PATHOLOGICAL STUDY OF ACUTE FASCIOLOSIS IN A BUFFALO

Rakesh Kumar^{1,*}, Devina Sharma², Rupali Masand¹, Abhishek Kumar¹ and Rajesh Kumar Asrani¹

ABSTRACT

A 12 year old female buffalo with the history of fever, anorexia and hemoglobinuria was presented to Multispecialty Veterinary Hospital at DGCN College of Veterinary and Animal Sciences, CSKHPKV, Palampur. The necropsy examination of the animal showed severe hepatomegaly with dirty fibrin threads attached with the liver, yellowish discoloration, necrohemorrhagic tracts, presence of leaf like flukes in the liver and hydro peritoneum. The petechial hemorrhages were evident in the peritoneal surface and intestinal mucosa. The epicardial and endocardial hemorrhages were quite evident in the heart. Severe hydrothorax and pulmonary oedema were also recorded during the necropsy. Histopathological evaluation of liver showed coagulative necrosis, hemorrhages, extrahepatic jaundice, mild bile duct hyperplasia, hydropic degeneration and hemosiderosis. The parasite was later on identified as F. gigantica leading to cholangio-hepatitis creating anaerobic conditions and fastidious growth of clostridial organisms. The hypoproteinaemia and toxemia all together led to the death of animal.

Keywords: Bubalus bubalis, buffaloes, F.

gigantica, Bovine bacillary hemoglobinuria, liver, hypoproteinaemia

INTRODUCTION

Fasciolosis, due to Fasciola hepatica and F. gigantica, is an important parasitic disease and affects the animal health adversely due to mortality, liver condemnation, reduced weight gain (up to 20%) and reduced quality and quantity (3 to 15%) loss) of milk production (Piedrafitta et al., 2010). Globally, more than 700 million domestic animals are at risk and millions of ruminants are infected worldwide with economic loss exceeds US\$2 billion per year (Mas Coma et al., 2005). Fasciola species seem to have uneven geographic distribution. The major infected areas by F. gigantica are the tropical regions of Africa and several Asian countries including Uzbekistan, Turkmenistan, Iran, Iraq, India and Pakistan (Mogatham et al., 2004; Raina et al., 2013). Adult worms are the inhabitants in the biliary system and cause chronic inflammation and obstruction eventually leading to cholangitis, cholecystitis and cholelithiosis. Before they mature in the biliary ducts, the larval forms can migrate for 10 to 12 weeks in the liver. Therefore, the acute syndrome due to the lesions may last for several

¹Department of Veterinary Pathology, College of Veterinary and Animal Sciences, Himachal Pradesh, India *E-mail: rkvetpath@gmail.com

²Department of Veterinary Parasitology, DGCN College of Veterinary and Animal Sciences, CSK HP Agricultural University, Himachal Pradesh, India

months (Spithill and Dalton *et al.*, 1988; Robinson and Dalton *et al.*, 2009). Recently, exposure to helminth infection (*Fasciola hepatica*) has been associated negatively with the disclosure of Bovine tuberculosis (Byme *et al.*, 2018) and *Clostridium novyi* infection (Smith *et al.*, 2014). The present study emphasizes the importance of regular deworming in the swampy regions of Himachal Pradesh, where the intermediate hosts like snails for *Fasciola* spp. development often plays an important role.

Case presentation and observation

A 12 year old female buffalo was presented to Multispecialty Veterinary Hospital at DGCN COVAS CSKHPKV, Palampur with the history of anorexia with sudden onset of pyrexia and passage of coffee coloured urine, which was confirmed as haemoglobinuria, later on. Animal was presented to the Department of Veterinary Pathology for the systemic necropsy examination. A thorough necropsy examination of the carcass was done, and gross lesions were recorded. Approximately 0.5 cm thick tissue sections were collected in 10%neutral buffered formalin, processed, sectioned at 4 microns and stained with Haematoxylin and Eosin (H&E) stain as per the standard protocols given by (Luna et al., 1968). Samples from the cut section of liver were collected in sterile swab for the microbiological investigation. Adult flukes were recovered from the parenchyma of liver then washed 3 to 4 times with phosphate buffer saline (PBS). After washing, samples were preserved in 10% buffered formalin, carefully labelled with proper details. Thereafter, the adult flukes were flattened, put between two slides, pressed and stained in Borax carmine, for detailed morphological studies according to the procedure given by Singh and Srivastava et al. (1977).

RESULT AND DISCUSSIONS

detailed necropsy examination The revealed severely enlarged dirty greenish brown coloured liver with fibrinous threads hanging on the parenchymal surface forming adhesions with peritoneal surface along with distended gall bladder. On cut section, the liver showed hard consistency, diffuse areas of necrosis along with calcification and hemorrhagic tracts formed by the migration of immature flukes. On the examination of bile ducts leaf like flukes were evident throughout. The cut section of liver showed complete dark yellowish discolouration due to cholestasis and sixty percentage of affected liver showed blackish appearance which on cut section showed the presence of foul smelling gas bubbles (Figure 1). One of the portion of liver showed the presence of solitary abscess lined by fibrous tissue capsule. The peritoneal surface showed petechial hemorrhages along with the accumulation of approximately 4 liters of sero-sanguineous fluid in peritoneal cavity. Spleen was shrunken. The intestinal mucosa showed the presence of petechial hemorrhages throughout. On opening the thoracic cavity approximately 2 liters of serous fluid was recovered. A lemon sized diffuse serous fluid filled pulmonary cyst was present on the cranio-ventral aspect of the lung. The trachea was filled with froth. The endocardial surface of heart showed suffusive reddish coloured hemorrhages while petechial hemorrhages were recorded on the epicardium of the heart.

Histopathological examination of the liver showed large areas of coagulative necrosis with profusehemorrhages.Degenerativechangesranging from cellular swelling to hydropic degeneration were evident. Mild bile duct hyperplasia along with accumulation of bilirubin inside the bile ducts



Figure 1. Liver showing the presence of fluke (arrow) in the bile duct along with hemorrhagic areas and cholestasis.



Figure 2. Liver showing diffuse area of coagulative necrosis, mild bile duct hyperplasia, hemosiderin and extrahepatic bile accumulation (H&E* 200x).



0.6 to 1.14



indicating extrahepatic icteric condition (Figure 2). Excessive erythrophagocytosis as a result of hemorrhages in the liver resulted to diffuse hemosiderosis. Increased kupffer cell activity and infiltration of neutrophils with few mononuclear cells (MNCs) was quite evident in the liver.

The flukes were identified as *Fasciola* gigantica. Their length and width of the fluke varied from 3.5 to 5.7 cm and 0.6 to 1.14 cm respectively. The shape was oblong with a longer rounded posterior end as compared to broadly pointed posterior end of *F. hepatica* (Figure 3). It had a shorter cephalic cone, a larger ventral sucker and a more anterior position of the testes. Gram staining of the swab taken from the portion of liver with gas bubbles formation showed the presence of bluish rods.

The clinical, gross, microscopic, microbiological and parasitological observations all together puts into record a case of buffalo suspected of bovine bacillary hemoglobinuria (BBH) aggravated due to the anaerobic conditions created by the massive damage of liver by *Fasciola gigantica*. Flaring up of clostrdial organisms causing toxemia and profound hypoproteinaemia (ascites and hydrothorax), a sequel to liver damage might be the cause of death of the animal.

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