

is an inflammatory process and the number of cells in the synovial fluid will be increased. Increased cellularity causes the synovial fluid to appear more turbid than expected. Also, joint effusion dilutes the hyaluronic acid in the synovial fluid, causing a decrease in viscosity. Decreased viscosity is detected when the synovial fluid strand breaks before 2 cm in length. A suggestion of decreased viscosity also may be visualized on a smear when the cells are randomly distributed instead of appearing in rows.

Mucin Clot Test

Mucin is hyaluronic acid. A mucin clot test is an assessment of the quality and quantity of hyaluronic acid. With this test, synovial fluid is expelled into 7N glacial acetic acid. The acetic acid causes the mucin to form a clot. Synovial fluid containing normal mucin will appear as clear fluid with a tight, ropy clot. Joint effusion from inflammation will dilute the hyaluronic acid, resulting in the formation of turbid fluid with a more friable mucin clot.

Determination of Protein Concentration

Synovial fluid protein concentration is often measured by refractometry. Normal synovial fluid has a protein content of <1.0 g/dL (this value will be less than the bottom of the scale of most hand-held refractometers). Joint trauma and inflammation will increase the protein concentration of the synovial fluid. Therefore, an animal with rheumatoid arthritis will have an increased protein concentration in their synovial fluid.

Nucleated Cell Count

Only nucleated cells are counted either by manual methods or automated analyzer. Healthy dogs may have a synovial fluid nucleated cell count of up to 1,500 cells/ μ l. Rheumatoid arthritis is an inflammatory process, causing an inflammatory synovitis. The total number of nucleated cells in the synovial fluid is moderately to markedly increased in an animal with rheumatoid arthritis.

Cytologic Evaluation

The most important part of the synovial fluid analysis is cytologic evaluation. Often, only a small amount of synovial fluid can be obtained. In this case, it should be used for cytologic study in preference to other forms of analysis. A wedge smear is made of the synovial fluid and stained with Romanowsky stain. Normal synovial fluid contains many mononuclear cells including macrophages and a few small, well differentiated lymphocytes. Less than 10% of the total nucleated cell count consists of neutrophils.

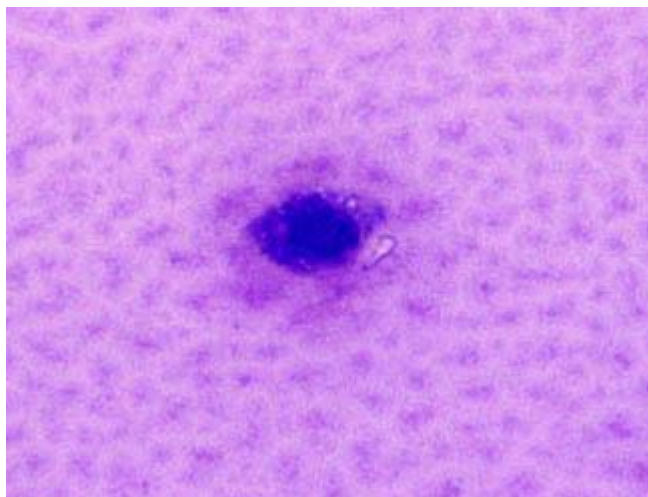


Figure Infrequent large mononuclear cell in the synovial fluid from a healthy dog (Wright stain).

Depending on the cellular content, synovial fluid can be classified as one of the following: normal, degenerative, inflammatory, or acute hemorrhage. Synovial fluid with inflammation can be further classified as either infectious or noninfectious, depending on the presence of microorganisms and the appearance of the neutrophils. Noninfectious inflammation in small animals is often associated with trauma or an immune-mediated process. In rheumatoid arthritis, the nucleated cell count is markedly increased (from $>10,000$ cells/ μl to $100,000$ cells/ μl). The neutrophil is predominant cell type found in the synovial fluid (Fig. 2). Mononuclear cells also may be increased in number, but the neutrophil count alone can be $>5,000$ cells/ μl .

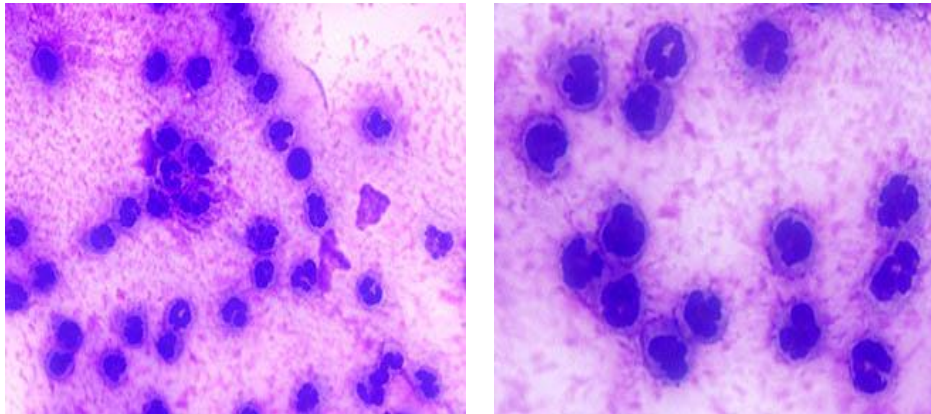


Figure: Numerous neutrophils in the synovial fluid of a dog with rheumatoid arthritis (Wright stain).

Serology

In addition to routine synovial fluid analysis, serology also can aid in the diagnosis of canine rheumatoid arthritis. Serology will detect the rheumatoid factors (RF; autoantibodies directed against the altered IgG). A RF titer = 1:16 is considered a positive test result that is suggestive of rheumatoid arthritis. Positive serological tests are found in 20-70% of affected dogs; however, false positive test results can occur in dogs with other systemic inflammatory disorders. For this reason, a positive RF titer is not a definitive diagnostic test for rheumatoid arthritis. All of the clinical signs and diagnostic findings must be taken into account when interpreting the serological test.

Therapeutics and control

The autoimmune diseases are receiving increasing attention in the pharmaceutical industry as progress is made in the understanding of immune and inflammatory processes. It is predicted that the annual value of the market for drugs used to treat autoimmune disease will exceed \$20 billion in the next few years. Rheumatoid arthritis is one of the more common and difficult to treat autoimmune diseases and there is a great deal of interest in the discovery of novel drugs to treat this condition.

Rheumatoid arthritis is a chronic syndrome characterized by non-specific, usually symmetrical inflammation of the peripheral joints, manifested by the formation of hypertrophied synovia known as panni. Pannus formation mirrors the destruction of articular and peri-articular structures, with or without generalized manifestations. The

condition differs from osteoarthritis not only through the obligatory involvement of the immune system but also because disease onset occurs early on in life, generally between the ages of 20 and 50, although it can begin at any age.

Complications during treatment

Since the birth of the modern pharmaceutical industry just over 100 years ago with the synthesis of aspirin, non-steroidal aspirin-like anti-inflammatory drugs (NSAIDs) have been the mainstay of the treatment of rheumatoid and other forms of arthritis. It is generally accepted that NSAIDs relieve the symptoms of arthritis such as pain and swelling without changing the course of underlying disease. There have been considerable efforts to develop drugs which modify disease progress and these have met with variable success. Immunosuppressants such as cyclosporine or anti-metabolite drugs such as methotrexate are effective but have dose-limiting adverse effects.

During the last few years however, basic research efforts have significantly progressed our understanding of autoimmune disorders such as rheumatoid arthritis. The disease is caused by increased chemotactic and immunostimulatory activity within the joints of sufferers resulting in an influx of inflammatory cell. The presence of activated immune cells increases local levels of cytokines and other inflammatory mediators propagating this process and supporting pannus proliferation and neovascularization, cartilage and bone erosion and eventual joint destruction.

Present scenario of therapeutics

Although significant efforts have resulted in the development of the COX2 inhibitors celebrex and viox, one of the most exciting developments in the management of rheumatoid arthritis has been the introduction of the anti-TNF biopharmaceuticals remicade and enbrel. These drugs represent a real step forward in the development of anti-inflammatory therapies. There is now an increasing number of inflammatory cytokines which have become targets for therapeutic intervention with biologicals such as monoclonal antibodies or immuno-fusion proteins. There is also encouraging evidence that cytokines can be targeted by orally active medicinal chemicals. The increasing knowledge of the immune system has revealed a number of targets for specific regulation of immune cells and in particular the process of antigen presentation, lymphocyte activation and leukocyte chemotaxis. The disease modifying arthritis drug arava selectively suppresses lymphocyte activation by inhibiting nucleotide synthesis and new drugs with similar mechanisms of action are in development. Methotrexate remains one of the most widely used disease modifying drugs in rheumatoid arthritis and attempts are being made to discover improved anti-proliferatives that are more specific and better tolerated. Biologicals which block lymphocyte cell surface receptors thereby preventing activation or adhesion, and molecules that limit chemotaxis are also showing encouraging therapeutic activity. Pannus proliferation is a primary feature of rheumatoid arthritis and therefore the development of anti-proliferative agents represents an opportunity for treating this disease. Another strategy for obtaining the same result is the development of anti-angiogenic agents to prevent the vascularization of inflamed joint tissues. One of the cardinal manifestations of rheumatoid arthritis is joint destruction. This feature results from the infiltration of various inflammatory cells into the pannus and the progression of synoviocytes into a phenotype that supports matrix destruction. This process involves the release of various proteases and the inhibition of these enzymes represents a further strategy in the treatment of rheumatoid arthritis. The report therefore concludes with an analysis of matrix metalloproteinases, elastases and cathepsins; how

these enzymes contribute to disease progression and how the drug development sector is targeting these enzymes. Major components of rheumatoid arthritis including cytokine production, leukocyte chemotaxis and adhesion, lymphocyte activation, synovial proliferation, angiogenesis and matrix degradation are now targeted for drug production.

Drugs in Development for the Treatment of Rheumatoid Arthritis

- Drugs targeting the metabolism of arachidonic acid
 - Cyclooxygenase (COX) Inhibitors
 - Drugs targeting other sites of arachidonic acid metabolism
 - Phospholipase A2 Inhibitors
 - Lipoxygenase Inhibitors
 - Variations on NSAIDs
- Drugs targeting inflammatory cytokines
 - Biological Inhibitors of cytokines
 - Anti-TNF Monoclonal Antibodies
 - TNF receptors and binding proteins
 - Other TNF receptor family members
 - RANK
 - Osteoprotegerin
 - Interleukin-1 antagonists
 - Other cytokine-targeted biologicals
 - Interleukin-6
 - Interleukin-8
 - Interleukin-10
 - Interleukin-12
 - Interleukin-15
 - Interleukin-18
 - Lymphotoxin β
 - B-lymphocyte stimulator
 - New Chemical Entities (NCEs) designed to inhibit cytokines
 - Inhibitors of p38 mitogen activated protein kinase (MAPK)
 - Inhibitors of cytokine activating enzymes
 - Inhibitors of Phosphodiesterase type 4
 - Other approaches to cytokine inhibiting NCEs
- Drugs targeting the movement and activation of inflammatory leukocytes
 - Inhibitors of chemotaxis
 - Antagonists of the complement fraction 5a
 - Chemokine Antagonists
 - Drugs targeting Adhesion Molecules
 - Selectins
 - Lymphocyte Function Antigen-1 (LFA-1, CD11a) antagonists
 - Very Late Antigen-4 (VLA-4) antagonists
 - Other Adhesion Molecule Targets
- Drugs designed to specifically regulate lymphocyte activation
 - Inhibitors of lymphocyte intracellular enzymes
 - Macrolide immunosuppressants
 - Selective inhibitors of nucleotide metabolism
 - Drugs targeting cell surface receptors on lymphocytes
 - CD2

- CD3
 - CD4
 - CD20
 - CD25
 - CD40 and CD40 Ligand
 - CD80 (B7)
 - CD152 (CTLA4)
- Vaccines and desensitizing agents
- Anti-proliferative Agents for Rheumatoid Arthritis
- Protease Inhibitors
 - Matrix Metalloprotease Inhibitors
 - Elastase Inhibitors
 - Cathepsin Inhibitors
- Anti-angiogenic Agents for Rheumatoid Arthritis
 - Targets for novel anti-angiogenic molecules
 - Growth Factors
 - Cytokines
 - Chemokines
 - Matrix Metalloproteases
 - Plasminogen activators
 - Angiostatic molecules
 - Cell adhesion molecules
- Analgesics for Rheumatoid Arthritis

Specialization: **Animal health**

Subject :**Disease of livestock**

Disease: Ring Worm

Species: **Buffalo/Cattle (Specially calves)**

Source of the material: *The Merck Veterinary Manual, Eighth Edition, CD-ROM*

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Country of origin: India

Author's name

Institution:

Organization:

Content: Dermatophytosis is an infection of keratinized tissue (skin, hair, and claws) by one of the three genera of fungi collectively called dermatophytes—*iddermophyton*, *Microsporum*, and *Trichophyton*. These pathogenic fungi are found worldwide, and all domestic animals are susceptible. A few dermatophyte species are soil inhabitants (geophilic), eg, *M gypseum* and *T terrestre*, and cause disease in animals that are exposed by digging or rooting. Other species are host-adapted to man (anthropophilic), eg, *M audouinii* and *T rubrum*, and infect other animals rarely. The zoophilic species are transmitted primarily by contact with infected individuals and contaminated fomites such as furniture, grooming tools, or tack. Contact with a dermatophyte does not always result in infection. Whether infection is established depends on the fungal species and host factors, including age, immunocompetence, condition of exposed skin surfaces, host grooming behavior, and nutritional status. Infection elicits specific immunity, both humoral and cellular, that confers incomplete and short-lived resistance to subsequent infection or disease. Under most circumstances, dermatophytes grow only in keratinized tissue, and advancing infection stops on reaching living cells or inflamed tissue. Infection begins in a growing hair or in the stratum corneum, where threadlike hyphae develop from the infective arthrospores or fungal hyphal elements. Hyphae can penetrate the hair shaft and weaken it, which together with follicular inflammation, leads to patchy hair loss. As the infection matures, clusters of arthrospores develop on the outer surface of infected hair shafts. Broken hairs with associated spores are important sources for spread of the disease. As inflammation and host immunity develop, further spread of infection is inhibited, although this process may take several weeks. Thus, for most healthy adult hosts, dermatophyte infections are self-limiting. In young or debilitated animals and, to some extent, in longhaired breeds of domestic cats, infection may be persistent and widespread.

1. **Symptoms**

2. **Diagnosis**

(i) **Barnyard diagnosis**

(ii) **Laboratory diagnosis:** Dermatophytosis is diagnosed by fungal culture, examination with a Wood's lamp, and direct microscopical examination of hair or skin scale. Fungal culture is the most accurate means of diagnosis. Dermatophyte test medium (DTM) may be used in a clinical setting. Selected lesions should have the hair clipped to a length of ~0.3 cm. The area should be gently patted with an alcohol-moistened sponge and then patted dry to reduce contamination with saprophytic fungi. Hair stubble and skin scale are collected for placement on the agar, which is then lightly covered to prevent drying out. Incubation at room temperature is sufficient except when culturing for *T verrucosum* from food and fiber animals, in which case incubation at 37°C is necessary. Dermatophyte growth is usually apparent within 3-7 days but may require up to 3 wk. Dermatophytes growing on DTM cause the medium

to change to red at the time of first visible colony formation. Dermatophyte fungi have white to buff-colored, fluffy to granular mycelia. Saprophytic contaminant colonies are white or pigmented and almost never produce an initial color change on DTM. Definitive diagnosis and species identification require removal of hyphae and macroconidia from the surface of the colony with acetate tape and microscopical examination with lactophenol cotton blue stain. The Wood's lamp is useful in screening examinations for *M canis* infections in cats and dogs. Infected hairs fluoresce yellow-green; however, only 80% of *M canis* infections fluoresce, and other fungal species in animals do not. Therefore, negative Wood's lamp examinations are not meaningful. False-positive examinations may occur and are especially likely in oily, seborrheic skin conditions. Fluorescing hairs should always be cultured to confirm the diagnosis. Direct microscopical examination of hairs or skin scrapings may allow early diagnosis by demonstration of characteristic hyphae or arthrospores in the specimen. The technique is more useful in diagnosing dermatophytosis in large animals than in small animals. Hairs (preferably white ones) and scrapings from the periphery of lesions are examined for fungal elements in a wet preparation of 20% potassium hydroxide that has been gently warmed or has incubated in a humidity chamber overnight.

Treatment

- (i) **At farmer level**
- (ii) **At village level**
- (iii) **District level**
4. **Precaution**
5. **International status**
6. **Advisory - Seasonal, etc**

Specialization: **Animal health**

Subject :**Disease of livestock**

Disease: Theileriasis: East Coast Fever

Species: **Buffalo/ cattle**

Source of the material: *The Merck Veterinary Manual, Eighth Edition, CD-ROM*

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Country of origin: India

Author's name

Institution:

Organization:

Content: East Coast fever is an acute disease of cattle characterized usually by high fever, swelling of the lymph nodes, dyspnea, and high mortality. **Etiology,:** *Theileria parva* infects cattle and African buffalo (*Syncerus caffer*) but is nonpathogenic in the buffalo. Sporozoites are injected into cattle by infected vector ticks several days after attachment. Although three subtypes of *T parva* are recognized on the basis of clinical and epidemiologic parameters, they are probably not true subspecies. *Theileria parva parva*, transmitted mainly between cattle, and *T parva lawrencei*, transmitted mainly from buffalo to cattle, are both highly pathogenic. *Theileria parva bovis*, transmitted between cattle, is much less pathogenic. In some endemic areas, indigenous cattle have a degree of resistance to the virulent subtypes. Mortality in such stock is relatively low, but introduced cattle are particularly vulnerable and may suffer mortality of >90%.

An occult phase of 5-9 days follows before infected lymphocytes can be detected in Giemsa-stained smears of the local drainage lymph node. Subsequently, the number of parasitized cells increases rapidly throughout the lymphoid system, and from about day 14 onwards, cells undergoing merogony are seen. This is associated with widespread lymphocytolysis, marked lymphoid depletion, and leukopenia. Piroplasms in RBC infected by the resultant merozoites assume various forms, but typically they are small and rod-shaped or oval.

1. **Symptoms:** Clinical signs vary according to the level of challenge and range from inapparent or mild to severe and fatal. Typically, fever occurs 7-10 days after injection of parasites by infected ticks, continues throughout the course of infection, and may be >107°F (42°C).

2. **Diagnosis:**

(i) **Barnyard diagnosis**

(ii) **Laboratory diagnosis:** Lymph node swelling becomes pronounced and generalized. Lymphoblasts in Giemsa-stained biopsy smears of lymph nodes contain multinuclear schizonts. Anorexia develops, and the animal rapidly loses condition; lacrimation and nasal discharge may occur. Terminally, dyspnea is common. Just before death, a sharp fall in temperature is usual, and pulmonary exudate pours from the nostrils. Death usually occurs after 18-24 days. The most striking necropsy lesions are lymph node enlargement and massive pulmonary edema and hyperemia. Hemorrhages are common on the serosal and mucosal surfaces of many organs, sometimes together with obvious areas of necrosis in the lymph nodes and thymus. Anemia is not a major diagnostic sign (as it is in babesiosis) because there is minimal division of the parasites in RBC, and thus no massive destruction of them. Animals that recover are immune to subsequent challenge with the same strains but may be susceptible to some heterologous strains. Most recovered or immunized animals remain carriers of the infection

Treatment

(i) **At farmer level**

(ii) **At village level:** The naphthaquinone derivatives parvaquone and buparvaquone and the lactate salt of the coccidiostat halofuginone have antitheilerial activity. Immunization of cattle using an infection-and-treatment procedure is gaining acceptance. This procedure uses a cryopreserved sporozoite stabilate of the appropriate strain(s) of *Theileria* derived from infected ticks and a single dose of either long-acting oxytetracycline or buparvaquone given simultaneously, or of parvaquone given ~8 days after infection. Oxytetracycline is ineffective as a treatment when administered after infection has been established. Cattle should be immunized 3-4 wk before being turned out to infected pasture. Incidence of East Coast fever can be reduced by rigid tick control, but in many areas, this means biweekly acaricidal treatment.

(iii) **District level**

4. Precaution
5. International status
6. Advisory -Seasonal, etc

Specialization: **Animal health**

Subject :**Disease of livestock**

Disease: Tick infestation

Species: **Buffalo**

Source of the material: *The Merck Veterinary Manual, Eighth Edition, CD-ROM*

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Country of origin: India

Author's name

Institution:

Organization:

Content: Ticks are obligate ectoparasites of most types of terrestrial vertebrates virtually wherever these animals are found. Ticks are large mites and thus are arachnids, members of the subclass Acari. The ~850 described species are exclusively blood-sucking in all feeding stages. Ticks transmit a great variety of infectious agents. Some of these agents are only slightly pathogenic to livestock but may cause disease in man; others cause diseases in livestock that are of tremendous economic importance. In addition, ticks can harm their hosts directly by inducing toxicosis (eg, sweating sickness tick paralysis caused by salivary fluids and toxins), skin wounds susceptible to secondary bacterial infections and screwworm infestations, and anemia and death. International movement of animals infected with the tick-transmitted blood parasites *Theileria*, *Babesia*, *Anaplasma*, and *Cowdria* spp is widely restricted. Primary factors in the extensive distribution and prevalence of many tick species and tick-borne disease agents are movement of tick-infested livestock over great distances, and introduction of livestock to tick species and tick-borne agents that they have not previously experienced and against which they have no immunity or innate resistance. A number of introduced tick species thrive in the vast grazing and browsing environments established during recent centuries as a result of human and livestock population explosions. Two of the three families of ticks parasitize livestock: the Argasidae (argasids, "soft ticks") and the Ixodidae (ixodids, "hard ticks"). Although they share certain basic properties, argasids and ixodids differ in many structural, behavioral, physiologic, ecologic, feeding, and reproductive patterns. Tropical and subtropical species may undergo one, two, or rarely three complete life cycles annually. In temperate zones, there is often one annual cycle; in northern regions and at higher elevations in temperate regions, 2-4 yr are required by most species. There are four developmental stages: egg, larva, nymph, and adult. All larvae have three pairs of legs; all nymphs and adults, four. Adults have a distinctive genital and anal area on the ventral body surface. The foreleg tarsi of all ticks bear a unique sensory apparatus—the Haller's organ—for sensing chemical stimuli (odor), temperature, humidity, etc. Pheromones stimulate group assembly, species recognition, mating, and host selection in ticks. Certain tick species that parasitize livestock can survive several months, and occasionally a few years without food, if environmental conditions permit. Tick host preferences are usually limited to a certain genus, family, or order of vertebrates; however, certain ticks are exceptionally adaptable to a variety of hosts, so each species must be evaluated separately. The larvae and nymphs of most ixodids that parasitize livestock feed on small wildlife such as birds, rodents, small carnivores, or even lizards. In the Argasidae, the leathery dorsal surface lacks a hard plate (scutum). Male and female argasids appear to be much alike, except for the larger size of the female and differences in external genitalia. The argasid capitulum (mouthparts) arises from the anterior of the body in larvae but from the ventral body surface in nymphs and adults. In the Ixodidae, the male dorsal surface is covered by a scutum. The scutum of the ixodid female, nymph, and larva covers only the anterior half of the dorsal

surface. The ixodid capitulum arises from the anterior end of the body in each developmental stage.

Argasid Parasitism: The Argasidae are highly specialized for sheltering in protected niches or crevices in wood or rocks, or in vertebrate host nests or roosts in burrows and caves. Some argasid species are known to survive unfed for several years. Most of these leathery parasites inhabit tropical or warm temperate environments with long dry seasons. Hosts are those that either rest in large numbers near the argasid microhabitat, or return from time to time to rest there or seasonally to breed there.

Ixodid Parasitism: The Ixodidae number over 650 species (versus about 155 argasid species), occupy many more habitats and niches than argasids, and parasitize a greater number of vertebrates in a wider variety of environments. More than 600 ixodid species have a three-host life cycle; others have a two-host cycle, and a few have a one-host cycle. Each ixodid postembryonic development stage (larva, nymph, adult) feeds only once but for a period of several days. Males and females of most species that parasitize livestock mate while on the host, although some mate off the host on the ground or in burrows. Males take less food than females but remain longer on the host and may mate with several females. During inactive seasons, few or no females are found feeding, even though males are still attached to the hosts. Larval and nymphal population activity generally peaks during the “off seasons” of adults, although in some species, there is more or less of an overlap in the seasonal dynamics of immatures and adults. The ixodid males, except those in the genus *Ixodes*, become sexually mature only after beginning to feed, after which they mate with a feeding female. Only after mating does the female become replete and fully sexually mature. She then detaches, drops from the host, and over a period of a few days, deposits a single batch of numerous eggs on or near the ground, usually in crevices or under stones or debris. Depending on species and quantity of female nourishment, the egg batch usually numbers 1,000-4,000 but may be >12,000. The female dies after ovipositing. Notably, ixodids (except one- and two-host species, which use vertebrate host animals as habitat for much of their life cycle) spend $\geq 90\%$ of their lifetime off the host, a fact of utmost significance in planning control measures. The several-day feeding process progresses slowly; the balloon shape characteristic of engorged larvae, nymphs, and females develops only during the final half day of feeding and is followed by detaching. The dropping time at certain hours of the day or night is governed by a circadian rhythm closely associated with the activity cycle of the chief host. It is also vital to know whether immatures of an ixodid species feed on the same host species as do the adults, or on smaller vertebrates. Where acceptable smaller-sized hosts are scarce, immatures of some ixodid species can feed on the same livestock hosts as adults; immatures of other species seldom or never do so. The proximity of acceptable hosts, air temperature gradients, and atmospheric humidity during “resting” and questing periods are among the numerous factors that regulate the development of each stage and, in the case of females, oviposition.

Three-host Ixodids: Most ixodids have a three-host cycle. The recently hatched larvae quest for a suitable host, usually from vegetation, feed for several days, drop, and molt to nymphs, which repeat these activities and molt to adults. Of the three-host species that parasitize livestock, a few have immatures and adults that parasitize the same kind of host; these often develop tremendous population densities. The success of ixodid species that require smaller-size hosts for immatures depends on the availability of those hosts in the livestock browsing and grazing grounds. The numerous natural hazards inherent in the three-host cycle have been compensated for by the numerous benefits afforded adaptable tick species by animal husbandry practices.

Two-host Ixodids: Some ixodids, especially those that parasitize wandering mammals (and also birds in certain cases) in inclement environments of the Old World, have

developed a two-host cycle in which larvae and nymphs feed on one host, and adults on another. As in three-host species, both hosts may be different or may be the same species. Two-host parasites of livestock thrive in both inclement and clement environments and are difficult to control. This is especially true of two-host species that feed in the ears and anal areas of livestock.

One-host Ixodids: Among the most economically important ticks are several one-host species. These parasites evolved together with herbivores that wandered in extensive ranges in the tropics (*Boophilus* spp, *Dermacentor nitens*, etc) or in temperate zones (*D albipictus*, *Hyalomma scupense*). Larvae, nymphs, and adults feed on a single animal until the mated, replete females drop to the ground to oviposit.

Feeding Sites: Each species has one or more favored feeding sites on the host, although in dense infestations, other areas of the host may be used. Some feed chiefly on the head, neck, shoulders, and escutcheon; others in the ears; others around the anus and under the tail. Other common feeding sites are the axillae, Udder, male genitalia, and tail brush. Immature and adults often have different preferred feeding sites. Attachment of the large, irritating *Amblyomma* spp is regulated by a male-produced aggregation-attachment pheromone, which ensures that the ticks attach at sites least vulnerable to grooming.

1. **Symptoms:** ticks can harm their hosts directly by inducing toxicosis (eg, sweating sickness tick paralysis caused by salivary fluids and toxins), skin wounds susceptible to secondary bacterial infections and screwworm infestations, and anemia and death.

2. **Diagnosis**

(i) **Barnyard diagnosis:** Each species has one or more favored feeding sites on the host, although in dense infestations, other areas of the host may be used. Some feed chiefly on the head, neck, shoulders, and escutcheon; others in the ears; others around the anus and under the tail. Other common feeding sites are the axillae, udder, male genitalia, and tail brush. Immature and adults often have different preferred feeding sites.

(ii) **Laboratory diagnosis**

Treatment

(i) **At farmer level**

(ii) **At village level**

(iii) **District level**

4. **Precaution**

5. **International status**

6. **Advisory -Seasonal, etc**