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Canine ehrlichiosis: A comprehensive review

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Abstract
Canine vector borne diseases are group of globally distributed and rapidly spreading illnesses which are generally caused by a wide range of infectious pathogens and transmitted by variety of vectors (Otranto *et al.*, 2009) [19].
Vector is a living agent that carries and transmit infectious pathogen from one living organism into another living organism. Vectors can transmit the infections from animals to humans or humans to animals, so vectors are also responsible for the transmission of many zoonotic diseases.
Canine Monocytic Ehrlichiosis (CME) is one of the important and globally significant vector-borne diseases of animals of family *canidae*. It is tick borne and usually caused by intracellular bacterium (rickettsial organism) *Ehrlichia canis* and transmitted by the brown dog tick (*Rhiphicephalus sanguineus*). There are many synonyms of the disease i.e. Canine rickettsiosis, canine hemorrhagic fever, canine typhus, tracker dog disease, tropical canine pancytopenia.
This overview emphasize on current knowledge on the Etiology, clinical manifestations, diagnostics, and evolving treatment challenges associated with the disease.

Keywords: Canine monocytic ehrlichiosis, *Ehrlichia canis*, vector-borne diseases, *Rhipicephalus sanguineus*, canine health

Introduction
The disease is primarily caused by intracellular bacterium (rickettsial organism) *Ehrlichia canis* and primarily transmitted by the brown dog tick (*Rhiphicephalus sanguineus*).
Ehrlichia canis was the first species recognized to infect dogs and is the principal cause of Canine Monocytic Ehrlichiosis (CME) (Sainz *et al.*, 2015) [21]. *Ehrlichia ewingii*, is the cause of canine granulocytic Ehrlichiosis (Nair *et al.*, 2016) [17]. *Ehrlichia chaffeensis*, which is indistinguishable from *E. canis* and the cause of Human Monocytic Ehrlichiosis, has also been identified as an infrequent cause of clinical disease in the dog. Lone star tick (*Amblyomma americanum*) has been found as vectors of *Ehrlichia ewingii* and *Ehrlichia chaffeensis.*
The organisms are considered as leukocytophillic bacteria and they multiply within cytoplasmic vacuoles of circulating monocytes and tissue macrophages. Organisms are gram-negative, obligatory intracellular and appears as small clusters of cocci within mononuclear cells (Sainz *et al.*, 2015, Chakrabarti A., 2017) [3, 21].
Risk factors includes History of tick infestation, living in endemic regions, and lack of preventive measures for ectoparasites. German shepherds are reportedly more predisposed to severe, chronic form of the disease.

Pathogenesis
Upon transmission via a tick bite, the organism enter the bloodstream and infect circulating leucocytes. The organism has imcubation period of 8-20 days. *Ehrlichia* organism has mainly three life stages i.e. initial body, elementary body and morulae. The infection is generally begins with the initial body which is extracellular and after getting uptake by the cells through endocytosis, it transforms into elementary body and subsequently clusters into the morulae. This morulae leaves the host cell and spread the infection in the other cells and organs of the animal body and produces the pathological signs in different cells and organs.
After an incubation period of 8-20 days, the course of *E. canis* infection, can be sequentially divided into acute (2-4 weeks), subclinical (several months to years) and chronic phases (Harrus *et al.*, 2012) [7], but the distinction among these phases is not straightforward in the naturally-occurring disease. After 8-20 days following the entry of organism acute phase starts where in the organism multiply within the mononuclear leukocytes of the blood and

thus they spread in the liver, spleen and lymph-nodes. Following the acute phase, sub-clinical phase will set in when the animal appears to be healthy and if the immunity is strong enough then the animal throw the organisms. Otherwise chronic phase develops. The severe chronic form of the disease is known as tropical canine pancytopenia. There is impairment in the production of bood cells. (Chakrabarti A., 2017) [3].

Clinical findings

Table 1: Clinically, the disease can be seen into 3 phases:

Phase	Duration	Key clinical features
Acute	1-4 weeks	Fever, lethargy, anorexia, lymphadenopathy and mild thrombocytopenia
Sub-clinical	Months to years	Asymptomatic, the parasite persists in the spleen. Low platelets may be present in bloodwork.
Chronic	Persistent	Pancytopenia (reduction in all blood cells), severe weight loss, epistaxis (nose bleeding), uveitis and neurological changes.

Tick infestation may be seen, especially in the acute phase of the disease, while ulcerative stomatitis and necrotic glossitis, hind limb and/or scrotal edema, bacterial pyoderma, icterus and central nervous system signs such as seizures, ataxia, vestibular dysfunction and cervical pain, have been more frequently reported in chronic CME (Mylonakis *et al.*, 2004) [14].

Bleeding diathesis also more common and severe in the chronic phase of CME. It is manifested typically as cutaneous and mucosal petechiae and ecchymoses, hyphema, epistaxis, hematuria, melena, prolonged bleeding from venipuncture sites or intraoperative bleeding. (Mylonakis *et al.*, 2004) [14].

Neuromuscular signs generally exhibited as central nervous system symptoms i.e. ataxia, seizures, paresis, twitching, hyperaesthesia etc are caused due to meningitis or meningeal bleeding (Sykes J. E., 2017) [23].

Ocular lesions are commonly seen in CME, and may be the sole presenting complaint. Anterior or posterior uveitis is the most prevalent manifestation. Ocular discharge, blepharitis, conjunctivitis, corneal ulceration, painful necrotic scleritis, secondary glaucoma and retinal hemorrhage and/or detachment leading to blindness have also been (Leiva *et al.*, 2005) [12].

During sub-clinical phase of disease, there is no or very mild evident clinical signs may be present those also may go unnoticed (Fourie *et al.*, 2015) [4]. Organisms may sequester within the spleen and evade the host immune systems through antigenic variation (Mavromatis *et al.*, 2006) [11].

Clinical pathology

Moderate to severe thrombocytopenia is considered as most consistent hematological finding during the any phase of CME.

During the acute phase of the disease there will be significant thrombocytopenia where Platelet counts will be as low as 20,000 to 52,000/µl. Mild anemia and mildly reduced white blood cell counts also may be found during the acute phase of the disease.

During the sub-clinical phase, there will be mild thrombocytopenia and mild reduction in RBCs and WBCs count. (Harrus *et al.*, 2011) [8].

Marked pancytopenia is considered as hallmark of the chronic phase of the disease (Harrus *et al.*, 2011) [8] and it occurs due to following reasons:

1. Increased consumption: Inflammatory changes in the blood vessels.
2. Splenic sequestration: Trapping of platelets in the enlarged spleen
3. Immune mediated destruction of platelets

Severe chronic phase of the CME is also known as myelosuppressive CME, where there will be severe bone marrow aplasia and severe pancytopenia, septicaemia due to secondary infections, weakness due to anemia and severe hemorrhagic tendencies due to thrombocytopenia (Harrus *et al.*, 1996) [5]. In the chronic stage, bone marrow hypoplasia often leads to non-regenerative anemia and leukopenia, resulting in guarded prognosis.

Liver disease in CME may be primary or secondary to hypoxia, intrahepatic hemorrhage, or septicemia in the myelosuppressive CME (Mylonakis *et al.*, 2017) [16].

Hyperproteinemia, hyperglobulinemia, hypoalbuminemia and mildly elevated alkaline phosphatase and alanin aminotransferase activities are common biochemical abnormalities in CME. Creatinine concentration is elevated in some dogs while glomerular proteinuria may be present, attributable to glomerulonephritis with or without immune-complexes deposition in the chronic and acute CME, respectively (Mylonakis *et al.*, 2010) [15].

Diagnosis

The diagnosis of CME can be achieved by a combination of clinical findings, hematology and serological tests and molecular techniques (Nakaghi *et al.*, 2008) [18].

Currently the diagnosis of CME is based on following methods:

1. **Anamnesis and Clinical findings:** History of tick infestation and clinical findings are very important for the assumption of the diagnosis of CME.
2. **Laboratory investigation:** Hemato-biochemical analysis is very important for the presumption of the disease (Kottadamane *et al.*, 2017) [10].
3. **Cytology:** Demonstration of ehrlichia morulae can be done in monocytes, macrophages and lymphocytes in giemsa stained blood smear or romanowsky-type stained buffy coat smears. Less frequently smears made from other aspirates of other tissues i.e. lymph node, BM, spleen, liver and cerebrospinal fluid, is helpful in establishing a definitive diagnosis of CME (Mylonakis *et al.*, 2003) [13]. Cytology has very low sensitivity for the diagnosis of CME. Intracytoplasmic *Ehrlichia* organism were found only in blood smear of one dog out of 40 samples (Parmar *et al.*, 2013) [20].
4. **Serological findings:** Indirect fluorescent antibody assay (IFA) is most widely uses technique for the diagnosis of and considered as gold standard technique after its development in 1972 (Belanger *et al.*, 2002) [1]. Another serological technique that may be useful for the detection of IgG antibodies against *ehrlichia* is ELISA (Harrus *et al.*, 2002) [6].

5. **Molecular techniques:** Polymerase chain reaction is mostly used molecular diagnostic technique and it overcome the serological techniques and cytology as it is highly sensitive and enable the detection of *Ehrlichia* DNA as early as 4 -10 days post-infection prior to sero-conversion or development of antibodies against *Ehrlicia*.
6. **Rapid test:** Snap 4Dx plus kit is a new method of diagnosis for CME. It can be used as pet side ELISA as it can be used in any clinic or at field condition as it works on the basic principle of ELISA technique. It is fast in providing results and has higher sensitivity almost up to 97.5% and higher specificity (Stillmann *et al.*, 2014) [22].

Treatment

Doxycycline, a semi synthetic tetracycline is considered as the first line choice of drug for the treatment of CME. It has bacteriostatic activity and acts by inhibiting the protein synthesis of the bacterial ribosomes and altering the permeability of the cytoplasmic membrane of the organisms. The dose recommendation are 5 mg/kg, PO, bid, at least for 21-28 days (Mylonakis *et al.*, 2017) [16].

Tetracyclines i.e. oxytetracycline has also found yield spectacular result and it can be given IV route @ 5-10 mg/kg, for 5-7 days or 22 mg/kg, PO, for at least 21-28 days. Dogs with uremic condition should not b given oxytetracycline since it is a nephrotoxic drug but in that case doxycycline can be used (Chakrabarti A., 2017) [3].

An anti-protozoal drug imidocarb dipropionate is suggested for the treatment of CME @ 5-7 mg/kg, two doses, two weeks apart (Chakrabarti A., 2017) [3].

Supportive therapy that should be given are glucocorticoids @ 1-2 mg.kg, daily for a week, balanced crystalloid solution should be given intravenously. *Carica papaya* leaf extract has a good activity in the thrombocytopenic patients (Gowda *et al.*, 2015). Pancytopenic patients may need whole blood transfusion and aggressive antibiotic therapy for prevention or control of secondary infections (Mylonakis *et al.*, 2010) [15]. Platelets reach plasma transfusion, shown a clinical improvement in a patient that had no recovery from chronic CME and no response to the routine therapy. Platelet count shown increase in 24 hours of transfusion (Vignesh *et al.*, 2014) [24].

Prevention and control

After recovery from the disease, animal can't develop immunity against it and it may be re-infected (Breitschwerdt *et al.*, 1998) [2]. There is still no vaccines are available so prevention of disease must be done by use of proper acaricidal preparations at a proper time (Jongejan *et al.*, 2016) [9].

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