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REVIEW ARTICLE

A Comprehensive Review of Pancreatic Adenocarcinoma, Lymphosarcoma and Adrenocortical Carcinoma in Dogs: Prevalence, Breed Predisposition, and Ultrastructural Insights

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Abstract

Canine malignancies present significant diagnostic and therapeutic challenges in veterinary oncology, profoundly impacting patient survival and quality of life. This review synthesizes current knowledge on three aggressive neoplasms: pancreatic adenocarcinoma, lymphosarcoma, and adrenocortical carcinoma. Systematic literature searches (PubMed/Google Scholar, 1976–2025) identified 35 peer-reviewed studies. Data were extracted on epidemiology, breed predisposition, ultrastructural features, and therapeutic outcomes. While there are limited published data specifically on breed-specific incidence of these cancers in dogs in Iran, global research suggests that certain breeds have a higher predisposition. Pancreatic adenocarcinoma is rare (incidence: 17.8/100,000 dogs; 0.011% of all canine tumors), with no consistent breed predisposition but a predominance in older dogs (mean age: 10 years). Its clinical challenge lies in its silent progression; patients often present with nonspecific signs like lethargy and vomiting only after the cancer has metastasized, leading to a poor prognosis with a median survival of less than three months, even with surgery. Ultrastructural alterations include abnormal zymogen granules and disrupted intercellular junctions. In contrast, lymphosarcoma accounts for 7–24% of all canine cancers, with an elevated risk in Boxers, Bernese Mountain Dogs, Labrador and Golden Retrievers. It is one of the most responsive cancers to chemotherapy; over 80% of dogs achieve remission with multi-agent protocols, yielding a median survival time of 12–14 months, which makes it a highly treatable, though often not curable disease. Adrenocortical carcinoma (prevalence: 0.1–0.3%) shows breed predispositions in German Shepherds and Poodles. A major clinical challenge is its frequent association with hormonally active syndromes, such as Cushing's syndrome, which can complicate diagnosis and management. For metastatic cases, the adrenal-selective drug trilostane has demonstrated efficacy in controlling clinical signs. While surgery can be curative for localized masses, the overall prognosis is guarded when invasion or metastasis is present. These neoplasms exhibit distinct epidemiological patterns and ultrastructural features. Breed-specific risks underscore genetic components, while novel therapies, such as toceranib phosphate and trilostane, show promise. Comparative oncology approaches, leveraging these spontaneous canine cancers as models for their human counterparts, may benefit both veterinary and human medicine. This review aims to enhance understanding of the clinical behavior and outcomes of these cancers' clinical behavior and outcomes to guide future research into improved diagnostic and therapeutic strategies.

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Introduction

Cancer remains a leading cause of morbidity and mortality in companion animals, with dogs exhibiting a diverse spectrum of malignancies (1). Advancing the management of these complex diseases requires a multifaceted approach, with two areas holding particular promise for improving outcomes: the study of breed predispositions and ultrastructural cellular characteristics. Understanding breed-specific risks provides a powerful epidemiological tool for refining differential diagnoses and identifying heritable genetic factors, while detailed ultrastructural analysis reveals the fundamental cellular machinery of tumors, uncovering potential targets for novel therapies. This integrative review applies this dual focus to three significant yet challenging canine cancers: pancreatic adenocarcinoma, lymphosarcoma, and adrenocortical carcinoma. While there is limited published data on their breed-specific incidence in dogs in Iran, global research confirms that certain breeds have a higher predisposition, underscoring the value of this line of inquiry (1-6). Pancreatic adenocarcinoma, an exocrine pancreatic tumor, is rare, accounting for approximately 0.011% of all canine tumors. Its clinical challenge lies in its silent progression and high aggression, often presenting with nonspecific signs such as vomiting, inappetence, and abdominal pain only after metastasis, leading to a poor prognosis with a median survival of less than three months even with surgery (1, 7-9). Unlike in humans, in whom pancreatic neoplasia is the fifth most common cause of cancer-related mortality, no association with household smoking has been identified in dogs (10). Recent studies suggest potential therapeutic advances, with toceranib phosphate, a tyrosine kinase inhibitor, demonstrating biologic activity (11, 12). In contrast, lymphosarcoma is one of the most common canine cancers (7–24% of all cases), characterized by well-documented breed predispositions and responsiveness to chemotherapy (6, 13, 14). Adrenocortical carcinoma, arising from the adrenal cortex, is less frequent but notable for its endocrine effects, often manifesting as hyperadrenocorticism (15). The scarcity of comprehensive epidemiological data, particularly for pancreatic and adrenocortical tumors, underscores the need for this review. We integrate key studies, including a pivotal ultrastructural analysis of canine pancreatic acinar cell carcinomas (16), clinicopathological reviews (8), and case reports highlighting breed-specific presentations (1, 4) and therapeutic potential (11). By synthesizing data on epidemiology, breed-specific risks, geographic variations, and ultrastructural characteristics, we aim to inform clinical practice and highlight future research priorities for these complex neoplasms.

Materials and Methods

A systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Electronic databases (PubMed, Google Scholar) were searched for records published between 1976 and 2025. The search strategy utilized Boolean operators with the following key terms: ("canine" OR "dog") AND ("pancreatic adenocarcinoma" OR "lymphosarcoma" OR "adrenocortical carcinoma") AND ("breed predisposition" OR "prevalence" OR "ultrastructure" OR "treatment"). The retrieved records were screened in a two-phase process against predefined eligibility criteria. The study selection process is summarized in the PRISMA flow diagram (Figure 1). Case reports, retrospective studies, epidemiological analyses, and ultrastructural studies involving dogs were included.

Studies were excluded if they lacked definitive diagnostic confirmation (e.g., histopathology or electron microscopy) or focused exclusively on non-canine species. Two independent reviewers screened titles and abstracts, followed by a full-text assessment of potentially eligible articles. Discrepancies were resolved through consensus. Data were extracted from included studies into a standardized form. Key extracted data included tumor prevalence, breed predisposition, age and sex associations, clinical presentation, geographic distribution, ultrastructural characteristics, therapeutic outcomes, and survival data. Data were synthesized both qualitatively and quantitatively. A meta-analysis was deemed inappropriate and was deferred due to significant heterogeneity among the included studies. Qualitative synthesis summarized the main findings across studies. Quantitative data on prevalence, breed-specific risks, morphometric measurements, and survival times were tabulated. Specific data from key studies were incorporated for detailed analysis: morphometric data from Banner et al. (1978) were analyzed to compare normal and neoplastic pancreatic acinar cells (16); biochemical data from Aupperle-Lellbach et al. (2019), histological classifications from Kircher and Nielsen (1976), and clinical response data from Musser and Johannes (2021), and clinical and pathological data from Oskouizadeh et al. (2008, 2011, 2020, 2024) were incorporated to assess diagnostic markers, tumor morphology, and treatment efficacy (1, 2, 4, 5, 8, 9, 12).

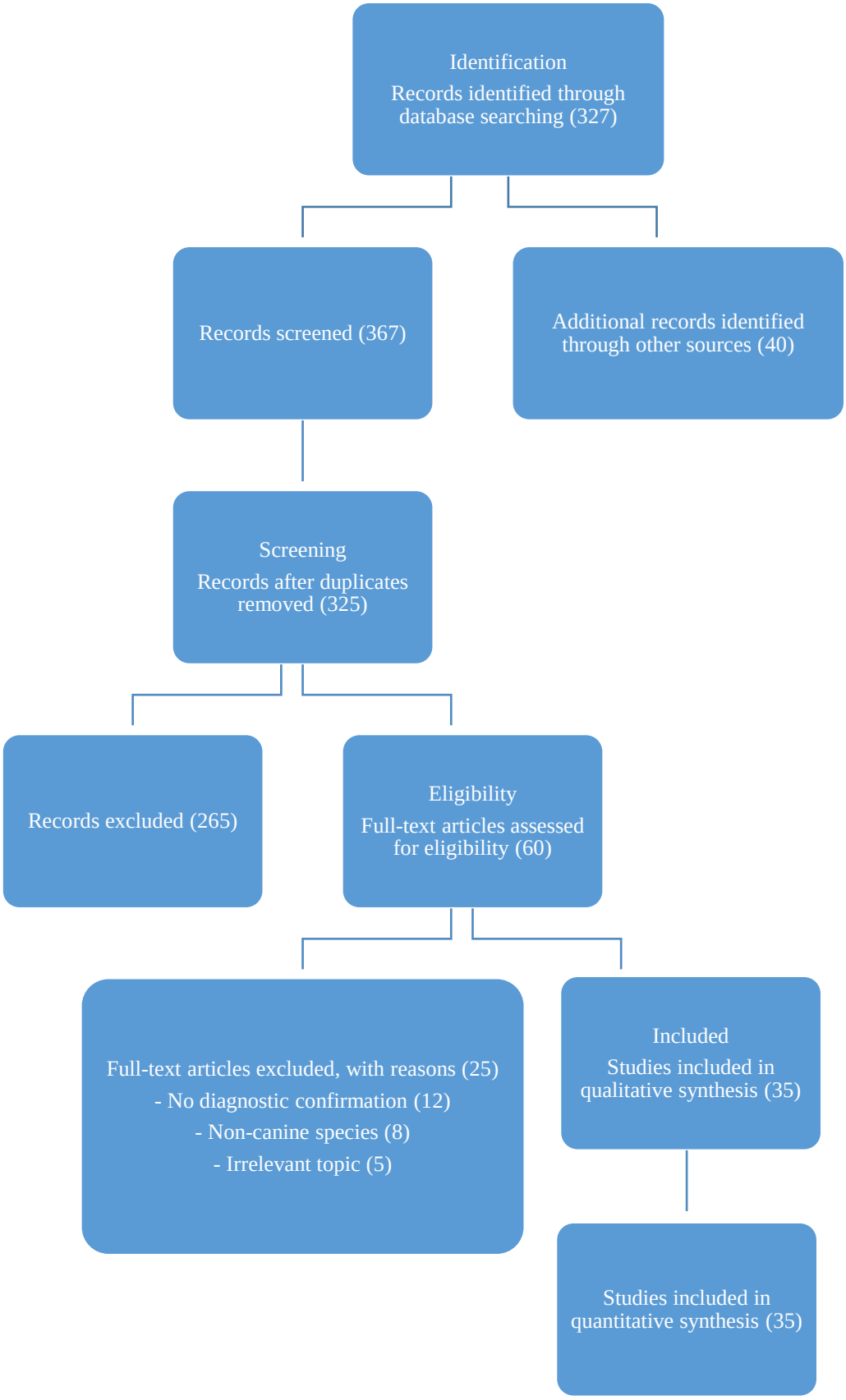

Figure 1. PRISMA Flow Diagram of Study Selection Process

Results

Pancreatic Adenocarcinoma

Pancreatic adenocarcinoma is a rare malignancy in dogs, constituting approximately 0.011% of all canine tumors (1, 7-9). It originates from the exocrine pancreas, with subtypes including small tubular (ductal), large tubular, and acinar adenocarcinomas, as well as undifferentiated carcinomas (1, 8, 9, 16). It predominantly affects older dogs (mean age: 10 years) and shows a slight female bias. No consistent breed predisposition has been identified, although Airedale Terriers may be overrepresented. Clinical signs are nonspecific and include vomiting (55–75%), weight loss or anorexia (30–40%), jaundice (15–25%), and steatorrhea (10–15%). Concurrent pancreatitis occurs in 64% of cases, complicating diagnosis. Laboratory abnormalities include elevated pancreatic lipase immunoreactivity (cPLI >400 µg/L) in 100% of acinar carcinomas and increased liver enzymes (ALP, ALT) in 60–80% of cases (1, 7-9). A comprehensive study of 55 pancreatic tumors across domestic animals, primarily dogs (approximately 140 exocrine adenocarcinomas reported) and cats, identified exocrine adenocarcinomas to be the most common pancreatic neoplasm (9). In dogs, these tumors occur predominantly in the duodenal limb (1, 9). Morphological patterns include small tubular (most common), large tubular, and rare acinar cell adenocarcinomas. Small tubular adenocarcinomas are characterized by tubules with prominent lumina, cuboidal cells, and delicate stroma, often transitioning to poorly differentiated areas exhibiting a "starry sky" pattern due to clear histiocytes interspersed among tumor cells. Large tubular adenocarcinomas are well-differentiated, with columnar cells forming ductal structures, occasionally accompanied by papillary projections or squamous differentiation. Acinar cell adenocarcinomas are rare, exhibit abnormalities of zymogen granules, and were not observed in the 55-case study. Undifferentiated carcinomas, reported only in dogs, lack epithelial differentiation and glandular architecture but exhibit the "starry sky" pattern and frequent metastases to the liver, lymph nodes, duodenum, and omentum (9). A detailed study of 22 canine pancreatic carcinomas found acinar carcinomas predominant (19/22), with ductal carcinomas in three cases (8). A case report of a 4-year-old female Miniature Pinscher with exocrine pancreatic adenocarcinoma highlighted severe clinical and pathological features (1). The dog presented with a 40-day history of weight loss, lethargy, vomiting, severe jaundice, chronic diarrhea, and steatorrhea, and ultimately died from hypovolemic shock and electrolyte imbalance. Radiographic findings revealed pancreatic enlargement, lateral deviation of the duodenum, and a soft tissue opacity medial to the duodenum. Histopathologic examination revealed pleomorphic neoplastic cells with large oval nuclei, prominent nucleoli, coarse chromatin, and a high nuclear-to-cytoplasmic ratio (1:1 compared with 1:5 in normal cells). The tumor formed acinar and tubular structures, and exhibited karyomegaly, atypical mitotic figures, necrosis, hemorrhage, and thrombus formation. Masson's trichrome staining identified acidophilic zymogen granules in some cells, and the mitotic index was five mitoses per field at ×400 magnification. Metastases to lymphatic and blood vessels, particularly the liver, were prominent, with normal hepatic parenchyma replaced by neoplastic cells (1). Exocrine adenomas exhibit tubular or acinar patterns, are encapsulated, and compress normal tissue (9). Nodular hyperplasia, common in older dogs, consists of non-encapsulated nodules resembling normal acinar cells, often associated with duct-like structures. Ultrastructural analysis of four acinar carcinomas revealed abnormal zymogen granules containing 24-nm microtubules, in contrast to normal granules (16). Neoplastic cells demonstrated increased nuclear volume (0.2508 ± 0.0418 vs. 0.0857 ± 0.0209 cu µ/cu µ), a higher nuclear-to-cytoplasmic ratio (0.3625 ± 0.0872 vs. 0.1004 ± 0.0263), and reduced nuclear pore density (8.54 ± 0.84 vs. 12.05 ± 0.71 pores/sq µ) (Table 1). Intercellular junctions exhibited fragmented tight junctions, reduced gap junctions, and sparse desmosomes, with discontinuous basal lamina and irregular tumor microvasculature. Tubuloreticular inclusions in endothelial cells, resembling distemper virus, were noted.

A retrospective study of 23 dogs reported a median survival time of 8 days, with 78% presenting with metastases at diagnosis (7). In contrast, a 10-year-old Beagle with ductal adenocarcinoma achieved a survival time of 445 days following partial pancreatectomy and adjuvant therapy with toceranib phosphate (2.4 mg/kg, three days per week) and firocoxib (5 mg/kg, every other day) (11), suggesting the potential of early intervention. Another study of eight dogs treated with toceranib phosphate (mean dose, 2.5 ± 0.1 mg/kg, three days per week or every other day) for confirmed pancreatic carcinoma reported a clinical benefit rate of 75% in the gross disease setting (12). Toceranib-specific median overall survival was 89.5 days (range: 14–506 days). Adverse events occurred in 37% of dogs and included grade 1 nausea, grade 1–2 diarrhea, grade 1 anorexia, and grade 2 neutropenia, with two dogs requiring dose holidays or reductions. Toceranib was well-tolerated, with discontinuation in 7/8 dogs due to progressive disease (n = 4), owner decision (n = 1), or

unspecified reasons (n = 2) (12). Therapeutic advances with toceranib phosphate (Palladia®), a tyrosine kinase inhibitor, show promise, with a clinical benefit rate (partial response and stable disease) of 75% and a median survival of 89.5 days compared with 8 days in untreated cases.

Table 1. Morphometric Comparison of Normal and Neoplastic Acinar Cells (16).

	V_v cell (cu μ/cu μ)	V_v nucleus (cu μ/cu μ)	V_v nucleus/ V_v cytoplasm	S_v nucleus (sq μ/cu μ)	Nuclear pores per sq μ NM
Control (Mean±SD)	0.9467±0.0153	0.0857±0.0209	0.1004±0.0263	0.1066±0.0309	12.05± 0.71
Neoplastic (Mean±SD)	0.9696±0.0147	0.2508±0.0418 *	0.3625±0.0872 *	0.2983±0.0593 *	8.54±0.84 *

Vv = volume density, Sv = surface density, NM = nuclear membrane. ∗Significant difference ($p < 0.05$).

Lymphosarcoma

Lymphosarcoma accounts for approximately 7–24% of canine cancers, with the multicentric subtype being the most common (80–85% of cases), followed by alimentary (5–7%), cranial mediastinal (5%), and cutaneous forms (4, 17). Extra-nodal forms affecting the central nervous system, eyes, bone, testes, or nasal cavity are rare (18). Its prevalence is higher in North America and Europe, likely reflecting differences in diagnostic capabilities (19). Geographic variations in risk have been reported: Golden Retrievers show no overall predisposition except in the UK; Golden Retrievers, Scottish Terriers, and Bulldogs show elevated risks in North America; Bullmastiffs and Airedale Terriers have top breed-specific rates in Croatia; and in Iran, urban, purebred dogs over 10 kg are at higher risk (6, 19, 20, 21). Lymphosarcoma shows notable breed predispositions, particularly in Boxers, Labrador Retrievers, and Golden Retrievers, suggesting a potential genetic influence, possibly involving apoptotic regulatory pathways (13, 22).

In a study of 130,684 insured dogs, 103 had a diagnosis of lymphoma recorded. A significant breed effect was identified, with Boxers, Bulldogs, and Bullmastiffs all showing a high incidence of lymphoma. The incidence of lymphoma increased with age, peaking at 10 years of age. (19). European data (n=1,529 cases) reveal significant breed predispositions (Table 2):

Table 2: Breed-Specific Risk for Lymphosarcoma in European Dogs (20)

Breed	Odds Ratio (95% CI)	Predominant Subtype
Bernese Mountain Dog	5.26–14.39	B-cell
Boxer	6.89	T-cell (high/low grade)
Rottweiler	4.59–9.39	B-cell
Golden Retriever	2.16 (UK only)	None consistent (vs. non-EU)
Labrador Retriever	3.33 (Switzerland)	High-grade T-cell

*p<0.01. Note: Golden Retrievers showed no significant predisposition in continental Europe.

A case study of a Labrador Retriever with multicentric lymphoma (Stage Va) in Iran demonstrated leukocytosis ($84 \times 10^3/\mu L$), lymphocytosis (90%), thrombocytopenia on hematology; elevated liver enzymes (AST, ALP, GGT) on biochemistry; and B-cell origin (CD20+) on Immunophenotyping. COP protocol (Cyclophosphamide, Oncovin, and Prednisone) for treatment induced remission within 8 weeks. COP chemotherapy achieves a 60–70% complete response rate and a median survival of 6–7 months (4, 20, 22). Nutritional management enhances treatment tolerance and reduces chemotherapy toxicity (4). Radiographic findings, such as sub-lumbar lymph node enlargement and hepatosplenomegaly, aid diagnosis but have variable prognostic value (4). Paraneoplastic syndromes occur in 30–50% of cases and include hypercalcemia (T-cell), anemia, and thrombocytopenia (4). While lymphoma can affect dogs of all breeds and ages, smaller breeds, including Chihuahuas, are generally less frequently diagnosed with lymphoma compared to some larger breeds. Lymphoma risk in dogs seems to be influenced by a combination of innate (genetic) factors and modifiable environmental factors linked to owner habits.

However, lymphoma can still occur in any dog, and some smaller breeds may still be predisposed (6, 14).

Adrenocortical Carcinoma

Adrenocortical carcinoma has a prevalence of 0.1–0.3% and commonly causes hyperadrenocorticism. Clinical signs include polyuria/polydipsia (90%), alopecia (85%), abdominal distension (75%), and muscle weakness (60%), driven by excessive cortisol production (2, 15). In rare cases, tumors secrete deoxycorticosterone, leading to mineralocorticoid excess and symptoms such as hypertension and hypokalemia, as reported in a single case of a dog with adrenocortical carcinoma, underscoring the need for comprehensive hormonal profiling to guide treatment (23, 24). German Shepherds, Labrador Retrievers, and Standard Poodles are overrepresented breeds, with German Shepherds accounting for 22% of cases (15, 25). Diagnosis typically involves hormonal assays, such as the ACTH stimulation test (elevated post-ACTH cortisol >18 µg/dL) or the low-dose dexamethasone suppression test, combined with imaging (ultrasound or CT/MRI) for tumor localization, vascular invasion assessment, and differentiation of benign adenomas from malignant carcinomas (2,5). Histopathologically, carcinomas are characterized by capsular or vascular invasion, with metastases most commonly affecting the liver, lungs, or kidneys (2, 23). Treatment with mitotane was evaluated in 32 dogs with cortisol-secreting adrenocortical neoplasms, achieving partial or complete remission in 75% of cases, though side effects (including gastrointestinal upset and neurologic signs) were frequent and required dose adjustments or treatment discontinuation in some dogs (24). Trilostane, with its less frequent and milder adverse effects, may be preferred as the primary medical treatment, particularly when adrenalectomy is not feasible. At a dose of 2 mg/kg twice daily, trilostane reduced metastatic lesions in one case, with a survival of more than one year (5, 26). Surgical adrenalectomy remains the primary treatment for non-metastatic tumors, but recurrence is common in malignant cases, with a median survival of 12 months (35% are metastatic at diagnosis) (2, 15, 25).

Discussion

The epidemiology and clinicopathological features of pancreatic adenocarcinoma, lymphosarcoma, and adrenocortical carcinoma in dogs reveal distinct patterns. Comparative epidemiology and pathogenesis of pancreatic adenocarcinoma highlight its marked rarity in dogs (0.011% vs. 3.3% in humans), which contrasts with its similarly aggressive behavior and typically late diagnosis. The absence of known environmental risk factors (e.g., smoking) suggests intrinsic biological differences (1, 9, 10). Ultrastructural abnormalities in neoplastic acinar cells, particularly disrupted intercellular junctions and abnormal zymogen granules, may contribute to metastatic propensity (1, 16). These features, combined with the gland's retroperitoneal location, create substantial diagnostic challenges. Early detection is exceptionally difficult as clinical signs are non-specific (e.g., lethargy, vomiting) and often only manifest with advanced disease. A practical diagnostic protocol for suspected cases should therefore combine advanced imaging (high-resolution ultrasonography with Doppler and contrast-enhanced CT) with guided fine-needle aspiration (FNA) or core biopsy for cytological and histopathological examination. However, the risk of pancreatitis and seeding must be considered. Serial monitoring of serum markers like pancreatic lipase immunoreactivity (PLI) may aid in identifying pancreatic inflammation or neoplasia, though it lacks specificity for malignancy. Ultimately, a high index of suspicion in older dogs with vague gastrointestinal signs is crucial for timely intervention (7, 27).

Lymphosarcoma breed predispositions highlight genetic underpinnings: Boxer dogs have a predisposition to T-cell lymphoma, which is associated with germline mutations in the PTEN gene and activation of the mTOR-PI3K-AKT pathway. These genetic factors contribute to the development of a more aggressive form of lymphoma in this breed (28). Golden Retrievers have a high incidence of lymphoma, and Genome-Wide Association Studies (GWAS) have identified several genes (MCC, FGFR4, TRPC6) potentially linked to this increased risk (29). European data contradict North American studies on Golden Retriever risk, suggesting regional genetic or environmental influences (19). The success of GWAS in elucidating breed-specific risks in lymphoma provides a strong foundation for future genetic research across all canine tumors. Future directions should include whole-genome sequencing to identify causal variants and structural alterations, the development of multi-breed genetic risk panels for clinical use, and comparative oncology studies to pinpoint conserved pathways across species. For example, applying similar GWAS approaches to pancreatic adenocarcinoma or adrenocortical carcinoma could uncover novel susceptibility genes, such as those involved in DNA repair (e.g., BRCA), inflammation, or steroidogenesis, which are poorly characterized in dogs but well-established in human medicine (8, 30-33).

Adrenocortical carcinomas in dogs frequently invade the caudal vena cava (50%), complicating surgical management

(25). The efficacy of trilostane in metastatic cases parallels human studies targeting steroidogenesis (5, 26).

The diagnostic and therapeutic implications of these neoplastic conditions underscore that, in Pancreatic Adenocarcinoma, Ultrasonography and CT are essential for the detection of pancreatic adenocarcinoma but lack sensitivity for early lesions (1, 27). Toceranib extends median survival nearly 10-fold (89.5 vs. 8 days), supporting targeted therapy trials (11, 12).

In contrast, for lymphosarcoma, Immunophenotyping of canine lymphomas is increasingly being performed because of its prognostic value (4, 19, 21, 34). Dogs with T-cell lymphomas are generally at higher risk of early relapse (52 vs. 160 days) and shorter survival times (150 vs. 330 days) compared with dogs with B-cell lymphomas (34). Breed-specific subtype risks (e.g., Boxers and T-cell) warrant genetic screening (28).

For adrenocortical carcinoma, Trilostane may be beneficial in inoperable cases, although prospective studies are needed (5, 26). Hormonal profiling (e.g., deoxycorticosterone) can identify atypical presentations (23, 24).

Limitations and Research Gaps, particularly geographic biases, appear to be present in the current literature. Adrenal tumors are relatively uncommon in dogs, making it challenging to gather large datasets for comparison. A high percentage of adrenal tumor data originates from North America, Australia, and European countries such as the UK, Germany, and Sweden, which have strong veterinary healthcare systems and research institutions, potentially leading to higher reporting (15, 24, 25). This bias is even more pronounced in underrepresented regions such as Iran, the Middle East, and parts of Asia and Africa. The true incidence and breed predispositions in these regions remain largely unknown, potentially skewed by differences in access to care, cultural attitudes towards pet ownership, and a lack of centralized cancer registries. To address these gaps, future studies should prioritize the establishment of multi-institutional tumor registries in these regions, utilizing standardized reporting protocols. Collaborative efforts between Western and local institutions could facilitate widespread, low-cost screening campaigns and the biobanking of samples for genetic analysis. This would not only provide a more comprehensive global understanding of canine cancer but also identify unique regional risk factors (e.g., environmental exposures, local breed genetics) that are currently underrecognized (13, 35).

Limitations related to therapeutic evidence further constrain the interpretation of outcomes, as most studies are retrospective with small cohorts. Breed genetics also appear to represent a research gap, as causal mutations remain unidentified outside lymphoma.

Conclusion

The comparative analysis of these three cancers underscores the aggressive nature of pancreatic adenocarcinoma, the genetic complexity of lymphosarcoma, and the invasive potential of adrenocortical carcinoma. Key findings include: Pancreatic adenocarcinoma exhibits distinct ultrastructural alterations (e.g., abnormal zymogen granules, junctional defects) that correlate with aggressive behavior. Lymphosarcoma breed risks vary geographically, with Boxers consistently predisposed to T-cell subtypes; and adrenocortical carcinoma responds to trilostane in metastatic settings, offering an alternative to mitotane. Future research should prioritize several areas. The diagnostic protocol for pancreatic cancer must evolve beyond conventional imaging to incorporate advanced techniques and biomarker research, including prospective trials of toceranib for these tumors. The remarkable progress in genetic mapping of lymphoma should be expanded through functional validation and translated into breed-specific targeted therapies, while also being applied to other cancers. Finally, a concerted global effort is required to address the significant geographical data gaps. By supporting comprehensive registries and molecular epidemiological studies in underrepresented regions, a more complete and equitable understanding of canine cancer can be achieved, ultimately benefiting dogs and informing human oncology.

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Authors' Contributions

Both authors contributed to the study conception and design. The first draft of the manuscript was written by Katayoon Oskouizadeh and both authors reviewed and approved the final manuscript.

Data Availability

All data supporting this review are derived from previously published sources cited in the reference list. Extraction templates are available from the corresponding author upon

reasonable request. The PRISMA flow diagram is presented as Figure 1.

Ethical Approval

Not applicable

Conflict of Interest

The authors declare no competing interests.

Consent for Publication

Not applicable

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