



Drug hypersensitivity causing organizing eosinophilic pneumonia in a pediatric patient



Christopher L. Jenks, MD, MS, FAAP^{a,*}, Askin Uysal, MD, FACP, FCCP^b,
Michael F. Papacostas, MD^a

^a Pediatric Critical Care Medicine, University of Texas Southwestern Medical Center at Dallas, USA

^b Pulmonology and Critical Care Medicine, Department of Anesthesia/Critical Care, Houston Methodist, USA

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ABSTRACT

Objective: To describe a relatively rare hypersensitivity reaction with pulmonary manifestations in a pediatric patient.

Data sources: Electronic medical records.

Study selection: Patient treatment in the pediatric critical care unit.

Data extraction and synthesis: Electronic medical records.

Conclusions: Eosinophilic pneumonias are rare in the pediatric population. Peripheral eosinophilia is not necessary to make the diagnosis. Bronchoalveolar lavage is the diagnostic study of choice. Lung biopsies are rarely needed to make the diagnosis. The treatment of choice is steroids. If steroids fail to improve the patient's condition, consider IVIG, and cyclosporine A.

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Case report

A 9 year old female with no significant past medical history presented to our emergency department for evaluation of dyspnea and fever. She is normally an active girl, with no smoking history, no second hand smoke exposure at home, and no history of illicit drug use. About 2 weeks prior to her presentation, she was seen by her primary care physician for an abscess on her thumb. She started a course of sulfamethoxazole/trimethoprim. Shortly thereafter, she developed a fever, cough, and a diffuse rash. She stopped taking the sulfamethoxazole/trimethoprim after 2 days due to decreased oral intake.

She returned to her primary care physician for re-evaluation, and was diagnosed with an acute otitis media (although no ear symptoms were present at the time). She was started on amoxicillin. She stopped the amoxicillin after one day due to nausea and vomiting. Her amoxicillin was then changed to azithromycin. She promptly stopped taking it due to profuse vomiting and an urticarial type rash. She then presented to an urgent care facility. Her

work up included a rapid flu and strep both of which were negative. She was given a prescription for topical hydrocortisone and oral diphenhydramine. She started to improve so her mother re-started the original sulfamethoxazole/trimethoprim. Shortly after re-starting the sulfamethoxazole/trimethoprim, she developed diffuse myalgias, dyspnea, and fever to 39.8 °C. She then went to an emergency department. Work up included another rapid flu (negative), a rapid strep (negative), WBC 3 thousand/mm³, and a chest roentgenogram (CXR) which was consistent with a "viral process." She was discharged on oral prednisone 60 mg a day. Her dyspnea worsened necessitating ambulance transport to our facility.

On presentation, she complained of cough, dyspnea, upper abdominal pain worse with deep inspiration, and anxiety to the point that she was afraid to go to sleep. She had no problems with confusion, seizures, emesis/diarrhea, chest pain, or swollen joints. There was no history of recent travel, tick bites, or sick contacts. Her blood pressure was 104/70, heart rate 120, respiratory rate 24, temperature 37.4 °C, height 140 cm, weight 31.5 kg, body mass index 16.07, pulse oximetry 97% on 2 L nasal cannula. Physical exam was significant for a clear oropharynx, coarse breath sounds which were decreased in the bases bilaterally, tachypnea and tachycardia.

She was admitted to the general pediatric floor. Her oxygen was continued. CXR showed bibasilar interstitial prominence. A

The patient was treated at Children's Medical Center of Dallas.

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* Corresponding author. 1935 Medical District Drive, Dallas, TX 75235, USA.
Tel.: +1 214 456 7591.

E-mail address: Christopher.Jenks@UTSouthwestern.edu (C.L. Jenks).

respiratory viral panel was negative (checks for influenza A subtype 1, influenza A subtype 3, influenza A subtype H1, influenza B, parainfluenza types 1–4, respiratory syncytial virus, adenovirus, metapneumovirus, coronavirus, rhinovirus, and enterovirus), WBC 14 thousand/mm³ (elevated but on steroids). The hospitalist discontinued her corticosteroids, and started her on levofloxacin for empiric coverage of an atypical pneumonia. Her condition rapidly deteriorated necessitating transfer to the pediatric intensive care unit.

A CXR revealed pneumomediastinum, pneumopericardium, and bilateral pneumothoraces. Oxygen saturations noted to be 50%. Intubation, pleural decompression followed by bilateral chest tube placement helped stabilize her condition. Oxygen saturations returned to normal. A peripherally inserted central catheter (PICC) line was placed in her left upper extremity. The following day a computerized tomography (CT) Scan of the chest with pulmonary embolism protocol revealed saddle thrombus located in the right pulmonary artery seen within the middle and lower lobe lobar, segmental, and subsegmental arteries with a probable tiny amount of thrombus within the left lower lobe segmental and subsegmental arteries, also noted to have ground glass appearance of the lower lobes bilaterally. Hypercoagulable work up included homocysteine level, protein C and S, antithrombin III, Factor II, Factor V Leiden, and Factor VIII all which were normal. Lupus anticoagulant anticardiolipin antibodies, B2 glycoprotein were all negative. Ultrasound of her left upper extremity showed an occlusive thrombus associated with the PICC line involving the axillary and basilica vein. She did not have any thrombosis in her lower extremities. She was placed on a continuous heparin drip with goals to keep her anti xa levels 0.35–0.7. Blood and urine cultures remained negative. Tests for *tularensis*, *rickettsi*, *erlichia*, *hanta virus*, *human immunodeficiency virus*, *herpes simplex virus* (HIV), *pneumocystis* were negative. Bronchoscopy with bronchoalveolar lavage (BAL) was negative for *cytomegalovirus*, *legionella*, *ebstein barr virus*, yeast, and acid fast bacilli. Her BAL showed 5% eosinophils.

An open lung wedge biopsy was negative for *mycobacterium*, *mycoplasma*, and *herpes simplex virus*. The surgical pathology returned as organizing eosinophilic pneumonia consistent with a drug hypersensitivity reaction. No evidence of infectious organisms, vasculitis, or evidence of aspiration seen. We re-started her on corticosteroids after which she showed continuous improvement over the next 4 weeks. Her pneumomediastinum and pneumothoraces resolved, and her chest tubes were removed. We discharged her on enoxaparin for her pulmonary embolism.

Discussion

The eosinophilic pneumonias encompass a wide variety of different interstitial lung disease, but all typically have eosinophilic infiltration of the lungs.¹ The exact role of the eosinophil in these disease processes are unknown, but may be related to tissue injury from inflammation and eosinophilic degranulation.¹ The eosinophilic pneumonias are generally placed into two large categories: Idiopathic and Secondary.² The idiopathic group contains eosinophilic granulomatosis with polyangiitis, idiopathic chronic eosinophilic pneumonia, and idiopathic acute eosinophilic pneumonia.² The secondary group includes parasites, drugs, toxins, allergic bronchopulmonary aspergillosis and neoplasms.²

Many drugs have an association with eosinophilic pneumonia, but not all have a proven causal relationship.³ Some of the associated drugs include the following: Antibiotics (beta lactams, tetracycline, sulfonamides, nitrofurantoin, isoniazid, ethambutol, pentamidine),

antiepileptics (carbamazepine, phenytoin), Illicit drugs (heroin, cocaine), non-steroidal anti-inflammatory drugs (ibuprofen, naproxen, indomethacin, diclofenac, piroxicam), antidepressants (amitriptyline, trazodone, venlafaxine), chemotherapeutic agents (bleomycin, camptothecin), cardiovascular drugs (angiotensin converting enzyme inhibitors, amiodarone, B-blockers), biologics (infliximab, interferon alpha, granulocyte – macrophage colony stimulating factor), and nutraceuticals (L-Tryptophan).³

Eosinophilic pneumonia is not common in the pediatric population. The mean age is typically in the late 20s. The exact incidence is unknown. There appears to be an association with HIV, smoking, and hypersensitivity to drugs or toxin exposure. Our patient seemed to have a causal relationship with Sulfamethoxazole/trimethoprim. The presentation is typically rapid over the course of 1–5 days, and generally involves fever, myalgias, pleuritic chest pain, crackles on lung exam, plus or minus peripheral eosinophilia as was the case in our patient. A CXR typically shows non-specific findings but includes consolidation, hilar adenopathy, pleural effusions, and reticulonodular densities.³ CT scans of the chest usually show ground glass opacities, nodules, or irregular lines.³ The CT chest in our patient demonstrated a ground glass appearance as well as a pulmonary embolism. Bronchoalveolar lavage is the diagnostic study of choice to diagnose an eosinophilic lung disease as it may be the only clue revealing a high eosinophil count (typically >25% when the normal in BAL fluid is <1%). Our patient had a slight elevation in her BAL eosinophils likely due to the fact that she had been partially treated with corticosteroids prior to her hospital presentation. A recent search of the available literature has revealed only a few papers that describe an association with a pneumothorax related to an eosinophilic pneumonia (in the setting of a Paragonimiasis infection).⁴ There have been very few reported cases of organizing eosinophilic pneumonia being associated with pulmonary embolism or a pneumomediastinum. Our patient's condition was associated with a pneumothorax, pneumopericardium, and pneumomediastinum. The exact etiology of the pulmonary embolism is unclear. Some possible etiologies could include the PICC line, an undiagnosed hypercoagulable disorder, or related to the inflammation of the eosinophilic disorder. Her hypercoagulable work up was negative but an undiagnosed mutation is always a possibility. Eosinophilic pneumonia has no obvious association with pulmonary embolism but still could be the possible etiology. The PICC line was present for less than 24 hours, but theoretically could have been the cause.

An interesting component of our patient is that she did not readily improve on her initial corticosteroid treatment, but did clinically respond to the corticosteroids later on in her hospital course. Corticosteroid refractory eosinophilic lung disease has been previously reported in the pediatric literature. Ronald Mortan et al reported a neonate at 2 weeks of life that had a protracted course of pneumonia, received several courses of corticosteroids with no improvement.⁵ At 8 weeks of life the patient had a lung biopsy which showed the eosinophilic pneumonia.⁵ The patient was then treated with an empiric course of intravenous immunoglobulin (IVIG).⁵ The IVIG was ineffective and a trial of cyclosporine A was instituted resulting in rapid clinical improvement.⁵

Summary

Eosinophilic pneumonias are rare in the pediatric population. Peripheral eosinophilia is not necessary to make the diagnosis. When this diagnosis is entertained, a BAL should be performed as early as possible because it is the diagnostic study of choice. Lung biopsies are rarely needed to make the diagnosis, but may be necessary if the primary diagnostic study of choice is unrevealing. The treatment of choice is corticosteroids. If corticosteroids fail to

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