

Co- infectious Cytomegalovirus and Pneumocystis Jiroveci Pneumonia in a Polyarteritis Nodosa Patient: a Case Report

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ABSTRACT

Our report discusses a patient diagnosed with PAN since 3 years ago. He presented with fever, chills and nonproductive cough. He was a long time receiver of immunosuppressant drugs for his underlying condition. Upon examination he was febrile, had cushingoid appearance and crackles in both lungs. Lung CT scan showed opacities in right upper lobe lung and multiple bilateral nodules and ground glass opacity along with mild thickening of pleura. A bronchoscopy was ordered to assess PCP, and without hesitation empirical therapy was started. However, his clinical condition did not improve as expected. At this time, suspecting another infection at play, a PCR and BAL specimen was ordered for CMV. After receiving the result of BAL analysis, our suspicion was confirmed for both PCP and CMV pneumonia. CMV is an important opportunistic infection in immunocompromised individuals. This case highlights this importance in immunocompromising conditions. In this setting, presence of respiratory signs and symptoms point out to PCP as the first differential diagnosis; but at the same time it's crucial for clinicians to consider the possibility of CMV as a co-infective agent.

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Introduction

Polyarteritis Nodosa (PAN) is described as a systemic vasculitis mainly involving medium sized arteries. Although PAN can occur idiopathically at

times, triggering agents have been found to cause this autoimmune disease (1)). Among these factors viruses such as Hepatitis B and C virus, and Cytomegalovirus (CMV) can be named,

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taking into account that the latter mostly occurs in immune compromised patients (2)). CMV is one of the most important life-threatening opportunistic infections, causing pneumonia or diffuse alveolar damage (3)). Pneumocystis Jiroveci is another opportunistic infective agent reported to be seen in immunosuppressed individuals including patients receiving immunosuppressive treatment for vasculitis e.g. PAN causing serious infections such as pneumocystis pneumonia (PCP) (4)). Interestingly enough, our report covers the case of a PAN patient with co-infectious of Pneumocystis and CMV.

Case presentation

A 35 years old man presented with fever, chills and nonproductive cough. He had been diagnosed with classic PAN since 3 years ago, and was initially treated with prednisolone 25 mg two times a day. This corticosteroid regiment was then gradually tapered. Due to the relapse of the disease about 3 months prior to his current hospitalization, he was administered one dose of Cyclophosphamide. After the first dose, the patient developed pancytopenia, bringing his treatment with Cyclophosphamide to a halt. Before receiving Cyclophosphamide due to mild fibrotic changes in superior lobe of the right Lung in imaging findings, an elective fiber optic bronchoscopy was performed. Bronchoscopy did not show any endobronchial lesion, and was clear about tuberculosis.

At the time of admission his vital signs were assessed. His respiratory rate was 30 per minute, blood pressure 120/80 mm/hg, body temperature 38.5, and oxygen saturation was 87 percent. He had fine crackles in lungs auscultation and cushingoid face in examination and the rest of his examination was normal. His first blood laboratory results were reported as following: WBC 3.6×10^3 , neutrophil 79 percent, lymphocyte 9

percent, platelet 134×10^3 , and LDH 523 and the rest of laboratory result was normal. After considering the history of prolonged corticosteroid use along with his other signs and symptoms including fever, hypoxemia, respiratory distress, elevation of LDH, and our first differential diagnosis was PCP. Following this differential diagnosis HRCT imaging of the lungs (Figure 1,2.) and sinus CT imaging (Figure 3) were ordered for assessing the origin of fever and Co-trimoxazole was prescribed empirically to treat PCP until the hypothesis is confirmed. HRCT results showed opacities in right upper lobe and multiple bilateral nodules, ground glass opacity along with mild thickening of pleura. Based on the HRCT results, clinical picture and immune compromised condition of the patient, he was deemed a candidate for a bronchoscopy after stabilization to look for Pneumocystis Jiroveci infection in BAL specimen. Although the patient received widespread antibiotics and corticosteroids for five days, his fever did not resolve which was considering his immune compromised status, an indicator for a possible co-infective agent, most likely CMV. At this time bronchoscopy was performed. Bronchoscopic study did not show any endobronchial mass and Broncho alveolar lavage analysis for Gram, and fungi stain was negative. Confirming our clinical diagnosis, qualitative Pneumocystis Jiroveci PCR, and PCR of CMV were positive. Subsequently, blood specimen was collected for CMV detection. PCR analysis of Blood for CMV showed 1.9×10^5 copies/ml. with this new data in the light, the CMV pneumonia was confirmed. Accordingly, Ganciclovir was ordered immediately and continued for about 3 weeks. After 72 hours of treatment both fever and pancytopenia resolved. We continued the treatment with Co-trimoxazole for 3

weeks. After this period we prescribed Co-trimoxazole 3 times a week prophylactically to prevent recurrence and continued CMV treatment with daily oral Ganciclovir for three months. We should also point out that an ophthalmologic consult was done and his eye examination for CMV retinitis was negative.



Figure 1. Opacities and nodules in Right upper lobe



Figure 2. Lung HRCT showed multiple bilateral nodules, ground glass opacity along with mild thickening of pleura

Discussion

Immuno compromised patients are at risk for opportunistic infections. The use of corticosteroids in rheumatologic disorders make patients who are involved with this spectrum of diseases susceptible for said infections (5-7). CMV disease is common in immune compromised patients (8). CMV is

usually seen in non-invasive, dormant form and is left untreated in the background of another opportunistic respiratory tract infection found in BAL analysis. Although sometimes it can become invasive and, as dangerous as it may be missed (9), this may lead to life threatening morbidity and even mortality in immune compromised patients (10). High dose corticosteroids might reactivate a dormant CMV infection, turning a non-invasive form to a clinically invasive CMV infection (9). It was reported that using steroids in PCP patients could double the risk of mortality in presence of a latent CMV infection (11).



Figure 3. Sinus CT image

In this context, it is of utmost importance to distinguish non-invasive and invasive CMV infection since either one demands a specific approach of management and not rush neither the diagnosis nor the treatment. Our patient was treated with corticosteroids because of his underlying vasculitis. Although at first we assumed that PCP was the main problem and treated the patient accordingly, he did not respond to the treatment. The failure of response to appropriate PCP treatment brought us to suspect that there might be another component to his illness such as CMV infection. After changing the treatment

regimen to cover PCP and CMV co-infection, patient's clinical condition improved dramatically.

Although co-infection of CMV and PCP in large-vessel vasculitis was reported in Vetter report for first time (12), according to our knowledge, our patient is the first documented report of such co-infection in medium-sized blood vessel vasculitis.

Conclusion:

As mentioned above, CMV is an important opportunistic infection in immunocompromised individuals. Detection and appropriate treatment of this infection can be lifesaving at times. In the background of classic PAN as an immune compromising disease, presence of respiratory signs and symptoms point out to PCP as the first differential diagnosis, but at the same time it's crucial for clinicians to consider the possibility of CMV as a co-infective agent.

Conflicts of Interest:

The authors declare that there is no conflict of interest.

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