Review Article

Cytomegalovirus infection of gastrointestinal tract

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ABSTRACT

Gastrointestinal tract infection with CMV can occur from mouth to anal canal. In the immunocompetent subjects, the upper GI tract is most commonly involved while immunocompromised individuals have colon as the most common site of involvement. In a study of CMV infection among AIDS patients, the most common site of CMV infection in the GI tract was the colon (55%), while gastric involvement was reported in 40% of cases. CMV mononucleosis, hepatitis and pneumonitis are among the most common manifestations.' GI tract involvement is common and it is easy to access for taking the diagnostic biopsies. Severity and extent of involvement within the GI tract is variable.

Key words: Cytomegalovirus, opportunistic infection, ulcerative colitis

INTRODUCTION

Gastrointestinal (GI) tract infection with cytomegalovirus (CMV) can occur from mouth to anal canal. In the immunocompetent subjects, the upper GI tract is most commonly involved while immunocompromised individuals have a colon as the most common site of involvement. In a study of CMV infection among AIDS patients, the most common site of CMV infection in the GI tract was the colon (55%), while gastric involvement was reported in 40% of cases. The antrum (25%) was the most common site of upper GI tract CMV infection whereas the sigmoid colon and rectum (35%) were the most common sites in lower GI tract involvement. Colonoscopy is recommended for patients suspected of having CMV infection with colon involvement.

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CMV mononucleosis, hepatitis, and pneumonitis are among the most common manifestations. GI tract involvement is common, and it is easy to access for taking the diagnostic biopsies.[1,2] Severity and extent of involvement within the GI tract are variable.[3,4]

GI CMV infection occurs in two distinct clinical settings: (i) Immunocompetent host; and (ii) immunocompromised patients.

CYTOMEGALOVIRUS INFECTION IN THE IMMUNOCOMPETENT

CMV infection can occur as reactivation of latent virus, or it may be acquired from the exogenous source. CMV induces Fc receptors and the infected cells, and CMV-infected cells bind to beta 2 microglobulin thus evading the humoral immune response. By its interaction with the immune system of the body, CMV has an immune-suppressive effect.

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This immune-suppressive effect of CMV is transient and thus, may not have overt clinical expression.

GI tract infection with CMV may result in diarrhea, acute colitis, and malaise. CMV infection may be superimposed on chronic, preexisting disease in the GI tract. As majority of GI tract infections are self-limiting, and there is a lack of serological evidence of infection in majority of the cases, the real incidence is difficult to establish.

Within GI tract, CMV infects endothelial cells. It may occur as super-infection in a preexisting inflammatory bowel disease, and CMV superinfection is associated with risk of developing toxic megacolon and may even require surgery and has high associated mortality.[5] Several cases of CMV vasculitis have been described in association with chronic ulcerative colitis.[6-10]

CYTOMEGALOVIRUS AS AN OPPORTUNISTIC PATHOGEN IN THE IMMUNOCOMPROMISED

CMV infection as an opportunistic pathogen in post organ transplant patients on immunosuppressive treatment and in AIDS. Posttransplant infection can occur in renal transplant, liver transplant, and bone-marrow transplant recipients.[11-13] Congenital immune deficiencies can also result in CMV infections in the bowel. Steroid therapy also predisposes to opportunistic CMV infection. Steroids at low doses used in the management of arthritis and chronic obstructive airway disease also pose the risk of infection.[14]

Symptoms

Symptoms of GI tract CMV infection vary depending on the site, severity and the extent of involvement within the GI tract. Diarrhea is the most common presenting symptom in the immunocompetent subjects.[4] Weight loss and fever are the common manifestations. Other common symptoms are hematochezia, dysphagia, abdominal distension, pain abdomen, and hematemesis. Perforation has been described especially in the organ transplant recipients.[15-17]

Pathology

CMV infection of GI tract can cause erythema, erosions, ulcerations, pseudotumor formation and perforation.[15,18-21]

Among the various macroscopic lesions due to CMV infection of GI tract, the ulcerative colonic lesions are the most common manifestation. Esophageal involvement can occur with deep ulcerations.[22] Colonic skip ulcers with normal intervening mucosa can endoscopically appear like Crohn's disease.

CMV can cause toxic megacolon in patient with preexisting inflammatory bowel disease, but CMV itself can cause vasculitis with toxic megacolon.

Bowel perforation has been described, most commonly involving terminal ileum and splenic flexure of the colon.[20,23,24]

Bowel obstruction due to inflammatory pseudotumor involving the ileocaecal region has been described.[21] Features of gastric outlet obstruction may occur because of inflammatory mass involving gastric antrum.

Ulcero-stenotic lesion in colon may have a radiological resemblance of neoplasm.[25]

Hemorrhagic colitis from active proctocolitis is well described.[26]

Pseudomembrane formation as a manifestation of CMV colitis has been described.

Pneumatosis intestinalis has been reported in renal transplant recipient with CMV colitis.[27,28]

Biliary tract involvement has been reported in form of cholangitis, acalculous cholecystitis, and papillary stenosis.[29]

PATIENT PROFILE AND ENDOSCOPIC FEATURES OF PATIENTS WITH GASTROINTESTINAL TRACT CYTOMEGALOVIRUS INFECTION

In our patients of GI tract CMV disease [Table 1], three patients were immunocompromised, and one had preexisting underlying ulcerative colitis.

First patient was a diagnosed case of common variable immunodeficiency (CVID) on replacement dose of intravenous (IV) immunoglobulin (Ig). He presented with

Table 1: Patient profile, site involved, and treatment outcomes in gastrointestinal cytomegalovirus disease

Underlying disease	Age/ gender	Organ involved	Treatment given	Outcome	Endoscopy features	Indication for endoscopy
CVID	52/male	Duodenum	Ganciclovir (oral) × 3 weeks + intravenous immunoglobulin	Good		Abdominal pain and diarrhea
AIDS	41/female	Esophagus	Ganciclovir (intravenous) × 3 weeks + HAART	Good	Erythema and diffuse ulcers	Odynophagia
Renal transplant	38/male	Stomach	Ganciclovir (oral) × 3 weeks	Good	Erythema and antral ulcer	Abdominal pain
Ulcerative colitis	37/female	Colon	Ganciclovir (oral) × 2 weeks	Good	Erythema and confluent ulcers in rectum and sigmoid colon	Bloody stools

CVID: Common variable immunodeficiency, HAART: Highly active antiretroviral therapy

3 weeks history of diarrhea associated with epigastric pain. Endoscopy was done and duodenal biopsies were taken to rule out gluten-enteropathy. Figure 1 shows the microscopic image of duodenal biopsy with CMV inclusion bodies. A similar case has been reported where duodenal CMV infection was reported complicating the CVID associated sprue.[30]

Our second patient was HIV-positive female with CD-4+ cell count 7/mm^3. She had fever and odynophagia for 3 weeks. She was empirically treated with anti-fungal drugs elsewhere. Endoscopy was done for the evaluation of esophagus, showed diffuse erythema and large ulcers involving the entire esophagus [Figure 2]. There was a large, oblong, deep ulcer in the distal third of the esophagus, from which multiple biopsies were taken. The biopsy showed stratified squamous epithelium with plenty of inflammatory cells with large cells showing typical intranuclear inclusion bodies suggestive of CMV esophagitis [Figure 3]. Ophthalmology review was done to rule out concomitant retinal involvement. There was no retinal involvement in the present case. She was started on IV gancyclovir along with antiretroviral therapy (ART).[31,32]

The third patient was postrenal transplant on immunosuppressive therapy for last 6 months. He presented with upper abdominal pain with a history of occasional vomiting for 2 weeks. Endoscopic evaluation showed erythema with multiple small clean-based ulcers in the gastric antrum. Biopsy from the ulcer base showed large cells with intranuclear inclusion bodies typical of CMV infection [Figures 4 and 5] GI symptoms-pain abdomen, nausea, and diarrhea are common postrenal transplant patients. There have been few case reports of CMV gastric ulcer in renal-transplant recipient in the literature.[33,34]

The fourth patient was a known case of ulcerative colitis on maintenance mesalamine treatment. She had increased stool frequency with urgency and tenesmus. Sigmoidoscopy showed erythema with confluent ulcers

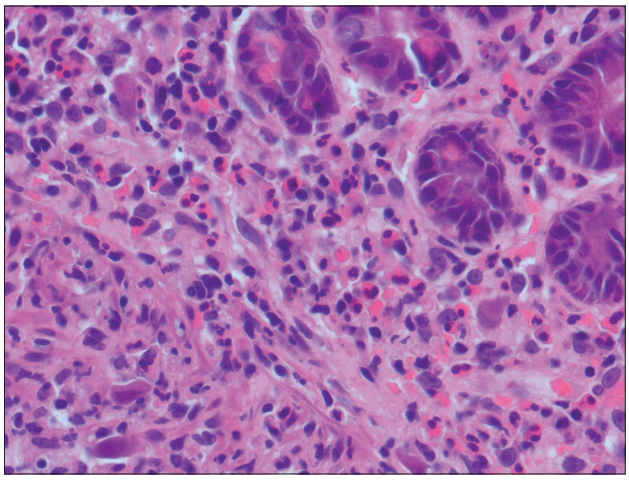

Figure 1: Microscopic image of duodenal biopsy, showing large cells with cytomegalovirus intranuclear inclusion bodies

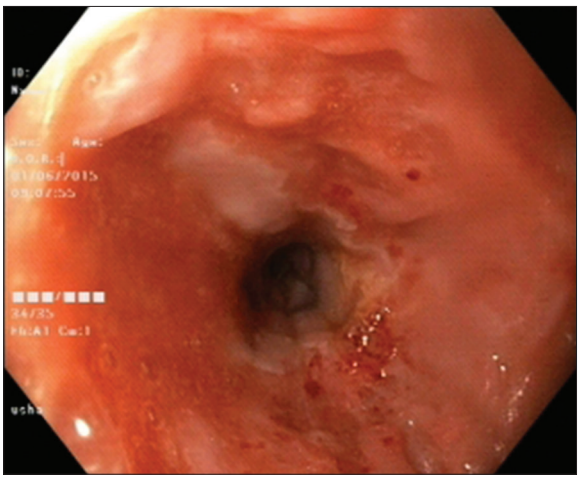

Figure 2: Endoscopy image showing large, deep punched-out ulcer in the lower esophagus

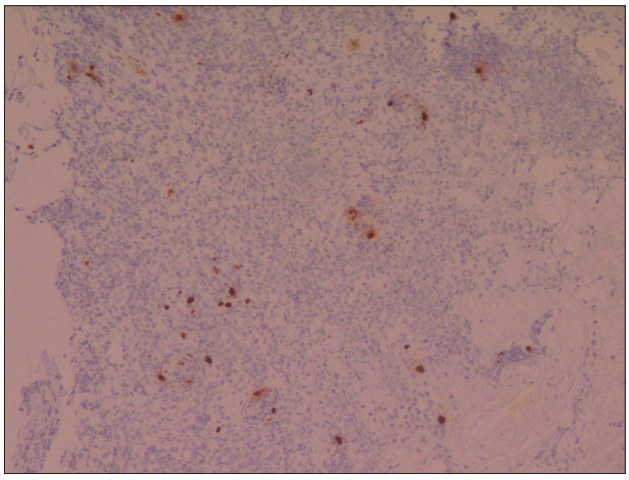

Figure 3: Esophageal biopsy showing positive immunohistochemical for cytomegalovirus

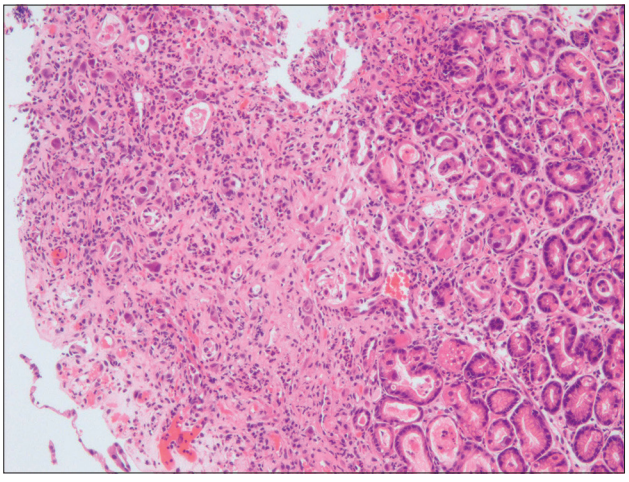

Figure 4: Microscopy image of antral biopsy showing large intranuclear inclusion bodies typical of cytomegalovirus infection

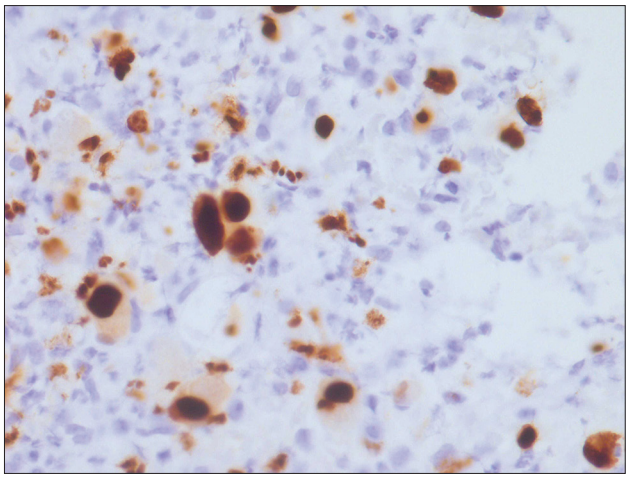

Figure 5: Photomicrograph showing cytomegalovirus - immunohistochemical positive stain on antral biopsy

in recto-sigmoid. Biopsy from the ulcers confirmed super-imposed CMV infection. She responded well to oral ganciclovir treatment. Increased re-activation of CMV in patients of ulcerative colitis has been reported in the literature.[35]

DIAGNOSIS OF CYTOMEGALOVIRUS INFECTION OF THE GASTROINTESTINAL TRACT

During the active CMV infection, there is the stage of early antigen production which is associated with IgM antibody response. This is followed by late antigen production and stage of latency during which CMV genome is incorporated in the host cell nucleus.

Serology

High titer of IgM antibodies or increasing titers of IgG antibody indicates present infection. In the immunocompromised persons, the interpretation of serology may be difficult.

Culture

CMV can be cultured in the medium with human embryo fibroblasts. Positive culture does not necessarily mean an active infection as virus can be excreted for months after the primary infection.[36]

Cytomegalovirus antigenemia

Detection of CMV antigen is a marker of an active infection. Antigen can be detected in circulation in the early stage of infection thus CMV antigenemia helps to detect active infection at an early stage.[37]

Histology and immunohistology

"Owl's eye" intranuclear inclusions are the hallmark of CMV in routine hematoxylin, and eosin staining of the tissue. These inclusions may be seen in the endothelial cells, stromal cells, and epithelial cells.

Presence of inclusion bodies in the stained tissue indicates present CMV infection. Atypical inclusions may cause diagnostic difficulty due to confusion with HSV inclusion body. In cases with diagnostic difficulty immunohistochemical staining helps to confirm the CMV infection. Both, early and late antigens can be detected by immunocytochemistry. Therefore, immunocytochemical staining is more sensitive for diagnosing active infection.[38]

Detection of viral nucleic acid

Detection of CMV nucleic acid by polymerase chain reaction (PCR) is a sensitive method but it does not always indicate the active infection. The PCR technique requires the homogenized tissue sample, so the histological details are not available. Contrary to this, *in-situ* hybridization allows the viral detection within the cells, thus histological details are available. PCR and *in-situ* hybridization positivity does not necessarily mean active infection as these may be positive in latent infection.[39,40]

Light microscopy for a demonstration of CMV inclusion bodies is an easy technique, but viral inclusion bodies are not always demonstrable.[41]

LOCATION AND MORPHOLOGY OF CYTOMEGALOVIRUS INFECTED CELLS

In the esophageal biopsy, CMV was detected in the endothelial cells, pericytes, and the inflammatory cells but it was not demonstrable in the epithelial cells.

In the gastric biopsy, virus was found to infect the glandular epithelial cells. While in the colonic biopsy, endothelial cells, lamina propria cells, and pericytes were infected.

MANAGEMENT OF CYTOMEGALOVIRUS INFECTIONS IN THE GUT

For the treatment of documented CMV infection of GI tract, gancyclovir is considered the drug of choice regardless of the cause of the underlying immunosuppression.[42] The recommended dose is 250 mg IV, twice in a day for at least 3 weeks.[43-46]

Oral ganciclovir prophylaxis is given to HIV-infected persons with CD-4+ count $<50/mm^3$.[47]

Cessation of prophylaxis may be considered when the CD-4 count becomes $>100–150/mm^3$ in response to highly active ART and the prophylaxis restarted if the CD-4 count becomes $<100/mm^3$.[48-50]

Foscarnet can be used as an alternative to ganciclovir.[51] It is a pyrophosphate derivative and has virostatic action. Esophageal ulcers heal well with the foscarnet treatment. Relapses may occur after the treatment is stopped.

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Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Hinnant KL, Rotterdam HZ, Bell ET, Tapper ML. Cytomegalovirus infection of the alimentary tract: A clinicopathological correlation. Am J Gastroenterol 1986;81:944-50.
2. Rotterdam H. Contributions of gastrointestinal biopsy to an understanding of gastrointestinal disease. Am J Gastroenterol 1983;78:140-8.
3. Cheung AN, Ng IO. Cytomegalovirus infection of the gastrointestinal tract in non-AIDS patients. Am J Gastroenterol 1993;88:1882-6.
4. Teixidor HS, Honig CL, Norsoph E, Albert S, Mouradian JA, Whalen JP. Cytomegalovirus infection of the alimentary canal: Radiologic findings with pathologic correlation. Radiology 1987;163:317-23.
5. Cooper HS, Raffensperger EC, Jonas L, Fitts WT Jr. Cytomegalovirus inclusions in patients with ulcerative colitis and toxic dilation requiring colonic resection. Gastroenterology 1977;72:1253-6.
6. Keren DF, Milligan FD, Strandberg JD, Yardley JH. Intercurrent cytomegalovirus colitis in a patient with ulcerative colitis. Johns Hopkins Med J 1975;136:178-82.
7. Levine RS, Warner NE, Johnson CF. Cytomegalic inclusion disease in the gastro-intestinal tract of adults. Ann Surg 1964;159:37-48.
8. Powell RD, Warner NE, Levine RS, Kirsner JB. Cytomegalic inclusion disease and ulcerative colitis; report of a case in a young adult. Am J Med 1961;30:334-40.
9. Sidi S, Graham JH, Razvi SA, Banks PA. Cytomegalovirus infection of the colon associated with ulcerative colitis. Arch Surg 1979;114:857-9.
10. Wong TW, Warner NE. Cytomegalic inclusion disease in adults. Report of 14 cases with review of literature. Arch Pathol 1962;74:403-22.
11. Esforzado N, Poch E, Almirall J, Bombí JA, López-Pedret J, Revert L. Cytomegalovirus colitis in chronic renal failure. Clin Nephrol 1993;39:275-8.
12. McDonald GB, Sharma P, Hackman RC, Meyers JD, Thomas ED. Esophageal infections in immunosuppressed patients after marrow transplantation. Gastroenterology 1985;88 (5 Pt 1):1111-7.
13. Dummer JS. Cytomegalovirus infection after liver transplantation: Clinical manifestations and strategies for prevention. Rev Infect Dis 1990;12 Suppl 7:S767-75.
14. Aukrust P, Moum B, Farstad IN, Holter E, Bjørneklett A, Kremer D. Fatal cytomegalovirus (CMV) colitis in a patient receiving low dose prednisolone therapy. Scand J Infect Dis 1991;23:495-9.
15. Dieterich DT, Rahmin M. Cytomegalovirus colitis in AIDS: Presentation in 44 patients and a review of the literature. J Acquir Immune Defic Syndr 1991;4 Suppl 1:S29-35.
16. Rubin RH. Impact of cytomegalovirus infection on organ transplant recipients. Rev Infect Dis 1990;12 Suppl 7:S754-66.
17. Frank D, Raicht RF. Intestinal perforation associated with cytomegalovirus infection in patients with acquired immune deficiency syndrome. Am J Gastroenterol 1984;79:201-5.
18. Wilcox CM, Robert F. Prospective endoscopic characterization of cytomegalovirus esophagitis in AIDS. Gastrointest Endosc 1994;40:481-4.
19. Goodgame RW, Genta RM, Estrada R, Demmler G, Buffone G. Frequency of positive tests for cytomegalovirus in AIDS patients: Endoscopic lesions compared with normal mucosa. Am J Gastroenterol 1993;88:338-43.
20. DeRiso AJ 2nd, Kemeny MM, Torres RA, Oliver JM. Multiple jejunal perforations secondary to cytomegalovirus in a patient with acquired immune deficiency syndrome. Case report and review. Dig Dis Sci 1989;34:623-9.
21. Wisser J, Zingman B, Wasik M, Duva-Frissora A, Beazley R, McAneny D. Cytomegalovirus pseudotumor presenting as bowel obstruction in a patient with acquired immunodeficiency syndrome. Am J Gastroenterol 1992;87:771-4.
22. Connolly GM, Hawkins D, Harcourt-Webster JN, Parsons PA, Husain OA, Gazzard BG. Oesophageal symptoms, their causes, treatment, and prognosis in patients with the acquired immunodeficiency syndrome. Gut 1989;30:1033-9.
23. Tatum ET, Sun PC, Cohn DL. Cytomegalovirus vasculitis and colon perforation in a patient with the acquired immunodeficiency syndrome. Pathology 1989;21:235-8.
24. Kram HB, Shoemaker WC. Intestinal perforation due to cytomegalovirus in patients with AIDS. Dis Colon Rectum 1990;33:1037-40.
25. Elta G, Tumage R, Eckhauser FE, Agha F, Ross S. A submucosal antral mass caused by cytomegalovirus infection in a patient with acquired immunodeficiency syndrome. Am J Gastroenterol 1986;81:714-7.
26. Bennett MR, Fine AP, Hanlon JT. Cytomegalovirus hemorrhagic colitis in a non-transplant patient. Postgrad Med J 1985;77:227-9.
27. Frager DH, Frager JD, Wolf EL, Rand LG, St Onge G, Mitsudo S, *et al.* Cytomegalovirus colitis in acquired immune deficiency syndrome: Radiologic spectrum. Gastrointest Radiol 1986;11:241-6.
28. van Son WJ, van der Jagt EJ, van der Woude FJ, Slooff MJ, Meijer S, The TH, *et al.* Pneumatosis intestinalis in patients after cadaveric kidney transplantation. Possible relationship with an active cytomegalovirus infection. Transplantation 1984;38:506-10.
29. Blumberg RS, Kelsey P, Perrone T, Dickersin R, Laquaglia M, Ferruci J. Cytomegalovirus- and Cryptosporidium-associated acalculous gangrenous cholecystitis. Am J Med 1984;76:1118-23.
30. Medlicott SA, Coderre S, Horne G, Panaccione R. Multimodal immunosuppressant therapy in steroid-refractory common variable immunodeficiency sprue: A case report complicating cytomegalovirus infection. Int J Surg Pathol 2006;14:101-6.
31. Whitley RJ, Jacobson MA, Friedberg DN, Holland GN, Jabs DA, Dieterich DT, *et al.* Guidelines for the treatment of cytomegalovirus diseases in patients with AIDS in the era of potent antiretroviral therapy: Recommendations of an international panel. International AIDS Society-USA. Arch Intern Med 1998;158:957-69.
32. Wilcox CM, Straub RF, Schwartz DA. Prospective endoscopic characterization of cytomegalovirus esophagitis in AIDS. Gastrointest Endosc 1994;40:481-4.
33. Ishiguro S, Ikeda T, Shimamoto K, Tanji N, Ohoka H, Yokoyama M. Cytomegalovirus infection in peptic ulcer in renal transplant recipient: A case report. Nihon Hinyokika Gakkai Zasshi 2004;95:777-80.
34. Gupta T, Agarwal D, Mandot A, Desai D, Joshi A, Abraham P. Cytomegalovirus involving gastric antrum in immunocompromised hosts: A report of 5 cases. Indian J Gastroenterol 2005;24:258-60.
35. Mariguela VC, Chacha SG, Cunha Ade A, Troncon LE, Zucoloto S, Figueiredo LT. Cytomegalovirus in colorectal cancer and idiopathic ulcerative colitis. Rev Inst Med Trop Sao Paulo 2008;50:83-7.
36. Gregory WW, Menegus MA. Practical protocol for cytomegalovirus isolation: Use of MRC-5 cell monolayers incubated for 2 weeks. J Clin Microbiol 1983;17:605-9.
37. van der Bij W, Torensma R, van Son WJ, Anema J, Schirm J, Tegzess AM, *et al.* Rapid immunodiagnosis of active cytomegalovirus infection by monoclonal antibody staining of blood leucocytes. J Med Virol 1988;25:179-88.
38. Zweygberg Wirgart B, Landqvist M, Hökeberg I, Eriksson BM, Olding-Stenkvist E, Grillner L. Early detection of cytomegalovirus in cell culture by a new monoclonal antibody, CCH2. J Virol Methods 1990;27:211-9.
39. Wakefield AJ, Fox JD, Sawyerr AM, Taylor JE, Sweenie CH, Smith M, *et al.* Detection of herpesvirus DNA in the large intestine of patients with ulcerative colitis and Crohn's disease using the nested polymerase chain reaction. J Med Virol 1992;38:183-90.
40. Niedobitek G, Finn T, Herbst H, Gerdes J, Grillner L, Landqvist M, *et al.* Detection of cytomegalovirus by *in situ* hybridisation and

immunohistochemistry using new monoclonal antibody CCH2: A comparison of methods. J Clin Pathol 1988;41:1005-9.
41. Schwartz DA, Wilcox CM. Atypical cytomegalovirus inclusions in gastrointestinal biopsy specimens from patients with the acquired immunodeficiency syndrome: Diagnostic role of *in situ* nucleic acid hybridization. Hum Pathol 1992;23:1019-26.
42. Buhles WC Jr., Mastre BJ, Tinker AJ, Strand V, Koretz SH. Ganciclovir treatment of life- or sight-threatening cytomegalovirus infection: Experience in 314 immunocompromised patients. Rev Infect Dis 1988;10 Suppl 3:S495-506.
43. Dieterich DT, Chachoua A, Lafleur F, Worrell C. Ganciclovir treatment of gastrointestinal infections caused by cytomegalovirus in patients with AIDS. Rev Infect Dis 1988;10 Suppl 3:S532-7.
44. Buhles WC, Mastre BJ, Tinker AJ. Syntex collaborative ganciclovir treatment study group. Ganciclovir treatment of life- or site-threatening cytomegalovirus infection: Experience in 314 immunocompromised patients. Rev Infect Dis 1988;10 Suppl 3:S495-504.
45. Spector SA, McKinley GF, Lalezari JP, Samo T, Andruczk R, Follansbee S, *et al.* Oral ganciclovir for the prevention of cytomegalovirus disease in persons with AIDS. Roche Cooperative Oral Ganciclovir Study Group. N Engl J Med 1996;334:1491-7.
46. Brosgart CL, Louis TA, Hillman DW, Craig CP, Alston B, Fisher E, *et al.* A randomized, placebo-controlled trial of the safety and efficacy of oral ganciclovir for prophylaxis of cytomegalovirus disease in HIV-infected individuals. Terry Beirn Community Programs for Clinical Research on AIDS. AIDS 1998;12:269-77.
47. Masur H, Kaplan JE, Holmes KK; U.S. Public Health Service; Infectious Diseases Society of America. Guidelines for preventing opportunistic infections among HIV-infected persons – 2002. Recommendations of the U.S. Public Health Service and the Infectious Diseases Society of America. Ann Intern Med 2002 3;137 (5 Pt 2):435-78.
48. Vrabec TR, Baldassano VF, Whitcup SM. Discontinuation of maintenance therapy in patients with quiescent cytomegalovirus retinitis and elevated CD4+ counts. Ophthalmology 1998;105:1259-64.
49. Macdonald JC, Torriani FJ, Morse LS, Karavellas MP, Reed JB, Freeman WR. Lack of reactivation of cytomegalovirus (CMV) retinitis after stopping CMV maintenance therapy in AIDS patients with sustained elevations in CD4 T cells in response to highly active antiretroviral therapy. J Infect Dis 1998;177:1182-7.
50. Jouan M, Savès M, Tubiana R, Carcelain G, Cassoux N, Aubron-Olivier C, *et al.* Discontinuation of maintenance therapy for cytomegalovirus retinitis in HIV-infected patients receiving highly active antiretroviral therapy. AIDS 2001;15:23-31.
51. Nelson MR, Connolly GM, Hawkins DA, Gazzard BG. Foscarnet in the treatment of cytomegalovirus infection of the esophagus and colon in patients with the acquired immune deficiency syndrome. Am J Gastroenterol 1991;86:876-81.