

Concurrent Traumatic Reticulo Pericarditis (TRP) with Theileriosis in HF-Cow

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ABSTRACT

A 4-year-old Holstein Friesian cow was presented to the Department of Veterinary Pathology, GADVASU, Ludhiana for postmortem examination. On external examination, brisket edema and enlargement of the prescapular lymph nodes were observed. Grossly, there was traumatic reticulo-pericarditis with suppuration, hepatic congestion and punched-out ulcers in the abomasum. Histopathologically, the heart showed myocardial fibrosis the abomasum revealed lymphoplasmacytic ulcerative abomasitis, the lymph nodes exhibited lymphadenitis and the liver showed features of chronic venous congestion. Blood smear examination revealed the presence of *Theileria* piroplasms within the red blood cells.

Keywords: Cow, fibrosis, *Theileria*, TRP, ulcer

Traumatic reticulo-pericarditis (TRP), also known as hardware disease is a condition caused by the ingestion of sharp metallic objects such as nails and pieces of wire^{1,2,3}. These foreign objects penetrate the reticulum and can damage nearby anatomical structures including the liver, diaphragm, spleen and heart. The ingestion of such objects is common in cattle grazing in areas where fodder is grown near industrial sites. TRP is a painful and progressive condition that can ultimately lead to the animal's death and results in significant economic losses due to decreased productivity^{2,4}. Theileriosis is a tick-borne febrile disease caused by protozoan parasites such as *Theileria annulata*, *T. parva*, *T. lawrencei* and *T. orientalis*, all of which contribute to production losses, morbidity and mortality in cattle^{5,6,7}. It affects approximately 39 million crossbred cattle in India, causing substantial economic losses annually⁸. Transmission is primarily mediated by hard ticks belonging to the genera *Hyalomma* and *Rhipicephalus*^{5,8}. *Theileria* has a complex life cycle that requires two hosts. During tick feeding, sporozoites are inoculated into the host and are subsequently taken up by mononuclear cells (MNCs), such as lymphocytes and macrophages. Inside lymphocytes, sporozoites undergo schizogony to produce schizonts (Koch blue bodies)^{5,7}. The infection of T-lymphocytes by *Theileria* reprograms the host cells by upregulating the NF- κ B pathway, leading to the expression of anti-apoptotic genes and promoting cell survival⁹. These transformed, proliferating lymphocytes cause lymph node enlargement. Merozoites released from infected lymphocytes then invade red blood cells, causing hemolysis that leads to severe anemia and jaundice¹⁰.

A 4-year-old female Holstein Friesian (HF) cow was presented to the Department of Veterinary Pathology, Guru Angad Dev Veterinary and Animal Sciences University (GADVASU), Ludhiana, Punjab with history of respiratory distress and high fever (104°F) persisting for six days. The animal suddenly collapsed during clinical examination. Bilaterally enlarged prescapular lymph nodes were noted and the animal exhibited abduction of the forelimbs while walking. At necropsy, marked subcutaneous ventral edema was observed in the brisket region. In the abdominal cavity, the abomasum was adhered to the diaphragm. Within the thoracic cavity, the heart was enlarged to approximately three times its normal size and showed adhesions to the ribs. The pericardial sac was markedly thickened and contained approximately 500 ml of yellowish,

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turbid, fibrino-suppurative fluid. A fibrino-purulent, cheesy mass was adhered to the epicardium, giving the classical "bread and butter" appearance (Fig. 1). A 12-cm long metallic wire was discovered in the reticulum, piercing through the diaphragm into the heart, forming a fistulous tract. The needle's migration tract was discoloured black. Liver and spleen were enlarged; the liver was notably icteric. The liver's surface showed congestion and widespread petechiae. On cut section, the liver exhibited a prominent reticular pattern, imparting a characteristic "nutmeg" appearance (Fig. 2). The prescapular lymph nodes were enlarged, edematous and hemorrhagic (Fig. 3). The abomasum showed severe multifocal punched out ulcerations with associated edema and hyperemia (Fig. 4).

Microscopically, the

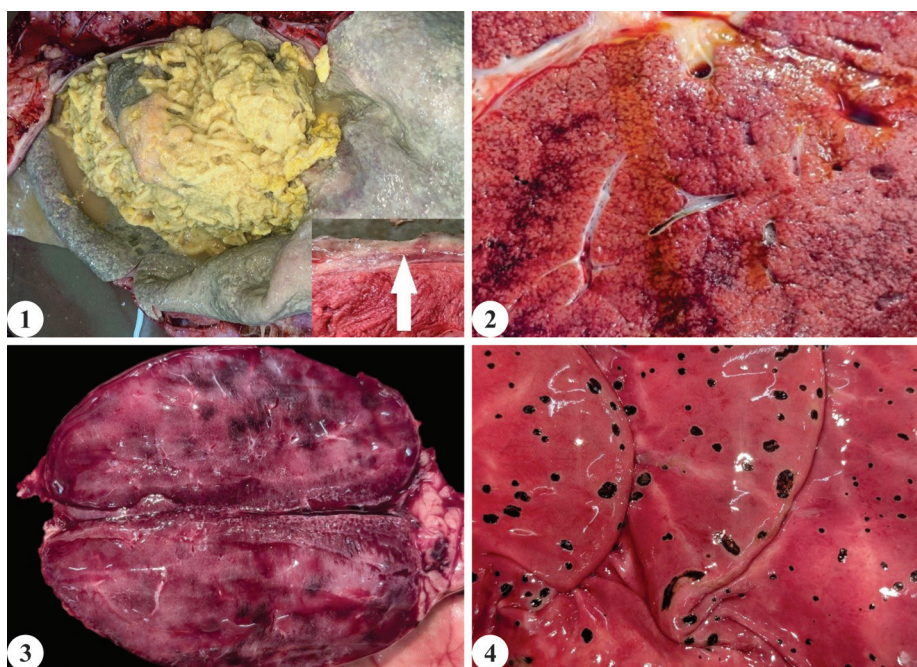


Fig. 1. The pericardial sac covered with fibrin giving bread butter appearance. Inset: Wide band of fibrosis on the surface of the epicardium (arrow); **Fig. 2.** Chronic passive congestion liver cut section shows enhanced lobular pattern; **Fig. 3.** Multiple punched out mucosal ulcers in abomasum with areas of haemorrhages; **Fig. 4.** Oedematous prescapular lymph nodes associated with multifocal coalescing petechiae and ecchymosis.

sections of heart revealed extensive epicardial fibrosis accompanied by suppurative inflammation (Fig. 5). Myocardial cells displayed degenerative changes such as sarcoplasmic swelling, eosinophilia, vacuolation and multifocal lymphoplasmacytic infiltration. The sections

of liver showed chronic passive congestion, evidenced by centrilobular hepatic atrophy and periportal sinusoidal congestion (Fig. 6). The section of prescapular lymph nodes exhibited follicular hyperplasia with fibrin, hemorrhage and mononuclear cells within

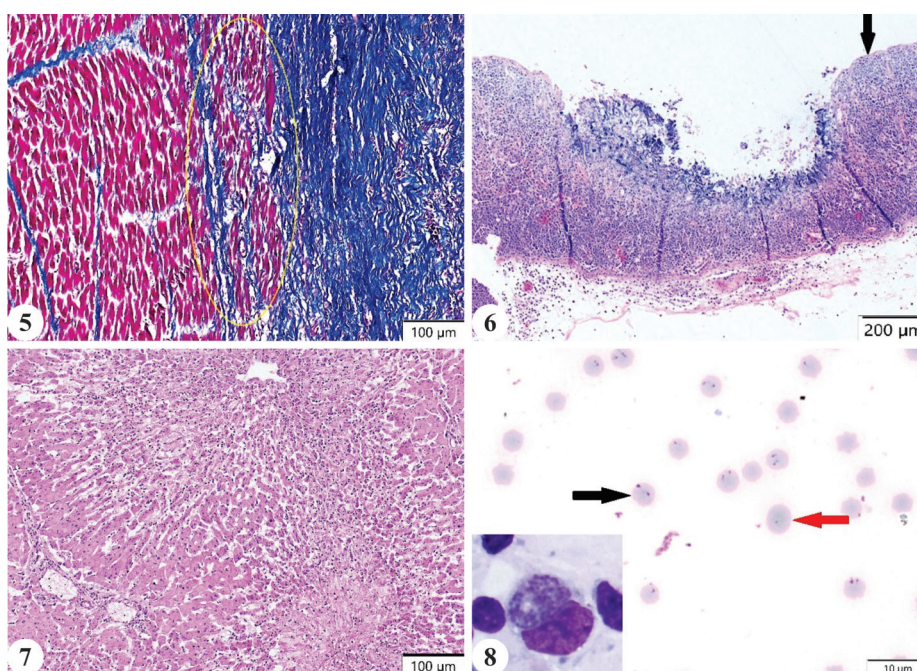


Fig. 5. Heart showing organized granulation tissue having extensive fibrosis (Masson's trichrome stain x100); **Fig. 6.** Focal mucosal abomasal ulcer with raised edges due to proliferation of lymphocytes (arrow) and central area of haemorrhage (H&E x40); **Fig. 7.** Liver shows centrilobular bridging necrosis (H&E x100); **Fig. 8.** Blood smear showing single (red arrow) and multiple (black arrow) *Theileria* piroplasm inside RBCs. Inset: Koch blue body inside lymphocyte pushing the nucleus (Giemsa stain x1000).

the subcapsular sinus. Impression smears from lymph nodes showed numerous schizonts (Koch blue bodies) indenting the nuclei of lymphoblasts. In the sections of the spleen, proliferation and infiltration of macrophages, plasma cells and lymphocytes were evident. The sections of abomasum showed lymphoplasmacytic infiltration in both the mucosa and submucosa with multifocal ulceration of the mucosal surface (Fig. 7). Blood smear prepared from heart blood revealed numerous *Theileria*'s piroplasms within many red blood cells, ranging from one to three piroplasms per RBC (Fig. 8).

The pathogenesis of TRP involves the migration of pyogenic bacteria through a fistulous tract created by a sharp foreign object (e.g., needle), originating from the reticulum and extending to the heart. This leads to fibrino-purulent pericarditis characterized by yellowish-green villous projections, imparting a shaggy heart or "bread and butter" appearance^{2,3,11,13}. Chronic constrictive pericarditis develops due to organization and fibrosis of the exudate, resulting in thick fibrous connective tissue bands, as observed in the present case (Fig. 1). The constrictive pericarditis, combined with cardiac tamponade due to excessive purulent fluid, contributed to the animal's death¹³. A generalized pathological change in TRP is chronic venous congestion, which causes persistent hypoxia in centrilobular regions of the liver, leading to hepatocellular atrophy, degeneration and necrosis giving rise to the characteristic "nutmeg liver" appearance (Fig. 2).

Anemia and jaundice are common clinical signs in cattle affected by *Theileria* infection, both of which were observed in this case^{5,7,10,12}. Prominent pathological findings in Theileriosis include splenomegaly, generalized lymphadenomegaly, abomasal edema and ulceration, edema, epicardial haemorrhages, diarrhoea and/or constipation, hepatic congestion and petechiation on liver^{1,9,10}. In this case, jaundice was likely due to a combination of widespread hemorrhages and centrilobular hepatic necrosis resulting from both *Theileria* schizont infection and chronic venous congestion.

Based on gross and histopathological findings, the present case was diagnosed as a concurrent case of TRP and Theileriosis in a Holstein Friesian cow.

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