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Case report

Disseminated *Nocardia cyriacigeorgia* causing pancreatitis in a haploidentical stem cell transplant recipient

Jason Chen[a], Jonathan Pan[a], Joanne Filicko-O'Hara[b], Margaret Kasner[b], Phyllis Flomenberg[c,*]

[a] Department of Medical Oncology, Sidney Kimmel Medical College of Thomas Jefferson University, 834 Chestnut St., Suite 320, Philadelphia, PA 19107, United States
[b] Department of Medical Oncology, Sidney Kimmel Medical College of Thomas Jefferson University, 925 Chestnut St., Suite 420A, Philadelphia, PA 19107, United States
[c] Division of Infectious Diseases, Department of Medicine, Sidney Kimmel Medical College of Thomas Jefferson University, 1015 Chestnut St., Suite 1020, Philadelphia, PA 19107, United States

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ABSTRACT

We report the first published case of acute pancreatitis secondary to disseminated nocardiosis in a hematopoietic stem cell transplant (HSCT) recipient on chronic immunosuppression for graft-versus-host disease (GVHD). Nocardiosis in the HSCT population is relatively rare, and has not yet been described in haploidentical HSCT recipients. Our patient is a 28-year-old male with a history of haploidentical HSCT and GVHD of the skin and lung who was admitted to the hospital with acute pancreatitis. The workup for the etiology of his pancreatitis was initially unrevealing. He subsequently developed worsening sepsis and respiratory failure despite broad spectrum antimicrobials. After multiple bronchoscopies and pancreatic fluid sampling, he was found to have disseminated nocardiosis with *Nocardia cyriacigeorgia*.

Introduction

Nocardia spp. is an aerobic gram-positive filamentous branching bacterium that is an uncommon cause of opportunistic infections in patients receiving hematopoietic stem cell transplantation (HSCT). While the incidence of nocardiosis in the allogeneic HSCT population is unclear, it is estimated around 0.3 to 1.7% according to two studies in the 1990s [1,2]. Risk factors for infection include immunosuppression for chronic GVHD and use of inhaled pentamidine for pneumocystis prophylaxis [3].

Nocardia can be found throughout the environment, including in water and soil. Due to its ability to aerosolize, it commonly causes a primary pulmonary infection, with a majority of patients presenting with fever, cough, and pleuritic chest pain [4]. However, *Nocardia* can disseminate to other organs, particularly the skin and central nervous system. Nocardial infection of the pancreas has been reported in an immunocompetent patient who had a resection and radiotherapy for bladder cancer [5]. We report the first published case of acute pancreatitis secondary to disseminated nocardiosis in a HSCT recipient on chronic immunosuppression for GVHD.

Patient presentation

The patient is a 28-year-old male who was diagnosed with acute myelogenous leukemia after presenting with fevers and chills. He underwent induction chemotherapy on clinical trial with cytarabine, idarubicin and sirolimus, and subsequent consolidation therapy with high-dose cytarabine. After achieving complete remission, he underwent a haploidentical HSCT from his mother. His conditioning regimen consisted of high-dose total body irradiation and cyclophosphamide. His transplant course was complicated by neutropenic fevers without positive cultures and skin GVHD. He was discharged on tacrolimus, mycophenolate mofetil and high-dose prednisone. His prophylactic antimicrobials included penicillin VK, valacyclovir, voriconazole and inhaled pentamidine every four weeks, due to a sulfonamide allergy. His post-transplant course was unfortunately complicated by staphylococcal bacteremia and pneumonia, influenza and respiratory syncytial viral infections and pseudomonas sinusitis requiring debridement. In addition to skin GVHD, he also developed pulmonary GVHD confirmed by video assisted thoracic surgery (VATS) biopsy 10 months after his HSCT. The biopsy was negative for aerobic, anaerobic, fungal and acid-fast bacilli (AFB) cultures.

The patient then presented in October, two years after his transplant, with fevers and abdominal pain. At that point, he was on

* Corresponding author.
E-mail addresses: Jason.Chen@jefferson.edu (J. Chen), Jonathan.Pan@jefferson.edu (J. Pan), Joanne.Filicko@jefferson.edu (J. Filicko-O'Hara), Margaret.Kasner@jefferson.edu (M. Kasner), Phyllis.Flomenberg@jefferson.edu (P. Flomenberg).

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dexamethasone 6 mg daily and ruxolitinib 5 mg twice daily for his pulmonary GVHD, and inhaled budesonide and montelukast for his chronic sinusitis. He had been previously on high-dose prednisone and sirolimus, with multiple failed attempts at weaning. He was also on prophylactic posaconazole, valacyclovir, atovaquone and azithromycin. His listed allergies at time of admission were: amoxicillin causing rash, vancomycin causing acute kidney injury, sulfonamide drugs causing rash, and voriconazole causing transaminitis. He was diagnosed with acute pancreatitis with an elevated lipase of 1204 units/l (normal range 13–60 units/l), though CT scan of the abdomen did not show any peripancreatic inflammation.

Workup of the etiology of the pancreatitis was unrevealing. While steroids are a known cause of pancreatitis, the patient had been on both high dose prednisone and dexamethasone in the past without GI complications. The patient denied alcohol use and abdominal ultrasound was negative for cholelithiasis. He had normal triglyceride and calcium levels, negative CMV titers, galactomannan, viral hepatitis serologies, Epstein-Barr virus and adenovirus titers. Stool studies were also negative for infectious diarrheal pathogens. His initial CT scan of the chest to evaluate his fevers showed multiple pulmonary nodules, which were attributed to progression of chronic GVHD (Fig. 1: CT chest on hospital day 4, showing multiple pulmonary nodules and tree-in-bud opacities). He also had a CT of the abdomen that showed a nebulous, hypoattenuating 1 cm lesion in the pancreatic body (Fig. 2: CT abdomen on hospital day 4, with vague 1 cm lesion in pancreatic body).

Due to persistent fevers, he was started on linezolid and meropenem in addition to his prophylactic antimicrobials. Shortly after, he underwent bronchoscopy on hospital day 5, which showed grossly normal airways. Cultures for bacteria, fungus and AFB were initially negative. Bronchoalveolar lavage analysis showed predominantly macrophages, without signs of malignancy, viral or fungal infection. Despite resolution of his abdominal pain and fevers, he had progressive respiratory failure requiring intubation and presumed septic shock of unknown origin.

On hospital day 12, he underwent a second bronchoscopy with both lavage and transbronchial biopsies. His cultures were initially negative although the transbronchial biopsy pathology demonstrated focal interstitial and organizing pneumonia. His antibiotics were broadened by adding amikacin and voriconazole. He was continued on high dose steroids for refractory shock and chronic GVHD and initially showed clinical improvement. His first bronchoscopy cultures showed *Mycobacterium avium* complex (MAC). However, given that no subsequent cultures were positive and he clinically improved, this was thought to be due to colonization.

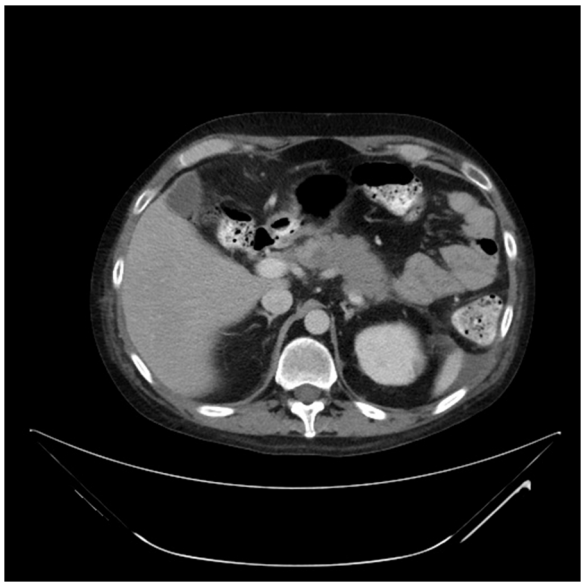

Fig. 2. CT abdomen on hospital day 4, with vague 1 cm lesion in pancreatic body.

On hospital day 20, he developed another episode of acute abdominal pain. Repeat CT of the abdomen showed a new pancreatic tail fluid collection measuring 6 cm x 4.5 cm x 4.1 cm (Fig. 3: CT abdomen on hospital day 20, showing worsening pancreatic collection). Repeat CT of the chest to evaluate his persistent respiratory failure showed progressively worsening pulmonary nodules with a miliary appearance.

The gastroenterology team evaluated the patient and thought his abdominal CT scan was consistent with a simple pancreatic pseudocyst related to his earlier pancreatitis. His lipase level was normal during this episode and his symptoms resolved without intervention. After being weaned off antibiotics and steroids, he developed recurrent abdominal pain with severe guarding concerning for an acute abdomen. On hospital day 25, a CT angiography of the abdomen was performed. It did not show any mesenteric vascular occlusion but noted that the pancreatic fluid collection had become multi-lobed and enlarged in size, measuring 13.8 cm × 7.2 cm × 6.8 cm. In addition, there was a new intraperitoneal loculated collection near the liver (Fig. 4: CT

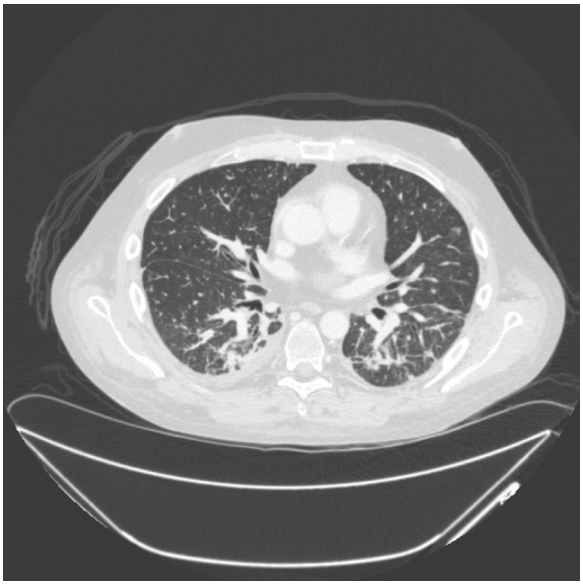

Fig. 1. CT chest on hospital day 4, showing multiple pulmonary nodules and tree-in-bud opacities.

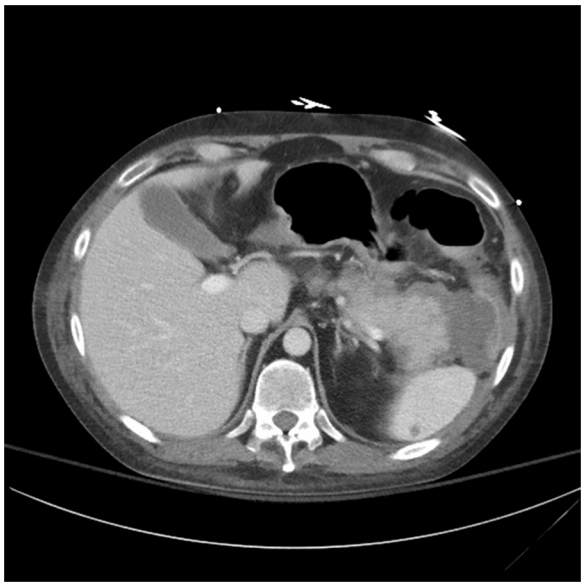

Fig. 3. CT abdomen on hospital day 20, showing worsening pancreatic collection.