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ANALYSIS OF ETIOLOGICAL, CLINICAL MANIFESTATIONS AND GROSS LESIONS ASSOCIATED WITH YOUNG PIGEON DISEASE IN A PIGEON LOFT OUTBREAK

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Abstract

Young Pigeon Disease Syndrome (YPDS) is a multifactorial condition that poses a significant health challenge to young pigeons, particularly those between the ages of 3 and 12 weeks post-weaning. In rare cases, it can also affect older pigeons. This syndrome is characterized by high morbidity and mortality, leading to considerable losses in the pigeon breeding and racing industries. The primary viral agent responsible for YPDS is pigeon circovirus (PiCV), known for its immunosuppressive effects, which increase susceptibility to secondary infections caused by Escherichia coli and other opportunistic pathogens like Candida albicans and Trichomonas gallinae. Additionally, pigeon aviadenovirus (PiAdV) and Columbid herpesvirus-1 (CoHV-1) are associated with the syndrome, contributing to its complex pathogenesis. Clinical signs of YPDS are non-specific and include lethargy, weight loss, vomiting, diarrhoea, and respiratory distress. The disease is most severe in juvenile pigeons, with rapid progression often leading to death within 3 to 5 days, while adult birds that develop clinical signs may take up to 8 days to die.

Key words*: pigeon, young pigeon disease syndrome, pigeon circovirus, pigeon adenovirus, co-infection.*

INTRODUCTION

Young Pigeon Disease Syndrome (YPDS) represents a significant health challenge in the avian world, particularly affecting young pigeons between the ages of 3 to 12 weeks (Raue et al., 2005). This multifactorial disease is characterized by high morbidity and mortality rates, often leading to severe losses in pigeon breeding and racing industries (Raue et al., 2005). The ethology of YPDS is complex, involving various viral, bacterial, and parasitic infections that can compromise the immune system of affected birds (Raue et al., 2005).

Among the viral agents implicated, the pigeon circovirus (PiCV) has emerged as a primary contributor to the syndrome, due to its immune-suppressive properties, increasing susceptibility to secondary infections and overall poor health outcomes in young pigeons (Sahindokuyucu et al., 2022; Stenzel & Pestka, 2014; Cságola et al., 2011).

Furthermore, there is pigeon aviadenovirus (PiAdV) and Columbid herpesvirus-1 (CoHV-1) that can also contribute to the onset and progression of YPD (Sahindokuyucu et al., 2020; Abdulrasool et al., 2022).

Pigeon circovirus (PiCV) was first identified as a potential cause of YPDS in the early 1990s, prompting extensive research into its role in disease pathogenesis. PiCV is now recognized as globally widespread, frequently detected in flocks with clinical signs of YPDS (Stenzel & Pestka, 2014; Stenzel & Koncicki, 2017). Studies have also documented significant genetic diversity among PiCV strains, suggesting varying levels of virulence and pathogenicity, which complicates both diagnosis and treatment (Cságola et al., 2011; Stenzel et al., 2014).

In addition to viral agents, bacterial pathogens like Salmonella spp. and Escherichia coli contribute to the progression of YPDS. These bacteria can worsen clinical symptoms, leading to severe gastrointestinal and systemic infections in young pigeons (Stenzel et al., 2014; Al-jumaili, 2023). Mixed infections involving multiple pathogens are associated with increased disease severity, highlighting the need for a comprehensive diagnostic approach by veterinarians (Hamouda et al., 2017; Nath et al., 2023).

Among the diverse pathogens associated with YPDS, adenoviruses have emerged as significant contributors to the disease's pathogenesis.
Adenoviruses are non-enveloped, double-stranded DNA viruses that can infect various avian species, including pigeons. Their involvement in YPDS has attracted growing research attention, particularly due to the intricate interactions between adenoviral infections and other co-infecting pathogens, such as bacteria and additional viruses (Vereecken et al., 1998).
The clinical presentation of adenoviral infections in pigeons varies significantly based on the host's age and immune status. In juvenile pigeons, adenovirus infections often result in severe gastrointestinal disturbances, immune-suppression and systemic illness, key features of YPDS. In pigeons older than one year, adenovirus infections typically lead to necrotizing hepatitis (Vereecken et al., 1998).
The clinical manifestations of YPDS range from lethargy and weight loss to severe respiratory and neurological symptoms.
Without prompt intervention, the disease progresses rapidly, often leading to high mortality (Sahindokuyucu et al., 2022; Stenzel & Koncick, 2017).
Effective management of YPDS involves identifying causative agents and implementing preventive strategies, such as vaccination and biosecurity measures, to minimize outbreak risks in pigeon lofts (Stenzel et al., 2012; Stenzel & Koncicki, 2017).
Research has shown that environmental stressors related to racing and breeding can weaken immune responses in young pigeons, increasing their susceptibility to infections and YPDS (Zigo et al., 2017; Zigo et al., 2019). Additionally, the presence of zoonotic bacteria underscores the public health implications of YPDS, beyond its impact on avian health (Teske et al., 2013; Karim et al., 2020).

MATERIALS AND METHODS

Research materials

Samples were collected over one year (2023-2024) from a breeding facility in southwest Bucharest. Ten pigeons 7 males and 3 females were examined, including 7 juveniles (3 to 12 weeks old) and 3 pigeons older than 1 year. All pigeons were closely monitored to prevent external factors or unauthorized medications from affecting the study.
Ante-mortem the pigeons were closely monitored for any sign of illness. Initially, we were looking for birds that refused to eat with all the others, birds that had a bad posture or any respiratory distress. Clinical examination continued without removing the birds from the loft for better understanding their behavior in distress.
The necropsy began with an examination of external signs such as fluid discharge, pectoral muscle condition, and feather abnormalities. Upon entering the coelomic cavity, attention focused on the macroscopic appearance of the viscera, particularly the liver, while all internal organs were examined for notable findings.

Sampling method

Real time PCR was used to identify the viral portion of disease like pigeon circovirus (PiCV), pigeon aviadenovirus (PiAdV), Columbid herpesvirus-1 (CoHV-1) and also for the diagnosis of *Trichomonas galinae*. For RT-PCR testing, cloacal swabs (sterile swabs without medium) were taken, and also pieces of liver, spleen, trachea, heart, lung, kidneys, small and large intestines were preserved in tightly sealed sterile containers and sent. The PCR test was performed in an external laboratory and the sample collection, and transport were done accordingly to the laboratory's recommendations.
Swabs for bacteriological and mycological cultures were collected, and tissue samples from previously excised organs were used for aerobic, anaerobic, and mycological cultures.
For the detection of aerobic bacteria and respiratory tract swabs, Columbia agar, Endo agar, and Chocolate agar were used.
For stool samples, Columbia agar, MacConkey agar, Hektoen agar, and XLD agar were employed. Additionally, XLD and Chromagar were used for the enrichment of *Salmonella* detection.
For the cultivation of anaerobic bacteria, we used Schaedler and Schaedler KV agar plates.
The methodology for mycological cultures varied based on the sample source:
For respiratory tract samples, Sabouraud agar supplemented with chloramphenicol and gentamicin, as well as malt extract agar, were used.

For stool samples, Sabouraud agar with gentamicin and chloramphenicol, along with Chromagar for yeasts, were employed.
Matrix-Assisted Laser Desorption Ionization–Time of Flight Mass Spectrometry (MALDI-TOF MS), a valuable tool in veterinary medicine, was utilized for the identification of various bacterial and fungal species.

RESULTS AND DISCUSSIONS

The general clinical signs observed in all affected birds included depression, lethargy, reluctance to fly, and varying degrees of weight loss, some leading to emaciation (Figure 3). Other symptoms were vomiting, dark-green droppings, diarrhoea, and an enlarged crop exhibiting signs of stasis with undigested food and water. A fluid-filled crop, as described by Rüdiger Raue and Volker Schmidt, was also noted (Raue et al., 2005).
Additionally, respiratory distress and neurological signs, including an abnormal forward position of the head and neck, were observed in rare cases (Figure 1).

Figure 1. Abnormal position of the head without tremor

Clinical signs varied, with pigeons aged 3 to 12 weeks post-weaning experiencing more severe symptoms and faster progression to death, typically within 2 to 5 days. In contrast, adult pigeons could survive up to 7 or 8 days, often showing severe pectoral muscle emaciation. Most fatalities occurred at night, with birds found in secluded areas of the loft, positioned forward with extended necks and wings by their sides. Gross lesions observed during necropsy are detailed in Table 1.

Table 1. Gross lesions observed during necropsy

Organs and systems	Types of gross lesions	Proportion with lesion
Gastrointestinal system	○ Green to yellow fluid in the crop, proventriculus, ventriculus and intestin.	9/10
	○ Enlarged, fluid with mucus filled crop.	5/10
	○ Proventriculitis, with numerous hemorrhagic spots on the entire mucosa.	7/10
	○ Hemorrhages on the ventricular mucosa.	4/10
	○ Enteritis.	9/10
	○ Necrotic hemorrhagic enteritis.	6/10
	○ Elevated gas content.	2/10
	○ Intestinal obstruction due to a granulomatous mass formation.	1/10
Lungs and air sacs	○ Air sacs covered with yellow mucus.	7/10
	○ Fibrinous airsacculitis.	8/10
	○ Accumulation of yellowish serous fluid in thoracic air-sacs.	4/10
	○ Bilateral enlargement of lungs.	4/10
	○ Fibrosed and dark-red in color lungs.	2/10
	○ Pulmonary hemorrhages.	4/10
Liver and pancreas	○ Liver friable with mild to severe enlargement.	7/10
	○ Discoloration of the liver and pancreas from yellow to red-black.	5/10
	○ Liver and pancreas with diffuse punctate to sever hemorrhages.	7/10
	○ Multifocal whitish lesions in pancreas.	4/10
	○ Necrosis of the liver and pancreas.	9/10
Heart	○ Multifocal submiliar white lesions in the myocardium.	2/10
	○ Serous pericarditis with a clear fluid or cloudy yellow liquid.	4/10
Kidneys	○ Diffuse green in colour.	3/10
	○ Diffuse yellow in colour.	2/10
Lymphatic system	○ Splenomegaly.	3/10
	○ Necrotic hemorrhage of the spleen.	4/10
	○ Atrophy of the Bursa of Fabricius, in some cases severe.	4/10

Cloacal feathers were clumped with faecal matter, and some birds exhibited yellow-green secretions from the oral and nasal cavities (Figure 2).

Figure 2. Yellow watery liquid with mucus flowing from the oral cavity post-mortem

Various degrees of emaciation of the pectoral muscles were observed during the external examination (Figure 3 a). Additionally, some birds were in the process of moulting, with new follicles emerging. Post-mortem examination revealed a significant loss of subcutaneous connective adipose tissue and generalized muscle atrophy (Figure 3 a, b).

Figure 3.(a) Deformation of the sternum together with sternal amyotrophy. (b) Sternal amyotrophy

In all examined cases, liver involvement was substantial, with hepatomegaly being the most common finding, accompanied by discoloration, increased friability and haemorrhagic necrosis (Figure 4). In several cases, multifocal whitish lesions were identified in the pancreas (Figure 4).

Focal haemorrhages on the ventricular mucosa (Figure 5) were observed in some cases; however, haemorrhagic proventriculitis, characterized by numerous haemorrhagic spots dispersed across the entire mucosal surface (Figure 5), was more frequently documented. The proventriculus and ventriculus were often filled with a yellow-green fluid and mucus.

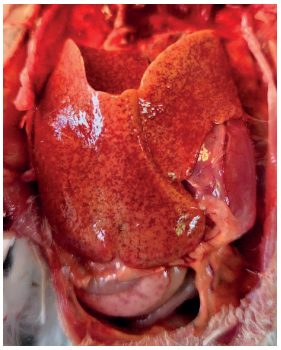

Figure 4. Liver with hepatomegaly, widespread discoloration indicating severe necrosis and diffuse punctate hemorrhages, which can also be observed on the pancreas

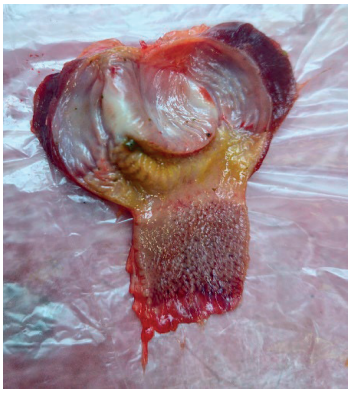

Figure 5. Proventriculitis with numerous hemorrhagic spots on the entire mucosa and focal hemorrhages on the ventricular mucosa

Results following necropsy and laboratory examination are presented in Tabel 2.

Out of 10 pigeons, only 1 was not positive for PiCV and it was a 3 years old female. It showed poor condition for a few weeks without any concrete signs of illness. Once clinical signs appeared, the bird's condition deteriorated rapidly, leading to death within 4 days, marked by progressive weight loss. Some articles suggest that pigeons older than 1 year may be able to clear the infection without exhibiting clinical signs (Abdulrasool et al., 2022).

Table 2. Age-related distribution of pathogens

Pathogen	3-12 weeks old	older than 1 year
PiCV	7/7	2/3
PiAdV	2/7	0/3
CoHV-1	0/7	0/3
Escherichia coli	7/7	3/3
Enterococci	3/7	0/3
Enterobacter hormaechei	1/7	0/3
Chlamydophila spp.	1/7	0/3
Aeromonas caviae	1/7	0/3
Acinetobacter baumannii	1/7	0/3
Candida sp.	4/7	2/3
Aspergillus flavus	0/7	1/3
Mucor sp.	1/7	0/3
Trichomonas gallinae	1/7	0/3
Ascaridia columbae	0/7	2/3

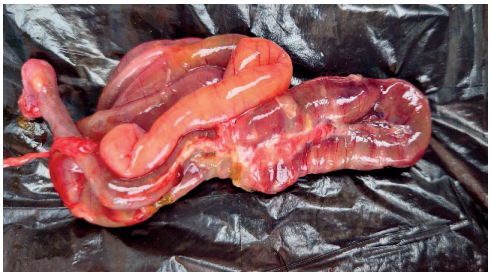

Figure 6. Impacted intestinal tract with *Ascaridia columbae*. Necrotic hemorrhagic enteritis and pancreatitis

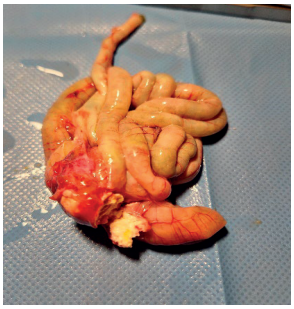

Figure 7. Intestinal tract showing enteritis and elevated gas content with a yellowish white granulomatous mass formation in the duodenum adhering to the cell wall causing obstruction

Haemorrhagic enteritis and pancreatitis (Figure 6) were frequently observed among the lesions. Additionally, the pancreas exhibited necrosis, while intestinal stiffness (Figure 6) was also noted, primarily due to parasitic infestations, including *Ascaridia columbae*.

In one instance, a yellowish granulomatous tumour mass adhering to the intestinal wall (Figure 7) was observed, resulting in intestinal transit obstruction. The intestinal tract was distended with gas, and the intestinal wall appeared thin, displaying signs of inflammation and haemorrhage (Figure 7).

The pericardium, along with the thoracic air sacs, exhibited inflammation, accompanied by moderate to substantial accumulations of transparent or slightly yellowish serous fluid (Figure 8). Furthermore, multifocal submiliary white lesions were observed in the myocardium on two separate occasions.

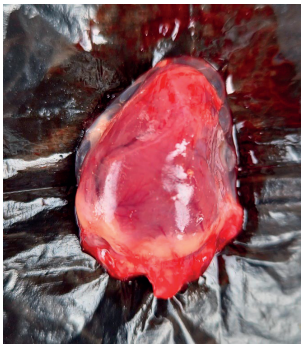

Figure 8. Serous pericarditis with a clear fluid

In relation to the lungs, gross lesions such as mild bilateral enlargement and the presence of a fibrinous covering on the serosal surfaces (Figure 9 a, b) were observed with increased frequency. Additionally, severe bilateral haemorrhages (Figure 9 a, b) were noted, originating centrally and progressively extending radially to involve the entire lung (Figure 9 b).

In one case, pulmonary congestion caused a complete discoloration of the lungs, rendering them dark red internally (Figure 9 a), while a fibrinous layer on the serosal surfaces contributed to a white-grey appearance (Figure 9 a).

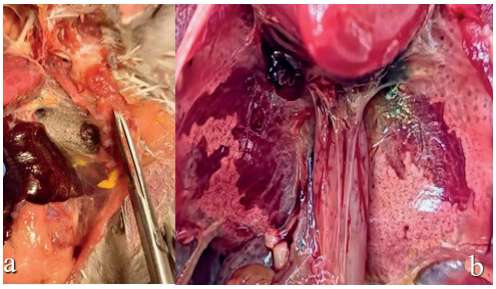

Figure 9. Pericarditis and airsacculitis with a yellowish, fibrinous covering, present on the serosal surfaces of the air sacs and heart. (a) The lung is fibrosed and gray in color on the outside and on the inside the color is dark-red indicating congestion and edema. (b) The lung shows bilateral, extensive hemorrhagic areas, with irregular edges localized predominantly centrally

Together with the liver, the air sacs exhibited the most frequent pathological changes. Predominantly, these alterations included fibrinous aerosacculitis characterized by enhanced adhesion of the air sacs to adjacent cavity organs and the accumulation of yellowish serous fluid (Figure 10). In severe cases, a yellowish, fibrinous exudate was observed on the serosal surfaces of the air sacs and various organs. These lesions are indicative of an *E. coli* infection.

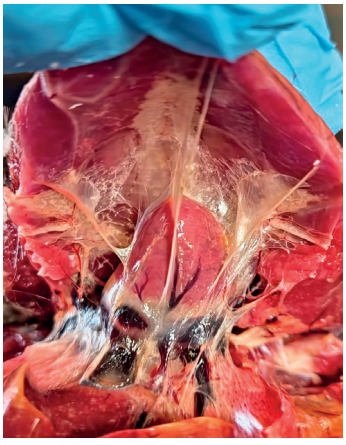

Figure 10. Fibrinous airsacculitis and pericarditis with increased adhesion of the air sacs to cavity organs and accumulation of yellowish serous fluid

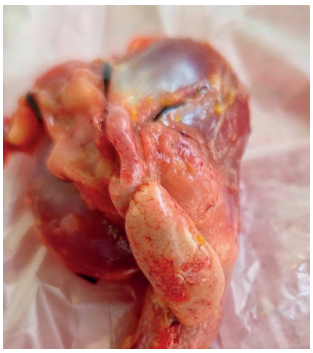

Figure 11. Splenomegaly with discoloration and petechial hemorrhages

The spleen was generally of normal size; however, in some cases, it was observed to be enlarged and exhibited a yellowish-white discoloration, with or without petechial hemorrhages (Figure 11). In certain instances, despite appearing of normal size, necrosis was present.

Examination of the Bursa of Fabricius revealed atrophy, with some cases demonstrating severe atrophy, thereby underscoring the immunosuppressive impact of circovirus.

Figure 12. Opened intestine with underlying hemorrhagic enteritis showing yellowish content and presence of *Ascaridia* worms

Several birds were affected by more than one secondary pathogen, either bacteria, fungi or parasites (Figure 12), highlighting the multifactorial nature of this pathology and its complexity.

CONCLUSIONS

This study confirms that YPDS primarily affects juveniles between 3 and 12 weeks post-weaning, with non-specific clinical signs.

Adults can also develop symptoms, which can sometimes lead to death.

Major stressors such as transportation, overcrowding, and moulting can contribute to the onset and spread of the disease.

PiCV was identified as the primary viral pathogen in 9 out of the 10 cases, sometimes co-infecting with PiAdV, while CoHV-1 was not detected. *Escherichia coli* was the most common co-pathogen, isolated from all samples, while other opportunistic pathogens like *Candida albicans* and *Trichomonas gallinae* suggested a compromised immune system.

The study highlights the multifactorial nature of YPDS, with PiCV playing a key role in its pathogenesis.

REFERENCES

Abdulrasool, M. T. A., Khaleefah, A., & Najem, H. A. (2022). A brief review of pigeon circovirus infection worldwide. *Multidisciplinary Reviews*, *5*(4), 2022018-2022018.

Al-jumaili, M. (2023). Pharmacokinetic profile of norfloxacin in pigeons. *Revista De Ciências Agroveterinárias*, 22(3), 470-474.

Cságola, A., Lőrincz, M., Tombácz, K., Wladár, Z., Kovács, E., & Tuboly, T. (2011). Genetic diversity of pigeon circovirus in hungary. *Virus Genes*, 44(1), 75-79.

Hamouda, E., Mohamed, N., Eid, A., Ismail, A., El-Sisi, M., Hassanin, O., … & Abou-Hashem, N. (2017). Utilization of rt-pcr and restriction enzyme analysis in detection and differentiation of pigeon paramyxovirus-1 and newcastle disease virus in pigeons. *Zagazig Veterinary Journal*, 45(Supplementary 1), 37-47.

Karim, S., Islam, M., Sikder, T., Rubaya, R., Halder, J., & Alam, J. (2020). Multidrug-resistant *Escherichia coli* and *Salmonella* spp. isolated from pigeons. *Veterinary World*, 13(10), 2156-2165.

Nath, B., Das, S., Tidd, N., Das, T., Forwood, J., & Raidal, S. (2023). Lesions and viral loads in racing pigeons naturally coinfected with pigeon circovirus and columbid alphaherpesvirus 1 in australia. *Journal of Veterinary Diagnostic Investigation*, 35(3), 278-283.

Raue, R., Schmidt, V., Freick, M., Reinhardt, B., Johne, R., Kamphausen, L., ... & Krautwald-Junghanns, M. E. (2005). A disease complex associated with pigeon circovirus infection, young pigeon disease syndrome. *Avian Pathology*, *34*(5), 418-425.

Sahindokuyucu, I., Turkmen, M. B., Antepliogˇlu, T., Alcigir, E., YAZICI, Z., Barry, G., ... & Kul, O. (2020). Young Pigeon Disease Syndrome (YPDS) in Turkish Pigeons (*Columba livia domestica*) stemming from coinfection with Pigeon aviadenovirus A and Pigeon circovirus: The first isolation and molecular characterization. *Authorea Preprints*.

Sahindokuyucu, I., Yazıcı, Z., & Barry, G. (2022). A retrospective molecular investigation of selected pigeon viruses between 2018–2021 in turkey. *Plos One*, 17(8), e0268052.

Stenzel, T. and Koncicki, A. (2017). The epidemiology, molecular characterization and clinical pathology of circovirus infections in pigeons – current knowledge. *Veterinary Quarterly*, 37(1), 166-174.

Stenzel, T. and Pestka, D. (2014). Occurrence and genetic diversity of pigeon circovirus strains in poland. *Acta Veterinaria Hungarica*, 62(2), 274-283.

Stenzel, T., Bancerz-Kisiel, A., Tykałowski, B., Smiałek, M., Pestka, D., & Koncicki, A. (2014). Antimicrobial resistance in bacteria isolated from pigeons in Poland. *Polish Journal of Veterinary Sciences*, 17(1), 169-171.

Stenzel, T., Pestka, D., Tykałowski, B., Smiałek, M., & Koncicki, A. (2012). Epidemiological investigation of selected pigeon viral infections in poland. *Veterinary Record*, 171(22), 562-562.

Stenzel, T., Piasecki, T., Chrząstek, K., Julian, L., Muhire, B., Golden, M., … & Varsani, A. (2014). Pigeon circoviruses display patterns of recombination, genomic secondary structure and selection similar to those of beak and feather disease viruses. *Journal of General Virology*, 95(6), 1338-1351.

Teske, L., Ryll, M., Rubbenstroth, D., Hänel, I., Hartmann, M., Kreienbrock, L., … & Rautenschlein, S. (2013). Epidemiological investigations on the possible risk of distribution of zoonotic bacteria through apparently healthy homing pigeons. *Avian Pathology*, 42(5), 397-407.

Vereecken, M., De Herdt, P., & Ducatelle, R. (1998). Adenovirus infections in pigeons: a review. *Avian Pathology*, *27*(4), 333-338.

Zigo, F., Takáč, L., Zigová, M., & Takáčová, J. (2017). Changes in bacterial microflora in young carrier pigeons during the race season. *Int J Avian Wildlife Biol*, *2*, 41-4.

Zigo, F., Ondrašovičová, S., Zigová, M., Takáč, L., & Takáčová, J. (2019). Influence of the flight season on the health status of the carrier pigeons. *Int J Avian & Wildlife Biol*, *4*(2), 26-30.