Seminars in Diagnostic Pathology ∎ (∎∎∎∎) ∎∎∎–∎∎∎

Contents lists available at ScienceDirect

Seminars in Diagnostic Pathology

journal homepage: www.elsevier.com/locate/serndb

Review article

Histopathology of parasitic infections of the lung

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Introduction

Parasitic infections are important but uncommonly encountered causes of pulmonary disease in most resource-rich settings. Given that infections are generally diagnosed using conventional microbiology methods, many pathologists are uncomfortable identifying parasites in histological and cytological preparations. However, with increased global travel, pulmonary parasitic infections such as cysticercosis, malaria, paragonimiasis, and schistosomiasis may be encountered by pathologists outside of endemic areas. Furthermore, some parasites such as *Dirofilaria immitis, Echinococcus granulosus, Paragonimus kellicotti, Strongyloides stercoralis, Toxoplasma gondii,* and the free-living amebae are found in parts of North America and Europe, and may occasionally be observed in tissue sections. Thus, pathologists can play an important role in diagnosing parasitic infections, particularly when parasites are not considered by the clinical team and the appropriate microbiology tests have not been ordered. A basic understanding of the parasitic infections that occur in the human lung will aid in formulating a directed differential diagnosis or provide a specific identification, which can be very helpful to direct patient care.

Parasitic pulmonary infections may be caused by a variety of organisms, which can be divided into three main categories: those caused by protozoa, helminths (worms), and arthropods.[1–5] Of these, the first two categories are by far the most common, and will be the subject of this review. Understanding the clinical scenario in which the infection occurs is important, since factors such

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http://dx.doi.org/10.1053/j.semdp.2017.06.004
0740-2570/

as immune status, age, gender, and exposure history are often critical when constructing a differential diagnosis.[1,2,4,5] It should be noted that parasitic infections can cause a variety of histopathologic patterns in the lung, which are summarized in Table 1 and will be discussed in more detail below, including eosinophilic lung disease (eosinophilic pneumonia, etc.), eosinophilic abscess, granulomatous inflammation, vasculitis, pleuritis, empyema, and acute lung injury patterns (diffuse alveolar damage, organizing pneumonia, fibrinous pneumonia, etc.).[6,7] These patterns should alert surgical pathologists to the potential presence of parasites, and guide further pathological evaluation.

Protozoa

Lung infections caused by protozoa are usually encountered in the setting of disseminated systemic disease. Worldwide, the most common pulmonary protozoal infections include amebiasis,

Table 1
Summary of the histologic characteristics observed in the most common parasitic infections of the lung.

Organism	Typical histologic patterns	Identifying parasite characteristics	Helpful stains or techniques
Protozoa			
Entamoeba histolytica	• Necrotic abscess with few neutrophils • Non-specific inflammation, granulation tissue	• Trophozoites only (no cysts), usually $<35\ \mu m$ in greatest dimension • Abundant dense bubbly cytoplasm • Round eccentric nucleus with peripheral chromatin and central karyosome (nucleolus-like structure) • Erythrophagocytosis may be seen	• H&E • PAS to highlight trophozoites
Toxoplasma gondii	• DAD • Necrotizing acute pneumonia	• Tachyzoites (round, oval or arc-shaped organisms), 2–8 μm in length • Intracellular (macrophages or alveolar epithelium), or extracellular within necrosis	• H&E • Commercial immunohistochemical stain
Helminths			
Nematodes			
Dirofilaria immitis	• Discrete necrotic nodule • Necrotizing granulomas with increased eosinophils	• One or more larvae seen in longitudinal and cross-sections, 100–350 μm diameter • Usually within a medium-sized artery in the area of necrosis • Thick (5–25 μm) cuticle with transverse striations • Usually degenerating/calcified, but internal lateral ridges can often still be seen.	• H&E
Strongyloides stercoralis	• Mixed inflammation +/- granulomas • DAD • Diffuse alveolar hemorrhage	• Larvae within alveoli; 400–700 μm in length and 10–20 μm wide • Rows of small internal nuclei; no identifiable organs in larvae • Notched tail (seen primarily in cytology specimens)	• H&E
Cestodes			
Echinococcus granulosis	• Cystic lesion; may be quite large and contain smaller cysts ("Cysts within a cyst" appearance) • Surrounding fibrotic rim and compressed host tissue • Rupture leads to granulomatous and acute inflammation • Aspirate fluid has gritty texture ("hydatid sand")	• Cysts have a lamellated acellular wall with an underlying germinal layer • Multiple protoscoleces (immature tapeworms with an inverted scolex) may be seen within cysts; 100 μm in diameter, free hooklets are 15–30 μm • Protoscoleces with two circular rows of hooklets and four cup-shaped suckers • Free hooklets are refractile and weakly birefringent	• H&E • Narrowed condenser to identify hooklets • GMS and PAS to highlight laminated layer • Ziehl-Neelsen positive hooklets
Taenia solium	• Thin-walled small cysts (cysticerci) • If organism is degenerating, associated mixed inflammation including neutrophils and eosinophils	• Cysticerci are 3–10 mm in greatest dimension • No outer laminated layer like *Echinococcus granulosus* • One protoscolex per cysticercus; attached at an infolded neck region • Invaginated protoscolex with four suckers and refractive hooklets	• H&E • Narrowed condenser to identify hooklets
Trematodes			
Paragonimus sp.	• Adult flukes in cystic cavity • Chronic eosinophilic pneumonia • Eosinophilic pleuritis and vasculitis • Necrotizing granulomas around eggs	• Eggs and/or adult flukes may be present • Eggs with refractile, strongly birefringent wall, shouldered operculum; 90 μm in length • Spines present on the tegmentum of the fluke • Ventral acetabulum/sucker of the fluke	• H&E • GMS (eggs)
Schistosoma sp.	• Intravascular eggs • Granulomatous endarteritis • Granulomas with increased eosinophils • Vascular changes of pulmonary hypertension	• Eggs seen; adults are not present in the lung (in venules of intestine or bladder) • 90–180 μm eggs with internal small nuclei (miracidium/larval form) • Spines present on eggs, which may be lateral, terminal, or rudimentary (Not always seen) • Eggs have thin wall, commonly collapsed in tissue, little to no birefringence • Eggs may have surrounding Splendore-Hoeppli phenomenon	• H&E

Abbreviations: DAD – diffuse alveolar damage.

Please cite this article as: Boland JM, Pritt BS. (2017), http://dx.doi.org/10.1053/j.semdp.2017.06.004