Canine Ehrlichiosis

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Ehrlichiosis is an infectious rickettsial disease characterised by reduction in cellular blood elements. Under the genus *Ehrlichia*, various species affecting domestic animals and man have been identified. Canine ehrlich iosis is caused by *E.canis*, *E.equi*, *E.ewingii* and *E.platys*. *E.canis* mostly affects monocytes and lympho cytes. *E.equi* and *E. ewingii* affects neutrophils and *E.platys* affects platelets. The disease produced by *Ehrlichia* in canines is also called as canine typhus, tropical pancytopenia, Nairobi bleeding disease, tracker dog disease and idiopathic haemorrhagic syndrome.

Canine ehrlichiosis is a tick borne disease and the causative organism is located intra-cytoplasmically within a plasma-lined membrane in leucocytes. The disease is sporadic in nature and is noticed in almost all age groups and male dogs are affected 1.5 times more than the females. Donatien and Lestoquard at the Pasteur Institute in Algeria first reported the disease in 1935. In India, it was first reported in 1944 by Mudaliar in Madras city. At present, the disease is very much prevalent in India. Case fatality rate is low and most reported mortality is in association with inter-current diseases. Even though the disease occurs in all breeds of dogs, German Shepherds are most susceptible. *E.canis* infections have been reported in dogs with concurrent infections with *Babesia canis* and *Hepatozoon canis* suggesting simultaneous transmission of organisms from vector ticks.

Canine infections occur when salivary secretions from the tick contaminate the attachment site during ingestion of blood meal. *E.canis* can be introduced in susceptible dogs by blood transfusion. Ticks are thought to be the primary reservoirs and adult brown dog tick, *Rhiphicephalus sanguineus* is capable of transmitting *E.canis* transtadially for at least 155 days following detachment from the host. After an incubation period of 8 to 20 days, the infected dog enters into acute phase of ehrlichiosis, which lasts for 2 to 4 weeks. During this time, the organism multiplies within the circulating mononuclear cells and mononuclear phagocytic tissues of the liver, spleen and lymph node and causes lymphadenomegaly and lymphoreticular hyperplasia of liver and spleen. Infected cells are transported via blood to other body organs especially lungs, kidneys and meninges. Infected cells adhere to vascular endothelium producing vasculitis and subendothelial tissue infection. Platelet sequestration induced by a platelet migration inhibition factor is considered to be the cause of thrombocytopenia and probably haemorrhage. Experimentally, it is found that dogs with adequate immunocompetence are reported to eliminate the parasite and do not develop the chronic phase.

Acute phase is characterised by depression, anorexia, fever, weight loss, occular and nasal discharges, dyspnoea, lymphadenopathy, oedema of the limbs and scrotum. Thrombocytopenia and leucopenia occur 10 to 20 days following infection. Central nervous system signs like hyperaesthesia, muscle twitching etc. as a result of inflammation of the meninges are also noticed. Epistaxis is considered to be the hallmark of the disease and it occurs infrequently. Chronic signs include abdominal tenderness, anterior uveitis, retinal haemorrhage etc. Secondary bacterial infections are also noticed. Affected puppies may remain as carriers for long time. *E.canis* also affects both wild and domestic animals and

symptomless infection can be produced in jackals and Macaca monkeys.

Neutrophilic morulae appear in infection with *E.equi* and E. *ewingii*. *E.equi* has a broad host range which includes horses, dog, cats and non-human primates. Clinical signs are muscular stiffness, lameness, stilted gait and arched back posture. Polyarthritis accompanied by fever is the most characteristic symptom. Haematologic abnormality same as that of *E.canis*. Because of serologic cross-reactivity, dogs affected with *E.ewingii* develop humoral antibody response to *E.canis* antigen.

Canine cyclic thrombocytopenia is caused by *E.platys* which replicates only in platelets. Co-infection with *E.canis* and E.platys is common and the latter can intensity the clinical course of monocytic ehrlichiosis.

The diagnosis of acute ehrlichiosis is by microscopic detection of organism in Giemsa stained blood smears or buffy coat preparation. The morular rosettes takes a light blue or lilac tint in the cytoplasm of leucocytes. There will be increase in serum Alanine Amino Transferase (AAT) and Alkaline Phosphate (AP) and total bilirubin. Ehrlichia are fragile organisms and do not remain viable in defibrinated blood at room temperature for longer than 48 hours. *E.canis* can be propagated in canine macrophage cell line ($DH8_2$) and less successfully in Murine Macrophage Cell line (P_388D_1). Indirect fluorescent antibody test is the highly sensitive and specific test for detecting Ehrlichia. Elisa and western blot assays can also be used for the purpose. Diagnosis is capricious because the period of parasitemia is generally short and may precede the peak of clinical signs.

Regarding treatment, tetracyclines are the most effective drug for the treatment of *E.canis* and other ehrlichial infections of dogs. The commonly used drugs with dose and details of administration are given below.

Drug	Dose	Route	Remarks
Tetracycline	22 mg/ kg body wt.	I/V, oral	At 8 hr interval for 14 days
Doxycycline	5-10 mg/ kg body wt	I/V, oral	At 12-24 hr interval for 7-10 days
Imidocarb	5 mg / kg body wt	Single I/M inj.	Repeated in 2-3 wks
Chloramphenicol	15-20 mg/ kg body wt.	I/V, S/C, oral	At 8 hr interval for 14 days

Prognosis is generally good. Dramatic clinical improvement occurs within 24 to 48 hours after initiation of tetracycline administration. Supportive therapy includes fluids, blood transfusion, vitamins and steroids. Dogs with pancytopenia, characteristic of chronic disease, have guarded prognosis and show gradual / no improvement.

Regarding control tetracyclines at low dosage of 6.6 mg / kg body weight is the only therapeutic preventive measure. This can be tried in dogs travelling in enzootic areas. Tick control can be encouraged by the use of acaricides, but does not provide an effective means of preventing the disease. Vaccine is not currently available for *E.canis*. Infection does not confer protective immunity and so subsequent exposure to infected ticks after treatment will result in disease condition.

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the defect causes production of defective hormones or enzymes biochemical profiling can help in the detection.

Another potential option for future could be use of molecular markers for genome screening of suspect parents and progeny to diagnose predisposition to the anomaly at birth itself. Although great strides in genetic research have been made researchers are yet to map the location of genes causing the anomalies to the specific canine chromosomes. Thus elimination of genetic anomalies is possible only by the shared concern of conscientious purebred breeders, veterinarians and researchers for genetic health of dogs.

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