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A cytomegalovirus induced cavitary pneumonia case in a patient with idiopathic thrombocytopenic purpura under corticosteroid treatment

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ABSTRACT

Objective: Cytomegalovirus infection is common in the neonatal period, during childhood, while in adults, it is encountered in immunosuppressed patients. CMV infection is a serious cause of mortality. CMV pneumonitis is seen most frequently in adults, even if ganciclovir treatment is given prophylactically after stem cell transplantation and it is seen at least after kidney transplantation. In this article, we present a case of severe cavitary pneumonia that develops in a patient with prolonged prednisolone therapy due to ITP.

Keywords: Idiopathic Thrombocytopenic Purpura, Pneumonia, Cytomegalovirus

INTRODUCTION

Cytomegalovirus (CMV) is a DNA virus from the herpesvirus family. CMV is found in saliva, upper respiratory tract, leukocytes, breast milk, urine, stool, in all body fluids and is transmitted by contact with one of them (1). Once ingested, CMV remains infected for life. It is also known to be transmitted by blood transfusions and transplantation organs. Virus infections are divided into two as primary and secondary. Primary infection is asymptomatic in people with good immunity. Secondary infection is seen in individuals with suppressed immunity. For example, opportunistic infections associated with CMV are frequently seen in patients after the organ transplantation (2). In these patients, CMV causes fever, lymphadenopathy, rash, and mononucleosis-like symptoms accompanied by lymphocytosis, causing severe mortality and morbidity by involving organs such as the eye, kidney, gastrointestinal system, liver, and even lung (3).

CASE

A 46-year-old male patient was admitted to our internal medicine outpatient clinic with weakness, cough, sputum, fever and chest pain complaints for about 4 weeks. The patient applied to various physicians for the last 3 weeks and used amoxicillin-clavulanic acid 1000 mg 2x1 tablet, clarithromycin 500 mg 2x1 tablet for 10 days, moxifloxacin 500 mg 1x1 tablet for 7 days. In his physical examination, his general condition was poor. He was conscious, his abdomen was comfortable, his respiratory rate was 18 / min, his pulse was 100 / min, his blood pressure was 100/60 mmHg, traube was closed in the abdominal examination, crepitant ral was found in both lung breath sounds, in the first examinations performed upon hearing murmur in the right hemithorax WBC was 18.0 10.3/ql (normal value: 4.1-11.0), (40% neutrophils, 50% lymphocytes, 5% monocytes, 5% other), CRP was 260 mg/L (normal value 0-6), Hb was 12.1 g/dl (normal value 11-15), platelet count was 102.0 10.3/ql (normal value 150-400). As there were consolidated areas in the right lung parenchyma in his anteroposterior lung radiography, he was hospitalized in the internal medicine service with a prediagnosis of pneumonia. The patient has a history of the stent in known coronary arteries and was followed up in our service 3 months ago to investigate the etiology of rash and thrombocytopenia. The patient was diagnosed with idiopathic thrombocytopenic purpura (ITP) due to the examinations.

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Acute viral markers for etiogenesis were negative (EBV IgM negative, IgG positive, CMV IgM negative, IgG positive, HSV-1-2 negative, Anti-HIV negative…). Malignancy screening were negative (gastroscopy, colonoscopy, whole abdominal ultrasonography, contrast-enhanced thoracic and abdominal tomography, tumor markers). As a result of the bone marrow biopsy, ITP diagnosis was made. Blood, urine, sputum, and stool cultures were sent from the patient because of 39 degrees fever in the first 24 hours of the patient's intermission to the service. It was planned to search for ARB in sputum for 3 days. No characteristics were observed in other biochemical and coagulation parameters sent. Pantoprazole 40 mg 1x1, inhaler salbutamol 4x1, wide spectrum intravenous piperacillin-tazobactam 3x 4.5 g with intravenous antipseudomonal activity, Intravenous trimethoprim sulfamethoxasol with pneumoecystis jiroveci sensitivity 400mg 3x1, N-acetyl-cysteine 600 mg peroral 2x1 were started prophylactically. The dose of corticosteroid used by the patient for ITP treatment was reduced by half, and IVIG at 1 gr/kg was given for 2 days. Viral markers were sent. His fever reached 39 degrees in the first 72 hours, and maculopapular rashes were observed in the upper extremities. Spleen size was reported as 130 mm in the whole abdominal ultrasound.

No vegetation was observed on electrocardiography and transthoracic echocardiography. Tuberculin skin test was performed. The test resulted in 2mm at 48th and 72nd hours. Unenhanced thoracic tomography was performed. A cavitary lesion in the right hemithorax and a consolidated area in the parenchyma were observed. Salmonella, toxoplasma, and brucella agglutination tests were negative. HAV, HBV, HCV and HIV, heterophile antibody, EBV, and VCA IgM were negative. No reproduction was observed in blood, urine, and sputum cultures. WBC increased to 22.0 10.3/ql and CRP to 580 mg/l on the 4th day of the patient's hospitalization. Since among the viral markers sent, CMV avidity was low, Anti CMV IgM was 1.40 index positive, Anti CMV IgG was >250.0 AU/mL, CMV PCR was sent, ganciclovir 5mg/kg 2x1 was initiated for the patient in consultation with the infectious diseases specialist. CMV PCR was requested to be sent in bronchoalveolar lavage, but it could not be sent because the patient did not accept the bronchoscopy procedure. Clinical response was observed in the first 48 hours. WBC decreased to 14.0 10.3/ql and CRP 172 mg/L. Thrombocyte count was above 100.0 10.3/ql during follow-up. Ganciclovir treatment was continued for 15 days. He had no fever in his follow-up.

Table 1. Course of CMV IgM and IgG Antibody Titers by Weeks in the Case

	CMV IgM	CMV IgG
First admission	negative	1.8
Second admission	>250	1.4
In the 2nd week of treatment	11	18
In the 4th week of treatment	10	20

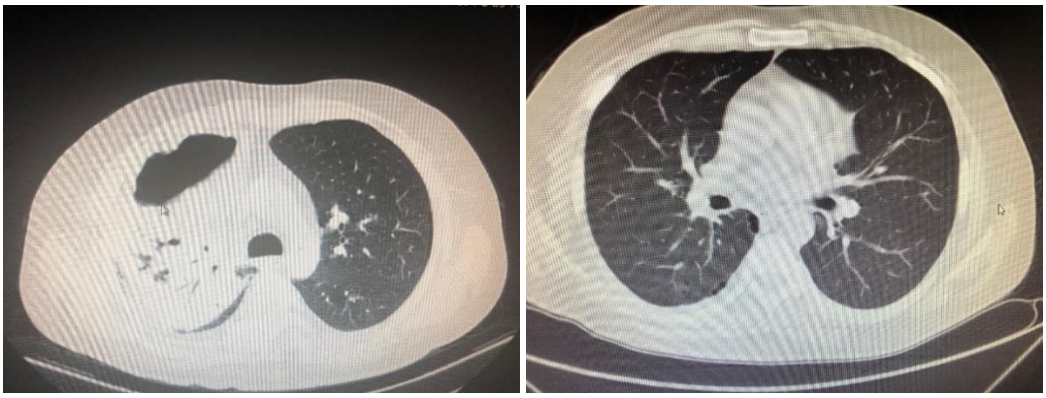

Figure 1. Thoracic examination of the case at the time of diagnosis and after treatment (1st month) due to pneumonia

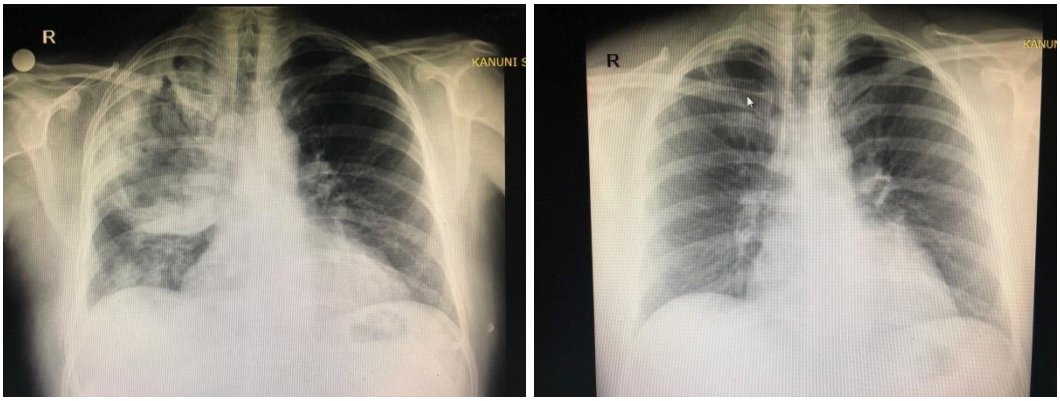

Figure 2. Anteroposterior lung radiography examination of the case at the time of diagnosis and after the treatment (1st month)

In the physical examination, murmur and crepitant rales that were heard by listening to the respiratory system disappeared. Consolidated areas were observed to be regressed, and the cavity to become smaller in the non-contrast thoracic tomography performed as a control. Since the titer of the CMV IgG sent increased, CMV IgM decreased, WBC was 8.2 10.3/ql, CRP was 6 mg/L, thrombocyte count was 104.0 10.3/ql, the patient was transferred to the general surgery unit of our hospital for splenectomy operation.

DISCUSSION

While CMV IgM was negative and CMV IgG was positive, which was sent in our patient's first hospitalization due to thrombocytopenia, the fact that CMV IgM was positive, CMV IgG was positive, CMV PCR was positive, low CMV avidity, which was sent at the second hospitalization due to pneumonia after 3 months of prednisolone use, supports the diagnosis of CMV pneumonia. Since CMV pneumonia can be confused with interstitial pneumonia such as Pneumocystis carinii and Pseudomonas aeruginosa, which are other opportunistic microorganisms, the antibiotic treatment of our patient was continued, but no bacterial growth was observed.

Most people infected with acute CMV have no symptoms suggestive of infection. Clinically indistinguishable symptoms from mononucleosis caused by Epstein Barr virus may develop in 7% of cases with primary CMV infection. Since the immunity is suppressed, reactivation develops when the CD4 count decreases (3). The emerging severe disease manifestation can usually manifest itself with colitis, interstitial pneumonia, hepatitis, meningoencephalitis, myelopathy, bone marrow suppression or retinitis findings. Besides, nonspecific skin rashes may accompany them. In most of the cases, a microbiologically proven source of infection cannot be found (4). Hemorrhagic colitis, abdominal pain, fever may mimic inflammatory bowel disease in gastrointestinal involvement. While mild liver enzymes increase in patients with hepatitis, bilirubin levels generally maintain their normal range. Portal vein thrombosis associated with CMV colitis has been shown in some cases (5). In some immunosuppressed patients, anterior uveitis was observed in the eye examination performed for eye-related complaints, and among the causative agents is CMV after HIV (6). Cardiac involvement is among the mortality of CMV infections in some patients who have received immunosuppressed therapy for a long time, and it was shown that CMV was isolated in endomyocardial biopsy specimens in autopsies (6).

In CMV pneumonia, fever, cough, sputum, and shortness of breath complaints like pneumonia caused by other factors develop. The consolidated area on lung radiography is irregular, and diffuse. Clinical findings, bronchoalveolar lavage (BAL) quantitative culture and CMV PCR method and viral cultures can be used for diagnosis.

The frequent detection of CMV, along with other bacterial pathogens in BAL has made the role of CMV controversial. The majority of CMV infections occur with the reactivation of the latent virus within the first three months after transplantation. (7) Studies conducted so far have shown that 14-day ganciclovir treatment is the most effective antiviral agent in CMV pneumonia. In cases where ganciclovir is insufficient or if there is eye involvement, the use of valganciclovir, foscarnet sodium and cidofovir may included (8).

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Ethical approval: All procedures performed in studies involving human participants were in accordance with the institutional and/or national research committee's ethical standards and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

REFERENCES

1. Lanzieri T, Chung W, Flores M, et al. Hearing loss in children with asymptomatic congenital cytomegalovirus infection. Pediatrics 2017. March; 139(3): e20162610 10.1542/peds.2016-2610

2. Orhan A, Hatice A. Investigation of The Rubella and Cytomegalovirus Seroprevalences by Elisa Method in Pregnant Women. Balikesir Health Sciences Journal. Cilt 6, Sayı 1, 11 - 15, 30.04.2017

3. Leruez-Ville M, Ville Y. Fetal cytomegalovirus infection. Best Practice Res Clin Obstet Gynaecol 2017;38:97-107

4. Manara R, Balao L, Baracchini C, Drigo P, D'Elia R, Ruga EM. Brain magnetic resonance findings in symptomatic congenital cytomegalovirus infection. Pediatric Radiology 2011;41:962-70

5. Bardanzellu F, Fanos V, Reali A. Human breast milk-acquired cytomegalovirus infection: certainties, doubts and perspectives. Curr Pediatr Rev 2019;15:30-41.

6. Monique M, Tejabhiram Y. Cytomegalovirus Retinitis in HIV and Non-HIV Individuals. Microorganisms. 2020 Jan; 8(1): 55.

7. Nakase H, Herfarth H. Cytomegalovirus Colitis, Cytomegalovirus Hepatitis and Systemic Cytomegalovirus Infection: Common Features and Differences. Inflamm Intest Dis. 2016;1:15–23

8. Lancini D, Faddy HM, Flower R, Hogan C. Cytomegalovirus disease in immunocompetent adults. MJA. 2014; 201:578-580.