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Trypanosomiasis in Wild Animals of Tropical and Sub-tropical Region: A Review

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ABSTRACT

Trypanosomiasis is a frequently reported disease in wild and domestic animals and it is a major cause of death in them. It has been detected in most wild species, including deer, elephants, capybaras, jaguars, tigers, lions and other species of domestic animals commonly known as surra in tropical and sub-tropical countries. It causes tremendous economic loss due to illness, decreased productivity, reduced weight gain, decreased milk yield and productivity and death in domestic and wild animals. Early detection and effective management strategies are crucial for controlling the spread of the disease and minimizing its impact on wildlife populations. By providing comprehensive information, such a review article can serve as a valuable resource for researchers and practitioners working in the field of wildlife health and conservation. The data from several article related with trypanosomiasis in wild animals were collected between a period of 1971 to 2022. Out of several article dealing with trypanosmiasis in wild animals, seventy-five articles are included as references for the compilation of this review. The article selected for this review are dealing about various aspects of trypanosomiasis in wild animals. The academic research databases PubMed, Scopus, Web of Science, Science direct etc besides google search engine was used for the selection of various article by using key words *viz*., trypanosomiasis, prevalence, wild animals, tropical trypanosomiasis, oxidative stress, anaemia, trypanocides *etc*. The article directly or indirectly related to wildlife trypanosomiasis were selected for this review. The prevalence of trypanosomiasis were reported in various species of wild animals *viz*., Tigers, Antelopes, Vampire birds, Deer, Shamber, Elephants *etc.* Transmission of trypanosomiasis to wild animals is comparatively easier in comparison to other animals as it may get infected either by biting flies or by ingesting infected flesh. Pathogenesis of trypanosomiasis in wild animals are mostly associated with cutaneous lesions, anaemia, emaciation, neuropathy, oxidative stressand lymphadenopathy.

Key words: Epidemiology, Tiger, Trypanosomiasis, Vector, Wild animals.

Trypanosomiasis is a disease caused by parasitic protozoans of the genus Trypanosoma. It affects both humans and animals, including domestic and wild animals. It is caused by unicellular flagellar protozoa belonging to the family of Trypanosomatidae of sub-genus Trypanozoon of the Salivarian section of the genus Trypanosoma. *Trypanosoma evansi* are found in two isolates *i.e.* type A and type B, in which type B is rare in occurrence. The genus Trypanosoma comprises many species which causing diseases called trypanosomiasis in domestic and wild animals, as well as in humans (Mekata *et al.,* 2013; Wiikowsky, 2022). *Trypanosoma evansi* was the first pathogenic mammalian trypanosome described in 1880 by Griffith Evans in the blood of Indian equines and dromedaries (Hoare, 1972). This species evolved from *T. brucei* by adaptation to mechanical transmission which allowed it to spread beyond the tsetse belt in Africa, triggering a wasting form of disease in livestock and wild animals commonly known as “Surra” in Asia and Africa. Trypanosomiasis has been detected in most wild species including deer, elephants, capybaras, jaguars (Choudury *et al.,* 1972) and tigers (Manohar *et al.,* 2023). Surra causes tremendous economic loss due to illness, decreased productivity, reduced weight gain, decreased milk yield and productivity and death in domestic animals (Kumar *et al.,* 2018) and wild animals. Surra is considered as major impediment in the health and productivity of animals (Kumar *et al.,* 2012). Among wild animals, the

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surra is often fatal in captivity (Parija *et al.,* 2001) whereas it is usually reported in sub-clinical form (Mc Culloch, 1967; Evans, 1910). The sub-clinical cases in some wild animals may act as the reservoir of infection for the domestic animals. The animals do not develop immunity against the parasitic diseases, the morbidity and mortality rates in infected population is usually high. The zoonotic potential of Trypanosoma evansi has been reported in India and Vietnam (Joshie *et al.,* 2005; Powar *et al.,* 2006; Dakshinkar *et al.,* 2007; Van chau *et al.,* 2016). The World Organization for Animal Health (OIE) classified Surra as a notifiable multi-species animal disease (OIE, 2018). Given the global significance of trypanosomiasis, particularly in tropical and subtropical regions where it affects both domestic and wild animals, consolidating available information on its various aspects in wild animal species can greatly aid research efforts. Early detection and effective management strategies are crucial for controlling the spread of the disease and minimizing its impact on wildlife populations. By providing comprehensive information, such a review article can serve as a valuable resource for researchers and practitioners working in the field of wildlife health and conservation. This article describes in details about the epidemiology, pathogenesesis, clinical pathology, diagnosis, treatment and prevention and control of trypanosomiasis in wild animals.

Trypanosome species in free-living wild animals

Several pathogens including various Salivarian trypanosomes, such as *Trypanosoma brucei* sp., *Trypanosoma congolense* and *Trypanosoma vivax* (causing "Nagana" or animal African trypanosomiasis [AAT]), *Trypanosoma evansi* ("Surra") and *Trypanosoma equiperdum* ("Dourine") and *Trypanosoma cruzi* (stercorarian trypanosome) are responsible for trypanosomiasis in wild animals. Trypanosome species commonly found in wildlife species include *T. vivax*, *T. brucei*, *T. congolense* and *T. evansi.* Multiple wildlife hosts carry the human-infective zoonotic trypanosome strain *T. rhodesiense*, including bush bucks (*Tragelaphus scriptus*), impala (*Aepyceros melampus*), lion (*Panthera leo*), zebra (Equus quagga boehma), warthog (*Phacocoerus africanus*) and duiker (*Sylvicapra grimmia*) (Anderson *et al.,* 2011). *Trypanosoma conglense*, *Trypanosoma simiae* and *Trypanosoma godfreyi* were reported in Rhinocerus (Obanda *et al.,* 2011).

Epidemiology

Surra and its causative agent, *Trypanosoma evansi* are widely distributed throughout the tropical and subtropical regions of Northern Africa, Southeast Asia, as well as Central and South America (Sanchez *et al.,* 2015; Desquesnes *et al.,* 2022). Chronic trypanosomiasis infections are commonly seen in cattle and buffaloes. These animals can act as reservoirs for the parasite, which can be transmitted to other animals, including wildlife. The interaction between domestic animals, such as cattle and buffaloes and wildlife, such as tigers, can indeed create opportunities for disease transmission.

The complexity of the susceptibility of many domesticated and wild animals to surra and the possibility of involvement of some of the wild animals as silent carriers or reservoirs of infection leads to the continuous geographical spread and emergence of this disease in nature. This causes recurrent outbreaks of devastating cases of trypanosomiasis in wild mammals when they are translocated from one region to another for the strengthening of captive breeding or exhibition (IUCN, 1998).

Prevalence of trypanosomiasis in wild animals

Trypanosoma evansi may affect more than 30 species of wild animals (OIE, 2005). The disease is endemic in the Indian subcontinent and has been reported from more than 48 countries. Its major outbreaks and sporadic cases have been reported in India, Pakistan and Bangladesh. The incidence of vector-borne diseases is very common in India (Sarma *et al.,* 2019). *Trypanosoma* mostly affects a member of the family Camelidae and Equidae but have a widespread host range amongst the salivarian trypanosomes including domestic and wild animals like horses, mule, donkey, camel, cattle, buffaloes, sheep, goat, dogs, pig, elephant, chital, jungle cat, mithun, deer, foxes, tiger and jackals (Sarma *et al.,* 2012; Kumar *et al.,* 2020; Kasozi *et al.,* 2021).

Tigers

The tiger (*Panthera tigris*) is classified under the genus *Panthera* and is the largest, most iconic wildlife species among the Felidae. About 93-95 per cent of the world's tiger population has been decimated in the last 100 years. The number of tigers in the wild varies between 3,726 and 5578 worldwide as per the International Union for Conservation of Nature (IUCN) recently released red list data and if this trend continues, tigers may cease to exist. Among the various diseases affecting the tigers, trypanosomiasis is one of the important diseases that play a major role in decreasing the tiger population. Several deaths in tigers reported due to trypanosomiasis in India (Sengupta, 1974; Dasgupta *et al.,* 1979; Ziauddin *et al.,* 1992; Chaudhuri *et al.,* 1996).

Rhinoceros

The *Trypanosoma evansi* outbreak in a captive population of Sumatran rhinoceros housed in peninsular Malaysia at the Sungai Dusun Conservation Centre was reported in 2003. This epidemic showed biphasic die-offs of animals with clinical signs of anorexia, depression, inco-ordination, rear limb paralysisand recumbency. Surra was suspected in five captive Sumatran rhinoceroses (*Dicerorhinus sumatrensis sumatrensis*) in Malaysia presenting depression, anorexia, incoordination, muscle tremor, nasal haemorrhage, recumbencyand laboured breathing followed by death. During this outbreak, *Trypanosoma*

evansi was reported in 3 out of 5 animals and all them died due to clinical Surra (Khan *et al.,* 2004). *T. vivax* infection reported in black rhinoceros (*Diceros bicornis*) from Africa (Mihok *et al.,* 1992).

Vampire bats

Trypanosoma evansi was also reported in wild hosts like vampire bats, capybaras and coatis. The vampire bats act as vector and can transmit the infection among themselves, thus functioning as a reservoir (Hoare, 1965; Ayala and Well, 1974). The report on indirect evidence of *Trypanosoma evansi* in a nectar-feeding bat (*Leptonycteris curasoae*), a feeding habit that precludes direct transmission of the parasite to other animals reported (Silva-Iturriza, 2013).

Deer

In deer, several reports gathered showed acute and sub-acute infection. The mortality rate of trypanosomiasis among captive deer in Malaysia was 27% (Adrian *et al.,* 2010). In Asia, many outbreaks associated with high morbidity and mortality were reported in Timor rusa deer *(Rusa timorensis*) and hog deer (*Axis porcinus*). *Trypanosoma evansi* reported in the brain of naturally infected hog deer that ranged free on a farm in Samut Prakarn province, Thailand died after showing nervous signs (Adrian *et al.,* 2010).

Antelopes

In *Antilope cervicapra*, Surra is more chronic and shows anaemia, loss of weight and abortion. Acute signs were reported from outbreaks in Mauritius, in *Cervus unicolor*, with acute fever, rapid loss of condition, emaciation, anaemia and death. In Thailand, in *Cervus porcinus* (hog deer), nervous signs were reported with paresis, lateral recumbency, excitation, convulsion and a high mortality rate. Similarly, an outbreak in the Java deer (*Cervus timorensis*) was reported from Malaysia (Perak) with anaemia, inappetence, respiratory distress and recumbency. Besides, several other haemoparasites were pre sent together with *Trypanosoma evansi* infection (Nurulaini *et al.,* 2017).

Sambar

The occurrence of trypanosomiasis in sambar deer, both native and imported, is concerning. The reports from Maharashtra State Forest Department (Sudan *et al.,* 2017) Thailand (Indrakamha *et al.,* 1996) and Malaysia (Abdullah *et al.,* 2014) highlight the presence of *Trypanosoma evansi* in these deer species. This indicates that sambar deer, whether native or imported, can be susceptible to this parasitic infection.

Elephant

The presence of *Trypanosoma evansi* in elephants, both in India (Stephen, 1986) and Thailand (Hin-On *et al.,* 2004), indicates that these animals can be infected by this parasite with some seropositive cases. The reports from Mahendra Chaudhury Zoological Park in Punjab further support this finding, showing sero-positivity for *Trypanosoma evansi* antibodies in Indian elephants (Moudgil *et al.,* 2022).

Other wild hosts

Capybara (*Hydrochoerus hydrochaeris*), the biggest rodent in the world is a major potential reservoir (Morales *et al.,* 1976). The capybaras proved to be of low susceptibility and did not develop clinical signs (Toro *et al.,* 1980). Amongst camelids, *Lama glama* and *Lama pacos* are occasionally found to be disease-ridden and under experimental conditions *Lama guanicoe* proved to be fully receptive and susceptible to infection. The trypanosomiasis in Marsupials like omnivorous *Didelphis* sp., *Monodelphis sp. and* bats eating fruits and arthropods such as *Platyrrhinus* sp., *Carollia* sp. and *Myotis* sp. in South American coatis (*Nasuanasua*), sometimes with a prevalence as high as 16% reported (Herrera *et al.,* 2004). Wild dogs (*Canis azarae*), red howler monkeys (*Alouatta seniculus* and *A. ursina*), white tail deer (*Odocoileus virginianuschiriquensis*), brocket deer (*Mazama satorii*), wild pigs (collared peccary, *Picari tajacuand* white-lipped peccary, *Tayassupecari*), New World mouse (*Oryzomys* sp.), ocelots (*Leopardus pardalis*) Have been reported. The statement suggests that although trypanosomiasis has been reported in certain animals, such as wild pigs, mouse, deer or monkeys, their specific role in the epidemiology of the disease is not well understood. This indicates a gap in knowledge regarding how these animals contribute to the spread and maintenance of the parasite within a given ecosystem (Desquesnes, 2004).

The clinical cases and mortality due to surra were reported in Asian or Himalayan black bear (*Ursus thibetanus*), Asian elephants (*Elephas maximus*), leopards (*Panthera pardus*), tigers (*Panthera tigris*), jaguar (*Panthera onca*) and Sumatran rhinoceros (*Dicerorhinus sumatrensis*). These wild animals are considered as endangered wild species in Asia (More *et al.,* 2017). The prevalence of Trypanosoma evansi in wild ruminants and the possibility of oral transmission pose a potential threat to wild carnivores, including endangered species. Wild carnivores that prey on infected animals or scavenge their carcasses may become infected with the parasite (Herrera *et al.,* 2011; Panigrahi *et al.,* 2015). The various species of wild rodents in which the parasite was detected in Laos, Cambodia and Thailand, may play a reservoir role in the region (Cristina *et al.,* 2013). *Nasua nasua* and *H. hydrochaeris* are reservoirs of *Trypanosoma evansi* and act as a source of infection in domestic animals (Da Silva *et al.,* 2016). The capybara is a large rodent found in tropical to temperate freshwater wetlands of South America may act as source of infections (Eberhardt *et al.,* 2014). It is concerning that Trypanosoma evansi has been detected in a variety of wildlife species, including pampas deer (*Ozotocerus bezoarcticus*), marsh deer (*Blastocerus*

dichotomus), ocelots (*Leopardus pardalis*), marsupialsand rodents. This broad host range suggests that the parasite can infect a wide range of animals, potentially contributing to its persistence and transmission within ecosystems. However, except for capybaras and vampire bats, the role of the diverse wild animal species in the epidemiology of *Trypanosoma evansi* could not be ascertained (Hoare *et al.,* 1965; Toro *et al.,* 1980; Shukla *et al.,* 2001). Other wild animals like jaguars (Sinha *et al.,* 1971), leopards (Sinha *et al.,* 1971), wolves (Sengupta, 1974), jungle cat (Ziauddin *et al.,* 1992) and feral cattle (Sudan *et al.,* 2017) are reported to be suffered from trypanosomiasis.

Transmission

Trypanosoma evansi, the parasite that causes surra, is mechanically transmitted by hematophagous flies from the genera Tabanus and Stomoxys in natural conditions. The success of transmission depends on the survival of the parasites in the oral cavity of the vector. The shorter the time interval between the vector feeding on infected animals and then feeding on uninfected animals, the greater the likelihood of successful transmission. This highlights the importance of vector control measures in preventing the spread of surra. (Wiikowsky, 2022). Glossina species are the main arthropod vectors in tsetse-infested areas, while other haematophagus-biting flies enable mechanical spread, particularly in tsetse-free areas of the world (Mbaya *et al.,* 2008a). In tsetse belts of Africa, *T. brucei, Trypanosoma congolense, Trypanosoma simiaeand Trypanosoma uniforme* are commonly transmitted and cannot be spread by mechanical vectors (Aregawi *et al.,* 2019). *T. vivax* and *Trypansoma evansi* can occur within and outside tsetse fly-infested zones and transmitted mechanically (Büscher *et al.,* 2019 Trypanosomes, is mechanically transmitted by blood-sucking insects such as Tabanus, Stomoxys, Atylotusand Lyperosia. In India, transmission is predominantly by tabanid biting flies. These insects can mechanically transmit the parasite from infected animals to uninfected ones through their feeding behavior, particularly if they feed on multiple hosts in quick succession, allowing the parasites to be carried on their mouthparts or mouthparts (Veer *et al.,* 2002).

In wild animals, Trypanosoma may be transmitted in four ways which include the bite of a blood-sucking insect from a local reservoir host (pigs, cattle, sheep, goat), the bite of a blood-sucking insect from an exotic animal species in the zoo, iatrogenic transmission through the infected blood-contaminated needles and surgical instruments (Parija *et al.,* 2001) and carnivores might have been infected by ingestion of freshly cut meat from infected dead animals. Oral transmission to carnivores when feeding on fresh infected meat has been described as well (Sinha *et al.,* 1971; Raina *et al.,* 1985). The vampire bats have unique ability to act as vector and host of this disease (Gingrich *et al.,*1983). Besides, these methods of transmission, oral transmission to predatory flesh-eaters *via* abrasions in the oral mucosae caused by bone splinters of diseased prey have been reported (Baker, 1968). Vector-borne disease, African trypanosomiasis of human and animals is recently identified as disease likely to spread due to climate change but scientific data are lacking in wildlife trypanosomiasis.

Transmission of infection to carnivorous animals is comparatively easier in comparison to other animals as it may get infected either by biting flies or by ingesting infected flesh (Raina *et al.,* 1985). The oral transmission through ingestion of meat and blood of animals infected with trypanosomiasis has been considered as an important mode of transmission (Baker, 1968; Fig 1).

Pathogenesis of trypanosomiasis in wild animals

The main pathogenesis of surra may be illustrated by following clinical significance (Fig 2).

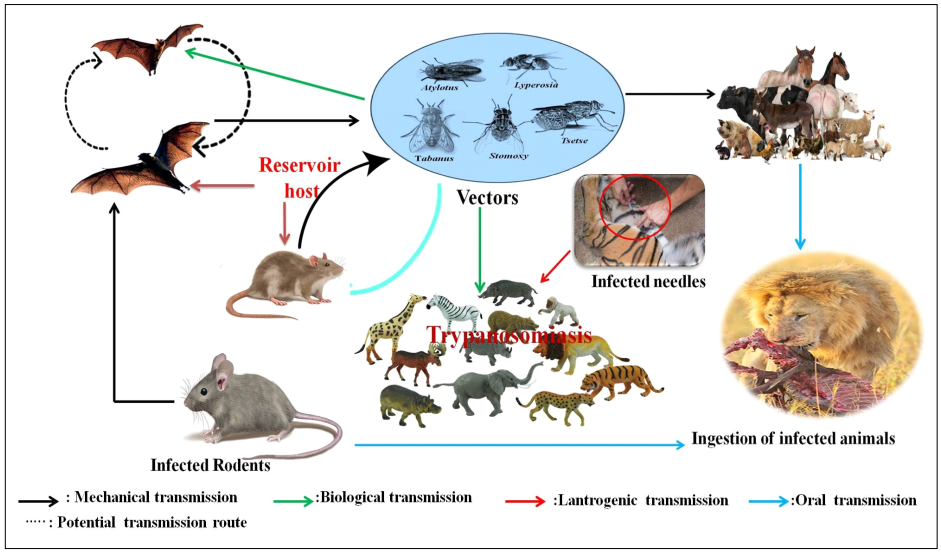

Fig 1: Illustration of transmission of trypanosomiasis in wild animals.

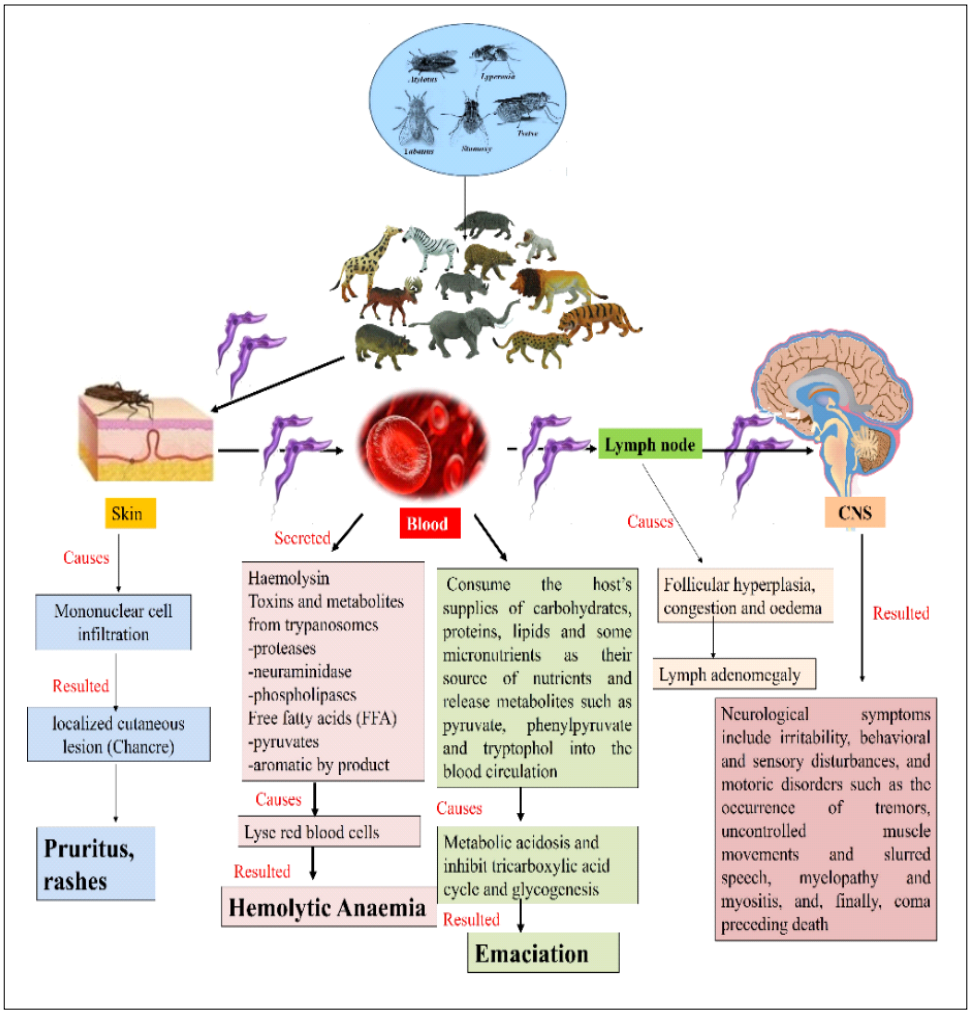

Fig 2: Illustration of pathogenesis of trypanosomiasis.

Cutaneous lesion

Extravascular trypanosomes have been known in several organs including eyes, brain, skeletal muscle, lungs, adipose tissue, heartand reproductive organs (Crilly and Mugnier, 2021). Even though, some cutaneous signs such as pruritus, rashes and skin lesions have been described in mammals carrying trypanosome infections, the existence of skin-dwelling trypanosomes remains poorly recognized in naturally infected mammals because skin infections have been unnoticed. Trypanosome infections instigate in mammalian hosts when tsetse mouthparts go in the skin to inoculate infected saliva during a blood meal. During this course, Metacyclic forms injected into the skin of a mammalian host by *Glossina* spp./ tsetse flies develop and replicate causing a regional cutaneous lesion (Chancre) which contains mononuclear cell infiltration (Dwinger, 1986).

Lymphadenopathy

The trypanosomes pass through the lymph vessels to the local lymph nodes, triggering lymph adenomegaly (Anosa and Isoun, 1983). The lymph adenomegaly is due to follicular hyperplasia, congestionand oedema. The parasite travels to all the body organs through blood circulation. In the circulation, infection is characterized by waves of parasitaemia as trypanosome populations replicate, then most of the parasites die, but the few that live begin multiplying again.

Anaemia

The pathophysiology of anaemia in trypanosomosis is multi-faceted and multi-factorial in origin. It initiates a cascade of actions leading to haemolytic anaemia and cardiovascular collapse. The haemolysin, a sensory/ excretory product of living trypanosomes is one of the several complex and multi-factorial etiologies associated with anaemia. This haemolysin is known to lyse red blood cells in the absence of antibodies (*in vitro*) and haemo-dilution (*in vivo*). The interaction of several factors acting either individually or synergistically pays important role in the development of haemolytic anaemia in animal trypanosomiasis. Anaemia is a major cause of death in haemoprotozoan diseases (Kumar *et al.,* 2022).

Neuropathy

The *T. evansi* infiltration and spreading in the central nervous system (CNS) have been defined which causes severe and potentially fatal clinical symptoms in the second stage of the disease. Trypanosomes penetrate the BBB is unidentified, but several mechanisms have been proposed *viz*., entrance through sites where the BBB is incomplete, such as sensory ganglia and circumventricular organs, deposition of immune complexes in the choroid plexus, with the resultant increase in vascular permeability (Rodrigues *et al.,* 2009) and release of toxic substances by trypanosomes that cause the opening of intercellular tight junctions in the ependymal lining of the ventricular system.

Emaciation

Trypanosomes, including *Trypanosoma evansi*, consume carbohydrates, proteins, lipidsand some micro-nutrients from the host's body fluids as their source of nutrients. This can lead to a decrease in the host's reserves of these nutrients, resulting in emaciation or weight loss in infected animals. The metabolites released by the trypanosomes, such as pyruvate, phenylpyruvateand tryptophol, can also contribute to the metabolic disturbances observed in infected animals (Igbokwe *et al.,* 1989).

Oxidative stress

Oxidative stress is a mechanism of pathogenicity in animals infected with *T. evansi*. Oxidative stress is the result of imbalances in cellular redox regulation and the inability of the antioxidant defense system to regulate reactive oxygen species (ROS). Studies conducted by (El-Deeb and Elmoslemany*,* 2015) using camels infected with *T. evansi* have suggested a correlation between increased service levels of cardiac injury biomarkers and oxidative stress. The damage to cellular proteins and membranes is a mechanism through which myocardial oxidative stress may compromise cardiac function. Increased serum malondial-dehyde and erythrocyte malondialdehyde levels in infected camels and consequently, increased service levels of cTnI, CK and CK-MB observed.

Clinical-pathology

Trypanosome infections in wild animals may display signs like those exhibited by domestic species or may succumb without showing any clinical signs. The classical signs of trypanosomiasis are intermittent fever and anaemia owing to haemolysis of red blood cells and erythron-phagocytosis, loss of appetite and weight loss condition, nervous symptoms, cachexia and death. The clinical signs of trypanosomiasis can vary widely in affected animals, with the intensity often related to the host species and the strain of the parasite. Some animals may show no apparent signs of infection, while others may exhibit severe symptoms.

Trypanosoma evansi infections lead to anaemia in tigers (Parija *et al.,* 2001; Panigrahi *et al.,* 2015) and wolves (Ziauddin *et al.,* 1992). The clinical signs include anorexia, intermittent temperature, watery eyes and pale mucous membrane, bilateral enlargement of prescapular lymph nodes, reluctant to walk, head pressing, uncontrolled movements in the body and sometimes a transitory change in consciousness, oedema of hind limbs (Shukla , 2001). The haematological (Hb, TEC, PCV, MCV and MCHC) and biochemical parameters (serum creatinine, calcium and phosphorus) levels are found to be decreased whereas TLC, serum bilirubin, ALP and AST increased in trypano-somiasis-infected tigers. Neutropenia and Lymphophilia are also observed in infected tigers (Khan *et al.,* 2004).

Postmortem finding includes mononuclear cellular infiltration in the central nervous system in elephants (Mbaya *et al.,* 2008), myocarditis in tigers, petechial haemorrhages in the liver, spleen and kidney in leopards (Chaudhri *et al.,* 1996), hepatomegaly in wolves (Shukla, 2001). The enlargement of the spleen and lymph nodes, an increase of pericardial fluid, congested liver, spleen, kidney, lungand brain were noted and they also observed, diffuse infiltration of lymphocytes, macrophages and plasma cells in the liver, spleen, kidneys, lungs, stomach, intestine and testes in tiger died of trypanosomiasis (Shukla, 2001).

Diagnosis

Diagnosis of a wild animal trypanosomiasis is a big challenge as the collection of samples required for diagnosis is not possible, however, during an outbreak of any disease samples collected from the dead carcass in postmortem examination could throw some light. Different diagnostic techniques from traditional to molecular are available for the diagnosis of trypanosomiasis. Diagnostic techniques for surra include microscopic examination, DNA detection by PCR, Card Agglutination Test and ELISA. Each diagnostic method has its advantages and disadvantages.

The parasites can be detected mainly by blood samples but sometimes other biological materials such as cerebrospinal fluid (in case of nervous signs), synovial fluid or lymph nodes are also useful. Microscopic examination of fresh blood is a common method for detecting living trypanosomes. Wet blood smears, thin blood smears and thick blood smears are commonly used techniques. Wet blood smears allow for the observation of living trypanosomes in a drop of fresh blood under a microscope, which can be useful for rapid detection. Thin blood smears are fixed and stained to improve visibility, allowing for easier identification of the parasites. Thick blood smears are used to concentrate the parasites, making them easier to detect, especially when the parasitemia (parasite concentration) is low. While these methods are relatively simple and inexpensive, they have limitations in terms of sensitivity, especially for detecting low levels of parasitemia (less than 10 trypanosomes per milliliter of blood).

In case of low parasitaemia, the buffy coat method increases the sensitivity of the test down to 100-200

trypanosomes per milliliter (Murray *et al.,* 1977). The concentration method is a low-cost substitute for direct microscopy. The mouse inoculation test is considered the most sensitive parasitological test for the detection of scanty trypanosomes. Polymerase chain reaction (PCR) with several primers specific for the sub-genus Trypanozoon or to species levels is the confirmatory molecular diagnosis for surra. This PCR-based detection of *Trypanosoma evansi* is very sensitive but not validated in the field. The antigen-antibody reactions-based tests like the Card Agglutination test and ELISA are helpful for the diagnosis of *Trypanosoma evansi*. ELISA and its antigen-detection variant are more sensitive and specific in the detection of trypanosomes. *Trypanosoma evansi* was detected only by PCR in blood samples of armadillos (*Euphractus* spp.), grey brocket (*Mazama gouazoubira*), crab-eating raccoon (*Procyon cancrivorus*) in Brazil (Herrera *et al.,* 2004). However, all these diagnostic techniques apply to the diagnosis of surra in wild animals in captivity.

Treatment

Treatment of wild animals is not a practical approach. Hence, one should only discuss about its control. However, treatment of wild animals in captivity can be discussed, but before designing a therapeutic schedule one must understand that wild animals are highly sensitive to drugs and its administration is a challenging task.

The most widely used trypanocide compound among various chemicals in infected tigers is diminazene aceturate (Upadhye and Dhoot, 2000). The other drugs like melarsomine hydrochloride (Cymelarsan) (Parija *et al.,* 2001) and quinapyramine salts (Triquin) in tigers (Dasgupta *et al.,* 1979), Wolves (Shukla, 2001), black bucks and jungle cat (Mbaya *et al.,* 2008a) are also effective against trypanosoma infected wild animals. Isometamidium hydrochloride an anti-protozoan drug has been used recently for treatment of *T. evansi* in India. The choice of anti-protozoan drugs for treatment of trypanosomiasis depends on the species of *Trypanosoma*. Drug resistance may occur and should be considered in cases refractory to treatment.

Prevention and control

T. evansi control in wild animals could only be possible by control of fly vectors and by control of surra in domestic animals which act as a source of infection for vector flies and wild animals as well. The control of its vector tabanid flies is difficult because of its high mobility and fertility and the larval stages of flies are generally spread over a wide area (Foil and Hogsette, 1994). However, control of tabanid flies in small closed deforest areas is efficient by insecticide sprays. This fly is closely related to the farming systems of livestock and it can be controlled by trap systems and insecticide sprays on animals. Many Indian Zoos are commonly practicing Integrated Pest Management (IPM) systems for vector control. Alternative measures should be designed to cope with trypanosomiasis in big cats as efficient vaccine and practical chemo-prophylactic procedures is not available. In captivity, trypano-tolerant individuals could produce through a selective breeding plan.

CONCLUSION

Our conclusion highlights the significant impact of trypanosomiasis on both wild and domestic animals, as the disease being responsible for a considerable number of deaths among infected animals. Controlling the vectors, such as hematophagous flies, is crucial for preventing the spread of trypanosomiasis, especially in wild populations where vectors play a key role in transmission. In addition to vector control, efforts should be directed towards developing efficient vaccines and chemoprophylactic measures to protect animals from infection. Furthermore, identifying and breeding trypano-tolerant or resistant wild animals can help mitigate the impact of the disease in susceptible populations. By implementing these measures, we can work towards reducing the prevalence and impact of trypanosomiasis in both wild and domestic animal populations, ultimately improving animal health and welfare.

Conflict of interest

The authors declare no competing interest.

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