JOURNAL OF VETERINARY AND APPLIED SCIENCES
VOLUME 15, ISSUE 1: 1050 – 1058 (2025)
Published by: Faculty of Veterinary Medicine, University of Nigeria, Nsukka, Nigeria
ISSN: 2315-6856; e-ISSN: 2636-5553; Website: www.jvasonline.com

Lymphoid leukosis in a commercial layer chicken flock: A case report with detailed necropsy, cytology and histopathological findings

Chukwudi C. Ezeugwu [1], Olanrewaju S. Olaifa [1], Olatunde B. Akanbi [2], Fatima Sanusi [3] *

[1] Department of Veterinary Pathology, Faculty of Veterinary Medicine, University of Ibadan, Oyo State, Nigeria.

[2] Department of Veterinary Pathology, Faculty of Veterinary Medicine, University of Ilorin, Kwara State, Nigeria.

[3] Department of Veterinary Physiology and Biochemistry, Faculty of Veterinary Medicine, University of Ilorin, Kwara State, Nigeria.

==========

Abstract

Avian lymphoid leukosis remains a leading neoplastic disease in commercial layer chicken flocks worldwide, and represents an emergent threat to Nigeria's rapidly expanding poultry industry. We report an outbreak of lymphoid leukosis in a 33-week old ISA Brown layer chicken flock (n = 20,000) in Southwestern Nigeria, characterized by progressive mortality exceeding 25% over three weeks, a 40% decline in egg production, with clinical signs of emaciation, pale combs, and greenish diarrhoea. Ten representative carcasses (mean body weight 1050 g) were examined. Grossly, nine of ten birds were severely emaciated; eight exhibited hepatomegaly (liver up to 11 × 8 cm; mean weight 80 g), and all livers bore multiple grey-white nodules (0.1 – 3 cm). Splenomegaly (up to 6 cm; 52 g) and disseminated neoplastic nodules in spleen, ovary, kidney, mesentery and caecal tonsils were recorded, and this confirmed multi-systemic involvement. Giemsa-stained impression smears of the hepatic nodules revealed a monomorphic population of medium to large lymphoid cells with high nuclear-to-cytoplasmic ratio, vesicular chromatin, prominent nucleoli, basophilic cytoplasm and occasional mitoses. Histopathology demonstrated well-circumscribed hepatic nodules of lymphoblasts replacing hepatic plates (3 – 4 mitoses/HPF), adjacent hepatocellular vacuolar degeneration and periportal lymphoid aggregates. Similar lymphoid effacement was observed in the spleen and ovaries, while the peripheral nerves remained intact; features pathognomonic for lymphoid leukosis and distinguishing it from Marek's disease and Reticuloendotheliosis. In Nigeria, where rearing of commercial layer chickens underpins food security and livelihoods, lymphoid leukosis outbreaks precipitate substantial economic losses via mortality, reduced egg production and carcass condemnations. This case report underscores the need for accurate diagnosis and control of the disease to safeguard the poultry sector from the huge economic losses.

Keywords: Lymphoid leukosis; Case report; Layer chicken flock; Nigeria; Pathology; Cytology; Histopathology.

*** Correspondence:** Fatima Sanusi; Email: sanusi.f@unilorin.edu.ng; Phone: +2348067650602

Article History: Initial manuscript submission received – May 03, 2025; Final revised form received – June 19, 2025; Accepted for publication – June 26, 2025; Published – June 30, 2025.

Introduction

Avian lymphoid leukosis (LL) is one of the most significant neoplastic diseases of chickens, and remains a pertinent concern particularly in commercial layer flocks. It is caused by a Retrovirus belonging to the avian leukosis/sarcoma virus (ALSV) complex, and it is transmitted vertically from breeder to chicks, and horizontally between infected birds. While significant progress has been made in eradicating it from many primary breeder stocks, LL continues to cause sporadic outbreaks and economic losses in commercial layers due to tumour-related mortality, decreased egg production and meat condemnation at processing (Hinojosa *et al*., 2016; Li *et al*., 2021; Hossain *et al*., 2024).

The clinical signs of LL in commercial layer chickens are often non-specific and insidious, becoming apparent only after the onset of tumours, typically around or after peak egg production (20 – 40 weeks of age), but can occur later (Bhutia and Singh, 2017). The avian leukosis virus (ALV) causes a neoplastic transformation of the bursa associated lymphoid cells (B-cells) early in life, and disseminates as neoplastic infiltrations throughout the body at maturity. Its pathology is characterized by nodular to diffuse lymphoid infiltrations in visceral organs including the liver, spleen, bursa of Fabricius, kidneys, ovaries, mesentery, lungs and heart (Soujanya *et al*., 2019; Elmeligy *et al*., 2024). The neoplastic lymphoid cells consist of a uniform population of medium to large lymphocytes and lymphoblasts. It has been reported that sparing of peripheral nerves with absence of paralytic conditions in affected birds is a distinguishing feature of LL from other avian leukosis such as Marek's disease (Balachandran *et al*., 2009).

The non-availability of effective vaccines against LL underscores the critical importance of prevention by sourcing chicks from virus-free breeders, implementing impeccable biosecurity protocols to prevent horizontal transmission and conducting vigilant monitoring and surveillance within flocks, which are the only effective strategies to control this economically significant neoplastic disease of poultry. The present case report presents an outbreak of LL in a 33-weeks old Isa brown commercial layer chicken flock, detailing the gross pathology, cytological and histopathological alterations, which are significant in definitive diagnosis of the disease in the field. The salient diagnostic features differentiating LL from other avian sarcomas such as Marek's disease are discussed. Recommendations are also made for the management and control of the disease in poultry flocks.

Case Report

History: The affected flock was a 33-weeks old Isa brown commercial layer chicken flock, with a flock size of about 20,000 birds in Southwestern Nigeria. The farmer reported a progressive daily mortality that has lasted for over 3 weeks, cumulating to over 25% mortality in the flock. There was also a 40% drop in egg production, and the size of eggs was significantly reduced. There was reduction in feed intake, and severely affected birds were emaciated, lethargic and had pale combs with greenish diarrhoeic pasted vent. The birds were sourced from a local hatchery within the region.

Gross lesions: Ten carcasses were presented for necropsy, and the following gross lesions were observed: The carcasses weighing average of 1,050g were severely emaciated, with prominent keel bones in nine out of the ten carcasses. The liver of eight out of the ten carcasses were markedly enlarged (hepatomegaly), measuring about 11cm in length and 8cm in diameter, and weighing about 80g in six out of the ten carcasses. There were multiple, variably sized, soft, greyish-white, glistening nodules on the liver

in all the ten carcasses (Figure 1 and 2). The neoplastic nodules from various carcasses measured from pinpoint sizes to 3 cm in diameter. The nodules were also present on the ovaries, kidneys, mesentery and caecal tonsils in all carcasses, indicating a visceral metastasis and multi-organ involvement of the neoplastic condition. The spleen was markedly enlarged (splenomegaly), measuring about 6 cm in diameter and weighing up to 52g in seven out of the ten carcasses due to a diffuse neoplastic infiltration (Figure 3). There was moderate enteritis with watery intestinal contents in all the ten carcasses.

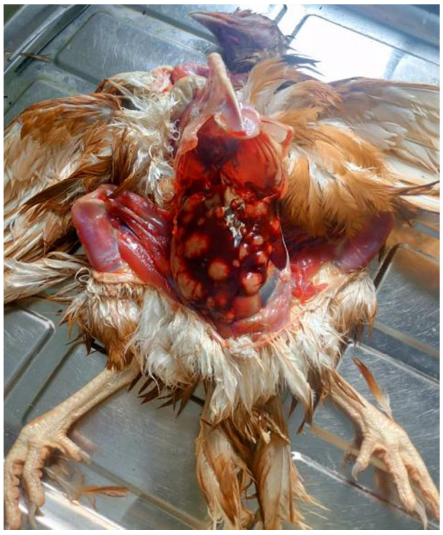

Figure 1: Whole carcass of a layer chicken with lymphoid leukosis, showing the enlarged liver in-situ with multiple, variably sized, soft, greyish-white, glistening neoplastic nodules. The keel bone is also very prominent.

Cytology: Giemsa-stained impression smear of the hepatic nodules (Figure 4) yielded a highly cellular smear predominantly composed of a monomorphic population of large to medium-sized, discrete lymphoid cells (lymphoblasts and prolymphocytes), with scant to moderate basophilic cytoplasm, high nuclear-to-cytoplasmic ratio and round to slightly indented nuclei containing finely distributed chromatin and prominent nucleoli. There were numerous clusters of lymphoglandular bodies with deeply basophilic naked nuclei in the smear. Occasional mitotic figures were seen. The background was pinkish, containing scattered erythrocytes, cellular debris and rare apoptotic bodies.

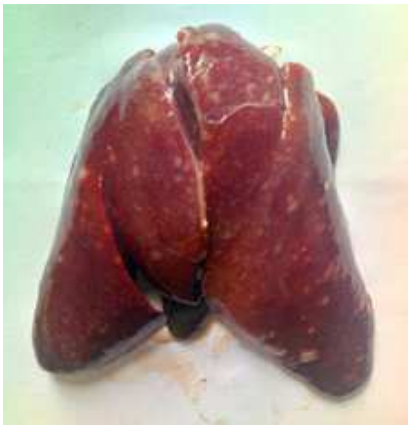

Figure 2: Liver of a layer chicken with lymphoid leukosis showing multiple smaller sized neoplastic nodules distributed throughout the hepatic tissue.

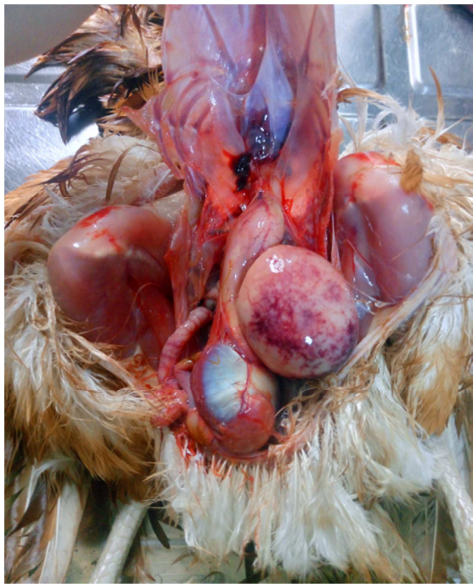

Figure 3. Markedly enlarged spleen in-situ (splenomegaly) due to diffuse neoplastic infiltration in layer chickens with lymphoid leukosis.

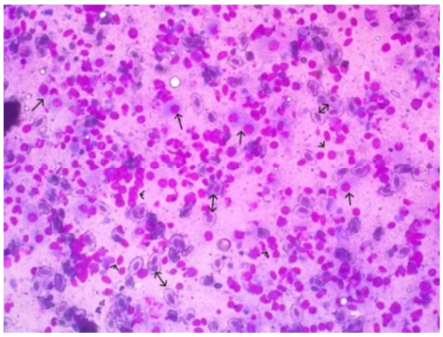

Figure 4. Touch impression smear of the hepatic nodules of layer chicken with lymphoid leukosis showing high cellularity with pinkish background. The cells consist of large discrete lymphoblasts, with round vesicular nuclei, high nuclear-to-cytoplasmic ratio, prominent nucleoli and scant to moderate basophilic cytoplasm (arrows); clustered to discrete, deeply staining, round naked nuclei of lymphoglandular bodies (arrowheads); nucleated red blood cells scattered within the field (double arrows); and cellular fragments within the pinkish dirty background. [Giemsa, ×400]

Histopathology: Hematoxylin and Eosin (H & E) stained sections of the liver revealed well-circumscribed nodular lesions separated from the normal hepatic parenchyma by mild fibrous stroma, which was composed of sheets of a mixed population of large to medium-sized lymphoid cells with round to indented nuclei, finely stippled chromatin, and prominent nucleoli (Figures 5 and 6). These neoplastic lymphocytes diffusely replaced the hepatic plates, with minimal stromal support and frequent mitotic figures (3 – 4/HPF). The nodular area showed a marked disruption of hepatic architecture. The adjacent hepatic parenchyma showed mild vacuolar degeneration of hepatocytes and periportal lymphoid aggregates. The central veins also show marked congestion. The spleen was markedly infiltrated by neoplastic lymphoid cells, leading to complete effacement of the normal red and white pulp architecture. The infiltrating cells were morphologically similar to those seen in the liver, with moderate fibroplasia of the trabeculae (Figure 7). The ovary exhibited moderate lymphoid infiltration within and around degenerating/necrotic follicles (Figure 8). Several primary and pre-ovulatory follicles displayed disrupted granulosa layers, with neoplastic lymphocytes infiltrating the thecal stroma (Figure 9) and in some cases, replacing the follicular architecture entirely. Follicles were necrotic, and had their centres replaced by a collection of infiltrating lymphoid cells. The sciatic nerve showed normal histoarchitecture, with well-myelinated axons, uniform nerve fibre diameters and intact perineurium and epineurium, without any evidence of inflammation or neoplastic infiltration (Figure 10).

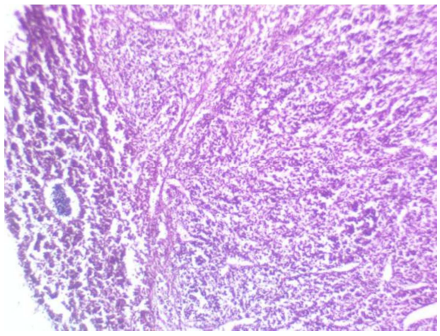

Figure 5. Section of the liver of layer chicken with lymphoid leukosis, showing diffuse neoplastic infiltration that has effaced the hepatic architecture. A mild fibrous stroma runs through to partly surround the neoplastic area. [H & E, ×100]

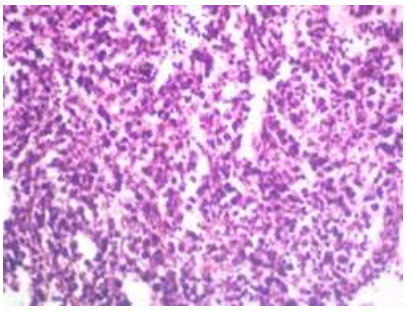

Figure 6. Section of the liver of a layer chicken with lymphoid leukosis, showing the neoplastic infiltrates consisting of medium to large lymphocytes and lymphoblasts. [H & E, ×400]

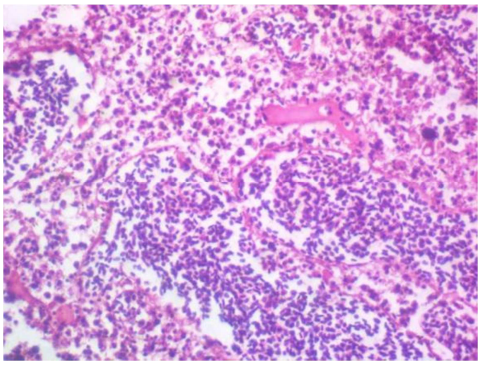

Figure 7. Section of the spleen of a layer chicken with lymphoid leukosis, showing severe infiltration of the parenchyma by neoplastic lymphocytes, surrounded by mild fibroplasia. [H & E, ×400]

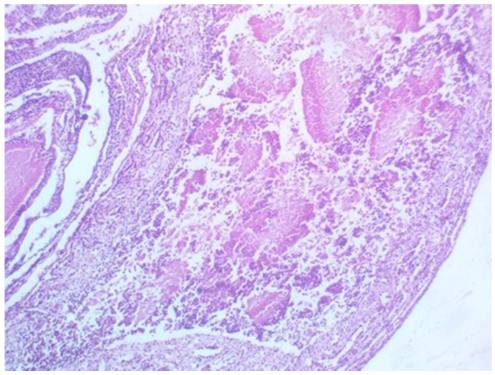

Figure 8. Section of the ovary of a layer chicken with lymphoid leukosis, showing lymphoid infiltration within the thecal stroma and around necrotic follicles. [H & E, ×100]

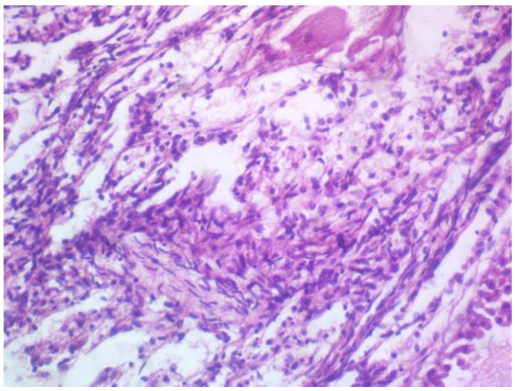

Figure 9: Section of the ovary of layer chicken with lymphoid leukosis, showing moderate neoplastic lymphoid infiltration within the thecal stroma. [H & E, × 400]

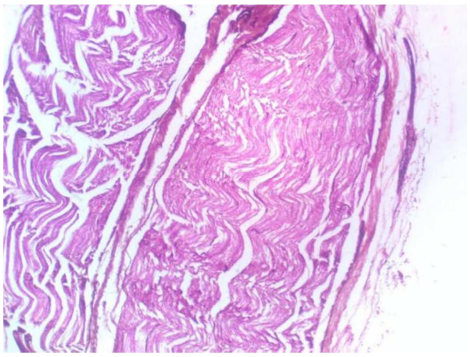

Figure 10: Section of the sciatic nerve of layer chicken with lymphoid leukosis, showing normal histoarchitecture, without any evidence of neoplastic infiltration. [H & E, × 100]

Case diagnosis: Visceral lymphoid leukosis with multi-systemic lymphoid neoplasia, significantly involving the liver, spleen and ovary, with notable sparing of the peripheral (sciatic) nerve – important in differentiating avian lymphoid leukosis from Marek's disease.

Discussion

The well documented hallmark of LL is the presence of discrete to diffuse tumours in various visceral organs. Lesions are reportedly usually soft, smooth, glistening and greyish-white to cream-colored, and the liver and spleen are the most consistently and severely affected organs (Bhutia and Singh, 2017). In the present case report, the liver from each carcass examined showed neoplastic nodules of varied sizes, which were both deep in the hepatic tissue and above the capsule, giving the liver an irregular surfaced appearance. The hepatomegaly recorded in the present case report is a characteristic feature of earlier reports (Bhutia and Singh, 2017). A previous survey on LL showed that the spleen of affected chickens is usually markedly enlarged (splenomegaly), sometimes several times normal size, with a smooth capsule and uniform pale tan to greyish cut surface (Sagarika *et al.*, 2017). In the present case report, the spleen was about 10 times larger

than normal, weighing 52g which was about 5% of the total body weight.

The difference between the lesions observed in the liver and spleen was that the neoplastic infiltration in the liver was nodular (nodular neoplastic hepatomegaly), but in the spleen, there was a more diffuse infiltration (diffuse neoplastic splenomegaly). It is thought that these pathologies culminated in the clinical signs observed which included anorexia, emaciation with prominent keel bone, and pale combs resulting from anaemia (Sagarika *et al*., 2017).

From earlier literatures reviewed, the bursa of Fabricius (BOF) of affected chickens was almost always reported to be involved, with discrete nodular tumours within the plicae (Fadly, 1987; Purchase, 1989). In the present case being reported, no lesions were observed in the BOF. In fact, the BOF were resorbed in most carcasses examined. We believe that at 33 weeks age, the BOF would have been totally resorbed/involuted and inactive to favour neoplastic progression. Although it is fact that the neoplastic B-cells originate from the BOF, the absence of tumours in the BOF in this case opened up prospects of considering other aetiologies in the avian leukosis-sarcoma complex. Marek's disease (MD) is the most common differential that always comes to mind. Peripheral nerve neoplastic infiltration is pathognomonic for MD (Fadly, 1987), as affected birds will show various degrees of paralysis. In the present case being reported, the sciatic nerve showed normal histoarchitecture. Grossly, the sciatic and brachial nerves were not enlarged and they retained their normal striations, as opposed to what is normally seen with MD (Balachandran *et al*., 2009). No paralysis was reported in the flock.

Histopathology is reportedly useful in definitive diagnosis and differentiation of LL from MD. Neoplastic cells in LL are more uniform than the pleomorphic infiltrates of MD (Kumar *et al*., 2018) and cell types in LL consists of a population of generally monomorphic population of large to medium-sized lymphoblasts and prolymphocytes (Witter *et al*., 2010). The findings in the present case report are consistent with these earlier reports.

Avian reticuloendotheliosis is another differential diagnosis that could be considered because its gross and histologic features can resemble both LL and MD (Payne and Venugopal, 2000). It has however been reported that natural cases of avian reticuloendotheliosis are very rare, as earlier studies has been only with experimental infections (Witter and Crittenden, 1979; Witter *et al*., 2010). Table 1 summarily compares the findings in the present case with earlier reported characteristic features of lymphoid leukosis, Marek's disease and reticuloendotheliosis of chickens.

The neoplastic involvement of the ovaries is one of the salient features highlighted in the present case. This finding is very important when one considers the economic implications of LL outbreaks in commercial layer flocks (Sagarika *et al*., 2017). A significant drop in egg production (up to 40%) and egg size was reported in the present case even before mortalities ensued. It is believed that compression atrophy and necrosis of ovarian follicles and thecal stroma cells due to infiltrating lymphoblasts is one of the underlying pathologies behind the egg production losses.

In lymphoid leukosis of chickens, cytologically, cells are typically round with a high nuclear-cytoplasmic ratio. Nuclei are round to slightly indented and vesicular, with coarse chromatin and one or more prominent nucleoli (Zhao *et al*., 2018). Cytoplasm is moderately basophilic and may contain vacuoles. Mitotic figures are common (Nair *et al*., 2019). The background often contains scattered erythrocytes and occasional normal lymphocytes or

macrophages. Necrosis or inflammation is minimal unless secondary infection occurs. The cytological findings in the Giemsa-stained touch impression smears of the visceral nodules in the present case report are consistent with these earlier reports.

Understanding the transmission dynamics is crucial for the control of LL in poultry flocks. It has been reported that vertical transmission is the primary route for establishing infection within a flock (Fadly, 1987). Viraemic hens transmit the virus trans-ovarially to a proportion of their offspring. These congenitally infected chicks are immunotolerant, become persistently viraemic for life ("shedders"), and are at high risk of developing LL later (Fadly, 1987). They also continuously shed virus, infecting flock mates. Horizontal transmission occurs readily through contact with infected birds, primarily via the faecal-oral route (shedding in saliva and faeces). The virus is also been reported to be present in the feather dander and albumen of infected hens (Spencer *et al*., 1984). Horizontal spread has been reported to be efficient in young chicks but becomes less efficient as birds age and develop immunity (Payne and Venugopal, 2000). Also, is has been reported that genetic susceptibility varies between chicken lines. Commercial layers, especially white-egg strains, often exhibit higher susceptibility than broilers or some brown-egg layers. Age susceptibility is highest in embryos and young chicks; older birds are more resistant to infection but susceptible to tumour development if infected early. It has been reported that the avian leukosis virus (ALV) is relatively labile in the environment but can survive for weeks in litter and poultry house dust under optimal conditions, facilitating horizontal transmission between flocks if biosecurity is breached (Fadly, 2000).

As there is no treatment for LL, management/control focuses entirely on prevention and eradication. The use of avian leukosis virus free breeders is the cornerstone of control. It is recommended that chicks and hatchable eggs should be purchased only from breeder flocks that are part of a rigorous, validated ALV eradication program. Biosecurity is critical to prevent horizontal introduction into ALV-free flocks. Regular testing of layer flocks (especially during rearing) for ALV antibodies can indicate exposure or infection within the flock. Regular necropsy of mortality, particularly during the critical period (20 – 40+ weeks), with histopathology on suspicious birds is essential for early detection of outbreaks. In the event of an outbreak within a commercial layer flock, identification and removal of viraemic or tumour-bearing birds can be attempted but is often impractical and economically challenging. Focus should therefore be on minimizing spread and preventing recurrence in subsequent flocks via enhanced biosecurity and sourcing avian

Conclusion: Avian lymphoid leukosis remains a threat to commercial layer chicken flocks, causing significant economic losses through tumour induced mortality and decreased egg production. Since vaccines are unavailable, prevention cannot be over-emphasized. Timely and accurate diagnosis is important to quickly detect outbreaks while differentiating other related avian sarcomas. This case report has tried to comprehensively present the pathology of a suspected outbreak of avian lymphoid leukosis in a 33 weeks old commercial layer chicken flock, emphasizing its clinical picture, gross lesions, cytology and histopathological alterations.

Conflict of Interest

The authors declare no conflicts of interest.

Table 1: Comparison of findings in the present case of lymphoid leukosis and earlier reported findings in the different lymphoid tumours of chickens.

Feature	Present case	Marek's Disease	Lymphoid Leukosis	Reticuloendotheliosis
Etiologic agent	No isolation *	Gallid herpesvirus 2 (Alphaherpesvirus)	Avian leukosis virus (Retrovirus)	Reticuloendotheliosis virus (retrovirus)
Age of onset	Adult birds (33 weeks of age).	Young birds (4–12 weeks).	Adult birds (>14 weeks).	Variable; often young to adult, may cause runting syndrome in chicks.
Cell lineage	No characterization *	T-cell lymphocytes.	B-cell lymphoblasts.	Mixed lymphoid /reticulum cells; heterogeneous populations.
Gross hepatic lesions	Multiple, variably sized, soft grey-white nodules (pinpoint to 3 cm).	Multifocal to coalescing white or grey nodules; less well-circumscribed; often accompanied by pale to yellow discoloration.	Diffuse or nodular white tumours in liver (often uniform miliary distribution)	Hepatomegaly with variable pale foci; may be swollen, friable; lesions less nodular and more diffuse
Spleen involvement	Marked splenomegaly with complete effacement by similar lymphoid nodules.	Splenomegaly common but nodules often less discrete; diffuse infiltration of white pulp.	Splenomegaly with nodular white foci, similar to that in the liver.	Splenomegaly with reticuloendothelial cell proliferation; may show haemorrhage and necrosis.
Peripheral nerve lesions	Absence of lesions on the sciatic nerve.	Prominent enlargement and/or yellowing of peripheral nerves (polyneuritis, paralysis).	Absent; nerves grossly and histologically normal.	Absence of lesions on the sciatic nerve; nerve involvement unusual.
Bursa of Fabricius	No significant bursal involvement noted.	Involvement rare; tumour formation uncommon in bursa.	Bursa often involved; bursal lymphomas considered pathognomonic.	Bursa may show lymphoid depletion or occasional neoplastic foci but not characteristic.
Histologic pattern	Well-circumscribed nodules of uniform medium-sized lymphoid cells with fibrous stroma; frequent mitoses (3–4/HPF); minimal inflammatory background.	Pleomorphic T-cell infiltrates; perivascular cuffing in nerves; mixed inflammatory infiltrate; mitoses variable.	Monomorphic lymphoblasts forming diffuse or coalescing foci; minimal stroma; frequent mitoses.	Heterogeneous cell populations (reticulum cells, lymphocytes); variable mitoses; may see acute necrosis and macrophage infiltration.
Clinical signs	Emaciation, hepato-splenomegaly, and decreased egg production.	Leg/wing paralysis, muscle atrophy, vision impairment, neuropathic signs.	Depression, weight loss, enlarged abdomen/organs, decreased egg production.	Runting, immunosuppression, poor growth, occasional paralysis.

* Test not done.

Sources: Payne and Venugopal, 2000; Balachandran *et al*., 2009; Witter *et al*., 2010; Kumar *et al*., 2018; Zhao *et al*., 2018; Nair *et al*., 2019.

References

Balachandran C, Pazhanivel N, Vairamuthu S, and Manohar BM (2009). Marek's disease and lymphoid leucosis in chicken - a histopathological survey. *Tamilnadu Journal of Veterinary and Animal Sciences*, 5(4): 167 – 170.

Bhutia LD, and Singh YD (2017). Occurrence of lymphoid leukosis in poultry population of Mizoram. *Indian Journal of Veterinary Pathology*, 41(3): 228 – 231.

Elmeligy AA, Ghania AA, and Fotouh A (2024). Pathological and immunohistochemical studies of lymphoid leukosis in pigeons in Egypt. *Open Veterinary Journal*, 14(8): 1952.

Fadly AM (1987). Differential Diagnosis of Lymphoid Leukosis. Springer, Boston, MA; pp. 197 – 211.

Fadly AM (2000). Isolation and identification of avian leukosis viruses: A review. *Avian Pathology*, 29(6): 529 – 535.

Hinojosa Y, Acevedo AM, Relova D, and Perera CL (2016). Virus de la Leucosis Aviar, 38(3): 187–193.

Hossain MDG, Pathan R, Hasan SN, Mozumder A, Mou MJ, Akter M, Sikder C, Reshad RAI, Mia R, Saha S, Islam T, and Akter S (2024). Molecular detection and genetic characterization of Avian Leukosis Virus from field outbreaks in Bangladesh. *Veterinary Medicine and Science*, 10(6).

Kumar MA, Palanivelu M, Barathidasan R, Kumar D, Singh SD, Lateef SK, Singh R and Dhama K (2018). Cytological and immunocytological detection and differentiation of Marek's disease and lymphoid leucosis in poultry. *Virus Disease*, 29(3): 349 – 354.

Li HQ, Meifang T, Zhang F, Huayuan J, Yanbing Z, Yang Q, Tan J, Jiangnan H, Su Q, Huang Y, and Zhaofeng K (2021). Diversity of Avian leukosis virus subgroup J in local chickens, Jiangxi, China. *Scientific Reports*, 11(1): 4797.

Nair V, Gimeno I and Dunn J (2019). Leukosis/Sarcoma Group. In: Swayne DE, Boulianne M, Logue CM, McDougald LR, Nair V, Suarez DL (Eds.). *Diseases of Poultry*, 14th edition, Wiley, pp. 587 – 625.

Payne LN and Venugopal K (2000). Neoplastic diseases: Marek's disease, avian leukosis and reticuloendotheliosis. *Revue scientifique et technique (International Office of Epizootics)*, 19(2): 544 – 564.

Purchase HG (1989). Progression of Avian Lymphoid Leukosis. Springer Netherlands; pp. 87 – 90.

Sagarika S, Das D, Panda SK, Das S, Jena B and Sahu RK (2017). Avian leukosis in chickens: A Clinico-pathological survey. *Journal of Entomology and Zoology Studies*, 5(6): 1697 – 1701.

Soujanya S, Lakshman M, and Madhuri D (2019). Occurrence of lymphoid leukosis in chicken. *The Pharma Innovation Journal*, 8(1): 159 – 162.

Spencer JL, Gilka F, Gavora J, and Wright P (1984). Distribution of lymphoid leukosis virus and p27 group-specific antigen in tissues from laying hens. *Avian Diseases*, *28*(2): 358.

Witter RL and Crittenden LB (1979). Lymphomas resembling lymphoid leukosis in chickens inoculated with reticuloendotheliosis virus. *International Journal of Cancer*, 23(5): 673 – 678.

Witter RL, Gimeno IM, Pandiri AR and Fadly AM (2010). Tumor Diagnosis Manual: The Differential Diagnosis of Lymphoid and Myeloid Tumors in the Chicken, 1st edition. Jacksonville, Florida: American Association of Avian Pathologists, Inc. pp. 1 – 84.

Zhao Z, Rao M, Liao M and Cao W (2018). Phylogenetic analysis and pathogenicity assessment of the emerging recombinant subgroup K of avian leukosis virus in South China. *Viruses*. 10(4): 194.