Incidence of *Trypanosoma evansi* in Thrissur town

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Abstract

There were report of two cases of Trypanosomiasis in thrissur town. It is a disease complex caused by several species of blood-and tissue-dwelling protozoan parasites. It affects cattle, sheep, goats, pigs, horses, camels wild animals and man. Severity of disease varies with species and age of the animal infected and the species of trypanosome involved. Confirmation depends on demonstrating trypanosomes in stained blood smears or wet mounts. Animals can be given drugs prophylactically in areas with an incidence. Drug resistance must be carefully monitored by frequent blood examinations for trypanosomes in treated animals.

Keywords: Trypanosomiasis, Protozoa, Thrissur, animal, Cattle.

Introduction

Trypanosomiasis is a disease complex caused by several species of blood-and tissue-dwelling protozoan parasites. The disease occurs throughout the tropical regions of Africa and in large areas of Asia and South America. It affects cattle, sheep, goats, pigs, horses, camels and man. Wild animals can also be infected with the parasites but generally do not suffer from disease. They are the source (reservoir) of infection for domestic animals

Case1

There is a private cattle farm in Thrissur housing about 30 animals, (25 buffaloes and 5 cows). The owners are milk vendors. The system of rearing is such that animals in advanced pregnancy are sent to Pollachi and are brought back after calving and reared in sheds facing each other. The dung pit is in the middle of the farm. In the months of June-July, there was disease incidence in this farm, with symptoms simulating e-fever. The symptoms shown include pyrexia 105 to 106°F, enlargement of superficial lymph node, anaemia, progressive weakness, the animals were showing anorexia, off feed, lachrymation, salivation, difficulty in bearing weight, intermittent diarrhoea, respiratory distress and drop in milk yield. The animals were treated with antibiotics, antipyretics, Inj Berenil and liver tonics. Though the animals responded to treatment, the symptoms reappeared once the treatment was discontinued. Around 40% of the animals in the farm were affected one after the other. The total milk production had reduced to less than half over a period of 15 days.

Wet film examination did not reveal any motile blood parasite. A blood smear was forwarded to Department of Parasitology, College of Veterinary and Animal Sciences, Mannuthy for further investigation, and the reports revealed the presence of *Trypanosoma evansi* (+++). All the animals were treated with Triquin (Quinapyramine Sulphate and chloride) @ 4mg/kg body weight s/c. The unaffected animals were given the medication at the same dosage as a prophylactic measure. The affected animals started responding to treatment the very next day itself, the temperature had dropped to normal, the animals started feeding normally and within ten days, the pre-incidence milk yield was reattained.

The disease had started in the rainy season in which the population of flies and mosquitoes is comparatively higher. The unhygienic dung pit in the middle of the farm, probably would have served as the breeding ground for mosquitoes and other biting flies. The owners were advised to clean the dung pit immediately and use insecticides to control flies. A screening was carried out after 1 month, and all the animals were found to be free from the parasite. This was repeated for 2 more months to ascertain any reoccurrence.

Case 2

There is also another incidence of trypanosomiasis in a cattle brought for slaughter. The cow, a Jersey cross-bred aged 6 years, was brought to the Thrissur Corporation slaughterhouse, Kuriachira on 16/06/07 by a pick-up vehicle at

around 2 p.m.The animal appeared to be in reasonably good condition. Within half an hour of unloading, the animal showed signs of restlessness, collapsed and died.

External signs

The carcass was bloated. There was bleeding from the nostrils and the rectum. There were no wounds on the surface of the carcass or any marks suggestive of snake-bite. Blood smears taken from the ear tip were collected and sent to the College of Veterinary and Animal Sciences, Mannuthy -No organisms suggestive of *Bacillus anthracis* could be found and hence the carcass was declared free of Anthrax. The sample was also negative for Pasteurella multocida organisms.Blood smears were also examined at the District Veterinary Centre, Thiruvambady, where numerous long, slender organisms having a free flagellum, suggestive of Trypanosoma evansi, could be seen. The stain used was Giemsa.

Pathogenesis of Trypanosomosis

Anaemia is the principal pathological change seen, which is attributable to the extravascular destruction of RBC's. In cattle, by the second week of infection, there is a sharp drop in RBC and haemoglobin levels and anemia becomes predominant. Concurrent thrombocytopenia of moderated degree may occur. RBC lifespan is reduced to half or less.

There are many theories for the cause of death of the animal:

1. Sugar consumption theory (Shern, K.,1925) - Sugar consumption by *Trypanosomes* might reduce blood sugar too rapidly for the liver to maintain the normal level, leading to a breakdown of its function and fatal intoxication.
2. Andrews, J.C. and Johnson, C.M. suggested that death was due to asphyxia caused by pulmonary oedema following partial obstruction of capillaries by agglutinating trypanosomes.
3. Trypanosomal toxins have also been suggested.
4. Respiratory distress due to elevated blood lactate levels

Transmission

By biting insects: The process is purely mechanical. A biting insect pass the blood forms from an infected animal to another in the course of interrupted feeding. The time between the two feeds is crucial for effective transmission because the trypanosomes die when the blood dries. The importance of this mode of transmission is variable from place to place, depending on the numbers of hosts and biting insects present, and also on the species of trypanosome. Large biting insects such as tabanids carry more blood and are more likely to act as mechanical vectors than for example mosquitoes.

Clinical Findings and Lesions

Severity of disease varies with species and age of the animal infected and the species of trypanosome involved. The incubation period is usually 1-4 wk. The clinical signs of surra, are indicative but are not sufficiently pathognomonic and diagnosis must be confirmed by laboratory methods. The disease may occur in peracute, acute or in chronic form. There is a transient rise in the body temperature, enlargement of superficial lymph glands up to 2-4 times, emaciation, progressive weakness, diarrhoea, anorexia and anemia. A sudden drop in milk yield is also evident. Oedema, particularly of the lower parts of the body, urticarial plaques and petechial haemorrhages of the serous membranes are often observed. Abortions have been reported in buffaloes. There are indications that the disease causes immunodeficiency. Cattle usually have a chronic course with high mortality, especially if there is poor nutrition or other stress factors. Ruminants may gradually recover if the number of infected tsetse flies is low; however, stress results in relapse.

Necropsy findings vary and are nonspecific. In acute, fatal cases, extensive petechiation of the serosal membranes, especially in the peritoneal cavity, may occur. Also, the lymph nodes and spleen are usually swollen. Ulceration of the tongue and gastric mucosa may be seen as in the second case report. Congestion of bone marrow, blood vessels on the surface of stomach andintestines are also present. In subacute and chronic cases, swollenlymph nodes, serous atrophy of fat, and anemia subcutaneous oedema,excessive fluid in abdominal cavities, emaciation, petechial haemorrhage and an enlarged liver are present.

Diagnosis

A presumptive diagnosis is based on finding an anemic animal in poor condition. Confirmation depends on demonstrating trypanosomes in stained blood smears or wet mounts. The most

sensitive rapid method is to examine a wet mount of the buffy coat area of a PCV tube after centrifugation. Other infections that cause anemia and weight loss, such as Babesiosis, Anaplasmosis, and Theileriosis, should be ruled out by examining a stained blood smear. Lymph node biopsies,Mercuric chloride test ,Stilbamidine test) etc. are also useful. Various serologic tests measure antibody to trypanosomes, but their use is more suitable for herd and area screening than for individual diagnosis. Indirect fluorescent antibody (IFA) test, Indirect enzyme-linked immunosorbent assay (ELISA), Polymerase chain reaction (PCR). Tests for detection of circulating trypanosome species-specific antigens in peripheral blood are becoming available for both individual and herd diagnosis, although their reliability remains unproven. Treatment and Control Several drugs can be used for treatment Most have a narrow therapeutic index, which makes administration of the correct dose essential. Drug resistance occurs and should be considered in refractory cases. Drug Dosage Route Remarks Suramin 10 mg/kg (1 ml/10 kg) IV Mainly used against T. evansi Diminazene aceturate 3.5-7 mg/kg (1-2 ml/20 kg) IM Mainly used in cattle and small ruminants Quinapyramine methyl sulphate 5 mg/kg (1 ml/20 kg) S/c Now mainly used against *T. evansi* and *T. brucei* Isometamidium chloride 0.25-0.5 mg/kg IM Used mainly in cattle, as a curative at lower rates, as a prophylactic at higher rates.

Control Control can be exercised at several levels, including eradication of biting flies and use of prophylactic drugs. Flies can be partially controlled by frequent spraying and dipping of animals, spraying of insecticides on fly-breeding areas, use of insecticide-impregnated screens, bush clearing, and other methods. Animals can be given drugs prophylactically in areas with an incidence. Drug resistance must be carefully monitored by frequent blood examinations for trypanosomes in treated animals.

Future prospects Control of *T. evansi* is likely to remain heavily dependent on chemotherapeutic intervention principally with Suramin The short to medium-term outlook for this approach, however, is now uncertain as commercial production of suramin has ceased, therefore, future disease control will depend on the availability of new trypanocidal drugs. At present there is very little information on vector ecology that could be used as an alternative or part of integrated control regimes, although there is ample evidence that link disease outbreaks with conditions that lead to high population densities of (potential) vectors.

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