

Cytomegalovirus pneumonia and pulmonary haemorrhage in a patient with polyarteritis nodosa

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Abstract

Cytomegaloviruses are opportunistic pathogens that cause lung infection in immunocompromised individuals. A 24-year-old male was admitted to the hospital with complaints of cough, fever and dyspnoea. He was receiving immunosuppressive therapy for polyarteritis nodosa. A chest X-ray showed heterogeneous right-sided opacity in the middle and lower lung zones. The diagnosis of cytomegalovirus pneumonia was confirmed by positive test for serum cytomegalovirus IgM antibodies. One day after admission, haemoptysis developed and patient with hemoptysis who had shortness of breath was intubated. Computed tomography (CT) showed bilateral alveolar opacity.

Keywords: Cytomegalovirus, Polyarteritis nodosa, Immunosuppression.

Introduction

Cytomegalovirus (CMV) is a well-known cause of life-threatening opportunistic infection in immunocompromised patients, such as those with acquired immunodeficiency syndrome (AIDS), receiving immunosuppressive therapy and those having undergone organ transplantation.¹ In immunocompromised patients, CMV infection can cause interstitial pneumonia, nodules (inflammatory or haemorrhagic), organizing pneumonia and (in severe cases) diffuse alveolar damage.²⁻⁴

Polyarteritis nodosa is a systemic autoimmune vasculitis characterized by necrotizing inflammatory lesions of the medium-sized and small muscular arteries. It is most commonly treated with a combination of drugs including Corticosteroids and immune suppressants.

Here, we present the case of a patient who was

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receiving immunosuppressive drugs for polyarteritis nodosa and developed cytomegalovirus pneumonia with pulmonary haemorrhage.

Case Report

A 24-year-old male was admitted to the emergency department (ED) of Yuzuncu Yil University, School of Medicine, Van, Turkey, in March 2015 with complaints of cough, fever and dyspnoea. His medical history revealed that, ten years prior, he had been diagnosed with polyarteritis nodosa, which was associated with familial Mediterranean fever, and was treated with azathioprine and prednisolone. On admission, the patient had a blood pressure of 150/70 mmHg, a temperature of 38.0°C, a heart rate of 112 bpm, a respiratory rate of 20 breaths/min and peripheral oxygen saturation of 86%. Auscultation revealed bilateral diffuse crackles. The abdominal exam was unremarkable.

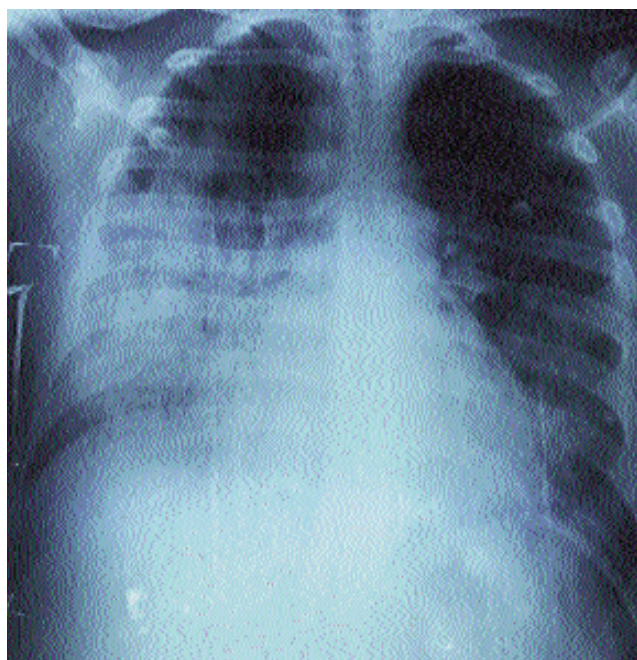


Figure-1: Chest X-ray heterogenous opacity right-sided mid and lower zone were observed.



Figure-2: Computed axial tomography (CT) scan alveolar opacity were observed.

The initial laboratory values were as follows: haemoglobin, 12.9 g/dl; white blood cell count, 16,500/ml; platelet count, 112,000/ml; erythrocyte sedimentation rate, 74 mm in the first hour; C-reactive protein, 166 mg/dl (normal range, 0-5 mg/dl); and creatinine, 2.53 mg/dl. Hepatitis serology was negative for hepatitis B surface antigen and antibodies thereto; anti-hepatitis C virus antibodies; antinuclear antibodies; antineutrophil cytoplasmic antibodies; anticardiolipin antibodies; and lupus anticoagulant. A chest X-ray showed heterogeneous right-sided opacity in the middle and lower lung zones (Figure-1). The diagnosis of cytomegalovirus pneumonia was confirmed by positive test for serum cytomegalovirus IgM antibodies. Combined therapy was started with empirical antibiotic therapy and antiviral therapy with ganciclovir (2.5 mg/kg, twice a day). One day after admission, haemoptysis developed and patient with haemoptysis and shortness of breath was intubated. Computed tomography (CT) scan of the lung was performed which showed bilateral alveolar opacity (Figure-2). Bronchoscopy showed fresh blood flowing out of both lungs. There was an abrupt drop in haemoglobin and haematocrit levels to 6.6 g/dl and 18.4%, respectively, which is suggestive of alveolar haemorrhage. There was no evidence of haemolysis or gastrointestinal bleeding. On the basis of the clinical condition of the patient, pulse therapy with methyl

prednisolone was initiated. Erythrocyte suspension was given, Plasma exchange was performed and intravenous immunoglobulin therapy (20 mg/day) was started. The haemoptysis decreased within a few days. However, the patient died from respiratory failure 3 weeks later.

Discussion

In immunocompromised patients, such as those with acquired immunodeficiency syndrome (AIDS), those receiving immunosuppressive therapy and those who have undergone organ transplantation, CMV exposure can lead to a life-threatening opportunistic infection. Suppression of the cellular immune response is the major

underlying predisposing factor for CMV disease.⁵ Our case was also under long-term immunosuppressive therapy.

In immunocompromised patients, CMV infection can cause interstitial pneumonia, inflammatory nodules and organizing pneumonia.^{2-4,6} Patients with CMV pneumonia usually have high fever, a dry cough, tachypnoea, rales and infiltrations on chest X-rays.^{7,8} Because CMV has significant tropism for the vascular endothelium, CMV infection can induce alveolar haemorrhage. A previous study described a fatal case of fulminant diffuse alveolar haemorrhage in an immunocompetent patient with CMV pneumonia, in whom high-resolution CT demonstrated bilateral perihilar consolidation with air bronchograms.⁹

The radiographic manifestations of CMV pneumonia are variable. Chest X-ray typically shows diffuse interstitial infiltrates.^{2,10} Findings on CT of the chest include ground-glass opacities, dense consolidation, pulmonary nodules and, less commonly, irregular linear opacities.^{3,4,11} In our case, the most pronounced CT finding was bilateral alveolar opacity.

Conclusion

Albeit uncommon, CMV pneumonia can complicate the underlying disease in patients treated with immunosuppressive agents.

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Conflict of Interest: None.

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