

revealed a 2 cm rupture in the distal third of the trachea. The lesion was situated at the end of the orotracheal tube, so the tube was advanced with the fiberoptic bronchoscope until the distal end was in the right main bronchus (Fig. 1B). Despite this maneuver, the patient's poor respiratory status, consistent with acute respiratory distress syndrome (ARDS) persisted, so we decided to administer support therapy with venovenous extracorporeal membrane oxygenation (VV-ECMO). After the patient's hemodynamic and respiratory situation stabilized, a self-expanding fully-coated nitinol tracheal stent was placed and the orotracheal tube was relocated to the site of the stent, re-establishing bipulmonary ventilation (Fig. 1C). After 13 days of support, ECMO was withdrawn and bronchoscopy-guided tracheostomy was performed. The patient remained stable and was discharged, almost 3 months after admission, after withdrawal of the cannula and removal of the stent by bronchoscopy.

Tracheobronchial lesions caused by tracheostomy are rare, occurring at a rate of around 0.2%–0.7%.¹ In our case, the procedure may have been complicated by the patient's history of repaired distal tracheoesophageal fistula and the resulting distortion of the tracheal pars membranosa in this region, increasing the chance of injury. Self-expanding prostheses are a useful therapeutic option, particularly in patients with a high surgical risk, or in those in whom conservative treatment is ineffective.²

ARDS is associated with a mortality of 45%–55%.³ A protective pulmonary ventilation strategy using low tidal volumes and prone positioning of the patient are the only therapeutic measures shown to improve survival.⁴ In this context, ECMO-VV support provides good oxygenation and ventilation, therefore minimizing ventilatory support and barotrauma associated with mechanical ventilation.⁵

Our patient's situation was complicated by lesions caused by aspiration, and we found it impossible to keep him properly ventilated, so lung function support with VV-ECMO was required before the tracheal stent could be placed. This case demonstrates the benefit of ECMO in the management of patients with ARDS, and its utility as a bridge to the definitive treatment of tracheal rupture.

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Pulmonary Dirofilariasis: A Diagnostic Pitfall in Clinical Practice*



Dirofilariasis pulmonar: un reto diagnóstico en la práctica clínica

To the Editor,

Human pulmonary dirofilariasis is an uncommon parasitosis caused by *Dirofilaria immitis*. The usual hosts and reservoirs are domestic and wild carnivores, while humans are inadvertent hosts through the vector by mosquito bites. Pulmonary infection is extremely rare, often asymptomatic and usually discovered on routine chest radiograph as a solitary lesion, commonly mislabeled as malignant tumor.¹

A 45-year-old Caucasian female was referred to the family doctor complaining of influenza-like symptoms and chest pain. She was a smoker (1 pack-day over the last 15 years), but had no contact with dogs or other pets. She denied any travel outside Greece. Chest X-ray revealed a small peripheral solitary pulmonary nodule in the right upper lobe. Clinical examination was unremarkable. White blood cell count, erythrocyte sedimentation rate and C-reactive protein were normal. Tuberculin skin test was negative, as was sputum testing for common bacteria, and so empirical antibiotic therapy was initiated. One month later, symptoms were resolved

but a new X-ray showed no radiological change, so lung cancer was suspected.

Computed tomography of the chest showed a well-defined 1.5 cm non-calcified nodule located peripherally on the right upper lobe abutting the parietal pleura with no lymphadenopathy. Fiberoptic bronchoscopy was normal. Microbiological and cytological analyses of bronchoalveolar lavage were negative. Immunological tests and screening for vasculitis were also negative.

Surgical resection was performed to rule out the tumor hypothesis. A 1.5 cm soft grayish yellow nodule was found on the upper lobe, not directly in contact with the pleura. A wedge resection of the nodule was performed. Frozen sections showed a benign lesion characterized by necrosis. Final microscopic examination documented a well-circumscribed necrotic nodule containing fragments of a non-viable parasite that had features of *D. immitis*, including a smooth surface and internal longitudinal ridges. The nodule was surrounded by a fibrous wall with inflammatory reaction composed of histiocytes and multinucleated giant cells. The patient had an uneventful postoperative course and did not require further medical treatment.

In Europe, human pulmonary dirofilariasis caused by the canine heartworm *D. immitis* is rare. Extra-pulmonary infection has also been reported.² Humans become infected through mosquito bites but infection is not transmitted either from person-to-mosquito-to-person or from person-to-person, because humans are the "dead-end" hosts.³

Differential diagnosis of a pulmonary coin lesion between malignant tumor and benign lesion is always a diagnostic challenge. Existing serologic assays lack sensitivity and specificity, and should be used to supplement other data.⁵ Histologic identification of the

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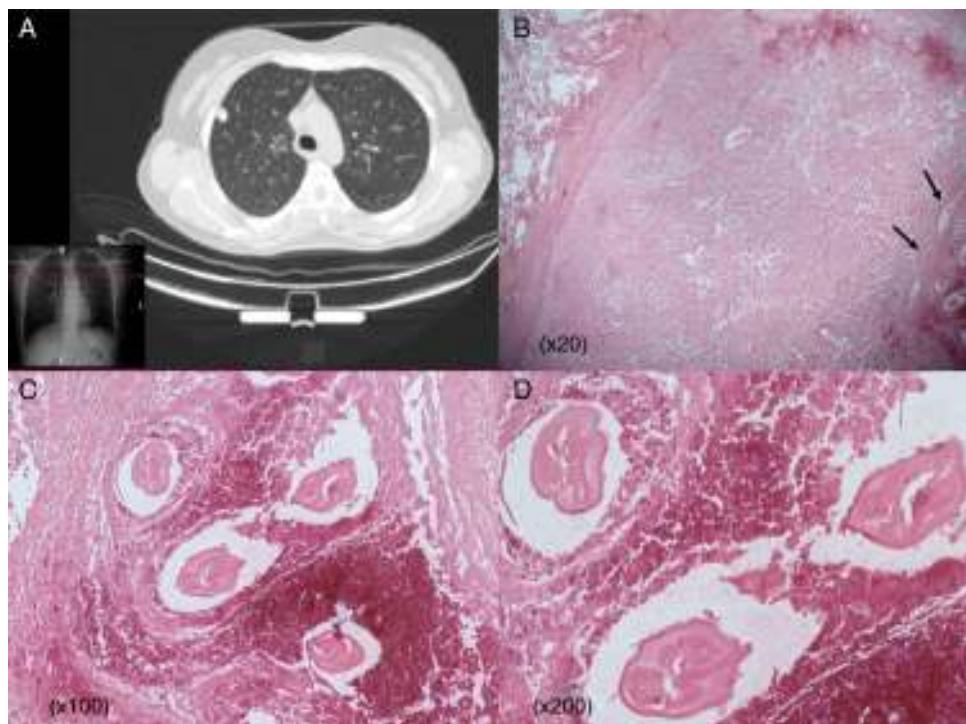


Fig. 1. (A) Chest CT scan showing an apparently malignant solitary nodule in the right upper lobe abutting the pleura. (B) Well-defined necrotic pulmonary nodule invading normal lung parenchyma. Worms in necrotic tissue (arrows) (20×). (C and D) Typical dirofilariasis worms (*D. immitis*) embedded in necrotic material (C: hematoxylin and eosin staining 100×; D: hematoxylin and eosin staining 200×).

nematode through surgical biopsy is still considered the best diagnostic approach, despite the high iatrogenic risk. Since humans are the “dead-end hosts”, definitive therapy is surgical resection, and further medical treatment is not required.³

Dirofilaria is endemic in the Mediterranean region, but the distribution of the disease is changing due to increasing environmental temperatures.⁴ However, knowledge regarding incidence is limited by the lack of registry and data are derived only from isolated reported cases. In Europe, fewer than 40 cases of pulmonary dirofilariasis have been reported, 3 of which were in Greece (Fig. 1).³

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