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Case Report

Coinfection of *Pneumocystis jirovecii* and Nocardia in an AIDS Patient

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Abstract

A 62-year old man with HIV/AIDS (CD4 count = 15 cells/mm3) presented with fevers, chills, dry cough and weight loss. He had been recently diagnosed with HIV and had not yet started anti-retroviral treatment. Imaging on admission showed right upper lobe and lingular mass-like consolidations. Bronchoscopy revealed *Pneumocystis jirovecii* and he was discharged on trimethoprim-sulfamethoxazole (TMP-SMX). Interestingly, a few weeks later as he continued to improve, cultures from the same bronchoscopy grew 3 different *Nocardia* species, all sensitive to TMP-SMX. Here we present a case of coinfection by *Pneumocystis* and *Nocardia*, the latter of which was vicariously managed by treatment of the former.

Keywords: *Pneumocystis jirovecii; Nocardia; AIDS Patient*

Introduction

Simultaneous lung infection by *Pneumocystis* and *Nocardia* is rare. While there are several species of each, *Pneumocystis jirovecii* and *Nocardia asteroides* are the principal pathogens in humans. Both are largely opportunistic organisms causing, at most, subclinical disease in immunocompetent individuals. In the immunocompromised host, however, infection can be life-threatening.

Pneumocystis causes predominantly respiratory symptoms and even frank respiratory failure and death. *Nocardia* also causes pneumonia and can disseminate throughout the body; a notorious site of infection is the brain. Thankfully, both tend to be susceptible to TMP-SMX. However, detection of the former is much easier than the latter. *Pneumocystis* can be detected on cytologic staining from a respiratory specimen but *Nocardia* is rather slow-growing and can take a few weeks to detect.

Co-infection by these two organisms has been rarely reported. This may be due to it truly being a rare phenomenon though other plausible pathophysiologic explanations exist. Here we report a rare case of *Pneumocystis* and *Nocardia* co-infection. The patient improved significantly with treatment of *Pneumocystis* even before *Nocardia* was found.

Case Presentation

A 62-year old man with a history of human immunodeficiency virus/acquired immune deficiency syndrome (HIV/AIDS; CD4 count = 15 cells/mm^3), diagnosed two months prior but not yet having started antiviral treatment, presented with symptoms of fever, chills, dry cough, and 35-pound weight loss for the last 6 months. He had a temperature of 39 degrees Celsius but otherwise normal vital signs. He was in no respiratory distress and physical examination was largely unremarkable. He was a never-smoker. Laboratory studies included a normal white blood cell count, procalcitonin 0.1 ng/mL, negative influenza PCR, and negative urinary antigens for *Streptococcus* and

Legionella species. Computed tomography (CT) scan of the chest without contrast showed dense, mass-like consolidations in the right upper and middle lobes and in the lingula (Figure 1).

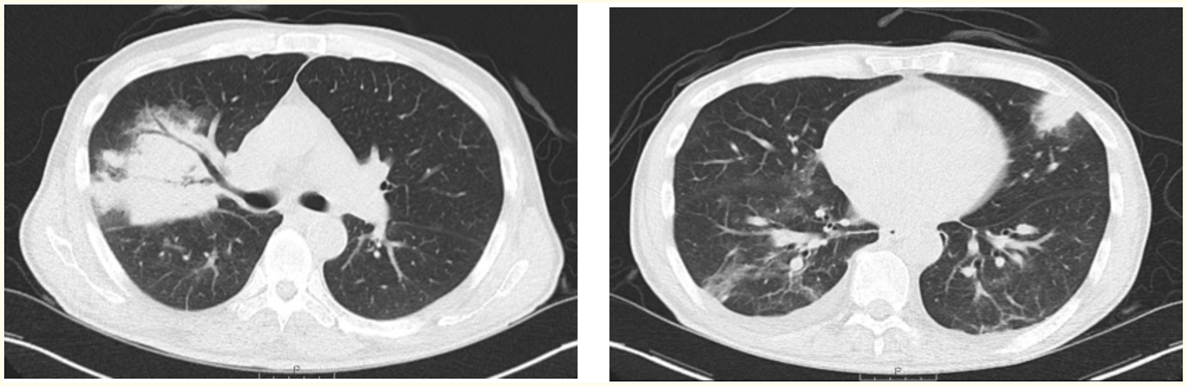

Figure 1: *Computed tomography scan on admission showing mass-like consolidations in the right upper lobe (left panel) and lingula (right panel).*

The differential diagnosis included opportunistic infections but malignancy was highly suspected given the appearance of the lesions. He was started on antibiotics for community-acquired pneumonia. Anti-retroviral therapy consisting of elvitegravir-cobicistat-emtricitabine-tenofovir was started on the fourth hospital day. One daily double-strength tablet of trimethoprim-sulfamethoxazole (TMP-SMX) was started for PCP prophylaxis as well. The pulmonology service was consulted and bronchoscopy was planned to both biopsy the masses and also perform endobronchial ultrasound (EBUS)-guided transbronchial needle aspiration (TBNA) of any abnormal mediastinal or hilar nodes given the concern for malignancy. However, just prior to bronchoscopy, chest X-ray appeared to show improvement in the bilateral infiltrates. This was confirmed on CT scan (Figure 2). As a result, only bronchoalveolar lavage of the affected segments was performed rather than biopsy or EBUS-TBNA since there was a much lower suspicion of malignancy. Indeed, cytologic studies showed organisms consistent with *Pneumocystis* and he was discharged on therapeutic doses of TMP-SMX.

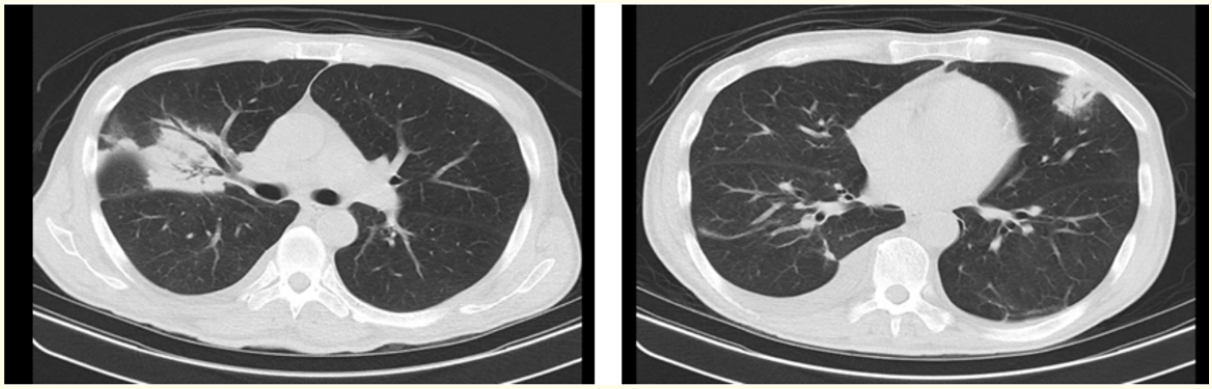

Figure 2: *Computed tomography scan 5 days following the initial scan showing evolution and improvement in the consolidations.*

Interestingly, a few weeks later, bronchoscopic cultures grew *Nocardia farcinica/cyriacigeorgica/otitidiscaviarum* group. Antibiotic sensitivities showed susceptibility to TMP-SMX. Magnetic resonance imaging of the brain was normal. In follow-up over the ensuing few months, serial CT scans of the chest showed resolution of his infiltrates (Figure 3). All symptoms resolved and his weight returned to baseline. He continues on highly active anti-retroviral therapy with significantly improved CD4 and viral load levels.

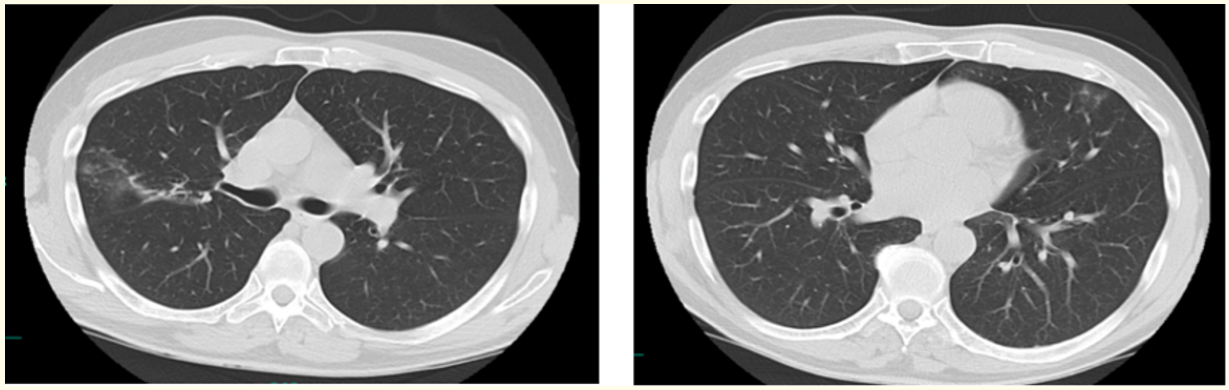

Figure 3: *Computed tomography scan two months later showing marked improvement.*

Discussion and Conclusion

Co-infection in patients with AIDS is a known entity but simultaneous lung infection by *Pneumocystis* and *Nocardia* is rare. To our knowledge, this has only been reported six other times in the literature in the last 30 years [1-6]. Only seven similar cases have been reported in other immunosuppressed, non-HIV conditions [7-12].

Pneumocystis is a fungus of which there are numerous species that infect mammals [13]. *Pneumocystis jirovecii* (PJP) infects and colonizes humans. While there is no clear environmental reservoir, it appears to be spread by airborne transmission. In immunocompetent individuals, this organism poses no clear clinical threat. In immunocompromised hosts, however, PJP can cause severe and life-threatening pneumonia.

Nocardia is a slow-growing, gram-positive actinomycete bacterium that is partially acid-fast [14]. Found in dust, soil, and stagnant water, human infection usually arises from direct inoculation of skin or soft tissues or by inhalation [2]. Numerous species have been identified but *N. asteroides* complex is the most predominant and notorious member of the genus worldwide. Nocardia can disseminate, particularly to the brain, and cause fatal disease.

It is unclear why co-infection by these two organisms has been very infrequently reported. It may truly be a rare occurrence. However, while PJP is readily diagnosed by cytologic studies, *Nocardia* is a slow-growing bacillus the isolation of which may take several weeks. Thus, it may be that co-infection is, indeed, more common than reported but it is not detected. By the time that growth of *Nocardia* comes to attention in a given case, it is conceivable that the patient has been discharged and results may not be followed up or the patient is lost to follow-up.

The ensuing question, then, would be: if *Nocardia* infection is present but not diagnosed, why are there not more patients failing treatment when being treated for PJP? At least a partial explanation would be that *Nocardia* is typically treated with TMP/SMX as first-line

therapy [15]. Thus, patients being treated for PJP with TMP/SMX are also often vicariously being treated for *Nocardia* even if the presence of the latter is not known. Moreover, *Nocardia* requires long-term treatment (~1 year) [15]. Towards that end, PJP pts are maintained on secondary prophylaxis with TMP/SMX after completing treatment. This may suppress *Nocardia* to a subclinical degree. In the case of HIV/AIDS, TMP/SMX prophylaxis is usually continued until the CD4 count is above 200 cells/mm^3. Hence, TMP/SMX is typically not discontinued for at least several months. By the time that TMP/SMX is discontinued, immune reconstitution has occurred, and it is likely that a patient's immune system then can suppress or clear the infection.

In sum, our case demonstrates that in patients with advanced immunosuppression, co-existence of more than one opportunistic infection must be kept in mind. Even when only a single organism has been identified, attention must be given to the possibility of slow-growing organisms that may be isolated over time in the laboratory. If *Pneumocystis* is to be treated, a favorable regimen would include trimethoprim-sulfamethoxazole as this could treat concurrent *Nocardia* until the latter is discovered as well.

Disclosures

Dr. Sabath and Dr. Ombada have no commercial or financial conflicts of interest nor any relevant funding sources.

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