

## Gastrointestinal Tract Cytomegalovirus Disease in Southwestern Taiwan: A Clinical Study of 1950 Endoscopic Biopsies

Seng-Kee Chuah, MD; Chi-Sin Changchien, MD; Chung-Mou Kuo, MD; Keng-Liang Wu, MD; King-Wah Chiu, MD; Yi-Chun Chiu, MD; Jien-Wei Liu<sup>1</sup>, MD; Hock-Liew Eng<sup>2</sup>, MD; Shue-Shian Chiou, MD

**Background:** Gastrointestinal (GI) cytomegalovirus (CMV) disease occurs in adult patients with immune suppression. This study reviews and discusses the clinical settings, endoscopic features, and locations of GI CMV lesions.

**Methods:** In total, 1950 endoscopic biopsy reports for all GI tract lesions in Chang Gung Memorial Hospital, Kaohsiung were retrospectively reviewed for CMV disease from 1999 to 2002. Only those patients found to be positive for viral inclusion bodies in tissue specimens, with further confirmation by special immunohistochemical staining, were enrolled in this study.

**Results:** Our series showed that all 5 patients were immunosuppressed when attacked by the virus. The esophagus (n = 2) and colon (n = 2) were infected in 4 patients, while stomach was involved in one patient. Those patients with lesions of the esophagus and stomach followed a more-benign clinical course. Endoscopic examination showed 1 or more prominent ulcers in the distal esophagus and at the antrum of the stomach. Rare endoscopic findings of diffuse esophageal CMV disease and severe and extensive colitis were presented. The overall mortality rate was 20%, and all but 1 patient responded well to ganciclovir treatment.

**Conclusions:** We emphasize that GI CMV disease is still rare in Taiwan. A high degree of suspicion for CMV disease is important when diagnosing immunosuppressed patients suffering from GI symptoms.

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**Key words:** GI tract cytomegalovirus disease, cytomegalic inclusion body.

All primary CMV infections usually resolve and enter a state of latency in healthy individuals.<sup>(1-3)</sup> The GI tract is at risk for subsequent CMV disease, and may contain latent viruses which could lead to local disease with reactivation.<sup>(4-6)</sup> This occurs partic-

ularly in adult patients with immune deficiencies, such as those suffering from acquired immunodeficiency syndrome (AIDS), organ transplant recipients, cancer patients undergoing chemotherapy, and those receiving steroid therapy.<sup>(7-14)</sup> Although CMV

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From the Division of Hepato-Gastroenterology; <sup>1</sup>Division of Infectious Diseases, <sup>2</sup>Department of Internal Medicine, Department of Pathology, Chang Gung Memorial Hospital, Kaohsiung.

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Address for reprints: Dr. Shue-Shian Chiou, Division of Gastroenterology, Department of Internal Medicine, Chang Gung Memorial Hospital, No. 123, Dabi Rd., Niasung Shiang, Kaohsiung, Taiwan 833, R.O.C Tel.: 886-7-7317123 ext. 8301; Fax: 886-7-7322402; E-mail: kyutarou@hotmail.com

may be present in the gastrointestinal tracts of 30%~43% of AIDS patients, only about 7.4% of these patients develop clinically apparent CMV disease.<sup>(6)</sup> Therefore, the only reliable marker for CMV infection is typical viral inclusion bodies,<sup>(15,16)</sup> thus making a diagnosis of GI tract CMV disease quite difficult. The number of reported AIDS cases is relatively low in Taiwan.<sup>(17)</sup> However, with the rapid increase in the incidence of the human immunodeficiency virus (HIV) since 1988, and rapidly growing numbers of organ transplants,<sup>(18)</sup> an increase in opportunistic infections such as cytomegalovirus should be expected in clinical practice. A few Asian reports about CMV disease over different sites of the GI tract have described individual case studies.<sup>(19,20)</sup> One report from Cheung et al. studied CMV infections of the GI tract of non-AIDS patients 10 years ago.<sup>(20)</sup> An important question is whether CMV is really still a rare disease in Asia. In this study, we attempted to locate patients who suffered from GI CMV disease, and compare and discuss their clinical and endoscopic features, with a view to continuing our work and helping such patients in the early stages of their illness.

## METHODS

In total, 1950 endoscopic biopsy reports from the Gastroenterology Division, Chang Gung Memorial Hospital, Kaohsiung for all GI tract lesions were retrospectively reviewed for CMV disease from January 1999 to December 2002. We restricted our enrollment criteria to the gold standard of diagnosis for GI CMV disease, i.e., the presence of typical viral intranuclear inclusion bodies further confirmed by special immunohistochemical staining methods.<sup>(1,6)</sup> At the same time, pathologists excluded other possible infections, such as fungi, other viruses, parasites, and bacteria before making a final diagnosis.

In total, 5 patients were enrolled (4 males and 1 female). The medical records of these 5 patients with GI CMV disease were also reviewed for their clinical background, laboratory data, treatment course, and clinical outcomes. The video recordings were reviewed, looking for any special characteristics of individual lesions and their locations and extents. All upper GI studies were performed by a free-hand technique using a forward-viewing electric endo-

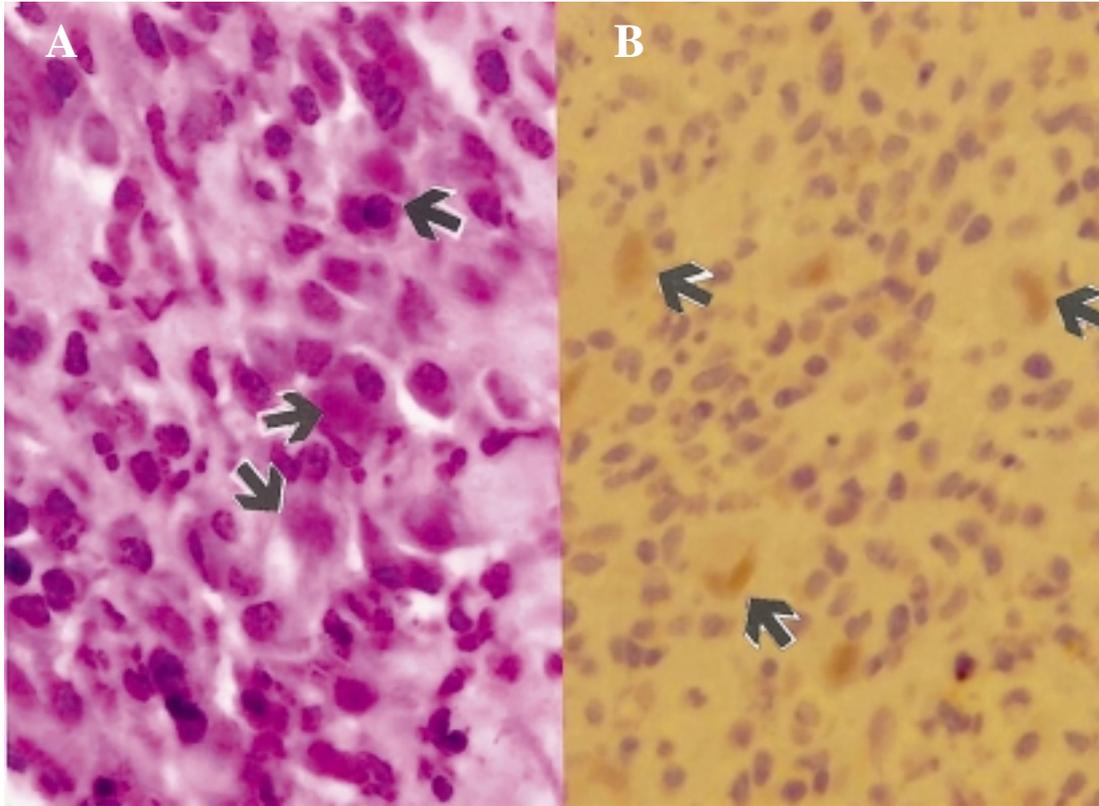
scope (Olympus GIF-XQ230 or XQ200). Lower GI tract studies were carried out by colonoscopy. After the patients were prepared by ingestion of an electrolyte lavage solution, an endoscopic examination was conducted using a CF200Z electronic videoendoscope (Olympus, Tokyo, Japan). Tissue specimens were obtained in multiple sessions by endoscopic snare biopsy forceps from the bases of abnormal mucosal lesions. These multiple biopsies obtained by the snare forceps technique were necessary to obtain sufficient tissue specimens. The diagnosis was confirmed by pathologists using hematoxylin and eosin (H&E) staining, and immunohistochemical staining with anti-CMV monoclonal antibody (Fig. 1A, B).

## RESULTS

Our review discovered 5 documented cases of GI CMV disease, with a male to female ratio of 4: 1. The mean age was  $59.2 \pm 22.5$  (range, 40~92) years. All 5 patients were immunocompromised at the time they were diagnosed with GI CMV disease. The mean serum albumin level was  $1.68 \pm 0.44$  (range, 1.2~2.4) g/dl, while the mean serum hemoglobin level was  $8.48 \pm 1.91$  (range, 6.1~10.1) g/dl. Three patients (60%) had positive urine or blood anti-CMV antibody assays (IgM).

Two male patients suffered from esophageal CMV disease (one had AIDS while the other had long been bedridden by a stroke and multiple organ failure). Only 1 instance of gastric CMV disease was observed in a male patient who had undergone long-term steroid treatment for systemic lupus erythematosus disease. Two CMV colitis patients, one suffering from prostate cancer with bone metastasis and the other multiple myeloma, were referred by a hematologist and an oncologist, respectively (Table 1).

Our youngest patient was a 40-year-old male AIDS patient receiving treatment at the Infectious Diseases Department. He had suffered from unrelieved sensations of heartburn and lower chest pain for 6 months, even after being treated with high dosages of omeprazole. The delay in conducting an endoscopic study was due to the patient's initial reluctance. The endoscopy picture showed prominent ulcers in the lower esophagus (Fig. 2A). The patient was treated with 250 mg ganciclovir administered intravenously every 12 h for 3 weeks, and was subse-



**Fig. 1** Intranuclear inclusion body of cytomegalovirus (CMV) in the gastrointestinal tract. (A) Hematoxylin and eosin-stained sample (arrow, original magnification, 400x). (B) Immunohistochemical staining with anti-CMV monoclonal antibody (arrow, original magnification, 100x).

**Table 1.** Patient Backgrounds and Outcomes of Treatment for Gastrointestinal Cytomegalovirus Disease

Organ involved	Underlying disease	Treatment course	Outcome
Esophagus (n = 2)	(1) AIDS	Ganciclovir x 3 weeks	good
	(2) Elderly stroke victim	Ganciclovir x 3 weeks	good
Stomach (n = 1)	(1) SLE	Ganciclovir x 3 weeks	good†
Colon (n = 2)	(1) Prostate cancer	Ganciclovir x 3 weeks	good
	(2) Multiple myelomas	Ganciclovir x 1 week	expired

**Abbreviations:** AIDS: acquired immunodeficiency syndrome; SLE: systemic lupus erythematosus.

† Death was caused by bacterial pneumonia 1 month after the gastric CMV was cured (the actual overall CMV-related mortality was 20%).

quently found to be free from symptoms. He has been prescribed triple anti-HIV viral medications regularly since then, and has been symptom-free for more than 1 year.

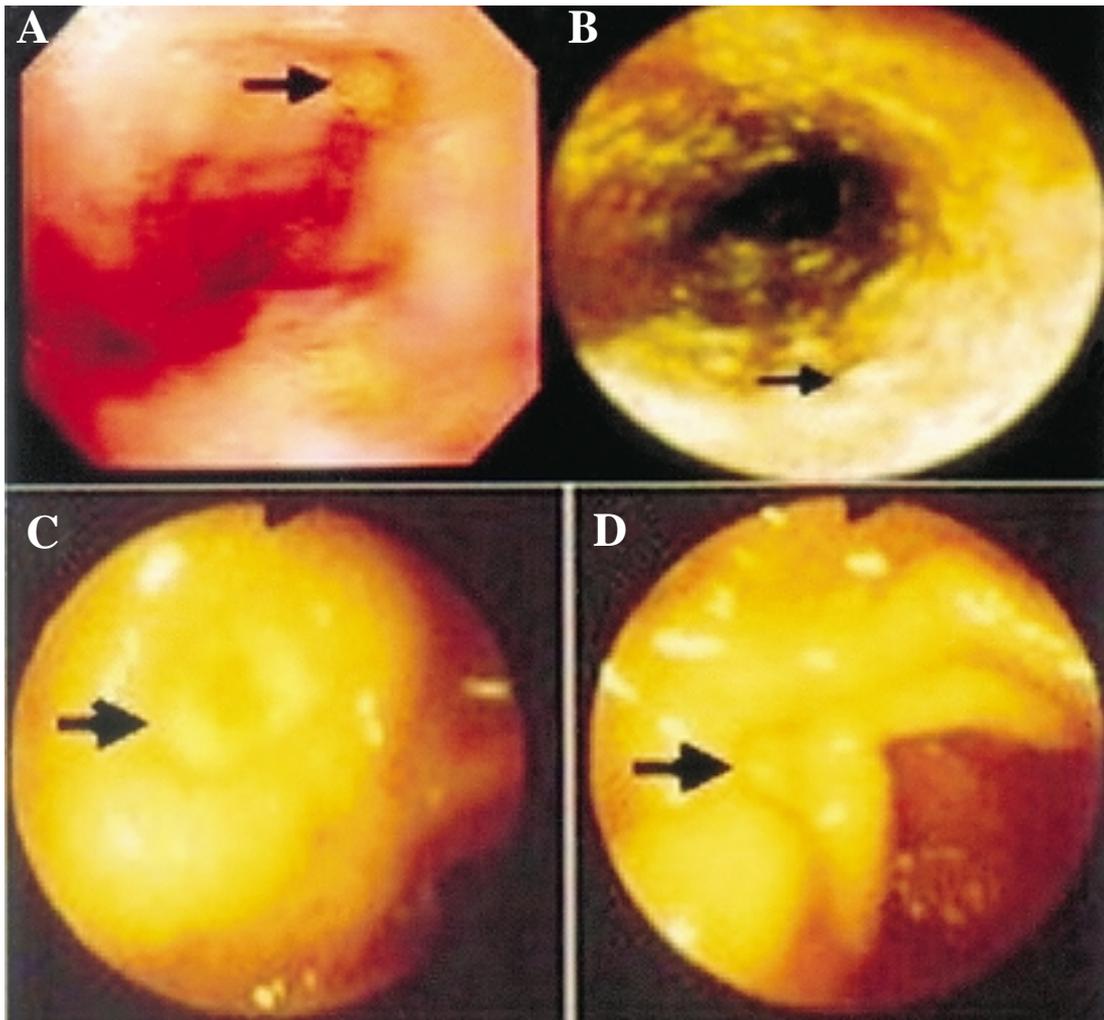
We also found a rare endoscopic presentation of esophageal CMV disease in a 75-year-old woman. She suffered from a stroke, diabetes mellitus, hypertension, and repeated infections of the urinary tract, and had been bedridden for many years. Endoscopy was performed due to the patient's severe vomiting, bleeding, and intractable chest pain. Diffuse and severe esophagitis with small ulcers were found over almost the entire esophagus (Fig. 2B). Initially, she was diagnosed as having an esophageal fungal infection, but sufficient biopsy tissue specimens allowed the pathologist to make a correct diagnosis. She responded well to a 3-week course of 250 mg ganciclovir administered intravenously every 12 h.

The only gastric CMV disease in our series was a 54-year-old male systemic lupus erythematosus

(SLE) patient with long-term steroid usage. He had suffered from epigastric pain for at least 2 months. We found 2 deep, round, ulcers, each about 1.0 cm in diameter at the antrum of the stomach (Fig. 2C, D). The patient was symptom-free after a 3-week course of intravenous ganciclovir treatment, but he died of pseudomonas-induced pneumonia 1.5 months later.

Two CMV colitis patients were diagnosed. A 93-year-old patient with prostate cancer was admitted to our oncology ward for a urinary tract infection. However, he began suffering from progressive bloody diarrhea 1 week after admission. A colonoscopy was planned, but the patient could only

tolerate a sigmoidoscopic examination. Marked inflammatory mucosal changes, with ulcerations of the rectosigmoid colon were found (Fig. 3A, B). CMV colitis was confirmed, and ganciclovir treatment was initiated, curing his viral disease. A 72-year-old female patient with multiple myelomas who received chemotherapy had episodes of bloody stools in the latter stage of her hospital course. A colonoscopic examination was performed when she finally gave her consent. Severe infection involving almost the entire colon, except for the cecum and rectum, was discovered, and was proven to be CMV colitis by biopsies. The endoscopic morphology showed



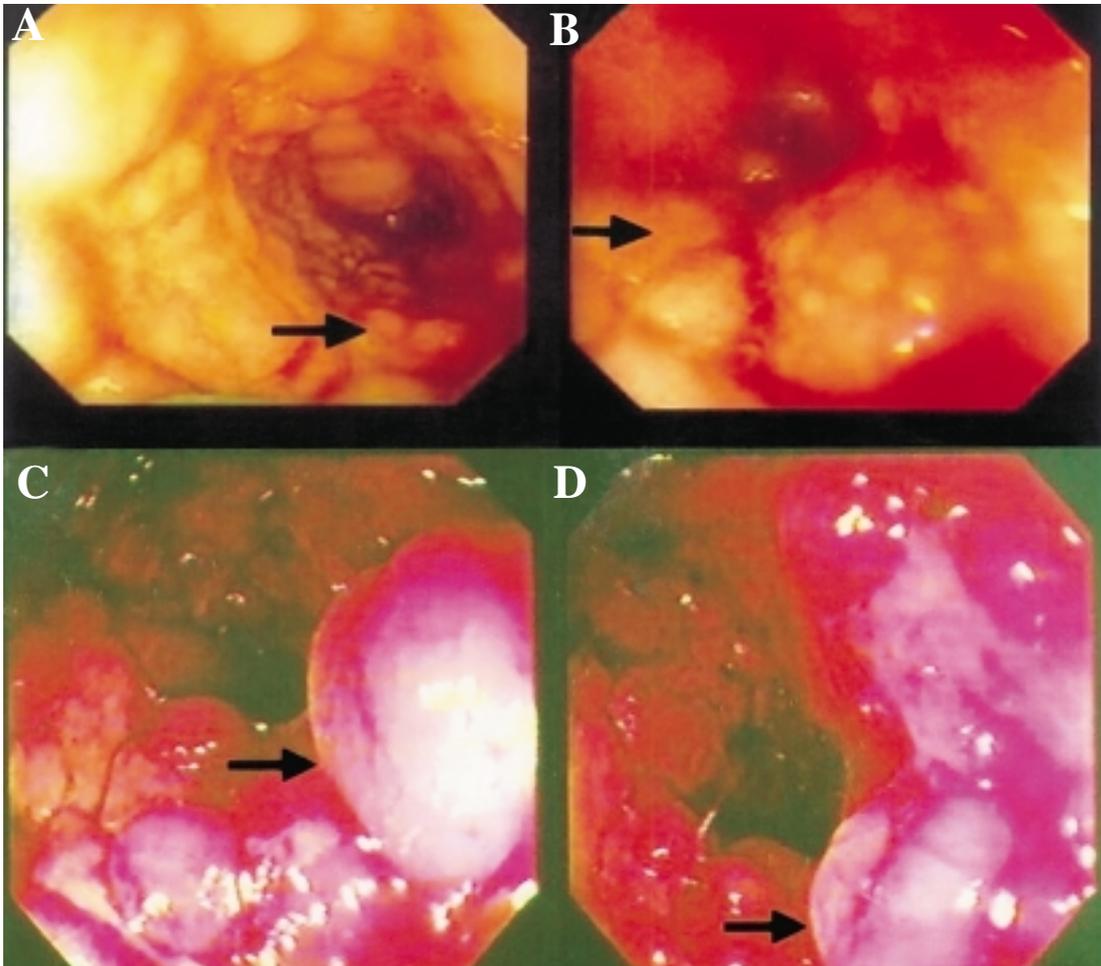
**Fig. 2** (A) Typical round ulcers of lower-esophagus cytomegalovirus (CMV) disease, each about 0.8 cm in size (arrow). (B). A rare feature of diffuse esophagitis with small CMV ulcers resembling lesions caused by herpetic and fungus infections. (C, D) Round and deep ulcers at the antrum of the infected stomach (arrow).

extensive nodular surfaces and ulcerations throughout the involved segments, resembling an exacerbation of ulcerative colitis, but with a rare presentation of pseudo-polyp formation at the hepatic flexure site (Fig. 3C, D). Mortality due to massive bleeding and sepsis occurred later, despite having received ganciclovir treatment.

## DISCUSSION

The principal risk factor in the development of CMV disease in these patients is immunosuppression and the presence of other opportunistic infections.<sup>(6,17,21)</sup> In Taiwan, HIV infection is becoming an increasingly more-serious public health problem.

Data show that more than 60% of these infected patients are in a serious condition when they are first diagnosed; that is, with a CD4<sup>+</sup> lymphocyte count below 50/mm<sup>3</sup>.<sup>(21)</sup> This was true for our only HIV patient suffering from esophageal CMV disease (who had a CD4<sup>+</sup> lymphocyte count of 34/mm<sup>3</sup>). In Taiwan, there is also an increase in the number of recipients of solid organ and bone marrow transplants, and of cancer patients receiving chemotherapy.<sup>(18)</sup> Therefore, it is very important to recognize the symptoms of opportunistic infectious diseases, such as oral and esophageal candidiasis, pneumocystis carinii pneumonia, tuberculosis, cytomegalovirus infection, and Kaposi sarcoma, and conduct careful histological reviews.



**Fig. 3** (A, B) Marked inflammatory changes with ulcerations over the infected sigmoid colon and rectum (arrow). (C, D) Colon severely infected with extensive nodular surfaces and ulcerations throughout the involved segments, resembling an exacerbation of ulcerative colitis, with rare presentations of pseudo-polyp formation at the hepatic flexure site (arrow).

CMV is a member of the herpes virus family.<sup>(1-3)</sup> It may be a new infection or the consequence of superimposed infections in previously damaged tissue.<sup>(1-3)</sup> CMV is highly prevalent in America, with seropositivity in the range of 53%~79% of adults.<sup>(22,23)</sup> However, serological markers of active CMV infection often do not appear. The only reliable marker of CMV infection is typical viral inclusion bodies<sup>(15,16)</sup> The target organs of CMV disease in immunodeficient adults are the lungs, adrenal glands, liver, and less commonly, the GI tract.<sup>(16)</sup> We also observed the rarity of GI tract CMV disease, with only 5 cases discovered for a 3-year period after reviewing 1950 endoscopic biopsies. GI tract CMV may be an underdiagnosed condition in immunocompromised populations. The main reason could be the difficulty in diagnosis due to inadequate biopsies which may account for the under-representation.

In our series, 4 of the 5 patients were senior citizens, with one 40-year-old male AIDS patient (with a low CD4<sup>+</sup> lymphocyte count when discovered). All of them were immunocompromised in different ways, as determined by their clinical backgrounds, low serum albumin levels (mean value of  $1.68 \pm 0.44$  g/l), and anemia (mean serum hemoglobin level of  $8.48 \pm 1.91$  g/dl) when diagnosed with GI CMV disease. A poor host immune background is the most-important factor accounting for GI CMV disease.

It appeared that the 2 patients with lower GI tract involvement were more seriously affected, one of whom bled to death (Table 1). However, these 2 patients were 72 and 93 years old, and the number of cases studied was too small to form a hypothesis. Interestingly, we observed 2 rare morphologies of GI CMV disease. One infected the esophagus and the other involved almost the entire colon.

We treated 2 cases of esophageal CMV disease. To our surprise, only 1 patient was HIV positive. A previous report identified these lesions as large, solitary, and situated in the distal esophagus but most lesions presented as multiple, well-circumscribed, shallow ulcerations larger than 1 cm located in the middle to distal section of the esophagus.<sup>(7)</sup> Our AIDS patient had typical ulcers in the lower esophagus (Fig. 2A). This patient has had no relapse 1 year after a course of ganciclovir treatment, which may also be due to the effective control of his underlying disease with anti-HIV agents.<sup>(24)</sup>

A rare presentation of esophageal CMV disease was observed in the other elderly, bedridden patient. The disease more-closely resembled an esophageal fungal infection and herpes-viral infections than CMV disease. Diffuse and severe esophagitis with minute ulcers and a whitish coating on the mucosa were found over almost the entire esophagus (Fig. 2B). The patient responded well to a 3-week course of intravenous ganciclovir treatment.

The colon is the most-common portion of the GI tract infected by CMV, especially in patients with AIDS.<sup>(2-4,6)</sup> The typical morphology may present as large, giant ulcers, predominantly of the right colon. There can also be multiple but smaller ulcers with colonic fold thickening which blurs or eliminates the mucosal vascular pattern, usually involving the right, but occasionally the entire, colon.<sup>(25)</sup> However, diffuse mucosal lesions with pseudo-polyps are less-commonly observed.<sup>(26)</sup> One of our 2 colitis patients suffered from a rare severe infection involving almost the entire colon except the cecum and rectum. The endoscopic morphology showed extensive nodular surfaces and ulcerations throughout the involved segments (Fig. 3C, D). The disease severity, and its rare morphology led to an unfortunate delay in diagnosis, and the patient bled to death.

The gastric morphology of the 54-year-old male SLE patient was typically deep, round ulcers about 1.0 cm in diameter over the antrum of the stomach, as has been reported in the literature.<sup>(3,19)</sup>

Ganciclovir is the treatment of choice for dealing with GI CMV disease, with a recommended 250 mg of ganciclovir administered intravenously every 12 h for at least 3 weeks.<sup>(27-30)</sup> Prophylaxis with oral ganciclovir has been studied for use by HIV-infected adults who are CMV-seropositive with low CD4<sup>+</sup> T lymphocyte count of  $< 50$  cells/mm<sup>3</sup>.<sup>(21)</sup> However, concerns about the side effects of neutropenia and anemia, conflicting reports of its efficacy, a lack of proven survival benefits, risks of experiencing ganciclovir-resistant CMV, and costs should be addressed.<sup>(21)</sup> The other alternative is valacyclovir, but an unexplained increase in death rates has been reported with its use.<sup>(31)</sup> CMV ulcers of the esophagus respond particularly well to foscarnet, a pyrophosphate derivative, with a rapid resolution of clinical symptoms and healing of the ulceration, and it could be an alternative medication if ganciclovir fails.<sup>(32)</sup> However, foscarnet is virostatic, and reactivation of

CMV could be expected in an immunosuppressed host even after a complete course of therapy.

CMV disease is not considered cured regardless of whatever courses of available antiviral agents are used. Maintenance therapy is still recommended in cases of rapid relapse of CMV esophageal ulceration after the initial treatment. Discontinuing maintenance therapy should be done with care among HIV-infected patients unless sustained CD4<sup>+</sup> T lymphocyte counts > 100~150 cells/mm<sup>3</sup> in response to HAART are achieved.<sup>(33,34)</sup> However, reinstitution of secondary prophylaxis should be given when the CD4<sup>+</sup> T lymphocyte count drops to < 100~150 cells/mm<sup>3</sup>.<sup>(35)</sup>

In conclusion, GI CMV disease is still rare, but an increase in the prevalence can be expected in the near future. Therefore, a high degree of suspicion is mandatory for any physician treating a patient from among these immunosuppressed populations who is suffering from GI disorders. Endoscopy and tissue biopsy confirmation of the specific inclusion body by a pathologist still comprise the gold standard of diagnosis.

## REFERENCES

1. Goodgame RW. Gastrointestinal cytomegalovirus disease. *Ann Int Med* 1993;119:924-35.
2. Chetty R, Roskell DE. Cytomegalovirus infection in the gastrointestinal tract. *J Clin Pathol* 1994;47:968-72.
3. Patra S, Samal SC, Chacko A, Mathan VI, Mathan MM. Cytomegalovirus infection of the human gastrointestinal tract. *J Gastroenterol Hepatol* 1999;14:973-6.
4. Tyms AS, Taylor DL, Parkin JM. Cytomegalovirus and the acquired immunodeficiency syndrome. *J Antimicrob Chemother* 1989;23:89-105.
5. Klatt EC, Shibata D. Cytomegalovirus infection in the acquired immunodeficiency syndrome. *Arch Pathol Lab Med* 1988;112:540-4.
6. Jacobson MA, Mills JCM. Serious cytomegalovirus disease in the acquired immunodeficiency syndrome (AIDS). *Ann Intern Med* 1988;108:585-94.
7. Wilcox CM, Straub RF, Schwartz DA. Prospective endoscopic characterization of cytomegalovirus esophagitis in AIDS. *Gastrointest Endosc* 1994;40:481.
8. Orton DI, Orteu CH, Rustin MH. Cytomegalovirus-associated gastric ulcer in an immunosuppressed patient with pemphigus vulgaris. *Clin Exp Derm* 2001;26:170-2.
9. Monkemuller KE, Wilcox CM. Esophageal ulcer caused by cytomegalovirus: resolution during combination antiretroviral therapy for acquired immunodeficiency syndrome. *South Med J* 2000;93:818-20.
10. Wilcox CM, Staub RF, Clark WS. Prospective evaluation of oropharyngeal findings in human immunodeficiency virus-infected patients with esophageal ulceration. *Am J Gastroenterol* 1995;90:1938-41.
11. Wilcox CM, Zaki SR, Coffield LM, Greer PW, Schwartz DA. Evaluation of idiopathic esophageal ulceration for human immunodeficiency virus. *Modern Pathol* 1995;8:568-72.
12. Poles MA, McMeeking AA, Scholes JV, Dieterich DT. Actinomyces infection of a cytomegalovirus esophageal ulcer in two patients with acquired immunodeficiency syndrome. *Am J Gastroenterol* 1994;89:1569-72.
13. Murray RN, Parker A, Kadakia SC, Ayala E, Martinez EM. Cytomegalovirus in upper gastrointestinal ulcers. *J Clin Gastroenterol* 1994;19:198-201.
14. Buckner FS, Pomeroy C. Cytomegalovirus disease of the gastrointestinal tract in patients without AIDS. *Clin Inf Dis* 1993;17:644-56.
15. 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;23:1019-22.
16. Henson D. Cytomegalovirus inclusion bodies in the gastrointestinal tract. *Arch Pathol* 1972;93:477-82.
17. Hung CC, Chen MY. HIV infection and AIDS in Taiwan: epidemiology, clinical spectrum and management. *Gastroenterol J Taiwan* 2001;18:22.
18. Chen CL, de Villa VH. Split liver transplantation. *Asian J Surg* 2002;25:285-90.
19. Suzuki M, Ochi Y, Hosokawa S, Uemura N, Hosoi K, Kuniyoshi N, Inoue S, Matsuda M, Kishi S, Matsuoka R. A multiple gastric ulcer case caused by cytomegalovirus infection. *Tokushima J Exp Med* 1996;43:173-6.
20. Cheung AN, Ng IO. Cytomegalovirus infection of the gastrointestinal tract in non-AIDS patients. *Am J Gastroenterol* 1993;88:1882-6.
21. 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 Int Med* 2002;137:435-78.
22. Tendero DT. Laboratory diagnosis of cytomegalovirus (CMV) infections in immunodepressed patients, mainly in patients with AIDS. *Clin Lab* 2001;47:169-83.
23. Dorigo-Zetsma JW, Van der Meer JT, Tersmette M, ten Kate FJ, Wertheim-van Dillen PM, van der Noordaa J. Value of laboratory investigations in clinical suspicion of cytomegalovirus-induced upper gastrointestinal tract ulcerations in HIV-infected patients. *J Med Virol* 1996;49:29-33.
24. Monkemuller KE, Wilcox CM. Esophageal ulcer caused by cytomegalovirus: resolution during combination antiretroviral therapy for acquired immunodeficiency syndrome. *South Med J* 2000;93:818-20.
25. Rene E, Marche C, Chevalier T, Rouzioux C, Regnier B,

- Saimot AG, Negesse Y, Matheron S, Leport C, Wolff B. Cytomegalovirus colitis in patients with acquired immunodeficiency syndrome. *Dig Dis Sci* 1988;33:741-50.
26. Freeman HJ, Shnikta TK, Piercy JR, Weinstein WM. Cytomegalovirus infection of the gastrointestinal tract in a patient with late onset immunodeficiency syndrome. *Gastroenterology* 1977;73:1397-403.
  27. 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(Suppl3):S532-7.
  28. 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(Suppl3):S495-504.
  29. Spector SA, McKinley GF, Lalezari JP, Samo T, Andruczk R, Follansbee S, Sparti PD, Havlir DV, Simpson G, Buhles W, Wong R, Stempien M. 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.
  30. Brosgart CL, Louis TA, Hillman DW, Craig CP, Alston B, Fisher E, Abrams DI, Luskin-Hawk RL, Sampson JH, Ward DJ, Thompson MA, Torres RA. A randomized, placebo-controlled trial of the safety and efficacy of oral ganciclovir for prophylaxis of cytomegalovirus disease in HIV-infected individuals. Terry Bein Community Programs for Clinical Research on AIDS. *AIDS* 1998;12:269-77.
  31. Feinberg JE, Hurwitz S, Cooper D, Sattler FR, MacGregor RR, Powderly W, Holland GN, Griffiths PD, Pollard RB, Youle M, Gill MJ, Holland FJ, Power ME, Owens S, Coakley D, Fry J, Jacobson M. A randomized, double-blinded trial of valaciclovir prophylaxis for cytomegalovirus disease in patients with advanced human immunodeficiency virus infection. AIDS Clinical Trials Group Protocol 204/Glaxo Wellcome 123-014 International CMV Prophylaxis Study Group. *J Infect Dis* 1998;177:48-56.
  32. Nelson R. Foscarnet in treatment of cytomegalovirus infection of the esophagus and colon in patients with acquired immune deficiency syndrome. *Am J Gastroenterol* 1991;86:876-81.
  33. Vrabec TR, Baldassano VF, Whitcup SM. Discontinuation of maintenance therapy in patients with quiescent cytomegalovirus retinitis and elevated CD4<sup>+</sup> counts. *Ophthalmology* 1998;105:1259-64.
  34. 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.
  35. Jouan M, Saves M, Tubiana R, Carcelain G, Cassoux N, Aubron-Olivier C, Fillet AM, Nciri M, Senechal B, Chene G, Tural C, Lasry S, Autran B, Katlama C. RESTIMOP study team. Discontinuation of maintenance therapy for cytomegalovirus retinitis in HIV-infected patients receiving highly active antiretroviral therapy. *AIDS* 2001;15:23-31.

## 南台灣巨細胞病毒引起的消化道疾病之臨床經驗： 1950 件腸胃道病灶之組織切片所見

蔡成枝 張簡吉幸 郭仲謀 吳耿良 趙景華 邱逸群 劉建衛<sup>1</sup> 邢福柳<sup>2</sup> 邱世賢

**背景：** 文獻報告中，巨細胞病毒引起的消化道疾病相當少見。但隨著免疫力低下的病患如愛滋病，癌症以及器官移植日益增加，巨細胞病毒感染的增加是可預期的，但少見的消化道感染是否也一樣呢？本研究乃討論這些病患的臨床表現，尤其是內視鏡所見，以及其治療之經驗。

**方法：** 1999 年一月至 2002 年十二月期間，我們配合有消化道疾病症狀的病患接受內視鏡檢查，發現腸胃道病灶之組織切片共 1950 件。臨床診斷必須在病理組織裡找到典型的巨大細胞聚合體 (cytomegalic inclusion body)，並經特殊免疫染色法確認且須排除其他感染的可能如其他病毒，黴菌，寄生蟲，或細菌感染。

**結果：** 我們只發現 5 例消化道受感染的病患，食道和大腸各 2 例，胃部 1 例。上消化道受感染的病患臨床表現似乎比大腸受感染病患輕。其中一例食道受感染的內視鏡所見相當罕見。一例幾乎整段大腸嚴重感染者因延誤而死亡，死亡率為 20%，其他四位病患皆對 ganciclovir 有很好的療效。

**結論：** 我們強調消化道之巨細胞病毒疾病仍然少見，因此，對於有消化道症狀的病患接受內視鏡健查，尤其是免疫力低下的病患如愛滋病，癌症以及器官移植者，提高警覺並針對腸胃道病灶做組織切片檢查是必要的。  
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**關鍵字：** 消化道巨細胞病毒疾病，巨大細胞聚合體。

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長庚紀念醫院 高雄院區 內科部胃腸肝膽系，<sup>1</sup>感染科，<sup>2</sup>病理科

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索取抽印本處：邱世賢醫師，長庚紀念醫院 胃腸肝膽系。高雄縣833鳥松鄉大埤路123號。Tel: (07) 7317123轉 8301; Fax: (07) 7322402; E-mail: kyutarou@hotmail.com