

Cytomegalovirus pneumonitis in an immunocompromised host

Sir,

A 41-year-old man, nonsmoker, known to have chronic glomerulonephritis and IgA nephropathy, came to our outpatient department with complaints of persistent dry cough for 1 year and weight loss of 10 kg over 6 months. He had been diagnosed to have chronic glomerulonephritis and IgA nephropathy for the past 3 years and has been taking mycophenolate mofetil 500 mg twice daily and prednisolone 5 mg once daily. On examination, he had pallor, bilateral pitting pedal edema, and bilateral scattered rhonchi. Blood investigations showed hemoglobin 6.2 g/dL, total white blood cell count 2400 cells/mm³, platelet count 2.7 lakh/mm³, blood urea nitrogen 70.7 mg/dL, and serum creatinine 7 mg/dL. Liver function test and blood glucose were within normal limits.

Chest X-ray posteroanterior view showed ill-defined nonhomogenous infiltrates on both lower zones [Figure 1]. HRCT chest showed centrilobular nodules with tree-in-bud appearance as well as cylindrical bronchiectatic changes with subsegmental consolidation involving middle lobe and bilateral lower lobes [Figure 2]. As India is a high prevalence country for pulmonary tuberculosis, our provisional diagnosis was also the same. Being an immunocompromised host, we also considered opportunistic viral and fungal infections as the differential diagnosis. Sputum smear for AFB, CBNAAT, and COVID-19 RTPCR were all negative. Mycophenolate mofetil was stopped, and he was started on intravenous (IV) amoxicillin–clavulanic acid and oral doxycycline. After correcting anemia, he was subjected

to a flexible bronchoscopy. There were no intraluminal lesions. Bronchial washings were taken and sent for Gram stain, bacterial culture, AFB smear, CBNAAT, AFB culture, fungal smear, fungal culture, cytology, Galactomannan assay, *Nocardia* stain, *Pneumocystis jiroveci* stain, and *Cytomegalovirus* (CMV) quantitative PCR. All investigations including fungal culture were negative. However, CMV quantitative PCR (bronchial wash) was 6 lakh copies/ml. Plasma CMV quantitative PCR was 473 copies/ml. Hence, we arrived at a diagnosis of CMV pneumonitis. The patient was started on renal adjusted dose of valganciclovir (450 mg twice daily). Level of immunosuppression was reduced by stopping mycophenolate mofetil, but prednisolone was continued. On follow-up after 3 weeks, he had a dramatic clinical and radiographic improvement [Figure 3].

DISCUSSION

CMV (also known as HHV 5) is a DNA virus, belonging to *Herpesviridae* family. Von Glahn and Pappenheimer described first adult case of CMV in 1925.^[1] CMV infection is asymptomatic or manifests as mononucleosis-like illness in immunocompetent hosts, but it can cause significant morbidity and mortality in immunocompromised hosts. While CMV infection refers to viremia, the term CMV disease implies evidence of CMV infection along with attributable clinical features, which can manifest as a viral syndrome or as tissue invasive diseases such as pneumonitis, retinitis, encephalitis, hepatitis, esophagitis, and colitis. Some patients develop CMV syndrome, a nonspecific presentation characterized by fever and myelosuppression in association with CMV viremia.^[2]

Lungs are the second most common site of acquired CMV infection, after kidneys.^[1] CMV pneumonitis presents with nonspecific symptoms such as low-grade fever, shortness of breath, and dry cough. Radiographic features are also nonspecific, including patchy or diffuse ground-glass opacities, patchy consolidation, small nodular opacities,

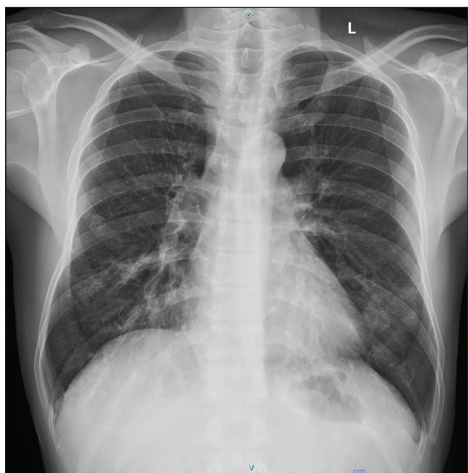


Figure 1: Chest X-ray posteroanterior view on admission showing ill-defined nonhomogenous opacities involving bilateral lower zones

thickened bronchovascular bundles, tree-in-bud opacities, reticular opacities, and small pleural effusions.^[3] Snyder *et al.* observed that despite treatment, CMV pneumonitis in lung-transplant recipients increases the risk of bronchiolitis obliterans syndrome and death.^[4]

The gold standard for diagnosis of tissue invasive CMV disease is biopsy, identifying Owl's eye inclusion bodies or detection of viral nucleic acid in the specimen.^[5] Although biopsy is the gold standard for tissue invasive disease, a study done by Chemaly *et al.* shows that higher viral loads in bronchoalveolar lavage fluid (100% specificity with viral load >5 lakh copies/ml) can be used to identify CMV pneumonitis.^[6] Quantitative PCR done in the plasma along with a pulmonary specimen (biopsy/bronchial wash/bronchoalveolar lavage) can reliably diagnose CMV pneumonitis.

The first-line treatment for CMV disease is IV ganciclovir or its prodrug, oral valganciclovir which acts by inhibiting viral DNA polymerase, thereby interfering with elongation of DNA.^[7] Reduction of level of immunosuppression is an important adjunct in the treatment of CMV disease.^[8]

Our patient responded well to renal adjusted dose of valganciclovir (450 mg twice daily). On follow-up after 3 weeks, he had a dramatic clinical and radiographic improvement.

This case is being presented because of its rarity. One should always keep in mind the possibility of viral infections such as CMV in immunocompromised patients and workup should be tailored accordingly.

Declaration of patient consent

The authors certify that they have obtained all appropriate

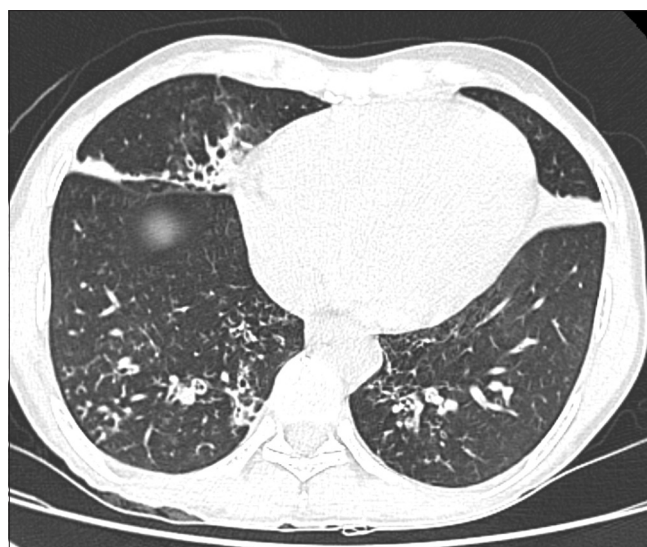


Figure 2: HRCT chest showing centrilobular nodules with tree in bud appearance as well as cylindrical bronchiectatic changes with subsegmental consolidation involving middle lobe and bilateral lower lobes

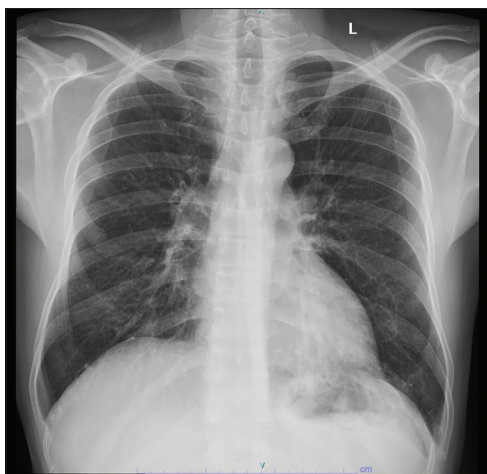


Figure 3: Repeat Chest x ray PA view after 3 weeks of treatment with Valganciclovir showing clearance of infiltrates

patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

**Rabitha Balakrishnan¹, Arjun Padmanabhan¹,
K. A. Ameer¹, Rajalakshmi Arjun²,
Praveen Muralidharan³**

¹Department of Respiratory Medicine, Kerala Institute of Medical Sciences, Trivandrum, Kerala, India, ²Department of Infectious Diseases, Kerala Institute of Medical Sciences, Trivandrum, Kerala, India, ³Department of Nephrology, Kerala Institute of Medical Sciences, Trivandrum, Kerala, India.
E-mail: dr.p.arjun@gmail.com

REFERENCES

1. Cunha BA. *Cytomegalovirus* pneumonia: Community-acquired pneumonia in immunocompetent hosts. *Infect Dis Clin North Am* 2010;24:147-58.
2. Reid GE, Lynch JP 3rd, Weigt S, Sayah D, Belperio JA, Grim SA, et al. Herpesvirus respiratory infections in immunocompromised patients: Epidemiology, Management, and outcomes. *Semin Respir Crit Care Med* 2016;37:603-30.
3. Franquet T, Lee KS, Müller NL. Thin-section CT findings in 32 immunocompromised patients with *Cytomegalovirus* pneumonia who do not have AIDS. *AJR Am J Roentgenol* 2003;181:1059-63.
4. Snyder LD, Finlen-Copeland CA, Turbyfill WJ, Howell D, Willner DA, Palmer SM. *Cytomegalovirus* pneumonitis is a risk for bronchiolitis obliterans syndrome in lung transplantation. *Am J Respir Crit Care Med* 2010;181:1391-6.
5. Kotton CN. CMV: Prevention, diagnosis and therapy. *Am J Transplant* 2013;13 Suppl 3:24-40.
6. Chemaly RF, Yen-Lieberman B, Chapman J, Reilly A, Bekele BN, Gordon SM, et al. Clinical utility of cytomegalovirus viral load in bronchoalveolar lavage in lung transplant recipients. *Am J Transplant* 2005;5:544-8.
7. Razonable RR, Humar A. Cytomegalovirus in solid organ transplant recipients-Guidelines of the American Society of Transplantation Infectious Diseases Community of Practice. *Clin Transplant* 2019;33:e13512.
8. Gugliesi F, Coscia A, Griffante G, Galitska G, Pasquero S, Albano C, et al. Where do we stand after decades of studying human *Cytomegalovirus*? *Microorganisms* 2020;8:E685.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Access this article online

Quick Response Code:



Website:

www.lungindia.com

DOI:

10.4103/lungindia.lungindia_662_21

How to cite this article: Balakrishnan R, Padmanabhan A, Ameer KA, Arjun R, Muralidharan P. *Cytomegalovirus* pneumonitis in an immunocompromised host. *Lung India* 2022;39:202-4.

© 2022 Indian Chest Society | Published by Wolters Kluwer - Medknow