

A Monthly e Magazine
ISSN:2583-2212

Popular Article

March, 2025 Vol.5(3), 6689–6684

Riemerellosis : An Overlooked Disease of Ducks

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[DOI:10.5281/ScienceWorld.15516525](https://doi.org/10.5281/ScienceWorld.15516525)

Abstract

“Riemerellosis” is a septicaemic disease of ducks known by different names like New duck disease, duck septicaemia, anatipestifer septicaemia and infectious serositis. In India, the disease has been reported in ducks from Assam, Kerala, Meghalaya and Odisha (Majhi *et al.*, 2020). Riemerellosis is caused by the bacteria *Riemerella anatipestifer* is a member of the *Flavobacteriaceae* family. It is a Gram-negative small capsulated, microaerophilic, nonmotile, nonspore-forming rod that occurs singly, in pairs, and occasionally in chains. There are 21 known serotypes that have been identified. (Li *et al.*, 2023). The disease affects both domestic and wild birds globally, with ducks being particularly susceptible, followed by geese, turkeys, pheasants, partridges and chickens. Transmission is mainly direct, bird-to-bird, via toenail scratches, especially of the duckling foot, or through respiratory epithelium during respiratory disease. Clinical signs of *Riemerella anatipestifer* infection usually develop after an incubation period of 2–5 days and comprise ocular and nasal discharges, mild coughing and sneezing, greenish diarrhoea, tremors of the head and neck, depression and incoordination progressing to loss of consciousness and death. The post-mortem lesions of *R. anatipestifer* are characterized by septicaemia, fibrinous pericarditis and perihepatitis, airsacculitis, fibrinous pneumonia, catarrhal rhinitis and enteritis, enlarged spleen and enlarged liver, caseous arthritis and salpingitis, skin necrosis as well as serous-fibrinous meningitis (Chikuba *et al.*, 2016). The most frequently affected organ is the heart. The epicardium is thick due to the edema, hemorrhage, abundant fibrinous exudates and infiltration of heterophils and macrophages. *R. anatipestifer* grows on the selective blood agar under micro-aerophilic conditions to produce dew drop, small (1–2 mm in diameter), transparent, glistening and non-haemolytic colonies. Confirmative diagnosis of *R. anatipestifer* infection is based on laboratory methods including PCR and gene sequencing. It has been found that outer membrane protein (Omp) A is a major immunogenic protein for *R. anatipestifer* and it is important for the organism virulence even after mutation or attenuation. This protein has been developed for serological detection of all *R. anatipestifer* (RA) serotypes (Li *et al.*, 2023). Various antibiotics are currently used to prevent and control RA infection in ducks, but the abuse and misuse of antibiotics lead to the emergence of drug-resistant strains. Vaccination is currently considered the

best strategy to control the disease in ducks. Inactivated bacterins, live vaccines and concentrated culture filtrates have been reported to be effective for prevention of the disease. However, there is little or no significant cross-protection between the serotypes. (Li *et al.*, 2023).

Keywords: Duck farming, History and Lesions.

Introduction

“Riemer” first described an epizootic septicaemia of domestic fowl in 1904 and subsequently “Riemerellosis” a septicaemic disease of growing ducks assumed worldwide importance. Over the years, this bacterial infection of ducks has been known by different names and this has led to confusion surrounding the incidence and treatment of the Riemerellosis. New duck disease, duck septicaemia, riemerellosis, anatipestifer septicaemia and infectious serositis are different synonyms for the infection of ducks with *Riemerella anatipestifer* (*R. anatipestifer*). *R. anatipestifer* causes septicaemia disease in ducks resulting in serious economic losses through high mortality, reduced growth rate, poor feed conversion and increased condemnations of carcasses (Gong *et al.*, 2020). Given the potential threat of the spread of *Riemerella* bacteria among poultry and the increasingly observed phenomenon of bacterial resistance to antibiotics, it is particularly necessary to know the epidemiology and pathology of the disease.

PATHOGEN

The causative bacterium was isolated and characterized by Hendrickson and Hilbert (1932), who called it *Pfeifferella anatipestifer*. Bruner and Fabricant (1954) observed that the organism had more in common with *Moraxella* sp. and suggested the name *Moraxella anatipestifer*. The bacterium shares phenotypic characteristics with *Flavobacter* spp. and has been historically and incorrectly known as *Moraxella anatipestifer* and *Pasteurella anatipestifer* due to their similar ecology and morphological/cultural characteristics. Comparison of its DNA base composition, DNA-DNA homology, and cellular fatty-acid profile indicated its exclusion from the genus *Moraxella* as well as *Pasteurella*. Its current classification as *Riemerella anatipestifer* was established by Segers and his colleagues, working in Belgium in 1993 by Perez (2016).

Morphology

R. anatipestifer is a member of the *Flavobacteriaceae* family. It is a Gram-negative bacteria, microaerophilic, non-motile, nonspore forming rod that occurs singly, in pairs and occasionally in chains (Gong *et al.*, 2020). The cells vary from 0.2 to 0.4 mm in width and 1 to 5 mm in length. Many cells stain bipolar with Wright’s stain, and a capsule can be demonstrated in preparations with India ink. The organism grows well on chocolate agar, blood agar or trypticase soy agar. Colonies on blood



agar, when grown for 24-48 hours at 37°C in a candle jar, are 1–2 mm in diameter, convex, entire, transparent, glistening and butyrous.

R. anatipestifer isolates have been serotyped using agglutination and agar-gel precipitin (AGP) reactions. Both of these tests involve surface antigens that are presumed to be polysaccharides. Plate agglutination is rapid and convenient, tube agglutination is favored over AGP as it is quantitative in terms of antibody titers. There are 21 known serotypes that have been identified but the lack of cross-protection between different serotypes leads to poor immunisation effects with existing vaccines (Li *et al.*, 2023). Virulence factors of *R. anatipestifer* include the capsule-capsular polysaccharide (CPS), lipopolysaccharides, gelatinases, the type IX secretion system (T9SS), outer membrane proteins (OMPs), and other putative virulence factors, which are associated with the viability of this bacterium in blood (Li *et al.*, 2023). Outer membrane proteins play a very important role in virulence and induce a strong antibody response. Omp A gene of *R. anatipestifer* is an important target to differentiate, between *R. anatipestifer* and other bacterial species.

Host Range

The disease affects both domestic and wild birds globally, with ducks being particularly susceptible, followed by geese, turkeys, pheasants, partridges and chickens. The bacterial infection in addition has also been reported from pigs.

History and Geographical Distribution

R. anatipestifer infection was first described in 1932 in Pekin ducks from three farms on Long Island, New York. The report referred to a new disease, which became known in the area as New duck disease. The disease started in 7 to 10-week-old ducks with about 10% mortality and later spread to younger ducklings of about 3 weeks of age. Six years later, the disease was observed in ducks from a commercial farm in Illinois and was reported as duck septicemia. The designation infectious serositis was given by Dougherty and coworkers after a comprehensive pathologic study. In the past *R. anatipestifer* infection has been reported from many countries that produce ducks, geese and turkeys. *R. anatipestifer* pathogenic infections in domestic ducks (*Anas platyrhynchos*) have been reported in Japan.

The disease is widespread in intensive duck producing areas of China, Thailand, Taiwan, USA, UK, Germany and Hungary. The infection has also been reported from Canada, Denmark, Italy, France, Netherlands and Australia. Sporadic cases have been reported from India, Bangladesh, Singapore, Israel, Korea, Egypt, Russia and New Zealand.



In India, the disease has been reported in ducks from Assam and Kerala. Although, *Riemerella anatipestifer* infection in an organized duck farm of Kerala with 12.5 % mortality and an outbreak in Meghalaya with 16 % mortality were reported but epidemiological investigations have not been undertaken to unravel the extent of the disease in India.

Transmission

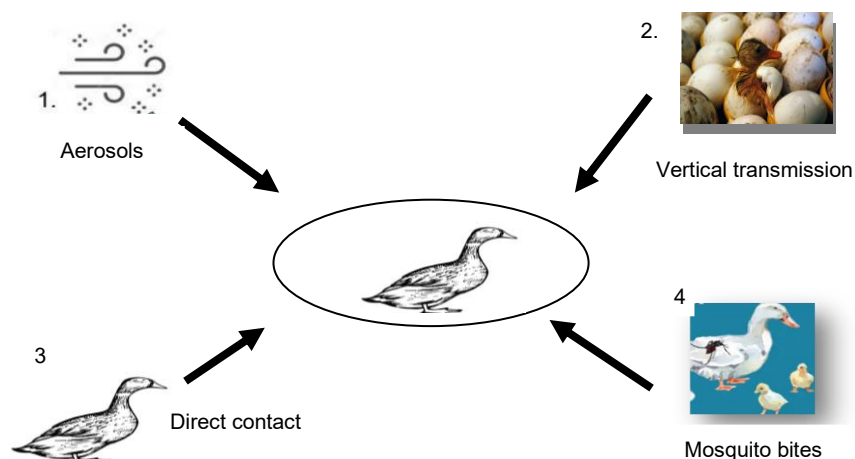


Fig: 01 Different routes of transmission of a *R. anatipestifer*

Transmission is mainly direct, bird-to-bird via toenail scratches, especially of the duckling foot, or through respiratory epithelium during respiratory disease. It can also be by faecal contamination of feed, water or the environment where survival of the infectious agent may be prolonged. Adverse environmental conditions and pre-existing disease are predisposing factors (Chikuba *et al.*, 2016).

R. anatipestifer have been isolated from pharyngeal mucosa of clinically normal ducklings. Some scientists regarded *R. anatipestifer* as a normal inhabitant microflora in the upper respiratory tract of some domestic and wild ducks. Infection takes place horizontally via the birds respiratory tract or through skin wounds, particularly on the feet. Vertical infection through ovaries or oviducts is controversial.

Pathogenesis

Although the pathogenesis is poorly understood ducks get infected from the environment by the respiratory route or when *R. anatipestifer* is introduced into lesions of the webbed foot. Turkeys may be infected by injuries or by the respiratory route when another pathogen disrupts the respiratory

epithelium. Once the infection is established on a farm, it frequently becomes endemic. It is common for multiple serotypes to be present on a single facility, with co-infections possible.

Many outer membrane proteins in pathogenic bacteria are virulence factors that enable or facilitate bacterial attachment to host cell surfaces, as part of the pathogenicity process (Caruana and Walper, 2020). Several *R. anatipestifer* virulence factors have been identified, including outer membrane protein A, glycosyltransferase VapD, CAMP cohemolysin, nicotinamidase PncA, and iron acquisition protein SprA.

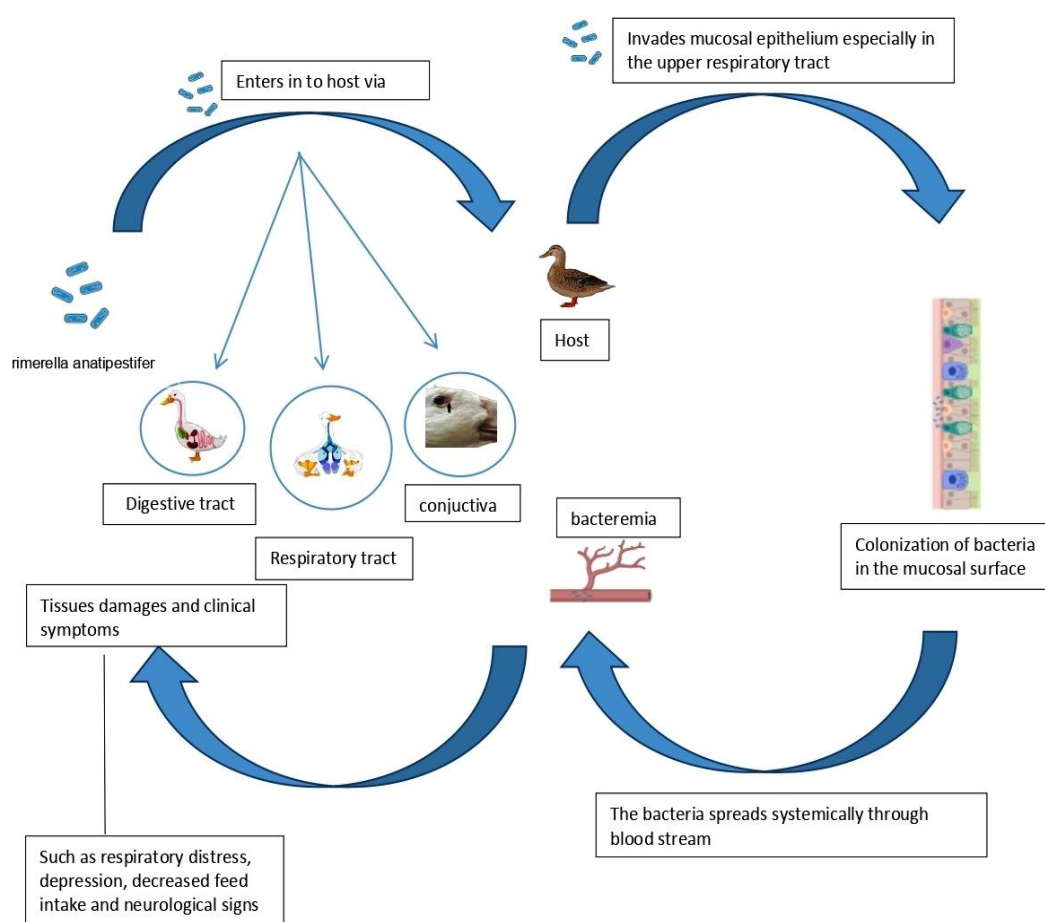


Fig 02 : Pathogenesis of *Riemerella anatipestifer*

Interaction between bacteria and host :

Outer membrane receptors, also known as TonB-dependent (TBdRs) receptors, are a family of proteins named for their localization in the outer membrane of gram-negative bacteria. Thirty-one TBdRs have been identified in *R. anatipestifer*, which may play important roles in the interaction between this bacterium and the host. External factors needed by bacteria, include iron, zinc, and copper ions and haem that enter the cytoplasm through the active transport of TBdRs, thereby maintaining



normal cellular activity. Outer membrane proteins of Gram-negative bacteria are known virulence factors and pathogenic species use these surface proteins to recruit complement regulators, including vitronectin (Vn), C4 binding protein, and complement factor H for immune escape. The Vn protein is an inhibitor of the Membrane attack complex system via the lytic pathway, thereby hindering the terminal pathway of complement activation. Moreover, Vn also plays important roles in mediating bacterial adhesion, cell signalling, and complement escape in pathogenic bacteria (Li *et al.*, 2023). Although the specific tactics used by *R. anatipestifer* to circumvent the bactericidal effect of complement are uncertain however it is established that the OMP76 acts as a virulence factor in *R. anatipestifer* and a complement escape-associated OMP that plays an important role in pathogenesis by this bacterium (Li *et al.*, 2023). Nevertheless, the molecular pathogenic mechanisms of *R. anatipestifer* are still enigmatic.

CLINICAL SIGNS

Clinical signs of *Riemerella anatipestifer* infection usually develop after an incubation period of 2–5 days. Affected ducklings, usually 1–7 weeks old, often have ocular and nasal discharges, mild coughing and sneezing, greenish diarrhoea tremors of the head and neck, depression, and incoordination progressing to loss of consciousness and death. In typical cases, affected ducklings in the terminal stages of disease lie on their backs, paddling their legs. Mortality may vary from 5% to 75%, Morbidity is usually higher.



FIG. 03: Affected Birds Showing Greenish White Diarrhoea (A), Ocular and Nasal Discharge (B), Tremors of Head and Neck (C), Mucous Exudates in the Oral Cavity (D), And Necrotic Dermatitis.



PATHOLOGY

Riemerellosis (RA) is a bacterial septicaemia and the lesions in affected birds may resemble *streptococcus* or *E. coli* infections.

GROSS LESIONS

The post-mortem lesions of *R. anatipestifer* are characterized by septicaemia, fibrinous pericarditis perihepatitis and airsacculitis, Fibrinous pneumonia, catarrhal rhinitis and enteritis, enlarged spleen and enlarged liver, caseous arthritis and salpingitis, skin necrosis as well as serous-fibrinous meningitis (Leibovitz, 1972 and Chikuba *et al.*, 2016). In addition edematous swelling around the tibio-tarsal joints of affected birds was reported by Tzora *et al.* (2021) The lesions of *R. anatipestifer* in ducks is similar to other bacterial infections like *Pasteurella multocida*, *Escherichia coli* and *Salmonella enterica*. Therefore, it is difficult to diagnose *R. anatipestifer* infection through the pathological features.

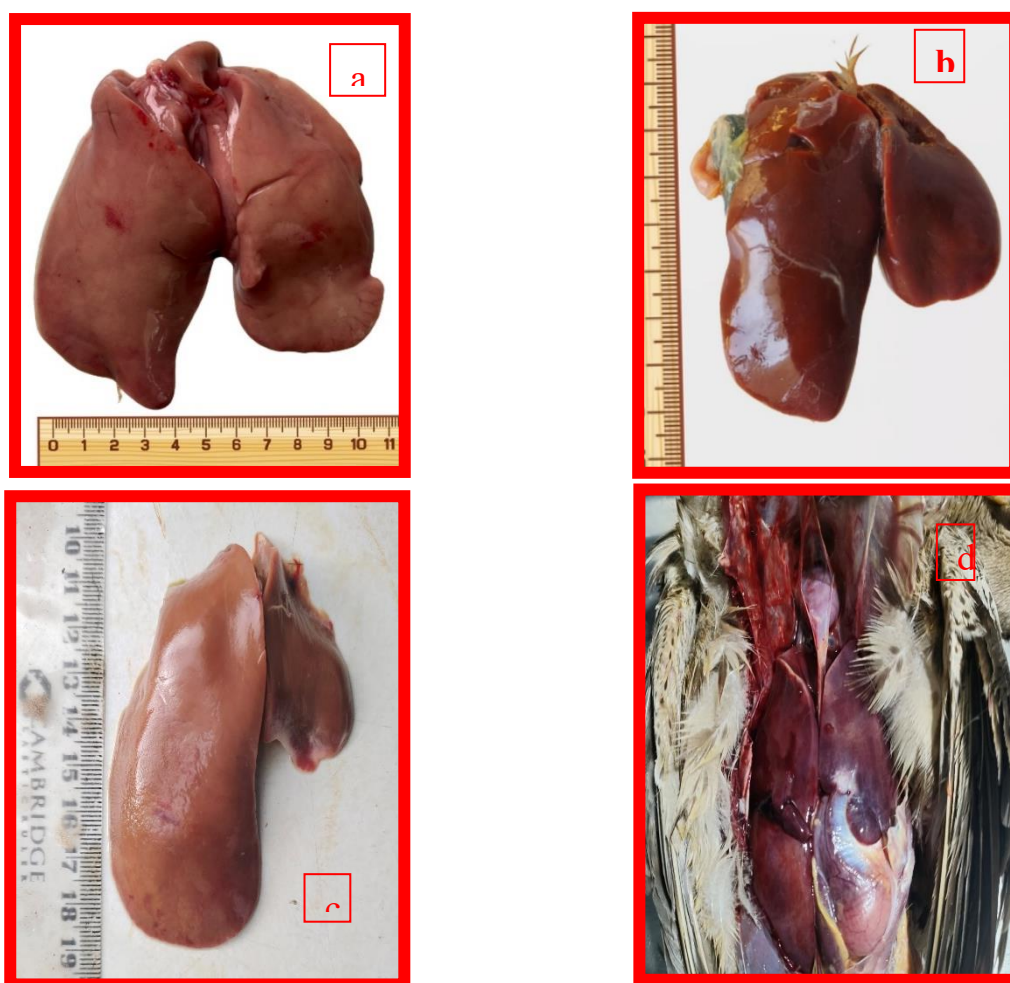


FIG. 04: Liver Showing Focal Area of Petechial to Ecchymotic Haemorrhage, Congestion and Hepatomegaly (Arrow) (A), Mild Fibrinous Perihepatitis (Arrow) (B), Fibrosis of Left Lobe of Liver (Arrow) (C & D).



HISTOPATHOLOGY

Histologically, heterophilic and fibrinous inflammation are observed at the serosal surface of the heart, liver, air sac and intestine. The most frequently affected organ is the heart. The epicardium is thick due to the edema, hemorrhage, abundant fibrinous exudates, and infiltration of heterophils and macrophages. Fibrinous exudates are found to be often surrounded by multinucleated giant cells. In acute cases, severe focal necrosis of the heart muscle is present. Lesions of the liver, air sac and intestine are essentially similar to those of the heart. Liver lesions observed in the acute stage of the disease are mild periportal mononuclear leukocytic infiltration, cloudy swelling, and hydropic degeneration of parenchymal cells. In less acute cases, moderate periportal lymphocytic infiltration may be observed. Fatty degeneration and some degenerated and necrotic hepatocytes were reported in livers of diseased ducks. Focal to massive hepatocellular necrosis and increased numbers of macrophages in the spleen with occasional fibrin thrombi are also reported.. In air sacs, mononuclear cells are the predominant cell type in the exudate. Multinuclear giant cells and fibroblasts may be observed in chronic cases Gram-negative stained bacterial colonies may be found in the fibrinous exudates. The lungs of infected ducks may be unaffected or at times there may be interstitial cellular infiltration and proliferation of lymphoid nodules adjacent to parabronchi, or there may be an acute fibrinopurulent pneumonia. Heterophilic and granulomatous bronchopneumonia have been reported in the most severe cases. Another characteristic lesion observed is heterophilic and fibrinous ventriculitis and meningitis in the brain. Brain lesions are noticed to be more severe in the brain stem, including the optic lobe, than in the cerebrum, cerebellum and spinal cord.

Currently, the disease is mostly a mixed infection or a secondary infection following pathogens infection in the clinic, such as influenza virus. In a study, it was found that *R. anatipestifer* infection caused damage to the intestinal system of ducks, affecting the intestinal immune barrier and causing a decrease in immunity, and then the ducks can be infected with *E. coli* and duck circovirus.

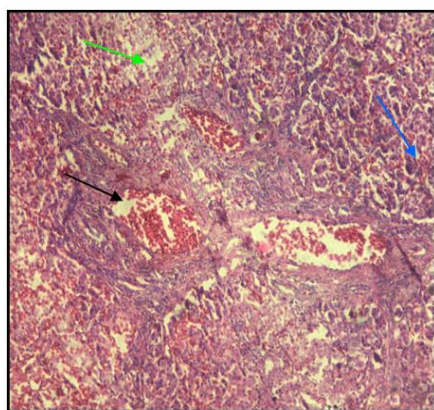


FIG. 05: PHOTOMICROGRAPH SHOWING HAEMORRHAGE (BLUE ARROW), CONGESTION (BLACK ARROW) AND NECROSIS OF LIVER PARENCHYMA (GREEN ARROW) (H & E 10X)

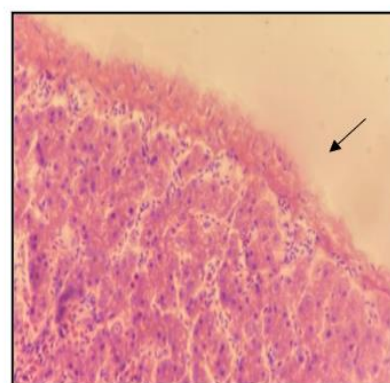


FIG.06: PHOTOMICROGRAPH SHOWING DEPOSITION OF FIBRINOUS EXUDATES ON THE CAPSULAR SURFACE (BLACK ARROW) OF THE LIVER AND INFILTRATION OF MONONUCLEAR CELLS (RED ARROW) (H & E 40X)

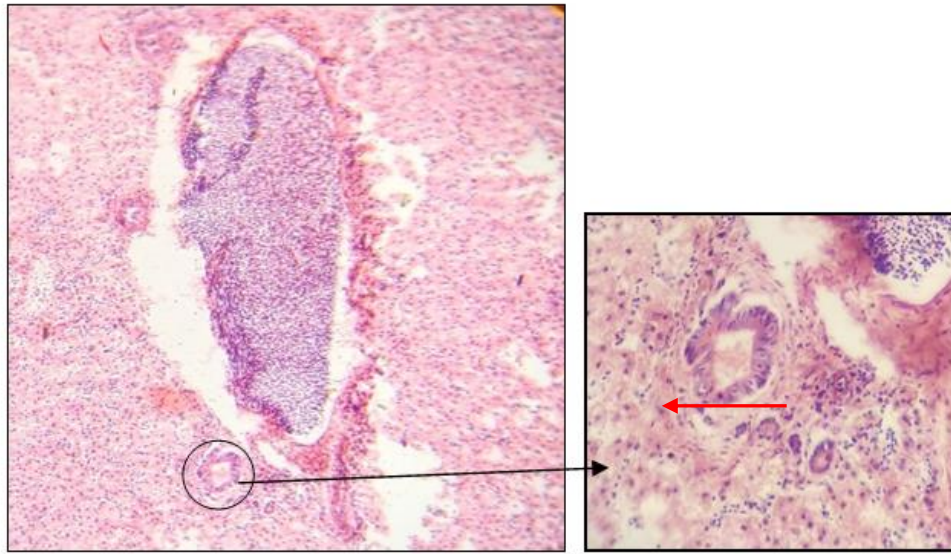


FIG. 07: PHOTOMICROGRAPH SHOWING HYPERPLASIA OF BILE DUCT ALONG WITH SEVERE CONGESTION AND HAEMORRHAGE OF LIVER PARENCHYMA (H & E 10X) (ARROW) (H & E 40X)

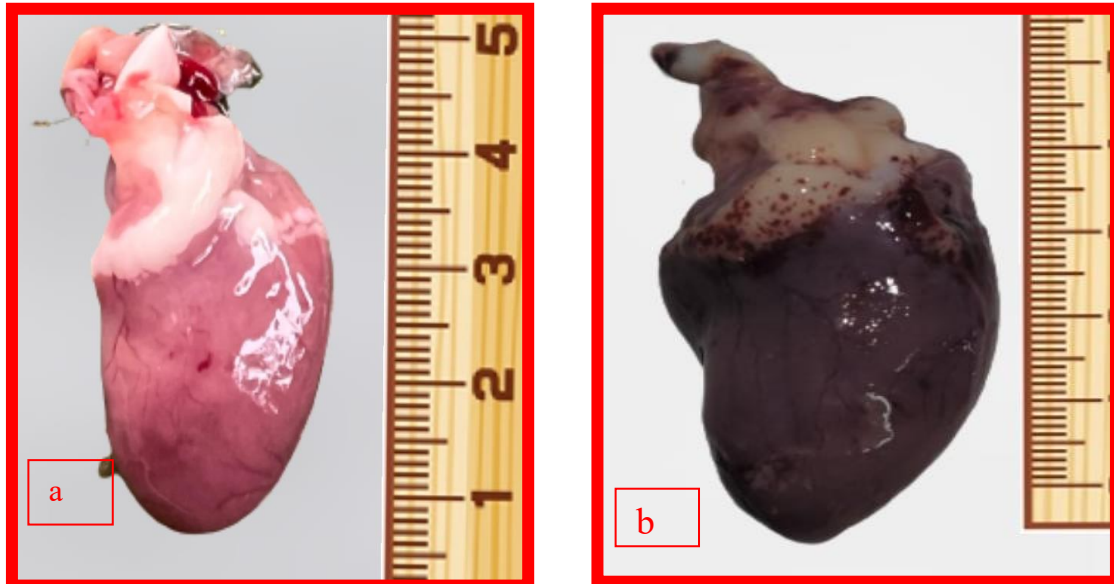


FIG. 08 : HEART SHOWING ENLARGEMENT DUE TO DILATATION (BLACK ARROW) (a), FOCAL PETECHIAL TO ECCHYMOTIC HAEMORRHAGE (RED ARROW) AND CONGESTED BLOOD VESSELS (a & b)



LABORATORY DIAGNOSIS

Confirmative diagnosis of *R. anatipestifer* infection is based on laboratory methods.

Cultivation and identification of *R. anatipestifer*.

Molecular methods.

➤ Differential diagnosis

R. anatipestifer infection should be differentiated from other septicemic diseases caused by *Pasteurella multocida*, *Coenonia anatina*, *Escherichia coli*, *Streptococcus faecium* and *Salmonellae*. Because these diseases produce gross lesions indistinguishable from those caused by *R. anatipestifer*, diagnosis must include isolation and identification of the causal organism. Differential diagnosis should also include chlamydiosis, especially in turkeys and in areas where the latter is a serious problem.

Treatment

Antibiotics are still an effective treatment approach for *R. anatipestifer* infection. However, the abuse and/or misuse of antibiotics have led to the emerged and evolved of bacterial resistance including multidrug resistance (MDR), extensive drug resistance, and pandrug resistance, which has seriously hampered the treatment of bacterial diseases. Various antibiotics are currently used to prevent and control RA infection in ducks, but the abuse and misuse of antibiotics lead to the emergence of drug-resistant strains.

Currently, *R. anatipestifer* has developed resistance to a variety of antibiotics, such as chloramphenicol, florfenicol, aminoglycosides, fluoroquinolone, posing a serious threat to the treatment of *R. anatipestifer* infection (Shousha *et al.*, 2021; Lyu *et al.*, 2023). Broad-spectrum cephalosporin ceftiofur decreased mortality in experimentally infected ducklings after a single subcutaneous dosage of 2 mg/kg bodyweight 5 hours post-infection.

It is advocated that Sulfaquinoxaline or a combination of penicillin and streptomycin can be used for initial treatment. However, antimicrobial susceptibility testing should be performed because multidrug-resistant strains are becoming more prevalent due to antimicrobial use and development of antimicrobial gene resistance (AGR). Horizontal gene transfer of ARGs across bacteria has gradually become the main mode of spread of antibiotic resistance (Lyu *et al.*, 2023).

Studies conducted by (Yang *et al.*, 2024), demonstrated the severe resistance status of *R. anatipestifer* and the extremely high carriage of resistance genes. Their observations highlight the



prevalence of *tet(X)* and beta-lactamase genes in *R. anatipestifer* and reveal the uneven distribution of resistance genes among lineages.

Control and prevention

Improved biosecurity

An important factor in preventing infection is good management and sanitation. The risk of contracting *R. anatipestifer* infection is higher in ducklings raised in stressful environments. Maintaining hygienic conditions is crucial when housing limited flocks of various ages. Depopulating the entire house is necessary for a thorough cleaning and disinfection.

Vaccination

As a result of increasing the emergence of drug-resistant strains of *R. anatipestifer* alternative measures such as vaccination has been encouraged. Inactivated, living attenuated and sub-unit vaccines are currently used to prevent *R. anatipestifer* infections in ducks farms. Inactivated vaccines have been used to prevent or reduce ducks mortalities and to develop serotype specific immunity. Proper autogenous vaccines can protect ducks from infection. The protective efficacy of the vaccine depends mainly on the used strains and the protection developed only against the homologous. The serotypes of *R. anatipestifer* present in any vaccine showed no cross-protection with other serotypes. The frequent changes of serotypes in the farms and the presence of more than one serotype in one farm make problems in application of vaccines against *R. anatipestifer*. Therefore, the vaccines should contain all the predominant *R. anatipestifer* serotypes to provide effective broad-spectrum protection. Multivalent inactivated vaccines have been used for the prevention of *R. anatipestifer* in ducks, especially against serotypes 1 and 2. Moreover, inactivated *R. anatipestifer* vaccine containing levamisole and chaperonin GroEL as adjuvants were successfully protected ducks from the infection. It is important to note that the immune response of vaccinated ducklings at very young age can interfere with the maternal immunity.

CONCLUSIONS

R. anatipestifer infection causes significant losses for the global duck business. Despite a great deal of research on this type of sickness, flocks are still afflicted with the disease. The emergence of medication resistance poses significant challenges in the management of *R. anatipestifer*. Furthermore, there is no cross-protection across the various serotypes and the existing vaccinations only produce homologous immunity. Therefore, more studies and research are required to develop treatment and immunisation procedures against *R. anatipestifer*. Proper and rapid identification of *R. anatipestifer* with detection of their antimicrobial susceptibility and its virulence potential is essential for



understanding the epidemiology of *R. anatipestifer* and to apply the effective control strategies. Finally, a continuous and frequent monitoring of antimicrobial resistant is necessary to determining the most effective antibiotic for the control of *R. anatipestifer* duckling infections.

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