Journal of Investigative Medicine High Impact Case Reports
Volume 10: 1–4

DOI: 10.1177/23247096211063337
journals.sagepub.com/home/hic
SAGE

Chronic Eosinophilic Pneumonia Masquerading as a Lung Mass

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Abstract

Lung masses are becoming more common, and although most are tumors, benign or malignant, some are not solid masses. Many pathologies can present as lung nodules, including lung cancers, hamartomas, lung abscesses, granulomas, and eosinophilic pneumonia, to name a few. A 40-year-old woman with a long history of smoking presented with cough and left-sided chest pain. After multiple imaging studies, she was thought to have a lung malignancy; however, multiple biopsies proved this was not the case. The histology reports of 3 to 4 biopsies at separate times indicated chronic inflammation ongoing in the lungs without any cancer cells present. She was treated for chronic eosinophilic pneumonia with a resolution of symptoms. The purpose of this case report is to discuss a case that was initially thought to be a lung mass but found to be chronic eosinophilic pneumonia manifesting as a lung mass.

Keywords

pathology, pulmonary critical care, radiology/imaging, lung, mass, eosinophilic, chronic, pneumonia

Introduction

The presence of a lung mass in adults usually raises concerns that an underlying malignancy is present. However, keeping in mind such a worse prognosis, other differential diagnoses must be ruled out first.

The size of a lung nodule is proportionately related to the risk of lung cancer, as discussed by Wahidi.[1] In addition, monitoring nodule/mass growth in 30 days is usually a good indicator of the possibility of malignancy.[2] The presence of risk factors such as diet, tobacco, and alcohol; exposure to other pollutants; and chronic lung conditions are additional factors to consider when evaluating lung cancer.

Since the 1920s, the medical society linked smoking to cancer; however, secondary to World War II, the efforts were distracted till later.[3] It was not until the 1950s where multiple studies showed a link between smoking as a risk factor and lung cancer.[4,5] Interestingly, some studies have linked asthma and a higher risk of developing lung cancer. In a meta-analysis done by Qu,[6] there was a statistically significant relationship between asthma and the risk of developing lung cancer with high power.[7]

We present a patient with multiple risk factors for developing lung malignancy who presented with a lung mass.

Case Presentation

A 40-year-old woman with a medical history of asthma and 11-pack-year smoking history presented to our emergency department with a cough of 1-day duration. She worked as a transit worker in the state and had regular exposure to dust but she denied any recent travel out of the state in the last year prior to her presentation. The patient's cough was associated with fever and left-sided chest pain that worsened with breathing. At the time of presentation, she was hemodynamically stable. Initial labs were significant for a white blood cell count of 13.1 × 109/L, D-dimer of 669 µg/mL, and alkaline phosphatase of 155 IU/L. The patient underwent a chest x-ray (CXR) that revealed a new left basilar infiltrate versus atelectasis, associated with a slight left pleural effusion and an increase in left perihilar opacity. The patient subsequently underwent a chest computed tomography (CT) angiogram showing a suprahilar mass measuring roughly 5 cm with peripheral consolidation and an infrahilar mass measuring 10 × 5 cm with peripheral atelectasis. There was also extensive mediastinal, suprahilar, hilar, and infrahilar adenopathy on the left with likely malignant effusion and peripheral consolidation highly suggestive of a malignant process.

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Received September 2, 2021. Revised November 8, 2021. Accepted November 10, 2021.

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The patient was treated with levofloxacin for postobstructive pneumonia. Among the initial differentials were small cell lung cancer, non-small cell lung cancer, lymphoma, pulmonary tuberculosis, and sarcoidosis. The patient underwent a CT-guided biopsy of the left lower lobe mass to rule out malignancy, which came back negative. In addition, cytology sent from a pleural aspirate revealed no malignant cells. Workup for metastasis was done via CT abdomen/pelvis and magnetic resonance image (MRI) abdomen, revealing subtle 10 mm hypodense right hepatic lobe lesion. At the same time, a bone scan was negative for metastatic lesions. An MRI brain was also negative for any metastasis. In addition, the patient had a negative TB Quantiferon test and her ACE level was within the normal range.

Given the increased suspicion for malignancy, the patient underwent bronchoscopy, demonstrating a necrotic endobronchial lesion in the left lower lobe and lingula. Biopsies and bronchoalveolar lavage (BAL) fluid were collected and were negative for malignancy and showed benign mesothelial cells, histiocytes, and acute inflammatory cells. The patient underwent a repeat bronchoscopy and biopsy by cardiothoracic surgery, which also returned negative for malignancy. However, it showed benign bronchial columnar cells, macrophages, and associated markedly severe acute and subacute inflammation with partial cell degeneration. She was scheduled for video-assisted thoracoscopic surgery (VATS) and biopsy. However, preoperative imaging showed a decrease in mass burden, making the procedure no longer necessary. The patient had a repeat CT-guided biopsy of the left upper lobe, which showed benign tissue with chronic inflammation, mild fibrous changes, intra-alveolar accumulation of mucin, no granuloma seen, negative for acid-fast bacilli (AFB), and negative for fungi. The specimen was sent to an outside, more specialized facility for confirmation of the histology findings. The repeat histology confirmed the previous results with the summary that the biopsy showed cellular interstitial chronic inflammation with alveolar mucus extravasation, bronchiolitis with prominent eosinophils, and Charcot Leyden crystals all suggestive of an eosinophilic syndrome such as allergic bronchopulmonary aspergillosis (ABPA), with differentials including chronic eosinophilic pneumonia, collagen vascular disease, hyper-eosinophilic syndrome drug reaction, and infection particularly parasitic.

Six months later, the patient presented at a different hospital facility where she had a repeat bronchoscopy with biopsy and extensive workup, which all came back negative. Her workup included Quantiferon gold, hypersensitivity screen, *Aspergillus* immunoglobulin G (IgG), rheumatology serologies (antinuclear antibodies [ANA], rheumatoid factor [RF]/cyclic citrullinated peptide [CCP], SSA/B, anti-neutrophil cytoplasmic antibody [ANCA]), fungal workup (*Aspergillus* galactomannan, *Cryptococcus*, blastomycosis), asthma profile (with *Aspergillus* immunoglobulin E [IgE] and total IgE), stool ova and parasites, which were all negative. Further parasitic workup was not done because the serum eosinophils were within normal limits.

Bronchoscopy done at a second facility revealed a left upper lobe mass completely obstructing airway abnormality in the anterior medial segment of the left lower lobe (B7 & B8). Transbronchial lung biopsies, BAL, and cryotherapy were performed. The results revealed mucus with marked acute inflammation with many eosinophils and necrosis, respiratory tissue with inflammation, and blood clot. The BAL was positive for 28% of eosinophils.

The patient later returned to our hospital with persistent fever, chest pain, and cough 2 days after the bronchoscopy at the outside facility. The CT chest revealed similar findings of a left lung mass with possible small lung abscesses. Her CXR showed an opacified left lung. Treatment commenced with cefepime, metronidazole, and vancomycin for health care–associated pneumonia versus postobstructive pneumonia. Our infectious disease team recommended a repeat bronchoscopy with biopsy; however, the patient refused. She later left against medical advice, discharged on antibiotics and prednisone taper.

Approximately 7 months following her initial presentation, the patient reported subjective improvement at our outpatient pulmonary clinic visit. She had a repeat CXR that showed near resolution of the lung mass, consolidation, and pleural effusion.

Discussion

The presence of nodule/mass in the lung usually raises suspicion for malignancy and is considered until proven otherwise. However, the presence of a vast differential diagnosis prompts adequate testing to ensure reaching an accurate diagnosis. In this case, our differentials were limited to eosinophilic pneumonia, fungal infections, and of course, lung cancer.

Usually, a mass around airways will lead to common local complications such as obstruction, leading to infection. As previously mentioned, this is how our patient presented and was found to have a lung mass.

Eosinophilic lung diseases include a heterogeneous group of disorders characterized by peripheral eosinophilia, eosinophilic infiltration of lung parenchyma as evidenced by biopsy or increased eosinophils in BAL fluid. Further categorization divides eosinophilic lung diseases into primary and secondary disorders based on identifiable etiology. In addition, primary conditions are either systemic or lung-limited. Idiopathic acute eosinophilic pneumonia (AEP), idiopathic chronic eosinophilic pneumonia (CEP), and simple pulmonary eosinophilia form part of the lung-limited disorders.[8] It is important to note that idiopathic eosinophilic lung diseases are usually a diagnosis of exclusion. Other risk factors should be excluded, including drugs, immune disorders, myeloid and lymphoid neoplasms, and, more interestingly, new onset of smoking or flavored cigars.[9] Idiopathic CEP is due to infiltration of pulmonary parenchyma by eosinophils

resulting in symptoms of dyspnea, cough, a prolonged course of febrile illness, and hypoxemia of varying severity. It usually presents with central sparing on CXR and may manifest as a reaction to certain drugs. Idiopathic CEP was first reported in 1969 when Carrington and colleagues presented 9 women with dyspnea, fevers, and weight loss. Further workup revealed peripheral opacities on CXRs and eosinophilic airspace infiltration on lung biopsy specimens. These patients had a favorable clinical outcome after initiating corticosteroid therapy.[10]

Among the common causes of lung masses, particularly chronic asthmatic patients, is developing ABPA.[1] On repeat diagnostic testing, our patient's CT-guided biopsy sample was sent to a specialized facility to compare the accuracy of our lab testing. Both samples revealed similar findings—cellular interstitial chronic inflammation with alveolar mucus extravasation, bronchiolitis with prominent eosinophils, and Charcot Leyden crystals all suggestive of an eosinophilic syndrome such as allergic bronchopulmonary aspergillosis, with differentials including chronic eosinophilic pneumonia and collagen vascular disease. However, studies did not meet the diagnostic criteria for ABPA. In addition, autoimmune serology, including ANA, was negative.

Occasionally, patients with underlying lung malignancies may initially present with a lung abscess. Such presentation occurs when malignancy causes postobstructive pneumonia. In other instances, few malignancies may appear as lung abscesses, for example, excavating bronchial carcinoma. Hendricks demonstrated 2 cases where patients initially thought to have lung abscesses were later diagnosed with lung cancer.[5,11] The high suspicion of eosinophilic pneumonia, yet not diagnosed, led us to think more about malignancy or a premalignant state. As supported by Travis, eosinophilic pneumonia is sometimes associated with some malignancies, mostly lung mucinous adenocarcinoma 0.99.[9,12]

Given multiple negative bronchoscopies, treating the patient's current problem, infection, was reasonable. Further surveillance and the decision of whether genetic testing is of importance needs to be determined. Multiple genetic mutations have been outlined in the past years to their association with lung cancer. In 2004, it was found that a small exposure to tobacco and the carriers of a locus on chromosome 6q23-25 led to increased susceptibility for lung cancer. In addition, autocrine growth factors, epigenetic gene inactivation, and tyrosine kinase inhibitors might be linked to lung cancers and carry a broad scope of the search in genetic analysis to use in patients who could be misdiagnosed or missed for lung cancer during their early stages.

Conclusion

The differential diagnoses in a patient presenting with possible lung mass are vast. However, differentials can be narrowed according to a patient's history and presenting illness. Our patient has a moderate smoking history, with asthma, lives at a shelter, who presented with a cough of 1-day duration, associated with fever and left-sided chest pain. With such symptomatology, the list includes primary pneumonia, lung abscess, tuberculosis (TB), or ABPA. Malignancy was highly considered, given the extensive adenopathy on imaging with associated pleural effusion. Fluorodeoxyglucose (FDG)-positron emission tomography (PET) was positive, which can be the case in malignancies and lung abscesses. However, despite extensive workup and repeated bronchoscopies with biopsies, findings were negative for malignancy, TB, and ABPA, as well as autoimmune disorders and sarcoidosis. Surgical resection would have been the best modality for diagnosis in our patient. However, her lung mass was found to be reduced during an extensive workup, and VATS was canceled. She was ultimately diagnosed with and treated for lung abscess following the second hospitalization to our facility. Following her second hospitalization, her follow-up CXR revealed near resolution of her lung mass, raising the suspicion for eosinophilic pneumonia. However, it is important to continue following the patient for resolution or progression of her mass.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical Approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed Consent

Verbal informed consent was obtained from the patient for her anonymized information to be published in this article.

References

1. Wahidi MM, Govert JM, Goudar RK, et al. Evidence for the treatment of patients with pulmonary nodules: when is it lung cancer? ACCP evidence-based clinical practice guidelines. 2nd ed. *Chest*. 2007;132(3 suppl):94S-107S.
2. Yao D, Zhang L, Wu PL, et al. Clinical and misdiagnosed analysis of primary pulmonary lymphoma: a retrospective study. *BMC Cancer*. 2018;18(1):1-7. doi:10.1186/s12885-018-4184-1.
3. Pahal P, Penmetsa GK, Modi P, Sharma S. *Eosinophilic Pneumonia*. https://www.ncbi.nlm.nih.gov/books/NBK537169/. Published May 2021. Accessed July 2, 2021.
4. Malhotra J, Malvezzi M, Negri E, Vecchia CL, Boffetta P. Risk factors for lung cancer worldwide. *Eur Respir J*. 2016;48:889-902. doi:10.1183/13993003.00359-2016.
5. Hendriks LE, Hochstenbag MM, Lalji UC, Dingemans AMC. A pulmonary abscess, beware of lung cancer! *Respir Med CME*. 2011;4(4):157-159. doi:10.1016/j.rmedc.05.001.
6. Qu YL, Liu J, Zhang LX, et al. Asthma and the risk of lung cancer: a meta-analysis. *Oncotarget*. 2017;8(7).
7. Müller FH. Tobacco abuse and lung cancer. *Z. Krebs-forsch*. 1940;49(1):57-85. doi:10.1007/BF01633114.

8. Sisodia J, Bajaj S. *Allergic bronchopulmonary aspergillosis*. https://www.ncbi.nlm.nih.gov/books/NBK542329/. Published March 2021. Accessed July 2, 2021.
9. De Giacomi F, Vassallo E, Yi ES, Ryu JH. Acute eosinophilic pneumonia. Causes, diagnosis, and management. *Am J Respir Crit Care Med*. 2018;197(6):728-736. doi:10.1164/rccm.201710-1967CI.
10. Travis WD, Brambilla E, Noguchi M, et al. International association for the study of lung cancer/American Thoracic Society/European Respiratory Society international multidisciplinary classification of lung adenocarcinoma. *J Thorac Oncol*. 2011;6(2):244-285. doi:10.1097/JTO.0b013e318206a221.
11. Wynder EL, Graham EA. Tobacco smoking as a possible etiologic factor in bronchiogenic carcinoma: a study of six hundred and eighty-four proved cases. *J Am Med Assoc*. 1950;143(4):329-336. doi:10.1001/jama.1950.02910390001001.
12. Yankelevitz DF, Gupta R, Zhao B, Henschke CI. Small pulmonary nodules: evaluation with repeat CT—preliminary experience. *Radiology*. 1999;212(2):561-566. doi:10.1148/radiology.212.2.r99au33561.