Case Report

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The Problem Of Cytomegalovirus and Inflammatory Bowel Disease

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1. Abstract

Ulcerative colitis is a chronic, inflammatory disease of unknown etiology. In this group of diseases that progress with remissions and relapses, many factors can cause disease activation. **Cytomegalovirus (CMV) is a latent viral agent that affects 40%-100% of the population. In patients with ulcerative colitis, CMV can present with similar clinical and endoscopic findings to disease activation. In particular, CMV infection screening is recommended for ulcerative colitis patients who are resistant to steroids.** There are many diagnostic methods available for CMV disease; however, the lack of standardization of diagnostic methods and the incomplete determination of cut-off values for PCR values in blood and tissue continue to cause difficulties in diagnosis. **CMV reactivation in patients with severe UC increases the rates of colectomy, and these rates can be reduced with appropriate antiviral therapy. Therefore, the diagnosis and treatment of CMV disease is of great importance.** There is no established standardization for the most appropriate diagnostic method and viral load for making treatment decisions. Further research is required to determine the best approach to antiviral therapy.

2. The Problem Of Cytomegalovirus İn Ulcerative Colitis

Inflammatory bowel diseases (IBD) are a group of chronic inflammatory disorders that affect the digestive tract. The exact cause of IBD is unknown, but it is thought to be caused by a combination of genetic and environmental factors. Although the gastrointestinal system is the primary site of involvement, extraintestinal manifestations may also occur. The diseases often present with abdominal pain, diarrhea, weight loss, and bloody diarrhea. In patients with IBD, many factors can cause disease flares. Some of these flares are caused by disease activation due to inadequate or ineffective medical treatment. Others are caused by bacterial or viral infections that occur in addition to the underlying IBD.

Cytomegalovirus (CMV) is the most common viral infection that can cause damage to the intestinal mucosa in patients with IBD, especially ulcerative colitis. It should be considered in cases of medical treatment resistance, worsening of disease progression after steroid therapy, and perforation [1].

3. Cytomegalovirus

Cytomegalovirus (CMV) is a double-stranded DNA virus that is a member of the herpesvirus family. It is typically acquired in childhood and remains latent for life. Seropositivity for CMV increases with age, with about 40-100% of adults being infected [2]. **In individuals with a compromised immune system, the virus can reactivate and cause organ damage [3]. In individuals with an intact immune system, CMV infection is usually asymptomatic. In symptomatic individuals, it can cause a clinical syndrome similar to infectious mononucleosis, with fever, sore throat, and lymphadenopathy. Depending on the type and severity of immunosuppression, it can also cause hepatitis, colitis, esophagitis, and retinitis [4,5]**. In healthy individuals, CMV reactivation is kept under control in a latent state by CD8-positive T cells and CMV-specific antibodies. In UC patients, continuous inflammation in the colonic mucosa can trigger CMV reactivation. In addition, immunosuppressive drug use, including corticosteroids, can trigger CMV reactivation. The relationship between UC and CMV reactivation was first reported in the literature by Powell et al. in 1964 [6]. Since then, numerous studies have been published on the relationship between CMV infection and UC. However, the prevalence of latent infection was not different between the healthy population and UC patients, so no association between CMV and UC development was detected (69.6% vs 51.8%, odds ratio [OR] = 1.36, 95% confidence interval [CI] = 0.45–4.14, p = 0.59) [7]. The idea that CMV causes UC flares or worsens the severity of the disease is controversial.

Lopes et al. prospectively evaluated 95 patients with endoscopically active UC and compared them with a control group. The rate of positive tissue CMV-DNA in UC patients was 12.1%, while it was 0% in the healthy control group of 50 individuals. Viremia was detected in only 1 patient with IBD [8,9]. The idea of whether CMV is responsible for the severity of colitis or is simply an innocent bystander to the underlying disease is controversial [10]. **The prevalence of CMV reactivation in severe UC ranges from 4.5% to 16.6%. In severe colitis cases that require colectomy, this rate can reach 25%.** This suggests that CMV reactivation

may increase the severity of colitis and the likelihood of surgery, but the issue remains controversial [11,12]. **According to studies, female sex, pancolitis, advanced age, steroid and azathioprine treatments have been found to be risk factors for CMV reactivation [13,14].** The optimal diagnostic method, the best time to start treatment, the duration of treatment, and the issue of changing immunosuppressive therapy have not been clearly established when CMV infection is detected in severe UC.

4. Diagnostic Methods

It is clinically difficult to distinguish between UC activation and CMV colitis. Both UC activation and CMV colitis present with similar clinical symptoms such as hematochezia, abdominal pain, weight loss, and diarrhea. There is no endoscopic finding that can definitively diagnose concomitant CMV colitis in UC activation, as the colonoscopy findings of the two diseases are similar. However, CMV colitis should be considered in UC that is resistant to immunosuppressive therapy. In cases of widespread mucosal defects, longitudinal ulcers, perforated ulcers, and geographic ulcers (Figure 1), CMV colitis should be suspected [15,16]. The diagnostic methods for CMV infection include histology, serology, CMV pp65 antigen, and CMV DNA polymerase chain reactions (PCR) in blood and tissue [17]. Positive CMV serology, antigenemia, or even positive PCR does not necessarily mean CMV infection. The correlation of these tests with active CMV infection is weak. Therefore, the distinction between CMV infection and CMV disease should be made carefully in clinical practice [18]. The prevailing view is that CMV disease should be associated with tissue damage.

5. Serum Antibody Test For Cmv

The titers of CMV-IgM antibodies can increase from the 2nd week to the 6th week after the initial infection and remain positive for 2-3 months. The sensitivity of these antibodies varies from 15% to 60%. [14] CMV-IgM antibodies rarely become positive during reactivation, therefore their diagnostic value in reactivation is low [19]. CMV-IgG antibodies in people who have had CMV infection usually remain positive for life, and the fluctuations in their levels in reactivation situations are low, making them a poor predictive parameter for reactivation.

6. Cmv Antigenemia Test

The sensitivity of CMV antigenemia tests that detect the CMV pp65 protein in circulating leukocytes has been found to be between 60% and 100%, and the specificity has been found to be between 83% and 100%. [20] The detection of the level of antigenemia allows for the measurement of the degree of viremia; however, it should be noted that viremia in the peripheral blood may not reflect CMV reactivation in the gastrointestinal system. CMV antigenemia tests have high specificity but low sensitivity in CMV colitis associated with moderate-severe UC. In studies, the sensitivity of antigenemia tests for the diagnosis of CMV colitis was reported to be 66.7% and the specificity was 87.1%. [21] **A recent study conducted in Korea found a strong association between CMV antigenemia and CMV inclusion bodies in the colonic mucosa, which are considered to be a sign of CMV colitis [22].**

7. Cmv Dna Pcr Testing

The measurement of CMV-DNA in the blood is primarily used to monitor CMV viremia and infection after solid organ and bone marrow transplantations. However, it has been suggested that it be included in the diagnostic algorithm for CMV colitis in UC patients [23]. CMV-DNA measurement in the blood is a highly sensitive and specific method for the detection of CMV viremia. Sensitivity ranges from 65% to 100%, and specificity ranges from 40% to 92% [24,25]. A study found that CMV colitis was predicted in 78.9% of cases with CMV DNA levels of 1150 copies/mL or higher [26]. Studies have shown that the level of CMV-DNA in tissue may be associated with disease activity, and that antiviral therapy can be initiated based on the level of CMV-DNA in tissue [27], therefore, it is recommended that the CMV PCR value in tissue be determined quantitatively, not qualitatively [28]. **Due to the difficulties in measuring the level of CMV-DNA in tissue, the number of biopsies needs to be increased. It has been shown that at least 11 biopsies are needed for the CMV PCR measurement to be 80% successful in patients with UC. Taking biopsies from ulcer bases, where the virus is better detected, will increase the accuracy rate [29,30]. Although there is no established cut-off value for the PCR value in tissue, it has been reported that values higher than 250 copies/mg tissue may predict resistance to steroids and immunosuppressants [28,31,32]**.

8. Histopathological Examination

One of the standard methods for diagnosing CMV infection in UC patients is the histological presence of inclusion bodies. **CMV-infected cells typically contain large inclusion bodies with a halo appearance (Figure 2).** These cells are difficult to show with only hematoxylin and eosin (HE) staining, so they must be stained immunohistochemically (IHC) (Figure 3). The combination of HE and IHC increases sensitivity by 78% to 93% [33].

There is no agreement on the number of IHC-positive cells needed to diagnose CMV colitis. Some studies have reported that 1 in 5 cells must be positive [34], while others have reported that 1 in 10 cells must be positive [35]. It has been recommended that 11 biopsies be taken in UC and 16 biopsies in Crohn's disease for the identification of CMV with IHC. CMV-positive cells are more commonly found at the base of ulcers [36]. However, Zidar et al. showed that there is no difference in CMV-positive cells between the edge and base of ulcers [37]. It is important to note that the detection of CMV-infected cells in tissue does not necessarily mean CMV disease, as CMV can be found in normal mucosal samples.

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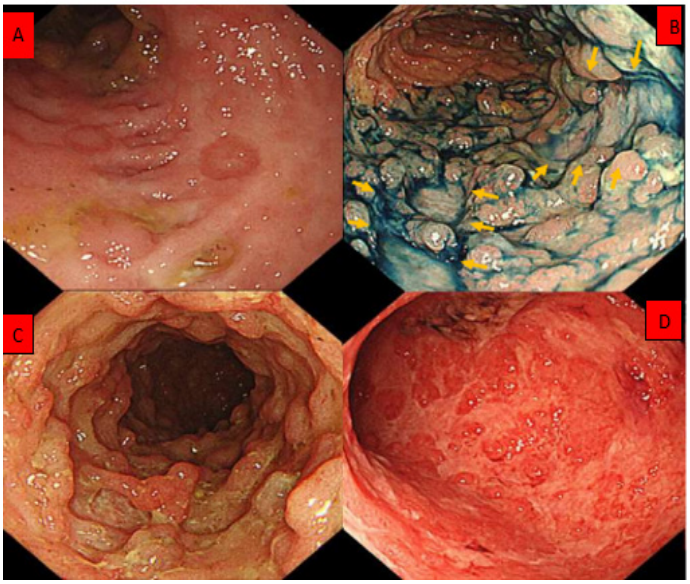

Figure 1: Characteristic endoscopic findings of ulcerative colitis complicated by human cytomegalovirus infection. (a) Punched-out ulcers: the round shape of ulcerationswith clear demarcation. (b) Longitudinal ulcers: ulcerations running along the lumen of the colon (arrows). (c) Di_use mucosal defect: many mucosal defects with surrounding reddish and erythematous mucosa. (d) Geographic ulcers: shallow and widespread mucosal defects with unclear demarcation.

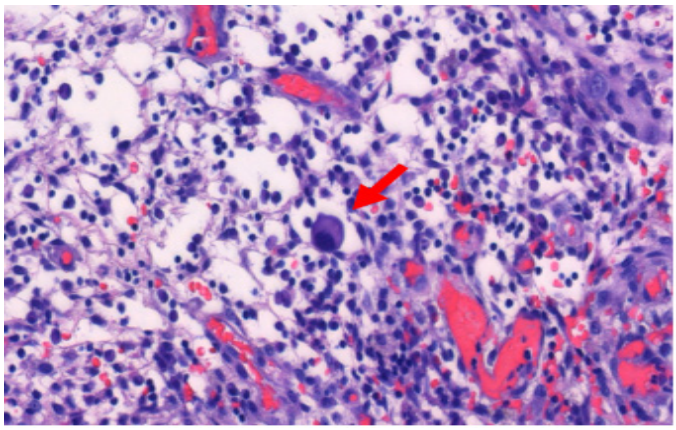

Figure 2: Haematoxylin and eosin [H&E] staining of colonic mucosal biopsy showing enlarged cells with thickened nuclear membrane and intranuclear inclusion bodies

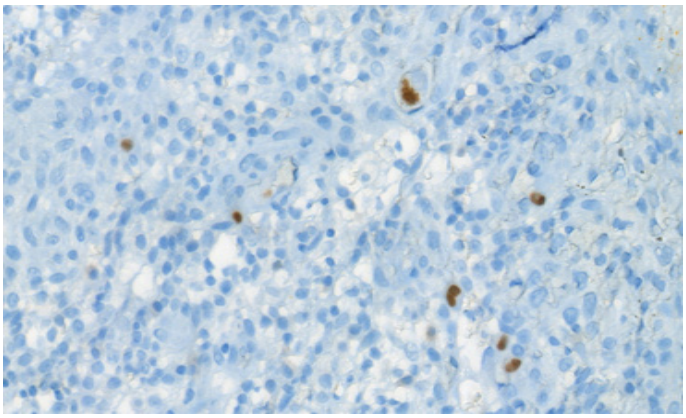

Figure 3: immunohistochemical(IHC) examination CMV antigen positive colon mucosal system

9. Effect Of Treatment

Various studies have been conducted on the efficacy of antiviral treatment in patients with CMV colitis. While some studies have shown that ganciclovir and foscarnet therapy improve outcomes [31,38], there are also studies that show no benefit [39]. **A meta-analysis of 15 studies evaluating steroid-resistant cases found that the rate of colectomy was significantly lower in the antiviral treatment group than in the control group [40].** Jones et al. retrospectively evaluated patients by dividing them into low- and high-density groups according to the density of inclusion bodies in the colonic mucosa using the IHC method. Antiviral treatment significantly reduced the surgical rate in the high-density group [41]. In contrast, Yılmaz et al. published a meta-analysis of the efficacy of ganciclovir in patients with CMV colitis. The analysis showed that ganciclovir treatment did not reduce the rate of surgical resection or mortality [42]. Based on the available data, it can be said that antiviral treatment can reduce the rate of surgical resection in a subset of active UC patients with CMV infection. There is no established standardization for the most appropriate diagnostic method and viral load for treatment decision-making. More research is needed to make decisions about antiviral treatment.

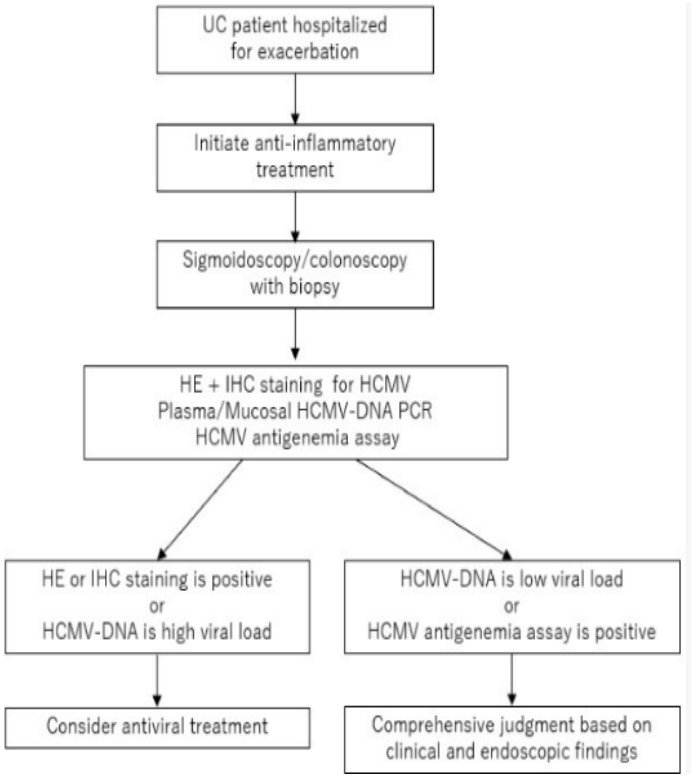

Figure 4: Proposed diagnostic and therapeutic strategy for the patients with ulcerative colitis and the patients with a suspected human cytomegalovirus colitis. UC: ulcerative colitis, HE: hematoxylin and eosin, IHC: immunohistochemistry, HCMV: human cytomegalovirus, PCR: polymerase chain reaction.

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In patients with ulcerative colitis (UC) who are also infected with cytomegalovirus (CMV), anti-inflammatory treatment can suppress the immune system and increase the risk of CMV reactivation and possible complications. The complication rate is especially high when steroids and cyclosporine are used [43]. The European Crohn's and Colitis Organization (ECCO) guidelines do not recommend routine screening for CMV infection before immunomodulatory treatment; however, they recommend evaluation in steroid-resistant colitis [44]. Reducing steroid treatment is recommended to reduce the risk of CMV colitis [45]. Adding azathioprine to steroid treatment further increases the risk of CMV colitis. It is thought that getting rid of steroid dependence can reduce the risk of CMV colitis. The effects of the use of anti-tumor necrosis factor-alpha (TNF-alpha) agents on CMV colitis have also been investigated. Studies have shown that the use of anti-TNF alpha drugs does not cause an increase in CMV colitis. TNF-alpha treatment did not adversely affect the results in CMV colitis patients [46]. Although there are no studies specifically investigating the efficacy of anti-TNFs, they may theoretically lead to a decrease in macrophage differentiation and CMV reactivation in patients with CMV colitis. Vedolizumab is a gut-specific anti-integrin agent that prevents the passage of circulating lymphocytes into the gut. When the safety data of the studies were examined, it was shown that vedolizumab did not increase the risk of CMV colitis [47]. In the UNIFI study, two cases of CMV colitis were reported in patients with UC who received ustekinumab every 12 weeks [48]. Only one case of CMV colitis has been reported in a Crohn's disease patient who received ustekinumab, and it was resolved with ganciclovir therapy [49]. With regard to the risk of CMV colitis in patients with UC who receive tofacitinib, only one case of CMV colitis has been reported in a patient who received 10 mg tofacitinib twice daily during the OCTAVE induction phase [50].

10. Conclusion

There are numerous meta-analyses, recommendations, and algorithms that have been proposed for the treatment of CMV colitis in patients with UC. However, there is no standardized approach to the optimal diagnosis, diagnostic methods, treatment candidates, and treatment duration of CMV colitis. There are still unanswered questions about the use of steroids, immunomodulators, and TNF-alpha agents in patients with CMV colitis. Prospective studies are needed to clarify CMV diagnosis, treatment strategies, and treatment duration in patients with active colitis who have CMV detected in tissues or blood.

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