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AFLATOXICOSIS IN DOG- A CASE REPORT

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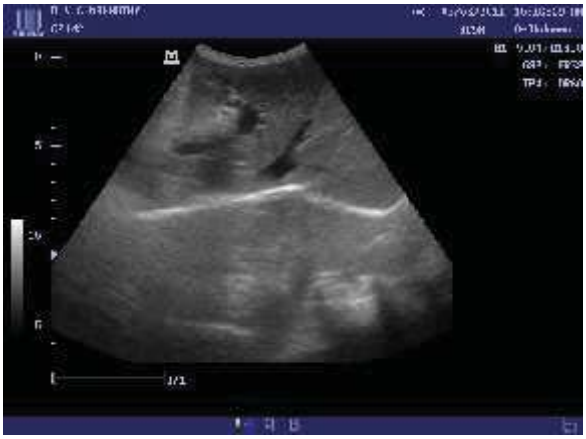
Introduction

Aflatoxicosis is due to ingestion of performed fungal toxin in the feed. It is common in domestic animals .However reports of aflatoxicosis in dogs are scarce. Most cases of aflatoxicosis occur when animals ingest moldy food stuff susceptible to contamination with toxins from *Aspergillus flavus* or *Aspergillus parasiticus.* Disease is characterized by icterus, lethargy, anorexia, petechiae, melena, epistaxis, and hematemesis.

Case history and observation

An eight month old male Great Dane was presented to University Veterinary Hospital, Mannuthy, Thrissur with a history of anorexia, distended abdomen and extreme depression. Clinical examination revealed temperature 102.2^0F, pulse rate 90/min, pale roseate mucous membranes, anterior abdominal pain and mild fluid thrill on tactile percussion of abdomen. Owner reported that another 2 year old female Great Dane dog is also showing same symptoms. Both the dogs were feeding with the same dog food. Hematological analysis revealed a low total RBC count 1.74million/Cu.mm, Hemoglobin 3.50 g/dl,and Platelet count 0.50lakhs/Cu.mm with slightly higher total WBC count 15000cells/Cu.mm.



(Fig.1-Hepatomegaly with engorged blood vessel)

Blood biochemistry showed normal total protein 9.72g/dl with low Albumin 2.33 g/dl high Globulins 5.39 g/dl and low A/G ratio 0.4:1 increased SGPT 1790 IU/L and Alkaline Phosphatase 439.00 IU/L. Urea, Creatinine value were within the normal range. Electrocardiography showed no abnormality. Fecal sample and blood smear examination were negative. On ultrasonography hepatomegaly could be detected (fig 1). Liver biopsy was not attempted due to low platelet count and resultant bleeding. Blood vessels in liver were engorged. On feed analysis using thin layer chromatography presence increased level of aflatoxin was detected (100ppb-200ppb). So the condition was diagnosed as aflatoxicosis.

Treatment, Management and Discussion

Dog was treated with Inj.DNS I.V@ 10 ml/kg b.wt, Inj. Lasix 2 ml I.M, Inj. Neurobion 2ml I.M. The owner was advice to give Tab. Silybon (Sylimarin) 70 mg twice daily, orally. Owner was also advised to stop the food which was contaminated with aflatoxin. After 7 days, owner reported that appetite came to normal and after 20 days there was complete recovery and disappearance abdominal distension.

Aflatoxins are not only potent toxins but also are carcinogenic, mutagenic and immunosuppressive. There are four natural aflatoxin (B1, B2, G1, and G2) responsible for food contamination and among these B1 is the

most hepatotoxic. Aflatoxin also can be nephrotoxic, and they can cause hemolytic anemia and coagulopathies. Aflatoxins are lipid soluble and readily absorbed from the gastrointestinal tract into the portal blood. They are then transported to the liver for metabolism. Necropsy finding associated with aflatoxicosis are hepatomegaly and diffuse yellow friable appearance of the liver. There may be serious hemorrhage in the gastro intestinal tract, on serosal surface, on the epicardium endocardium, in skeleton muscle, perirenal and urinary bladder (Jakhar and Sudana 2004). Addition of hydrated sodium calcium aluminosilicate, clay that tightly and selectively adsorbs aflatoxins, is effective in protecting broiler chicken feed from aflatoxin-contamination (Kubena *et al*., 1998). Signs include anorexia, lethargy, vomiting, and jaundice. Hematochezia, melena, and hematemesis are sometimes present, as well as mucosal or more widespread petechiae and ecchymosis (Dereszynski 2008). Patients may have peripheral edema or ascites. Polyuria and polydipsia may also be noted. In some cases of toxicity, acute death occurs before clinical signs are noted. The disease is progressive, and the case fatality rate is high. Histological examination of liver biopsy samples reveals hepatocellular fatty vacuolation, hepatic necrosis, periportal necrosis, portal fibrosis, and perivenular necrosis and inflammation. Bile duct proliferation is present, and bile canaliculi may be plugged with bile cast (Stenske 2006). Cullen and Newbernr (1994) observed that animals on low protein diet are more susceptible to aflatoxicosis.

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