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Chest CT Features of North American Paragonimiasis

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Abstract

OBJECTIVE—The purpose of this study was to characterize the chest CT findings of North American paragonimiasis due to *Paragonimus kellicotti* in the largest (to our knowledge) case series reported to date and to compare the findings with those reported for paragonimiasis infections in other regions.

MATERIALS AND METHODS—A retrospective review was performed of chest CT examinations of eight patients with North American paragonimiasis treated at our institution between 2006 and 2010. Findings were characterized by site of involvement, including lungs and pleura, heart and pericardium, lymph nodes, and upper abdomen.

RESULTS—The most common chest CT findings in this case series were pleural effusions and internal mammary and cardiophrenic lymphadenopathy. Pulmonary parenchymal findings included peripheral lung nodules of 1–3.5 cm in size with surrounding ground-glass opacity; many nodules had a linear track to the pleural surface that may correspond to the worm's burrow tunnel. Pericardial involvement (5/8 patients) and omental inflammation (5/7 patients), which are uncommon in Asian paragonimiasis, were common in this series.

CONCLUSION—Pleural and pulmonary features of North American paragonimiasis are generally similar to those reported from Asia. The presence of a track between a pulmonary nodule and the pleura may help distinguish paragonimiasis from mimickers, including chronic eosinophilic pneumonia, tuberculosis, fungal infection, or malignancy. Pericarditis, lymphadenopathy, and omental inflammation were more common in our series than in reports on paragonimiasis from other regions. These differences may be related to the infecting parasite species or to the fact that radiologic examinations in the present series were performed relatively early in the course of infection.

Keywords

eosinophilia; paragonimiasis; *Paragonimus kellicotti*; parasite; worm

Paragonimiasis is a parasitic infection caused by trematodes (or lung flukes) of the genus *Paragonimus*. The parasite has a complex life cycle including two intermediate hosts and a definitive mammalian host. Humans are accidental hosts who become infected by eating raw or improperly cooked crab or crawfish. Worldwide, several million people are believed to be chronically infected with various *Paragonimus* species, the most common being *P. westermani*, which is endemic to parts of Asia [1]. The radiographic and CT findings of

pleuropulmonary infection from *P. westermani* have been described in numerous studies [2–8]. In contrast, only six cases of infection caused by the native North American species, *P. kellicotti*, were reported between 1984 and 2007 [9–15]. Between 2006 and 2010, however, there have been at least 10 clinically confirmed cases of infection by *P. kellicotti* in the midwestern United States, eight of which were treated at our institution [16, 17]. We retrospectively reviewed the CT and clinical findings in these eight patients to identify specific chest CT features that may help in the diagnosis of this uncommon infection.

Materials and Methods

Between July 2006 and September 2010, eight patients (7 male and 1 female; age range, 10–32 years; mean, 25 years) with serologically confirmed ($n = 5$) or clinically diagnosed ($n = 3$) paragonimiasis were identified at our institution. The Washington University School of Medicine Human Research Protection Office approved our retrospective review of the electronic medical records and radiologic imaging. A waiver of consent was granted for this HIPAA-compliant study.

All eight patients underwent chest CT before the diagnosis of paragonimiasis was made (range, 2–33 weeks between onset of symptoms and performance of CT; mean, 10.5 weeks). All eight studies were performed on MDCT scanners from multiple manufacturers. However, because of the retrospective nature of this study, there was no standard protocol. Seven of the eight studies were performed with iodinated IV contrast, and the eighth study was not contrast enhanced. Images were reviewed by two board-certified radiologists with a focus in cardiothoracic imaging with 12 and 2 years of experience, respectively. Final interpretations were determined by consensus.

Image Analysis

Findings were categorized by organ system. Pleural findings included pleural effusion, pleural thickening (i.e., a visible pleural stripe thicker than 1 mm), and increased extrapleural fat. Pulmonary nodules were evaluated by size, location, margins, attenuation, and surrounding ground-glass attenuation. Peripheral nodules were defined as either abutting pleura or as surrounded by lung distal to segmental bronchi. Additional parenchymal findings, including linear opacities and areas of atelectasis, were characterized. Lymph nodes were considered enlarged if the short-axis measurement was greater than 10 mm (mediastinal and hilar), greater than 5 mm (cardiophrenic), or visible (internal mammary). Pericardium was considered thickened if it measured at least 2 mm, and pericardial effusion size was subjectively classified as physiologic, small, moderate, or large. When included, the upper abdomen was also evaluated for the presence of ascites and peritoneal inflammation.

Establishing the Diagnosis

In the United States, the two most commonly used serologic tests for the diagnosis of paragonimiasis are an immunoblot assay offered by the Centers for Disease Control and Prevention and a commercially available ELISA. Both tests detect antibodies to antigens of *P. westermani* [1]. Because there is no definitive serologic test for the diagnosis of pleuropulmonary paragonimiasis due to *P. kellicotti* infection, the *P. westermani* tests are frequently used. Five of the eight patients in this study had a positive serologic test (four by immunoblot and one by ELISA). Three of the patients had negative serologic studies. However, the strong clinical history, including prior consumption of raw crayfish from Missouri rivers in all patients, characteristic laboratory and imaging findings, and rapid response to appropriate therapy, were sufficient to establish a clinical diagnosis in these

patients. The sensitivity and specificity of the immunologic tests targeted for *P. westermanni* are unknown when they are used for the diagnosis of *P. kellicotti* infection [1, 16].

Results

Clinical Findings

All eight patients had consumed at least one raw crawfish from rivers in southeastern Missouri before the onset of their symptoms. Patients ranged in age from 10 to 32 years. All adult cases occurred while on float trips where alcohol use was present. The sole pediatric patient was demonstrating his “survival skills” to his relatives. All but one of the patients were male.

Illness onset occurred 2–12 weeks after ingestion of raw crayfish. Most patients presented with fever (8/8 [100%]), cough (8/8 [100%]), night sweats (5/8 [62.5%]), and malaise (5/8 [62.5%]), and many of the patients were initially considered to have pneumonia. Two patients experienced upper quadrant abdominal pain 6–8 weeks after crayfish consumption. One of these patients underwent emergent cholecystectomy for suspected acute cholecystitis. However, the gallbladder was normal.

The time from onset of symptoms to diagnosis was 3–40 weeks (mean, 16 weeks; median, 12 weeks). During this time, patients often received multiple courses of antibiotics, corticosteroids, and other invasive procedures without significant improvement in their clinical symptoms.

Eosinophilia is a hallmark laboratory finding in patients with paragonimiasis. All patients had elevated absolute blood eosinophil counts of 600–3900 cells/ μ L (5.6–40%). All five patients who underwent thoracentesis had eosinophilic pleural effusions, and all three patients who underwent bronchoalveolar lavage had eosinophilia in the bronchoalveolar lavage fluid. One patient had a pericardiocentesis with an exudative effusion that contained 60,000 WBCs with 50% eosinophils.

Pleural and Parenchymal Imaging Findings

All eight patients had pleural effusions that ranged from small to moderate in size. One patient presented with a spontaneous right-sided hydropneumothorax. Six of the eight patients (75%) had pleural thickening, and of those patients, four also had increased extra-pleural fat indicative of more long-standing pleural inflammation (Fig. 1). One patient with pleural thickening (12.5%) had associated rounded atelectasis.

Four patients (50%) had at least one “worm nodule,” a lesion we defined as having two components: a pulmonary nodule at least partially surrounded by ground-glass attenuation and a linear opacity connecting the nodule to the pleural surface (Fig. 2). The nodules were typically lobulated, measured 10–35 mm in diameter, and had attenuation intermediate to fluid and skeletal muscle. Two patients had peripheral nodules abutting the pleura with surrounding ground-glass attenuation without linear tracks (Fig. 3). Five patients (62.5%) had isolated linear opacities (those without associated parenchymal nodules) emanating from the visceral pleura and abruptly terminating within the lung parenchyma (including the two patients with nodules not meeting the criteria listed previously in this article). These tracks were 5–70 mm long and were at least 2 mm thick (Fig. 4). Three patients (37.5%) had at least one indeterminate pulmonary nodule smaller than 6 mm, including one 5-mm nodule with a central cavitation. These indeterminate nodules did not have associated tracks to the pleural surface or surrounding ground-glass attenuation. The pleuropulmonary findings are summarized in Table 1.

Additional Imaging Findings

Additional chest CT abnormalities included pericardial thickening or pericardial effusion in five patients (62.5%) (Fig. 5) and thoracic lymphadenopathy. Notably, internal mammary and cardiophrenic lymphadenopathy was present in all eight patients (Fig. 6). In the seven patients in whom portions of the upper abdomen were included, inflammation and stranding of the greater omentum were present in five patients (71.4%) (Fig. 7), and perihepatic ascites was present in one patient (12.5%). These additional findings are summarized in Table 2.

Discussion

Life Cycle of *Paragonimus* Species

P. kellicotti has a life cycle similar to that of other *Paragonimus* species in that it includes two intermediate hosts (i.e., snails and crustaceans) and a definitive host (i.e., mammals) (Fig. 8). Our study highlights some of the unique CT features that may contribute to the diagnosis of paragonimiasis resulting from *P. kellicotti* infection, and understanding the life cycle is helpful to explain the spectrum of clinical and radiologic findings we observed.

Metacercariae (immature organisms) of *P. kellicotti* live in the heart of crayfish, and this stage is infective for mammals that ingest the crayfish [1, 18, 19]. Metacercariae excyst in the digestive tract and penetrate the small-bowel wall to enter the peritoneal cavity within hours of ingestion [1, 8]. Approximately 2–3 weeks later, the organisms pass through the diaphragm and enter the pleural space. The organisms penetrate through the visceral pleura and (later) burrow into the lung parenchyma. The linear opacities observed by radiography and CT probably represent burrow tracks of the worms, and experimental studies support this explanation [4]. Once in the lung parenchyma, a cavity of 1–2 cm in diameter forms around the worms in the vicinity of bronchioles [18], where the organisms produce eggs. The eggs penetrate the bronchioles and exit the host—by either being coughed into the air or by being swallowed and passed in the stool—thus completing the life cycle [1, 18]. Alternatively, eggs may end up in the pleural space (a dead end for reproduction), where they may incite an intense inflammatory host response [1].

Imaging Findings in the Context of Other *Paragonimus* Species

Only six cases of North American paragonimiasis infections were reported in the literature between 1984 and 2007. The imaging findings for these cases are summarized in Table 3. These case reports focused on the clinical presentation or pathology, rather than imaging. However, imaging results for these cases are consistent with our findings, including effusions, spontaneous pneumothoraces, and areas of consolidation that are difficult to further characterize by chest radiography.

In 2005, Kim et al. [3] reported chest CT findings for 31 patients with pleuropulmonary paragonimiasis caused by *P. westermani*, the most common cause of paragonimiasis in Asia. The most frequent finding in their study was a poorly marginated (74%) peripheral or subfissural nodule of approximately 2 cm in diameter with a necrotic center (87%). Many nodules had a “subpleural streaky opacity” connecting the nodule to the pleural surface (48%) and adjacent ground-glass attenuation (58%). Pleural findings included focal pleural thickening adjacent to a nodule (87%) and fissural thickening (32%). Pleural effusions, hydropneumothorax, or pneumothorax were seen in only 29% of patients, and mediastinal or hilar lymphadenopathy was reported in only 13% of patients. There was no mention of cardiophrenic or internal mammary lymphadenopathy.

The parenchymal findings reported by Kim et al. [3] in patients with paragonimiasis caused by *P. westermani* are similar to our findings in North American patients with infection

caused by *P. kellicotti*. Four of our eight patients had the typical worm nodule connected to the pleura by a linear track, analogous to the “streaky linear opacity” previously described with *P. westermani* infection, and an additional two patients had similar appearing nodules in continuity with the visceral pleura (these nodules may have had tracks that were too short to see on CT). Kim et al. [3] and other authors [2, 6, 8] have described pulmonary lesions with air-filled cavities, lung cysts, or bronchiectasis. These findings were not observed in our cases, and we suspect this is because of a difference in chronicity of the infections. Although some of our patients were ill for several months before diagnosis, other studies included patients who were probably infected for several years. The acute presentation of our patient population may also explain the higher incidence of pleural effusions (100%) than reported in prior series. Pleural effusions appear to be more common in early stages of the infection.

Three findings in our series—lymphadenopathy, pericardial disease, and omental inflammation—were observed with a much higher frequency than has been previously reported. All eight of our patients had enlarged cardiophrenic and internal mammary lymph nodes. The cardiophrenic nodes are known to drain the anterior mediastinum and anterior liver, and they are contiguous with the internal mammary chains [20]. Because none of these lymph nodes was biopsied, we can only speculate that they are reactive to inflammation in the upper abdomen, diaphragm, or pleural membranes.

Pericarditis is a rare manifestation of paragonimiasis that has been sparsely reported in the literature. Saborio et al. [21] described two pediatric patients in Costa Rica with pericarditis due to *P. mexicanus*, a species that is endemic to Central and South America. Neither patient underwent CT, but echocardiography showed moderate-to-large effusions.

Pericardiocentesis revealed exudative effusions with eosinophilia in both cases. It is notable that a high percentage of our cases had apparent pericardial involvement; five of our eight patients had pericardial thickening and three had pericardial effusions. None of our patients reported prior episodes of pericarditis. Perhaps *P. kellicotti* has a greater affinity for the pericardium than other *Paragonimus* species; however, none of the six previous cases of North American paragonimiasis outlined in Table 3 were reported to have had pericardial involvement. Another possibility is that pericardial effusions are transient during the early stages of infection—an unintended consequence of the worms taking a “wrong turn” in the chest as they migrate to the lungs—and that they are uncommonly seen in patients with chronic infections.

Abdominal Paragonimiasis

Ectopic paragonimiasis has been widely reported with species other than *P. kellicotti*, and the incidence of these findings is unknown on the basis of the limited number of cases. Peritoneal paragonimiasis has been reported with *P. westermani* [22–25], but not with *P. kellicotti*. The imaging appearance of peritoneal paragonimiasis consists of inflammation or nodules, and it can resemble peritoneal carcinomatosis or tuberculous peritonitis. The nodules may be cystic or calcified. Of note, CA-125 levels may be elevated [26], leading to further confusion with peritoneal carcinomatosis.

Differential Diagnosis

The radiologic differential diagnosis of thoracic paragonimiasis includes malignancy, atypical infection (i.e., fungal or mycobacterial), and eosinophilic pneumonia. It is important to note that patients with paragonimiasis generally present with a clinical picture that is mistaken for community-acquired pneumonia, and it is only after failure of the initial treatment with antibiotics that further evaluations (e.g., laboratory studies and CT) are performed.

Multiple case reports have described false-positive ^{18}F -FDG PET results in *P. westermani* infection [26–29], where malignancy was suspected. *P. westermani* infection causes increased metabolic activity in both pulmonary nodules and associated lymph nodes. We assumed that *P. kellicotti* lesions would have a similar appearance, but none of the patients in our series had FDG PET studies.

Fungal infection and tuberculosis can have similar presenting symptoms and imaging findings, with nodules or consolidation, lymphadenopathy, and effusions. However, the presence of a linear track to the pleura would be very unusual in patients with these types of infection.

Paragonimiasis shares several clinical and imaging features with chronic eosinophilic pneumonia, including chronic symptoms, eosinophilia, and areas of peripheral consolidation. Chronic eosinophilic pneumonia is almost always bilateral, whereas only three of our eight patients had bilateral parenchymal disease. Moreover, pleural effusions and lymphadenopathy are seen in only 10% and 10–15%, respectively, of cases of chronic eosinophilic pneumonia [30]. Other uncommon causes of eosinophilia with pulmonary disease include other parasites (e.g., ascariasis, strongyloidiasis, and tropical pulmonary eosinophilia) and Churg-Strauss vasculitis [15].

Limitations

Our study has limitations. First, only five of the eight patients had positive serologic tests for *Paragonimus* species infection. However, the history of raw crawfish ingestion, eosinophilia, and rapid response to praziquantel in each of the other three cases makes the diagnosis of paragonimiasis overwhelmingly likely. Furthermore, currently available serologic tests are based on *P. westermani*. However, metacercariae consistent with *P. kellicotti* were detected in the majority of crayfish tested from rivers from which our patients consumed crayfish. DNA sequencing confirmed the species identification in the crayfish, and identical parasite sequences in clinical specimens from two of our patients [19].

Second, none of the pulmonary lesions was resected to offer pathologic proof that worms or burrow tracks were present. Our observations are made in the context of clinical symptoms, laboratory values, and imaging features that have been described in prior studies of paragonimiasis.

Conclusion

Pleural and pulmonary parenchymal findings in *P. kellicotti* infection are generally similar to those reported in patients with *P. westermani* infection. These include pleural effusions and peripheral nodules with surrounding ground-glass attenuation. The presence of a worm track connecting the nodule to the pleura, if present, can help to distinguish this entity from malignancy or other infections in the appropriate clinical context of eosinophilia and a history of raw crawfish ingestion. Internal mammary and cardiophrenic lymphadenopathy, pericardial disease, and omental inflammation were also common in this series. The extrapulmonary findings help differentiate this entity from chronic eosinophilic pneumonia.

Finally, this series serves as a reminder that parasitic infections can occur in patients without foreign travel. Although all of our patients were residents of either Missouri or Illinois who consumed crayfish while in Missouri, *P. kellicotti* is widely distributed throughout North America. The prior cases of North American paragonimiasis included patients from Iowa, Colorado, Oklahoma, and Michigan (in addition to Missouri). Physicians should consider the possibility of paragonimiasis in patients with fever, pulmonary symptoms, and eosinophilia.

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References

1. Procop GW. North American paragonimiasis (caused by *Paragonimus kellicotti*) in the context of global paragonimiasis. Clin Microbiol Rev. 2009; 22:415–446. [PubMed: 19597007]
2. Im JG, Whang HY, Kim WS, Han MC, Shim YS, Cho SY. Pleuropulmonary paragonimiasis: radiologic findings in 71 patients. AJR. 1992; 159:39–43. [PubMed: 1609718]
3. Kim TS, Han J, Shim SS, et al. Pleuropulmonary paragonimiasis: CT findings in 31 patients. AJR. 2005; 185:616–621. [PubMed: 16120908]
4. Im JG, Kong Y, Shin YM, et al. Pulmonary paragonimiasis: clinical and experimental studies. RadioGraphics. 1993; 13:575–586. [PubMed: 8316665]
5. Singcharoen T, Silprasert W. CT findings in pulmonary paragonimiasis. J Comput Assist Tomogr. 1987; 11:1101–1102. [PubMed: 3680703]
6. Mukae H, Taniguchi H, Matsumoto N, et al. Clinicoradiologic features of pleuropulmonary *Paragonimus westermani* on Kyusyu Island, Japan. Chest. 2001; 120:514–520. [PubMed: 11502652]
7. Jeon K, Koh WJ, Kim H, et al. Clinical features of recently diagnosed pulmonary paragonimiasis in Korea. Chest. 2005; 128:1423–1430. [PubMed: 16162738]
8. Kagawa FT. Pulmonary paragonimiasis. Semin Respir Infect. 1997; 12:149–158. [PubMed: 9195680]
9. Pachucki CT, Levandowski RA, Brown VA, Sonnenkalb BH, Vruno MJ. American paragonimiasis treated with praziquantel. N Engl J Med. 1984; 311:582–583. [PubMed: 6749230]
10. Mariano EG, Borja SR, Vruno MJ. A human infection with *Paragonimus kellicotti* (lung fluke) in the United States. Am J Clin Pathol. 1986; 86:685–687. [PubMed: 3776924]
11. Procop GW, Marty AM, Scheck DN, Mease DR, Maw GM. North American paragonimiasis: a case report. Acta Cytol. 2000; 44:75–80. [PubMed: 10667165]
12. DeFraín M, Hooker R. North American paragonimiasis: case report of a severe clinical infection. Chest. 2002; 121:1368–1372. [PubMed: 11948081]
13. Castilla EA, Jessen R, Scheck DN, Procop GW. Cavitary mass lesion and recurrent pneumothoraces due to *Paragonimus kellicotti* infection: North American paragonimiasis. Am J Surg Pathol. 2003; 27:1157–1160. [PubMed: 12883250]
14. Madariaga MG, Ruma T, Theis JH. Autochthonous human paragonimiasis in North America. Wilderness Environ Med. 2007; 18:203–205. [PubMed: 17896846]
15. Boé DM, Schwarz MI. A 31-year-old man with chronic cough and hemoptysis. Chest. 2007; 132:721–726. [PubMed: 17699148]
16. Centers for Disease Control and Prevention. Human paragonimiasis after eating raw or undercooked crayfish: Missouri, July 2006–September 2010. MMWR Morb Mortal Wkly Rep. 2010; 59:1573–1576. [PubMed: 21150864]
17. Lane MA, Barsanti MC, Santos CA, Yeung M, Lubner SJ, Weil GJ. Human paragonimiasis in North America following ingestion of raw crayfish. Clin Infect Dis. 2009; 49:e55–e61. [PubMed: 19681705]
18. Garcia, LS. Diagnostic medical parasitology. 5. Washington, DC: ASM Press; 2007.
19. Fischer PU, Curtis KC, Marcos LA, Weil GJ. Molecular characterization of the North American lung fluke *Paragonimus kellicotti* in Missouri and its development in Mongolian gerbils. Am J Trop Med Hyg. 2011; 84:1005–1011. [PubMed: 21633042]
20. Aronberg DJ, Peterson RR, Glazer HS, Sagel SS. Superior diaphragmatic lymph nodes: CT assessment. J Comput Assist Tomogr. 1986; 10:937–941. [PubMed: 3782563]

21. Saborio P, Lanzas R, Arrieta G, Arguedas A. *Paragonimus mexicanus* pericarditis: report of two cases and review of the literature. J Trop Med Hyg. 1995; 98:316–318. [PubMed: 7563258]
22. Kim JY, Kang CM, Choi GH, et al. Laparoscopic excision of intra-abdominal paragonimiasis. Surg Laparosc Endosc Percutan Tech. 2007; 17:556–558. [PubMed: 18097324]
23. Tomashefski, JF.; Dail, DH. Dail and Hammar's pulmonary pathology. 3. New York, NY: Springer-Verlag; 2008.
24. Yoo E, Kim JH, Kim MJ, et al. Greater and lesser omenta: normal anatomy and pathologic processes. RadioGraphics. 2007; 27:707–720. [PubMed: 17495288]
25. Kim SY, Ha HK. Peritoneal manifestations of parasitic infection. Abdom Imaging. 2008; 33:172–176. [PubMed: 17924162]
26. Kim KU, Lee K, Park HK, Jeong YJ, Yu HS, Lee MK. A pulmonary paragonimiasis case mimicking metastatic pulmonary tumor. Korean J Parasitol. 2011; 49:69–72. [PubMed: 21461272]
27. Yoo IR, Park HJ, Hyun J, et al. Two cases of pulmonary paragonimiasis on FDG-PET CT imaging. Ann Nucl Med. 2006; 20:311–315. [PubMed: 16856575]
28. Watanabe S, Nakamura Y, Kariatsumari K, et al. Pulmonary paragonimiasis mimicking lung cancer on FDG-PET imaging. Anticancer Res. 2003; 23:3437–3440. [PubMed: 12926086]
29. Osaki T, Takama T, Nakagawa M, Oyama T. Pulmonary *Paragonimus westermani* with false-positive fluorodeoxyglucose positron emission tomography mimicking primary lung cancer. Gen Thorac Cardiovasc Surg. 2007; 55:470–472. [PubMed: 18049856]
30. Johkoh, T. Eosinophilic lung diseases. In: Müller, NL.; Silva, CIS., editors. Imaging of the chest. Philadelphia, PA: Saunders/Elsevier; 2008. p. 853-865.

Fig. 1. 20-year-old man with North American paragonimiasis. Axial contrast-enhanced CT image through lower thorax shows bilateral pleural effusions (*straight arrow*) with pleural thickening and increased extrapleural fat in left hemithorax, indicative of chronic inflammation. Mild pericardial thickening (*arrowheads*) and anterior diaphragmatic lymphadenopathy (*curved arrow*) are also present.

Fig. 2. Three patients with North American paragonimiasis and nodules connected to visceral pleura by linear tracks

A and B, 30-year-old man. Oblique-axial CT image (**A**) shows ill-defined nodule (*arrow*) in left upper lobe with surrounding ground-glass opacification. Nodule is connected to visceral pleura anteriorly by small linear track (*arrowhead*). Also note bilateral pleural effusions. Oblique-axial unenhanced CT image (**B**) shows thin linear track to pleura (*arrowhead*) and ill-defined nodule (*straight arrow*), which is intermediate attenuation to fluid and muscle. Also note bilateral pleural effusions (*curved arrow*) with thickening of mediastinal pleura in left hemithorax.

C and D, 31-year-old man. Axial CT (**C**) shows lobulated nodule (*arrow*) in apical segment of right upper lobe with surrounding ground-glass opacification. Thin linear track (*arrowhead*) connects nodule to visceral pleural surface. Axial contrast-enhanced CT (**D**) more clearly defines linear track (*arrowhead*) extending to visceral pleura and intermediate attenuation of lobulated right upper lobe nodule (*arrow*). Also note right pleural effusion and high right paratracheal lymph node.

E and F, 22-year-old man. Oblique-axial CT image (**E**) shows lobulated left upper lobe nodule (*arrow*) and areas of surrounding ground-glass opacification. Nodule is connected to visceral pleura by curvilinear track (*arrowhead*). Oblique-axial contrast-enhanced CT (**F**) shows that nodule (*arrow*) has lobulated contour and heterogeneous attenuation, and linear track (*arrowhead*) connecting it to pleural surface is faintly visible. Small left pleural effusion was present (not shown).

Fig. 3. Two patients with North American paragonimiasis and peripheral nodules abutting visceral pleura

A, 28-year-old man. Axial contrast-enhanced CT shows peripheral nodule (*arrowhead*) in apical segment of right upper lobe, abutting visceral pleura. Note large halo of ground-glass attenuation and posteriorly layering right pleural effusion.

B, 10-year-old boy. Axial contrast-enhanced CT shows peripheral nodule (*arrowhead*) abutting visceral pleural in apicoposterior segment of left upper lobe. Note moderate left pleural effusion.

Fig. 4. Two patients with North American paragonimiasis and isolated linear tracks. (Courtesy of Graham WK, St. Frances Medical Center, Cape Girardeau, MO)

A, 32-year-old man. Axial contrast-enhanced CT shows isolated linear track (*arrowhead*) in anterior segment of left upper lobe extending from anterior pleural surface. Note subtle ground-glass attenuation surrounding this track. Patient had bilateral lung involvement, including multiple tracks with nodules in right lung (not shown) and right hydropneumothorax.

B, 26-year-old woman. Oblique coronal multiplanar reformation of contrast-enhanced CT shows long linear track (*arrowheads*) in left upper lobe emanating from visceral pleural at left lung apex. Track was approximately 5.5 cm long as it meandered toward left hilum. Note ground-glass attenuation surrounding portions of this track.

Fig. 5. Two patients with pericardial involvement of North American paragonimiasis

A, 10-year-old boy. Axial contrast-enhanced CT scan through heart shows moderate-sized pericardial effusion (*arrows*) with pericardial thickening and enhancement. Echocardiography (not shown) revealed increased echogenicity within fluid, suggestive of exudative effusion.

B, 32-year-old man. Axial contrast-enhanced CT shows mild pericardial thickening (*arrowheads*). Patient also had bilateral pleural effusions and pleural thickening (*arrows*).

Fig. 6. 28-year-old man with North American paragonimiasis. Axial contrast-enhanced CT scan through midthorax shows bilateral internal mammary lymphadenopathy (*arrowheads*), typical finding in our series of patients. Also note right pleural effusion and pericardial effusion tracking into superior pericardial recess.

Fig. 7. 22-year-old man with North American paragonimiasis. Axial contrast-enhanced CT scan at soft-tissue windows shows extensive inflammation of greater omentum (*arrowheads*). Also note areas of peritoneal thickening and enhancement (*arrow*). Patient also had small volume of perihepatic ascites (not shown).

Fig. 8. Life cycle and intermediate host of *Paragonimus kellicotti*

A, Graph illustrates life cycle of *P. kellicotti*. Reprinted from Centers for Disease Control and Prevention Web site (www.dpd.cdc.gov/dpdx/HTML/ImageLibrary/Paragonimiasis_il.htm).

B, Photograph of golden crayfish (*Orconectes luteus*), one of several crayfish species that serves as intermediate host for *P. kellicotti*. (Courtesy of Fischer P, Washington University School of Medicine, St. Louis, MO)

TABLE 1

Pleural and Lung Parenchymal Findings in Eight Patients With North American Paragonimiasis

Finding	Percentage (No.) of Patients
Pleura	
Pleural effusion	100 (8)
Bilateral	62.5 (5)
Unilateral (2 left, 1 right)	37.5 (3)
Hydropneumothorax	12.5 (1)
Pleural thickening	75 (6)
Increased extrapleural fat	50 (4)
Rounded atelectasis	12.5 (1)
Lung parenchyma	
Worm nodule (nodule with pleural track)	50 (4)
Single	25 (2)
Multiple	25 (2)
Nodule without linear track	25 (2)
Isolated linear track	75 (6)
Single	62.5 (5)
Multiple	12.5 (1)
Indeterminate nodule	37.5 (3)

TABLE 2

Summary of Additional Findings in Eight Patients With North American Paragonimiasis

Finding	Percentage (No.) of Patients
Pericardium	
Thickening	62.5 (5)
Effusion	37.5 (3)
Lymphadenopathy	
Internal mammary	100 (8)
Cardiophrenic	100 (8)
Mediastinal	87.5 (7)
Hilar	87.5 (7)
Upper abdomen	
Inflammation of greater omentum	71.4 (5) ^a
Perihepatic ascites	12.5 (1)

^aOnly seven of eight patients had visible greater omentum.

TABLE 3Summary of Radiologic Findings in Prior Reports of *Paragonimus kellicotti* Infection

Patient, Study	Radiologic Findings
19-year-old man [9, 10]	Initial presentation with spontaneous left hydropneumothorax; subsequently developed bilateral pleural effusions, right upper lobe "infiltrate," pleural thickening, and right hilar lymphadenopathy
21-year-old man [11]	"Nonspecific linear streaking" on one chest radiograph; all other radiographs were described as normal
18-year-old man [12] ^a	Bilateral effusions and left lung "infiltrate" on chest radiographs; bilateral pleural effusions and left pleural thickening on subsequent CT
35-year-old man [13] ^a	Initial presentation with spontaneous right hydropneumothorax; "cavitary mass lesion" on CT 1 year later
71-year-old man [14]	Right pleural effusion
31-year-old man [15] ^a	Right lower lobe "consolidation," "necrosis," and "nodule formation" on CT

^aThese case reports included CT findings; other case reports included only chest radiograph findings.