Clinical vignette: Mycoplasma pneumonia: A novel risk factor for thromboemboli?

Mary Seiler
Jennifer Jernigan

Follow this and additional works at: https://digitalrepository.unm.edu/hostpitalmed_pubs

Recommended Citation
https://digitalrepository.unm.edu/hostpitalmed_pubs/42

This Presentation is brought to you for free and open access by the Internal Medicine at UNM Digital Repository. It has been accepted for inclusion in Hospital Medicine by an authorized administrator of UNM Digital Repository. For more information, please contact disc@unm.edu.
**Mycoplasma pneumonia: A novel risk factor for thromboemboli?**

Mary Seiler, MD; Jennifer Jernigan, MD
Albuquerque, NM

**Introduction**

Pulmonary emboli are serious, life-threatening events and their diagnosis can be complicated by the presence of other significant pulmonary pathology.

When patients do not respond to initial medical treatment as expected it is always important to reevaluate and reconsider other possible etiologies of complaints (avoiding anchoring bias and premature closure). With pulmonary complaints, it is pertinent to keep in mind that the presence of a pulmonary infection does not exclude a thrombotic event.

**Case Description**

**History**

A 54 yo male with a history of tobacco use presented to the ED with one week of malaise, nonproductive cough and two days of shortness of breath, pleuritic chest pain and headaches. He denied any other past medical history and had not been smoking since he became ill.

**Pertinent Physical Exam Findings**

T: 37.6, HR: 92, BP 126/77, RR: 24, O2sat: 92% on 15 L via nonrebreather

His initial physical exam was unremarkable, including a clear lung exam

**Pertinent Laboratory Findings**

WBC 13.6 with 83% neutrophils and a left shift

Total protein 8.5 (6.1-8.2 g/dL), albumin 2.7 (3.4-4.7 g/dL), AST 108 (6-58 units/L), ALT 152 (14-67 units/L), Alkaline phosphatase 176 (38-150 units/L), total bilirubin 1.5 (0.3-1.2 mg/dL), direct bilirubin 0.5 (0.1-0.4 mg/dL)

CK 563 (37-242 units/L), Troponin normal x 2

Influenza PCR was negative

**Imaging**

Chest xray showed a left lower lobe bronchial infiltrate consistent with a pneumonia vs aspiration

**Hospital Course**

- Patient was admitted to the hospital and started on community acquired pneumonia treatment with azithromycin and ceftriaxone.
- On hospital day 4 he had no improvement so a CT-Chest without contrast was performed which showed extensive inflammatory bronchiolitis that favored a viral or atypical pneumonia, thus more serologies including mycoplasma pneumoniae, coccidioidomyocosis and histoplasmosis were ordered
- On hospital day 7 he continued to have profound hypoxia and a CT-Chest Angiogram was performed which revealed multiple segmental pulmonary emboli in his right lower lobe.
- He was switched from prophylactic anticoagulation to therapeutic anticoagulation and the next day his mycoplasma IgG and IgM both came back positive
- He was treated with 14 days of azithromycin and discharged on oxygen and a three month prednisone taper as recommended by Pulmonology

**Discussion**

Pulmonary emboli are a serious life-threatening diagnosis that physicians never want to miss. Due to the common and non-specific nature of symptoms associated with pulmonary emboli, risk stratification tools have been developed and widely implemented to improve diagnostic accuracy while limiting unnecessary imaging. As our knowledge of molecular medicine increases the question arises of previously unknown risk factors which are not included in traditional risk calculators.

Mycoplasma pneumoniae infections have been linked to thromboemboli in children (a normally very low risk population) in several case studies over the years. In all the reported cases children were otherwise healthy admitted with respiratory distress and found to have IgG and IgM antibodies to mycoplasma pneumoniae as well as a documented thromboembolism in the form of either a DVT, pulmonary embolism, cardiac thrombus or cerebral venous thrombosis. In 5 of the 6 cases children had positive prothrombic markers (such as antiphospholipid antibodies) during the acute phase that resolved with treatment of infection. In the 6th case the child was found to have a familial antithrombin deficiency.

Another case study chronicled the clinical course of a hypertensive 28 year old male found to have pulmonary emboli and serologies positive for mycoplasma pneumoniae. In his case he had positive lupus anticoagulant and anticardiolipin antibodies but there was not mention of follow-up antibodies tests returning to normal.

In our patient’s case he did not have other risk factors for thromboemboli and using traditional methods of risk stratification, such as Well’s criteria, he would have been low probability. This case demonstrates the importance of reevaluating diagnoses when a patient is not responding to treatment as expected. It also demonstrates that there are common conditions associated with the development of prothrombotic factors which are not accounted for in current risk stratification models.

**Conclusion**

*Risk stratification tools for pulmonary emboli should be used but providers should consider whether there is a prothrombotic condition that is not accounted for in the model.

*More investigations into infections as risk factors for thromboemboli are needed.

*Physicians should be aware of the association of mycoplasma pneumoniae infection with thromboembolism and have a low threshold for further evaluation when a patient is not improving as expected.

**References**