HEPATOCELLULAR CARCINOMA IN A DOG - A CASE REPORT

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Hepatocellular carcinoma (HCC) are the most common primary liver tumor in male dogs (The Merck Veterinary Manual 2010) and it is not uncommon in sniffer dog. These neoplasms typically affect younger dogs, more than two-thirds of the dogs are less than 10 years old (Patnaik et al. 2005). It occurs frequently in mammalian hosts including human (Bertino 1996). Several workers were reported hepatocellular carcinoma in different canine species (Patnaik et al. 1980, Evans 1987, Liptak et al. 2004, Sterczer et al. 2011).

A carcass of sniffer Labrador dog of Central Industrial Security Force, Kolkata Port Trust, aged about three years and six months was brought to Disease Investigation Laboratory, Institute of Animal Health & Veterinary Biologicals, Kolkata, India for postmortem examination to ascertain the cause of death. The dog was suffering in chronic hepatic disorders for last few months with the history of anorexia, lethargy, weakness, inactivity, apathy, weight loss, polydipsia, vomition, abdominal pain and ascites. It was finally unfit for training programme and duty before death.

Haematological and biochemical parameters primarily revealed Total Leucocytic Count (TLC) 6,800 cu. mm, Differential Leucocytic Count (DLC) (Neutrophil 84%, Lymphocyte 13%, Monocyte 1%, Eosinophil 2%, Basophil 0%), Haemoglobin 10.4 gm%, Serum urea 39.80 mg/dl, Blood Urea Nitrogen (BUN) 19.00 mg/dl, Serum creatinine 1.55 mg/dl, SGPT/ALT 83.10 u/l, SGPT/ASL 84.60 u/l, Serum potassium 4.60 mcg/l, Total Serum protein 6.80 gm% (Albumin 3.40 gm%, Globulin 3.40 gm%, Albumin Globulin ratio 1:1). The Thyroid function test by Chemiluminescence assay (CLIA) showed Total triiodothyroxin (T₃) 1.44 ng/ml, Total thyroxin (T₄) 10.87 µg/dl, Thyroid stimulating hormone (TSH) 0.34 ng/ml. Examination of aspirated peritoneal fluid revealed presence of blood cells predominantly lymphocytes with nucleated total cell 780/cu.mm, Sugar
107.0 mg/dl, Total Protein 5.6 gm/dl, Chloride 89.6 mcg/l. The smear of ascitic fluid showed presence of inflammatory and sheets of reactive mesothelial cells.

Ultrasonography of whole abdomen revealed overall gross ascites with a small right sided pleural effusion. Hepatic veins were apathy and dilated. Gall bladder was distended and walls were thin, measured 2 mm at the porta hepatis. Inferior vena cava (IVC) behind the liver was distended.

Fig.1: Irregular masses of enlarged hepatocytes with proliferation of reticular fibers showed pseudo-lobulation (10X).

Fig.2: The large hepatocytes showed few mitotic figures with proliferation of reticular fibers there in. Some places the nuclei of few hepatocytes showing hyper-chromasia with vesicular cytoplasm indicating irregular cell division (40X).

Fig.3: Well developed proliferating hepatocytes with large nuclei and few mitotic figures (40X).

Fig.4: Periportal fibrosis and perivascular proliferation of bile duct, hepatic artery and portal vein. Necrosis of surrounding hepatocytes due to pressure of irregularly scattered hepatocytes and reticular proliferation (10X).
Primarily considered ascites of inflammatory or neoplastic etiology.

The dog was treated for last few months with several medicines without showing any improvement and that gradually became off-fed, reluctant to move, unfit for any work and finally died.

Postmortem examination revealed presence of moderate amount of sero-sanguinous fluid in thoracic cavity and hyperemic thoracic wall in general. Lower part of trachea and broncho-tracheal portion were anastomosed with oesophagus, blood vessels, nerve along with surrounding tissues adhered with spine. Trachea was mild haemorrhagic. Lower part of trachea and broncho-tracheal portion were anastomosed. Right lung was partly solidified, suppurative and haemorrhagic. Left lung was pneumonic, cyanosed and consolidated. Heart was hypertrophic and chambers were filled with clotted blood. Pericardium found congested. Diaphragm was severely congested. Spleen was enlarged and anaemic. Liver was hypertrophic, cirrhotic along with perihepatitis. Stomach was empty. Pancreas was atrophic and anaemic. Intestine was partly haemorrhagic. Mesenteric lymph nodes were enlarged and haemorrhagic. Kidneys were hypertrophic and congestion in cortex. Urinary bladder was thickened with scanty haemorrhages in mucosa. A large amount of dark straw coloured ascitic fluid was revealed in abdominal cavity, which was collected aseptically and Escherichia coli was isolated.

Hepato-cellular carcinoma was detected in histopathological examination. The architectural details showed irregular masses of enlarged hepatocytes with proliferation of reticular fibers given pseudo-lobulation (Fig.1). The large hepatocytes showed few mitotic figures with proliferation of reticular fibers there in. In some places the nuclei of few hepatocytes showed hyper-chromasia with vesicular cytoplasm indicating irregular cell division (Fig.2). The section depicted a well developed proliferating hepatocytes with large nuclei and few mitotic figures (Fig.3). Periportal fibrosis and perivascular proliferation of bile duct, hepatic artery and portal vein were evident. The surrounding hepatocytes were almost necrosed due to pressure of irregularly scattered hepatocytes and reticular proliferation(Fig.4). The liver cells were anaplastic, arranged in trabecular or acinar pattern, mitosis and enlarged nuclei commonly seen, which corroborate with the finding of Vegad (1995). The trabecular growth pattern was 2-8 cells wide layer of tumour cells, which showed similarities with the findings of Mohan (1953). Tumour cells were composed of nest and end to malignant appearing hepatocytes separated by dense bundle of collagen, which corroborate with the findings of Cotran et al. (1994). Suppurative pneumonia and myocarditis were revealed during histopathological examination of lungs and heart muscles respectively. Considering all, it was finally concluded that the dog was died due to multi-organ failure as a consequence of hepato-cellular carcinoma.

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REFERENCES


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