THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
Popular article
June, 2026 Vol.6(6), 1772-1775

Bovine Anaplasmosis

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doi.org/10.5281/ScienceWorld.20992772

Abstract

Bovine anaplasmosis, caused by the rickettsial organism *Anaplasma marginale*, is a globally distributed tick-borne disease of cattle with significant economic importance. The disease is characterized by severe anemia, reduced growth rates, weight loss, and, in severe cases, death, all of which adversely affect animal welfare and impose substantial financial losses on cattle producers. Although highly sensitive and specific diagnostic tools, such as competitive ELISA for detecting antibodies and real-time PCR for identifying *A. marginale* DNA, are available, interpreting these test results in the context of clinical disease remains challenging. Current treatment and control strategies rely primarily on the use of oral and/or injectable tetracyclines. However, increasing concerns over antimicrobial use in livestock raise questions about the long-term sustainability of this approach.

Key Words: *Anaplasma marginale*, anaplasmosis, cattle, rickettsia, tick-borne disease

INTRODUCTION

Anaplasmosis is an infectious, non-contagious, tick-borne disease which is caused by obligate intraerythrocytic parasites of the order Rickettsiales, genus Anaplasma (Radostitis et al., 2000) and occurs in tropical and subtropical regions worldwide. Different species causing anaplasmosis includes *A. marginale*, *A. centrale*, *A. bovis* etc. The disease is transmitted mainly by tick vectors and mechanically by contaminated needles or dehorning or other surgical instruments. There is a strong correlation between age of cattle and severity of disease

as adults are more susceptible to the infection (Radostitis et al., 2000). Bovine anaplasmosis is endemic in regions of Asia and Africa. Hasanain et al. (2015) revealed that there were 72.9% persistently infected cases or chronic carriers and 27.1% were acutely infected cases of cattle during their study.

CLINICAL SIGNS

The disease is characterized by haemolytic anemia associated with fever, weight loss, abortion, decreased milk production and in some cases death of the infected cattle (Palmer et al., 1989).

GROSS LESIONS

Gross lesions like pale mucus membranes and icteric serosal surfaces of abdominal organs with emaciated carcass were observed. Splenomegaly along with congestion and discoloration of spleen was seen. Splenomegaly may be due to excessive destruction of erythrocytes by phagocytic cells. In anaplasmosis, anaemia develops due to extensive erythrophagocytosis initiated by parasite-induced damage of red blood cells and anti-erythrocytic antibodies (DeVos et al., 2006). Lungs showed pulmonary congestion, haemorrhage, oedema and emphysema. Hepatomegaly with thickening of bile duct and gall bladder was distended with dark colored bile. Jaundice and anemia in anaplasmosis may be due to the destruction of the blood cells and their contents being released into the blood stream (Egbe-Nwiyi et al., 1997).

HISTOPATHOLOGICAL LESIONS

Microscopically, lungs showed variable extents of interstitial pneumonia, marked thickening of inter alveolar septa characterized by proliferation of type-I pneumocytes and mild infiltration of mononuclear cells. The emphysema observed may be compensatory to terminal dyspnoea. The heart revealed disorientation of myocardial fibres. Mild to moderate multifocal myocardial degeneration and coagulative necrosis was seen which may be due to severe anaemia (Hasanain et al., 2015). The spleen showed lymphoid depletion and widespread erythrocytes as evident by increased haemosiderosis. Kidney revealed tubular degeneration and necrosis besides interstitial 18 hemorrhages. Liver showed fatty changes and mild hemorrhages besides biliary retention (Singh et al., 2014). Coagulative necrosis of the hepatocytes and the bile ducts hyperplasia have been reported earlier (Egbe-Nwiyi et al., 1997). Hepatic necrosis is a feature of this disease. Anderson and Hurtado (1989) also reported centrilobular hypoxic hepatic necrosis and splenic hemosiderosis which may be due to erythrocyte destruction. DeVos et al. (2006) also reported biliary retention in most cases of

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Published 20/06/2026

anaplasmosis as there is accumulation of haemosiderin in cells of mono-nuclear phagocytic system

DIAGNOSIS

Diagnosis of bovine anaplasmosis is usually based on signalment and presenting clinical signs as well as laboratory tests such as light microscopic examination of Giemsa-stained blood smears or serological/molecular diagnostic procedures (Radostits et al., 2007). A competitive ELISA has been used for diagnosis of A. marginale infection in various ruminants including cattle, sheep and deer (Kocan et al., 2010). Currently cELISA test used for diagnosis of bovine anaplasmosis is based on use of a monoclonal antibody that recognizes MSP5 of Anaplasma marginale (de la Fuente et al., 2004). This antigen is conserved among all known species of Anaplasma (Salih et al., 2009). The extracted DNA from blood cells were analyzed by Anaplasma marginale specific PCR using primers derived from msp5 gene. Primers for the PCR were

Forward primer GTGCTACGATCGCGCCTGCT,

Reverse primer GCCCATGCCACTTCCCACGG (Radwan et al., 2013).

Moumouni et al. (2015) revealed the sequences of Msp5 gene of A. marginale showed 99.8% identity with each other and were homologous to the sequences of isolates from Australia, the Philippines and China. Dahmani et al. (2017) observed that rpoB gene sequences from A. ovis positive samples were identical to each other and showed 100% identity with A. ovis strain KMND Nayes-14. Also, rpoB sequences from two A. marginale positive R. bursa ticks and the four other sequences obtained from cattle blood samples were identical to each other and showed 100% identity with A. marginale strain Dawn and Gypsy Plains and 99% with A. marginale strain Florida and St. Maries.

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