

AN UNUSUAL CASE OF IDEOPATHIC FIBRINOUS PERICARDITIS IN NILI-RAVI BUFFALO-A CASE REPORT

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ABSTRACT

An unusual case of idiopathic fibrinous pericarditis in Nili-Ravi buffalo was presented at Veterinary Teaching Hospital, Department of Clinical Medicine and Surgery (CMS), University of Agriculture Faisalabad. The clinical signs and postmortem findings were suggestive of idiopathic fibrinous pericarditis which are discussed in detail.

Keywords: Nili-Ravi buffalo, clinical signs, postmortem findings, idiopathic, fibrinous pericarditis

INTRODUCTION

Nili-Ravi buffalo is also known as Black Gold of Pakistan, because of its adaptability in hot climates and major share in milk and meat production. Inflammation of pericardium along with accumulation of fibrinous material is called as pericarditis (Grunder, 2002). Clinical signs associated with pericarditis are tachycardia, muffled heart sounds, distention of jugular vein, edema of submandibular, brisket and ventral abdominal region. This is usually a disease of developing countries because of poor managemental and feeding practices, affecting ruminants through loss of production and death. This condition is mostly

seen in old, pregnant and recently parturated animals. Ingestion and penetration of any sharp object from gastrointestinal tract to heart usually leads to development of this disease. Incidence of foreign body associated pericarditis higher in buffalo than in cattle (Misk *et al.*, 2001). Among animals, dogs and horses are usually affected by idiopathic fibrinous pericarditis (Jesty *et al.*, 2005). This is infrequently seen condition in buffalo especially Nili-Ravi buffalo (Summet *et al.*, 2012).

CASE PRESENTATION

A 7 year old, Nili-Ravi buffalo was presented in comatosed position at Veterinary Teaching Hospital, Department of Clinical Medicine and Surgery (CMS), University of Agriculture Faisalabad Pakistan. The buffalo was from a dairy farm with 20 other milking animals. She had calved 25 days earlier without any problem. Anamnesis did not reveal any significant abnormality but history of complete anorexia, scanty feces and sudden drop in milk production for the last 5 days. She was treated by referring veterinarian with fluid therapy, antibiotics, analgesics, laxatives and ruminotonic but buffalo did not respond; the condition of the animal had deteriorated despite therapy. On clinical examination, buffalo had severe tachycardia, dyspnoea and mild brisket

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edema. Temperature, respiration and pulse rate were 39.2 °C, 45 breaths/minute, 120 beats/minute, respectively. Before a diagnosis could be arrived, the animal died. Postmortem was conducted with aim of investigation of the cause of death.

NECROPSY FINDINGS

Thoracic and abdominal findings were insignificant except generalized edema most prominent in thoracic region, along with foul smelling straw colored fluid in pericardium having flakes of fibrinous material giving gelatinous and hirsute like appearance to heart especially epicardium and pericardium. Grossly there was no detectable lesion or spots on heart except markedly increased heart size and thickened pericardial wall. Reticulum was full of undigested fodder having four, 5 to 6 centimeter non penetrated metal wires along with six, 3 to 4 centimeter blunt ended nails.

Reticular wall was intact. No other lesions were detected in rest of the body.

DISCUSSION

The clinical signs and postmortem findings were suggestive of fibrinous pericarditis as described by Radostits *et al.*, (2007). Grossly, heart and reticulum were normal, as there was not a single spot or hole on the outer surface. The underlying cause of pericarditis was not of traumatic origin because of absence of fibrous tract from reticulum to pericardium. Generally pericarditis has three forms, effusive, fibrinous and constrictive pericarditis. Accumulation of protein rich fluid in the pericardial sac is called as effusive pericarditis. If there is fibrin deposition along with protein rich fluid then it is termed as fibrinous pericarditis, while maturation of fibrin along with the fibrosis of pericardium is known as constrictive



Figure 1. Accumulation of straw colored fibrinous material in pericardium of affected buffalo.

pericarditis (Pekins *et al.*, 2004). In ruminants, incidence of fibrinous and septic pericarditis is more in contrast to idiopathic and non septic pericarditis. In ruminants mostly pericarditis is traumatic in origin, where a sharp object such as wire and nail penetrates into pericardium through perforation in reticulum. Postmortem examination of affected animal usually reveals foreign body penetrating into pericardium but sometime the penetrating object may fall back into reticulum after contacting pericardium, so traumatic pericarditis cannot be ignored in this case. Nonetheless, the heart was bereft of any healing or healed trauma. Haematogenous route associated pericarditis has been reported in colibacillosis, salmonellosis and some anaerobic infections. It is less common and is masked by signs of septicemia. Radostits *et al.* (2007) reported that physical penetration of pericardial sac is not essential for development of pericarditis as in some cases traumatic mediastinitis acts as a vehicle for the development of infection. In humans, presence of large quantity of fibrinous material in the pericardial sac is considered to be a diagnostic parameter for pericarditis (Lewinter and Kabanni, 2005).

Clinical signs, hematological analysis, radiography, pericardiocentesis, ultrasonography and postmortem findings are diagnostic tools for confirmation of pericarditis. Different control measures such as routine administration of magnets to pregnant animals, keeping ruminants away from construction areas, routine analysis of crops for metallic objects, effective feeding and managerial strategies such as screening of fodder and processed feed before serving to animals. This unusual case of idiopathic fibrinous pericarditis has not been reported in Nili-Ravi buffalo in Pakistan. Our case study is based on clinical signs and postmortem findings as described

elsewhere (Radostits *et al.*, 2007; Sumeet *et al.*, 2012). However, this report lacks microbiological and histopathological investigations that need to be investigated to understand the etiopathogenesis of this condition.

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