CHAPTER 19

TICK BORNE-BACTERIAL AND VIRAL DISEASES

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INTRODUCTION

Ticks are obligatory blood-feeding parasites that belong to the order Parasitiformes and are widely distributed worldwide, especially in tropical and subtropical regions (Shaw et al. 2001). According to an estimate of total ticks known, about 10 % act as a vector (Jongejan and Uilenberg 2004). Ticks have three main families; Ixodidae, Argasidae, and Nuttalliellidae (monotypic species belonging only to South Africa), and approximately 899 tick species are included in these families (Shaw et al. 2001). The *Ixodidae* are hard ticks having scutum (hard plate) on the dorsal side and has estimated about 700 species and classified into 7 genera. The main genera are *Amblyomma*, *Dermacentor*, *Rhipicephalus, Haemaphysalis*, and *Hyalomma*, *Ixodes.* The *Argasidae* are the soft ticks without scutum and comprises of two main genera *Argas* and *Ornithodoros* and has approximately 193 species (Lagos-Quintana et al. 2001; Gondard et al. 2017). The *Nuttalliellidae* (have only one genus and species) and shares characteristics with the first two families (hard and soft).

Ticks transmit various pathogens of both medical and veterinary importance such as bacteria, rickettsiae, protozoans, helminths, spirochaetes, and viruses when they are compared to other arthropods vector groups (Yu et al. 2015). Furthermore, along with transmitting tick-borne disease, ticks are also responsible for serious allergy, irritation, abscesses, anemia, immunodeficiency, paralysis, hypersensitivity and toxic condition (Jongejan and Uilenberg 2004).

Ticks communicate with their hosts by detecting their breath, scents, body heat, and vibrations. The sites of predilection to bite animals' are ear, udder, and tail areas (Estrada-Peña, et al.,2004). In hard ticks the blood meal is long (3-10 h) depending on the growth phase and tick bites relatively occur in the day time. Conversely, in soft ticks (nymphs and adults) bites rather occur at night and only feed for a few minutes (Sun et al. 2010). They can survive for a long time without sucking blood, determined by the availability of hosts and tick species. Tick go through four development stages; eggs, larvae, nymphs, and adults (male/female). After hatching from eggs, they evolve into six-legged nymphs and then eight-legged adults (Doggett 2004).

In recent years, tick-borne zoonosis has become more common due to climatic change and the transportation or migration of tick-infested animals across the border. Other variables that contribute to the spreading of tick-borne infections include urbanization, deforestation, habitat destruction, biodiversity loss, wildlife immigration, bird migration, and other livestock-related trades that give ideal circumstances for ticks to multiply. The spread of vector-borne diseases is also influenced by variables such as biotic (host density and optimal places for tick species protection) and abiotic (habitat structure and global warming).

In the previous thirty years, the worldwide emergence of tick-borne pathogens, become an immense hazard to human health. The identification of new pathogens continuously is an increasingly globally threat of tick-borne diseases. Furthermore, the development of new molecular techniques has made reliable identification and attribution of correct phylogenetic positions to many pathogens that make tick-borne zoonoses possible (Khamesipour et al. 2018)

Tick surveillance is also becoming more important as the epidemiology changes as a result of climate and habitat, as well as increased host availability and travel of people with their companion animals. Ticks on companion animals have been the subject of investigations in several parts of Western Europe as a result of this, ticks from household pets are being monitored (Jongejan et al. 2019). In this short review of the literature, we will highlight the bacterial and viral diseases of animals that are spread by ticks.

Tick Borne Bacterial Diseases

Lyme Disease

The Lyme disease and its causative agent were reported in 1976 by Burgdorfer and his associates in the city of Old Lyme, Connecticut, USA by performing an epidemiology analysis on many children having complaints of arthritis (Stanek et al. 2012). In animals, this disease is frequently reported in dogs and horses, but its accurate diagnosis is still challenging (Bartol 2013).

Etiology

Borrelia species belongs to phylum Spirochaetes are thin, elongated, motile, gram-negative, flagellated bacteria that consist of 21 plasmids (12 linear and 9 circular), which are the

How to cite this chapter: Rehman TU, Zaman MA, Malik MI, Jawad H, Ehsan M, Rashid M, Tahir A and Shahid H, 2022. Tick borne-bacterial and viral diseases. In: Abbas RZ, Khan A, Liu P and Saleemi MK (eds), Animal Health Perspectives, Unique Scientific Publishers, Faisalabad, Pakistan, Vol. 2, pp: 148-156. https://doi.org/10.47278/book.ahp/2022.54

highest figure of plasmids in a bacterium (Stricker et al. 2005). *The B. afzelii and lusitaniae* are the predominant species in Austria and Italy respectively (Veronesi et al. 2012) (Muller et al. 2002).

Epidemiology

The black-legged tick (*Ixodes scapularis)* and *Ixodes ricinus* is the major vector of *B. burgdorferi* in the USA and Europe respectively (van der 2016). Along with rodents, birds as avian hosts transport these ticks from one area to another (Humair 2002). The ranges of geographical distributions depend on the vector adaptations (Becker et al. 2016). According to the findings of (Boseret et al. 2013) many strains of *Borrelia burgdorferi sensu lato* have been identified from songbirds' ticks from various regions globally. Canaries present relatively less spirochetemia which was reported in post-experimental infection of *B. burgdorferi* with no or mild clinical signs. So, passerines are not of great importance for *borreliosis* as long-term amplifying reservoirs (Boseret et al. 2013). Clinically healthy horses can carry *B. burgdorferi s.s.* (Chang et al. 2003), while older horses are more susceptible (Ebani et al. 2012; Funk et al. 2016). Along with vector (ticks), the specific climate conditions of an area also play role in diseases spread.

Pathophysiology

Horses may get an infection by the attached nymphs or larvae harboring the *B. burgdorferi* as a reservoir. The ticks attach with the host and the spirochetes from the mid-gut come in the salivary glands, and become part of the tick's saliva which is transferred into the host in two or three days after attachment. This bacterium protects itself from the humoral antibodies by predominant migration within connective tissues. Frequently, inoculation can happen before 2-3 days' period depending if the infected tick already has spirochetes in his salivary glands (van der 2016). In some cases, transmission of *B. burgdorferi* into the host body has been found within 18 to 24 hours of post-attachment (van der 2016). Concurrent infection through other tick-borne pathogens such as *Theileria equi* (Basile et al. 2015) and *Anaplasma phagocytophilum* (van der 2016) may also take place.

Clinical Presentation

The infected horses manifest clinical signs including low-grade fever, fatigue, reduced body weight, behavioral changes, dysphagia, lameness, arthritis, stiffness in the neck, episodic respiratory distress, anterior uveitis, cranial nerve deficits, ataxia, cardiac arrhythmias, meningoencephalitis, abortion and foal mortality. The variation of clinical signs in many infected horses has proved it as a multi-systemic disease and uveitis is the most frequent extra neural manifestation of *Borrelia* infection (van der 2016). The clinically infected horses show variations in the signs and symptoms because of co-infection with other pathogens such as *A. phagocytophylum* (Butler et al. 2005).

The incidence of tick-borne infections is more in dogs as they get more exposure to dirty places where ticks are in large numbers. The infected dogs show anorexia, lethargy, vomiting, reduced weight, generalized pain, joints swelling, fever and lameness. Occasionally, the kidneys are targeted straightly due to the formation of antigen-antibody complexes within the glomerulus which shows the chronic nature of the disease. The disease duration ranged from 2 to 730 days before death (Clark and Bidaisee 2021).

Differential Diagnoses

It is challenging because of the different clinical signs that can be associated with possible co-infection and diverse *B. burgdorferi* genospecies (van der 2016).

Diagnosis

The diagnosis is difficult in various species because *B. burgdorferi* infections are persistent devoid of any clinical signs and symptoms. The antibodies can be confirmed after 5-6 weeks of post-exposure, while the highest titers can be found after 3 months. It is preferred to culture *B. burgdorferi* from suspected horse skin biopsies (Chang et al. 2003) along with a 2-step serology protocol (ELISA or IFAT) (Butler et al. 2005). The new fluorescent bead-based multiplex assay serum analysis is an important quantitative tool for identification of the antibodies to outer surface protein antigens of *B. burgdorferi* symbolic for natural infection with and/or vaccination against the Lyme pathogen (Wagner et al. 2011). It is preferable to combine cytological assessment, antibody, and PCR testing of ocular fluids in highly suspected cases (Divers et al. 2012). Moreover, histopathology is the definitive way for the equine *borreliosis* diagnosis (Sircar et al. 2016).

The histopathology lesions of infected horses with leptomeninges include cranial neuritis, lymphohistiocytic leptomeningeal vasculitis, and peripheral radiculoneuritis with Wallerian degeneration, whereas the spirochetes are identified with the help of immunohistochemistry and Steiner silver impregnation (James et al. 2010; Imai et al. 2011).

Management & Treatment

The infected ponies are cured using the intravenous tetracycline @ 6.6mg/Kg body weight bid for weeks has proven better than per-oral use of doxycycline or sodium ceftiofur (Divers et al. 2003).

Above and beyond, the preventive measures like avoiding interactions with tick-infested areas and vigilant cleaning of the horse for early removal of all ectoparasites like ticks should be adopted. Tick infestation can be protected by several types of insecticidal sprays but many of them have not been approved for horses and also their effectiveness is undocumented up till now (Butler et al. 2005). The canines insecticidal can be used to kill ticks on horses because so far, no adverse effect has been reported (Divers et al. 2001).

Q Fever

This fever is a globally important socio-economic, public health and occupational zoonotic disease (animal searchers, slaughterers, farmers, and veterinarians) (Marmion et al. 2005; van der et al. 2011). This disease was reported by E.H. Derrick in 1937 and the term "Q fever" (query fever) was proposed to describe febrile illnesses in animal slaughter house workers in Brisbane, Queensland, Australia in the same year (Gwida 2012).

Etiology

The *Coxiella (C.) burnetii* (gram-negative) bacterium causes Q fever and is considered as a possible biological warfare source with the capability of windborne spread as well as durability in the environment (for more than 40 months) by adopting form like a spore (Dalton et al. 2014; Asamoah et al. 2020). In Ethiopia, the confirmed case of *C. burnetii* was reported in ticks collected from cattle (Philip et al. 1996).

Epidemiology

As *C. burnetii* is secreted in amniotic fluids, placental discharges, milk, feces, urine, and vaginal fluids of infected animals (Schimmer 2018). It is frequently spread in animals as well as in humans by contaminated dust inhalation, parturition or aborted placenta secretions, infected milk (unpasteurized) or meat, and wool (Salifu et al. 2019). If the environment is contaminated with parturition excretions of infected animal, then there are chances of an outbreak in that locality due to a vast spread of bacteria via contaminated dust particles (Asamoah et al. 2020). Dogs and cats are prone to *C. burnetii* infection and can spread to humans (McQuiston et al. 2002). In Ethiopia, *Coxiella* was suspected as the potential cause of abortion episodes, as it can affect all three ruminant species (Deressa et al. 2020).

Clinical Signs

This infection usually occurs in multiple species including sheep, goats, cattle, dogs, cats, rodents, birds, and other wildlife (Asamoah et al. 2020). The sheep and goat are the important causes of outbreaks in human, whereas cows are also a significant reservoir of the *Coxiella* (Rodolakis 2009). As *C. burnetii* multiplies in the trophoblasts of the allantochorion and placentomes of ruminants (Roest et al. 2012), therefore, Q fever causes reproductive issues like infertility, abortion, stillbirth, premature delivery, and weak offspring (Anderson et al. 2013; Hogerwerf et al. 2013). Generally, infected animals show anorexia, fever, rhinitis, and mild coughing (Asamoah et al. 2020).

Diagnosis

For diseases diagnosis in ruminants, enzyme-linked immunosorbent assay (ELISA) (OIE 2008) and phase-specific serology is a recommended tool to analyze the disease dynamics within herds (Bottcher et al. 2011; Sting et al. 2013). Although in dogs and cats, IFAT is recommended to detect antibodies of *C. burnetii* (Shapiro et al. 2016; Bauer et al. 2021).

Zoonosis

In recent years with an inclining rate, Q fever is reported as a re-emerging zoonotic disease in many European countries, particularly a huge number of human cases attributed to livestock have been diagnosed in the Netherlands (Roest et al. 2011; Georgiev et al. 2013). During epidemic of Q fever (2007 to 2010) 4000 human cases were diagnosed, as a result 50,000 small ruminants were culled with short-term restrictions on animal breeding. This fever causes $1 million losses annually to the Australian meat industry.

Treatment

In small ruminants, antibiotic treatments with two successive injections of oxytetracyclin (20 mg/kg body weight) have been successful (Van den et al. 2015). In many reports doxycycline and fluoroquinolones are also suggested as the most effective drug, hence in 1989 these were used together to treat acute meningitis in Q fever infection. The sulfamethoxazole-trimethoprim salts are also effective and no resistance reported so far (Alemneh and Melaku 2018).

Prevention

In the United States, human and animal vaccines are developed but not available commercially (McQuiston et al. 2002), while in Australia, whole-cell formalin-inactivated vaccine has been produced and licensed (Eldin et al. 2017). The preventive efforts should be considered by avoiding contact with infected animal excretions on the farm and try to apply maximum sanitation practices to reduce the risk of infection while dealing with parturition procedures. Serostatus of researchers, veterinarians, and farm workers should be analyzed periodically (McQuiston et al. 2002). General disinfection of farm and specially parturition pens by 10% sodium hypochlorite or 2% formaldehyde can be done (Gwida 2012).

Bovine Anaplasmosis

Etiological Agent

The highly pathogenic intra-erythrocytic rickettsia *Anaplasma (A.) marginale* causes Bovine Anaplasmosis (Pothmann et al. 2016) that mainly affects the health, reproduction, and production of infected animals (Kocan et al. 2010).

Clinical Signs

The characteristics signs of B. Anaplasmosis include fever, anorexia, decrease milk yield, depression, hemolytic anemia, mild to severe hemolysis, jaundice and abortion (Kocan et al. 2003; Kocan et al. 2010). The lactating and peri-parturient cows are more prone to infection due to associated stress and immunosuppression (Aubry and Geale 2011; Da Silva and Fonseca 2014). The recovered animals become reservoirs and spread the infection in remaining healthy herd (Aktas and Özübek 2017).

Transmission

The biological vector of *A. marginale* is ixodid tick mainly *Rhipicephalus microplus* (Al-Hosary et al. 2020). There are many other sources of transmission like the trans-placental route and direct transmission during tattooing, castration, dehorning or by infected blood transfusion (Inokuma 2007; Aubry and Geale 2011). The persistence of *A. marginale* infection is enabled by antigenic variation. The Major surface proteins (MSP) of *A. marginale* are extremely variable proteins that are responsible for the invasion of host cells (Al-Hosary et al. 2020).

Diagnosis

In acute stages of disease microscopic examination of Giemsa-stained blood smears is reliable but not for carrier or pre-

symptomatic animals (Inokuma 2007; Carelli et al. 2007) however, cross-reactions have been reported (Aktas and Özübek 2017)

Treatment

Earlier to the availability of antimicrobial (tetracycline), many preparations for example dyes, arsenic compounds, antimalarial, and antimony derivatives were implemented to treat acute anaplasmosis. However, due to less therapeutic effects, these compounds could not control mortality (Shaukat et al. 2019)

At initial infection, imidocarb dipropionate @ 5mg/Kg twice, seven days apart and tetracyclines such as oxytetracycline @22 mg/kg o.i.d for 5 days are effective. As these can only control the infection but cannot consistently eliminate *A. marginale*, the animals can become asymptomatic carriers. The extensive diversity and variability of *A. marginale* strains make it difficult to eliminate the disease (Almazan et al. 2018)

Prevention

Antigenic and genetic variations in the outer membrane proteins responsible for transmission make the prophylactic approaches less successful.

Canine Monocytic Ehrlichiosis (CME)

Canine ehrlichiosis is a fatal tick-borne disease caused by an obligate intracellular parasite, *Ehrlichia canis,* which resides and replicates within mononuclear cells (Zhu et al., 2009). At least five bacterial species are found in domestic dogs: *Ehrlichia canis, Ehrlichia chaffeensis, Ewingii, Anaplasma platys and Anaplasma phagocytophilum* (Hmoon et al. 2021).

Etiology & Epidemiology

The *Rickettsiales* group, which has been established by combining the *Rickettsiaceae* and *Anaplasmataceae* families under the genus name *Ehrlichia*, currently contains the etiological agent of ehrlichiosis. This genus presently contains 11 *Ehrlichia* species, which have been divided into three categories based on their serological, morphological, immunological, and genetic characteristics. *Ehrlichia* are small rickettsias with a round shape that can contain a wide range of pleomorphism, particularly in cell cultures, while their cell walls are identical to gram-negative bacteria. Moreover, monocytes, granulocytes, and thrombocytes are among their favorite target cells (Procajlo et al. 2011). The Giemsa staining method is preferable for identification and enables cells to become dark blue or navy blue.

All feeding stages of the brown dog tick, *Rhipicephalus sanguineus*, transmit *E. canis*, although nymphal and adult ticks can disseminate infection for at least 155 days after separating from an infected host. The *E. canis* was the first *Rickettsiales* species to be discovered in dogs and recognized in Brazil (1970s), has global distribution, particularly in tropical and subtropical regions.

Pathogenesis

The animal gets the infection after being bitten by a carrier tick *R. sanguineus*, as a result host's blood circulation is invaded and morulas are formed in blood cells. When an infected blood cell disintegrates, new elementary corpuscles attack the next blood cells, such patterns follow across the body, gaining access to the liver, spleen, bone marrow, and lymphatic nodes, where they can multiply repeatedly. The *E. canis* resides in monocytes while *E. phagocytophila* (act. *Anaplasma phagocitophilum*), and also *E. ewingii* prefer neutrophilic and acidophilic granulocytes of infected dogs.

Clinical Signs & Symptoms

Canine ehrlichiosis occur in three different clinical forms as acute, subclinical and chronic (Ristic and Holland 1993). The acute form of disease exhibits clinical sign and symptom e.g., Anorexia, high temperature, depression, emaciation, reduced weight (Iqbal et al. 1994). In sub-clinical phase the clinical signs and symptoms are not evident but still animal is suffering from diseases and exhibiting anemia, uneven leucopenia and thrombocytopenia (Greene and Harvey 1990). The chronic phase of disease is typically recognized by anorexia, fever, anorexia, emaciation, edema, epistaxis and shock finally reaching to death (Buhles et al. 1974).

Diagnosis

In laboratory examination canine ehrlichiosis is diagnosed by traditional method like hematology, serology and biochemistry parameters. In lab testing, thrombocytopenia, anemia, and leucopenia can be found in infected animal tests reports. The most reliable serological method includes plate latex agglutination and immune-fluorescent antibody test (Greene and Harvey 1990). The CME diagnosis is difficult, and it typically demands the employment of many diagnostic methods at once (Procajlo et al. 2011). Now the most recent and advanced molecular technique PCR (polymerase chain reaction) has been developed and consider as most sensitive as compared to traditional methods (Hmoon et al. 2021).

Treatment

For the management of acute CME, an antibiotic from tetracycline group for at least 28 days are indeed a preferred therapy and has a rapid recovery. The signs of acute ehrlichiosis normally diminish within 48 to 72 hours after the use of the antibiotics. Conversely, chronic ehrlichiosis, might be more difficult to cure since dogs do not react well enough to tetracycline therapy and antibiotic resistance has been reported in such cases (Procajlo et al. 2011). Tetracyclines are the most effective medications for treating ehrlichiosis, independent of the Ehrlichia species or the disease's type (Procajlo et al. 2011). The doxycycline, given orally at a dosage of 11 mg/Kg b.m. daily for at least 28 days, supplemented with imidocarb shots administered after 14 days at a rate of 3-6 mg/kg b.m is the most preferred antimicrobial drug against *E. canis* (McClure et al., 2010; Procajlo et al. 2011).

According to Price (1980), imidocarb's activity is 3 times higher than that of tetracyclines, but it has the added benefit of being effective against *Babesia canis*, which can accompany ehrlihiosis (Procajlo et al. 2011). Other than doxycyclines, oxytetracycline at 25 mg/kg b.m. every 12 hours, can be

administered. Liquids, corticosteroids, and vitamins are used as adjuvant treatments. Blood transfusions may be required in extreme conditions (Procajlo et al. 2011; Sharma et al. 2015)

Tick-Borne Viral Diseases

Approixmately10% of tick species are involved in the spread of tick-borne viruses (Kazimirova et al. 2017) and are generally known as Tiboviruses (Abubakar et al. 2018). These viruses belongs to nine tiboviruses families; among them eight are RNA families (*Flaviviridae, Reoviridae, Rhabdoviridae, Orthomyxoviridae, Nyamiviridae, Phenuiviridae, Nairoviridae*, and *Peribunyaviridae)* and one DNA family *(Asfarviridae*) (Abubakar et al. 2018). They require ticks and vertebrates as their host to complete their life cycles. Until now, about 19 tick-borne viral diseases in animals and 16 in human have been observed. The most common Tiboviruses belong to Flaviviridae family which include tick-borne encephalitis, West Nile, Louping ill, Powassan and Kyasanur Forest virus, that is transmitted by *Dermacentor reticulatus*, *Ornithodoros moubata, Ixodes ricinus, Ixodes scapularis, and Haemaphysalis punctata*, respectively. While *Asfarviridae family* is involved in the spread of the African swine fever virus (Abubakar et al. 2018).

Tick Born Encephalitis Virus

Epidemiology

Tick born encephalitis is a viral disease caused by arbovirus and transmitted via ticks i.e., *Ixodes ricinus* and *spersulcatus* in Asia and Northern Eastern Europe respectively (Leschnik et al. 2002). The mice, deer, foxes, sheep, cattle, and dogs act as a natural host (Stanek and Hofmann 1994). The disease mainly affects the nervous system (Leschnik et al. 2002) and spread depends upon the number of ticks in a specific season (Kirtz 1999).

Clinical Signs

After the virus has completed its incubation period (5-9 days), clinical signs like fever, anorexia, apathy, and neurological symptoms including (acute thalamic and cerebrocortical lesions cause alteration in behavior, consciousness, decreased tone in muscles of limbs, and seizures) start to appear. Further diffused brainstem lesions cause vestibular strabismus, nystagmus, facial paresis, and dysphagia. While meningeal inflammation leads to increased sensitivity to pain in the neck and spinal reflexes may alter due to lesions in the spinal cord (Tipold 1997; Reiner and Fischer 1998; Kirtz 1999). The hematological analysis revealed leukopenia and lymphopenia in the early stage of infection (Leschnik et al. 2002). Prognosis is good if the infected animal survives the first week of infection (Kirtz 1999).

Diagnosis and Treatment

The clinical signs and symptoms along with the history of the tick infestation are determined for the disease diagnosis. The virus identification is confirmed through various tests including PCR, ELISA, cytological, as well as serological tests of blood and cerebrospinal fluid. As no specific treatment is carried out, only supportive therapy is done (Leschnik et al. 2002).

Louping Ill Virus (Ovine Encephalomyelitis)

Etiology

The louping Ill virus caused ovine encephalomyelitis disease that is transmitted through I. *ricinus* tick in sheep. The disease is characterized by encephalitis and have high mortality rate 60% (Reid et al. 1981). However, the disease is less prevalent in cattle, but infected animal can also develop CNS signs and fatal illness (Dobler 2010).

Clinical Signs

The virus gets entry into the body of an animal through the saliva of the infected tick. After replication into lymphatic tissue, the virus comes into the bloodstream and remains there for 1-5 days. During this period, the animal may develop fever. After that, the virus enters the central nervous system of animals and starts replication. The clinical signs like muscle tremors, ataxia, nervous nibbling, and weakness start to appear and animal may collapse after 1-3 days of onset of signs (https://www.msdvetmanual.com/nervous-system/louping-ill/louping-ill-in-animals). The dogs that are guarding a sheep herd, infested with the ticks might develop CNS signs. (Mackenzie 1982).

Diagnosis and Treatment

Clinical signs along with the tick infestation help in the diagnosis of the disease. No specific treatment or vaccines are available (https://www.msdvetmanual.com/nervous-system/louping-ill/louping-ill-in-animals)

Powassan Virus

The Powassan virus caused fulminant meningo-encephalitis in horses. After the incubation period of 8 days, the animal exhibits clinical signs including frothy mouth, excessive chewing, ataxic, tremors in neck and head. Further neurological lesions like encephalomyelitis, brain necrosis, and necrotic foci in parenchyma were seen (Little et al. 1985) Goats don't show any signs however they shed virus in the milk from day 7-15 post-infection (Kokernot et al. 1969).

Diagnosis and Treatment

The diagnosis is made on the basis of clinical signs and symptoms along with the history of the tick infestation. Moreover, laboratory testing of blood and spinal fluid is recommended. There is no specific medication for the treatment of Powassan virus disease.

Kyasanur Forest Disease Virus

The disease is caused by the virus of Flaviviridae family and transmitted through hard tick (*Haemaphysalis spinigera*) is endemic to South-western India. The Blanford rat, striped forest squirrel, and house shrew are the natural acting hosts.

Clinical Signs

After the incubation period (3-8 days), the symptoms including chills, fever, muscle pain, vomiting, gastrointestinal

and bleeding problems start to appear. Animals infected with ticks can show high virus titers in their blood (Trapido et al. 1959). While other livestock and wild animals show no clinical signs (Dobler 2010).

Diagnosis and Treatment

The diagnosis can be confirmed during early stage through molecular tests (PCR) and in later stage by Elisa. As no specific treatment is carried out, only supportive therapy is done.

West Nile Virus

Etiology

The virus is present in birds without exhibiting the clinical signs, however horses may become infected and can develop a severe illness that can lead to death. (https://www.msdmanuals.com/professional/infectious-diseases/arboviruses,-arenaviridae,-and-filoviridae/west-nile-vir)

Clinical Signs

The infected animals manifest clinical signs including anorexia, stumbling, muscle twitching, paresis, hazy vision, noggin, teeth grinding, uncontrolled walking, convulsions, circling, and dysphagia. This may lead to coma and eventually death (https://www.oie.int/en/disease/west-nile-fever)

Diagnosis and Treatment

The disease is diagnosed by observing clinical signs and determining the presence of antibodies in the blood. No specific treatment is carried out, although vaccines in horses are available.

Control and Prevention of TBD

Chemical Control

Acaricides are used to control tick population at different growth phase of their life cycles. Dipping vats, sprays, pour-on and parenteral acaricides are used in chemical control. During the treatment with acaricides, taxonomy of the ticks as well as their susceptibility to that kind of treatment should be considered for effective results. Ticks can gain resistance against a particular type of acaricide which can be transferred genetically to the next generations (Almazan et al. 2018).

Immunological Control

Vaccines are an effective source for the control of TBDs. Depending upon the ticks whether they are one host ticks or multiple host ticks, vaccines are administered accordingly. This is because the immune response against the vaccination varies from host to host. This factor needs to be considered when preparing a vaccine or running its trial. Vaccines against tick-borne pathogens are more effective for the wild reservoir hosts that are transmitted by ticks from an infected host to a healthy host than those pathogens that are transmitted transovarially. Some tick-borne pathogens like bacteria and protozoans require much more time for transmission from the tick to the host during the feeding time of ticks than the viruses that are transmitted as soon as a tick bites the host. This factor should also be considered while designing a vaccine. In the case of viruses, vaccines are prepared by keeping in view tick attachment, its feeding time, and reduction in host tick-borne pathogens. Moreover, vaccines used for wild, domestic, or humans have different considerations (de la Fuente et al. 2017).

Vaccines for Domestic Animals

Vaccines like BM86/BM95 for tick infestation control in cattle are proved to be cost-effective and a reasonable approach. One host ticks like Rhipicephalus spp. which are the main vectors for bovine anaplasmosis and babesiosis, have been effectively controlled by the vaccinations. For designing a vaccine of bovine anaplasmosis, which is also transmitted by other means like biting insects and blood contaminated objects, other factors like tick pathogen infection, tick bite pathogen transmission, host tick bite pathogen should also be considered in addition to the reduction of tick infestation (de la Fuente et al. 2017). BM 86 is derived from a gut membrane protein found in the intestine of *R. microplus*. This vaccine controls the tick population by interfering with blood clotting and cell growth of the ticks. *R. microplus* feeding on cattle vaccinated with BM86 show a marked reduction in females, their weight and reproductive ability. Combined treatment with acricides and BM86 can give 100% tick control. BM95 is homologous protein to BM86, has 39 and 21 difference in nucleotide at amino acid level has 64% efficacy in cattle (Almazan et al. 2018).

Conclusion

Using acaricides for complete tick eradication seems to be impossible. Ticks can also be controlled by the safe formulations with repellency and parasiticidal activity (de la Fuente et al. 2017). Emerging pheromones base control and biological control agents like tick repellents and botanical acaricides respectively are also promising (Benelli et al. 2016). Vaccines are the safest and effective way to control tick-borne diseases, because they can induce a long-lasting immunity in the animal against tick infestation as well as pathogen infection. The vaccines derived from the combination of the tick as well as pathogen antigens seem to be more effective.

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