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https://dx.doi.org/10.4314/nvj.v46i1.7 **CASE REPORT**

Concurrent Canine Leptospirosis and Aflatoxicosis in A Kennel: A Case Study.

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ABSTRACT

Leptospirosis and aflatoxicosis are significant health concerns for dogs worldwide. The gross and histopathological lesions in both conditions often overlap and require confirmatory tests for differentiation. Recently concurrent leptospirosis and aflatoxicosis were suspected in a kennel in Ibadan housing nine dogs. Two dogs died without any premonitory signs, while four others died within two days with clinical signs of jaundice, anorexia, and bloody diarrhoea. A combination of necropsy, histopathology, Polymerase Chain Reaction (PCR) and high-performance liquid chromatography analysis (HPLCA) were employed to arrive at the confirmatory diagnosis of the cases. Necropsy findings revealed significant pathological changes in the liver suggestive of aflatoxicosis. Histopathological examination supported both aflatoxicosis and leptospirosis. PCR extraction and amplification was positive for the Leptospira 16s rRNA gene detected in the liver of both dogs. Toxicological screening of the feed revealed aflatoxin levels 39 ppb, nearly doubling the permissible limit of 20 ppb. The high level of aflatoxin observed probably induced immunosuppression, rendering the dogs susceptible to leptospirosis. This report underscores the importance of molecular detection and toxicological assessments of tissue and feed to differentiate between canine leptospirosis and toxic liver injury.

Keywords: Keywords: Aflatoxicosis, Leptospirosis, Histopathological comparison, PCR Testing, Toxicological Evaluation

INTRODUCTION

Canine leptospirosis presents a significant zoonotic threat in Nigeria, posing serious health risks to both dogs and humans. The disease, caused by *Leptospira* bacteria, is associated with systemic infections that primarily affect the kidneys and liver, leading to acute renal failure, hepatic dysfunction, and other life-threatening complications (Goldstein, 2014). Clinically, infected dogs may exhibit fever, lethargy, vomiting, diarrhea, jaundice, and, in severe cases, hemorrhagic manifestations and multi-organ

failure (Aswar et al., 2015). The disease's pathology is marked by interstitial nephritis, tubular degeneration, and hepatic necrosis, which contribute to its high morbidity and mortality rates.

From a public health perspective, leptospirosis remains a critical concern due to its zoonotic potential, with humans contracting the infection through direct or indirect contact with contaminated urine, water, or soil. Occupational exposure, particularly among kennel workers and veterinarians, increases the risk of infection, underscoring the need for stringent biosecurity and hygiene measures (Awosanya et al., 2013). While vaccination remains a key preventive strategy, variations in serovar prevalence necessitate continued surveillance and research to enhance vaccine efficacy and ensure broader protection (Griebsch et al., 2022; CC et al., 2024). Strengthening public health interventions, improving environmental sanitation, and increasing awareness among pet owners and veterinary professionals are crucial steps in mitigating the disease's impact.

Aflatoxicosis is a disease caused by mycotoxin-producing mycotoxins with Aflatoxin B1 being the most toxic (Akinrinmade *et al.,* 2012). It is very common in livestock and less in dogs (Akinrinmade *et al.,* 2012; Benkerroum *et al.,* 2020; Boermans *et al.,* 2007; Martínez-Martínez *et al.,* 2021). It is a non-infectious cause of liver failure, usually associated with hemorrhagic diathesis in 60% of cases (Bastianello, *et al.,*1987). It does not occur as an outbreak but as sporadic cases in dogs. This case came from a single dog breeder with 10 adults and 5 puppies in his kennel with a history of his dogs being fed moldy dried food that had been on stored for over a year. The breeder recorded 5 deaths within 7 days.

The aim of this report is to find out whether the primary cause of the spike in mortality was canine leptospirosis, canine aflatoxicosis or a combination of both conditions. This was done by gross and histopathological description of the lesions as well as PCR and toxicological evaluation of the dry feed.

MATERIALS AND METHODS

Case Presentation

Two dogs were submitted for postmortem and routine histopathological examination and one dog for clinical evaluation at the Veterinary Teaching Hospital, University of Ibadan, from Jericho area of Ibadan, Oyo State, Nigeria. Prior to the presentation, the kennel dogs (three) had been under evaluation and treatment by a private veterinaian within Ibadan. Both dogs had been ill for a short duration and died on the same day. The 3 other dogs died at various intervals within a four-day period prior to the presentation of the two carcasses. All the dogs had a similar history of jaundice, inappetence, and hematochezia. A summary of the history of the dogs presented is in Table 1. After the examination and therapy with liver tonic, there were no more deaths in the kennel.

Table I: Summary of the clinical findings observed in the dogs presented

CASE	BREED	AGE	SEX	DEPRESSION	ICTERUS	HEMATOCHEZIA	ANOREXIA	DIARRHOEA
1	Boerboel	9	F	+	+	+	+	+
2	Boerboel	4	F	+	+	+	+	+
3	Boerboel	3	F	+	-	-	+	-

Pathology

At necropsy, tissues from all organs were collected and stored in 10% formalin, routinely processed and stained with haematoxylin and eosin. Warthrin Starry silver stain was also applied to sections of the liver and kidney for confirmation of *Leptospira spp* organisms in trimmed sections. The liver and kidney samples from the two dogs that were examined at necropsy were also collected in a Petri dish and submitted for PCR evaluation for Leptospirosis using primers specific for pathogenic *Leptospira spp* organisms. Blank template reactions were used as the negative control.

Toxicology

Feed samples were submitted to Animal Care Laboratories, Ibadan, Nigeria, for determination of Aflatoxin levels using antibody capture ELISA method by thin layer chromatography (TLC) and high-performance liquid chromatography analysis (HPLCA) (Pilau *et al.,* 2022). For the aflatoxin detection, the dried feed was blended into powder before weighing and subsequently subjected to extraction for aflatoxins. Milili-Q H20 and 100 mL of ethyl acetate and acetonitrile were added before the mixture was filtered and underwent aliquoting (Ramón *et al.,* 2002). The final samples were dried and injected onto a calibrated gel permeation chromatography column. The quantification of the aflatoxin samples was done by HPLCA (Ramón *et al.,* 2002). The detection limit was 0.4 ppb.

Table 2: Summary of gross findings following necropsy of dogs 1 and 2.

Gross Lesion	**Dog 1**	**Dog 2**
Icterus	+	+
Anemia	+	+
Widespread petechial to ecchymotic haemorrahages	+	+

Pulmonary edema	+	+
Chronic hepatopathy	+	+
Hepatic Pallor	+	+
Gastric ulcers	+	+
Melena	+	+
Nephritis	+	+

Key: - = absent += present

POLYMERASE CHAIN REACTION

Liver and kidney samples were homogenized, and total DNA was extracted using a Universal Genomic DNA Extraction Kit (Solarbio® LIFE SCIENCES, China) following the manufacturer's instructions.

PRIMERS: Target a portion of the 16s ribosomal RNA gene.

LEP-16Sf - 5'- CAT GCA AGT CAA GCG GAG TA -3' (Rahman et al.,2021)

LEP-163R - 5- CTT AAC TGC TGC CTC CCG TA-3 (Ivanova et al.,2012)

PCR amplification was performed in a GeneAmp® PCR System 9700 (Applied Biosystems Waltham, MA).A mixture consisting of 25 µL of the 2X PCR Master Mix (Neb®), 1 µL of each primer at a concentration of 500 nM, 5 µL of the template DNA and 18 µL of nuclease-free water to set up a total reaction volume of 50 µL was used for the PCR. The following cycling conditions; initial denaturation at 94°C for 2 min, 35 cycles of denaturation at 95°C for 20 s, annealing at 50.2°C for 60 s, extension at 72°C for 90 s, and a final extension at 72°C for 7 min were employed for the PCR.

The amplicons were analyzed using agarose gel electrophoresis, with a 2.0% gel, stained with 0.005% ethidium bromide (Bio-Rad, Hercules, CA) and imaged using the Molecular Imager® Gel DocTM XR + System and Image LabTM Software (Bio-Rad, Hercules, CA).

RESULTS

The dogs had yellow livers which were firm to touch. This is consistent with hepatic failure and aflatoxicosis. They had generalized petechial to ecchymotic hemorrhages involving almost every organ including the lungs, urinary bladder, and subcutaneous tissues of the ventrolateral aspect of the abdomen. The second dog also had hemorrhages in the uterine body and horns. The stomach contained abundant dark tarry material mixed with blood in both dogs. There was copious amount of yellow tinged froth in the tracheobronchial passage in both dogs. There was mild scarring and renal cortical hemorrhages present in both dogs. Other gross findings have been summarized in table 2.

Histopathologically, there was severe and diffuse vacuolar degeneration of hepatocytes accompanied by moderate and random centrilobular hepatic necrosis. There was moderate bile ductular hyperplasia with bile retention within the canaliculi. Large bile ducts were seen to retain bile in both dogs. There was moderate proliferation of Kupffer cells and some macrophages were seen to contain hemosiderin pigments. The periportal area was thickened by fibrous connective tissue accompanied by mild pleocellular (neutrophils, macrophages, and lymphocytes) infiltration. There were areas of regenerative nodules with hepatocyte hyperplasia and moderate megalocytosis. There

was individualization of hepatocytes with cord dissociation in dog 1. The hepatic sinusoids were wider due to the thinning of hepatic cords. A few pyknotic hepatocytes were also evident and were more in dog 1 than 2. The summary and comparison between dog 1 and 2 are presented in Table 3.

Table 3: A summary of histopathological lesions found in Dog 1 and 2 and comparison with histopathological lesions commonly seen in Canine aflatoxicosis and leptospirosis.

CASE	DOG1	DOG 2	AFLATOXICOSIS	LEPTOSPIROSIS
Individualization Of hepatocytes	++	+	-	++
Dissociation of hepatic cords	+	-	-	+
Necrosis of hepatocytes	++	++	++	+
Vacuolar degeneration of hepatocytes	++	++	++	+
Megalocytosis	+	++	++	-
Cholestasis	+	++	+	++
Bile ductular proliferation	+	++	++	-
Liver Cirrhosis	-	-	+	++
Bridging hepatic Fibrosis	-	-	++	_
Regenerative hepatic nodules	++	++	++	+
Interstitial nephritis	++	++	-	++
Portal hepatic fibrosis	++	++	++	-
Bronchopneumonia	_	_	-	++

Key: -=absent, -+= may be present, += mild and present, ++= marked and present

The morphological changes were classified as follows;

- Hepato-cellular necrosis (random, multi-focal) with hepato-cellular vacuolar degeneration (severe, diffuse, lipid-type).
- Hepatitis: Mild, sub-acute, lymphoplasmacytic, periportal.
- Nodular regeneration and megalocytosis (moderate, multi-focal) with bile ductular reaction.

Sections of the liver and kidneys that were stained with Warthin Starry silver stain did not reveal the presence of spirochetes in the hepatic sinusoids or the proximal and distal tubules of the kidneys.

Three samples A, B, and C were submitted for testing. Samples A and C were from the batch of dried food (noodles) the dogs consumed while B was locally purchased noodles. Optical density reads @450nm for samples A, B and C were 1.830, 1.966 and 1.760 respectively with the corresponding total aflatoxin concentrations at 37ppb, 24ppb and 39ppb respectively. The samples from the dried feed consumed are almost double the acceptable amount of Aflatoxin permissible by NAFDAC (20ppb) in Nigeria (Akinrinmade et al., 2012; The Partnership for Aflatoxin Control in Africa, 2020).

$$\text{Aflatoxin } (\mu g/kg) = \frac{\text{Absorbance of sample} \times \text{concentration of standard}}{\text{Absorbance of standard} \times \text{weight of sample}}$$

The PCR amplification and extraction for canine leptospirosis was positive for 16s ribosomal RNA gene for two samples of the liver for dogs 1 and 2 and negative for the kidney sample for

dog 2. The kidney sample for dog 1 was unavailable for testing.

DISCUSSION

Leptospirosis is a zoonotic disease with numerous serovars in Nigeria (Pilau et al., 2022). Dogs are seen as important reservoir hosts and asymptomatic and symptomatic carriers of the disease (Azócar-Aedo et al.,2014; Iwamoto et al., 2009; Pilau et al., 2022). Canine Aflatoxicosis is also common in Nigeria with a lot of popular brands having some degree of aflatoxin present enough to cause subclinical to chronic aflatoxicosis with predisposition of the liver to neoplastic transformation (Akinrinmade et al., 2012; Benkerrou, 2020; Boermans and Leung, 2007; Martínez-Martínez et al., 2021) . Canine leptospirosis and acute aflatoxicosis are very strong differentials because the can be acute, present with icterus, elevated liver enzymes, depression, lethargy, widespread hemorrhages, and melena (Azócar-Aedo et al.,2014; Iwamoto et al., 2009; Kumar and Balachandran, 2009; Newman et al., 2007) . Both conditions are often used as differentials for each other but are not often reported as co-infection in a single case. Pilau et al. (2022) found that the prevalence of asymptomatic leptospirosis in dogs was approximately 16%, with serological tests indicating a rate of 13.5% and bacteriological tests showing 1.6%.The feed samples (A and C) analyzed showed aflatoxin levels almost double the acceptable 20ppb (37ppb and 39ppb) in feed and enough to cause acute hepatotoxicity in both dogs 1 and 2.

Even though the dogs were fed for a considerably short period of time (4-5 days), the high dose of the aflatoxin consumed by the well-fed and lactating bitches in the kennel was enough to cause immune suppression and predispose the animals to clinical symptomatic leptospirosis.

The gross and histological findings had not only overlapped features of leptospirosis and aflatoxicosis such as hepatic lipidosis with individualization of hepatocytes, centrilobular necrosis, regenerative nodules, and cord dissociation but features such as megalocytosis, marked bile ductular hyperplasia, and vacuolation, marked periportal fibrosis and reduced inflammatory infiltration were seen which is more common in aflatoxicosis or toxic liver injury (Martínez- Martínez et al., 2021). The histopathology of the liver which is the primary organ which was affected by the conditions strongly suggests that both conditions were occurring side by side with lesions from the aflatoxicosis overwhelming the lesions by the sub-acute leptospirosis (Martínez- Martínez et al., 2021). No major lesions were seen in other organs including the kidneys. Leptospirosis usually causes a tubulo-interstitial lymphoplasmacytic nephritis, pneumonia which was absent in both dogs, and lymphoplasmacytic hepatitis but in these two necropsied dogs a mild multifocal pleocellular infiltration (neutrophils and lymphocytes) was seen in the periportal areas associated with the fibrosis and ductular proliferation. The livers from Dogs 1 and 2 were both pale yellow and firm in consistency which could be attributed to bile retention and vacuolar degeneration of hepatocytes. Wouters et al. reported aflatoxin accumulation in the hepatocyte brings about an initial glycogen depletion within a day and increased fatty change in three days.

The PCR was positive for pathogenic group 1 Leptospira interrogans in two samples of the liver for dogs 1 and 2 and negative for the kidney sample for dog 2. This could point to the serovar affecting primarily the liver or that the dog was asymptomatic until the mycotoxin caused severe immunosuppression and turned the asymptomatic leptospirosis to a clinical presentation and exacerbating the signs associated with hepatic failure (hematochezia, icterus, and widespread hemorrhages). According to Bastianello et al. (1987), hemorrhagic diathesis occurs in up to 60% of cases of aflatoxicosis accompanied by marked thrombocytopenia and prolonged clotting time (Bastianello et al., 1987). It is also interesting to note that perivascular lymphocytic infiltration and Kupffer cell hyperplasia are also seen in chronic intoxication by aflatoxin (Martínez- Martínez et al., 2021). The Warthrin starry silver stain did not elaborate any spirochetes or organisms in the sections of the liver and the kidney for dogs 1 and 2 despite the PCR being positive for Leptospira spp. When dogs are initially diagnosed with aflatoxicosis, leptospirosis is frequently misdiagnosed. Dogs with leptospirosis display symptoms that tend to overlap with aflatoxicosis.

In conclusion, Aflatoxicosis is a potential differential diagnosis since it exhibits symptoms including icterus, depression, and hemorrhages with blood-tinged feces. In some dogs suspected of having aflatoxicosis, polymerase chain reaction (PCR) was actually utilized to rule out the likelihood of leptospirosis occurring (Newman et al., 2007). Once more, a lot of mycotoxin poisoning cases are typically misdiagnosed. The work by Boermans and Leung (2007) claims that veterinarians frequently fail to recognize mycotoxins as the root cause of chronic conditions such as cancer, immune-suppressed infections, liver and kidney fibrosis (Boermans and Leung, 2007).

The overlapping similarities are why more attention should be given to aflatoxicosis in diagnosis and improving the toxicological evaluation of feed, tissue samples, and blood

samples. Lesions especially at histopathological levels overlap up to 90% of the time and the difference is usually the scoring of the lesions which determines the likelihood of either Canine leptospirosis or toxic liver injury (Azócar-Aedo et al.,2014).

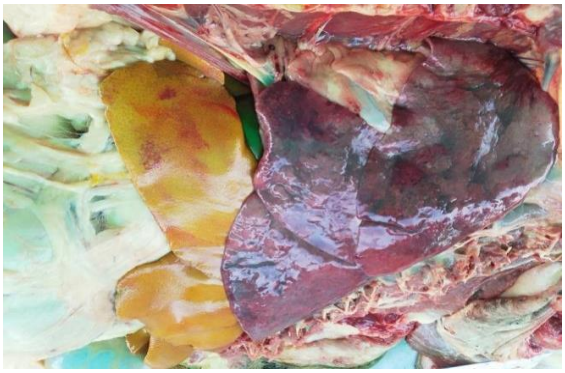

Figure I: Right Thoracic view of organs In-situ.

Figure II: Liver (Dog): Hepatic steatosis.

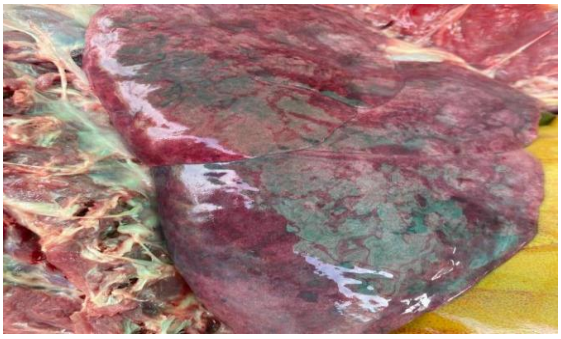

Figure III: Right lung (Dog): Diffuse congestion with multifocal areas of petechial to ecchymotic hemorrhages.

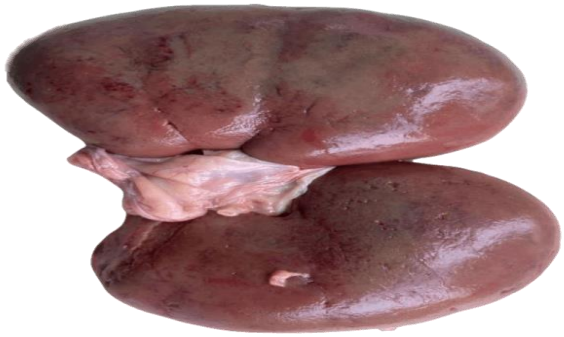

Figure IV: Kidney (Dog): Cortical surface of the kidney showing petechiation and scarring.

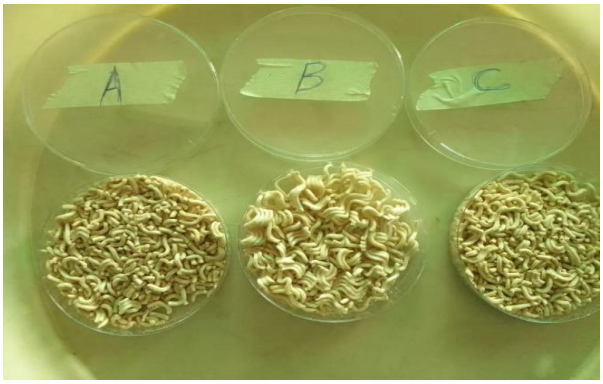

Figure V: Samples A and C are the suspected contaminated dried food. Sample B is the control. Notice how the dried foods A and C have a crumbled appearance and are soft to the touch and smelt moldy.

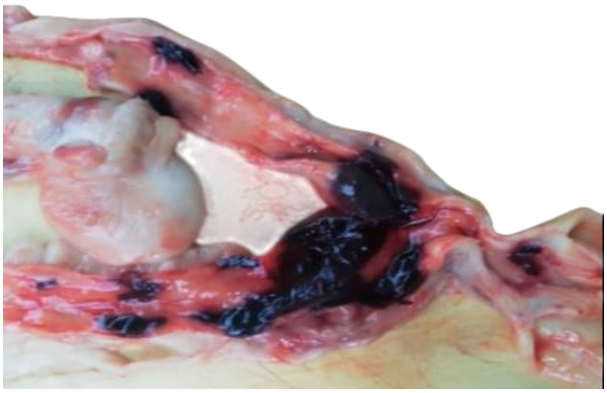

Figure VI: Uterus (Dog) showing variably sized hematomas in the uterine horn and body.

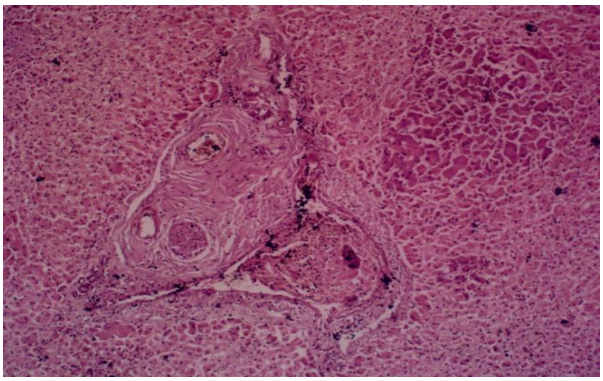

Figure VII: Liver (Dog): Low magnification of liver showing hepatocellular necrosis and severe periportal fibrosis (blue arrow) with a focus of regeneration (black arrow).

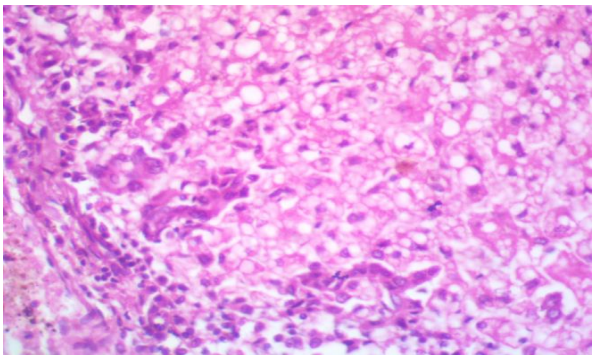

Figure VIII: Liver (Dog): Infiltration of periportal areas with few neutrophils and lymphocytes (black arrow), with lipid-type macrovacuoles (blue arrow) and bile ductular reaction (thick black arrow).

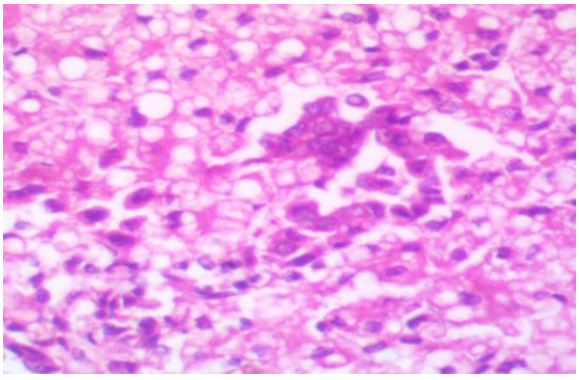

Figure IX: Liver (Dog): Black arrows showing marked Bile ductular reaction.

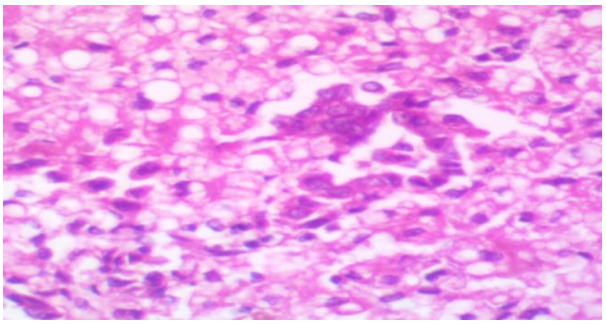

Figure X: Liver (Dog): Distinct hepatic megalocyte (black arrow) with deep eosinophillic cytoplasm.

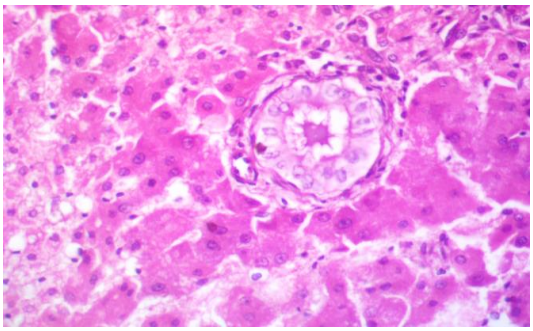

Figure XII: Dog): bile retention (cholestasis) in an intact bile duct. (All sections are stained with H&E).

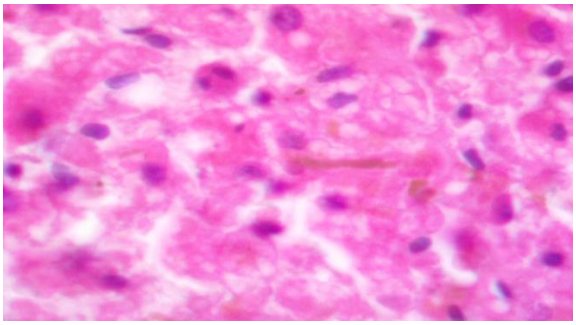

Figure XI: Liver (Dog): High magnification showing bile retention in the canaliculi (black arrow).

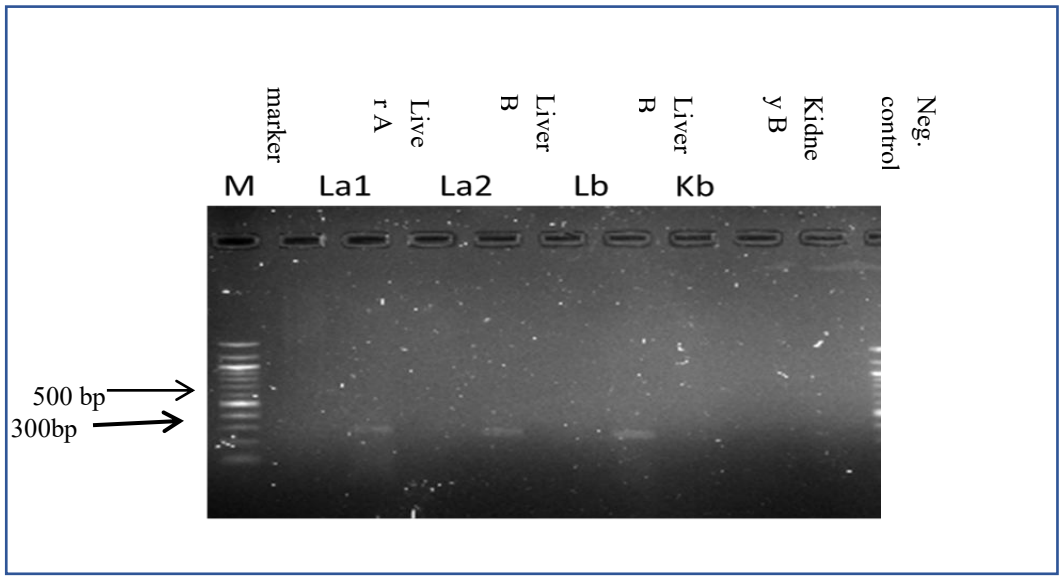

Figure 13: Agarose gel picture of PCR test done on liver and kidney samples using gel-specific primers for pathogenic *Leptospira spp*. Positive bands are seen on liver A1, A2 (dog 1), and Liver B (dog B) for 16S rRNA *Leptospira* gene.

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