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Research Article

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Nodular Typhlitis Associated with *Heterakis* spp. in Golden Pheasants (Chrysolophus pictus): A Pathological and Immunohistochemical Investigation

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ABSTRACT

This report details nodular typhlitis associated with *Heterakis* spp. in golden pheasants (Chrysolophus pictus) focusing on clinical and pathological findings. Necropsies found hemorrhagic ceca filled with multifocal white nodules. Histopathological analysis has shown necrotic foci, inflammatory infiltrations, and parasite fragments in the submucosa and tunica muscularis, and perivascular spindle-cell proliferation assumed a whirling pattern consistent with perivascular origin. Masson's trichrome staining revealed only limited peripheral collagen, whereas the core nodules consisted of mesenchymal spindle cells with vimentin and α-smooth muscle actin (α-SMA) positivity, along with focal S-100 reactivity in some sections and uniform desmin negativity. While leiomyomas have been reported in *Heterakis* infections, the immunophenotypic profile—characterized by diffuse vimentin and SMA, occasional S-100 positivity, and absence of desmin—suggests a lesion with perivascular or undifferentiated mesenchymal features rather than a classical smooth muscle neoplasm or neurogenic tumor. Our observations thus expand the spectrum of pathological outcomes following chronic *Heterakis* infection in pheasants and underscore the value of immunohistochemistry in characterizing such spindle-cell lesions.

1. INTRODUCTION

Heterakis species are members of the Ascaridida order, a large group of nematodes with direct life cycles that primarily inhabit the gastrointestinal tracts of chickens, pheasants, turkeys, and wild birds (Ayeni et al. 1983; Draycott et al. 2000; Fedynich, 2008; Menezes et al. 2003; Millán et al. 2004; Norton et al. 1999; Rizzoli et al. 1999; Tompkins et al. 2002). These small (5.5–31 mm), whitish-tan nematodes typically reside in the cecum of their host (Cram 1927; Madsen 1941; Maplestone 1932). There are three clinically significant species in poultry—*H. gallinarum, H. dispar,* and *H. isolonche* (Soulsby 1982). Among these, *H. isolonche* is more commonly linked to severe nodular or verrucous typhlitis, characterized by granulomatous and even neoplastic nodules in the cecal wall and submucosa. Although *H. gallinarum* was traditionally viewed as less pathogenic, recent reports document that this species, too, can provoke severe nodular typhlitis with granulomatous cecal lesions in domestic chickens (Menezes et al. 2003).

Nodular typhlitis is previously described most commonly in pheasants, with multiple nodular lesions in the cecal wall resulting in significant morbidity and mortality (Menezes et al. 2003). Infections with heavy burdens of *H. isolonche* in golden pheasants (*Chrysolophus pictus*) have been associated with diarrhea, emaciation, and mortality (JPC 2024). Grossly, the ceca are thickened and have many firm, small raised nodules (usually ≥1–2 mm), giving a prominent "cobblestone" appearance of the mucosal surface. In severe cases, widespread cecal affection may lead to intestinal intussusception (Menezes et al. 2003). Traditionally, these nodules have been considered nothing more than granulomatous or fibroplastic responses to *Heterakis* larvae. However, they often contain interwoven bundles and whirls of spindle cells consistent with a possible sarcomatous nature. Additionally, under similar infections, not all galliform birds (e.g., quail or grouse) form similar nodules sharing typical co-pathological response, suggesting a species-genus-confined pathologic response (JPC 2024).

Over time, a consensus emerged that many of these lesions represent true neoplasia rather than purely inflammatory changes. Benign smooth muscle tumors (leiomyomas) were identified in heavily parasitized pheasant ceca (Menezes et al. 2003), and Helmboldt and Wyand (Helmboldt & Wyand, 1972) documented "neoplastic nodules" in golden pheasants, categorizing them as leiomyomas induced by *Heterakis* infection—occasionally even when only *H. gallinarum* was present. Subsequent investigations demonstrated that *H. isolonche* as well as *H. gallinarum* can induce the development of these innocuous lesions, typically exhibiting little mitotic activity or invasion (Menezes et al. 2003). By the late 20th century, chronic parasitic typhlitis in pheasants had been widely acknowledged as leading to uncontrolled cellular proliferation, therefore serving as a prime example of parasitic neoplasia (Jones et al. 1997).

The pheasant parasite-associated neoplasia parallels that reported from other species, not the least of which is *Spirocerca lupi* infections in canids, where granulomas of the esophagus develop into malignant sarcomas (Boros et al. 2020). In pheasants, *Heterakis* infection usually causes cecal leiomyomas or leiomyosarcomas through chronic inflammation and tissue damage (Boros et al. 2020). However, a

population of infected pheasants does not go on to develop tumorous lesions, indicating that host determinants, such as immune response or genetic susceptibility, may contribute (Menezes et al. 2003). The association of *Heterakis* infection with cecal nodular tumors highlights nodular typhlitis as a classic example of inflammation-driven neoplasia in avian pathology.

This study aimed to investigate nodular typhlitis attributed to *Heterakis* species in a pheasant flock with high mortality and to characterize the resultant cecal lesions through histopathological and immunohistochemical analyses.

2. MATERIALS AND METHODS

2.1. Animals

The study material comprised 22 golden pheasants (*Chrysolophus pictus*) housed at Antalya Zoo. Two adult pheasants (approximately 1.5 kg body weight), which were among those that succumbed to the disease, were submitted for necropsy. No antemortem fecal examinations were recorded.

2.2. Histopathology

Formalin-fixed tissues were processed by routine methods and embedded in paraffin. Sections (4 µm) were cut and stained with hematoxylin and eosin (H&E) for light microscopic examination. Masson's trichrome stain (#04-010802; Bio-Optica, Milan, Italy) was used primarily to distinguish collagen (stained blue) from muscle-like tissue in the nodular lesions. Additionally, sections were closely examined for the parasites or parasite eggs within the lesions.

2.3. Immunohistochemistry (IHC)

An IHC panel was applied to the cecal nodular lesions to determine the phenotype of the proliferating spindle cells. After antigen retrieval, serial sections were immunolabeled with the following primary antibodies: vimentin [a broad mesenchymal cell marker (M7020, Dako; 1:200 dilution)], α-smooth muscle actin [α-SMA, marker of smooth muscle and myofibroblastic cells (M0851, Dako; 1:100 dilution)], desmin [marker of differentiated smooth and skeletal muscle (M0760, Dako; 1:100 dilution)], and S-100 protein [(marker of neural crest-derived cells such as Schwann cells (Z0311, Dako; 1:400 dilution)]. A standard streptavidin-biotin-peroxidase complex technique was used with appropriate positive and negative controls for each marker. The antibody reactions were visualized with 3,3'-diaminobenzidine (DAB) chromogen and hematoxylin counterstain.

No molecular assays were conducted in this case. However, morphological identification of any parasites in tissue was attempted using published criteria for *Heterakis* species. The few adult nematodes collected from the cecal content were preserved in 70% ethanol and later examined microscopically to aid in species identification based on morphological features (such as the presence of cecal ridges, and male spicule length, which differentiate *H. isolonche* from *H. gallinarum*). Because only a limited number of worms were present and they were female (lacking distinctive male features), definitive species identification was impossible; however, *Heterakis* sp. was confirmed.

3. RESULTS

3.1. Clinical Observations and Outcome

Of the 22 pheasants, clinical signs included white diarrhea, cachexia, weakness, and lethargy. Despite initial treatment with Sulphamezathine and B vitamins, followed by Amoxicillin and B vitamins, the flock experienced a mortality rate of 2–3 birds per day, ultimately leaving only three survivors. No antemortem fecal examinations were recorded. The high mortality despite antibiotic and supportive therapy underscored the severity of the underlying parasitic infection, suggesting that targeted anthelmintic intervention might have been crucial for reducing losses.

3.1. Gross Findings

The primary lesion was confined to the ceca, which were carpeted with multifocal to coalescing nodules (Fig. 1). Each nodule was firm and pale, ranging from pinpoint to about 0.5 cm in the largest dimension. The cecal walls were diffusely thickened and less pliable due to the dense nodules within them. The mucosal surface had a rough, nodular contour, while externally the serosa also appeared undulant due to transmural nodular expansion. No significant luminal obstruction was noted when incising the ceca, but the number of nodules indicated a severe chronic process. Several cross-sections of slender whitish nematodes (~1–1.5 cm long) were found in the cecal content. No other gross abnormalities were identified in the bird; the liver, spleen, and intestine (apart from the ceca) appeared grossly normal, and there were no ascites or peritonitis.

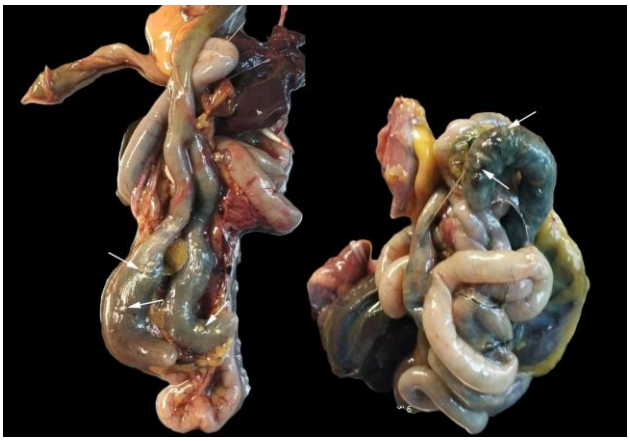

Fig. 1. Nodular lesions observed in the ceca of two pheasants (arrows).

3.3. Histopathologic Findings

Microscopically, the cecal nodules consisted of proliferative spindle cell tumors centered in the submucosa and muscularis (Fig. 2A-F). These expansile nodular masses disrupted the normal architecture of the cecal wall. The neoplastic cells were fusiform with elongate, blunt-ended nuclei and moderate pale eosinophilic cytoplasm. They were arranged in interwoven fascicles and whorled patterns, often concentrically around small blood vessels (perivascular whirling). The cell density was high in the nodules, but cellular atypia was low: the nuclei were fairly uniform with only mild to moderate pleomorphism, and mitotic figures were rare (averaging <1 per high-power field). No evidence of overt malignancy, such as invasive growth into adjacent tissues or metastatic spread to other organs, was observed. The boundaries of the nodules were generally pushing/compressive rather than infiltrative. Masson's trichrome staining showed minimal to moderate collagen at the edges of the nodules. The bulk of each nodule consisted of spindle cells that did not stain strongly for collagen, suggesting a primarily muscle-like or myofibroblastic composition (Fig. 2G, H).

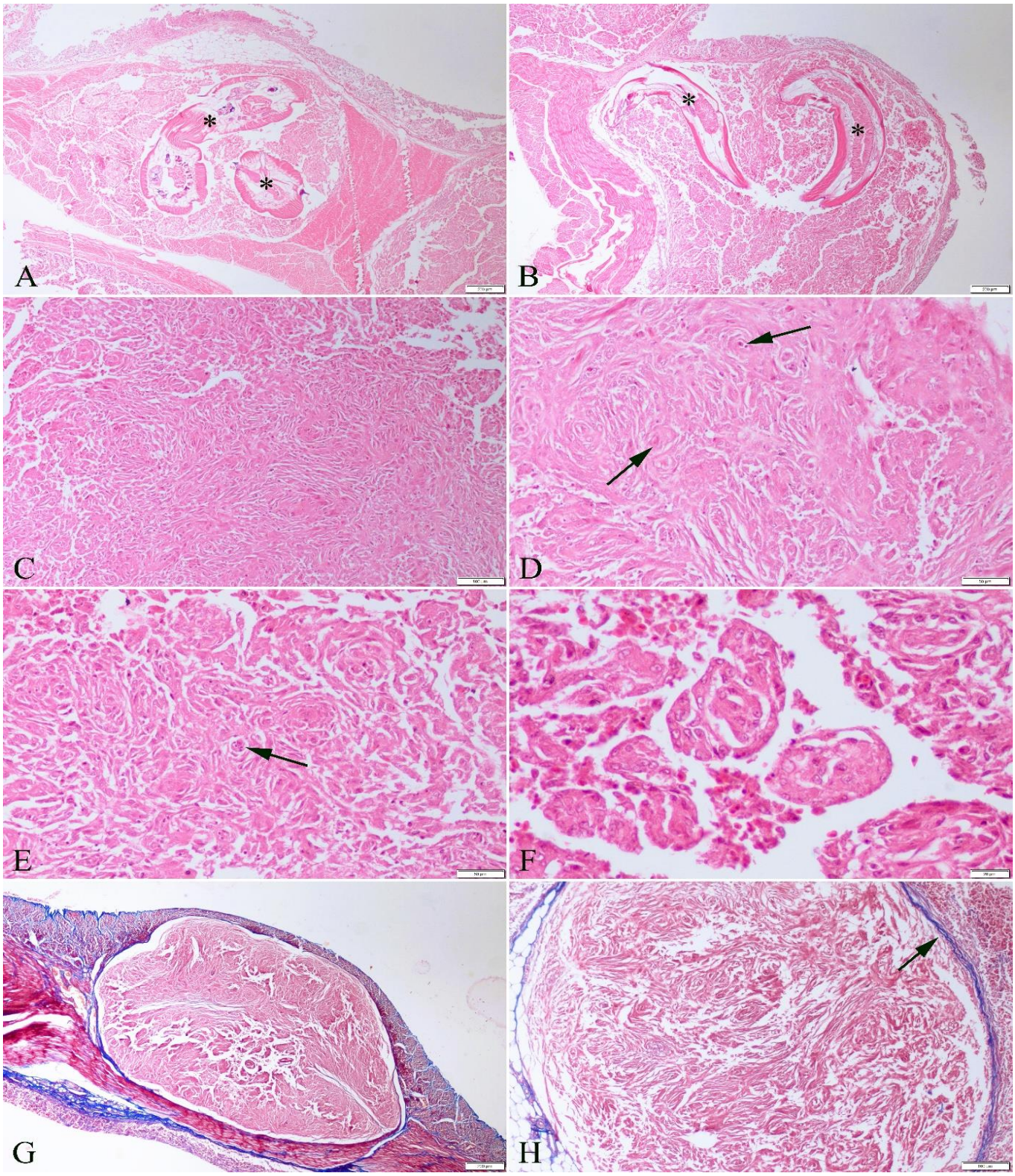

Fig. 2. Parasite cross-sections surrounded by neoplastic formations in microscopic sections of the cecum (stars) (A, B), H&E; Neoplastic cells exhibiting a whorled proliferation pattern around blood vessels (arrows) (C-E) with mild to moderate pleomorphism (F), H&E; Collagen staining observed around, but not within, the neoplastic areas (arrows) (G, H), Masson's Trichrome. Scale bars: 200 µm (A, B, G); 100 µm (H, C); 50 µm (D, E); 20 µm (F).

Interestingly, within some nodules there were central areas of necrosis containing remnants of nematode larvae. Affected nodules often showed a granulomatous reaction around these parasite remnants: multinucleated giant cells, epithelioid macrophages, and lymphoplasmacytic infiltrates were present at the core of such lesions. Cross-sections of a nematode with a thick cuticle, pseudocoelom, muscle layers, and an intestine were identified in a few sections, consistent with an immature *Heterakis* spp. worm. In nodules where parasite sections were found, the surrounding spindle cell proliferation appeared particularly well-developed, suggesting the parasite as a stimulus. Some nodules lacked any visible parasite, consisting purely of spindle cells and fibrous stroma – these may have been older lesions where the inciting larva died and was resorbed. No clusters of protozoal parasites (e.g., Histomonas) were seen in the cecal tissue. The other examined tissues (including liver and spleen) had no significant lesions, apart from mild reactive changes.

3.4. Immunohistochemistry Results

Immunohistochemistry revealed that the spindle cells exhibited diffuse cytoplasmic vimentin labeling, consistent with a mesenchymal phenotype. α-SMA reactivity was also prominent, suggesting myofibroblastic or pericytic differentiation. Desmin was entirely negative, ruling out a well-differentiated smooth muscle tumor (leiomyoma). S-100 showed sparse, patchy immunopositivity in a few scattered cells, potentially representing entrapped nerve elements or minimal neural differentiation; the majority of the neoplastic spindle cells were S-100–negative. This prominent α-SMA and vimentin reactivity coupled with weak and partial S-100 positivity and desmin negativity collectively points to a pericytic (perivascular) or myofibroblastic proliferation rather than a classic leiomyoma or neurofibroma. The perivascular whorling pattern further supports a perivascular wall tumor (PWT) origin. Figure 3 illustrates the immunohistochemical features observed.

3.5. Ancillary Findings

Given the histological evidence of larvae embedded in the cecal wall, it is likely that the *Heterakis* infection (regardless of species) was the inciting factor for tumor formation. No evidence of other infectious agents was found in association with the lesions. Additionally, a parasitological fecal examination of the intestinal content was performed using the Fülleborn flotation method, revealing the presence of *Heterakis* spp. eggs (Fig. 4). Therefore, the final diagnosis was nodular typhlitis with parasite-associated PWT of the cecum in a golden pheasant with *Heterakis* spp. identified as the etiological agent in the lesions.

4. DISCUSSION

Heterakis gallinarum has long been identified as a prevalent nematode of the ring-necked pheasant in the USA (Gilbertson & Hugghins, 1964; Olsen & Braun, 1980; Pence et al. 1980), European countries (Draycott et al. 2000; Madsen 1941), and Türkiye (Gürler et al. 2012). There is limited knowledge regarding the pathological impact of *Heterakis* in wild birds, but poultry and pheasants—particularly the latter—are known to be highly susceptible (Fedynich 2008). While H. gallinarum has been implicated in clinical signs ranging from inflammation to tumor-like lesions, H. isolonche is typically associated with the most severe clinical outcomes (Halajian et al. 2013).

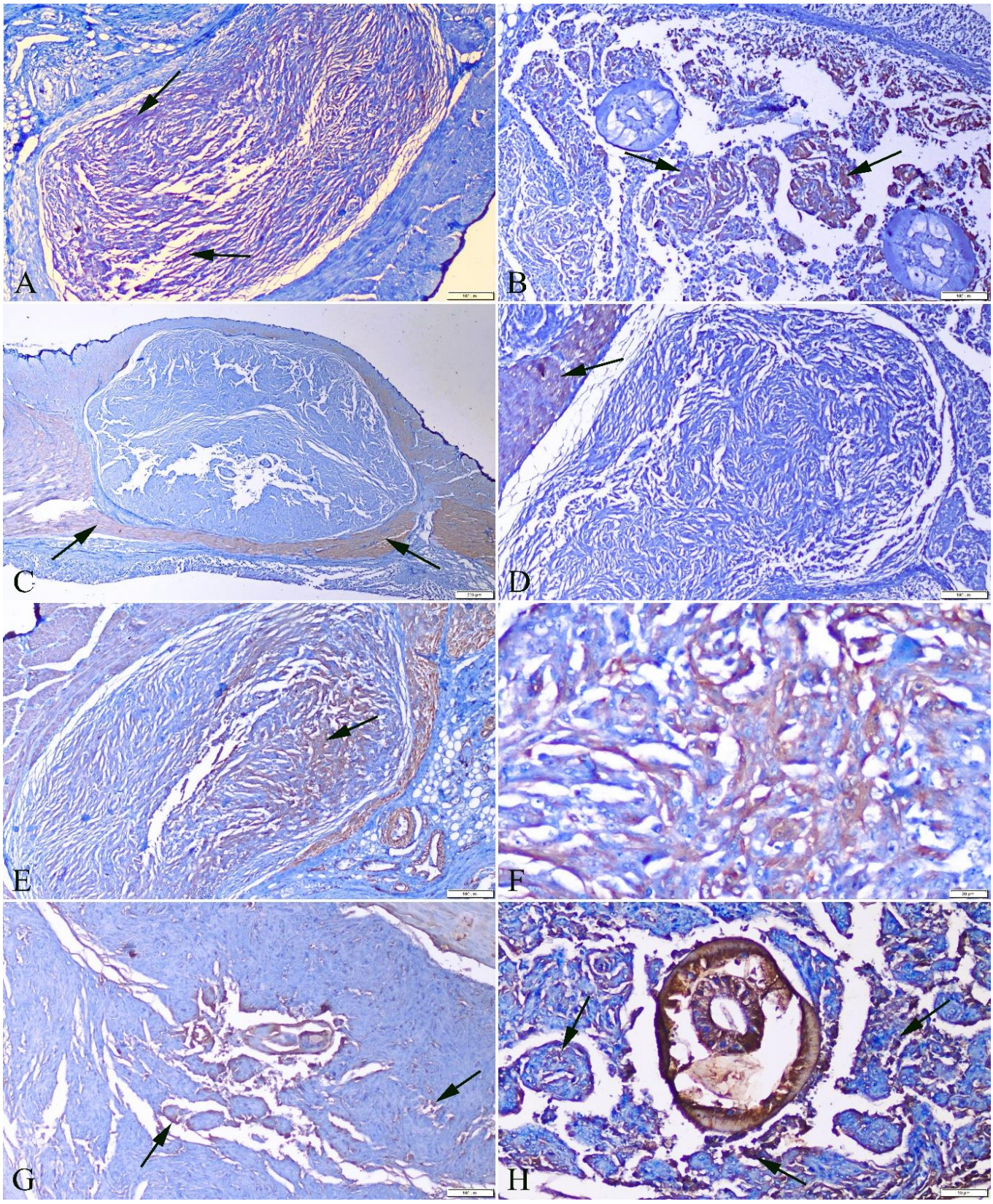

Fig. 3. Widespread vimentin positivity in neoplastic areas (A, B); desmin staining restricted to the muscle layer outside neoplastic regions (C, D); diffuse α-SMA expression in tumor cells (E, F); and partial S100 staining (G, H). Arrows indicate positive staining areas. DAB counterstain. Scale bars: 200 µm (C); 100 µm (A, B, D, E, G); 50 µm (H); 20 µm (F).

In this case study, definitive species identification was impossible because of the limited available worm material; however, the presence of larvae within lesions strongly implicates *Heterakis* spp. as the etiological agent.

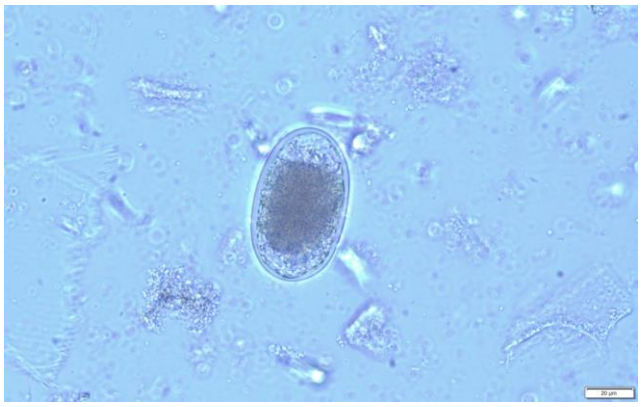

Fig. 4. Microscopic view of a *Heterakis* spp. egg using the Fülleborn flotation method. Scale bar: 20 µm.

Numerous reports have documented nodular typhlitis in pheasants caused by both *H. gallinarum* (Menezes et al. 2003) and *H. isolonche* (Balaguer et al. 1992; Callinan 1987; Griner et al. 1977; Schwartz 1924). This disease is characterized by inflammatory, granulomatous, or even neoplastic nodules in the cecal wall—especially the submucosa—ranging from 1 to 8 mm in diameter and varying in color from pale white or pink to dark brown (Griner et al. 1977; Menezes et al. 2003). After initial nodule development, the host mounts a vigorous immune response mediated by lymphocytes, macrophages, and fibroblasts surrounding the parasites (Balaguer et al. 1992; Callinan 1987). Later on, degenerate worms are surrounded by older nodules containing epithelioid cells, plasma cells, and giant cells (Balaguer et al. 1992). Our findings also showed similar nodular lesions with occasional necrotic foci and acute inflammatory reactions, but the predominant lesions were proliferative, tumor-like spindle-cell proliferations with occasional inflammatory cells, consistent with a neoplastic process rather than a purely inflammatory nature.

The primarily proliferative mesenchymal cell population seen in nodular typhlitis has been described using a variety of terminologies, including the classic "nodular typhlitis" (Schwartz 1924), "sarcoid leiomyoma" (Krahnert 1952), "parasitic granuloma" (Cohrs 1966), "parasitic neoplasia" (Helmboldt & Wyand, 1972), "verrucous typhlitis," and "leiomyoma" (Menezes et al. 2003). Divergent opinions persist in the literature regarding whether these changes represent granulomatous or fibrohistiocytic tissue, fibrovascular proliferations, fibrous hyperplasia, or bona fide neoplasms like leiomyomas (Balaguer et al. 1992; Halajian et al. 2013; Mendonça, 1953). Indeed, submucosal and muscular layers in the cecum frequently show necrotic areas, giant cell granulomas, and nematode remnants (Halajian et al. 2013; Mendonça 1953), sometimes accompanied by clear evidence of neoplastic nodules (Balaguer et al. 1992; Mendonça 1953). Our findings revealed a predominance of proliferative spindle-cell nodules with minimal inflammatory components, and immunohistochemical analysis confirmed these proliferations as predominantly myofibroblastic or pericytic rather than leiomyomatous, thus refining the current understanding of these parasite-induced proliferative lesions.

In a previous study, Himmel and Cianciolo (2017) detected nodular mesenchymal cell proliferations in the liver and lungs of pheasants, suggesting metastasis from the intestine despite no apparent granulomatous inflammation or intralesional nematodes in those tissues. Our histopathological investigation revealed necrotic foci, inflammatory cell infiltrations, and

connective tissue proliferations within the submucosa and tunica muscularis of the cecum but no metastatic spread.

Our immunohistochemical results differ from those previously reported by Himmel and Cianciolo (2017), who demonstrated widespread positivity for vimentin and S-100 along with negativity for desmin and SMA, indicative of a neurofibromatous origin. In our case, spindle cells were weakly reactive for vimentin and α-SMA, mostly negative for S-100 (only sporadic focal reactivity), and negative for desmin. Although prior studies described leiomyomas associated with *Heterakis* sp. (Balaguer et al. 1992; Griner et al. 1977), the immunophenotype observed here—prominent α-SMA and vimentin, combined with minimal or incidental S-100 staining—more accurately aligns with a PWT or possibly a myofibroblastic proliferation rather than a fully differentiated leiomyomatous or neurofibromatous tumor (Chijiwa et al. 2004). This aligns with the concept that *Heterakis* infections can prompt a range of mesenchymal proliferations, including PWT-like nodules in pheasants. Additionally, Masson's trichrome staining demonstrated only focal collagen deposition at the periphery of these parasite-associated nodules, whereas the central spindle cell proliferations lacked dense collagen and exhibited a mildly muscle-like or myofibroblastic appearance. This limited collagen further supports the notion of a perivascular or myofibroblastic origin rather than a purely fibrous hyperplasia.

Chronic *Heterakis* infection remains the principal driver of these cecal tumors, presumably via sustained local injury, prolonged inflammation, and growth factor release (Boros et al. 2020). Eventually, fibroblasts, pericytes, or precursors of vascular smooth muscle can undergo abnormal clonal proliferation, leading to a neoplasm (Menezes et al. 2003). Although H. isolonche typically invades the cecal wall and is more traditionally considered tumorigenic, H. gallinarum—from the cecal lumen—has also been involved in cecal nodules (Menezes et al. 2003). Host-specific immune responses likely determine whether fibroplastic or pericytic proliferation ensues. Previous differentials included purely inflammatory granulomas, leiomyomas, fibrosarcomas, schwannomas, and reactive myofibroblastic nodules. Immunohistochemistry is crucial to distinguish these entities: leiomyomas show strong desmin positivity, and nerve sheath tumors express S-100 reactivity (Chijiwa et al. 2004). In our case, the prominent vimentin and α-SMA positivity, rare S-100 labeling, and desmin negativity highlight a myofibroblastic lesion with minimal neurogenic differentiation. Additionally, the organized whorling pattern around blood vessels supports a perivascular origin rather than random fibroplasia. Although rare, metastatic behavior has been documented (Himmel & Cianciolo, 2017); our case did not show metastases, but repeated exposures or co-factors could potentially incite more aggressive transformations.

From a clinical standpoint, nodular typhlitis can initially go unnoticed until worm burdens become significant. Despite the use of antibiotics and supportive care, the current outbreak has been marked by a high mortality rate—underscoring just how severe this parasitic infection is and reinforcing the urgent need for targeted anthelmintic treatment. As the disease progresses, affected birds may present with chronic diarrhea, emaciation, or even intestinal obstruction, including intussusception (Menezes et al. 2003). By the time multifocal tumors form in the

cecum, conservative treatment is generally unavailing. Prevention and early intervention are crucial in managing *Heterakis* infections in pheasants. Regular fecal screenings for *Heterakis eggs*, strict deworming protocols, and strategies to disrupt the parasite's life cycle—such as minimizing soil exposure and controlling earthworm populations—are essential in maintaining healthy flocks. In cases where advanced nodular lesions develop, severely affected birds may need to be culled to safeguard overall flock health. While these tumors are benign in that they do not typically metastasize, they can significantly impair intestinal function and, if extensive, may prove fatal (Menezes et al. 2003). Veterinarians should include *Heterakis* infection in their differential diagnoses when evaluating pheasants with chronic weight loss and severe cecal pathology.

Additionally, recognizing that many of these lesions are perivascular wall tumors broadens our understanding of how chronic parasitic infections contribute to neoplastic transformation in birds. Similar to Spirocerca lupi in canines, *Heterakis* infections in pheasants demonstrate how persistent inflammation and tissue repair can lead to tumor formation (Boros et al. 2020). Future research could focus on the molecular characteristics of these pericytic proliferations to further elucidate the mechanisms driving parasite-induced neoplasia.

5. CONCLUSION

Overall, our results reinforce the notion that *Heterakis* nematodes can incite significant pathological sequelae, including PWTs, thereby highlighting the need for vigilant parasite management in pheasant flocks and greater awareness of infection-induced tumors in avian pathology.

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Conflict of Interest

The authors declare no conflict of interest.

Author Contributions

VI: Conception, histopathological diagnosis, interpretation of immunohistochemical results, and manuscript writing and revision.

ST, AM, GO: Necropsy examination, tissue processing, histopathological staining, and manuscript writing.

MFP: Parasitological examination and species identification.

References

Ayeni, J. S. O., Dipeolu, O. O., & Okaeme, A. N. (1983). Parasitic infections of the grey-breasted helmet guinea-fowl (Numida meleagris galeata) in Nigeria. Veterinary Parasitology, 12(1), 59–63. https://doi.org/10.1016/0304-4017(83)90088-2

Balaguer, L., Romano, J., Nieto, J. M., & Fernandez, J. P. (1992). Nodular typhlitis of pheasants caused by Heterakis isolonche: further evidence of a neoplastic nature. J. Zoo & Wildlife Med, 23(2), 249–253.

Boros, Z., Bojan, A., & Cozma, V. (2020). The relationships that occur between parasitic infections and neoplasmic formations in humans and animals. Sci Parasitol, 21(3), 142–149.

Callinan, R. B. (1987). Nodular typhlitis in pheasants caused by Heterakis isolonche. Australian Veterinary Journal, 64(2), 58–59. https://doi.org/10.1111/j.1751-0813.1987.tb16131.x

Chijiwa, K., Uchida, K., & Tateyama, S. (2004). Immunohistochemical evaluation of canine peripheral nerve sheath tumors and other soft tissue sarcomas. Veterinary Pathology, 41(4), 307–318. https://doi.org/10.1354/vp.41-4-307

Cohrs, P. (1966). Textbook of the Special Pathological Anatomy of Domestic Animals. Pergamon Press.

Cram, E. (1927). Bird Parasites of the Nematode Suborders Strongylata, Ascaridata, and Spirurata. Smithsonian Institution. U.S. National Museum Bulletin 140. U.S. Government Printing Office.

Draycott, R. A., Parish, D. M., Woodburn, M. I., & Carroll, J. P. (2000). Spring survey of the parasite Heterakis gallinarum in wild-living pheasants in Britain. The Veterinary Record, 147(9), 245–246. https://doi.org/10.1136/vr.147.9.245

Fedynich, A. M. (2008). Heterakis and Ascaridia. In C. Atkinson, N. Thomas, & D. HUnter (Eds.), Parasitic Diseases of Wild Birds (pp. 388–412). Wiley-Blackwell.

Gilbertson, D. E., & Hugghins, E. J. (1964). Helminth Infections in Pheasants from Brown County, South Dakota. The Journal of Wildlife Management, 28(3), 543. https://doi.org/10.2307/3798206

Griner, L. A., Migaki, G., Penner, L. R., & McKee, A. E. (1977). Heterakidosis and Nodular Granulomas Caused by Heterakis isolonche in the Ceca of Gallinaceous Birds. Veterinary Pathology, 14(6), 582–590. https://doi.org/10.1177/030098587701400605

Gürler, A. T., Bölükbaş, C. S., Pekmezci, G. Z., Umur, S., & Açici, M. (2012). [Helminths of pheasant (Phasianus colchicus) detected by necropsy and faecal examination in Samsun, Turkey]. Türkiye Parazitolojii Dergisi, 36(4), 222–227. https://doi.org/10.5152/TPD.2012.54

Halajian, A., Kinsella, J. M., Mortazavi, P., & Abedi, M. (2013). The first report of morbidity and mortality in golden pheasant, chrysolophus pictus, due to a mixed infection of heterakis gallinarum and h. isolonche in iran. Turkish Journal of Veterinary and Animal Sciences, 37(5), 611–614. https://doi.org/10.3906/vet-1206-31

Helmboldt, C. F., & Wyand, D. S. (1972). Parasitic neoplasia in the golden pheasant. Journal of Wildlife Diseases, 8(1), 3–6. https://doi.org/10.7589/0090-3558-8.1.3

Himmel, L., & Cianciolo, R. (2017). Nodular typhlocolitis, heterakiasis, and mesenchymal neoplasia in a ring-necked pheasant (Phasianus colchicus) with immunohistochemical characterization of visceral metastases. Journal of Veterinary Diagnostic Investigation, 29(4), 561–565. https://doi.org/10.1177/1040638717707555

Jones, T., Hunt, R., & King, N. (1997). Veterinary Pathology (6th ed.). Williams & Wilkins.

JPC. (2024, October). JPC Systemic Pathology. (2024, October). Digestive System (D-P11): Golden pheasant (Chrysolophus pictus) cecal heterakiasis. Joint Pathology Center. https://www.askjpc.org/vspo/show_page.php?id=VSs1MFljeTV3VC9SOENZQXJsYmp3Zz09

Krahnert, R. (1952). Sarkoides Leiomyom nach Heterakidenbefall. Monatshefte Für Veterinärmedizin, 7, 71–75.

Madsen, H. (1941). The Occurrence of Helminths and Coccidia in Partridges and Pheasants in Denmark. The Journal of Parasitology, 27(1), 29. https://doi.org/10.2307/3272883

Maplestone, P. (1932). The genera Heterakis and Pseudaspidodera in Indian hosts. Indian Journal of Medical Research, 20, 403–420.

Mendonça, J. M. de. (1953). Heterakis isolonche Linstow, 1906 e Heterakis gallinae (Gmelin, 1790), agentes causais da Tiflite verrucosa em faisões no Jardim Zoológico do Distrito Federal. Memórias Do Instituto Oswaldo Cruz, 51(0), 675–687. https://doi.org/10.1590/s0074-02761953000100021

Menezes, R. C., Tortelly, R., Gomes, D. C., & Pinto, R. M. (2003). Nodular Typhlitis Associated with the Nematodes Heterakis gallinarum and Heterakis isolonche in Pheasants: Frequency and Pathology with Evidence of Neoplasia. Memorias Do Instituto Oswaldo Cruz, 98(8), 1011–1016. https://doi.org/10.1590/S0074-02762003000800005

Millán, J., Gortazar, C., & Villafuerte, R. (2004). Ecology of nematode parasitism in red-legged partridges (Alectoris rufa) in Spain. Helminthologia, 41(1), 33–37. https://researchers.unab.cl/en/publications/ecology-of-nematode-parasitism-in-red-legged-partridges-alectoris

Norton, R. A., Hoerr, F. J., Clark, F. D., & Ricke, S. C. (1999). Ascarid-associated hepatic foci in Turkeys. Avian Diseases, 43(1), 29–38. https://doi.org/10.2307/1592759

Olsen, O. W., & Braun, C. E. (1980). Helminth parasites of band-tailed pigeons in Colorado. Journal of Wildlife Diseases, 16(1), 65–66. https://doi.org/10.7589/0090-3558-16.1.65

Pence, D., Young, V. E., & Guthery, F. S. (1980). Helminths of the ring-necked pheasant Phasianus colchicus (Gmelin) (Phasianidae), from the Texas Panhandle. Undefined.

Rizzoli, A., Manfredi, M., Rosso, F., Rosa, R., Cattadori, I., & Hudson, P. (1999). Intensity of nematode infections in cyclic and noncyclic rock partridge (Alectoris graeca saxatilis) populations. Parassitologia, 41(4), 561–566.

Schwartz, B. (1924). Occurrence of nodular typhlitis in pheasants due to Heterakis isolonche in North America. J Amer Vet Med Assoc, 65, 622–625.

Soulsby, E. (1982). Helminths, Arthropods and Protozoa of Domesticated Animals (7th ed.). Bailliere Tindall.

Tompkins, D. M., Parish, D. M. B., & Hudson, P. J. (2002). Parasite-Mediated Competition among Red-Legged Partridges and Other Lowland Gamebirds. The Journal of Wildlife Management, 66(2), 445. https://doi.org/10.2307/3803177