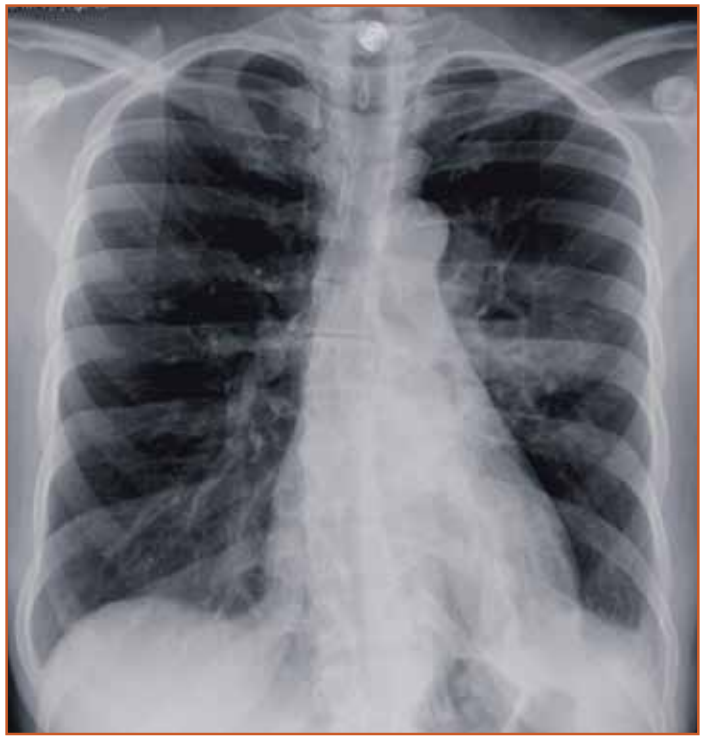

What is your diagnosis? Courtesy of the author.

Community-acquired pneumonia

The tyranny of a term

Bennett Lorber, MD, and Thomas Fekete, MD

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> What signifies knowing the name, if you know not the nature of things?
>
> —Benjamin Franklin

Picture this. It's a true story.

A twenty-nine-year-old woman enters a hospital emergency room with complaints of fever and cough. In the emergency room her fever is confirmed, she has a cursory exam and is sent for a chest X-ray that shows a left mid-lung infiltrate (see the X-ray above). A diagnosis of community-acquired pneumonia (CAP) is made, and the patient is discharged on azithromycin.

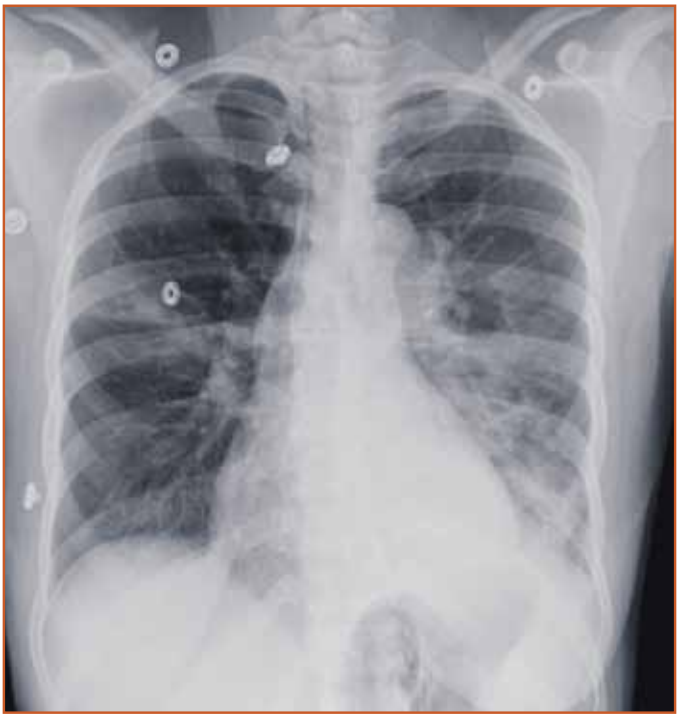
She faithfully takes the antibiotic, but after a week is no better and returns to the emergency room. This time the chest X-ray shows expansion of the left mid-lung infiltrate and a new right upper lobe infiltrate. She is admitted to the hospital. What is the admission diagnosis? It's community-acquired pneumonia. After all, she has evidence of pneumonia, and it occurred outside of a health care setting, so it is, in fact, a community-acquired pneumonia. This time she is treated with moxifloxacin, and, after four days, she is stable and discharged to complete her antibiotic course.

Two weeks later her fever and cough have not subsided so she returns to the emergency room. The infectious diseases consultation service is called to see her. They take a history! Among the information that is quickly obtained with just a few questions is the following: Her cough and fever had been present for almost two months before she made her first trip to the emergency room; she had lost nine pounds since the illness began; she had night sweats almost daily; and she resided in a recovery home for former drug users where another resident has had a bad cough for a few months. What is the diagnosis? Tuberculosis seems very likely. She is hospitalized, isolated, and a sputum sample has acid-fast bacilli and later grows *Mycobacterium tuberculosis*.

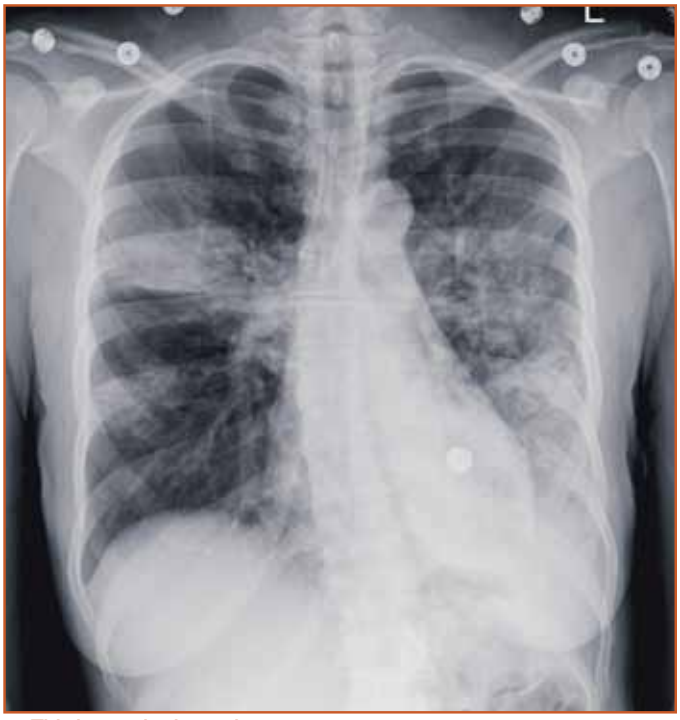
What should we make of this story? If one were to present the case of a patient with two months of cough, fever, night sweats, weight loss and a pulmonary infiltrate, any third-year medical student would consider the diagnosis of tuberculosis. Yet fully trained physicians missed the diagnostic boat on three occasions. Why? We believe it is the tyranny of the term "community-acquired pneumonia." All one needs is a cough, fever, and an abnormal chest X-ray and no further information is required. We have a diagnosis and a course of treatment. What could be simpler? Yet the hazards of this approach are obvious.

Language matters. The way we frame an issue, and the words we use to indicate it or describe it can dictate the way in which it is handled. The term CAP has eliminated thoughtful

investigation of a clinical syndrome and turned the response into a spinal reflex arc. Clearly those who developed the term CAP, and those who developed guidelines for management of this syndrome, never intended that the term should or would replace thoughtful clinical investigation; in practice, however, that seems to be what has happened. The fact that it can't be this simple is well illustrated by the fact that, not including references, the consensus guidelines for the management of community-acquired pneumonia jointly developed by the Infectious Diseases Society of American and the American Thoracic Society are more than thirty-five pages long.[1]



Second x-ray in the series. Courtesy of the author.



Third x-ray in the series. Courtesy of the author.

Most patients with fever, cough, and a pulmonary infiltrate on X-ray do have an acute infectious pneumonia that will be treated well with one of the regimens outlined in CAP guidelines. Cases of tuberculosis, endemic mycoses, and other infections that will require specific diagnostic and therapeutic approaches are uncommon, but we shouldn't miss these diagnoses when a few simple questions about duration of illness and exposures might point us in the right direction. Think of the potential health harm and the very real follow-up costs in both time and money because the tyranny of a term resulted in our patient with tuberculosis being hospitalized for four days without appropriate isolation.

The term "community-acquired pneumonia" first appeared as a published entity in the late 1970s. A Medline search showed limited references to community-acquired pneumonia in abstracts in 1978 and in titles in 1981, but the term must have been in common use since it was not formally defined. The concept of community-acquired pneumonia was used to distinguish infection in independent-living children and adults from that in nursing home patients (rather than hospitalized patients). Contrast this with 2009 when the term was used in 302 references and 172 titles that lump together patients with varying degrees of immune depression and residential situations and contrasts them to patients who acquired their pneumonia in the hospital.

Operationally, the transition from "bacterial pneumonia" and "atypical pneumonia" to "community-acquired pneumonia" appears to have been driven by two factors. First, there was an increased appreciation of the difficulty of making clear clinical distinctions among pneumonias of different etiologies. Limitations of culture and non-culture tests were more apparent when there was an imperative to begin treatment with information generated entirely by clinical evaluation and chest radiography. Second, there was a major shift from using narrow spectrum agents such as ampicillin, amoxicillin, and oral cephalosporins to macrolides and tetracyclines for the treatment of pneumonia. This trend increased further when respiratory fluoroquinolones and newer-generation macrolides were marketed in the early 1990s. The concatenation of these events made it possible to treat broadly with convenient and relatively nontoxic therapy without need to wait for diagnostic test results.

For a long time, one of the staples of house-staff training was the Gram stain. Residents were expected to be able to Gram stain sputum specimens from their patients with suspected pneumonia and, in many cases, they were tasked with recognizing common bacteria and starting pathogen-specific treatment. In the wake of the Clinical Laboratory Improvement

Amendments (CLIA) and changes in residency training, not only have the house staff labs disappeared, but also, in many hospitals, the entire microbiology venture has been outsourced. Doctors are so far removed from sputum Gram stain results that it is nearly impossible to picture treatment for pneumonia being guided by microscopy results. Cultures are slow and insensitive, so it is commonly believed that no information from stains and/or cultures would trump the combination of clinical presenting features and response to treatment. Consequently few sputum cultures are sent (even from patients with a strong suspicion of bacterial pneumonia).

The problem with this state of affairs is that we are much more assertive and certain about our diagnosis of pneumonia than is merited by the real world correlation between initial impression and final diagnosis. Our confidence lives in the distorted reality sometimes called *post hoc ergo propter hoc*. Once we conclude that a patient had pneumonia, we can go back through the record and study the choices made when the patient first presented for care. This approach has dominated the published studies of CAP. In the 2007 combined consensus guidelines for management of CAP,[1] there were sixteen Level I (randomized-controlled trial based) recommendations: six were related to validating the utility of specific antibiotics, five to influenza prevention and treatment, two to ventilator/oxygen management, and one each for development of local guidelines, obtaining blood cultures and the safety of a five-day course of treatment. None of them related to diagnostic uncertainty in the ER, diagnostic testing, or timing of antibiotics. On the other hand, retrospective studies have shown an outcome benefit for very early administration (e.g., within four hours of ER admission) of empiric antibiotics. The apparent benefit of this approach[2] led to guidelines and incentives for early treatment. In an urban U.S. hospital, comparison of pneumonia admissions in 2003 versus those in 2005 showed some telling changes: in 2005, more antibiotics were administered and were given earlier, but fewer patients given antibiotics actually had pneumonia (fifty-nine percent versus seventy-six percent).[3] Thus, very early treatment becomes, *de facto*, excessive treatment. We showed a similar result in our analysis of patients receiving very early ceftriaxone/azithromycin—a combination that seems tailored for community-acquired pneumonia.[4] Only thirty-nine percent of the patients who received this combination had a discharge diagnosis of pneumonia, although virtually all of the patients without pneumonia had respiratory symptoms resulting from congestive heart failure or chronic obstructive pulmonary disease.

Would it be better to describe pneumonias by their etiology, their clinical appearance, their pathophysiology, rather than by the current "site of acquisition?" Each of these models has potential downsides. The etiology can be hard to determine, the clinical appearance can be misleading, and we can rarely ascertain whether a pneumonia is acquired via aspiration of oral or GI tract contents versus inhalation versus arrival via bloodstream (the latter two are quite rare). Who would advocate turning back the clock to the halcyon days of the 1970s when these other terms were in wide circulation and still found wanting? So what alternatives do we have? We now have tools that may enable us to make a more specific microbiologic diagnosis within the first twenty-four hours of hospital admission. But, as one example, the use of antigen tests for pneumococcus and legionella is not universal, may not be as reliable as once thought and is probably not cost-effective. Future technological improvements, e.g., RT-PCR, may be better, but they are unlikely to be embraced unless the costs are reduced. Indirect tests such as procalcitonin might not be able to distinguish among the various bacterial etiologies of pneumonia, but might be useful to distinguish among the causes of acute respiratory symptoms (pneumonia versus congestive heart failure, for instance). Point-of-care or stat testing as is done for myocardial infarctions (troponin) and heart failure (BNP) could reduce the burden of unnecessary antibiotics. It might also allow us to unravel the skein of diseases currently managed with a combination of diuretics, antibiotics, and beta-agonists. Even if we don't have an elegant name, we should be able to avoid many of the blunders that our current system imposes.

In the meantime, we can be a little more thoughtful and, when faced with a patient having a cough and an X-ray infiltrate, ask a few more questions and perform a focused exam before reflexively applying the community-acquired pneumonia label and pulling the antibiotic trigger. This not only would have spared our patient with tuberculosis a series of unhelpful interventions and hospitalizations, but also would have reduced the chance of her spreading tuberculosis to those around her, including vulnerable hospitalized patients.

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