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Therapeutic management of infectious canine hepatitis in an unvaccinated puppy

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Abstract

A 3-month-old non-descript puppy was referred to small animal medicine outpatient unit of Madras Veterinary College Teaching Hospital with the history of Inappetence, vomiting, enlarged tonsils and lymphnodes from past 3 days. Clinical examination revealed pyrexia (39.7 °c), pale pink conjunctival mucous membrane, subcutaneous oedema of neck & abdominal palpation revealed hepatomegaly. Blood profile revealed moderate anaemia Hb (7.7 g/dl), PCV (24.2%), RBC (3.85m/cmm) mild leukopenia WBC (2800/cmm), Serum biochemistry revealed elevated ALT (204 U/L), ALP (1428 U/L), Total bilirubin (1.35 mg/dl) & direct bilirubin (1.28 mg/dl). Ultrasonography evidenced hepatomegaly & engorged portal vein. Symptomatically treated with ceftriaxone I/V infusion @10mg/kg along with DNS @ 5ml/kg I/V. Bilateral corneal edema observed on 6th day of presentation. PCR assay on nasal swab detected CAD1 virus and the disease diagnosed as ICH. Therapeutic management and successful recovery of the pet will be discussed further.

Keywords: ICH, Hepatitis, Blue eye, PCR

Introduction

Infectious canine hepatitis (ICH) due to canine adenovirus 1 (CAV-1), is a highly contagious viral disease. It causes Severe abdominal pain, vomiting, diarrhea, jaundice (yellowing of the eyes/gums), and bleeding disorders. The main route of infection is occurring through urine, feces, or saliva from infected dogs. It mainly damages some vital organs like liver, kidney and eyes. The diseases are very rarely occurred in dog due to routine vaccination. But it primarily seen in unvaccinated puppies (Carter *et al.*, 2004) ^[1].

Case History, Observation and Diagnosis

A 3-month-old non-descript unvaccinated puppy was referred to small animal medicine outpatient unit of Madras Veterinary College Teaching Hospital with the history of Inappetence, 2-3 episodes of vomiting per day, enlarged tonsils and lymphadenopathy from past 3 days. Clinical examination revealed pale pink conjunctival mucous membrane, subcutaneous oedema of ventral neck, woebegone expression and hepatomegaly on abdominal palpation. Hemogram revealed moderate anaemia Hb (7.7 g/dl), PCV (24.2%), RBC (3.85m/cmm) mild leukopenia WBC (2800/cmm). Serum biochemistry revealed elevated ALT (204 U/L), ALP (1428 U/L), Cholesterol (329 mg/dl), Total bilirubin (1.35 mg/dl) & direct bilirubin (1.28 mg/dl) and hypoglycemia (63mg/dL) (Table 1). Ultrasonography evidenced hepatomegaly with normal liver parenchyma (Fig 1) and engorged portal veins.

Animal symptomatically treated with broad-spectrum antibiotic ceftriaxone I/V infusion @10mg/kg, Inj: Amikacin 10 mg/kg I/V along with DNS @ 5ml/kg I/V and hepatoprotectants for 5 days. On 6th day of presentation bilateral corneal opacity noticed (Fig 2). Considering the corneal opacity along with presented clinical signs, differential diagnosis was trypanosomiasis, ICH and Ehrlichiosis. No microfilariae of *Trypanosoma evansi* noticed in the microscopic examination of fresh wet blood films (wet film examination) and Microhematocrit centrifugation (Woo test). No parasite or morula of *Ehrlichia canis* on peripheral blood smear

and no thrombocytopenia or monocytosis in hematology. Nasal swab was collected on that day for the detection of Canine Adeno Virus 1. By using primer 5'-CGCGCTGAACATTACTACCTTGTC-3' and ICHB 5'CCTAGAGCACTTCGTGTCCGCTT-3' amplifying E3 gene of CAV-1 and CAV-2 were used and the disease is confirmed as ICH by PCR (Fig 3). Confirmatory diagnosis made as Infectious Canine Hepatitis.

Treatment

Treatment continued with fluid therapy, antibiotics and supportive therapy using heamatinics, hepatoprotectants were given. After 10 days of treatment blood profile revealed normal range of Hemoglobin (12 g/dl), PCV (30%), RBC (4.6m/cmm), WBC (16000/cmm), ALT (115 U/L), ALP (160 U/L), Cholesterol (150 mg/dl), Total bilirubin (0.4mg/dl), Direct bilirubin (0.1mg/dl). Animal had an uneventful recovery.

Case Discussion

Infectious canine hepatitis is a systemic disease caused by canine adeno virus CAV-1 which replicates in vascular endothelial cells and hepatocytes and produces acute enterohemorrhagic hepatitis (Appel *et al.*, 1987) [2]. Infectious canine hepatitis commonly affects dogs less than 1 year of age (Greene *et al.*, 2006) [3]. Transmission occurs via direct contact or indirectly through exposure to infectious saliva, faces, urine, or respiratory secretions and ectoparasite such as fleas and ticks (Willis *et al.*, 2000) [4]. Viremia occurs up to 6 to 9 months after infection (Poppensiek & Baker *et al.*, 1951) [5]. virus initially replicates within the tonsils, after which it spreads to regional lymph nodes and the bloodstream via lymphatics. incubation period of CAV-1 in dogs is 4 to 9 days, although many dogs exhibit no indications of disease (Cabasso *et al.*, 1962) [6].

Biphasic fever is the earliest clinical sign. Some dogs recover after the first febrile peak (1–2 days). Dogs with a second peak of hyperthermia are more likely to develop a more severe type of ICH. Clinical signs include depression, inappetance, lethargy, dehydration, fever (up to 106°F or 41°C), tonsillitis, pallor, conjunctivitis, peripheral lymphadenopathy, tachypnea, tachycardia, weakness, polydipsia, vomiting, diarrhea, cough, and icterus. Abdominal palpation may reveal abdominal pain or hepatomegaly and distention can occur as a result of accumulation of serosanguineous or hemorrhagic fluid accumulation (Caudell *et al.*, 2005 and Appel *et al.*, 1987) [7, 2]. In chronic form, dogs with partial immunity undergoes hepatic failure over a period of weeks to months. The antibody response occurs 7 days after infection and reduces the tissue damage. Immune complex deposits in Viral persistence area such as cornea, uvea and glomeruli lead to development of corneal edema, corneal opacity (blue eye), severe uveitis and glomerulonephritis (Morrison *et al.*, 1975) [8].

Hematologic findings show leukopenia, neutropenia, anemia, increased nucleated red blood cells, and moderate to severe thrombocytopenia in case of severe form elevated ALT, ALP, hyperbilirubinemia, hypoglycemia, and hypoalbuminemia and coagulation disorders such as DIC can be evidenced (Wigton *et al.*, 1976) [9]. Diagnosis test for ICH includes complement fixation, hemagglutination inhibition, enzyme linked immunosorbent assay (ELISA), viral isolation, immunohistochemistry, and polymerase chain reaction (PCR). sample such as rectal, conjunctival and nasal swabs, blood, urine; tissues collected at necropsy can be sent for

PCR. Hu *et al.*, 2001 [10] stated that PCR can detect field virus (CAV-1) and vaccine virus (CAV-2) but false negative can occur if the animal is in sub-acute or chronic stage of ICH.

Treatment for ICH depends on the weight, age and severity of ICH. In case of acute cases, supplementation of crystalloid fluids and correcting electrolyte abnormalities are indicated. Other symptomatic treatment includes antiemetics, antacids, sucralfate, whole blood or plasma transfusions, and colloids such as hetastarch. In case of hepatic encephalopathy lactulose enemas, oral antimicrobial drugs such as ampicillin may also be indicated. Broad spectrum antibiotics preferred in case of gastroenteritis. corneal edema and uveitis can be treated with topical ophthalmic preparations that contain glucocorticoids and atropine to prevent development of glaucoma.

Prevention of ICH is mainly by properly vaccinating the animal. Active immunity produced by ICH is lifelong. Immunity after immunization with attenuated live vaccines lasts at least 3 years and probably longer. Early vaccines, which contained CAV-1, were associated with the development of corneal edema and glomerulonephritis in a small percentage (<1%) of immunized dogs (MacLachlan & Dubovi *et al.*, 2010) [11] and this complication removed by replacing live attenuated CAV-1 with CAV-2.



Fig 1: Ultrasonography evidenced hepatomegaly with normal liver parenchyma

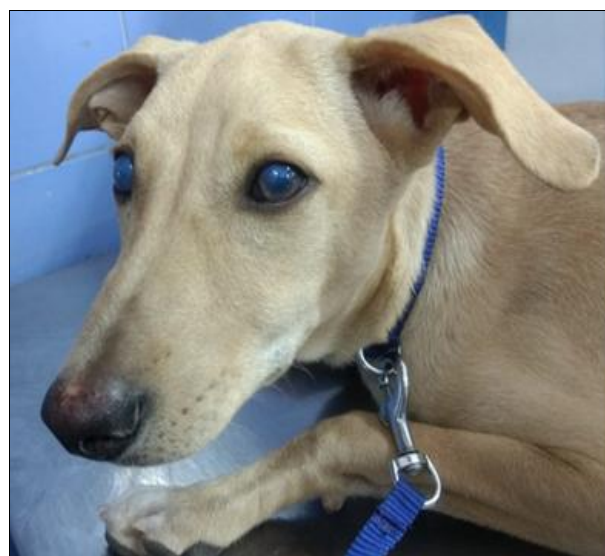


Fig 2: Bilateral corneal opacity

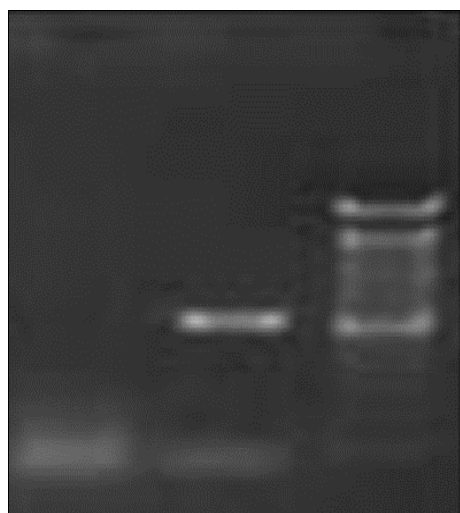


Fig 3: 5'-CGCGCTGAACATTACTACCTTGTC-3' and ICHB 5'CCTAGAGCACTTCGTGTCCGCTT-3' amplifying E3 gene of CAV-1 and CAV-2 were used and the disease is confirmed as ICH by PCR.

Table 1: Hematology & Serum Biochemical Parameters

Parameter	1 st day	5 th day	15 th day
Hemoglobin (g/dl)	7.7	9	12
PCV (%)	24.2	27.9	30
RBC (m/cmm)	3.85	4.55	4.6
WBC (/cmm)	2800	38,000	16,000
Neutrophil	69	90	80
ALT (U/L)	204	178	115
ALP (U/L)	1428	450	160
Cholesterol (mg/dl)	329	300	150
Total bilirubin (mg/dl)	1.35	1	0.4
Direct bilirubin (mg/dl)	1.28	0.8	0.1

Conflict of Interest

Not available

Financial Support

Not available

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