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Idiopathic pulmonary fibrosis in cats: Clinical presentation, diagnosis and management

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ABSTRACT

Idiopathic pulmonary fibrosis (IPF) is a progressive fibrotic lung disease characterized by a distinct histopathological pattern known as usual interstitial pneumonia (UIP) in human medicine. In recent years, cases showing clinical and histological features overlapping with those in humans have also been reported in the feline population. This presentation aims to highlight current knowledge on diagnosis, pathogenesis, clinical findings, differential diagnosis, treatment, and prognosis by comparing reported data in cats with their counterparts in human medicine. Available literature indicates that TGF-β-mediated fibrogenesis, epithelial-mesenchymal transition, miRNA, and other molecular regulators play roles in disease processes; and that radiological and histopathological findings (honeycomb pattern, ground-glass opacities, type II pneumocyte hyperplasia, fibroblastic foci) demonstrate similarities in both human and feline cases. However, in veterinary medicine, the lack of standardized diagnostic criteria, the overlap of radiological findings with other pulmonary pathologies, and limitations in performing invasive biopsies make it difficult to determine the true disease burden. Treatment is mainly supportive; antifibrotic approaches are still evaluated with limited data. In conclusion, prospective studies, biomarker research, and the standardization of advanced imaging protocols are required to better understand feline IPF, which will not only increase diagnostic accuracy but also strengthen knowledge transfer between veterinary and human medicine

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Introduction

Idiopathic pulmonary fibrosis (IPF) is a progressive fibrotic lung disease characterized by a distinctive histopathological pattern, commonly referred to in human medicine as usual interstitial pneumonia (UIP) (Raghu et al., 2011). In humans, the reported median survival following diagnosis ranges between only 2.5 and 3.5 years, and the limited therapeutic options available significantly compromise both quality of life and prognosis (Ley et al., 2011; Raghu et al., 2011; Strongman et al., 2018;).

In recent years, several studies have demonstrated that cases with similar clinical and histopathological features also occur in the feline population (Cohn et al., 2004; Evola et al., 2014). Reported cases in cats typically involve geriatric individuals and are associated with high mortality rates, underscoring the importance of early diagnosis and effective management (Cohn et al., 2004). However, diagnostic criteria for IPF in veterinary medicine have not yet been clearly defined, and the diagnostic boundaries of this disease in cats remain uncertain.

The aim of this review is to examine the current literature on IPF and related pulmonary disorders in cats and to highlight their similarities and differences in comparison with human medicine.

Literature Search Strategy

A comprehensive literature search was performed using the PubMed, Scopus, and Web of Science databases. Studies published between (1979-2023) were considered eligible for inclusion. The search strategy was based on combinations of keywords related to feline interstitial lung diseases, pulmonary fibrosis, and relevant biomarkers.

Studies were included if they (i) involved feline subjects, (ii) addressed interstitial lung disease, pulmonary fibrosis, or related pathological and pathophysiological mechanisms and (iii) were published as original research articles or

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Articles were excluded if they (i) involved non-feline species, (ii) were case reports lacking sufficient clinical or pathological detail, (iii) were conference abstracts without available full-text articles, or (iv) were published in languages other than English.

Definition and General Information: Fibrosis is defined as a chronic tissue response characterized by the excessive and uncontrolled accumulation of extracellular matrix, representing a pathological variant of the physiological wound-healing process. The underlying mechanism, fibrogenesis, is a complex and tightly regulated biological process in which numerous cellular components and signaling pathways interact (Moss et al., 2022).

Pulmonary fibrosis, most often of idiopathic origin but with a multifactorial etiology, is a severe interstitial disease associated with a poor prognosis (Evola et al., 2014). Interstitial lung diseases (ILDs), which include interstitial pulmonary fibrosis, represent a broad group of parenchymal disorders (Cony et al., 2019). In human medicine, ILDs are used as an umbrella term encompassing more than 200 distinct conditions, which are generally classified into two categories: those with an identifiable cause and those of unknown etiology. Among the idiopathic interstitial pneumonias, IPF is the most clearly defined entity (Mikolasch et al., 2017; Travis et al., 2013).

IPF is the most prevalent form of fibrotic interstitial lung disease in human medicine and is regarded as the prototypical and reference condition of this group (Raghu et al., 2022). Although widely recognized in humans, pulmonary fibrosis has more recently been reported in cats (Selman et al., 2010). While feline pulmonary fibrosis demonstrates clinical and histopathological similarities to human IPF, its pathogenesis remains incompletely understood (Cohn et al., 2004; Williams et al., 2004). In both human and veterinary medicine, pulmonary fibrosis is considered a disease with a poor prognosis, frequently culminating in death as a result of severe hypoxemia (Evola et al., 2014). The association between pulmonary fibrosis and lung carcinoma has been documented in both human and animal studies (Aubry et al., 2002). One human study revealed that concurrent pulmonary neoplasms associated with idiopathic pulmonary fibrosis occur at a nearly seven-fold higher rate compared with control groups, independent of common environmental risk factors such as smoking (Hubbard et al., 2000).

IPF is a progressive disease with a poor prognosis, with a reported mean survival in human medicine of only 2.5 to 3.5 years following diagnosis (Raghu et al., 2011; Strongman et al., 2018; Ley et al., 2011). Within the spectrum of idiopathic interstitial pneumonias, IPF represents the most common subtype and the one most strongly associated with high morbidity during its clinical course (Travis et al., 2013). In a study conducted on cats, no significant breed or sex predisposition for IPF has been identified (Cohn et al., 2004). Similar to the human condition, pulmonary fibrosis in cats has most often been reported in geriatric patients, suggesting a slowly progressive and insidious pathogenesis (Cohn et al., 2004). However, the presence of overlapping clinical and radiographic findings with other diseases that may mimic IPF significantly complicates accurate diagnosis, thereby limiting reliable determination of the true incidence and prevalence of the condition (Moss et al., 2022).

The pathogenesis of IPF is based on the interplay of molecular and cellular mechanisms. Disruptions in intracellular signaling pathways, autophagy, developmental reprogramming processes, metabolic remodeling, apoptotic mechanisms, microRNAs, and genetic and epigenetic regulators play decisive roles in the onset and progression of the disease (Moss et al., 2022). Transforming growth factor-β (TGF-β) is critical for both physiological and pathological wound healing and represents one of the key signaling molecules in fibrosis. TGF-β is secreted in a latent form by a variety of cell types, including epithelial cells, fibroblasts, and immune cells. Once secreted, various molecules facilitate its activation and binding to receptors on target cells, thereby initiating a fibrotic signaling cascade (Frangogiannis, 2020). As in humans, the pathogenesis of pulmonary fibrosis in domestic cats has not been fully elucidated. Although interstitial lung diseases in cats are most frequently associated with infectious agents, other potential etiological factors include inhaled substances, neoplasms, immune-mediated disorders, toxins, and certain drugs (Dungworth et al., 1982; Norris et al., 2002). A correlation between pulmonary carcinomas and pulmonary fibrosis has also been reported (Aubry et al., 2002).

In human medicine, disease onset is often linked to pulmonary injury, and viral agents such as herpesviruses have been implicated in triggering pulmonary fibrosis (Gross & Hunninghake, 2001). Moreover, asbestos and silica inhalation have been shown to contribute to the development of fibrotic foci (Oberdorster, 1996). Chronic inflammation, environmental chemicals, and inherited defects are also recognized as secondary causes of pulmonary fibrosis in humans (Baumgartner et al., 1997; Wagner et al., 1974; Noble et al., 2012; Wang et al., 2009). In addition, both in humans and cats, chemotherapeutic agents such as nitrosoureas have been reported to induce pulmonary fibrosis (Skorupski et al., 2008; Durant et al., 1979). Microscopic morphological features observed in type II pneumocytes of affected cats demonstrate a high degree of similarity to those described in humans (Thomas et al., 2002). Chronic pulmonary hypertension, increased pulmonary vascular resistance, or venous obstruction may lead to concentric collagen deposition in the lung parenchyma over time, thereby triggering the development of interstitial fibrosis (Caswell et al., 2016).

Although cigarette smoke exposure is a recognized etiological factor in the development of pulmonary fibrosis in humans, its role in feline pulmonary fibrosis has not been

established. However, environmental exposure to cigarette smoke has been suggested as a potential contributing factor in cats, warranting further investigation (Evola et al., 2014). Experimental models of pulmonary fibrosis in cats have been considered to most closely resemble human pulmonary fibrosis when compared with models in other animal species (Corcoran et al., 1999; Borzone et al., 2001; Lobetti et al., 2001; Cohn et al., 2004; Norris et al., 2005; Secrest et al., 2008; Phillips et al., 2012).

Clinical Findings: The primary clinical signs of the disease are respiratory in nature, including coughing, tachypnea, abdominal breathing, cyanosis, and open-mouth breathing. In addition, nonspecific signs such as anorexia, weight loss, and exercise intolerance may also be observed. Pulmonary auscultation typically reveals crackles, wheezes, and an increase in bronchovesicular sounds (Evola et al., 2014). In one study, the characteristic respiratory distress observed in fibrotic parenchymal lung lesions was found to be predominantly inspiratory, although in some cases both the inspiratory and expiratory phases were affected, producing a mixed pattern. This distinction highlights differences from conditions such as asthma and chronic bronchitis, which are generally bronchial in origin and primarily characterized by expiratory difficulty (Cohn et al., 2004).

Diagnosis and Differential Diagnosis: Variable radiographic findings have been reported in cats with pulmonary fibrosis, and the condition may be confused with pneumonia, neoplasia, pulmonary edema, and asthma (Evola et al., 2014). In one study, thoracic radiographs of cats diagnosed with pulmonary fibrosis (PF) revealed interstitial and bronchial infiltrates along with an alveolar pattern (Evola et al., 2014). Histopathologically, pulmonary fibrosis is consistent with interstitial pneumonia. However, differentiating idiopathic pulmonary fibrosis from secondary pulmonary fibrosis caused by factors such as asbestosis or toxicity is often challenging (Williams et al., 2004; Cohn et al., 2004; American Thoracic Society, 2001). Definitive diagnosis is established through histopathological examination of lung biopsy specimens and is often confirmed by pathological evaluation of samples obtained postmortem (Evola et al., 2014; Cohn et al., 2004).

In humans, diagnostic imaging techniques such as computed tomography (CT) may provide meaningful evidence toward an IPF diagnosis but remain insufficient for definitive confirmation (Krafft et al., 2011; Lynch et al., 2018). Human studies have reported CT findings such as honeycombing and ground-glass opacities (American Thoracic Society, 2001; Lynch et al., 2018). In another study conducted in cats, CT scans revealed focal increases in soft-tissue attenuation, ventral consolidation, and mass lesions. Histopathological examination of all affected cases demonstrated type II pneumocyte hyperplasia, pulmonary fibrosis, and smooth muscle hypertrophy (Evola et al., 2014). Although lung biopsy is a valuable tool in the diagnostic process, it may not always yield a definitive diagnosis. The patchy and heterogeneous distribution of histopathological changes characteristic of idiopathic pulmonary fibrosis within the lung parenchyma may result in diagnostic oversight or difficulty distinguishing IPF from other diseases during differential diagnosis (Dungworth et al., 1982; Flaherty et al., 2001).

Treatment and Prognosis: Although a variety of therapeutic procedures have been proposed (Noble et al., 2012; Garantziotis et al., 2004; Cottin et al., 2012), the treatment of pulmonary fibrosis remains limited regardless of its etiology, and in human medicine, lung transplantation is considered the only definitive therapeutic option for affected patients (Coll et al., 2013; Aurora et al., 2010). There is no specific curative therapy for the disease; therefore, treatment is primarily symptomatic. Oxygen supplementation, corticosteroids, bronchodilators, furosemide, and nebulization have been recommended (Evola et al., 2014). Inhibition of the TGF-β–mediated signaling pathway has been identified as one of the therapeutic mechanisms of pirfenidone, an antifibrotic agent (Moss et al., 2022).

Characteristic histopathological findings frequently observed inIPF include honeycombing, traction bronchiectasis, and fibroblastic foci, all of which are associated with disruption and remodeling of normal lung architecture. When these structural alterations, together with the underlying pathological processes, progress in parallel with clinical symptoms and radiographic abnormalities, the prognosis of the disease is generally poor (Raghu et al., 2011).

Pulmonary fibrosis has been investigated using various animal models, including species such as cats and dogs that are capable of developing spontaneous fibrotic processes. However, none of these models have been evaluated on a large scale, most likely due to the practical and ethical challenges associated with such studies. While current models have made important contributions to understanding the fundamental pathophysiology of fibrotic processes, it must be acknowledged that none can fully reflect the biological complexity of IPF. Therefore, future research should not only focus on improving existing models but also on testing and validating hypotheses across multiple model systems (Moss et al., 2022).

In humans, UIP demonstrates distinct findings on CT, with honeycombing and ground-glass opacities predominating in lung tissue images. In human medicine, if the patient's clinical presentation and physical examination findings are consistent with a CT scan showing a UIP pattern, a diagnosis of IPF may be established (Duffy et al., 2020). In another study, echocardiographic imaging revealed right ventricular dilation, hypertrophy, and pulmonary arterial hypertension in affected cats, with these findings thought to represent secondary changes due to primary pulmonary injury (Evola et al., 2014). In humans, pulmonary hypertension is known to frequently accompany pulmonary fibrosis (Smith et al., 2013; Pitsiou et al., 2011). The incidence of pulmonary hypertension in human IPF cases has been reported to range between 32% and 85% (Smith et al., 2013; Nathan et al., 2007).

The occurrence of the disease in both human and veterinary contexts underscores the necessity of therapeutic intervention. In cats suffering from IPF, clinical presentations

are generally unstable and tend to progress over time. Excluding differential diagnoses such as chronic bronchitis, asthma, infectious diseases and neoplastic processes plays a critical role in establishing the disease's specificity. The absence of a definitive therapeutic approach negatively impacts prognosis. More detailed classification of the disease, the development of novel treatment strategies, and comprehensive documentation of tomographic findings would facilitate diagnosis and contribute to more favorable prognostic outcomes.

Conclusion

Idiopathic pulmonary fibrosis in cats represents a progressive and often fatal interstitial lung disease that remains poorly understood and challenging to diagnose in clinical practice. The nonspecific nature of clinical signs and imaging findings frequently leads to delayed diagnosis or misclassification with other respiratory disorders. Although histopathology remains the definitive diagnostic method, its limited antemortem applicability highlights the need for improved non-invasive diagnostic approaches. Large-scale prospective studies integrating biomarker-focused investigations and standardized advanced imaging techniques are therefore essential to enhance diagnostic accuracy and disease characterization. Improved understanding of the pathogenesis and progression of feline pulmonary fibrosis may also facilitate earlier recognition and more informed prognostic assessment. Ultimately, such efforts have the potential to strengthen the translational value of feline pulmonary fibrosis as a comparative model, thereby bridging human and veterinary medicine.

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