

Persistent Pneumonia in an Infant

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abstract

A 4-month-old boy with past medical history of eczema presented with fever and cough; a chest radiograph showed lung consolidation, and he was initially treated with amoxicillin for presumed community-acquired pneumonia. After several days, his fever persisted. He was also profoundly anemic. Antibiotic coverage was broadened because of the concern for resistant organisms; he began to improve and was discharged from the hospital. However, at 5 months of age, his fever returned, and he continued to demonstrate lung consolidation on chest radiograph. Additionally, he had lost weight and continued to be anemic. Splenic cysts were noted on abdominal ultrasound. He was diagnosed with an unusual etiology for his pneumonia and improved with the appropriate therapy. An underlying immunodeficiency was suspected, but initial testing was nondiagnostic. At 12 months of age, he presented with another infection, and the final diagnosis was made.



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Dr Padilla led the initial writing of the manuscript, recruited various specialists for writing the manuscript, revised the manuscript, and was involved in the care of the patient; Dr Logan contributed to the writing of the manuscript, critically revised all versions of the manuscript, and was involved in the care of the patient; Dr Codispoti contributed to the writing of the manuscript, revised the manuscript, and was involved in the care of the patient; Dr Jones contributed to the writing of the manuscript and was involved in the care of the patient; Dr Van Opstal led the writing of the final manuscript, revised the manuscript, and was involved in the care of the patient; and all authors approved the final manuscript as submitted.

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DR PADILLA (PEDIATRICS)

The patient was a 4-month-old African American boy born at 37 weeks' gestation with a birth weight of 2.45 kg (10th percentile) and a past medical history of moderate eczema diagnosed at 2 months of age. He initially presented to his pediatrician's office with fever and cough for 7 days. Chest radiograph showed a consolidation involving the lateral portion of the right middle lobe that was concerning for pneumonia. He was prescribed amoxicillin; however, after 7 days of therapy, he continued to be febrile with a maximum temperature of 102°F. He was referred to the emergency department for further workup. In the emergency department, his vitals included a temperature of 103°F, pulse 173 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation of 96% on room air. His weight was 5.78 kg (15th percentile) and height was 59 cm (18th percentile). On physical examination, he appeared alert and comfortable. Head, neck, and oropharyngeal examination were normal except for

conjunctival pallor. Lung examination demonstrated diminished breath sounds over the right middle lobe. Cardiovascular examination revealed a II/VI nonradiating systolic ejection murmur, strong peripheral pulses, and brisk capillary refill. His abdomen was soft and nontender, without masses or hepatosplenomegaly. His skin was pale with eczematous patches on his face and trunk.

Dr Van Opstal (General Pediatrics), What would be your differential diagnosis and next steps in management for this child?

DR VAN OPSTAL

The child may have a resistant or atypical organism causing his pneumonia. First, I would ensure he had truly received the amoxicillin. I would also be concerned for a complication of pneumonia, such as parapneumonic effusion or empyema. I would also consider pulmonary congenital malformations, such as congenital cystic adenomatoid malformation, pulmonic sequestration,

or bronchogenic cysts. Additionally, the child has a murmur. I would want to better characterize the murmur, which could be related to his fever or a clinical sign of anemia, but infectious endocarditis should be considered. Finally, the child could have an additional source of infection, such as a urinary tract infection. Regarding management, he should have blood cultures drawn, a urinalysis and urine culture, and respiratory viral studies. Testing of an acute-phase reactant may also be helpful. Although the Infectious Diseases Society of America guidelines on community-acquired pneumonia¹ do not routinely recommend complete blood count with differential, in his case, I would check one, given the physical signs of anemia and the persistent signs of infection. A repeat chest radiograph would also be appropriate. The guidelines for community-acquired pneumonia also recommend admitting children aged 3 to 6 months with concern for bacterial infection. At this time, he appears stable for the general pediatrics floor.

DR PADILLA

Complete blood count was significant for a white blood cell count of 15.7 th/ μ L (normal, 5–15 th/ μ L), hemoglobin of 6.2 g/dL (normal, 9.5–13.5 g/dL), and platelets of 308 th/ μ L (normal, 150–399 th/ μ L). Of note, his hemoglobin had been 8.5 g/dL just 1 week earlier. Additionally, his mean corpuscular volume was 73 fL (normal, 76–111 fL) and red cell distribution width was 16% (normal, 11.5%–14.5%). C-reactive protein was 163.5 mg/L (normal, 0–8 mg/L), and his chest radiograph (Fig 1) continued to show a right middle lobe opacity. He was admitted to the general pediatrics floor, and the infectious diseases service was consulted. Ceftriaxone and clindamycin were begun to treat a presumed amoxicillin-resistant pneumonia.

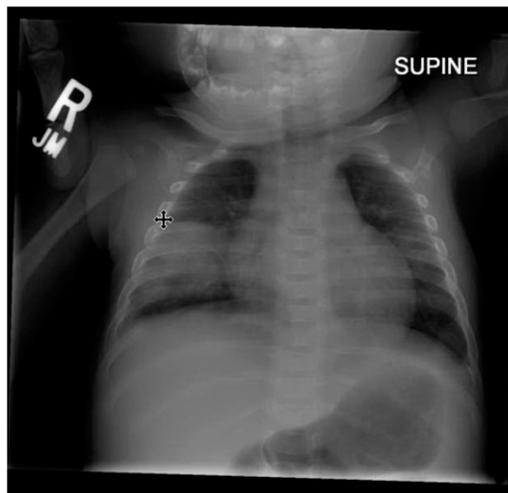


FIGURE 1

The patient's chest radiograph from his first admission demonstrated a consolidation in the lateral right middle lobe. The heart border is spared as the medial aspect of the right middle lobe is uninvolved.

Dr Logan (Infectious Diseases), what would your differential diagnosis be for this infant's pneumonia, given his failure to respond to typical antibiotic coverage? How should the clinician best evaluate for unusual etiologies of pneumonia?

DR LOGAN

In a previously healthy 4-month-old infant presenting with fever and a lobar consolidation, the first considerations are common organisms that include viral and bacterial pathogens. The lobar nature of the consolidation increases the likelihood of bacterial infection, and treatment should be directed against typical organisms such as *Streptococcus pneumoniae*, which would be the most common cause, but *Staphylococcus aureus* (including methicillin-resistant *S aureus*) and *Streptococcus pyogenes* should also be high on the differential. In the case of this infant, it is also significant to note disease progression despite correct initial therapy for community-acquired pneumonia (amoxicillin). In persistent pneumonia (especially in this age group where maternal antibodies are beginning to wane), the clinician must consider untreated

common causes of acute pneumonia, progressive disease including necrotizing pneumonia, lung abscesses, empyema, obstructive lesions, bronchiectasis, and congenital abnormalities, as well as myriad uncommon atypical pathogens in which routine antimicrobial therapy would not be effective such as *Mycobacterium tuberculosis*, *Pneumocystis jirovecii*, and several other bacteria, viruses, fungi, protozoa, and parasites. A definitive diagnosis should be sought through serologic testing, additional radiologic evaluation (such as ultrasound or computed tomography [CT] scan), and bronchoscopy for microscopy and culture.

DR PADILLA

Four days later, the patient continued to have intermittent fevers. A CT scan of the chest revealed large areas of consolidation in the lateral segment of the right middle lobe with a few smaller nodular opacities in the bilateral lower lobes (Fig 2). No findings suggestive of cavitory necrosis or empyema were seen. There was no suspicion for congenital conditions, such as bronchopulmonary sequestration or congenital cystic adenomatoid

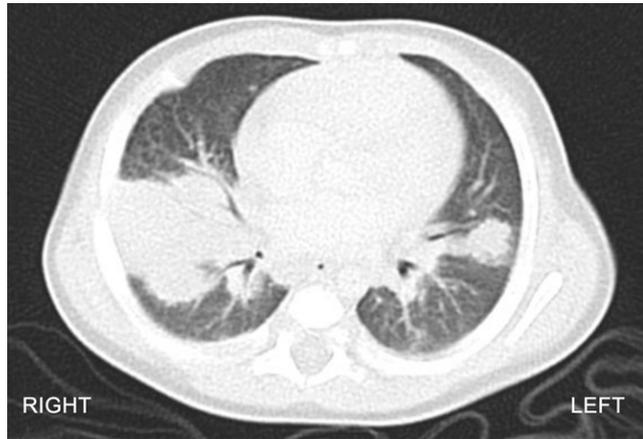


FIGURE 2

Chest radiograph revealed a large consolidation in the lateral right middle lobe and smaller areas of consolidation in the bilateral lower lobes.

formation. Clindamycin was changed to vancomycin, and azithromycin was added for atypical coverage. Over the course of the next 3 days, the patient's fever curve gradually began to improve, and blood cultures continued to show no growth. Infectious workup was negative for common respiratory viruses. Serum serology studies testing for *Chlamydia*, *Mycoplasma*, and urinary antigen testing for *Legionella* were also negative. Bronchoscopy was deferred at this time because he was clinically improving. The patient completed 5 days of azithromycin and was discharged from the hospital on 14 additional days of cefdinir and clindamycin. During his hospitalization, the patient's hemoglobin reached a trough of 5.3 g/dL with a mean corpuscular volume of 71 and red cell distribution width of 16%. Anemia workup was negative for hemoglobinopathies and glucose-6-phosphate dehydrogenase deficiency; parvovirus B19 serologies were also negative. Hematology was consulted and thought his anemia was most likely secondary to acute inflammation. At the time of discharge, he was well appearing, gaining appropriate weight, and had been afebrile for >48 hours. He was discharged from the hospital with close follow-up.

At 5 months old, the patient was noted to have lost 1 kg since hospital discharge (Fig 3), and parents reported the return of fevers. He had been followed closely as an outpatient by a nutritionist, his pediatrician, and infectious diseases. He was breastfeeding with formula supplementation and appeared to have appropriate caloric intake. On examination, he had persistent decreased breath sounds over the right middle lobe. His breathing remained nonlabored, and his oxygen saturation was normal. Hepatosplenomegaly was a new finding. He continued to have eczema and signs of anemia such as conjunctival pallor and pale skin. He was again admitted to the general pediatrics floor where infectious diseases, pulmonology, and immunology were consulted. Laboratory results demonstrated a white blood cell count of 25 th/ μ L, hemoglobin of 6.6 g/dL, and C-reactive protein of 179 mg/L. His chest radiograph revealed an unchanged right middle lobe consolidation. The patient underwent bronchoscopy, which yielded a positive smear for *P jirovecii*. He was started on intravenous (IV) trimethoprim-sulfamethoxazole (TMP/SMX) with rapid clinical improvement. An abdominal ultrasound revealed

hepatosplenomegaly with multiple hypoechoic nodular lesions in the liver and spleen thought to be consistent with disseminated *P jirovecii*.

Dr Codispoti (Immunology), when should a general pediatrician be suspicious of an immunodeficiency in a child, and what are the first steps in evaluation for possible immunodeficiency in this particular case?

DR CODISPOTI

When infections are unusual, severe, or recurrent, our suspicion rises. We need to see if there are other reasons for the severity or recurrent nature of the infections, secondary immunodeficiencies due to malnutrition, HIV, metabolic disorders (diabetes), environmental insults (radiation), inadequate antimicrobial therapy, and trauma are usually more common. A good starting point is what is recommended by the Jeffrey Modell Foundation in the Ten Warning Signs for Primary Immunodeficiency, which include failure of an infant to gain weight normally, ≥ 2 pneumonias in 1 year, and the need for IV antibiotics to clear an infection.² These signs, however, are by no means absolute. Although our patient did not have ≥ 2 pneumonias, he certainly had an unusual organism, which makes one more suspicious for an immunodeficiency.

The first step in evaluation of immunodeficiency is the complete blood count with differential. This will tell us which individual, or if multiple, cell lines (leukocytes, erythrocytes, or platelets) are affected. If the history is suggestive for deep-seated abscesses, granulomas, atypical mycobacterial infections, or severe infections, especially with opportunistic fungi and viruses, we would check markers of lymphocyte subsets (CD4, CD8, CD19, CD56). We would also

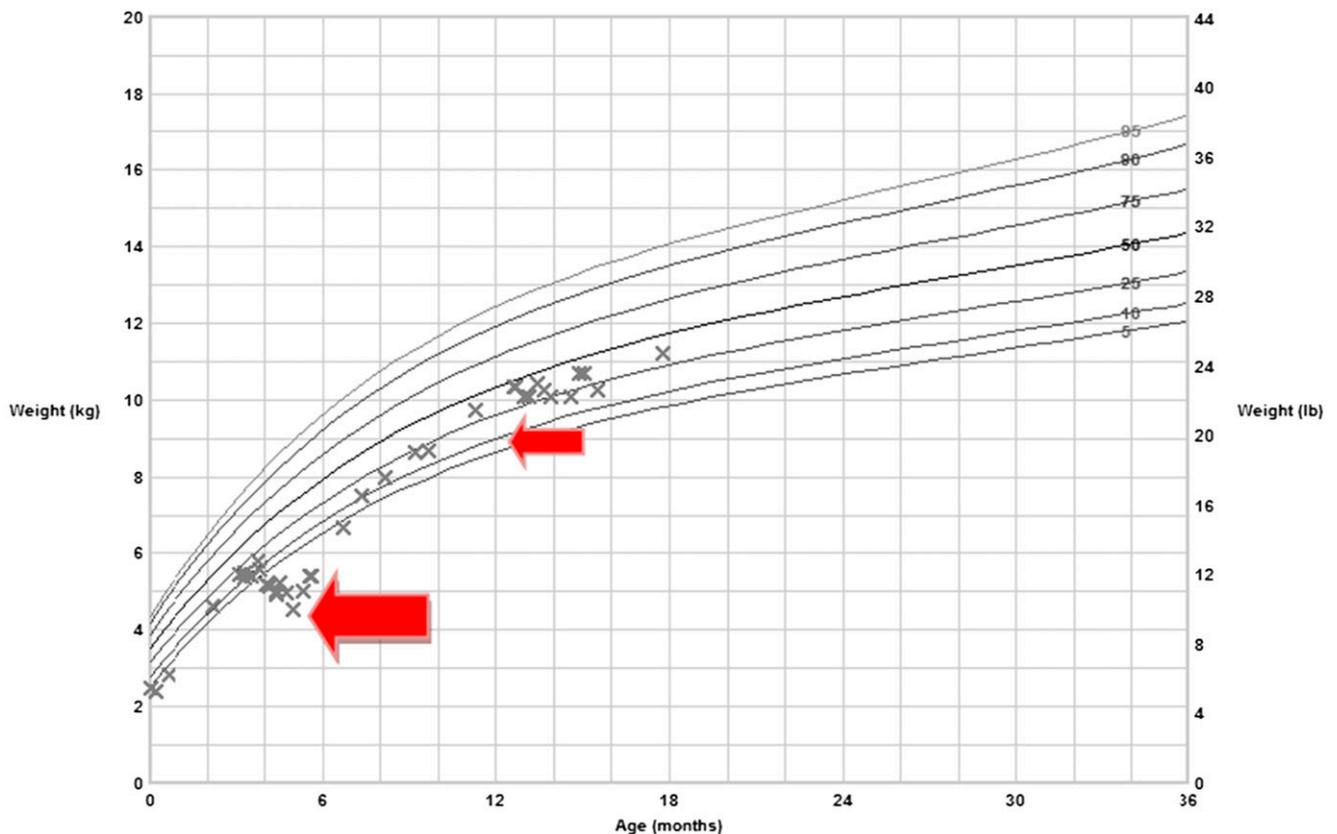


FIGURE 3

The patient's growth chart at the time of his second admission demonstrated a 1-kg weight loss over a 1-month period (larger arrow). The patient's growth chart after his treatment for PJP demonstrated healthy weight gain, and he began following the 50th percentile (smaller arrow).

check the proliferative capacity of these leukocytes after exposure to antigens (tetanus, *Candida*) or mitogens (pokeweed, phytohemagglutinin, concanavalin A). If the history is suggestive of sinopulmonary disease or chronic diarrhea, we would check quantitative immunoglobulins (immunoglobulin (Ig)M, IgG, IgA). If these Ig levels are adequate, we would check their functionality by checking antibody response to vaccines, usually both protein (tetanus, diphtheria, *Haemophilus influenzae*) and polysaccharide (23-valent pneumococcal).

DR PADILLA

Because of the patient's history and the etiology of his pneumonia, workup for an underlying immunodeficiency, specifically one affecting the T-cell lineage, was

pursued. Immunodeficiency profile (Table 1) revealed normal immunoglobulin, B-cell, and natural killer-cell levels. Absolute CD3, CD4, and CD8 counts were mildly depressed but not to the degree typical in a severe T-cell deficiency. A mitogen stimulation test showed normal T-cell activity, again arguing against a severe T-cell deficiency. HIV 1 and 2 antigen/antibody assays were negative. Fluorescence in situ hybridization probe for DiGeorge syndrome (22q11.2 deletion) was also negative.

After 7 days of IV TMP/SMX, the patient was doing well clinically and ready for discharge. As an outpatient, he completed 3 additional weeks of oral therapeutic doses of TMP/SMX before transitioning to a prophylactic dose. He rapidly gained weight and was following a growth curve just below the 50th percentile; his

hemoglobin rebounded to 10.1 g/dL. The patient underwent repeat abdominal ultrasounds to evaluate the hypoechoic structures in the spleen. They had significantly decreased by 6 months of age and were completely resolved at 11 months. Over the course of the next 7 months, he continued to be followed closely by his pediatrician, infectious diseases, and immunology. He underwent genetic testing for syndromes that would most likely explain his constellation of symptoms, including tests for a particular form of severe combined immunodeficiency called IL7R deficiency, which demonstrates a B + natural killer + T phenotype, as was suggested by his immunodeficiency profile, Hyper-IgM (CD40 ligand and CD40), hyper-IgE (STAT3/DOCK8), and Wiskott-Aldrich syndrome; all of these results were normal.

TABLE 1 The Patient's Immunodeficiency Profile

Component	Value	Reference
% Lymphocytes	19	
Absolute lymphocyte count	3705	1140–4430 CMM
% CD19, B cells	40	11%–45%
Absolute CD19 count	1482	430–3300 CMM
% CD3, T cells	54	55%–82%
Absolute CD3 count	2001	3500–5000 CMM
% CD4, T helper	45	50%–57%
Absolute CD4 count	1667	2800–3900 CMM
% CD8, T cells	9	8%–13%
Absolute CD8 count	333	350–2500 CMM
% CD56, NK cells	3	2%–18%
Absolute CD56 count	111	23–797 CMM

The patient's lymphocyte profile demonstrated depressed CD3, CD4, and CD8 subsets. Although not completely normal, these levels were not indicative of a severe T-cell deficiency. CMM, ; NK, natural killer.

The patient continued to do well until age 12 months, when he presented to his pediatrician with a red and tender cervical lymph node measuring $\sim 4 \times 4$ cm. There was no appreciable area of fluctuance, and empirical clindamycin was begun for presumed lymphadenitis. Upon repeat examination 10 days later, there was fluctuance and purulent drainage, but the lesion had decreased to 2.5×2.5 cm in size. A culture of the drainage grew TMP/SMX-resistant *Klebsiella pneumoniae*. His antibiotic was switched to amoxicillin-clavulanate, and he was sent to follow-up with infectious diseases.

Dr Logan, how does the child's presentation with lymphadenitis change the differential diagnosis for his particular underlying etiology?

DR LOGAN

In itself, having an acute lymphadenitis after being healthy and thriving for 7 months does not suggest any particular diagnosis, especially in a child with significant eczema. A more concerning issue when he presented with lymphadenitis was the organism recovered from the purulent drainage, *K pneumoniae*, an enteric Gram-negative bacteria from the Enterobacteriaceae family, which is an unusual pathogen to cause lymphadenitis and is most often seen in gastrointestinal and genitourinary

tract infections. Typical bacterial pathogens in acute lymphadenitis are *S aureus* and *S pyogenes*, common skin flora.⁴ This unusual Gram-negative bacteria (*K pneumoniae*) causing lymphadenitis in a male infant, along with a history of atypical pneumonia was highly suspicious for an undiagnosed primary immunodeficiency, in particular, chronic granulomatous disease (CDG), which was now at the top of the differential because of this new Gram-negative lymphadenitis infection in conjunction with his previous history. This disease may have been overlooked previously because other diagnoses more classically associated with *P jirovecii* were pursued first.

DR PADILLA

A neutrophil oxidation test revealed an absent neutrophil oxidative burst by dihydrorhodamine 123 fluorescence testing. A diagnosis of X-linked recessive chronic CGD was later confirmed by genetic testing. The patient is currently being treated with prophylactic doses of TMP/SMX, itraconazole, and interferon- γ to prevent bacterial and fungal infections. To date, he has required drainage of 1 additional infected lymph node but has otherwise been asymptomatic. His mother recently revealed that she is pregnant with a girl.

Dr Jones (Genetics), What is the genetic evaluation of a child with CGD? How would you recommend the mother be counseled, given her recent pregnancy?

DR JONES

There are 5 genes associated with CGD: *CYBB*, *CYA*, *NCF1*, *NCF2*, and *NCF3*. Mutation in the *CYBB* gene is the most common cause of CGD. The *CYBB* gene is located on the X chromosome. It is inherited in an X-linked recessive fashion, seen primarily in males. The *CYA*, *NCF1*, *NCF2*, and *NCF3* genes are inherited in an autosomal recessive fashion. A variety of changes have been identified in the *CYBB* gene, resulting in the CGD phenotype. Nonsense mutation or a deletion of the gene can result in a more severe disease phenotype. Missense mutations between base pairs 1 and 309 usually have milder disease with improved survival. Mutations distal of base pair 309 are more severe. The patient was identified to have a mutation of the *CYBB* gene at site 469. The risk for the family to have another affected child is dependent on whether this represented a de novo mutation in the oocyte or it was inherited from the mother. If it was inherited from the mother, there would be a 50% chance of passing the disease on to a future male child. In this case, the mother was not a carrier for the *CYBB* mutation, making it unlikely that she is a carrier (however, there are reported cases of mosaicism). In this case, the recurrence risk in a future pregnancy would be $<1\%$, but to be safe, I would still recommend genetic testing for the known mutation in any future male. There is no need to test a female because she has 2 X chromosomes and would not be symptomatic.

Summary

In summary, our patient presented with fever and cough at 4 months of

age with a presumed community-acquired pneumonia that was unresponsive to amoxicillin. He also had significant anemia, which was thought to be secondary to inflammation. The patient initially seemed to improve on an antibiotic regimen including coverage for atypical, methicillin-resistant *S aureus* and penicillin-resistant *S pneumoniae*. A CT scan of the lungs made congenital pulmonary malformations unlikely. However, despite therapy, after 1 month, he demonstrated poor growth, persistent anemia, and a continued right middle lobe consolidation. Upon bronchoscopy, he was found to have *P jirovecii* pneumonia (PJP). He also had evidence of hypoechoic splenic structures, initially presumed to be disseminated *P jirovecii*.

P jirovecii, formerly known as *Pneumocystis carinii*, is an atypical fungus that can cause pneumonia in susceptible populations.^{5,6} Serologic studies show that healthy children are infected with *P jirovecii* before age 2 years, but most infections are asymptomatic. Symptomatic children are generally immunocompromised. PJP commonly presents with nonproductive cough, tachypnea, dyspnea, fever, and hypoxia. The presentation can be acute or subacute depending on the underlying etiology. Pediatric patients who are predisposed to PJP include patients with HIV infection, underlying malignancy, primary immunodeficiency, or severe malnutrition or organ transplant recipients on immunosuppressive therapy.⁷ These underlying risk factors should be considered and investigated in any patient presenting with PJP.

Initially, no predisposing cause could be found for our patient. Congenital immunodeficiency syndromes associated with PJP are those where T-cell populations are affected. In one retrospective study of patients with PJP, associated

immunodeficiencies included Wiscott-Aldrich, severe combined immunodeficiency, hyper IgM, and 1-Stat5b deficiency. Our patient was tested for all of these, resulting in a negative workup.

At 12 months of age, the patient developed a more classic symptom of CGD, and the diagnosis was pursued. CGD is characterized by the inability of neutrophils and monocytes to eradicate catalase-positive microorganisms.^{8,9} This is generally because of a defect in respiratory burst causing the failure to generate reactive microbial oxygen metabolites and hydrogen peroxide. The majority of cases are X-linked recessive, whereas a minority are autosomal recessive. Clinical presentations of CGD are highly variable. Children can present with recurrent infections or atypical pneumonia and/or lymphadenitis and poor wound healing, as in the case of our patient. Other presentations include hepatic abscesses and/or other abscesses, osteomyelitis, and other atypical or severe infections due to bacteria or fungi. CGD patients also have inflammatory syndromes associated with granulomatous disease in the viscera that can cause mass effect and obstruction. In retrospect, the patient did have hypoechoic splenic structures that may have been granulomas associated with CGD. However, because the patient improved with therapy directed against *P jirovecii*, invasive measures to diagnose these structures were not pursued. Family history of infections can be an important part of the history. This patient did not have any family history of recurrent or unusual infections. Although the most common organisms involved are catalase-positive organisms, such as *S aureus*, *Burkholderia cepacia*, *Serratia marcescens*, *Listeria* species, *Escherichia coli*, and fungal pathogens such as *Aspergillus fumigatus*, *Candida glabrata*, and *Candida albicans*, various other

organisms have been associated with infections in CGD patients, including *P jirovecii*.⁸⁻¹⁰ Currently our patient is doing well on recommended management of CGD involving the use of interferon- γ and prophylactic antifungal agents/antibiotics. At this time, the only known cure for CGD is hematopoietic stem cell transplantation.¹¹

ABBREVIATIONS

CGD: chronic granulomatous disease
 CT: computed tomography
 Ig: immunoglobulin
 IV: intravenous
 PJP: *Pneumocystis jirovecii* pneumonia
 TMP/SMX: trimethoprim-sulfamethoxazole

REFERENCES

- Bradley JS, Byington CL, Shah SS, et al; Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. The management of community-acquired pneumonia in infants and children older than 3 months of age: clinical practice guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. *Clin Infect Dis*. 2011;53(7): e25–e76
- Jeffery Modell Foundation. Ten warning signs for primary immunodeficiency. Available at: <http://downloads.info4pi.org/pdfs/General10WarningSignsFINAL.pdf>
- Immunology deficiency search database. Available at: <http://www.immunodeficiencysearch.com/home>. 2015
- Thorell E. Cervical lymphadenitis and neck infections. In Long S, Pickering L, Prober C, eds. *Principles and Practice of Pediatric Infectious Disease*. 4th ed. New York, NY: Elsevier/Churchill Livingstone; 2012;135–147
- Gigliotti F, Wright TW. *Pneumocystis jirovecii*. In Kliegman R, Nelson WE, eds. *Nelson Textbook of Pediatrics*. 19th ed. Philadelphia, PA: Elsevier/Saunders; 2011:1069e1–1069e5

6. Gigliotti F, Wright TW. *Pneumocystis jirovecii*. In Long S, Pickering L, Prober C, eds. *Principles and Practice of Pediatric Infectious Disease*. 4th ed. New York, NY: Elsevier/Churchill; 2012:1230–1233
7. Saltzman RW1, Albin S, Russo P, Sullivan KE. Clinical conditions associated with PCP in children. *Pediatr Pulmonol*. 2012; 47:510–516
8. Buckley R. Evaluation of suspected immunodeficiency. In Kliegman R, Nelson WE, eds. *Nelson Textbook of Pediatrics*. 19th ed. Philadelphia, PA: Elsevier/Saunders; 2011; 715–719e1
9. Buescher ES. *Infectious complications of dysfunction of deficiency of polymorphonuclear and mononuclear phagocytes*. . In Long S, Pickering L, Prober C, eds. *Principles and Practice of Pediatric Infectious Disease*. 4th ed. New York, NY: Elsevier/Churchill Livingstone; 2012:619–626
10. Adinoff AD, Johnston RB Jr, Dolen J, South MA. Chronic granulomatous disease and *Pneumocystis carinii* pneumonia. *Pediatrics*. 1982;69(1): 133–134
11. Buckley R, Orange J. Primary immunodeficiency disease. Adkinson NF Jr, Busse WW, Bochner BS, et al, eds. *Middleton's Allergy: Principles of and Practice*. 8th ed. Elsevier; 2013: 1166–1168

FINDING THE RIGHT ONE: *My two eldest sons recently began looking for full-time jobs. My second son has a very specific skill set, so when he moved to a new city earlier this month, he simply showed a potential employer what he could do. My eldest son does not have such a specific skill set. He has filled out a few application forms, but so far he has not found the right job. As his father, I have a few ideas what might be best for him, but my views may not match with his – or with a prospective employer. As reported in The Wall Street Journal (Business: April 14, 2015), more employers are using specialized personality or pre-hire tests to find the best person for the job. In 2013, approximately 55% of employers used pre-hire tests to screen job applicants, compared with approximately 25% in 2001. The tests may be somewhat generic personality tests, but many are customized so that employers can screen for certain traits (such as friendliness or inquisitiveness) that may be most important to them. The result is that employers are taking their time filling open positions while waiting for the right person. From the employer’s point of view, a good match leads to longer productive employment with less rapid turnover. Data suggest that the process has had an impact. Over the past 15 years, labor market “churn,” the gap between job openings and hires, has declined by more than 25% – suggesting that more hires are staying on the job longer. For job applicants, however, the test may be yet another barrier to finding a job. The tests measure what applicants say they would do and not what they actually do. Moreover, applicants do not get feedback on what went well or not and how to improve. As for my eldest son, I do not know what type of pre-hire test he has taken. I do hope that if he does take one, the test will value critical thinking and empathy – traits in which he excels.*

Noted by WVR, MD

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