An Atypically Atypical Pneumonia: Breaking Down Cognitive Biases of Breathing

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Introduction

A 25 y/o male patient presents to the ED in November 2017 with a chief complaint of dyspnea and cough. He was previously seen by his primary care physician five days ago, who prescribed him a Z-pak for bacterial pneumonia. Over the past five days his symptoms of lethargy, dyspnea, and lingering cough continued to progress in part, he believes, due to his labor-intensive occupation. Deep breathing exacerbated his shortness of breath, with no alleviation of symptoms. Patient found no relief from Z-pak, of which he was compliant. Severity of discomfort was rated as moderate with worsening progression over time. Respiratory review of symptoms positive for cough and shortness of breath, GI ROS was negative. Patient has no pertinent family history or surgical history, denies smoking, and reports rare alcohol use. Past medical history indicates anxiety, chronic abdominal pain, encephalitis, meningitis due to adenovirus, and multiple bouts of pneumonia. After HPI, physical exam, lab work, and imaging, the ED also diagnosed the patient with atypical pneumonia due to an infectious organism and planned on releasing him later that day contingent on improvement of a mid-80% SPO_2 from administering stronger antibiotics and oxygen. However, his SPO_2 did not improve, and the staff did not know why. Our previously healthy patient was discharged from cardiology weeks later with cardiomyopathy. This poster will attempt to discuss this case within the context of EM management, and show the importance of cognitive debiasing in EM.

Case Description

HPI from ED Intake:

- CC: "shortness of breath and cough"
- Productive cough started five days ago
- Went to PCP and was started on Z-pak (Azithromycin)
- No relief of symptoms. More difficult to breathe secondary to fluid in lungs
- Gradual onset, with worsening progression. Moderate severity
- ROS: Respiratory (+ cough, SOB); GI (- pertinent findings)
- Context: URI

Physical Exam:

- Head: Normocephalic and atraumatic
- Eyes: Conjunctivae and EOM are normal. Pupils PERRL
- Neck: Normal range of motion. Neck supple
- Cardiovascular: Normal rate and rhythm
- Pulmonary/Chest: Decreased breath sounds in the right lower field and the left lower field
- Abdominal: Soft. Normal appearance. No tenderness
- MSK: Thoracic and lumbar back both normal
- Neurological: AOx3/3, GCS 15
- Psych: Normal mood and affect, judgement normal
- BP: 94/57, Pulse: 101, Temp: 98.5°F

Assessment and Plan:

- CBC, CMP, and Chest X-Ray
- Differential Dx: PNA (pneumonia)/URI (upper resp. infection)/Asthma

Given the differential of PNA and URI, the patient was started on ceftriaxone and doxycycline. Use of nebulizer in ED improved breathing, but X-Ray was "impressive in the setting of being on antibiotics." CXR showed "L PNA." The patient had multiple visits for PNA over the years for unknown reasons. Upon later re-evaluation, wheezing was improved at lung bases with 2L of 94% O_2, but despite five days of Z-pak from PCP and antibiotics from the ED he felt worse. After several hours of observation, the patient was admitted with the diagnosis of "PNA of both lungs due to infectious organism," and transferred to IM.

Initial CBC and BMP Lab Results

Patient CBC

Component	Value	Reference Range
WBC	8.8	4.0-10.0 x1000/µL
RBC	4.8	3.8-5.9 M/µL
Hemoglobin	13.4	12.0-18.0 g/dL
Hematocrit	40.8	37.0-52.0 %
MCV	84.8	78.0-94.0 fL
MCHC	32.8	31.0-36.0 g/dL
Platelets	275	140-440 x1000/µL
ANC	6.0	1.0-11.0 x1000/µL
Neutrophils	68.1	37.0-84.0 %
Lymphocytes	21.2	8.0-49.0 %
Monocytes	7.5	4.0-15.0 %
Eosinophils	2.6	0.0-7.0 %
Basophils	0.3	0.0-4.0 %

Patient BMP

Component	Value	Reference Range
Sodium	141	135-145 mmol/L
Potassium	4.5	3.3-5.0 mmol/L
Chloride	105	96-106 mmol/L
CO_2	21	22-30 mmol/L
Anion Gap	15	>7-<17
Glucose	99	70-100 mg/dL
BUN	14	8-18 mg/dL
Creatinine	1.10	0.50-1.20mg/dL
Calcium	9.5	8.8-10.2 mg/dL
BUN/Creatinine	12.7	10.0-20.0

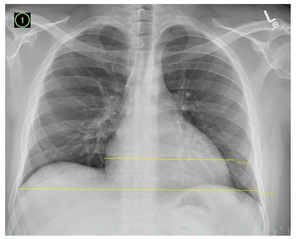
Tables 1 and 2. The patient's CBC (**Table 1**) and BMP (**2**) from the ER returned within normal limits across the board, only showing a slight decrease in CO_2 levels from reference values.

Additional Lab Test Results

Tests	Value	Notes
Procalcitonin	0.04	<0.1 Bacterial infection highly unlikely*
proBNP	2147.0	<300 pg/mL
Adenovirus by PCR	-	Real time Taqman PCR. Nasopharynx
Influenza A RT-PCR	-	Real time Taqman PCR. Nasopharynx
Influenza B RT-PCR	-	Real time Taqman PCR. Nasopharynx
PCR Human Metapneumovirus	-	Real time Taqman PCR. Nasopharynx
Parainfluenza PCR	-	Real time Taqman PCR. Nasopharynx
Respiratory Syncytial Virus PCR	-	Real time Taqman PCR. Nasopharynx
Rhinovirus PCR	-	Real time Taqman PCR. Nasopharynx
Legionella UA	-	+ indicates pleural effusion, EtOH, travel
S. pneumoniae UA	-	+ indicates above plus liver disease, leukopenia
Lower Respiratory Sputum Culture	>25 epi. cells	Gram stain shows abundant epithelial cells consistent with saliva

Table 3. Further lab workup after normal CBC and BMP panels. While procalcitonin (PCT) levels are low, it is noted that falsely low PCT can be a result of parapneumonic effusion or early phase of infection. proBNP is markedly elevated. For a proBNP of less than 300 pg/mL, acute CHF is an unlikely cause of dyspnea.[1] Here, proBNP is well over 300, indicating a potential etiology of CHF.[1] All viral and bacterial blood and urinary cultures returned negative.

Follow-Up PA and Lateral Chest X-Ray



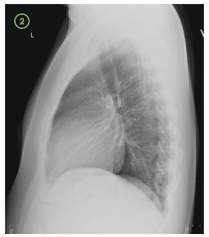


Figure 1 and 2. Follow-up PA (**Figure 1**) and Lateral (**2**) chest X-Ray taken two months after ED visit. In comparison to X-Rays taken in ED, reviewer reports "complete resolution of previously noted perihilar haziness. There is no congestive failure or pneumonia seen. There is no cardiomegaly. The hilar and mediastinal contours are unchanged from prior. There is no pleural effusion." However, enlarged cardiothoracic (CT) ratio (0.54), 3rd mogul sign (left atrial enlargement), prominence of left atrium, left ventricular predominance, and lack of retrocardiac space in lateral X-Ray are all still clearly visible two months after ED visit.

Methods/Procedure

Given negative cultures and subsequent lack of improvement from antibiotics, focus was shifted from bacterial to viral etiology. 2L of 94% O_2 was given in attempt to resolve SOB and improve SPO_2. However after observation and management, only marginal improvement was seen in the patient, and X-Ray showed:

> "bilateral patchy opacities concerning for infectious process… left greater than right. Given the patient's history, this finding is concerning for atypical/viral pneumonia. There is no large pleural effusion; a small amount of fluid is seen within the right minor fissure."

This confirmed the staff's assertations that the patient had atypical pneumonia. Yet, while looking for the positive findings for PNA, cardiomegaly (above) was missed in imaging despite elevated proBNP levels indicating possible cardiac pathology. Cognitive biases kept the medical team perplexed as to the etiology of the patient's non-improvement in symptoms. Due to this, the patient was admitted for observation and further evaluation, and care turned over from the ED staff to internal medicine. Two weeks after his ED visit, the patient was discharged with instructions to follow up with cardiology, prescriptions for furosemide, lisinopril, metoprolol succinate, and spironolactone, and a plan to implant an internal defibrillator contingent on potential improvement of LVEF (14%).

Discussion

Originally, a diagnosis of atypical pneumonia was made. This would fit the patient's dyspnea and could explain the cardiac problems, as carditis and pericarditis have resulted from *Mycoplasma pneumoniae* infections in young adults.[2,3] However, viruses are also a frequent etiologic finding in patients with community-acquired pneumonia, highlighting a possible viral cause of his symptoms upon a negative *M. pneumoniae* test result.[4] Viral pathogenesis is suspected in upwards of 30% of idiopathic dilated cardiomyopathy patients and fits with the patients progression.[5] Kearny et al. postulate that 60% of myocarditis patients have proceeding flu-like symptoms, and Quddus et al. show it is not unprecedented to have progression from a viral respiratory illness to cardiomyopathy.[6,7] However, premature closure bias likely occurred in this case because of the patient's age. PNA is caused by an infectious organism. The symptoms for an infectious organism of the lungs include dyspnea, but also fever, chills, sweating, and nausea, none of which were observed in this patient. As a result, this patient did not present with the classical symptoms of an infectious organism, but rather "dyspnea." A change in differential diagnoses was necessary to account for this chief complaint, which would include potential neurologic, MSK, renal, and cardiac etiologies. While it is uncommon to find CHF in healthy 25 y/o patients, HF can be found in a wide variety of patients, such as post-partum women.[8,9] Cognitive bias is considered to be a mistake in reasoning or cognitive process due to holding onto one's beliefs despite evidence to the contrary. Because of the cognitive biases occurring here due to the patient's age and dyspnea, other etiologies were not considered, and the patient was not treated for his true illness. Debiasing principles, such as being aware of one's biases, may have prevented this representative bias that occurred—while taking into account the dyspnea, past history, and perihilar haziness, the heuristics used by the medical staff bypassed important observations that may have led to improved patient outcomes.

Conclusion

This case illustrates that while having expectations about particular presentations are important in patient care, our biases may cloud our decision making capabilities. After the null hypothesis of the ED team was proven (negative for PNA), cognitive bias based on the patient's age and PMH still dictated the direction of care. Framing bias showed the care given to the patient was not working within the context of PNA, and further management was reactionary within this constraint. Representative bias allowed for incorrect presuppositions about the patient, a previously healthy 25 y/o with SOB and history of PNA, to be made, further clouding the complaint of "dyspnea." Without these mental heuristics and roadblocks, the medical team could have noted cardiac pathology and altered their treatment course. Cardiac involvement was confirmed by MRI five days later, showing:

> "Severely enlarged ventricular size [note: LVEDVI: 241 ml/m², severely increased >135ml/m²]. Severely depressed left ventricular systolic function. LVEF: 14%. Postcontrast images demonstrate delayed enhancement: focal mid wall region of the basal anteroseptum and mid wall segment of the basal anterior wall. This is a nonischemic pattern of enhancement and may by seen ininflammatory myocardial diseases."

Patients that present with seemingly simple presentations and diagnoses may not be as clear cut as we think. As future EM physicians, we should always be alert to the fact that even though we may believe a patient has a particular illness, we must continue to evaluate our cognitive biases and decision making processes. This is especially true in the ED, where we may fall into the habit of following representative, framing, and expectation biases and dismissing potential co-morbidities due to a number of factors like time, tiredness, and frustration.

Learning Points

- **Cognitive Debiasing**

A number of factors about this case led the medical team to the diagnosis of pneumonia. The patient history, imaging, and presentation were almost textbook, and the patient should have improved with treatment. However, because the team was set on this diagnosis, cardiomegaly was missed on the chest X-Ray. As future EM physicians, we must always examine cases with an open mind, and continually look for clues that may add to our differential. Recognizing our own cognitive biases will prevent us from using mental heuristics that thwart us from recognizing important information about our patients' illnesses. This recognition would have allowed for…

- **Earlier EKG**

As stated elsewhere, viral respiratory illnesses are known to cause cardiac issues. Even though this patient was a previously healthy 25 y/o, some EM physicians have stated their beliefs to always do an EKG in the ED for presentations like this, no matter the patient history. However…

- **Justice versus Beneficence**

Justice, the virtue/principle denoting the obligation of the physician to be fair with resources while giving patients what they are due, argues that while an EKG may have been beneficial to this patient (beneficence), would his presentation necessitate it given the resources available in the hospital?

References

1. Januzzi JL, et al. "The N-terminal Pro-BNP investigation of dyspnea in the emergency department (PRIDE) study," *American Journal of Cardiology* 2005;95(8):948-954.
2. Kenney RT, Li JS, Clyde WA, Wall TC, O'Connor CM, Campbell PT, Van Trigt P, and Corey GR. "Mycoplasmal Pericarditis: Evidence of Invasive Disease," *Clinical Infectious Diseases* 1993;17:558-562.
3. Farraj RS, McCully RB, Oh JK, and Smith TF. "*Mycoplasma*-Associated Pericarditis," *Mayo Clinic Proceedings* 1997;72:33-36.
4. Schultz JC, Hilliard, AA, Cooper LT, and Rihal CS. "Diagnosis and Treatment of Viral Myocarditis," *Mayo Clinic Proceedings* 2009;84(11):1001-1009.
5. Kuhl U, Pauschinger M, Noutsias M, Kapp JF, and Schultheiss HP. "Diagnosis and treatment of patients with virus induced inflammatory cardiomyopathy," *European Heart Journal Supplements (Supplement J)* 2002;4:173-180.
6. Kearney MT, Cotton JM, Richardson PJ, et al. "Viral myocarditis and dilated cardiomyopathy: mechanisms, manifestations, and management," *Postgraduate Medical Journal* 2001;77:4-10.
7. Quddus A, Afari ME, and Minami T. "Influenza Induced Cardiomyopathy: An Unusual Cause of Hypoxemia," *Case Reports in Cardiology*, vol. 2015, Article ID 738146, 2015; 3 pages.
8. Khaddash I, et al. "An Unusual Cause of Postpartum Heart Failure," *Annals of Cardiac Anaesthesia* 2017;20(1):102-103.
9. Hilfiker-Kleiner D, et al. "Postpartum Cardiomyopathy: A Cardiac Emergency for Gynecologists, General Practitioners Internists, Pulmonologists, and Cardiologists," *Deutsches Arzteblatt International* 2008;105(44):751-756.

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